CHAPTER 12

Imitation in Autism

Findings and Controversies

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The lack of natural, spontaneous imitative and interpersonally coordinated movements one observes when interacting with people with autism is striking. Yet, this aspect of autism is often camouflaged by the overall lack of social and emotional reciprocity and interpersonal engagement that is fundamental to autism. To what extent these two areas of difficulty may be related is a question that has barely been addressed in autism research to date.

The possibility that primary problems in imitating others could be a significant contributor to the social and learning deficits in autism was first suggested over 25 years ago, by Marian DeMyer (DeMyer et al., 1972) in the first comparative study of imitation in autism. Accumulating evidence of imitative deficits in autism was documented in an important research review (Prior, 1979) but ignored in cognitive theories of autism through the next decade. The lack of attention to imitation in the autism world is somewhat surprising, given the emphasis that major figures in psychology had placed on the roles of imitation, both immediate and deferred, in child development, among them Piaget (1962), Baldwin (1906), Skinner (1957), and Bandura (Bandura, Ross, & Ross, 1963). The landmark early imitation studies of Meltzoff and colleagues (Meltzoff & Moore, 1977, 1989) during the late 1970s and 1980s might have stimulated interest in imitation as a contender in the search for primary neuropsychological deficits in autism. However, the reorientation of autism theorists to the social aspects of autism (Fein, Pennington, Markowitz, Braverman, & Waterhouse, 1986) and the exciting new findings of Baron-Cohen, Frith, and colleagues (Baron-Cohen, Leslie, & Frith, 1985) focused

attention on theory of mind and related, more mature capacities as a primary explanation for the cognitive aspects of autism.

However, at the same time, papers began to appear that documented autism-specific developmental abnormalities in social cognitive skills that developed years before theory of mind. This pioneering work, led by Marian Sigman and a succession of graduate students and postdoctoral fellows, including Judy Ungerer, Peter Mundy, Connie Kasari, and Nurit Yirmiya, documented autism-related abnormalities in joint attention behavior, dyadic emotional responsivity, imitation, and symbolic play in preschool children (Kasari, Sigman, Mundy, & Yirmiya, 1990; Mundy, Sigman, Ungerer, & Sherman, 1987; Sigman & Ungerer, 1984; Ungerer & Sigman, 1987; Yirmiya, Kasari, Sigman, & Mundy, 1989). This work helped refocus the field to look for markers of autism that developed much earlier than theory of other minds.

In 1991, Rogers and Pennington suggested a developmental model of autism in which early cascading social–communicative impairments might stem from early deficits in motor imitation, affecting emotional mirroring and sharing and impeding growing awareness of the other as a subjective psyche. Such a cascade, they suggested, would severely affect the development of joint attention, verbal and nonverbal communication, and symbolic play. Furthermore, they suggested a brain–behavior link, hypothesizing that prefrontal cortex might play an important role in performance of motor imitation, and in autism-related difficulties, both in intentional imitation and in other executive acts.

Arguments for considering imitation as one possible primary deficit in autism must address four issues both in the theories and in empirical studies. First, if imitation is a building block of typical social development and a primary influence in autism symptoms, there should be supportive evidence of these interrelationships across time and across developmental acquisitions, in both typical development and autism. Second, studies of behavioral differences in autism need to consider the many levels or relations between a behavioral act and a neurobiological starting state difference. Third, imitation is not one behavior. Rather, there are a variety of different behaviors or skills that may be involved, as described in a taxonomy of matching behaviors developed by comparative psychologists that needs to infiltrate autism imitation research (Want & Harris, 2002). Finally, imitative deficits documented in lab studies need to be reconciled with imitative phenomena such as echolalia and imitative acts reported by parents and observed by clinicians. Understanding imitative performance in autism will require that we can explain the general imitative deficit while accounting for imitative performances.

To address these topics, we (1) review the evidence for a central imitation deficit in autism, (2) review existing theories that seek to explain the imitation problems and their supportive evidence, (3) integrate autism findings into comparative psychology's taxonomy of "imitative" behaviors, (4) examine evidence of brain–behavior relations regarding imitation in autism, and (5) direct attention to questions that need additional research.

FINDINGS FROM CONTROLLED STUDIES OF IMITATION IN AUTISM

We draw from the most recent and comprehensive review of imitation research in autism covering literature to 2002 (Williams, Whiten, & Singh, 2004), as well as the most recent findings from the empirical literature. The Williams and colleagues (2004) article provided the field with an exhaustive review that identified 124 articles concerning autism and imitation and reviewed in depth 21 controlled studies of hand or body movement imitations in autism that had been published up to March 2002. The review paper first considered the overriding question, "Is there an imitative deficit in autism?" Of the 21 studies, two found no group differences in an adequately designed study. Two more that reported no group differences were confounded by ceiling effects. Three studies did not report the statistical tests necessary for answering this question. The remaining 14 studies reported an autism-specific deficit, generally at very high levels of significance even though the groups were small. Thus, 14 of 16 methodologically adequate studies have reported an autism-specific deficit in imitation of body or hand movements. Furthermore, Williams and colleagues pooled the findings from these studies involving 281 subjects with autism spectrum disorders, using the Logit method, resulting in a combined p value of $p = .00005$ ($n = 248$ subjects, $t = -4.260$, 89 *df*).

Next, the authors considered whether the imitative deficit might be due to a secondary cause. They examined studies in which comparison groups had other clinical conditions, including undifferentiated mental retardation, Down syndrome, language impairment, and developmental dyspraxia. Each of these studies found significantly greater impairment in the group with autism than in the other clinical group. The authors concluded that there was widespread evidence of a specific impairment in imitation in autism.

Studies That Have Occurred Since 2002

Eight controlled studies of imitative performance, not previously reviewed and published since the Williams and colleagues (2004) review, are briefly reviewed here.

Bernabei, Penton, Fabrizi, Camaioni, and Perucchini (2003) compared a large group of 46 preschoolers with autism and very severe intellectual impairments to 45 age-mates with similar intellectual impairments on the Užgiris–Hunt scales (Užgiris & Hunt, 1975). The children with autism were significantly more mature than comparisons on the four object-oriented scales: object permanence, means–end, causality, and spatial relations. Intragroup examination of scores revealed an autism-specific deficit in the imitation subscales compared to the object-oriented subscales. The group with intellectual disability showed similar level of performance on all the scales (with lower performance on vocal imitation) and strong intercorrelations across the areas. In contrast, the group with autism demonstrated a statistically significant

weakness in imitation (with equivalent performance on gestural and verbal imitation) compared to their other scores. Furthermore, in the autism group, vocal imitation did not correlate with any other performance. Thus, the group with autism showed a relative weakness in imitation and a lack of overall integration of skills across the object and social (imitative) domains compared to the contrast group.

Ingersoll, Schreibman, and Tran (2003) examined the performance of 15 young children with autism, ages 23–53 months, and 14 typical toddlers, ages 16–32 months. This study examined imitative performance under varying object conditions. Three pairs of novel simple objects were carefully constructed. Each afforded a simple and familiar sensorimotor action, with half the objects provided visual and auditory sensory stimuli when the children carried out the action in imitation of the adult. Between-group performance did not differ significantly in either condition on these simple actions, though ceiling effects and very familiar acts may have masked underlying imitative differences. However, unlike the comparison group, children with autism showed a significant deficit in their imitative performance in the nonsensory feedback condition compared to the sensory condition. The authors interpreted the findings as demonstrating that imitating other people is intrinsically motivating to children who do not have autism but is less so for children with autism.

Avikainen, Wohlschlager, and Hari (2003) compared the performance of eight adults with Asperger syndrome or high-functioning autism and typical IQ with eight typically functioning adults on a simple object imitation task. In the task, the subject sat across from the experimenter. In front of each adult were two cups of different colors, with a magic marker between the cups. The model picked up the marker with one of two grip patterns (ulnar or radial) and inserted it into one of the two cups. The subject was told to either imitate the model as if the model was a mirror image or to imitate the model with the same-sided hand, which necessitated crossing the body to carry out the task. The typical group demonstrated an advantage in the mirroring condition as seen in fewer errors in all three target behaviors (hand, cup, grip) across the 80 trials. However, the group with autism showed no such advantage and differed from the typical group only in this condition, particularly in errors made involving hand choice or grip position. Given the very small group sizes and the huge error rates from these high-functioning adults (15–20%) on hand choice and cup choice, a replication with a larger sample will be extremely helpful for interpreting these findings.

Meyer and Hobson (2004) examined a particular aspect of imitation: self–other orientation (identification) in a study of 16 older children with autism and moderate mental retardation, and 16 comparison children matched on age and IQ. There were four object-oriented tasks like rolling a wheel and stacking objects. A line in front of the child and in front of the experimenter, who sat on the floor facing the child, marked each person's "personal space." Each task was demonstrated once in the child's "space" and

again in the adult's "space." After each model, the child was given the toy and encouraged to imitate. The children with autism made significantly fewer responses that modeled the self–other orientation to the object than did the comparison subjects. The patterns of relationships across groups also differed, with the delayed children demonstrating significant correlations between IQ and placement patterns that were absent in autism. This finding is consistent with data from several other studies demonstrating an autism-specific difficulty in correctly imitating the orientation of an action in relation to the model's body (e.g., Avikainen et al., 2003; Ohta, 1987; Smith & Bryson, 1998). Meyer and Hobson (2004) interpret this as a failure of identification and draw parallels between performance on this task and ability to understand others' perspectives and to mentally shift from one perspective to another. These authors are drawing attention to and replicating a very interesting phenomenon in autism imitation that needs to be studied and explained.

Rogers, Hepburn, and Stackhouse (2003) examined the performance of 24 2-year-olds with autism, compared to well-matched samples of children with fragile X (n = 18), other delays (n = 20) and typical development (n = 15) on three types of imitative tasks: gestural, oral–facial, and novel object imitations. The study was designed to examine (1) profiles of imitative performance across groups and (2) relations between imitative performance, other aspects of the autism phenotype, and tasks of skilled motor planning and execution. Significant relationships were found across the three types of imitation tasks, indicating a general imitative skill underlying all tasks. Children with autism showed a deficit in imitation in relation to both comparison groups, with the most deficient performances on the oral–facial and, surprisingly, the object-oriented tasks and their imitative performance correlated strongly with severity of autism symptoms. There was also a significant relation with initiation of joint attention behavior, and with language development. However, contrary to hypotheses, there was no autism-specific deficit on the motor planning/execution tasks, nor did performance on those tasks relate to imitative performance for the children with autism. Interestingly, the imitative performance of children with fragile X syndrome was strongly related to the presence and severity of their autism symptoms.

Bennetto (1999) examined five components of imitation in high-functioning children with autism (n = 19), compared to children with developmental dyslexia (n = 19) matched on age and verbal IQ. Specific tasks and within-subject experiments assessed basic motor functioning, body schema, dynamic spatiotemporal representation, memory, and motor execution of nonmeaningful hand and arm gestures. Consistent with previous research, participants with autism demonstrated overall worse imitation skill. Further analyses revealed a specific pattern of impairment characterized by difficulty with the kinesthetic aspects of postures and movements, particularly during complex sequences. Participants with autism also demonstrated impairments in basic motor skills, which appeared to account for some, but not all of their imita-

tion deficits. Participants with autism did not differ from comparison participants on body schema, spatiotemporal representation, or memory, suggesting that their imitation deficits were not secondary to problems in these areas.

McIntosh, Reichmann-Decker, Winkielman, and Wilbarger (in press) reported a psychophysiological study of automatic facial imitation, or mimicry, carried out with 14 high-functioning adolescents or adults with autism spectrum disorders and 14 typically developing age- and vocabulary-matched comparison subjects. Subjects viewed photos of eight happy and eight angry photos in two conditions: observation only and intentional imitation. Electromyographic recordings from brow and check muscles revealed an autism-specific impairment in specificity of the automatically mimicked emotion but no group difference in the intentional condition (though the response measured was muscle activation, not accuracy of imitation or normalcy of the expression).

Finally, Scambler and colleagues (in press) reported a study of automatic facial imitation involving affective expressions in 26 2-year-olds with autism, 24 children with other delays, and 15 children with typical development to four different emotional expressions of adults occurring in naturalistic displays (fear, joy, disgust, and distress). Microanalytic scoring techniques were used to rate intensity, hedonic tone, and latency of child response. Children with autism demonstrated fewer episodes of matching responses than did the comparison groups, and when there were matching responses, the intensity of their emotional expressions were much less intense, or more muted, than the two comparison groups.

Thus, consistent with a large number of previously reviewed studies, eight recent controlled studies of imitation demonstrate autism-specific difficulties in subject groups ranging from toddlers and severely disabled preschool children to adults with Asperger syndrome. The evidence continues to support the existence of a central deficit in motor imitation in autism. In addition to impairments in intentional imitations, we have two reports of impairments in automatic imitative responses as well. However, describing a problem does not explain it. What accounts for the imitative problems in autism? We next consider the major theories accounting for the imitative deficit in autism, and the existing support for those theories. We begin by briefly mentioning theories that have been suggested in the past but can be rejected based on the current body of evidence.

EXPLANATORY THEORIES OF IMITATION PROBLEMS IN AUTISM

Explanations for the imitation deficits in autism fall into five main areas: problems in representing the target action, problems executing the target action, problems with attention to the target action, problems with cross-modal integration of perceptual information, and problems with the motivation for producing the target action. We review each in turn here.

Impairments in Representation of the Target Act

The well-known difficulties that children with autism display in symbolic play and in language development have led many to hypothesize autism-specific problems in forming and manipulating representations. Several different kinds of representational problems have been suggested to underlie imitation difficulties, including problems with the symbolic nature of some of the tasks, problems representing the motor movements involved, problems representing one's body, and problems in coordinating representations between self and other. Each of these is discussed in turn.

Symbolic Representations

Earlier theories of imitation problems in autism focused on the more symbolic aspects of representations and the links between pantomime, imitation, and symbolic play (Curcio, 1978). Linking these skills together is supported both by cognitive theories of child development and by clinical studies of patients with apraxia (Heilman, 1979). However, several studies specifically tested this hypothesis and found no supportive evidence. As reviewed in Williams and colleagues (2004), two groups found that symbolic content improved the imitative performance of children with autism (Rogers, Bennetto, McEvoy, & Pennington, 1996; Stone, Ousley, & Littleford, 1997) and others have found no impairment related to symbolic representation (Smith & Bryson, 1998). Thus, no support has been found for a symbolic representation as the source of the imitative deficit.

Motor Representations

Another aspect of representation that might be involved, however, involves motor representational capacity. Three studies have specifically examined whether people with autism form accurate representations of the target movements and hold them on line in working memory for sufficient periods to act on them (Bennetto, 1999; Rogers et al., 1996, Smith & Bryson 1998). Bennetto's (1999) work is particularly informative here. Children's ability to discriminate correct from incorrect video models, with variable time delays, was carefully examined, with no evidence of autism-specific problems. No autism-specific differences in the ability to form mental representations of movements and hold them on line have been identified thus far, and thus this hypothesis lacks support.

Self-Representation

A further motor concept that arises has to do with representation of one's own body. There is a clinical hypothesis that children with autism lack appropriate awareness of their own bodies. Two studies provide some data on this.

Bennetto (1999) used a neuropsychological task specifically focused on representation of one's own body (Semmes, Weinstein, Ghent, & Teuber, 1963). There were no autism-specific group differences on accuracy of identifying points on one's body related to those on a two-dimensional model, compared to a carefully matched clinical group of children. However, this task is concerned primarily with self-concept of static locations rather than ability to represent dynamic changes in limb positions in relation to one another. A recent electroencephalography (EEG) study of response to self-generated versus other's generated movements did not detect any difference in mu wave suppression between the autism group and comparison group in response to self-generated hand movements (Oberman et al., 2005) Thus, we currently lack data that support this hypothesis.

Self–Other Representation or Mapping

Two theories have been put forward concerning difficulties with coordinating representations of self and other: the self–other mapping theory and the identification theory.

The self–other mapping hypothesis arose in 1991 by Rogers and Pennington, who suggested that an imitation deficit involving seeing "the other as a template of the self," or self–other mapping of representations, might be a primary behavioral/neuropsychological deficit in autism. Williams, Whiten, Suddendorf, and Perrett (2001) expanded on this idea, suggesting that mirror neurons could provide a neural mechanism underlying such a deficit. Several papers published in the last few years have used methods to examine self–other mapping.

Avikainen and colleagues (2003) isolated a phenomenon that had been previously reported but never before directly examined in various autism imitation studies—problems with direct mirroring of the model (other groups that have also identified this problem include Bennetto, 1999; Meyer & Hobson, 2004; Ohta, 1987; Smith & Bryson, 1998). The specificity of the imitation problem in these studies allows us to consider a very fundamental problem involving neuron systems that allow for direct mapping of specific movements between humans. At least in animal research, neurons involved in perceiving and executing action appear to have this level of specificity. Jellema, Baker, Oram, and Perrett (2002) report that, in the macaque, neurons in superior temporal sulcus (STS) are quite specific in their response to movements of individual body parts: mouth, eyes, head, legs, and so on, and specific in their response to specific actions: walking, climbing, crouching, etc. They suggest that different cell populations appear equipped to break down complex motor acts into their basic components, both in detection and in performance. Examination of mirror neuron activation in autism has begun and is summarized in the section on brain–behavior relations. At the current time, this hypothesis is still considered viable, with initial support from both brain imaging and neuropsychological studies.

Identification Theory

Hobson (Hobson & Meyer, Chapter 9, this volume) offers a contrasting interpretation that appears to challenge the self–other mapping perspective, suggesting that "identification" is the key deficit in autism. These two concepts appear quite similar; however, there are subtle but distinct differences. Identification refers to individuals recognizing aspects of themselves that are the same within other individuals. In contrast, mapping refers to establishing correspondences rather than sameness.

In development, the origin of these two processes might be very different. Correspondence starts from an assumption that two individuals are separate but bear some resemblances. The concept of self–other mapping appears to involve capacities seen in young infants' abilities to detect correspondences among stimuli, even across sensory modalities, and precedes infants' capacities for identification, which appear to require development of more advanced representational abilities. Identification may develop from earlier capacities to detect self–other similarities and mappings, but identification is unlikely to be a prerequisite for self–other mapping.

To summarize, hypotheses concerning autism-specific difficulties in representation of target actions lack empirical support. Hypotheses concerning autism-specific difficulties with representation of one's own body have just begun to be studied and thus can neither be accepted or refuted at this point. There is evidence from a number of studies to support difficulties in *coordinating* representations of self and other's movements, and this is a promising area for further investigation.

Motor Execution Problems

The suggestion that the imitation deficit in autism could reflect a neurologically based difficulty with producing the movements per se was first suggested by Damasio and Maurer (1978). There is a body of evidence (see Dewey & Bottos, Chapter 17, this volume, for a review) that documents abnormal movements, muscle tone, and balance in persons with autism compared to those with other conditions or with typical development.

Several methodological approaches have been used to test the hypothesis that difficulties with motor imitation may be due to more fundamental difficulties in producing precise and well-coordinated movements. One approach uses clinical comparison groups who are also known to have motor production problems. Many autism imitation studies have used clinical comparison groups (for examples, see Rogers et al., 1996; Sigman & Ungerer, 1984; Stone et al., 1997). In each of these studies, a comparison group of participants with another clinical condition (usually intellectual handicap) has been matched to the experimental group with autism in terms of age and IQ functioning, with occasional matching for motor development as well.

The findings from these studies are somewhat inconsistent and reflect the nature of the groups being studied. When high-functioning subjects with autism are used, and are compared to typically developing controls, autism-specific motor deficits are generally found. However, when lower-functioning and younger subjects are examined and are compared to subjects with equivalent levels of mental retardation, then autism specific motor differences are not necessarily found. Several groups have demonstrated that toddlers and preschoolers with autism demonstrate fine motor skills that are no more impaired than the clinical comparisons (e.g., Rogers et al., 2003; Stone et al., 1997). However, a legitimate criticism of this approach is that the children with autism may have a type of motor difficulty more severe than comparison children that is not being adequately measured or adequately controlled for in the design.

A second methodological approach has involved choosing a comparison group with known motor impairment, like developmental coordination disorder, and matching it to the autism group on motor measures. If the children with autism demonstrate poorer imitation performance than controls, a motor impairment hypothesis cannot really account for the findings. This approach has been followed in only one study (Green et al., 2002). These authors found imitation performance among individuals with autism spectrum disorder (ASD) to be worse than those with developmental coordination disorder.

A third methodological approach has involved measuring children's motor performance directly on tasks that tap motor coordination and examining the relations between the motor performance and imitative productions. This approach has been used by several different experimental groups (Bennetto, 1999; Rogers et al., 2003; Smith & Bryson, 1998) all of whom demonstrated that the diagnosis of autism continued to be related to imitation performance, even after the variance associated with motor performance was removed. A statistical point to note is that partial correlations and multiple regressions assume that the confounding variable has an equal effect on the variance of each group. However, at least some groups report that imitation has a different relationship with motor skills in autism than it does in control groups (see Rogers et al., 2003). Therefore, the approach of controlling for variance in motor ability may not fully address this concern. To summarize, the few studies that have addressed this hypothesis suggest that while motor impairment affects imitation performance, it does not fully account for the autism imitation deficit. This is an area in which much more research is needed.

Difficulties with Attentional Flexibility

Many structured imitation batteries require multiple shifts in visual attention, even in single-action stimuli, which require shifts from the model's face to an object and then to the model's body. Children with autism have been shown to have difficulties with attentional flexibility (Ozonoff, Strayer, McMahon,

& Filloux, 1994) and may have unusual attentional foci during social interaction tasks (Klin, Jones, Schultz, Volkmar, & Cohen, 2002).

Given the well-known lack of typical eye contact and attention to others, imitation studies need to determine whether they have the visual attention of their subjects. While a few studies have tried to examine this concept (e.g., Rogers et al., 2003), ensuring that a subject is attending does not guarantee that the subject is looking at the relevant aspects of the movement. As eye-tracking studies of autism are teaching us (Klin et al., 2002), visual attention may be focused on something other than the critical stimulus, even with attentive subjects. It will be quite important for future studies to monitor gaze within imitation studies in order to provide better information on orienting, visual attention, and gaze shifts during imitation tasks.

The relationship between visual attention and imitation in autism is likely to be complex. As discussed elsewhere (see Williams & Waiter, Chapter 15; Decety, Chapter 11; Mon-Williams, Chapter 14, this volume), visual attention and motor activity are usually tightly coordinated. It is quite possible that eye movement patterns can be shaped by repeated experiences of visual tracking of complex, goal-directed action sequences. Thus, repeated practice in joint attention and imitative exchanges may "train" the infant's attentional processes and foster attentional flexibility (Williams et al., 2001, 2006). This idea is supported by evidence from both McEvoy, Rogers, and Pennington (1993) and Griffith, Pennington, Wehner, and Rogers (1999), who found significant relationships between executive function and joint attention in young children with autism.

Cross-Modal Processing Abilities

Imitation requires that one coordinate visual–spatial information input from the partner's movements with proprioceptive and kinesthetic output regarding one's own body and thus rests on cross-modal information processing abilities. We lack a body of solid empirical information on the integrity of cross-modal processing in autism. Two recent studies have examined this problem in relation to imitation. As described previously, Bennetto's (1999) study found no differences on a task requiring a very similar kind of cross-modal transfer, from visual stimuli involving a two-dimensional representation of a body onto the child's own body. Williams, Massaro, Peel, Bosseler, and Suddendorf (2004) recently examined speech reading in children with autism, in which no deficits in cross-modal processing were documented. However, at this point, we do not have the data needed to accept or reject this theory.

Motivational Factors

Imitation deficits may be an epiphenomenon of a more general social disinterest in autism. This has been a popular way of discussing, or perhaps dismissing, the imitative findings in autism. The line of reasoning is this: People with

autism are less interested in other people than comparison groups, and thus look at them less, are less motivated to do what other people do, less motivated to cooperate with experimenters, and for all these reasons have less practice in imitating others, resulting in poorer performance on experimental batteries. This view has been described most recently by Trevarthen and Aitken (2001; see also Dawson et al., 2002; Zelazo, 2001).

Ingersoll and colleagues (2003) highlighted motivational aspects by manipulating object effects during imitation tasks. While this study provides an important reminder that motivational factors must be considered in every autism imitation study, other studies appear to indicate that the social motivation factor alone cannot account for the imitation performance deficit in autism. For example, a motivational problem would predict equally poor performance on easy and difficult imitation tasks (Williams, Massaro, et al., 2004). Yet the body of studies has consistently demonstrated differential performance patterns in autism based on task difficulty. Children with autism have been found to perform adequately on very simple imitative tasks (Charman & Baron-Cohen, 1994). Indeed, one study (Libby, Powell, Messer, & Jordan, 1997) found the children with autism to be less negative about performing imitations than comparisons. Even with very young children, Rogers and colleagues (2003) reported no differences between 2-year-olds with autism and two other developmentally matched groups on cooperative, contingent acts in response to a model. Another characteristic that may differentially affect motivation is the intentional nature of the task. Studies of automatic imitative processes (e.g., McIntosh et al., in press; Scambler et al., in press; Yirmiya et al., 1989) are probably not as dependent on motivation as tasks involving intentional gestural imitation. Thus, although the motivation question needs to be asked and considered in each study, it is also an area that can be addressed to some extent at the level of task design (including using methods to tap automatic processes and to enhance motivation) and examined in the analyses.

Another argument against the motivational hypothesis is that it suggests an experiential deficit accruing over time. Fewer imitative experiences day by day will over time result in increasing disparity of experience and lack of equivalent amount of practice in the wide range of skills that young children learn via imitation (Williams, Massaro, et al., 2004). An example would be poor ball-throwing skills because of reduced practice in reciprocal play (Hoon & Reiss, 1992). This leads to the hypothesis that very young children with autism should show less experiential imitative deficit, and that progressively older groups should show progressively larger deficits. However, imitation studies in autism find significant group differences at the earliest ages even when carefully controlled for visual attention and cooperative responses to the examiner (Charman et al., 1997; Kalmanson, 1987; Rogers et al., 2003; Sigman & Ungerer, 1984).

A final problem with the motivation hypothesis is that it assumes that imitation is a unitary, intentional phenomenon that results in some positive

interpersonal (and intrinsic) reward. Yet, different types of imitative behavior (to be discussed further later) lead to different consequences. Automatic imitation, or mimicry, is considered to be an unintentional behavior that occurs outside awareness, a relatively hard-wired phenomenon. Imitation used in the apprenticeship function involves intentional motivated acts to learn or accomplish a motivated skill, as in imitating someone's acts that open a candy box. The reward is in the accomplishment of a personal goal, and there is no basis for hypothesizing an autism-specific deficit in this type of motivation. The third type of imitation, intentional imitation of another's behavior in a social–communicative exchange like infant–parent games, could be considered less intrinsically rewarding for children with autism than others. However, the studies of response to being imitated conducted by Dawson and Galpert (1990) and Escalona, Field, Nadel, and Lundy (2002) have clearly demonstrated that children with autism enjoy being imitated, seek to continue the experience when it ends, and respond with increased imitation. The social imitative experience appears intrinsically rewarding to the children in these studies. Thus, while the motivational hypothesis needs to be considered, the construct of "motivation" itself seems too general to be very helpful in understanding autism-specific imitation difficulties. Much more precise hypotheses are needed.

To conclude this section, evidence supports theories regarding the contribution of both motor and motivational deficits in autism to imitative performance, but neither of these appears to fully account for autism-specific differences. Two aspects of representation of movement appear to be differentially affected: self–other orientation and affective quality of gestures. The evidence of specific difficulties with orientation of a movement in relation to the partner's body has been cited previously. Examination of "style," or affective quality of acts involving manual movements, has thus far only been reported by Hobson and colleagues. These aspects of "style" may well reflect affectively related automatic imitation, or mimicry, of body movements. Abnormal mimicry in autism is currently being reported. Studies of facial mimicry also demonstrate autism-specific deficits in affective mirroring of facial expressions (McIntosh et al., in press; Scambler et al., in press; Yirmiya et al., 1989).

The social variables involving attention to the model, experience and practice with imitation, and motivation to perform are critical variables and in some ways get to the heart of autism. Motivation to perform tasks needs to be addressed and examined in individual studies. Focus of attention needs to be examined with more sophisticated methods than have thus far been used. Experiential history is a very real consideration and may indicate the need for a training study or study in which groups are already matched on capacity to imitate movements precisely, to examine the role of additional processes affecting imitative performances. It does not appear at this time that the imitation deficit in autism is reducible to one simple deficient process. Imitating

another is a complex act with many degrees of freedom; currently the evidence suggests multiple affected subcomponents.

LESSONS LEARNED FROM COMPARATIVE PSYCHOLOGY: THE VARIETY OF WAYS THAT SOCIAL MODELS CAN INFLUENCE BEHAVIOR

One of the many contributions that the comparative psychologists have made to imitation research has been their careful dissection of the different kinds of "matching" behaviors. While researchers studying humans tend to use the generic term *imitation* to refer to any kind of matching behavior, comparative psychologists have differentiated at least five different kinds of matching behaviors that occur between conspecifics. As Want and Harris (2002) suggested, child researchers would be wise to learn these finer distinctions (as described by Whiten, Chapter 10, this volume). This careful taxonomy of copying behavior has been extremely helpful to the animal researchers who study social learning. What might we gain by applying this careful taxonomy of matching behavior to the autism imitation literature?

Mimicry and the Transmission of Affect

In the human literature, the term *mimicry* refers to automatic, (nonintentional) matching behaviors, particularly involving facial, postural, and gestural movements that occur rapidly and outside of awareness. The occurrence of mimicry in typically developing adults is well established in the social psychology literature; see Moody and McIntosh (Chapter 4, this volume) for a review. It can involve simple movements, as well as emotionally salient stimuli. The best-known examples are social smiling and yawning. Mimicry is closely related to emotional contagion, whereby the perception of another's expression of emotion elicits the same emotion in the observer (Hatfield et al., 1999).

There are only three controlled studies of mimicry in the published autism literature, all focusing on facial mimicry of emotional expressions. Two of the studies examined children's responses to experimenter's discrete emotion expressions. Both studies reported a deficient mimicry response (Scambler et al., in press; Sigman, Kasari, Kwon, & Yirmiya, 1992). The third controlled study examined the response of high-functioning adults to emotional displays delivered on a computer screen and measured using electromyography (EMG; MacIntosh et al., in press; see Moody & MacIntosh, Chapter 4, for a description). All three report autism-specific deficits in mimicry. In addition, tasks like Hobson's form versus function task (Hobson & Lee, 1999) can likely be included in this group. This study reported a large autism-specific deficit in mimicking the dynamic style of an object-directed action in two independent samples of people with autism.

These findings from four independent studies are exciting and provocative. A deficit in automatic mirroring of others' emotional behavior could have significant effects on social–emotional behavior, especially reciprocity and emotion expression via facial display and gesture. Mimicry is an important area for further study in autism, at both the behavioral and the brain levels.

Emulation

Emulation refers to completing an observed goal-directed task by achieving the modeled end state, but not necessarily modeling the specific behaviors used to achieve the goal state. Whiten further distinguishes between goal emulation and result emulation. In result, or end, emulation the end state is copied. In goal emulation, the individual copies what he or she considers to be the goal of the actor. Although there have not been studies in autism that specifically target result emulation, goal emulation has been investigated in several studies using Meltzoff's (1995) failed intentions task. The paradigm examines whether young children with autism can complete simple means–end tasks in both a modeled and in a disrupted, or failed, condition, in comparison to developmentally matched typical and/or clinical groups. Performance of the intended act in the failed condition demonstrates goal emulation.

Two published studies have demonstrated that the majority of children with autism complete the target task in the imitated condition (though in some of the studies they performed significantly fewer of these tasks than the comparison subject group), as well as in the failed condition (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001). In addition, several other studies have demonstrated lack of autism-specific differences in imitation tasks involving familiar and functional means–end acts on objects, like the Hobson studies. Thus, there does not seem to be evidence of autism-specific problems in goal emulation in the literature. It could be that intact goal emulation underlies many of the successful object imitation performances in autism involving simple (and usually familiar) acts.

Object Movement Reenactment or Intentional, Means–End Imitation

As discussed in depth by Whiten (Chapter 10, this volume) the distinction between emulation and imitation may be a fine one, as copying the action (imitation) and copying the effects of the action (emulation) may be indistinguishable. For example, Tomasello and colleagues (Nagell, Olguin, & Tomasello, 1993) used a rake task, in which subjects (toddlers and chimpanzees) observed an adult use a rake to retrieve an object in a box. In one condition the rake was turned prong-side down, and in the other condition, prong-side up—a less efficient way to use a rake to retrieve an object. This kind of task nicely separates copying means and copying goals but does not mean that

participants were imitating means–end behavior. They could simply be recreating the rake position and action. One might then ask if participants copied the overhand or underhand grip, but such a distinction is unlikely to be relevant to the action outcome. Whiten advocates introduction of the term *object movement reenactment* (OMR) to clarify the situation.

The number of papers reporting relative strengths in OMR compared to gestural or facial imitations might lead us to suspect that children with autism can carry out these tasks adequately. However, a study by Rogers and colleagues (2003) raises questions about this assumption, while also presenting a challenge to Whiten's classification. This study presented copying tasks to young children that required them to perform simple acts on objects, but the actions were novel in relation to the objects. They involved operating a squeak toy with the elbow, inverting a toy car and patting the underbody, and banging duplos together rather than stacking them. The car inversion, compression of the squeak toy, and duplo manipulation were clearly OMRs. Surprisingly, the children with autism demonstrated as impaired performance on these tasks as on the gestural or facial tasks. This recollects the Hammes and Langdell (1981) findings where the children would not imitate drinking from the teapot.

These findings suggest that the difficulty of bodily imitation in autism may indeed occur in object tasks as well, although the use of familiar or conventional acts or acts that are well-supported by the affordances of the object may mask this difficulty. An alternative interpretation is that OMR skills may be usually intact in autism but are susceptible to interference when they differ to those previously learned to be associated with the object being used. A methodological issue to note here is that identifying such problems requires a scoring system sensitive to the precision of the imitation: posture, the limbs used, directionality of the movement, timing of the action, orientation in space, and so on. While this level of coding may seem overly detailed, the precision of an imitation may have important effects in natural social interactions, in that a smoothly executed automatic mirroring movement may be so synchronous as to go almost unnoticed in an interaction, but rather "felt" as a natural social response, whereas an uncoordinated, poorly timed, or poorly reproduced movement may stand out as "odd" (see Stern, 1985, for a similar concept).

Gestural or Body-Level Imitation

How should intentional gestural acts be classified? Many of the autism imitation studies, perhaps the majority, have asked subjects to copy manual or bodily postures and movements and oral and/or facial movements or expressions. This kind of imitation differs from tasks in the mimicry paradigms in that in the intentional tasks, imitation is explicitly instructed and the resulting behavior is thus intentional rather than automatic. Response to instructed imitation is not typically part of the classification system used in the comparative

studies, and yet it occurs in humans in a very regular basis. In older children and adults it is frequent in learning situations, as in sports or dance lessons, learning musical instruments, and so on. In infancy and early childhood this kind of imitation is seen in parent–infant interaction games, and in toddlers and preschoolers in songs and chants involving finger or body movements, in imitation of actions on toys and outdoors in play.

As reported here, virtually every autism study involving gestural imitations, except for the work of Beadle-Brown and Whiten (2004), has reported autism-specific differences, and this includes studies that tap the full range of age groups and severity levels. Although most of the work has involved hand and body movements, those that include oral–facial movements also report autism-specific deficits. Again, a sensitive coding system is necessary to fully capture problems with this area. Clearly, persons with autism have great difficulties with this type of imitative behavior.

To conclude, the application of the comparative psychology classification system helps identify the nature of the imitative problems in autism with somewhat greater precision. The difficulty does not appear to involve understanding the intention of the model for the action, particularly involving objects. The imitative difficulty appears to center on mirroring of others' body movements, both automatic and intentional. To what extent these are independent is unknown. Although Rogers and colleagues (1996, 2003) demonstrated strong correlations across different types of simple intentional imitation tasks, Stone and colleagues (1997) revealed the opposite, and most studies of imitation have not provided the needed analyses. In terms of brain function, some neural mechanisms involved during imitation of purposeful use of objects may be independent of those involved in mimicry or in intentional bodily imitation. We need integrated studies involving behavioral science and neuroscience to help us drill down more deeply into the nature of these different types of imitative behavior.

BRAIN–BEHAVIOR RELATIONS INVOLVED IN IMITATION

The complex capacity for interpersonal imitation seen in human beings implies specific brain mechanisms evolved to support such capacities. How and why has such evolution occurred? This question was addressed early on by Bruner (1972), who observed the contributions of tool use, imitation, and play to cognitive development in humans. Byrne and Whiten (1988) argued that a selective advantage evolved from being able to manage and use information inherent to social complexity, and this advantage resulted in massive expansion of cerebral cortex over a period of about 2 million years. They consider three different social abilities—cooperation, deception, and imitation—as particularly important forces behind these evolutionary changes. Merlin Donald (1991) suggested four main epochs in social evolution of humans, with imitation, or mimesis, playing a fundamental role in the third epoch, in

which occurs the evolution of social–communicative behavior specific to our species. Drawing from the neurological and neuropsychological studies available at that time, he hypothesized a model of brain evolution of multiple structures with increasingly specialized roles in imitative behavior, which would require integration of multiple brain regions across both hemispheres.

Discovery of Mirror Neurons

In 1992, reports from single-cell physiological studies of monkeys demonstrated the first evidence of neurons with the property of firing both during observation and during execution of an action (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). These were dubbed "mirror neurons" and provided the first direct evidence of perception–action coupling at the level of a single neuron. This extremely important discovery stimulated a burst of brain-based studies involving imitation in humans. While these neurons were first identified in animals without the capacity for intentional imitation, the locus of these neurons in the monkey homolog to Broca's area allowed hypotheses for the role of mirror neurons in language, imitation, and other crucial human social–communicative abilities, suggested earlier by Donald, to develop rapidly (Rizzolatti & Arbib, 1998). Mirror neurons were quickly seen as a potential mechanism that might explain both imitative deficits and the greater social–communicative deficits associated with autism, particularly as a starting-state mechanism responsible for a slowly accruing series of social deficits (Williams et al., 2001). This discovery stimulated a wave of theorizing about potential roles of mirror neurons in human development and evolution (Rizzolatti & Arbib, 1998; Williams et al., 2001) and neuroimaging studies of brain responses to imitative tasks, both in typical development and in autism.

Neuroimaging Studies of Imitation in Typically Developing Subjects

Increasingly, perspectives on brain functioning do not see action and perception as served by separate apparatus. Rather, cortical functioning tends to be dependent on connectivity between brain areas. The "mirror neuron" discovery is important as one of the earlier experimental findings in accord with this perspective. Areas of visual cortex have now been identified that are sensitive to motor input (Astafiev, Stanley, Shulman, & Corbetta, 2004), and spatial attention appears to be dependent upon parietofrontal connectivity (Gaffan, 2005; Thiebaut de Schotten et al., 2005).

The neural substrate of imitation is reviewed by Decety (Chapter 11, this volume). To summarize very briefly, inferior and superior aspects of parietal lobe are likely to be important in relating visual aspects of movement to codings of actions derived from proprioceptive input. In addition, frontal brain areas are likely to be important in inhibiting and commissioning imitation. The STS is likely to be important in assigning intention to observed action (Jellema et al., 2000). Keysers and Perrett (2004) suggest that in form-

ing connectivity with inferior frontal lobe through the observation of self-generated action, the STS plays a vital role in developing the mapping of visual to self-codings that are required for the mirror neuron network and imitation. The insula is also a potentially important area that may be involved in more affectively laden imitation (Gallese, Keysers, & Rizzolatti, 2004).

The distinction between ventral and dorsal stream processing (Milner & Goodale, 1995) has also attracted interest recently, as researchers study basic visual processing in autism. In essence, the ventral stream has high sensitivity to spatial variance but a lower sensitivity to changes in temporal dynamics. It is thought to be associated with processing static visual content and processes information along ventral temporal cortex. The dorsal stream processes visual information that changes rapidly with time, along the dorsal stream involving mirror neurons in parietal lobe. Some authors argue that autism could be a dorsal stream problem (Milne et al., 2002; Spencer et al. 2000). The dichotomy is of interest to imitation research in understanding which aspects utilize these different streams and how they might relate to one another.

Neuroimaging Studies of Imitation in Autism

As reviewed in Williams and Waiter (Chapter 15, this volume), studies are beginning to explore relationships between imitative abilities and neural systems in autism. Nishitani, Avikainen, and Hari (2004) reported a magnetoencephalography (MEG) study of subjects with Asperger syndrome (AS) observing and imitating oral–facial movements from still photos (see Williams & Waiter, Chapter 15, for a more detailed description of this study). Compared to typical adult control subjects, those with AS demonstrated decreased activation of Broca's area during imitation, but no differences in STS activation implying that the AS difference in brain response was specific to the imitation condition and was not seen during observation.

Williams and colleagues (in press) utilized a functional magnetic resonance imaging (fMRI)–imitation protocol involving the Iacaboni and colleagues (1999) isolated finger movement task in a study of ASD and typical controls. They demonstrated several areas of autism-specific differences. One involved decreased activation of the mirror neuron (MN) regions of the right inferior parietal lobe in ASD during both the observation and imitation phases of the experiment. The authors suggest that this may represent problems with the parietal mirror neuron system involving a generalized poor proprioceptive input to self-generated movements, regardless of the stimulus, in ASD. A second very important finding involved decreased left amygdala activation during the imitation condition in ASD. Amygdala activation has not before been identified in fMRI–imitation studies. Williams and colleagues interpret this finding as possibly reflecting a different, and lessened, emotional experience associated with imitation in ASD. This hypothesis has direct connections to Dawson and colleagues' (2004) recent suggestion that deficient social behavior in early autism is due to lack of typical positive affective feedback—

the intrinsic reinforcement system, as well as to various studies reporting amygdala differences in autism (Abell et al., 1999; Bauman & Kemper, 1998). Finally, the lack of activation in the posterior aspect of STS in the ASD group during imitation but not observation (in the face of the opposite occurring in controls) was particularly interesting given that subjects with autism also show decreased responses is this same area of STS during mental state tasks (Castelli, Frith, Happe, & Frith, 2002).

Oberman and colleagues (2005) reported a pilot EEG study of mu wave suppression, which has been demonstrated to be correlated with MN activity. A group of high-functioning persons with autism observed videos of several stimuli, including a hand movement, and were asked to imitate the movement. The subjects demonstrated lack of mu wave suppression during imitation but not during observation, suggesting lack of activation of the MN system during imitation (which they performed adequately).

The final neuroimaging study of autism and imitation to be discussed here was recently published by Dapretto and colleagues (2006). This group reported a fMRI study of 10 high-functioning children with ASD and 10 typically developing comparison children, matched by age and IQ. The stimuli involved pictures of five different emotional expressions, which the participants either imitated or passively viewed in the scanner. Half of the group subsequently repeated the experiment outside the scanner on an eye tracker, with no differences in fixation times to faces. Precision of imitation was not measured, although judges did not rate the emotional expressions of the two groups differently. The group with ASD demonstrated many activations similar to controls, but did not demonstrate activity in the mirror neuron area of pars opercularis during both imitation and observation. During imitation, the typically developing group showed significantly greater activation in insula and amygdaloid areas than the ASD group, who showed greater parietal and visual association activation. Furthermore, Dapretto and colleagues found significant negative correlations between the intensity of social symptoms specific to autism and activity in pars opercularis, insula, and limbic structures, insula and social symptoms specific to autism, even with IQ controlled. Furthermore, there was adequate monitoring of visual fixations to ensure that this difference was not due to looking patterns. These findings suggested that children with autism use some different neural strategies during both imitation and observation of emotion faces, and this probably results in less felt emotion for them. This is the most convincing study yet that ties autism to MN system differences.

Although it is seductive to attribute many aspects of the autism profile to MN deficiency (Theoret et al., 2005; Williams et al., 2001), and although the evidence currently supports the hypothesis that the MN network functions abnormally in autism, experimental tests of this hypothesis need to consider some important issues. The most important of these is that MN function has been demonstrated at a single cell level in monkeys and not humans. Yet, the functions that are being ascribed to MNs such as theory of mind, language,

and imitation are only found in humans. MN circuitry in monkeys appears to assist with understanding others' actions, and perhaps with intention reading (Fogassi et al., 2005; Perrett et al., 1989). Two different research groups have used simple intention reading tasks with children with autism, and neither has reported a deficit in their response (Aldridge et al., 2000; Carpenter et al., 2001). These tasks, which should elicit MN activation, have not shown any autism-specific impairment.

A second issue for an MN hypothesis for autism has to do with a biological model for a MN deficit. Imagining some type of specific impairment of the mirror neurons at the core of autism is difficult given that the mirror neurons are unique only in their functions and not in their structure, location, development, and migration (V. Gallese, personal communication, April 2005; Petrides, Cadoret, & Mackey, 2005). Williams and colleagues (in press) suggested an alternative mechanism. The left anterior frontal lobe (serving what has been classified here as an MN function) is normally characterized by an unusually high level of pruning appropriate to its serving an integrative function. One biological function that could theoretically result in an impaired MN function would be an impaired neuronal pruning mechanism. An alternative suggestion is a general problem with connectivity, and general connectivity problems have been suggested in autism (Just, Cherkassky, Keller, & Minshew, 2004). However, a general connectivity theory better addresses problems that require higher-order skills than early skills like simple gestural imitation. In addition, and as discussed by Williams and Waiter (Chapter 15, this volume) there is poor evidence for a general connectivity impairment in ASD.

Therefore, although we have some tentative evidence in support of the MN hypothesis of autism offered by Williams and colleagues (2001), the function and neural substrate of the MN system is now being recognized as much more sophisticated than was previously imagined. As the fMRI studies have demonstrated, there are many brain responses to imitation tasks, and many ways in which autism-related imitation differences might be reflected in brain responses. Given current models of brain development and the crucial role of experience in developing expertise, we assume that neural responses to imitative tasks reflect learning and practice histories, as well as possible starting-state differences. The early mirroring seen in young infants suggests that there is a starting-state mechanism present and active at the time of birth. However, given the interactive differences that children with autism experience, we should be cautious about assuming a starting-state difference in infants with autism as the explanation for either the behavioral or the brain activation differences related to imitation studies. Prospective studies of infants who go on to develop autism will help us understand the interactions among starting-state capacities and developmental histories. Such studies are currently ongoing in several different countries.

To conclude, the aforementioned studies demonstrate our growing knowledge of the neuroscience of imitation. As expected, given the behavioral

evidence of imitation problems in autism, the initial neuroimaging studies of autism report differences in neural activity during imitation tasks in subjects with ASDs, and the Williams and colleagues (2006) study suggests that the differences do not involve one particular structure but, rather, appear widely distributed. Imitation involves the coordination of multisensory information with the execution of motor acts, automaticity of responses, the interpretation of self and others' behavior, and affective responses that may well influence frequency of response through differences in intrinsic emotional reward systems. What is emerging is a complex picture of neurology of imitation. Although there is tremendous emphasis at the moment on mirror neurons in Broca's area as the "site" of imitation and other kinds of self–other relations, their role in human ontogeny is still completely unknown. If, as Donald (1991) hypothesized, the whole cortical expansion was driven by the selective advantages conferred by being able to copy, communicate, and read actions, one would expect that imitation would be a property more of the whole cortex than of an individual area.

INTEGRATING CURRENT RESEARCH ON DEVELOPMENT OF IMITATION AND AUTISM

A long time ago, we suggested that the core social difficulty in autism involved the coordination of self–other schemas, at a bodily sense as well as a psychological sense. We suggested that impaired imitation early in autism might be the initial reflection of this difficulty and might also contribute to difficulties coordinating other kinds of self–other schemas (Rogers & Pennington, 1991). Having reviewed all the published studies to date, there is very strong evidence of autism-specific impairments in imitation of model's gestures, oral–facial movements, and actions on objects in participants ranging in age from 2 to adulthood and across the intellectual range of autism, as well as the severity range of autism and its milder variants. Imitation is not absent in autism but, rather, less frequent and less precise than in other groups.

Are imitative deficits related to other core features of autism? Several papers have examined concurrent relations. Imitative ability correlated strongly with the presence and severity of autism symptoms in very young children with autism (Rogers et al., 2003). Early imitation has demonstrated relations with language development (for which imitation is a strong predictor, in both typically developing children and those with autism; Charman & Baron-Cohen, 2003; Rogers et al., 2003; Stone & Yoder, 2001 and reviewed by Charman, Chapter 5, this volume). Only one study has examined relations between imitation, dyadic responsivity, and joint attention behavior in autism, reporting significant and moderately strong relations for children with autism (Rogers et al., 2003). In the only study to examine it, intentional imitation was not correlated with unintentional, or automatic, imitation (mimicry) of emotional expressions (Scambler et al., in press). Treatment studies have

begun to demonstrate collateral effects of imitation abilities on children with autism: increased social engagement with others, language skills (Wert & Neisworth, 2003), social initiations (Nikopoulos & Keenan, 2003), social initiations (Nikopoulos & Kennan, 2003), and generalized effects of imitation training (Garfinkle & Schwartz, 2002). Thus, while studies of concurrent relations support the hypothesis that imitation has links to other core features of autism, the prospective longitudinal studies of typical and atypical development from early infancy through the preschool years needed to test this model have not yet been published.

Do imitative deficits in autism precede development of other autism symptoms? Zwaigenbaum and colleagues (2005) have provided the first lab-based evidence of imitation problems in infants prior to diagnosis of autism. In a large study of the early development of infant siblings of children with autism, he and his colleagues followed 65 infant siblings from 6 to 24 months. Nineteen children presented with symptoms corresponding to an ASD at 24 months. Imitation items (along with several other indicators of social impairment) administered at 12 months predicted these 19 children at a p value of .003. This is the first direct lab evidence that imitation impairments exist in infants prior to the time the full syndrome emerges.

The imitation deficit associated with autism does not appear to be due primarily to motor dexterity though motor maturity and coordination contribute to poor-quality performances. Neither have imitation problems in autism been linked yet primarily to poor motivation to perform, though this has not been well studied and it is an extremely important variable to manage well in task design and procedures. The focus of attention during imitation tasks has been examined by a few: No abnormalities have yet been identified.

The types of imitative difficulties found, particularly involving self–other perspectives, body part orientations, and role reversals, provide support for autism-specific difficulties in forming and coordinating, or mapping representations of self and other—as originally proposed by Rogers and Pennington (1991) and later expanded by Williams and colleagues (2001). Self–other mapping in imitation tasks relies on connectivity across the entire brain, from visual to motor cortex and from right to left hemisphere. Therefore, to the extent to which autism involves white matter connectivity, skills that require widespread connectivity, like self–other mapping, are likely to be strongly affected. Several studies have described brain processes involved in imitative tasks, and the few comparative studies of autism document autism-specific differences in brain activation patterns during imitative tasks. White matter deficits of the type documented in autism by Piven, Bailey, Ranson, and Arndt (1997) and replicated by Hardan, Minshew, and Keshavan (2000) and Waiter and colleagues (2004) should have greatest impact on behavioral functions that depend on cortical integration between the most spatially disparate structures, like imitation. More than any other developmental or neuropsychological impairment in autism, imitation appears to meet the primary deficit crite-

ria of specificity, universality, precedence, and persistence (Pennington & Ozonoff, 1991).

In typical development, reciprocal mirroring between parent and infant appear to form an important part of the repertoire of reciprocal social interactions. These appear pleasurable to both partners, and we assume that repeated experiences of imitating and being imitated strengthen the infant's neural connections involved in self–other mappings through repetition and positive affect and lead to increased frequency across early childhood (the Hebbian model of MN learning described by Keysers & Perrett, 2004). Both automatic and intentional imitative processes appear in the first year of life, and the relations between them are unknown. Several starting-state conditions are at play: a capacity to produce some early motor imitations (which demonstrates starting-state neural coordination of perception–action circuitry), partners who imitate the infant and provide models for imitation, infant discrimination of reciprocal imitations, and positive affect experienced during reciprocal imitations.

Impairment in any one or more of these conditions may interfere with the development of coordinated and reciprocal motoric and affective interaction patterns, resulting in infrequent social imitations, lack of enjoyment in imitative exchanges, lack of expectation for reciprocity, and thus lack of practice and lack of developing automaticity. Similarities among infants who will develop autism, infants with blindness, infants with extreme deprivation, and infants of depressed mothers, similarities involving affective neutrality and decreases in contingent responsiveness, may mark the early difficulties in this general domain, even though the derailing variable differs for each group. Given the pleasure children with autism show during episodes of being imitated, and the apparent reinforcement value therein, being imitated provides positive experiences for them (Dawson & Galpert, 1990; Escalona et al., 2002; Harris, Handleman, & Fong, 1987). Lack of reinforcement does not appear to be a viable hypothesis concerning imitative deficits in autism.

For infants who will develop autism, developing cognitive and motor abilities support successful interactions with objects. Their interest and knowledge about objects develop, and they learn means–end relations and observe and comprehend others' intentions on objects, which we assume involves MN activation. Their ability to understand others' acts may well support the development of intentional imitation of interesting actions on objects. However, the neurobiology of autism impedes the development of imitation as a source of social communication and affects automatic mimicry processes as well. The final picture in autism would be of a capacity for intentional imitation of object skills, though lacking in precision (due to lack of practice, motor difficulties, and/or other causes), combined with a large deficit in automatic mimicry of other's facial, vocal, postural, gestural, and other expressive behaviors in appropriate social interactions. Altered neural responses to imitative stimuli may reflect lack of practice and lack of expertise, primary neural differences, and/or recruitment to other functional circuits. However, the evidence sug-

gests that in autism, the capacity for more typical imitative responses is present, as seen in imitations of acts on objects, echolalia, occasional echopraxia, video copying, and the capacity of some young children with autism to acquire much more typical imitative skills through treatment.

We continue to suggest that the "cause" of the imitation problem in autism lies in abnormal functioning of neural processes that entrain us to each other and result in coordination of bodily movements and actions, and later, entrainment of affective and social cognitive processes. This failure of entrainment, neurally and behaviorally, is core to the symptoms of autism. This could result from a deficient capacity to automatically map others' actions onto the self and produce imitative responses. Evidence seems more supportive of impairments in these areas than in detecting mirroring responses of the other. Longitudinal studies of imitative development from birth through the second year, in both typical and atypical development, are desperately needed. A great deal of developmental theorizing about the role of imitation in development, including our own, has been built on very little longitudinal data. We need to follow Heimann and Ullstadius's (1999) lead and put early imitation theories to the test.

ADDITIONAL TOPICS FOR RESEARCH AND RESOLUTION

Delay versus Deficit

Throughout this chapter we have addressed questions in need of further research. There is one other issue that needs to be resolved. Much discussion has hinged on the distinction between a *delay* in imitative development and a *deficit* in imitative ability in autism. Several studies have demonstrated specific imitative impairments in high-functioning adults (Avikainen et al., 2003; Rogers et al., 1996). Identification of unusual patterns of imitative performance in autism in relation to delayed groups also supports a deviance model, as seen problems with oral–motor impairments in Rogers and colleagues (2003), and with self-orientated movements in Smith and Bryson (1998), Avikainen and colleagues (2003), and Meyer and Hobson (2004), among others. However, the issue may well involve the semantics of the term *delay*. Although an immature performance in children may be considered a delay, adult differences, even when they reflect immaturities, are typically not referred to as delays. At some point, delay becomes deviance. Examination of the developmental sequences and trajectories of intentional and automatic imitation in early autism would help distinguish between delayed and deviant patterns of development.

IQ and Imitation

IQ is often significantly correlated with imitative ability, and imitation skills have been found to predict to later IQ in the very few longitudinal studies that

have been reported, as reviewed by Hepburn and Stone (Chapter 13, this volume). However, interpretation of this relationship is not straightforward. Given that an IQ score reflects a person's past learning rate, imitation may be an important determinant of IQ performance. If imitation ability is the powerful early learning tool that theorists suggest, then lack of imitative experiences would result in a diminished repertoire of skills and abilities and reflected in lower IQ scores. The nature of the relation between imitation and IQ score is not yet known, and experimenters should be cautious about statistically controlling for IQ in analyses, because they may be controlling for the very variable that they wish to study.

More Precise Conceptualization and Coding of Imitative Tasks

The range of tasks being used in autism imitation studies raises many questions. The comparative psychologists have given clinical researchers a taxonomy for tightening definitions and choice of tasks, and using such distinctions will help communication across studies. In terms of tasks, some of the imitative tasks used in the scanner (simple finger raising) are so simple that they barely seem to tap imitative phenomena. Is such a simple task really parallel to imitating a sequence of meaningless actions, or to automatic responses to natural emotional expressions of others? The "messiness" of the actions used in autism imitation studies makes it difficult to extract core difficulties, particularly given evidence that brain responses vary specifically and differentially to stimuli involving different body parts. Finally, most imitative tasks have multiple degrees of freedom involved, including body position, limb position, and movement dynamics, among others. Detailed coding systems that examine errors provide much more information about the nature of the performance than pass–fail systems.

Response to Being Imitated

We need greater understanding of people with autism's awareness of being imitated, which has begun to be examined by Nadel and her colleagues, Nielsen (Nielsen, Suddendorf, & Dissanayake, Chapter 7, this volume) and Decety (Chapter 11, this volume) but few others. Escalona and colleagues (2002) have developed further Dawson's early finding that children with autism responded differentially to being imitated (Dawson & Galpert, 1990; Harris et al., 1987), responding with attention to contingency but with approach and touch after being imitated. Sensitivity to being imitated brings to mind initial hypotheses concerning contingency raised early by Dawson and Lewy (1989) and more recently by Gergely and Watson (1999). Response to being imitated suggests awareness of self and other in important ways. It would be interesting to examine response to imitation in light of the self–other orientation problems highlighted by Meyer and Hobson (2004). To what extent children with autism demonstrate a typical or atypical response to others' imitations of

them is unknown, and this represents a very fruitful area for further investigation.

Echolalia and Echopraxia in Autism

Finally, we have very little information about the phenomena of echolalia and echopraxia, whether in autism or in other neurological disorders, and their relation to other types of imitative behavior. Decety (Chapter 11, this volume) has provided an intriguing theory concerning both phenomena, and we need both behavioral and imaging studies to help us understand the nature of these responses compared to other types of intentional and automatic imitative responses.

Treatment of Imitation Difficulties

One aspect of the general research agenda for autism focuses on developing more effective treatments for autism, and as such, imitation provides us with a potentially important tool. Intervention studies have demonstrated that imitation skills are quite responsive to contingencies and teaching (as are other early deficits in autism, including language, joint attention, and play skills, to name a few). If the developmental theories of imitation in autism continue to be supported by increasing evidence, then early development of imitative capacity in very young children with autism may have a marked effect on outcomes (see Sallows & Graupner, 2005, for positive evidence). The extent to which interventions that develop improved intentional imitation also result in more normalized automatic imitation is completely unexplored but of potentially great importance.

We have raised a number of areas in which further research is clearly needed. The importance of imitation problems in the ontogeny of the syndrome is unknown, and the question awaits research on starting-state imitation, examination of imitative responses during the period of symptom onset, in both regressed and early onset cases, and the role of various types of imitation in other aspects of both typical and atypical social development. However, 15 years of imitation research in autism have provided strong support for the centrality and pervasiveness of the difficulties that people with autism have in coordinating self with other at the bodily level as well as the psychological level.

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