Hyperimitation of Actions Is Related to Reduced Understanding of Others' Minds in Autism Spectrum Conditions

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Background: Anecdotal evidence has noted that individuals with autism spectrum conditions (ASC) frequently exhibit heightened spontaneous imitative behavior, with symptoms of echolalia and echopraxia. This is contrasted by empiric reports that ASC results in decreased imitation and an underlying deficit in the mirror system, leading to impaired social understanding. Thus, it remains unclear whether automatic imitation is enhanced in ASC and how this is related to poorer social abilities.

Methods: This study investigated spontaneous imitation in 18 high-functioning adults with ASC and 18 age- and IQ-matched control participants during a simple imitation inhibition task. Mentalizing was experimentally assessed in the same participants using both behavioral and functional magnetic resonance imaging measures, as was social interaction using an observational measure.

Results: Individuals with ASC showed increased imitation of hand actions compared with control participants and this was associated with reduced mentalizing and poorer reciprocal social interaction abilities. In the functional magnetic resonance imaging mentalizing paradigm, ASC participants with increased imitation scores showed less brain activation in areas often found to be active in mental state attribution, namely the medial prefrontal cortex and temporoparietal junction.

Conclusions: The results confirm the presence of hyperimitation in ASC, which is accompanied by reduced social cognition, suggesting that a general imitation impairment and a global mirror system deficit are absent. These findings offer an explanation for echopractic features based on theories of atypical functioning of top-down modulation processes in autism.

Key Words: Action, autism, fMRI, imitation, mirror neuron system, social cognition

Autism spectrum conditions (ASC) are neurodevelopmental disorders with a heritability rate of over 90% (1), which are characterized by abnormalities of social interaction, impairments in communication, and a restricted repertoire of interests and activities (2). A number of empiric studies have shown that children and adults with ASC perform poorly in a variety of imitation tasks, i.e., showing less or faulty copying of the observed movements (see [3] for a review). However, two frequently reported clinical features of autism are incompatible with an imitation deficit in ASC and rather indicate problems with increased imitation and imitation inhibition. Behaviors and speech may be involuntarily copied from others, including speech patterns (echolalia) and observed actions, which may be imitated without regarding the context and the meaning of this action (echopraxia) (3–5).

Such hyperimitation of actions may rely on the mirror system (MS), a set of cortical regions in the inferior frontal gyrus and inferior parietal lobe that are thought to provide the observer automatically with a matching motor representation in one's own motor system (6,7). Action mirroring is assumed to underlie imitation of observed actions and social understanding (8). In line with this idea, a prominent theory (9) has suggested that children with autism have an abnormal MS and that this deficit causes the often observed weaknesses in imitation performance (3), poor theory of mind (ToM) skills (10), and impaired social cognition in autism. Evidence for an atypical MS response in ASC comes from neuroimaging studies that have shown less activation of MS regions during the imitation of movements (11,12). Theoret *et al.* (13) observed in individuals with autism less transcranial magnetic stimulation-induced motor-cortical excitability when watching self-hand actions. Using electroencephalography, children with ASC showed less μ-wave suppression during the observation of another person's actions, which has been linked to MS functions (14). These results of a disturbed MS in ASC would thus suggest that ASC individuals show poorer imitation skills as a result of a functional mirroring deficit, i.e., the ability to automatically match observed behavior onto one's own motor representations. At a first glance, the evidence supporting this hypothesis seems to be at odds with the clinical features of enhanced imitation in ASC, as these observational reports would predict equal or increased activation of the MS compared with control subjects.

However, on closer inspection, studies of MS function in ASC have yielded rather inconsistent findings. Avikainen *et al.* (15) studied motor cortex excitability using magnetoencephalography and found no difference in activity between ASC and control participants when observing simple hand movements. Also, different studies have localized the MS deficit in ASC to different neurological areas. Dapretto *et al.* (11) found that individuals with ASC show normal activity in the parietal area but reduced activity in the inferior frontal gyrus, whereas Williams *et al.* (12) reported the opposite pattern of results. Several electroencephalography studies in children and adolescents have found no differences in MS functioning, measured by μ-wave suppression, between control subjects and an ASC group (16–18). Additionally, recent neuroimaging evidence confirmed also that individuals with ASC exhibited normal activity in the MS using a functional magnetic resonance imaging (fMRI) repetition suppression design (19). Interestingly, one fMRI study even revealed a hyperactivity of the MS in ASC during the observation of hand actions (20).

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Received Jul 14, 2010; revised Aug 27, 2010; accepted Sep 3, 2010.

0006-3223/$36.00
doi:10.1016/j.biopsych.2010.09.017

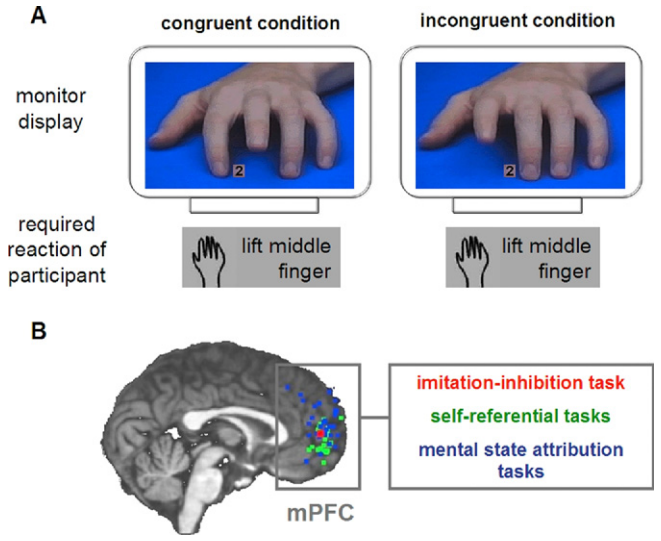

Figure 1. (A) During the imitation-inhibition task, participants had to lift their index or middle finger in response to a number (1: index finger, 2: middle finger) while watching video sequences of a hand that mirrored the hand of the subjects. The task consisted of two different conditions. In the congruent condition, the corresponding finger on the screen was lifted simultaneously with presentation of the digit (e.g., the index finger was lifted when 1 was presented). In the incongruent condition, the noncorresponding finger was lifted (e.g., the middle finger was lifted when 1 was presented). Displayed are two example stimuli for both conditions and the required response of the participant. **(B)** Meta-analysis of functional neuroimaging studies. The imitation-inhibition task (red dot) activates a similar region in the medial prefrontal cortex, as self-referential (green) and mentalizing tasks (blue points). (Reproduced with permission from Brass *et al.* [55].) mPFC, medial prefrontal cortex.

Furthermore, recent behavioral findings do not support the idea of a dysfunctional MS in ASC. This was indicated by intact automatic imitation in ASC (21,22), where an adult ASC group showed as much of a tendency to engage in spontaneous and uninstructed imitation as a matched control group. Similarly, Sebanz *et al.* (23) found no difference in children with ASC to automatically represent others' action in a joint action paradigm. Additionally, in intentional imitation tasks, both typical and autistic children had the same tendency to imitate an adult's goals (24). These results speak against a global failure in the mirror system in ASC and rather suggest that previous reports about deficits in more complex, voluntary imitation tasks are due to nonspecific, task-general impairments (25,26).

Based on these findings, which compromise the idea that the MS is generally broken in autism, and on the clinical observations of excessive mirroring of actions, an alternative hypothesis would suggest that the MS (and functions supported by this system, such as imitative behavior) is not deficient but rather that the control or top-down modulation of this system is impaired (27). This view would predict that autistic patients should have problems in the control of imitative behavior, rather than in imitation per se (28). Support for this account comes from investigations in typical populations and neurological patients using an experimental paradigm known as the imitation-inhibition task (Figure 1A). In this task, the observation of a movement while performing another opposite movement leads to an interference effect, by slowing down reaction times in incongruent compared with congruent trials, in which the perceived movement corresponds to the executed movement (29). Previous research has suggested that mechanisms needed to control such automatic imitation, induced by unintentional mirroring of observed actions, relies on specific processes dedicated to distinguish one's own action from someone else's action and to attribute intentional mental states (28,30). These latter social cognitive functions are consistently associated with increased activation in the medial prefrontal cortex (mPFC) and the temporoparietal junction (TPJ), the same regions that are also activated in the imitation-inhibition task (Figure 1B). In line with this idea, previous studies in ASC reported consistently impairments of mentalizing (31) and also weaker activations of typical ToM areas (e.g., [32]). Furthermore, support for this idea for an involvement of social cognitive functions in the control of imitation was found in a lesion study (33), in which poorer imitative control was associated with decreased mentalizing and perspective-taking abilities.

Our present study, hence, aimed to provide a more stringent test for hyperimitation of actions in ASC from clinical observations and its relationship to social cognitive abilities by using established experimental paradigms. Eighteen high-functional adults with ASC and 18 nonautistic, matched control participants completed the imitation-inhibition task, a story-based ToM task (34), and a second, animation-based ToM task (32) in the fMRI scanner. Specifically, our hypothesis of increased hyperimitation of actions in ASC and the relationship with social cognitive functions was twofold. It predicted, first, enhanced automatic imitation effects in ASC, expressed as an increased mean interference effect. Our imitation-inhibition paradigm is highly sensitive to produce interference effects, as it is a choice reaction-time task, compared with previous studies using a single-response task, which found, so far, only an equivalent interference effect in the ASC group and control subjects (21,22). Second, we predicted that in ASC an increased interference effect in the imitation-inhibition task, indexing a reduced ability to control automatic mirroring functions, should be related with decreased behavioral performance in the behavioral mentalizing task. This would support the idea that the control of MS functioning, rather than MS functioning itself, is associated with social cognitive abilities. In contrast, the alternative theoretical account by Williams *et al.* (9) would predict the reverse pattern of correlations, i.e., lower interference effect in the imitation-inhibition task, caused by a deficient MS, should be related to poorer mentalizing abilities. Concerning the mentalizing fMRI study, it was further hypothesized that an increased interference effect in the imitation-inhibition task will be associated with reduced neural activity in typical mentalizing regions, such as the mPFC and the TPJ. We further sought to support this by performing a meta-analysis on mentalizing activations to compare those with the obtained activations of the present fMRI study.

Methods and Materials

Participants

Eighteen participants with ASC and 18 matched control participants took part in this study (Table 1). All participants in the ASC group had previously received a diagnosis of autism or Asperger's syndrome from an independent clinician according to standard

Table 1. Descriptive Characteristics of the Control and Autism Spectrum Conditions Group

Group	*n*	Age[a]	Gender	FSIQ[a]
ASC	18	35.6 (12.4)	12 M/6 F	109.5 (19.2)
Control	18	33.0 (10.7)	12 M/6 F	110.0 (14.0)
Difference		$p = .5$	$p = 1.0$	$p = .9$

ASC, autism spectrum conditions; F, female; FSIQ, full-scale intelligence quotient; M, male.

[a]Mean (SD).

criteria (DSM-IV [2]). In addition, the Autism Diagnostic Observational Schedule-Generic (35) was used to characterize the participants and to obtain information about social interaction abilities using an observational measure. On this test, seven participants met the criteria for autism, while eight participants met the criteria for autism spectrum disorder. We were unable to classify three participants, as they had not been assessed with the Autism Diagnostic Observation Schedule (ADOS). Analyses excluding these individuals did not substantially change the results; thus, we elected to report the analyses including these patients, so the findings are based on all available data.

General Procedure

All participants gave their informed consent to participate in the study, which was approved by the local ethics committee and conducted in accordance with established ethical standards (1964 Declaration of Helsinki).

Behavioral Tasks and Questionnaires

Imitation-Inhibition Task. Every video sequence started with a frame showing the hand (without number) in a resting position (2000 msec), two consecutive frames (each lasting 34 msec) with the number and the finger movement of the videotaped hand, and then a picture (1500 msec) showing the number and the finger in the end position (Figure 1A). Between trials, a blank screen was presented for 2000 msec. Participants were instructed to immediately react to the presentation of the number (1: index finger lifting movement; 2: middle finger movement). The concurrently displayed hand actions could be either congruent or incongruent with respect to the number. Reaction times were recorded with a custom-built response device using light sensors. The imitation-inhibition task started with a 20-trial practice phase. Participants were instructed to give a quick and correct response. The conditions were presented randomly (50 trials each).

Theory of Mind Task: Strange Stories. Mentalizing was tested with 24 short stories, 8 of them requiring the comprehension of a mental state (ToM condition) (36). The physical and jumbled condition (eight stories each) were control conditions designed to control for making inferences, working memory demands, and story comprehension. Stories were presented blocked and in counterbalanced order. Participants read through the stories and when they felt that they understood it, turned over the sheet to reveal a question. Reaction time of the subjects (time for reading the story until the beginning of the answer) and verbal responses were recorded and rated (36). Answers were scored with two points if they gave a full account, one point for a partial response, and no points for incorrect answers.

fMRI Task

Mentalizing was evoked using the animations paradigm with eight silent videos (32,37). Four mentalizing videos (two triangles acting like humans) and four control videos (triangles moving randomly) were presented twice. Before each fMRI experiment, subjects were told to watch the animations and think about what the triangles were doing and thinking and they were shown two practice animations. After each video, the subjects were asked to describe "what was happening in this animation?" To adapt the length of the videos for an fMRI study, all videos were shortened to 20 sec. The stimuli were randomly presented in a block design. Additionally, four null epochs (40 sec) were presented (task length was 17 min).

fMRI Data Acquisition

The fMRI brain images were acquired with a 1.5 Tesla system (Siemens Sonata, Erlangen, Germany). Functional whole brain data were obtained using a T2* echo-planar sequence sensitive to blood oxygenation level-dependent contrast (44 slices, 2.5 mm thickness, gap 1.5 mm, echo time 90 msec, repetition time 3.96 sec per volume). Structural images were acquired with a T1 sequence using a phased-array head coil. Slices were angled in an oblique orientation 5° to the anterior-posterior commissure line.

fMRI Data Analysis

The fMRI data were analyzed using SPM2 (Wellcome Department of Imaging Neuroscience, London, United Kingdom). In a first preprocessing step, functional images were realigned to the first volume to account for motion artefacts and then spatially normalized to a standard template (Montreal Neurological Institute) with a resampled voxel size of $3 \times 3 \times 3$ mm. Afterward, images were smoothed with 9 mm full-width at half maximum. After preprocessing, functional images were analyzed using a box car function block design. Each stimulus category (mentalizing, random) was modeled as a separate regressor and used to derive contrast images for the correlation analyses. To investigate whether neural activity during mentalizing correlated with the participants' behavioral results in the imitation-inhibition task (interference score for reaction times [RTs]/errors), we correlated these scores with activity evoked by the contrast of the mentalizing condition minus the control condition. For regression analyses, a threshold of $p < .001$, uncorrected, with a cluster extent of ≥ 5 voxels was used.

Meta-Analytic Procedure

This study included the mPFC region of the regression analysis from the present neuroimaging study and activation peaks of neuroimaging studies on mentalizing from the literature. A method was employed (activation likelihood estimation [38]) that allows computing a statistical map for each voxel in the brain with an estimation likelihood of activation using activation peaks from previous studies. In a second step, it is then possible to overlay these two maps to test for overlap of brain regions (Supplement 1).

Statistical Analysis of the Behavioral Data

All statistical analyses were performed with SPSS (Version 15.0; SPSS Inc., Chicago, Illinois). None of the variables showed a significant difference from a normal distribution ($p > .05$); therefore, we used parametric tests. A Bonferroni correction was applied to adjust the significance level, to account for multiple comparisons (Supplement 1). For the ToM test, we computed four new variables. To control for the performance in the two control conditions in this task, the residuals for the accuracy score and RTs in the ToM condition were computed, after removing the variance of the performance in the control conditions, subsequently being referred to as ToM-jumbled and ToM-physical conditions.

Results

Behavioral Results

Differences in the Control of Automatic Imitation Between the ASC and Control Group. As expected, the interference effect (incongruent − congruent trials) in the imitation-inhibition task was significantly larger in the ASC group compared with the control subjects [$t(34) = 2.2, p = .034$] for errors (Figure 2A). Similarly, in the RTs, ASC participants showed a tendency to an enlarged interference effect [$t(34) = 1.8, p = .079$] (Figure 2B). Planned t tests indicated that this interference difference in errors was due to more errors in the ASC group in incongruent trials [$t(34) = 2.1, p = .036$] in contrast to no difference on congruent trials [$t(34) = .7, p > .4$].

Relationship Between Imitative Control and Social Cognition. We further inspected correlations with measures of social cognition

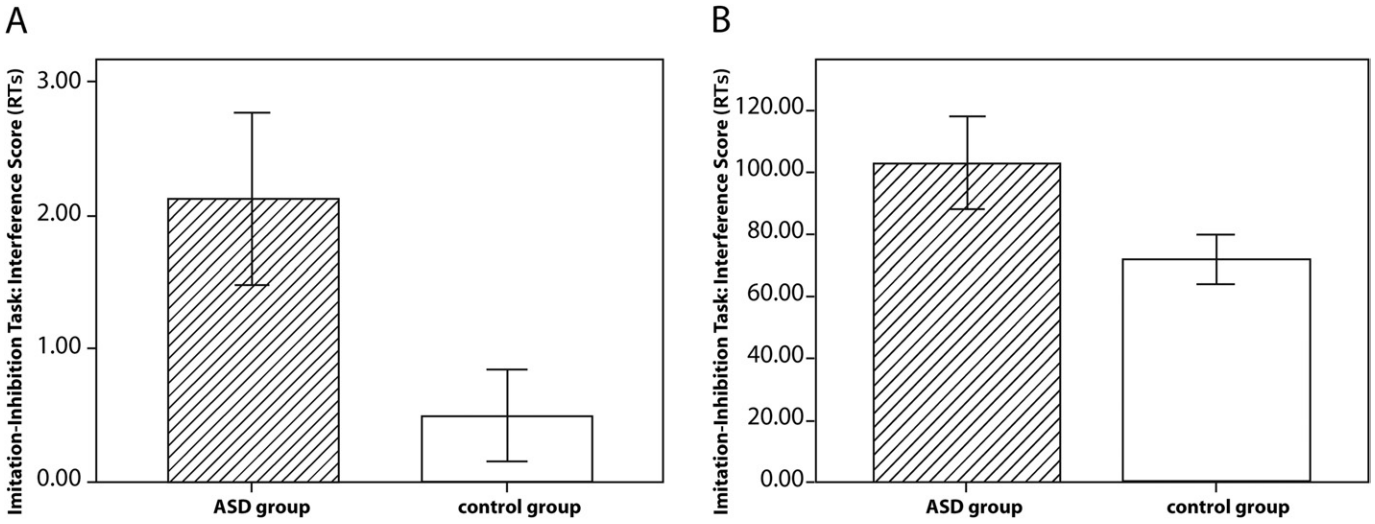

Figure 2. (A) Mean interference effect for errors (incongruent minus congruent trials) for the autism spectrum conditions and control group. **(B)** Mean interference effect for reaction times (incongruent minus congruent trials) for the autism spectrum conditions and control group. ASD, autism spectrum disorders.

and the imitation-inhibition task in the ASC group. In the advanced ToM task, a significant correlation was found between the interference score errors and the RTs in the ToM-jumbled condition [$r(15) = .67$, $p < .05$, Bonferroni corrected] (Figure 3A). The correlations ToM-physical condition [$r(15) = .48$, $p < .1$, uncorrected] and ToM RTs [$r(15) = .43$, $p < .1$, uncorrected] with the interference score errors also showed a trend toward statistical significance. When looking again at the separate conditions in the imitation-inhibition task, correlations were only found between the incongruent condition and ToM-jumbled [$r(15) = .65$, $p < .05$, Bonferroni corrected] (Figure 3B).

To assess the possible moderating effect of additional variables, such as age and IQ, on the foregoing, significant correlations between the imitation-inhibition task and the ToM task, partial correlations were computed. After accounting for the variance caused by these two variables, significant correlations were found selectively for the incongruent condition and the interference score errors with the ToM-jumbled variable [incongruent: $r(15) = .58$, $p < .05$, interference: $r(15) = .61$, $p < .05$]. No significant correlations were found in the control group.

However, it is important to ascertain that these bivariate correlations are not just due to outliers, which may artificially inflate a correlation. Two commonly used procedures (Cook's distance metric: cutoff > 1, and leverage values: cutoff $> .5$) were used. None of the data points of the above-reported correlations showed a value greater than .7 (Cook's distance) and .4 (leverage value) and most

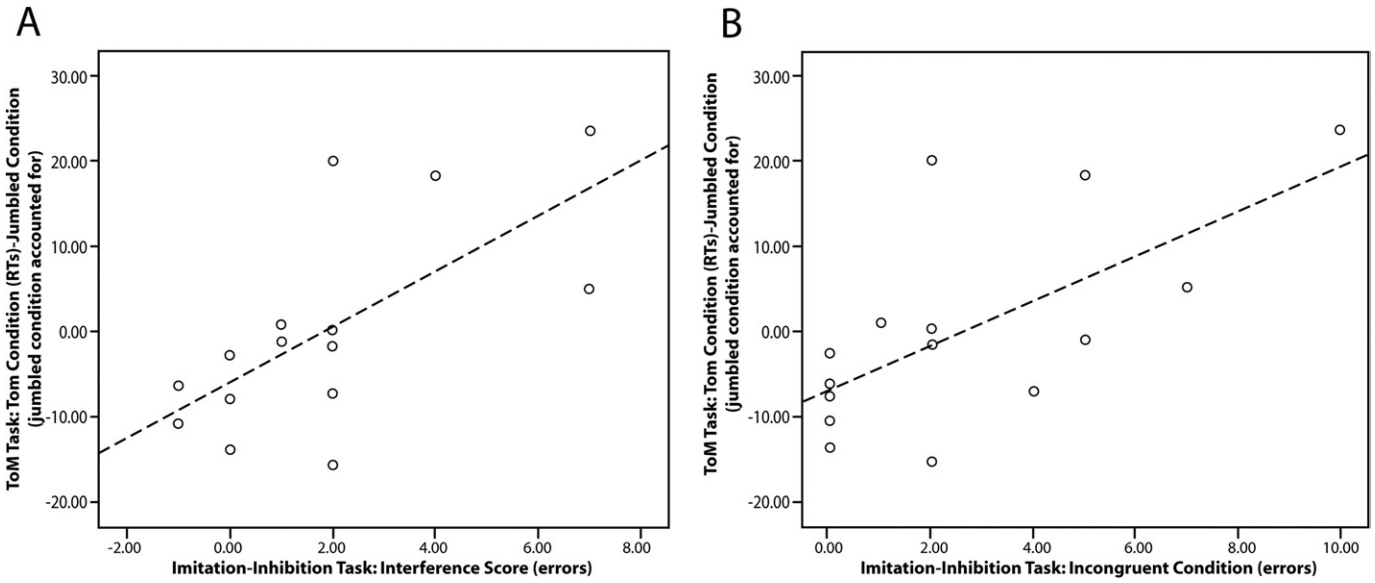

Figure 3. Scatter plots of the correlation between imitative control and mentalizing. **(A)** Scatter plot showing the significant correlation in the autism spectrum conditions group ($n = 15$) between the performance in the imitation-inhibition task (interference score) and the reaction times (theory of mind condition), after statistically eliminating the performance in the jumbled condition. **(B)** Scatter plot showing the significant correlation in the autism spectrum conditions group ($n = 15$) between the performance in the imitation-inhibition task (incongruent condition) and the reaction times (theory of mind condition), after statistically eliminating the performance in the jumbled condition. RT, reaction time; ToM, theory of mind.

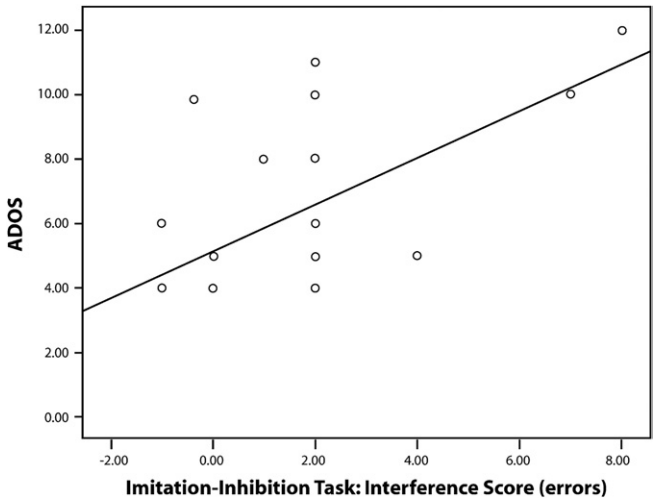

Figure 4. Scatter plots of the correlation between imitative control and the Autism Diagnostic Observational Schedule (ADOS). Scatter plot displaying the significant correlation in the autism spectrum conditions group ($n = 15$) between the performance in the imitation-inhibition task and the Autism Diagnostic Observational Schedule (reciprocal social interaction subscale), where a higher score on a subscale indicates a greater degree of impairment.

data points had values smaller than .1. Therefore, the above-reported, significant correlations are not caused by statistical outliers.

Correlations Between Imitative Control and the ADOS. The scores of two subscales of the ADOS, measuring reciprocal social interaction and social communication in interaction with the examiner, were correlated with the interference score of the imitation-inhibition task. Mirroring the results from the ToM task, a significant positive correlation was found between the reciprocal social interaction score and the interference score for errors [$r(15) = .51$, $p < .05$, corrected], showing that an increased interference effect (i.e., lower imitative control) was related to higher social impairment (Figure 4). Furthermore, none of the data points of this correlation showed an outlier measure value greater than .4 (both measures).

Neuroimaging Results

Whole-Brain Correlations Between Performance in Imitative Control and Neural Activity during Mentalizing. We investigated in a between-subjects regression analysis the brain areas that showed decreasing activity during mentalizing with increasing interference effect in the imitation-inhibition task. Three brain regions were found to be selectively activated. These activations were localized in the mPFC (x: 15, y: 54, z: −3), the TPJ (x: −51, y: −63, z: 24), and posterior superior temporal sulcus (x: 60, y: 39, z: 6) (Figure 5).

Meta-Analytic Results. To test whether the mPFC activations obtained in this study encompassed similar cortical regions as in previous neuroimaging studies on mentalizing, we compared meta-analytic data and our activation foci. As the focus of our interest was the mPFC, the coordinates included in the meta-analysis were restricted to this region. This revealed an overlap of the above-reported activation and of previously found mentalizing activation foci in the ventral part of the mPFC (Figure 6).

Discussion

Previous studies in ASC reported impairments of mentalizing (31) and also weaker activations of typical mentalizing areas in neuroimaging studies (32). Conversely, a second line of research focused on the integrity of the mirror system in autistic patients and has related this to poor social abilities and deficits in imitative performance in ASC (9,11,12). In contrast to this hypothesis, the view favored by the current work would predict that autistic patients should have problems in the control of imitative behavior rather than in imitation per se. It might be therefore possible that the mirror system is not deficient in ASC but that this system is not influenced by regions that distinguish between the self and other agents (31). Impairments of such a system could therefore lead to egocentrism, abnormalities in self-awareness, and limitations in mentalizing, as they can be found in autism (39). This would also predict that the control of imitation might be related to social abilities (e.g., performance in ToM tasks) in individuals with ASC.

The current results strongly supported this idea. Individuals with ASC showed a higher interference effect, that is, more deficits to inhibit automatic imitation compared with a matched control group. Secondly, a significant correlation was found between a theory of mind task and the imitation-inhibition task. Thirdly, in an associated fMRI study, decreased performance on imitative control was also associated with less activity in mPFC and TPJ during a theory of mind task (animated shapes [32]), regions believed to be crucial for the control of imitation. We furthermore conducted a meta-analysis and confirmed that the activation in the mPFC, which has been considered as the key node in ToM processing (e.g., [40]), falls within an area previously implicated in mental state attribution tasks.

In common with previous reports of equivalent automatic imitation effects and the clinical observation of overimitation of actions, the ASC group showed, in a more sensitive task, a significant hyperimitation of actions. This indicates that the ASC participants had problems with the control of MS functions, rather than with MS functioning itself. However, most studies using neuroimaging methods have found evidence for weaker activation of the MS in autism (e.g., [11,12]), although more recent studies showed equal or even hyperactivation of MS regions (17,19,20). This may partly be due to the reason that different effectors were used in different studies to detect MS activation in the ASC group. Studies using emotional facial expressions (e.g., [11,41]) cannot, for example, distinguish automatic imitation from emotional contagion. The results are also difficult to interpret because face stimuli were presented, and there is a growing body of evidence that gaze patterns to faces are abnormal in autism (42). Specifically, individuals with ASC spend less time looking at the eye region of the face, which has been shown to be crucial in emotion recognition (43). Thus, these results are hard to compare with the stimuli in our studies that rather relied on the more classical hand mirror system. In addition, a recent meta-analysis questioned whether all regions of the mirror system are equally involved in imitation (44). Until the results of studies investigating mirror system activity in ASC show a more consistent pattern, it may be difficult to relate them to the imitation abilities in this group.

Furthermore, our results from our ASC participants replicated a previous lesion study (33) that showed patients with frontal lesions showed a highly significant correlation between the interference score and the same ToM task, a pattern that was also found to a lesser extent in the control group, as the sample size of this group was much larger than in the present study, which might have prevented finding any significant results in the control group. Similar to the current study, it was shown statistically that the correlations were not based on single outliers and possible moderating variables, such as age, IQ or the control conditions of the ToM task, did not influence the obtained correlations. Additionally, previous neuroimaging and patient studies showed that the imitation-inhibition

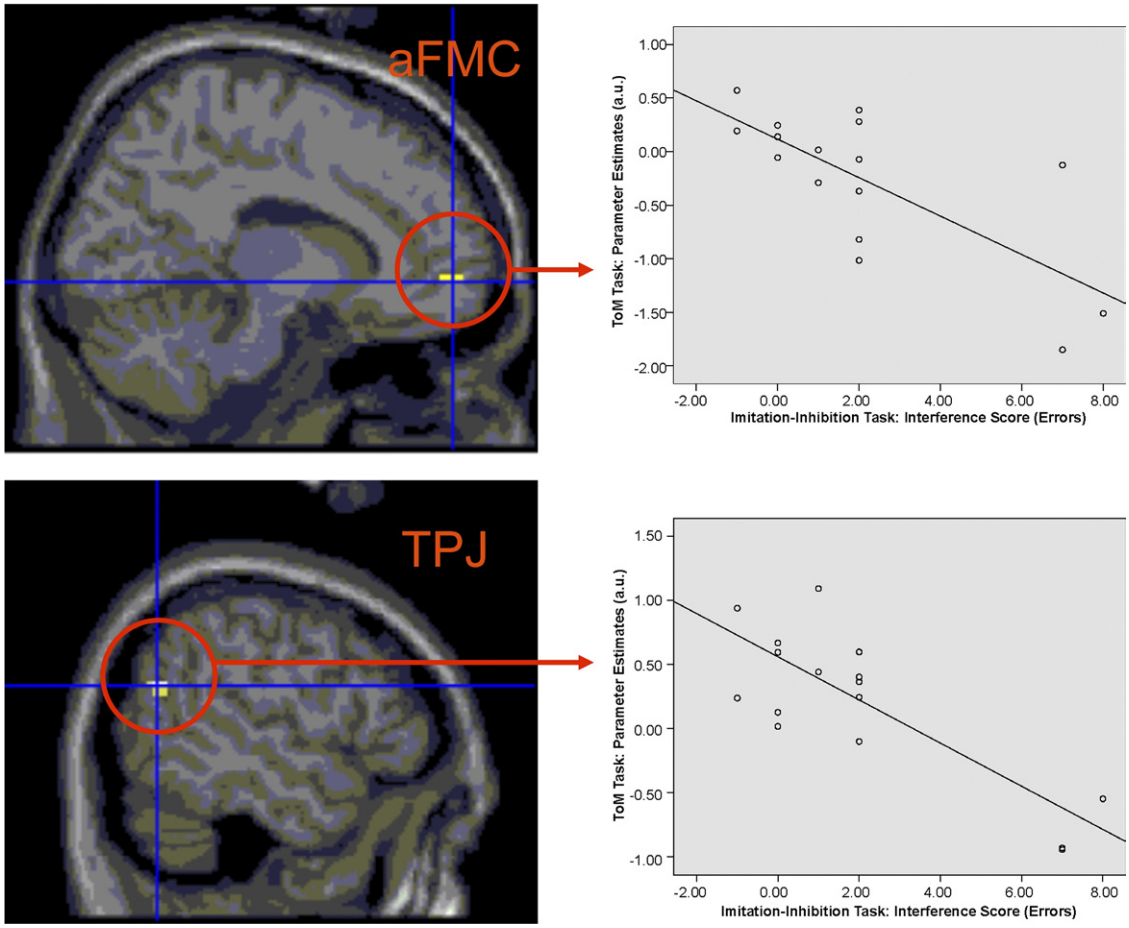

Figure 5. Correlation between brain activity in the theory of mine (ToM) task and the interference score in the imitation-inhibition task. Left: Areas (medial prefrontal cortex, temporoparietal junction) showing a negative correlation of activation with increasing interference score ($p < .001$, uncorrected). Right: Relationship between the parameter estimates (mean cluster value) ToM task (ToM − control condition) and the interference score ($n = 17$). aFMC, anterior fronto-median cortex; TPJ, temporoparietal junction.

task was not related to cognitive control tasks (45,46) and that even after controlling for executive functions, such as response inhibition, mental flexibility, and working memory, the correlations between ToM and imitative control remained significant (33). Although our previous work demonstrates that the association between self and other distinction with regard to ToM and imitation inhibition is not reducible to an executive function deficit, in other neurodevelopmental disorders echophenomena are associated with general disinhibitory behaviors. For example, in Gilles de Tourette's syndrome, catatonic symptoms, including echophenomena, are associated with the presence of comorbidities including attention-deficit/hyperactivity disorder. At present, this is still an underresearched area in ASC.

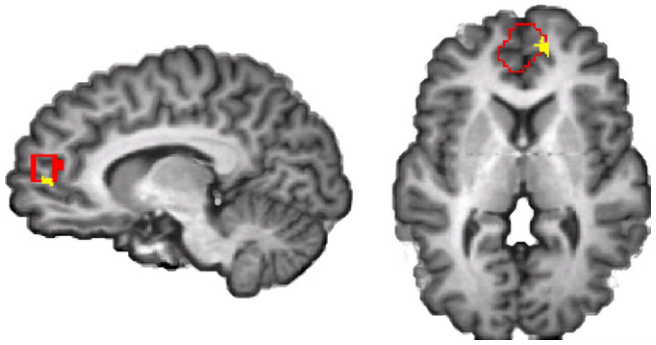

Figure 6. Overlap of areas activated in the present study (yellow: correlation interference score with brain activity in the theory of mind task) and previous studies on mentalizing (red), focusing on the medial prefrontal cortex. Displayed are areas overlapping in the ventral medial prefrontal cortex.

Similarly, a recent model has suggested that a route responsible for automatic imitation is not generally disturbed but that the modulation of this route is deficient in ASC (26,27). This top-down modulation account predicts that functions of mPFC and TPJ are crucial to regulate automatic mimicry. These specific areas might modulate activity in other cerebral regions and therefore mPFC and TPJ should show abnormal processing in ASC, rather than typical mirror regions. For example, differences in top-down modulation of face processing in autism has been shown (47), as well as a functional underconnectivity between frontal and posterior regions during ToM processing in autism (48). Thus, one intriguing, but still speculative, idea is that the mPFC is concerned with top-down modulation of posterior brain regions and possibly regions of the shared representational system (49). Similarly, it has been proposed that the mPFC is crucially involved in top-down control during empathic responses (50,51). In an fMRI study, naive participants reacted to the observation of painful stimuli with an activation of the pain matrix (anterior cingulate cortex, anterior insula), whereas experts did not

show this pattern but rather activations of the mPFC and the TPJ, in close proximity to the regions of the current study (52). Furthermore, controlling facial mimicry recruited an area in the posterior superior temporal sulcus, close to the TPJ region (53). Accordingly, automatic activation of shared representations for emotional experiences might be modulated by the mPFC network, which supports meta-cognitive, reflective awareness of these emotional states (54).

In summary, we present experimental evidence for the hyperimitation of actions in adults with ASC. Our findings further suggest, by showing a relationship between such increased action mirroring and behavioral and neural measures of social cognition, that the impairment of the control of MS functions shares common features with mental state attribution. This implies that top-down modulation MS functions may be compromised in ASC. These results may have important implications for future theorizing about the cause and nature of deficits observed in ASC.

This work was supported by the European Community's Sixth Framework Programme, New and Emerging Science and Technology/Evolution, Development and Intentional Control of Imitation (Contract Number 012929) and the Medical Research Council (United Kingdom).

All authors report no biomedical financial interests or potential conflicts of interest.

Supplementary material cited in this article is available online.

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