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A Systematic Review of Action Imitation in Autistic Spectrum Disorder

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Imitative deficits have been associated with autistic spectrum disorder (ASD) for many years, most recently through more robust methodologies. A fresh, systematic review of the significance, characteristics, and underlying mechanism of the association is therefore warranted. From 121 candidates, we focused on 21 well-controlled studies involving 281 cases of ASD. Overall, children with ASD performed worse on imitative tasks (Combined Logit p value $< .00005$). The emerging picture is of delayed development in imitation, implicating a deficit in mapping neural codings for actions between sensory and motor modalities, rather than in motivation or executive function. We hypothesise that ASD is characterised by abnormal development of these mappings, such that they are biased towards object-oriented tasks at the expense of those required for action imitation per se.

KEY WORDS: Imitation; gesture; action; autism; emulation; 'mirror neurons'.

INTRODUCTION

Imitation research in autistic spectrum disorder (ASD) has a long history. The first suggestion of a relationship was made in 1953 (Ritvo & Provence, 1953), just 8 years after Kanner (1943) had published his seminal paper on autism. Ritvo and Provence noted that:

A mother described the inability of one 21-month-old child to make pat-a-cake simply from watching her. The only way he could learn the game was to have the mother hold his hands and put them through the appropriate movements.

Since that time, several studies have explicitly investigated the relationship between imitation and autism. Rogers and Pennington (1991) were the first to review these, examining seven studies. Their influential review found strong evidence for the existence of a deficit affecting imitation of simple body movements, as well as imitation of actions with symbolic meaning. Using Stern's (1985) model of interpersonal development, they suggested that a biological impairment in autism restricted the capacity of the infant to "form and co-ordinate social representations of self and other at increasingly complex levels via amodal or cross-modal representational processes". They hypothesised that a primary deficit in such a capacity (referred hereon as self–other mapping) would lead to a cascade of effects including impaired imitation, social, communicative and affective skills, and that a deficit in the prefrontal-limbic neural system could form the basis for this impairment.

Later, Smith and Bryson (1994) carried out a comprehensive review of 15 studies conducted up to that point, including a number lacking control data. They hypothesised that the imitative deficit associated with autism could be due in part to impairment in the perceptual organisation of movements,

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manifesting in an abnormal representation of actions. They suggested that the ability of children with autism to recognise when they are being imitated precludes the problem from being one of self–other correspondence of the kind Rogers and Pennington (1991) had suggested (although one might argue that this should also preclude the possibility that the problem lies in abnormal perception of movements). Smith and Bryson concluded tentatively that children with autism might show different patterns of imitative deficit according to their functional level, that imitative tasks requiring bilateral integration may be especially affected, but that the specificity of the deficit to autism was still undemonstrated.

Rogers (1999) carried out a further review of studies up to 1997 (though just missing a further study by Charman *et al*. and Drew (1997) described further below) and took up some of the issues raised by Smith and Bryson. Rogers again found strong evidence supporting the existence of an imitative deficit, but concluded there was insufficient data to be precise about the components of imitation affected, or whether the deficit was due to a motivational, praxic or self–other matching impairment. She did not discuss whether the evidence pointed to the deficit being specific to ASD.

A hypothesis put forward by Williams, Whiten, Suddendorf, and Perrett (2001) attempted to build on the Rogers and Pennington (1991) model by linking it to the recent discovery of "mirror neurons" that code for the same action, whether it is perceived or enacted (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). However, rather than contrasting the imitative model of intersubjective development with the metarepresentational (or "theory of mind") model (Baron-Cohen, Leslie, & Frith, 1985) as Rogers and Pennington had done, Williams *et al*. considered that the "theory of mind" deficit associated with autism might be due to a "simulation" deficit (Gallese & Goldman, 1998; Carruthers & Smith, 1996). This would mean that people with autism have difficulty understanding the thoughts of others, because they find it hard to imagine themselves in their position by relating what they observe about others' behaviour to their own neural codings for similar behavioural memories. They suggested that such a simulation process is dependent upon a neurocognitive mechanism allied to that necessary for imitation. Williams *et al*. suggested that a developmental delay in such a neural mechanism could be the common factor that is core to autistic disorders. One implication of this proposal is that imitation may be a profitable focus in studying the integrity of the underlying neural mechanism involved in autism.

In recent years, a number of studies have been published that utilised more robust methodology than previously. Furthermore, the study of imitation itself has advanced, as a number of disciplines including primatology, robotics, neuropsychology, and developmental psychology have begun to cross-fertilise (Dautenhahn, 2002; Meltzoff & Prinz, 2002; Want & Harris, 2002). We therefore considered that a fresh, critical and systematic review of the literature in the light of recent thinking, and new, higher quality data, would address questions of current interest. In particular, these concern whether the imitative deficit associated with autism is:

1. Typically present and if so, to what extent?
2. Necessary for the development of autism and therefore ubiquitous in the condition?
3. Specific to autism or a non-specific feature of co-morbid neurodevelopmental disability?
4. Associated with a profile of performance that might suggest whether the underlying problem is with the process involved in translation from observation of acts to the execution of them, a general social learning impairment or some other less specific neurocognitive capacity utilised by imitative processes, such as executive function.

SEARCH DETAILS

Computerised literature searches of the databases EMBASE 1988–present, Medline 1966–present and Web of Science were performed. In addition, a manual search of core articles and books was conducted. Using the subject headings "imitative behaviour" and "autistic disorder" (as subject headings or key words), 104 references on Medline were located. The EMBASE search used subject headings "imitation" and "autism or infantile autism". This generated 26 references, of which 17 were novel in relation to the Medline survey. A further search run through Web of Science did not identify any additional references. Three further references (one in press) were identified through correspondence and articles. The search covered the period up to March 2002.

Inclusion Criteria

To include any of these studies in our further analyses, we set the criterion that it should at least compare performance on an action-imitative task between a group with ASD and a control group. Diagnostic groups could include subjects with autism or Asperger's syndrome. One study contained some subjects with atypical autism. We confined our review to the imitation of hand and body actions rather than including imitation in general. This is because our aim was to examine specifically the profile of the action–imitation deficit, rather than imitation as a whole, which may be associated with a broader range of neural mechanisms. Also it may not be valid to compare vocal, facial and affect imitation with action imitation, and the inclusion of such a diverse range of studies may confuse rather than clarify. Where studies did include other measures, we have confined our analysis to the data on action imitation. Twenty-one studies met these criteria. Nine of these have not been included in previous reviews. Some studies included in previous reviews were excluded due to lack of controls.

CLASSIFYING IMITATIVE TASKS

The ongoing multidisciplinary onslaught on imitation noted above has produced a profusion of alternative ways of classifying forms of imitation. Here, we emphasise just two principal distinctions that are made in the existing literature. First, if the act involves an object we have termed it an "action upon an object" (AO) and where no object is involved we have called it a gesture. Second, we distinguish "meaningful" from non-meaningful actions. This is less straightforward, yet appears important. The meaningful category typically includes pantomime acts, such as pretending to use a comb. It also includes meaningful and symbolic gestures such as waving "goodbye". For the purpose of this analysis we have called all meaningful, symbolic, familiar, transitive or pantomime acts and gestures "Meaningful gestures" (MG) and others non-meaningful gestures (NMG). Meaningful gestures are those with a semantic association, whilst NMGs can only be described in terms of posture and location.

This classification covers most tasks quite adequately for present purposes. One remaining anomaly is where an object is used to perform a meaningful action but not the one for which the object was intended (sometimes referred to as a placeholder in the literature). This we have called an "action-upon-a-substituted object" (ASO). In addition, where a *sequence* of actions or gestures is presented, we have added the term sequential (S). Finally, three other dichotomies will play a less central role in our analysis and will be further discussed below. They are: immediate vs. deferred; spontaneous vs. elicited; and structure vs. style.

REVIEW OF INDIVIDUAL STUDIES

A summary of the studies reviewed is shown in Tables I and II. In the following, which should be read in conjunction with Table I, which it complements, we address significant methodological issues and highlight details of the studies for which there is insufficient space in the table.

The first full study, by DeMeyer *et al.* (1972), used an extensive battery of imitative tasks, based on the do-as-I-do method (Hayes & Hayes, 1952). Problems include the absence of reliable diagnostic criteria available at that time, and the fact that because the children with autism had a lower mental age than the controls, they were exposed to a different (easier) set of tasks. DeMyer *et al.*, introduced the notion of including a simple imitative task as a control for "negativism" and also considered the impact of pre-task training. They noted that four autistic children had received extensive training in action imitation before the experiment, yet still failed these tasks.

Hammes and Langdell (1981) introduced the method of displaying tasks on videotape. As with DeMyer *et al.*, simple tasks were performed at ceiling levels by all individuals in the group. These were pantomime actions, all involving the pouring, drinking and stirring of soup or tea, and involving the use of real objects. Group differences then emerged when subjects were asked either to imitate these pantomimed actions in the absence of an object, or were asked to use an object for another purpose, such as drinking from the pot. Five out of eight children with autism used the object for its proper purpose rather than that which had been modelled.

Sigman and Ungerer (1984) examined well-matched groups and employed simple imitative tasks from the Motor Imitation Scale (MIS; Uzgiris & Hunt, 1975). Despite the simplicity of these tasks, group differences were still significant. However,

Table I. Studies discussed further in the text

	Subjects			Controls				Diagnosis		Tasks	
Paper	Number of subjects	Mean age of subjects (years)	Level of function	Controls (n)	Control for IQ	Control for age	Control tasks	Diagnosis	Diagnostic tools	Tasks (p value)	Reported group differences p value
DeMyer *et al.* (1972)	12	5.6	Verbal age = 2.60 years	MR (5 only)	No. of Mean verbal ages of controls = 4.4 years	Yes	Yes (Spontaneous Object use and three task difficulty levels	Autism (9) + schizophrenia (3)	No	MG NMG MAO NMAO	Easy AO = NS Possible AO < 0.02 All Gesture < .01
Hammes and Langdell (1981)	8	9.1	Snijders-Oomen test MA = 4.5	MR	Yes	Yes	Simple tasks	Autism Rutter (1978)	No	MAO MASO MG	MAO = NS MASO < 0.013 MG = .02
Sigman and Ungerer (1984)	16	4.3	MA = 24.8 IQ = 48.1	MR (16) Normal (16)	Yes	Yes	No	Autism DSM III	No	MAO MG NMG	MAO < 0.01 (vs. MR) M/NMG < .01 (vs. MR) < .001 (vs. Nor.)
Jones and Prior (1985)	10	8.6	MA = 4.3 IQ (Leiter) = 72	Normal (10) CA matched Normal (10) MA matched	Yes	Yes	Yes	Autism DSM III	No	NMG (Berges-Lezine)	< .001
Ohta (1987)	16	10.2	IQ = 72.1 VIQ = 64.9 PIQ = 85.3	Normal (8) ADHD (8) 189 preschool 3–6 years	Yes PIQ	Yes	No	Autism DSM III	No	MG NMG (Berges-Lezine)	Aut. vs. 3–3.5 years-NS Aut vs. 3.5–4 years- < .05 (PI score)
Stone *et al.* (1990)	22	4.6	IQ = 54.1 (Merril-Palmer Scale)	MR (15) Normal (20) Hearing impaired (20) LI (20)	Yes with MR group	Yes	Yes	Autism DSM III	CARS Mean = 36.5)	MAO NMG (tasks from DeMyer *et al.* 1972)	< .0001
Charman and Baron-Cohen (1994)	20	11.7	3.8 Verbal ability	DD	On VMA but not on NVMA	Yes	No	Autism DSM-III, DSM-IV, Rutter (1978)	No	NAO MG NMG (MIS & Meltzoff, 1988 a, b)	NS
Brown (1996)	27	24.7; 12.1; 5.7	MA = 6.75 MA = 4.75 MA'n/a' (BPVS, TROG)	MR (11) 5–6 years old (12) 3–4 years old (11)	Yes MR group	Yes	Easy and "motivating"	Autism DSM-III-R	No	MG, NMG MAO NMAO SNMAO Spont. AO Deff.AO	Reduced ability in youngest (p < .01)

Rogers *et al.* (1996)	17	15.5	FSIQ = 89 (WISC)	Dyslexia (10) Mixture (5)	Yes	Yes	Motor and memory (No diff.)	AD (9) PDDNOS (8)	CARS 34.25	MG NMG SMG SNMG	NS NMG .008 SMG <.001 SNMG .001
Stone *et al.* (1997) (Study 1)	18	2.3	EV = 1.8 DQ = 56	DD and Normal	Yes	Yes	None	Autism DSM-III, DSM-IV, Rutteasfr (1978)	CARS = 35.1	MIS Total AO MG NMG	<.01
Charman *et al.* (1997)	10	1.7	NVMA = 1.4 VC = .5 EL = .5	DD and normal	Yes	Yes	None	Autism	ADI-R + Expert clinician	NMAO (Meltzhoff, 1988)	0.04 (Aut vs MR) 0.01 (Aut vs. Norm.)
Roeyers *et al.* (1998)	18	4.8	3.2 MPS or McS	DD	Yes	Yes	None	DSM-III or DSM-IV	No	MG, NMG MAO, NMAO (MIS and Meltzoff tasks)	.0005 (MIS) .05 (Meltzoff)
Smith and Bryson (1998)	20	11.4	MA = 7.8	LI (20) Normal (20)	Yes	With LI	Gesture memory and dexterity task	Autism Bryson, Clark & Smith's diagnostic criteria	ABC	NMG (Deaf alphabet and Berges-Lezine)	NMG: .002 Reversal errors: .006 Left-right errors: NS Model present vs. absent .02 (no group X condition interaction)
Hobson and Lee (1999)	11	13.75	VMA = 5	MR	Yes	Yes	No	DSM-IV	CARS = 33.1	'style'	P < .005 "reversal errors"
Aldridge *et al.* (2000)	10	3.3	Nil	Normal	Object permanency task	No	Nil	DSM-IV	Nil	Gesture and emulation task	No tests but clear group differences on both
Green *et al.* (2002)	11	9.2	WISC = 107 M-ABC	SDDMF	Yes	Yes	No but note control group	DSM-IV	ADI-R	MG NMG (Cermak Coster, & Drake, 1980)	p < .01

Abbreviations to Tables I and II.

Imitative tasks.

MG = Meaningful Gestures; S = sequential. N = non-meaningful; H- as a prefix = hand; AO = actions with objects; ASO = Actions upon substituted objects; Spont. = spontaneous; Deff = deferred; MIS = Motor Imitation Scale (see text).

Others

VMA = verbal mental age; NVMA = nonverbal mental age; (Griffiths) VC = Verbal comprehension; (Reynell) EL = expressive language; (Reynell) DQ = Developmental Quotient; EV = expressive Vocabulary; LIPS = Leiter International Performance Scale; PPVT = Peabody Picture Vocabulary Test; SDDMF = specific developmental disorder of motor function; M-ABC = Movement Assessment Battery for Children; CARS = Childhood Autism Rating Scale; DSM-III = Diagnostic and Statistical Manual, 3rd edition; PI = Partial Imitation; Ohta 1987, described these as "peculiar" and seems to be referring to reversal errors (see text). NS = not significant. MR = mental retardation; MA = mental age; CA = chronological age; DD = developmental delay; LI = language impaired; Aut = autism group.

Table II. Studies not discussed any further in the text

	Subjects			Controls				Diagnosis		Tasks	
Paper	Number of subjects	Mean age of Subjects (years)	Level of function	Controls (n)	Control for IQ	Control for age	Control tasks	Diagnosis	Diagnostic tools	Tasks (p value)	Group differences p value
Wetherby and Prutting (1984)	4	9.5		Normal (4)	No	No	No	Autism US Nat. Soc. Aut. Children Criteria	No	MAO NMAO MG NMG	NS (tests only for overall communication)
Jones and Prior (1985)	10	8.6	MA = 4.3 IQ (Leiter) = 72	Normal (10) CA matched Normal (10) MA matched	Yes	Yes	Yes	Autism DSM III	No	NMG (Berges-Lezine)	$< .001$
Morgan *et al.* (1989)	10	8.4	MA = 3.03 (PPVT)	MR (10) Normal (10)	Yes	With MR only	Used all items on MIS	Autism DSMIII	No	MAO NMAO MG NMG (MIS)	NS (Ceiling levels in all groups)
Heimann *et al.* (1992)	5	4.3	Griffiths DQ 43–106	Normal (3)	No	Yes	No	Autism DSM III	No	MAO MG & MASO	No tests
Libby *et al.* 1997	10	12.5	VC = 6.2 EL = 5.5	Down's syndrome; Normal	Yes (VMA)	No Mean age controls = 4.6 & 2.3 years	None	Autism Wing and Gould (1979)	No	ASO SMG Script	Autism > control $P < .05$ + less likely to refuse
McDonough, Stahmer, Schreibman, and Thompson (1997)	6	4.6	LIPS = 2.7 PPVT = 2.2	LIPS & PPVT matched groups	Yes	No	No modelling vs. modelling	DSM-III-R	No	NMAO MAO	NS

much information is not reported in this study, including measures of IQ and reliability, and which of the MIS Items were used and which items elicited normal performance. The study noted a relationship between receptive and expressive language ability, and imitative ability. This meant that children with autism performed worse than those with mental retardation and much worse than those with normal intelligence.

Ohta (1987) appears to be the first author to describe perspective reversal errors in imitation, calling them "partial imitation". Such errors include that of the subject holding up the palm to face themselves when they have observed it facing towards them. This phenomenon did not appear in any of the age- and IQ-matched control groups, but for one task it did appear with equal prevalence in 3–3.5-year-old preschool children.

Stone, Lemanek, Fishel, Fernandez, and Altemeier (1990) published a large study that investigated 91 children in four groups (mental retardation, hearing impairment, language delay, and autism). Imitative tasks consisted of "pretend" actions and body movements. Only the group with autism showed an imitative deficit. The absence of reliability measures and control tasks are weaknesses in this study. This is of some concern as some of the tasks were simple and on the basis of earlier studies, normal performance might have been predicted.

Charman and Baron-Cohen (1994) tested a large number of children on the Uzgiris–Hunt Scales and tasks described by Meltzoff (1988a,b). The normal ages for passing these tasks are 7–20 months and 9 months, respectively. The intention of the study was to compare imitative ability on gestural imitation with "procedural" imitation (what we have called here imitation of actions on objects). Subjects had a mean chronological age (CA) of nearly 12 years, and a mental age of 4–7 years. It is perhaps not surprising then, that the study was confounded by ceiling effects, with high levels of passes on most tasks. However, one of the "procedural" tasks, which utilised a method most likely to be novel to the observer (touching a button with the forehead), did discriminate between the learning disabled and autism group. The authors suggest that whilst the other tasks could be completed by a problem-solving approach (emulation), the forehead-touching-button task was most dependent upon imitation.

Brown (Brown, 1996; Whiten & Brown, 1999) carried out a large study which included individuals with autism from three age groups, as well as 3- to 4-year-old, and 5- to 6-year-old normal children and children with mental retardation. Imitative ability was compared on a broad range of tasks, including 93 actions that were a mixture of actions on objects, (substituted and non-substituted), gestures (face, hands and body) and vocal expressions. Young children with autism performed worse than the other groups including the 3- to 4-year-old children. By contrast, the older autistic children and adults generally performed well, scoring the top score over 50% of the time.

Brown also investigated two aspects of imitation upon novel objects introducing the two-way method of testing for imitation described by Dawson and Foss (1965). The first concerned spontaneous imitation of opening an "artificial fruit" used in imitation research with non-human primates (Whiten, Custance, Gomez, Teixidor, & Bard, 1996). This task involves opening a reward-containing box in front of a child, using just one of various possible methods to remove a pin, handle and bolts. The reassembled "fruit" is then handed to the child and their method of opening it can be compared to the one that they were shown. The degree of fidelity they show in their method of opening to that they observed, provides a measure of spontaneous imitation. The young children with autism had a much lower inclination to imitate the method they saw, and differences between autistic and non-autistic populations once again diminished with increasing age of the autistic group. Finally, showing subjects one of two ways to open a drawer, and then giving them the drawer to open the following day tested deferred imitation. The subjects with autism were less inclined to imitate the method that they had observed than the controls.

Rogers, Bennetto, McEvoy and Pennington (1996) provided a methodologically thorough study of imitation in autism. Participants had high functioning autism and were compared with an age- and IQ-matched group. Control tasks were designed to ensure that memory and motor control problems were not confounders. There were some problems though: the control group was ill-defined with a diffuse range of neurodevelopmental impairments, and some of the patients had a diagnosis of atypical autism, not meeting full criteria for autism or Asperger's syndrome. The authors argued that such striking findings in such mildly affected individuals added weight to their case but it raises some doubts over the specificity of the deficit. Group differences

were greater with sequential imitation than with single imitation, and whilst adding meaning to the gestures hindered performance among the controls, it improved it for the group with an autistic disorder.

Stone, Ousley, and Littleford, (1997) compared 18 autistic children with a mean age of 28 months, with some even younger normal controls and children with developmental delay. The diagnosis in children of this age can be unreliable and Stone *et al.*, do not describe the use of a standardised method. Nevertheless, they report significant group differences on imitative ability. Again, the lesser magnitude of these differences may perhaps be explained by the age difference between the autistic group and the controls. In addition, no task type x group interaction was found, so all groups showed equally improved performance for meaningful actions on objects with non-meaningful action imitation being most difficult. Two other interesting findings to emerge were that: (a) imitation improved at follow-up a year later (though there was no later comparison with other DD children); and (b) the imitation score predicted expressive language one year later ($r = .55$, $p < .01$).

Charman *et al.* (1997) repeated their 1994 methods, but in much younger children identified as part of a screening programme and diagnosed using Autism Diagnostic Interview–Revised (ADI-R), Autism Diagnostic Observation Schedule-Generic (Lord, Rutter, & Le Couteur, 1994; Lord *et al.*, 2000) and clinical judgement. This time, in contrast to their earlier findings, they found a highly significant group difference between those with autism and the controls. Those with developmental delay fell between the other two groups. The ascertainment and diagnostic procedures employed make the findings particularly robust, but, as with Charman *et al.* (1994), it still remains debatable (as discussed further below) whether the actions on the objects were really tasks tapping the ability to achieve the same goal (to emulate) as opposed to copying the method to reach the same goal (to imitate). Charman *et al.* (1998) then reanalysed the data utilising the diagnoses made at 42 months. Those individuals with pervasive developmental disorder that did not meet full research criteria fell between those with autism and those with developmental delay, though post-hoc comparisons only revealed significant differences between the latter two groups.

Roeyers, Van Oost, and Bothuyne (1998) also used tasks involving actions upon novel objects to test what they called procedural imitation, with 5-year-old children. Whilst they found significant group differences on "procedural" imitation, those for gestural imitation were of a much greater magnitude. Half of the autistic subjects imitated unreliably on gestural tasks, compared with just one of 18 controls. The most powerfully discriminating gesture was a non-meaningful, invisible gesture, which involved clapping both hands onto the back of the head. Of the "procedural" tasks, the best discriminator was the task not associated with a sensory effect, involving transfer of a ring between two branches of a model tree. However, as with Charman *et al.* (1994, 1997, 1998), the experimental design does not exclude the possibility of using previously learned methods to obtain the same outcome (emulation), though in the action-object task where this seems least likely, there are greatest group differences.

Smith and Bryson (1998) conducted another sizeable study involving 20 children with autism, aged about 11 years, matched to language-impaired controls and to a group of verbally matched, younger controls aged about 6 years. The study examined imitation of meaningful and NMGs both singly and as two and three item sequences. Also included was a test of gesture memory and groups in which children performed equally well with both individual postures and their corresponding sequences. Children with autism imitated single postures much worse than the other children and all three groups performed significantly worse in the absence of the model. Children with autism also made significantly more 180^{o} reversal errors, although they did not show any greater reduction in performance with sequences than controls. Finally, these authors found that receptive language level accounted for 11%, and a measure of dexterity accounted for a further 37%, of variance associated with imitation ratings scores. However, the group differences in non-symbolic imitation of gesture still remained significant. Age was not included in the regression equation, which may have led to an underestimate of the effect size.

Hobson and Lee (1999) were interested in not just whether children with autism could imitate the essential configuration of an action but the "style" in which it is performed. They therefore compared autistic and well-matched mentally retarded children on tasks that were performed in a "harsh" or "gentle" way (meaningful objects used in a non-meaningful way). The results were partially confounded by the children's basic difficulties with imitation and

reversal errors, but despite this, there was still a very considerable difference between the groups in their tendency to imitate "style".

Aldridge, Stone, Sweeney, and Bower (2000) compared a group of 10 children with autism and a mean age of 3.3 years, with a group of normal children whose mean age was 11 months. They were matched by their performance on Piagetian object permanence tasks. There were three simple gestural imitation tasks including tongue protrusion and three emulation tasks using the materials described by Meltzoff (1995). These tasks involve the demonstrator performing an action but failing to reach the goal, such as pressing a button but missing it. The children with autism scored a uniform zero on the gestural imitation, but interestingly they managed on many of the emulation tasks.

Finally, Green, Baird, Barnett, Huber, and Henderson, (2002) have conducted a study designed to compare the profile of motor difficulties of children with Asperger's syndrome (AS) to those seen in children with Developmental Co-ordination Disorder (DCD). The comparison involved a range of motor tasks but included a test of NMG imitation. Green *et al.* found that whilst all nine children with AS (diagnosed using ADI-R) also met diagnostic criteria for DCD, they performed even worse than the DCD group, who did not have an autistic disorder, on this task.

OVERALL FINDINGS

Is There an Imitative Deficit in Autism?

The first finding of this review is a reinforcement of Rogers' (1999) message that "every methodologically rigorous study so far published has found an autism specific deficit in motor imitation". Of the 21 studies, 14 found an imitative deficit, three did not report the necessary tests and four showed no effect. Of these latter four studies, two were confounded by ceiling effects, one included a very small sample that used much younger children as controls and the other also used autistic children much older than controls. Most studies found group differences at very high levels of significance, despite having tested just 10–20 subjects per group.

How Significant is the Effect Overall?

There are many problems in combining the results from studies such as these, to arrive at a single measure of effect size (Smith & Egger, 1999). The groups differ in their membership and the tasks differ according to their type. Other methodological differences make some findings more reliable than others. Finally, the studies generally report levels of significance for differences between groups, rather than percentages of subjects passing tasks, which might have allowed the computation of a combined odds ratio. Therefore, we cannot compute a measure that estimates the magnitude of the group difference. However, as the studies described have now examined a total of 281 children with ASD in addition to controls, an overall, combined p value may still offer a fairly robust and useful indicator of performance differences between autistic and non-autistic groups. We used the Logit method (Britton *et al.*, 1998) to combine p-values from 17 studies where tests of group differences were carried out with respect to gesture or action compared to an appropriate control group (Green *et al.* was excluded due to the nature of the control group). Thirteen significant p values were combined for $k = 17$ studies. The logit method requires all p values to be independent, so only one p could be taken from each study. A hierarchy was set for p-value selection, namely NMG, followed by MG, MAO and finally NMAO. The combined p value, computed in this way, was $p = .00005$ ($n = 248$ subjects, $t = 4.260$, 89df). If those studies where age and IQ were not controlled for are excluded, only one study remains non-significant and this figure decreases further to $p = .00002$ ($k = 12$ studies, $n = 196$ subjects, $t = 4.56$, 69 df, 64 df).

Is an Imitation Deficit Specific to Autism?

Is it possible that the imitation deficit could be explained by non-specific neurodevelopmental delay rather than being specific to autism? Most studies that have tackled this issue have found an imitative deficit in children with autism compared to those with global developmental delay (usually of unknown origin). Rogers *et al.* (1996) employed young people with a range of specific developmental delays and still found a group difference. Some studies (Sigman & Ungerer, 1984; Charman *et al.*, 1997) have shown that the children with mental retardation perform at an intermediate level between those with autism and controls, while some report a correlation between verbal mental age (VMA) and imitative ability (Sigman & Ungerer, 1984; Stone *et al.*, 1997; Royeurs, Van Oost, &

Bothuyne, 1998). VMA was suggested to account for 11% of the variance by Smith and Bryson (1998). However, Stone *et al.* (1990), did not find an imitative deficit among either a hearing-impaired or language-delayed group.

An alternative explanation is that widespread, coincidentally co-morbid motor co-ordination difficulties, cause the deficit. Green *et al.* (2002) found that their entire autism group met the criteria for DCD and still performed worse on tests of NMG imitation than the controls who also had a diagnosis of DCD. This would be in accord with the larger study of Smith and Bryson (1998) who found there was still a group difference after controlling for dexterity; and Rogers *et al.* (1996) who found that the autism group had no difficulty with the motor control tasks.

In conclusion, it seems that non-specific neurodevelopmental delay and motor skill impairment can account for some impairment but by no means all of it. As VMA correlates with both, all three factors may be indicators of another common parameter important in brain development, but similarly motor impairment in autism may have a different cause to that in DCD. This will be discussed further below. Finally, Charman *et al.*'s (1998) finding suggests that the more mildly affected "sub-syndromal" individuals show a mild imitation deficit, raising the possibility that severity of deficit could correlate with severity of disorder.

Delay or Deviance?

Some differences between performance on different types of imitative task have appeared consistently in the studies described whilst in others results are apparently contradictory. The studies have used children of a wide range of ages and varying control groups. A closer examination of the findings may be helpful in further describing the course of imitative development in autism.

Rogers *et al.* (1996) and Green *et al.* (2002) found that adding meaning to gesture facilitated performance among their older groups of patients, in contrast to the findings of Smith and Bryson (1998), whose normal controls were either much younger or were language impaired, and of Stone *et al.* (1997), who studied much younger preschoolers. This suggests that the development of language might be necessary to utilise the meaning of a gesture to facilitate imitation, and that such an ability improves with age.

As mentioned, reversal errors have been seen in many studies of children with autism, but Ohta (1987) reported equal numbers of reversal errors to be present in the normal young pre-schoolers in his sample. In addition, in other studies (Morgan, Cutrer, Coplin, & Rodrigue, 1989; Libby Powell, Messer, & Jordan, 1997) there was an absence of group differences when groups were matched on VMA but were far apart on CA, and both Whiten and Brown (1999), and Royeurs *et al.* (1998) found that group differences diminished in the older population. Finally, if the negative findings of Morgan *et al.* and Charman and Baron-Cohen (1994), are compared with the positive findings of Charman *et al.* (1997) and Rouyers *et al.* (1998), it can be seen that the studies used very comparable methodology and the differences lay in the chronological ages of subjects. Therefore, it seems that autism is characterised by a *delay* of normal imitative development (Whiten & Brown, 1999) rather than an absolute deficit, a conclusion paralleling that of Baron-Cohen, (1989) in relation to theory of mind, which also appears delayed rather than existing as a fixed deficit.

Are Some Types of Imitation More Susceptible to Impairment?

A perusal of Table I (particularly with regard to Hammes & Langdell, 1981; Sigman & Ungerer, 1984; Royeurs *et al.* 1998; and Aldridge *et al.* 2000), shows that imitation of NMGs generally produced greater group differences than for AO tasks. However, it is likely that imitation of AOs can utilise mechanisms of social learning other than imitation (see Whiten and Ham, 1992, for a classification of types of social learning). The observation of a meaningful object (or gesture) might trigger the release of a previously rehearsed motor-programme, or the observation of a desirable outcome might lead the observer to reach this goal using their own problem-solving ability (emulation). Similarly, objects can have "affordances", in the sense that a button "invites" being pressed and a handle "invites" being picked up. As such, Hammes and Langdell's findings, that children were more inclined to carry out the action they associated with an object rather than that which they saw performed, suggests that other learning mechanisms were used in preference to imitation. In the study by Aldridge *et al.* (2000), the children may have used the objects' affordances to complete the tasks. Therefore, where older children

might use their knowledge of an action's meaning to counter an imitative deficit, younger children may similarly use affordance learning in AOs. Imitation itself is therefore required especially for copying NMGs, where there is no obvious goal, affordance or associated knowledge of the action.

In the various "Meltzoff" tasks used by Charman *et al.* (1994, 1997), and Royeurs *et al.* (1998), most tasks use actions which are unlikely to be novel to the observer, such as pulling something apart or pushing a button. This means that they could be also be completed using techniques that call for little imitative ability. Those tasks associated with the poorest copying (such as pressing the button with the forehead or transferring a ring between branches of a toy tree) did appear to use more novel actions, though it is always difficult to ascertain true novelty, beyond testing for baseline response tendencies. In contrast, Whiten and Brown (1999) found reduced fidelity of imitation of two alternative methods of opening an "artificial fruit" among young children with autism. This is the strongest evidence of an imitation deficit of actions upon novel objects. Therefore, it seems that some of the tasks in these studies have assessed types of social learning other than imitation but that where the latter has been assessed as well, imitation has been affected to the greater extent.

Does this Meta-analysis Suggest the Underlying Mechanism?

A number of hypotheses have been advanced over the years to account for the imitative deficit seen in autism. It has been suggested that it results from:

(1) A deficit in representational or symbolic functioning (Curcio, 1978).
(2) Poor engagement in the experimental tasks by the autism group (Trevarthen & Aitken, 2001).
(3) A long-term deficit in social interaction that leads to less practiced motor skills (Tantam, 1991).
(4) A dyspraxic problem (Jones & Prior, 1985).
(5) A disorder of action representation (Smith & Bryson, 1994).
(6) A specific deficit in self–other mapping ability (Rogers *et al.*, 1991).

The evidence presented above is not consistent with the first three hypotheses.

With respect to the first hypothesis, it is clear that meaning either facilitates or has no effect on performance. With respect to the second, Trevarthen and Aitken have proposed a model of autism based on the notion that these children have diminished motivation to interact socially. Such a model would predict equally poor performance on easy and difficult imitation tasks. However, these studies have consistently demonstrated that children with autism engage well with the simple tasks and indeed one study (Libby *et al.*, 1997) found the children with autism to be the least negative. On more difficult tasks, rather than becoming disengaged, the children make errors. Finally, in those studies involving control tasks (Rogers *et al.*, 1996; Smith & Bryson, 1998) subjects do not perform worse than controls on gesture recognition or memory. This also argues against a disinterest in the subject matter as the cause of the group difference. The third hypothesis is similar in suggesting that the deficit is a result of less practiced motor skills because of less social interaction. An example would be poor ball-throwing skills because of reduced reciprocal play (Tantam, 1991). If this was the sole reason for the imitative deficit, it should not be apparent at an early age and become more evident with development. This reverse seems to be the case (Whiten & Brown, 1999).

Another possibility (fourth hypothesis) is that children with autism do not have so much of a difficulty with imitation, as with generalised motor-planning and execution difficulties (Smith & Bryson, 1994; Rogers, 1999). However, Green *et al.* found that the children with an autistic disorder performed worse than those with dyspraxia. Motor-planning difficulties are a well-recognised feature of autism (Hughes, 1996) and are concerned with the execution of motor programmes in response to instruction or an attempt to reach a goal, rather than imitation. However, there is clearly overlap between imitation and motor execution. Williams *et al.* (2001) have suggested that through shaping neural mechanisms matching actions to their perceived counterparts, imitation may play an important and pervasive role in the development of motor skills and planning ability. Griffith, Pennington, Wehner, and Rogers (1999) found that their tests of executive function did not detect group differences between children with autism and controls until the age of 4 years, whereas studies discussed above show the imitative deficit to be present earlier, at the age of about 20 months. This is in keeping with the idea that executive dysfunction is secondary to a core imitative deficit, rather than *vice versa*. A final argument against this hypothesis is that it does not explain

why meaningful objects or gestures should facilitate the imitation process among children with developed linguistic ability.

The fifth hypothesis points to deficits in action representation. Whilst Bartak, Rutter, and Cox (1975) showed that children with autism showed poorer understanding and expression of gesture than children with specific developmental receptive language disorder, Smith and Bryson (1998) found no group difference in the recognition of postures and sequences. This suggests that the deficit is at a different level from the simple representation of action.

We suggest that the evidence presented is most consistent with sixth hypothesis: a specific imitation deficit reflecting deficits in self–other mapping. One particular finding consistent with a self–other matching deficit and reported in a number of studies (Brown 1996; Hobson & Lee, 1999; Ohta, 1987; Whiten & Brown, 1999) is the presence of reversal errors. In these, the basic components of the imitation are correct but subjects seem to be unable to alter the perspective accordingly. A self–other mapping problem offers, we suggest, the most parsimonious explanation.

Sequential Imitation

Another consistent finding (from two of the most methodologically thorough studies (Smith & Bryson, 1998; Rogers *et al*., 1996) which poses more of a puzzle, is that in imitating sequential meaningful gestures, the gap in performance between autistic individuals and controls widens considerably in comparison to imitating single meaningful gestures. However, the gap does not change when imitating *sequences* of NMG. An explanation may be that as one moves to a sequence, the task becomes novel and meaningless even if its components are meaningful, since it is the sequence rather than the components, which is being imitated. In such a case, calling on rehearsed action as a compensatory mechanism, as described above, collapses. Strings of NMGs will remain equally difficult as single ones but for the meaningful gestures, the task gets much more difficult than with single instances.

Excessive Imitation

The evidence for an imitation deficit in autism may seem at odds with the abnormal, ''enhanced'' imitative behaviours that are well-recognised features of autism. Such behaviours include excessive and stereotyped patterns of mimicked movement and vocal expression including echolalia. This could reflect the lack of connection between research into elicited imitation and patterns of spontaneous imitation, highlighting the need for the relationship to be explored, particularly in relation to *level* of imitation. When children show spontaneous echolalia or imitative gesturing, do they also have an elicited imitative deficit at that same level of imitative development or higher? It may well be that excessive imitation exhibited in a unimodal manner divorced from context, is actually reflecting a delay in imitative development. The latter includes developing the ability to integrate vocal, affective and motor expression in the imitation of an appropriate and possibly novel goal, identified through recognising others' intentions.

Relation to Neurocognitive Research

It may be helpful to place these findings in the context of recent developments in imitation research which have begun to explore the different neural pathways that may be involved in imitation. Vogt (2002) has drawn the distinction between imitation which involves early and late mediation. In late mediation, imitation succeeds the formation of a cognitive representation of the act to be copied. In early mediation, motor cortical structures directly influence visual processing (and vice versa) through the use of visuomotor couplings or ''mirror neuron'' mechanisms (Rizzolatti, Fogassi, & Gallese, 2001). Such ''couplings'' can take a variety of forms whereby the perception of different aspects of perceived actions is influenced by existing motor cortical representations. In particular ''either the elementary properties of an observed action specify the [motor] response in a piecemeal manner'', or ''a compound visual representation of a seen gesture activates an appropriate action'' (Vogt, 2002, p. 212). Rizzolatti *et al*. (2002) have found that ''mirror neurons'' can show high or low congruence. Highly congruent mirror neurons fire only on observation of an action such as a precision grip on a small object and when that same specific action is executed. Low congruence mirror neurons are less specific in matching observed actions to executed ones. Rizzolatti *et al*. (2002) suggest that highly congruent mirror neurons are required for matching

the compound representation of an action, whereas low congruent ones are involved in more piecemeal processing. Object-oriented imitation may simply consist of replaying the changes that actions have produced on the object (Byrne & Russon, 1998), and so it follows that object-oriented imitation might possibly be utilising low congruence visuomotor couplings and a semantic knowledge of object properties. In contrast, action-oriented imitation may be more dependent upon high congruence "mirror neuron" functions.

This might mean that autism is associated with predominantly low congruence visuomotor couplings, which could fit with the pattern of findings discussed in this review; relative sparing of object-oriented imitation and affordance learning, but with action-oriented imitation delayed in its development. To speculate, a factor which may affect the development of congruence could be apoptosis or programmed cell-death. Such cell-organising functions are possibly impaired during infancy in children with autism (Bailey *et al*., 1998; Fatemi & Halt, 2001; Fatemi, Halt, Stary, Realmuto, & Jalali-Mousavi, 2001).

CONCLUSION

We have reported an analysis of 21 studies, spanning nearly 30 years and involving 281 children with ASD. Much new and informative data have been generated in the last 5 years. The size of the imitative deficit is considerable and is most apparent in younger age groups. It is characterised by difficulty with NMGs more than with familiar, meaningful actions or actions with objects, and also in difficulty with reversal. The pattern is consistent with a delay in imitative development coupled with relative preservation of object-oriented imitation and linguistic mechanisms that may be used to compensate. The core problem could possibly be due to selective impairment of "high congruence" visuomotor coupling mechanisms. The imitative deficit seems to be specific to autism, as long as perceptual and motor capacities are intact, but performance in some clinical conditions such as Attention Deficit Hyperactivity Disorder still needs to be examined.

Further work is also required to clarify whether certain imitative tasks can better discriminate between autism and dyspraxia, and standardised autism diagnostic processes need to establish how the imitative impairment relates to severity. Further understanding of the neural bases of social learning may then productively relate to this line of research, to throw light on the neural basis of the disorder.

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