A theoretical approach to the deficits in infantile autism

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Abstract
Deficits specific to the syndrome of infantile autism appear in imitation, emotion sharing, theory of mind, pragmatics of communication, and symbolic play. Current competing theories of Hobson and of Baron-Cohen, Frith, and associates account for some, but not all, of these specific deficits. The present article suggests that early social capacities involving imitation, emotion sharing, and theory of mind are primarily and specifically deficient in autism. Further, these three capacities involve forming and coordinating social representations of self and other at increasingly complex levels via representational processes that extract patterns of similarity between self and other. Stern's theory of interpersonal development is offered as a continuous model for understanding the development and deficits of the autistic child and as a means for integrating competing theories about the primary deficits in autism. Finally, the article suggests a neuropsychological model of interpersonal coordination involving prefrontal cortex and executive function capacities that is consistent with the social deficits observed in autism.

Despite the myriad abnormal characteristics of autistic persons described in the scientific literature of the past 40 years, there are only a few characteristics specific to infantile autism, appearing whenever autistic persons are compared with persons matched on IQ and chronological age (CA) (Baron-Cohen, 1988; Prior, 1979; Sigman, 1989). Although various domains of human ability—cognition, language, perception—have been considered as the main area of deficit in the past, many current theorists have converged on the idea that the disruption of early developing social processes is basic to the disorder (Baron-Cohen, 1988; Dawson & Ley, 1989a, 1989b; Fein, Pennington, Markowitz, Braverman, & Waterhouse, 1986; Hobson, 1989, 1990; Mundy & Sigman, 1989a, 1989b).

Sigman (1989) recently demonstrated the theoretical importance and pragmatic utility of examining the deficits in autism within the framework of normal development, an approach that is essential to the interdisciplinary of developmental psychopathology (Cicchetti, 1990). The present article, likewise based in a model of normal development, will suggest the potential power of an early deficit in imitation to disrupt other early developing interpersonal processes. The suggested model will tie together this imitation deficit with two of the currently more promising theories of the primary deficit in autism: the affective deficit hypothesis proposed by Hobson (1989, 1990) and the metarepresentational
deficit hypothesis proposed by Baron-Cohen, Leslie, and Frith (1985, 1986). This integration will be accomplished via a continuous developmental model of autism based on Stern’s (1985) theory of interpersonal development. In addition, the model will suggest a way of viewing current neuropsychological findings concerning executive function deficits in autism that is consistent with the social deficits observed in autism.

In what follows, we will provide a review of the social processes that appear to be specifically impaired in autism and those that do not. We will then propose a theoretical model influenced by Stern that integrates the social deficits from a developmental framework. Next we will briefly review the current neuropsychological findings regarding autism in light of the described social deficits. We will end by making clear the unique and testable hypotheses of our model, describing the empirical studies needed to test these hypotheses, and indicating issues that are not addressed by our model.

**Deficits in Autism**

Autism, first described by Kanner in 1943, is a severe disability involving abnormalities in social relationships, language, behavior, and cognition. There is considerable agreement that basic social processes (either primarily or secondarily) are specifically deficient in autism (Baron-Cohen, 1988; Fein et al., 1986; Hobson, 1989; Mundy & Sigman, 1989a, 1989b); however, it is clear that not all social behavior and social drive are absent in autism. A model of autism must account not only for the social processes that are affected, but also for those that are not specifically affected by the disorder. We will first review autistic subjects' performances in basic social processes that are similar to comparison subjects matched on IQ and CA. (Note: throughout this article, studies referred to will always involve comparison subjects who are both age and IQ matched unless otherwise specified. Thus, autistic subjects with retardation will be compared with retarded subjects; autistic subjects without retardation will be compared with nonretarded subjects.) Then we will consider areas in which autistic subjects demonstrate specific deficits when compared with comparison subjects.

**Less Impaired Social Behaviors**

**Attachment behaviors.** Attachment has long been considered a specific area of deficiency in autism. However, recent studies by three independent groups have found that behaviors indicative of attachment security in autistic children are similar to those of mentally retarded comparison groups. Sigman and Mundy (1989) reported the results of three studies of discrete attachment behaviors in 40 young autistic children matched on mental age (MA) with mentally retarded subjects and normally developing subjects. Although all three groups demonstrated preferential behavior toward the mother, distress at separation, and positive reactions to reunion with the mother, both clinical groups were less involved with their caretakers and showed less distress on separation from their mothers than the normally developing group. However, no differences in reunion behavior between the autistic and the retarded groups were found. Similar results were found by Shapiro, Sherman, Calamari, and Koch (1987), who compared the attachment behaviors of unmatched groups of autistic, mentally retarded, developmentally language-disordered, and pervasively developmentally disordered (PDD) young children. Using an A-B-C classification system, they reported that 50% of the autistic and PDD groups were classified as secure, a higher percentage than in either of the other two clinical groups. Furthermore, security was unrelated to developmental quotients or severity of autistic symptoms. Finally, Rogers, Ozonoff, and Maslin-Cole (1991) reported no differences in overall security ratings on a continuous rating scale between a group of 17 young autistic and PDD children and a CA and MA matched
group of developmentally disabled children with other psychiatric disorders. They found security to be unrelated to severity of autism, but positively related to MA in the autistic/PDD subjects only.

These studies do not support the conclusions that attachment processes are "normal" in autism. Rather, they demonstrate that, in the Strange Situation paradigm, autistic children are not significantly different from nonautistic, developmentally disabled children in terms of discrete behaviors considered to reflect attachment security. None of these studies examined the broader notion of the autistic child's "internal working model" of the attachment figure, although Rogers et al. (1991) suggested how it might differ in its construction and final form from that of other children's. Attachment behaviors are quite old in the evolutionary sense, characteristic of all mammals and mediated by stimuli that do not necessarily require "intersubjectivity" in Stern's sense (cf. Hofer, 1987). So it should not be surprising that some aspects of this evolutionary old behavior are present in autistic children, even though other social behaviors (which may be evolutionarily newer) are impaired.

Self- and other-recognition. Earlier psychoanalytic theories suggested that a deficient sense of self and self/other differentiation were fundamental to autism (Goldfarb, 1965; Mahler, 1968; Ornitz & Ritvo, 1968), a view that Hobson (1990) recently developed further. There are many different aspects of one's representation of the self (Neisser, 1988), few of which have been studied in autism. One aspect of the self that has been studied in autism involves the physical self, or what Neisser would call the "ecological self." Studies of self-recognition in autism have used the standard mirror self-recognition task in which normal infants exhibit the development of self-recognition by 18–20 months of age (Bertenthal & Fischer, 1978). Using this task, several studies of autistic subjects reported appropriate behavior for MA (Dawson & McKissick, 1984; Ferrari, & Matthews, 1983; Spiker & Ricks, 1984). Hence, these studies suggest that autism does not preclude the development of a physical or ecological self.

Other-recognition appears relatively early in normal development, with infants demonstrating visual discrimination of mother from others by 4 months (Lamb & Campos, 1982). Empirical studies of other-recognition, using pictures of faces with autistic subjects, reported mixed findings. Studies that matched control subjects on verbal abilities reported no differences between autistic and control subjects (Braverman, Fein, Lucci, & Waterhouse, 1989; Langdell, 1978; Ozonoff, Pennington, & Rogers, 1990; Smalley & Asarnow, 1990). Some studies that matched subjects on nonverbal MA (which maximizes the chances of finding significant differences) report autistic deficits in face matching (Tantum, Monaghan, Nicholson, & Stirling, 1989), and others found no group differences (Volkmar, Sparrow, Rende, & Cohen, 1989), although there are some indications that autistic subjects use different strategies for processing facial information (Langdell, 1978; Tantum et al., 1989).

Thus, across the literature on perceptual recognition of self and other (at an "ecological self" level), autistic subjects demonstrated performances comparable to comparison subjects. Success on such perceptual tasks does not imply that autistic children are "normal" in terms of all aspects of self and other representation. However, the early theorists suggested that the autistic child does not develop an adequately differentiated sense of even a bodily self and bodily other, and this global view is not supported by recent research. Instead, it appears that the very basic social ability of perceptual recognition and differentiation of self and other is not specifically deficient in autistic subjects. Clinical examples of autistic people using other people to assist them in sensorimotor goals also demonstrate this knowledge of a differentiation between a physical self and a physical other, as well as some knowledge of different physical capacities among people.
Differential social responsiveness. A number of observational studies of autistic subjects found differential responsiveness to certain social cues. Both Sussman and Sklar (1969) and Freitag (1970) found autistic children to be differentially responsive to positive versus negative interactions. Moreover, autistic children have not been found to be less compliant or more negative than comparison subjects in their interactive style (Clark & Rutter, 1981; Volkmar, Hoden, & Cohen, 1985). Finally, social responsiveness in autistic samples has been found to vary in a normal way with both familiar and unfamiliar adults (McHale, Simeonsson, Marcus, & Olley, 1980) in terms of responsivity of their social partner (Clark & Rutter, 1981; Lord, 1984; Tonick, 1981). Two studies of young autistic children and their mothers in free play from Marian Sigman’s laboratory reported no differences between autistic subjects and mentally retarded controls (Sigman & Mundy, 1989; Sigman, Mundy, Sherman, & Ungerer, 1986) in frequency of social behaviors involving looking, vocalizing, and proximity-seeking directed to their caretakers during play, although the autistic subjects demonstrated specific deficits in joint-attention behaviors and speech.

In summary, a number of basic social processes do not appear to be specifically deficient in autism, including some behaviors indicative of attachment security, the perceptual processes of self-recognition and other-recognition, and differential social responsiveness. In contrast to the popular stereotype of the autistic child, the autistic subjects in these studies were not pervasively aloof, avoidant, or unattached, although there is certainly a wide range of social engagement/isolation in autistic children (Sigman, 1989). If all early social processes are not specifically impaired in autism, theorists are forced to develop a differentiated account of the kind and nature of social deficits in autism. Interestingly, none of these relatively intact social processes necessarily require imitation, emotion sharing, or awareness of another person’s mental state (which we will discuss next); three skills that Stern indicates are essential for normal interpersonal development to occur. The distinction between social processes that require one or more of these skills and those that do not is crucial for our theoretical model of autism.

Specifically impaired social processes

Imitation. Although imitation was historically seen as a skill that developed in the latter part of first year of life (McCabe & Uzgiris, 1983; Piaget, 1962), in the past 10 years a number of studies of neonatal imitation have been published (Meltzoff, 1988; Meltzoff & Moore, 1977) that have resulted in increasing acceptance of the idea that very young infants are capable of “real” imitation (Mandler, 1988) based on representational capacities. Thus, imitation of some body movements appears to be present essentially from birth. We will argue that imitation, even in neonates, can be thought of as a social skill.

Autistic children’s difficulty in imitating another person’s actions was first described by DeMyer and colleagues (DeMyer, Alpern, Barton, DeMyer, Churchill, Hingtgen, Bryson, Pontius, & Kimberlin, 1972), who compared 12 autistic (and schizophrenic) subjects with 12 mentally retarded controls. The autistic subjects were significantly poorer than controls on imitation of body movements, less deficient but still significantly poorer on imitations of actions on objects, and equivalent on nonimitated purposeful object acts; the last finding makes the alternative explanation of motor deficits unlikely.

Since then, there have been six replications and one nonreplication, which was flawed by ceiling effects. Four of the six replications used nonsymbolic, sensorimotor imitation tasks, and all found autism-specific deficits on these tasks; so it is unlikely that the imitation deficits in autism are simply secondary to symbolic deficits. We will now review these seven studies.

Jones and Prior (1985) used body imitation tasks that involved no objects and no
symbolic acts to compare 10 autistic subjects with 10 CA matched and 10 language matched normal children. They found specific deficits in autistic subjects on all tests of body imitation and suggested a primary imitation deficit that they felt reflected the presence of motor dyspraxia in autism. Likewise, Ohta (1987) studied simple body imitations unconfounded by objects or symbolic content in 16 nonretarded autistic subjects (mean age, 10 years) compared with two control groups, one consisting of 16 subjects matched on CA and Performance IQ and the other a group of 189 normal preschoolers. Ohta reported deficits in the autistic subjects in simple gestural imitations compared to both control groups. Further, the imitation deficit was not related to IQ or MA, and the autistic subjects performed like the youngest preschoolers (mean age, 36 months) in the study. Ohta suggested that the basis for the deficit was a deficient representational capacity as it applies to body image. Sigman and Ungerer (1984) demonstrated autism-specific imitation deficits on both motor and vocal imitation tasks in a group of 16 young autistic children compared with 16 mentally retarded and 16 normal MA matched controls. Finally, a recent study by Hertzig, Snow, and Sherman (1989), which compared 18 lower functioning, verbal autistic subjects with both 14 IQ matched retarded controls and 19 normal language matched preschoolers, demonstrated that the autistic subjects were poorer than both control groups at imitating both sensorimotor and symbolic actions of a model. Furthermore, they had even greater difficulty imitating when the content of the action was primarily affective. These tasks included imitation of a pictured model, pantomime of an emotion on command, and imitation of emotion in a live model. Contrary to these four studies, Morgan, Cutler, Coplin, and Rodriguez (1989) reported no evidence of a motor imitation deficit in 10 verbal autistic children compared with 10 language matched mentally retarded and 10 normal controls. However, all three groups in their study were at ceiling levels on all the measures given to them, so that there was a lack of discriminative power in the measures.

In addition to these studies of sensorimotor imitation, two studies of symbolic imitation, or pantomime, tasks involving IQ matched clinical comparison groups have been published (Bartak, Rutter, & Cox, 1975; Hammes & Langdell, 1981). Both studies found autistic subjects deficient on classic apraxia tasks involving symbolic pantomime when compared with controls.

Thus, although Baron-Cohen (1988) summarized the imitation literature as indicating specific impairments in higher order (symbolic) imitation, as opposed to nonautism-specific impairments in lower order (simple body movement) imitation, a wider sampling of the imitation literature suggests specific deficits (i.e., worse than mentally retarded MA and CA matched comparison groups) in imitation of simple motor movements and affect expressions, as well as in higher level symbolic imitations in autistic persons.

Given the importance that has been attributed to infant–mother imitations in terms of early social reciprocity and intersubjectivity (Stern, 1985), affective sharing (Malatesta & Izard, 1984), and social learning (Bruner, 1975; Baldwin, 1906), early deficits in imitation might have significant effects on social and affective development. Furthermore, imitation is a relatively new behavior evolutionarily, and, unlike other social behaviors such as attachment, there is still considerable debate about the presence of true imitation in nonhuman species (Galef, 1988). Thus, although the imitation deficits in autism have been explained in terms of motor dyspraxia and deficient body image, we stress the importance of viewing imitation as an early social skill. One can have an intact physical or ecological self and, yet, still not imitate.

Affective processes. There is fairly wide agreement that some capacity to discriminate emotions is present by 2–4 months of age (Field, Woodson, Greenberg, & Cohen,
Differential emotional responses to angry versus happy faces (Klinnert et al., 1983) as well as reliable cross-modal matching of emotional sounds with emotional pictures (Walker, 1982) are observable by at least 7 months of age.

Furthermore, important theoretical linkages between early imitation and affective development have been suggested. Stern suggested a prominent role of early imitation in the development of affect sharing, affective communication, and affect attunement. Other major theorists (Malatesta & Izard, 1984; Trevarthen, 1979; Uzgiris, 1981) also suggested that early mother-infant imitations form the basis for development of affective mutuality, reciprocity, and communication in the first year of life. In addition, Malatesta and Izard (1984) suggested that maternal imitations have an important role in patterning the infant's facial expressions of emotion. Finally, they theorized that the infant's experience of viewing the mother's imitation of the baby's facial expression allows the infant to integrate visual perception/recognition of facial affect patterns with subjective feelings, a foundation for later development of intersubjective awareness of others' internal states. This description is similar to Stern's description of early capacities for amodal or cross-modal representations: he vividly described maternal cross-modal matches to infant affective expressions—affect attunements.

Hobson (1989, 1990) suggested that the autistic child's deficits in cognition, language, and symbolic play stem from a primary affective disorder involving the child's constitutional emotional reactivity, including deficits in emotional expression and perception, on the one hand, and the child's inability to develop reciprocal, affectively based relationships with others, on the other. These deficits prevent the autistic child from developing intersubjective awareness of self or others, resulting in the autistic child's "failure to recognize other people as people with their own feelings, thoughts, wishes, intentions" (1989, p. 23), as well as in a severe impairment in the capacity to think abstractly or symbolically.

A number of studies have provided evidence confirming that there are specific deficits in autistic subjects' perception and understanding of affect (Hertzig et al., 1989; Hobson, 1986a, 1986b; MacDonald, Rutter, Howlin, Rios, Le Conte, Evered, & Folstein, 1989; Smallly & Asarnow, 1990). Other laboratories have reported evidence of specific deficits in affective expression and affect sharing in autistic children when compared with control groups. These include less positive affect, more negative affect, and more unreadable or mixed affective expressions, less direction of affect displays to a social partner, less use of gaze to communicate affect, and less mirroring of partner's social smiles (Dawson, Hill, Spencer, Galpert, & Watson, 1990; Hertzig et al., 1989; Kasari, Sigman, Mundy, & Yirmiya, 1990; MacDonald et al., 1989; Mundy, Sigman, Ungerer, & Sherman, 1986; Snow, Hertzig, & Shapiro, 1987; Tantam et al., 1989; Yirmiya, Kasari, Sigman, & Mundy, 1990).

However, other studies have found no differences between autistic and control subjects on static emotion perception tasks when the most conservative, verbally matched control groups have been used (Braverman et al., 1989; Hobson, Ousen, & Lee, 1988; Ozonoff et al., 1990). Moreover, emotion-perception deficits have been found in other clinical groups, particularly mentally retarded subjects (Hobson et al., 1988). These studies raise questions about the theorized primacy and specificity of the affective deficit to autism, but not the presence of an affective deficit in autistic persons.

There are other problems with Hobson's theory. Although Hobson has recently addressed the imitation deficit in autism (Hobson, 1990), he appears to suggest that the affective deficit in autism is related to motor imitation deficits in the following way. Deficits in early affective developmental processes prevent adequate development of self-other awareness, which im-
pairs the development of "basic intra- and interpersonal structures of bodily coordination." This impairment is reflected in poor motor imitation skills. Thus, Hobson argues that imitation deficits are secondary to affective deficits in autism. However, the neonatal imitation research would suggest that the capacity for simple motor imitations precedes affective development and development of self-other concepts. Some have suggested that neonatal imitation fosters the development of greater self-other awareness (Malatesa & Izard, 1984; Meltzoff, 1985; Stern, 1985).

In spite of these problems, there are important aspects of Hobson's theory that a comprehensive theory of autism needs to incorporate, even though the affective deficit may not be the one primary deficit in autism. In particular, Hobson (1989, 1990) and Stern both suggested a connection between development of affective mutuality and development of an awareness of other minds. Hobson (1990) also suggested an explicit model of development of some aspects of self-other awareness in the absence of other aspects of such awareness that addresses well the inconsistencies in social behaviors in autism described earlier.

**Metarepresentation.** A cognitive theory of autism involving metarepresentation has been developed by Baron-Cohen, Leslie, and Frith from a series of controlled studies published over the past five years. These studies have been very impressive in documenting deficits in autistic subjects on a wide array of converging measures of theory of mind—false belief tasks, appearance/reality tasks, and story sequencing tasks (Baron-Cohen, 1988, 1989a, 1989b; Baron-Cohen et al., 1985, 1986; Dawson & Fernald, 1987; Harris & Muncer, 1988; Leslie & Frith, 1988; Ozonoff et al., in press). Moreover, the rate of failure among autistic subjects (around 80%) is impressively high, as is the rate of success (again around 80%) among both mentally retarded and nonretarded controls with lower MA's than the autistic subjects. Clearly, these studies have identified a deficit that is at the core of autism and that is theoretically capable of explaining many features of the disorder. Any competing theory must accommodate these results.

The metarepresentational view suggests that autistic children cannot form a "theory of other minds" that is necessary to attribute mental states—thoughts, beliefs—to other people. This deficit in theory of other minds is viewed as resulting from a fundamental cognitive deficit in the ability to form second-order representations, or metarepresentations—representations of other mental representations. This theory assumes that the autistic child can form a primary representation of another person's physical self (or one's self) just as he or she would form a representation of a table or a chair. It assumes that the autistic child cannot understand another's mental state, because he or she cannot form a secondary representation, or metarepresentation, a mental representation of another's mental state. Thus, the metarepresentational account (correctly) predicts that autistic children should be unimpaired in self- and other-recognition, but markedly impaired in understanding other people's internal experiences. Hobson (1990) made a somewhat similar distinction between the physical and the interpersonal self and between I—It versus I—Thou relations to explain how autistic children could have well-developed knowledge of the physical world but very deficient knowledge of the social world.

The earliest example of metarepresentation in development is thought to be the use of symbolic substitutions in play (Leslie & Frith, 1988), which generally appears in the second year, although Baron-Cohen (1989c), among others, recently argued that joint attention may be an early precursor of metarepresentation in normal development. Baron-Cohen (1988, 1989c) accounted for autistic subjects' deficits in symbolic play, pragmatics of communication, joint attention, and theory of mind via the metarepresentational hypothesis.

Because of possible definitional confusion, we will use the term "metarepresentation" in the rest of this article to refer to
the complex cognitive capacity to form second-order representations. We will use "theory of mind" to refer to the child's awareness of internal cognitive processes in another, and we will use "intersubjectivity" to refer to reciprocal sharing of one's subjective experiences, whether affective or cognitive, with another.

Despite its great successes, there are several empirical and theoretical problems with the metarepresentational theory. First, the metarepresentational theory, focusing as it does on complex cognitive skills first supposedly demonstrated in symbolic play, has difficulty accounting for the imitation deficits, because imitation is a capacity that appears much earlier than joint attention and symbolic play in normal development. Second, the link between metarepresentational abilities and symbolic play has not been demonstrated empirically. Third, the metarepresentation theory posits a sharp discontinuity in the development of a theory of other minds, whereas other theories (such as Stern's) suggest a more continuous developmental course. In this way, the metarepresentational account neglects the importance of partial accomplishments (Haith, 1990). Although the metarepresentation theory can become less discontinuous by including joint-attention deficits as an early precursor, as Baron-Cohen (1989c) suggested, it would have to change drastically to accommodate sensorimotor imitation and affective deficits and the continuous development of intersubjectivity that they imply. Conceding this point would make their theory very similar to Hobson's.

Despite these shortcomings, the metarepresentational account has contributed important insights about the nature of autism. A majority of autistic children appear deficient in theory of mind, and this deficit can account for many of the social symptoms of the disorder. Second, this account treats awareness of other minds as a special kind of human interpersonal ability, a point alluded to by object-relations theorists, attachment theorists, ethologists, and philosophers. A developmental model of autism must incorporate these strengths of the metarepresentational theory.

Joint attention. According to Stern (1983) and Bretherton, McNew, and Beeghly-Smith (1981), the earliest behaviors that clearly indicate the infant's growing awareness of others' minds are joint-attention behaviors, behaviors that direct another person's attention to an object of interest in the environment: pointing, reaching, showing an object, looking back and forth between the object of interest and the adult's face, following another's gaze or point, first seen in the 9-12-month period. It is widely accepted that young autistic children are deficient in the use of joint-attention behaviors (Curcio, 1978; Loveland & Landry, 1986; Mundy & Sigman, 1989a; Mundy, Sigman, Ungerer, & Sherman, 1986; Wetherby & Prutting, 1984).

Several theorists argue that the joint-attention deficit is secondary, resulting from some already existing deficit in autism (e.g., Mundy & Sigman, 1989a, 1989b), but there is disagreement about what the joint-attention deficit is secondary to. Compelling theoretical arguments have been put forth both for viewing joint attention as an early cognitive, metarepresentational development (Baron-Cohen, 1989c), and for viewing it as an advancement in affect sharing and signaling (Hobson, 1989; Mundy, Sigman, & Kasari, 1990). Although there is not yet any empirical evidence to link joint-attention behaviors with other metarepresentational abilities, Mundy et al. (1990) demonstrated relationships between deficits in joint attention and abnormal affect sharing in young autistic children as compared with CA and MA matched comparison subjects. Early cognitive and affective development are not necessarily separate, and joint-attention behaviors may be both cognitive and affective milestones.

Other pragmatic processes. Abnormalities of speech and gesture usage in verbal autistic children involve deviance in the pragmatic functions (virtually all of which are
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impaired; Baron-Cohen, 1988), rather than deviance in phoneme, syntax, or semantics (Bartak et al., 1975; Beisler, Tsai, & Von, 1987; Wetherby & Prutting, 1984). The pragmatics of language usage involve social regulatory functions and social communicative aspects of conversation. Prinzant and Wetherby (1987) and Wetherby (1986) provided integrative reviews of the pragmatics literature in autism with emphasis on deficits in communicative intent. They described the intersection of developments in social, communicative, and cognitive domains in the development of intentional communication. They suggested that understanding these secondary pragmatic deficits will shed light on the primary impairments in autism.

Thus, both verbal and nonverbal communicative abnormalities of autistic children may be seen as being based in difficulties with the social (rather than linguistic or symbolic) aspects of communication. Studies document particular abnormalities of pragmatic functions that involve (a) coordinating attention of self and other or (b) sharing experiences or affective exchange. Thus, autistic people’s use of language mirrors other aspects of their social functioning, with some social capacities present and others, particularly those that require affective communication or knowledge of other minds, absent. Whether their pragmatic awkwardness also reflects absence of appropriate social use of imitation, normally seen in mirroring the partner’s posture, facial expression, vocal tone, and so forth, is unknown. However, Hoskins (1987) emphasized the importance of body imitation or mirroring in pragmatics of social conversation.

Symbolic play. The final area of specific deficit in autistic children to be reviewed here involves symbolic play. In normal children, symbolic play is believed to evolve out of three capacities: the capacity for deferred imitation; the capacity for referential communication; and the capacity for representational thought that children demonstrate in the second year of life (Fisch, 1962; Werner & Kaplan, 1963). Of these three, only the relationship between referential communication and symbolic play has been tested and demonstrated (Mundy, Sigman, Ungerer, & Sherman, 1987).

A variety of studies have consistently reported autistic deficits in symbolic play compared with control subjects (Mundy et al., 1987; Riquet, Taylor, Benaroya, & Klein, 1981; Sigman & Ungerer, 1984; Wing, Gould, Yeates, & Brierley, 1977). This is not to say that symbolic play skills are always absent in autism. There are reports of delayed symbolic play skills in autism (Lewis & Boucher, 1988; Rogers & Lewis, 1989). Thus, studies of symbolic play in autism indicate that there is a specific deficit (rather than a total absence) in symbolic play relative to other clinical populations.

The symbolic play deficit in autism is generally viewed as a secondary deficit, resulting from earlier appearing deficits specific to autism (Baron-Cohen, 1988; Hobson, 1989). Both the metarepresentation theory and the affective theory have accounted for the symbolic deficit, but there is as yet no empirical evidence that supports these hypothesized relationships. In fact, Mundy et al. (1987) demonstrated that in autistic children, symbolic play capacities were related to referential communication but independent of joint-attention skills. This finding presents problems for metarepresentational theory, which incorporates both joint attention and symbolic play as early evidence of metarepresentation. Studies that examine possible relationships among imitation, metarepresentation, affective function, and symbolic play are needed to help us understand the relationship among these skills in early development.

We have reviewed the autism literature regarding deficits specific to autism and the two conflicting current major theories—the affective theory and the metarepresentational theory. The imitation deficit appears to present a major challenge to both theories. The imitation deficit challenges Hob-
son's affective theory because imitation is present at birth in normal development, prior to affect sharing and thus does not seem to be secondary to affective development. Autistic deficits in both imitation and affect pose serious problems for the metarepresentational account, because the appearance of these skills in normal development is prior to the earliest manifestation of metarepresentation, and because they do not plausibly require metarepresentational ability. Both theories account equally well for the symbolic play and pragmatics deficits in autism, because both theories view pragmatic deficits and symbolic-play deficits as secondary to earlier, more primary deficits.

**Autism as a developmental disorder**

Before moving ahead, a caveat is in order. Although we speak of global deficits in autism, we recognize that autistic individuals vary considerably in the severity and variety of their symptoms. We agree with others (Harris, 1989; Sigman, 1989) that autism is a developmental disorder. We do not expect to find specific deficits in young autistic children that are maintained throughout development. Rather, we expect to see some signs of a deeper underlying deficit specific to autism stand out during a specific developmental stage, only to be accomplished to some degree at a later developmental stage and replaced by other symptoms of the underlying deficit. We suggest that symptoms will appear in whatever skills affected by the primary deficit(s) are most salient at a particular developmental point. Ours is a theory of heterotypic, rather than homotypic, continuity.

In fact, the current body of research supports this view. Joint-attention deficits in autism are clearest in preverbal, young autistic children and no longer appear in older, verbal autistic children (Stone & Caro-Martinez, 1990). Symbolic play deficits are clearest with low IQ and young autistic children, but symbolic play is certainly seen in higher IQ and older children and improves with treatment (Boucher & Lewis, 1989; Rogers & Lewis, 1989). Lower level theory of mind abilities have been found to be deficient in the majority of verbal, school-aged autistic subjects studied. Yet older, high IQ subjects with autism or PDD have demonstrated in some studies the accomplishment of first-order theory of mind at a level similar to controls and show comparative deficits only on second-order theory of mind tasks (Bowler, 1989; Ozonoff, Rogers, & Pennington, in press). Simple motor imitation skills are quite impaired in young autistic children, as described earlier. However, older, higher IQ autistic subjects demonstrate successes on infant-level motor imitation tasks that younger, lower IQ subjects have routinely failed (Morgan et al., 1989).

The notion of heterotypic (rather than homotypic) continuity is quite important in looking at studies that at first appear to be nonreplications. Nonreplications must be carefully examined to see whether the subjects and controls are of similar CA, MA, and verbal ages across studies. Furthermore, studies that find no differences between autistic subjects and comparison groups may only be indicating that both subject groups are too immature for the specific deficit to be demonstrated yet (i.e., theory of mind studies in young autistic and young retarded children), or that both groups have finally developed that skill (i.e., joint-attention behaviors in older verbal subjects).

Core to our thinking, then, is the premise that deficits on specific tasks, including tasks that tap the primary deficits in autism, will not be constant across development, but rather will change with development and experience. Further, we expect that improvement will occur in all deficit areas over time, from a small amount to a great deal in different individuals.

**A Continuous Model of Autistic Development Based on Stern**

What relationships might exist among the three deficient areas—imitation, affect sharing, and theory of mind—in early de-
velopment? Stern’s model of interpersonal and self-development suggests a variety of roles and linkages among these three aspects of infant social development. In addition, we (and others: see Hobson, 1990; Sigman, 1989) find that Stern’s model fits both the empirical and the clinical data regarding autism particularly well. Finally, Stern’s model suggests an integration of the affective and metarepresentational positions along a developmental continuum. In this section, we will review Stern’s four self domains, and we will suggest effects that might result from early deficits in imitation, affect sharing, and theory of mind, which will take the form of a suggested developmental model of autism.

Although we find Stern’s theorizing quite important to understanding autism, we recognize the danger of assuming that deficits that are demonstrated during the preschool or school-aged years in autistic children were also present during infancy. Because a diagnosis of autism is seldom made before age 2, understanding infant development of autistic persons is very difficult empirically. Thus, we recognize that this is only a hypothetical model.

**Sense of an emergent self**

The very young infant’s emergent sense of self involves the experience of emerging patterns of organization, of grasping relationships between separate experiences. The infant experiences an organization in the social world via early imitative exchanges and via the dialogue of “vitality” affects that the partners share (which may also allow the infant some innate level of processing human affective expressions, according to Malatesta & Izard, 1984). Stern postulates an ability of the infant in the first months of life both to form representations of other people’s behaviors and affective signals and to plan and execute an expressive response. The empirical basis for these concepts is found in Meltzoff’s work on neonatal imitation and on amodal perception, as well as in the observational studies of Trevarthen and of others documenting the complexity of infants’ social exchanges in the first few months of life (Meltzoff, 1988; Meltzoff & Moore, 1977; Trevarthen, 1979).

Autistic deficits in imitation and emotion sharing would greatly affect the baby’s ability to organize social information concerning other people by depriving the baby of primary sources of social data—mother–baby imitations and amodal perception of emotion via the mother’s bodily expression of emotion (in Stern’s terms, the vitality affects). However, the baby’s ability to organize information about the physical world through amodal perception would not be specifically affected. Thus, in this stage the preautistic infant would develop a sense of organization in the physical world, while lacking a parallel sense of organization in social interactions and in the social world.

**Sense of a core self**

The second domain of self-awareness, awareness of core self, involves a phase of intensive and almost exclusive sociability lasting from roughly 2–6 months. The normal infant is an organized, highly motivated social partner. The “topic of conversation” in social exchange is affect, and interactions are focused on sharing and regulation of affect and excitation. Caretakers regulate the infant’s self-experiences via regulating diverse aspects of the infant’s experience, including arousal, affect intensity, discrete affects, security, somatic state, attention, curiosity, and engagement with the physical world.

In this phase, the preautistic infant would develop more normally in areas that reflect an understanding of the relationships between the self and the physical world; these include a bodily self, a sense of agency, and memory for familiar experiences. However, the infant’s imitative and affective deficits would affect both the infant’s internal experience of affect and the infant’s ability to perceive and share affect with the caretaker. Finally, the preautistic infant could experience the mother as a reg-
ulator of some self-experiences, particularly involving agency, but the affective deficit would not provide the infant with the sense of the mother as a regulator of affective experience.

How would these differences affect infant social representations? We suggest that generalized representations of other people would have to be built up largely from invariants extracted from observed behavioral contingencies, without the huge affective data base available to other infants. Thus, we agree with Baron-Cohen (1989b) that autistic children (especially early in life) are essentially behaviorists, that their knowledge of others is most developed regarding the effect that other people have on themselves and the environment as causal agents. We expect that the relatively unafflicted social skills (reviewed earlier in this article) will be developed from their learning of behavioral sequences and consequences rather than intersubjective knowledge of shared affect or mental states. Thus, the autistic child could develop a behavioral “working model,” in attachment terms, of the mother, as demonstrated by autistic children’s differentiated social appeals for assistance and by observations of differential social responsiveness and attachment behavior in autistic children, but this working model would lack an organized affective core (Rogers et al., 1991).

**Sense of a subjective self**

In this third domain, the normally developing infant becomes aware of subjective mental states: feelings, focus of attention, motives, and intentions that lie behind physical actions in self and in others. These mental states now become the subject matter of relating. Infants and partners share subjective experiences through intentional communication of intentions, emotions, and the focus of their own attention.

What accounts for the shift from core self to intersubjective self? Stern suggests that shared affect “may be the first, most pervasive, and most immediately important form of sharing subjective experiences” (1985, p. 132). Stern feels that shared affect leads the infant to recognize that other internal states are also shareable with other humans.

Although Baron-Cohen (1988, 1989a) and others suggested that the development of the primary theory of mind deficit in autism is discontinuous, appearing late in infant development, Stern’s theory suggests that the infant who has not experienced social mutuality through imitated coordinations of physical selves, or emotional mutuality through coordinated affective exchanges, lacks the information needed to proceed to an awareness of and coordination of subjective experiences with another. Thus, we hypothesize that the preautistic infant would have difficulty developing a sense of interfacing minds or a theory of other minds, because he or she lacks so much social information and experience due to deficient developmental precursors, and because specific biologic autism-related deficits would directly interfere with the formation and use of intersubjective representations. The full effect of the deficits in autism are encapsulated in Stern’s description of his stage of intersubjectivity. We suggest that the deficit in the feeling of “relatedness” that characterizes autism reflects this deficit in intersubjectivity, which is made up of normal mirroring of other’s behavior, shared affect, and awareness and sharing of subjective experiences.

**Sense of a verbal self**

In intersubjective sharing, the infant uses communicative behaviors to refer to an unseen, subjectively experienced mental state. Stern sees such nonverbal communications as an essential step toward the use in this fourth domain of more formal symbols—words and symbolic play—to refer to mental content. Similarly, Miller (1990) suggested that human language represents the integration of two systems, one communicative and one symbolic, parts of which may exist separately in other species. Autistic deficits are seen in both of these sys-
tems. Let us first focus on deficits in symbolic play.

Symbolic play. Humphrey (1988) suggested that young children's symbolic play demonstrates their complex social knowledge and overriding interest and immersion in the social world. Even though interactions with the social world are more complex than interactions with the physical world, young children are absorbed by the social world as reflected in their symbolic play, which converts physical props into social scripts. The autistic child is shut out of the richness and complexity of the social world due to the impact of the imitation, affect, and intersubjective deficits. Autistic children are not immersed in the social world; rather, their mastery is of the physical world which is the focus of their play. Additionally, major theorists on symbolic play have stressed the primacy of the role of deferred imitation in the development of symbolic play (Baldwin, 1906; Piaget, 1962; Stern, 1985; Werner & Kaplan, 1963). Thus, the imitation deficit in autism may also play a primary role in the symbolic play deficit. Thus, to summarize, if the purpose of play is to practice and maintain newly mastered schemas, as Piaget (1962) suggested, the autistic child has too little knowledge of the social world to act it out in play and, instead, spends time in play that involves physical manipulation of objects.

Language. Now let us move to language. The earliest indication of a communicative deficit in young autistic children involves lack of joint-attention behaviors, which Mundy and Sigman (1989a, 1989b) suggested serve both affect sharing and theory of mind. We suggest that for the autistic child, (a) joint-attention behaviors are not developed as a means of sharing affect, since the capacity for affect sharing is primarily impaired, and (b) they are not developed as a communicative vehicle, since the autistic child is not aware of the partner's subjective experience and does not know of the partner's mind and attention, let alone how to command them.

Bruner (1975) suggested that joint-attention behaviors are crucial for coordinating adult and infant minds so that the infant can gain access to the meaning of the adult's spoken words. Without some knowledge of other minds, the autistic child cannot develop with the mother a nonverbal repertoire of sharing meanings and so has no communicative foundation to help interpret symbolic verbal behavior. This view would explain the common history in autistic children of early words that drop out or do not progress. We would suggest that in such cases, the biologic readiness for speech demonstrates itself, but without a nonverbal communication system already in place, the early words cannot be incorporated into an already existing communicative framework, meaning cannot be inferred, and, serving no function for the child, words drop out of the child's repertoire. However, as social knowledge and early communicative behaviors slowly increase, autistic children develop some shared meanings and many eventually develop verbal language.

This formulation also provides a way of understanding other pragmatic deficits. If the autistic child cannot grasp the speaker's meaning intersubjectively, words only come to mean something as they become associated with environmental events in a contingent fashion. "Do you want a cookie?" becomes associated with receiving a cookie, and, as the child's memory and verbal production permit, he or she uses "Do you want a cookie?" to request a cookie. Echolalia may be an alternate route to language learning, a detour around the intersubjective deficit that allows the autistic child to piece together the beginnings of a verbal language system. Once begun, the child's growing knowledge of language through use helps him or her discover the underlying rules for syntax and semantics (Tager-Flusberg, Calkins, Nolin, Baumberger, Anderson, & Chadwick-Dias, 1990). The phenomenon of pronoun reversal can be understood in the same way. The autis-
tic child does not have a foundation of shared meaning to help interpret deictic words, including pronouns, and instead uses associative learning to decode them—which will result in pronoun reversals.

Normal pragmatic use of language also requires that one grasp the underlying conveyance of affective information. Other pragmatic deficits of language seen in autism—inappropriate use of speech imitation and questioning, stilted speech, use of language as a means rather than an end, abnormal prosody—can all be seen as resulting from language learned to affect the physical environment without a foundation in affect sharing and in understanding of what might be in another persons' thoughts.

**Blind infants.** There is one other clinical population—blind infants—in which developments in Stern's fourth domain are significantly affected. In blind infants, symbolic play deficits are marked, and echolalia and pronoun reversal sometimes appear in the early stages of language development (Friedberg, 1977; Rogers & Puchalski, 1984). Blind infants are cut off visually from motor imitation, from visual affect mirroring, and from the mother's visually referenced nonverbal communications (i.e., mother-initiated joint-attention behaviors). The resulting lack of social information may be reflected in blind children's relative lack of symbolic play.

In terms of language development, the blind infant cannot use intersubjectively constructed, visually referenced shared meanings to interpret the mother's speech. The blind infant may use contingency learning and verbal memory as an alternate route to language acquisition, resulting in echolalia. However, once language is established, blind children may use language as an alternate route for development of interpersonal relatedness and intersubjectivity. We suggest that unlike autistic infants, blind infants are not missing biologic capacities for imitation, affect sharing, and intersubjectivity, but rather the sensory information that supports such developments in young infants.

**Theoretical integration**

We are suggesting a cascade model of autism in which lack of certain aspects of interpersonal development at every previous stage disrupts certain developments in the following stage. In the theoretical integration that follows, we view early imitation skills, emotion sharing, and theory of mind as increasingly complex expressions of the ability to form and coordinate certain representations of self and of another and to use those representations to guide the planning and execution of one's own behavior.

In terms of imitation, Meltzoff (1988; Meltzoff & Moore, 1977), Stern (1985), and Mandler (1988) suggested that the capacity of a neonate to imitate an adult facial movement denotes an innate capacity to form and act on an amodal representation, for the infant is matching a visual perceptual display to a kinesthetic movement on the basis of some more global representation of aspects of faces. It thus appears that the infant has an already existing template of some aspects of the human body that allows the infant to coordinate self and other body movements.

In terms of affect sharing, Malatesta and Izard (1984) suggested that affect sharing and mirroring likewise require a kind of amodal representational matching, in this instance between the adult's expressive display (facial affect, vocal tone, and Stern's "vitality" affects) and the infant's subjectively experienced emotional state. (This type of amodal matching must also underlie maternal "affective attunements," as described by Stern.) Further, Malatesta and Izard suggested that infant imitation allows the infant's prewired affective displays to be "shaped up" via perception and imitation of the adult's full affective display. (This suggests us a parallel between the "shaping up" of an infant's facial affect display through the adult-infant affect match-
ings and the phonetic "shaping up" of an infant's vocal productions through the adult-infant vocal imitations to match the cultural-linguistic phonetic system.)

In terms of theory of mind, Bretherton et al. (1981) and Bruner (1975) suggested that the infant's experience with shared affect combined with increasing cognitive and affective development leads continuously into the infant's sharing of representations of mental states, or theory of mind, as demonstrated in intentional and referential communication and the joint-attention behaviors. As the infant has increasing experience of self-agency through motor acts, the infant's subjectively experienced affects, motives, and intentions expand. The infant continues to share these subjective experiences with a receiving and mirroring partner via the ever-widening behavioral repertoire of face, hand, and body movements. This results in the gradual development of the joint-attention behaviors, which serve to share increasing meanings and motives with the partner, thus demonstrating the infant's growing awareness of "interfacing minds" (Bretherton et al., 1981), as well as to continue the dialogue of shared affect established earlier.

Thus, these three early social capacities that seem to be primarily and specifically deficient in autism—imitation of another's body movements, emotion sharing, and theory of mind—appear to involve forming and coordinating specific social representations of self and other at increasingly complex levels via amodal or cross-modal representational processes that extract patterns of similarity between self and other. Further, human infants appear to be biologically prepared to develop these social behaviors. Each of these capacities demonstrates the infant's increasing knowledge of others and opens doors for the acquisition of much more social knowledge of self and other.

The end result of the triad of core developmental deficits in autism is deficient awareness of other's affective states and subjective minds and deficient ability to represent one's own or others' subjective experiences—feelings, thoughts, intentions, hopes, beliefs—via pretend play, affective communion, or language. In choosing a label for this model, we cautiously turn to the term "intersubjectivity," not in the narrower sense (i.e., theory of mind) that Stern suggests, but in the broader sense that Trevarthen uses (including both primary and secondary intersubjectivity). As we defined it earlier in this article, this term refers to reciprocal sharing of one's internal, subjective experiences, whether affective or cognitive, with another via imitative, affective, and communicative modes. This intersubjective deficit in autism is, of course, combined with whatever other developmental deficits, such as retardation, the autistic person may also have. The autistic child is not seen as choosing to withdraw from the social world or to avoid it (although the difficulties of trying to live socially without shared affectivity or intersubjectivity may eventually lead to avoidance). Rather, the child cannot access the social world via physical mirroring, affective mutuality, shared meanings, and some understanding of the inner life of others.

A diagrammatic depiction of this view is presented in Figure 1, in contrast to Baron-Cohen's (1988) representation of his and Hobson's (1989) theories. It is important to note that in all three diagrams, the relationships between deficits are generally hypothesized and are, as yet, without empirical support. In the diagram of our intersubjectivity theory, we suggest that the basis for autism lies in the deficient capacity to form or manipulate the particular (generally amodal) representations of self and others that underlie infant body imitation, affect mirroring and sharing, and awareness of other's subjective states. We suggest that this disability affects all three capacities directly; hence, the arrows from the self-other representations box into imitation, emotion sharing, and theory of mind. However, we also suggest that in normal development these capacities appear sequentially in this order, with each
(a) the emotion theory of Hobson (Baron-Cohen, 1988)

Lack of innate ability to interact emotionally with others

- Failure to recognize other people's mental states
- Impaired ability to abstract and symbolize

Emotion-recognition deficits

Pragmatic deficits

Pretend play deficits

(b) the metarepresentational theory of Frith, Leslie, and Baron-Cohen (Baron-Cohen, 1988)

Impaired meta-representational capacity

- Impaired 'theory of mind'
- Impaired symbolic skills

Specific social skills impaired, others spared

Pragmatic deficits

Pretend play deficits

(c) the intersubjectivity theory (Rogers & Pennington)

Impaired formation/coordination of specific self-other representations

- Impaired imitation
- Impaired emotion sharing
- Impaired 'theory of mind'

Impaired pretend play deficits

Joint attention and pragmatic deficits

Figure 1. Contrasting theoretical models of the primary deficits in autism.
capacity influencing the development of the next. In autism, deficits in each capacity will negatively affect development in the next capacity, which results in a continuous, cascade model; hence, the horizontal arrows leading from imitation to emotion sharing and from emotion sharing to theory of mind. Arrows from the second to the third tier represent hypothesized relationships between the more primary deficits and the more secondary deficits. As described earlier, both imitation and theory of mind capacities are theorized to play important roles in the development of symbolic play. Likewise, imitation, affect sharing, and theory of mind are all theorized to be necessary for normal pragmatics development, both at the joint-attention stage and later in social speech. This model thus makes strong predictions about the intercorrelations of five developmental accomplishments.

As discussed earlier, the main problems with Hobson’s affective theory are its difficulty in accounting for the timing of the imitation deficit and the lack of specific deficits in some social processes such as attachment. The main problem with Baron-Cohen’s theory of mind hypothesis is its difficulty accounting for earlier deficits in imitation and emotion sharing. Our theory differs from Hobson’s in its portrayal of the role of the imitation deficit. It shares with Hobson the importance of emotion sharing in development of early and later self/other experiences. Our theory differs from Baron-Cohen’s in its view of theory of mind deficits as not primary and discontinuous, but rather one of several types of interpersonal capacities that are affected in autism. It shares with Baron-Cohen the importance of a theory of other minds in social development. The strengths of the present model are its ability to integrate all major areas of deficit in autism in a coherent developmental framework, its ability to encompass both intact and deficient social skills, its ability to integrate other competing theories of autism, and its ability to account for differing profiles of similarity and differences between autistic people and comparison groups across development. It also provides an alternative interpretation of the pragmatic and symbolic play deficits in autism. We agree with Hobson and Baron-Cohen that the social deficits in autism are primary, lasting, and appear to reflect dysfunction in the biologic substrate of human sociability, a topic we turn to next.

A Possible Biologic Model of Autism

We are suggesting a biologically based developmental model of childhood autism. Can a biologic model account for the triad of imitation, affect sharing, and theory of other minds? Let us turn briefly to some neuropsychological thinking and research concerning these aspects of social behavior.

Hypothesized role of prefrontal neocortex in social deficits

In 1978, Damasio and Maurer suggested that autistic children shared many characteristics with adults who had sustained damage to the prefrontal neocortex: “lack of initiative, a similar concreteness in thought and language, an inability to focus attention, shallowness of affect and lack of empathy.” Around the same time, MacLean (1978) proposed that development of the prefrontal cortex gives rise to the ability to look within oneself and evaluate one’s emotions and then to project those feelings onto others to allow us to feel and share their sorrows, joys, pain, happiness—in essence, intersubjectivity. Suggested neurological pathways for intersubjectivity involve prefrontal–limbic connections. Nauta (1971) called the frontal lobes the main neocortical projection of the limbic system. There are extensive reciprocal connections between the limbic system and the frontal lobes; some of these connections pass through the cingulate gyrus, which may have an important role in coordinating limbic and prefrontal functions. Through its connections with the limbic system (Damasio, 1985; Stuss & Benson, 1983), the prefrontal neocortex could
sample limbic regions that signal internal activities representative of emotion and combine the results with calculations about the future to generate understanding of others and shared awareness.

Neuropsychological data concerning autism

Now let us turn to empirical data concerning prefrontal deficits in autism. Although a variety of neurological and neuropsychological studies have been carried out over the years, a series of recent studies of frontal functions from several different labs are beginning to provide some consistent findings. One biological study has provided some evidence of frontal dysfunction in autism. Horwitz, Rumsey, Grady, and Rapoport (1988) reported results from a positron emission tomography (PET) scan study of autistic subjects in which correlations between frontal and other brain regions (parietal and certain subcortical structures—thalamus, caudate nucleus, and lenticular nucleus) were reduced relative to controls.

There have also been several neuropsychological studies using measures sensitive to frontal lobe deficits. On a variety of measures considered sensitive to frontal lobe deficits, deficits in nonretarded autistic subjects when compared with appropriate control groups have been a consistent finding (Ozonoff, Pennington, & Rogers, in press; Ozonoff, Rogers, & Pennington, in press; Prior & Hoffman, 1990; Rumsey, 1985; Rumsey & Hamburger, 1988, 1990; Szatmari, Bartolucci, Bremmer, Bond, & Rich, 1989). Such deficits have been found mainly on two tasks: the Wisconsin Card Sorting Task, which requires flexible shifting of cognitive sets and inhibition of prepotent responses, and the Tower of Hanoi, which requires planning and depth of search.

Social deficits of nonautistic frontal patients

In addition to evidence of autistic dysfunction on neuropsychological tasks thought to tap frontal functions, there are a number of studies of adults with early frontal injuries who evidence social impairments similar (but not identical) to those seen in autism. Price, Daffner, Stowe, and Mesulam (1989) described major social-cognitive deficits in role-playing tasks, moral reasoning, and formal operational thought in two adults with early bifrontal lesions. Damasio and Van Hoesen (1985) and Kolb and Milner (1981) reported that lack of pleasure in social interactions, disturbance in both experience and expression of emotion, and disturbance of communicative drive, including mutism, were routinely found in adults with lesions in the mesial or orbitofrontal surfaces of the frontal lobe. Furthermore, imitation deficits of both simple body movements and symbolic, or pantomime, movements have been reported in adults with frontal dysfunction (De Renzi, Motti, & Nichelli, 1980; Kolb & Milner, 1981). Thus, there is some evidence from the adult neuropsychological literature that deficits in imitation, emotion expression/experience, social and communicative initiative, and “intersubjectivity” (as seen in lack of empathy, role playing, and other social-cognitive deficits) result from frontal injuries. Although these case reports of early frontal lesions in humans (e.g., Price et al., 1989) do not include classical autism as an outcome, these patients were deficient in empathy and social perspective taking, so there was some overlap with autistic symptoms. Since it is beginning to appear that several different psychiatric disorders involve a frontal component (Goldman-Rakic, 1987), it is important to keep in mind the complexity of this large part of the cortex and to work toward more differentiated accounts of its role in normal and abnormal behavior. It may also be possible, as Prior and Hoffman (1990) suggested, that abnormalities of lower brain functions could secondarily affect frontal functioning in autism. Only continued neurobiologic and neuropsychological explorations of functioning in autistic persons will allow us to determine more precisely the relation between frontal functioning and
these recurring deficits in flexibility, anticipation, and representation in autistic subjects.

Difficulties with an executive function/frontal deficit hypothesis

While empirical data from neuropsychological studies of both autistic persons and frontally injured persons could lead one to hypothesize that frontal deficits might lie behind the social deficits in autism, there are a variety of potential difficulties with such a hypothesis. One apparent difficulty is the timing question. Although autism appears early in childhood, it has traditionally been accepted that frontally mediated neuropsychological skills appear later in childhood. Recently, however, views that the frontotemporal lobes are not particularly active in early life are giving way (Welsh & Pennington, 1988). PET scan data involving normal infants suggest that metabolic activity in frontal areas reaches adult levels at about 8 months postgestation (Chugani & Phelps, 1986). There are also telling across-species behavioral studies. Diamond and Goldman-Rakic (1985) demonstrated that object-search skills that normally develop in human infants between 9 and 12 months are most likely subserved by the prefrontal cortex in humans via cognitive operations involving representational thought and representational memory (Diamond & Goldman-Rakic, 1985; Diamond, Zola-Morgan, & Squire, 1987; Goldman-Rakic, 1987). Finally, Mandler (1988) suggested that even earlier in infancy there is evidence of representational thought and memory, skills generally considered to reflect frontal function.

Clearly, we do not yet have evidence that the frontal lobes are metabolically or behaviorally active in humans before 6 months of age. However, even if the frontal lobes demonstrate less than adult levels of metabolic activity in the first 6 months of life, this does not preclude the presence of some frontal activity. Moreover, the neural circuits that have been proposed to subserve the social behaviors affected in autism are not exclusively prefrontal, but include the limbic regions as well. Thus, there is evidence from behavioral studies to support the idea that some frontal functioning is present in the first year of life, when the neural deficits underlying autism are hypothesized to begin affecting development.

A second problem involves the question of relationship between social cognition and executive function as measured by tasks such as the Wisconsin Card Sort or the Tower of Hanoi. How might frontal functions involving executive function—planning, anticipation, inhibition of prepotent responses, flexible shifting among mental schemas, holding a mental representation on line while possible responses are compared (Welsh & Pennington, 1988)—be involved in the social deficits in autism? Humphrey (1988) argued that the greatest complexities of primate life occurred in the social, rather than the physical, realm. A variety of social behaviors of primates in complex social groups appear to involve both affect perception/sharing and theory of other minds. Primate skills thought to reflect theory of mind are particularly evident in deceptive acts in which animals appear to anticipate a conspecific’s (partner or antagonist) intentions and behavior, which will vary depending on the individual’s actions (Byrne & Whiten, 1988). Such abilities appear to require executive-function strategies involving foresight, flexible planning, and inhibition. Thus, Humphrey theorized that executive-function skills evolved to serve social planning in primates, and in humans came to be applied to both the social and the physical world. Deficits in such skills would appear to be in the social or cognitive realm, depending on whether the problem at hand was a social or a nonsocial problem, but would actually reflect a deficit in a neural system that can be applied to either social or nonsocial problems.

A third problem with applying an executive-function deficit hypothesis to autism is that autistic persons can demonstrate excellent planning and problem-solv-
ing skills in some domains, such as finding a favorite food or object that parents have tried to hide. More formally, autistic children have not been found to be impaired on the AB task, which is identical to the classic frontal task in nonhuman primates, the delayed response task. Should not autistic children be impaired on frontally mediated object-search tasks if, in fact, frontal dysfunction is present? Not necessarily. The AB task may simply be too easy to detect frontal deficits in most autistic children. Goldman-Rakic's (1987) theory of frontal function suggested that forming representations is mediated by posterior cortex, while using those representations to form future plans is a separate process mediated by the prefrontal cortex. Furthermore, she theorized that different parts of the prefrontal cortex specialize in planning future behaviors based on different kinds of representations—spatial, object-orientated, motivational, and so forth. From this differential view of prefrontal cortex, we would predict persons with frontal deficits would show impairments on some, but not all, measures of prefrontal function depending on the locus of the dysfunction. We would also predict that deficits on “frontal” tasks could be caused either by frontal dysfunction per se or by dysfunction in other brain regions that supply the particular representation on which prefrontal planning is based. This second notion is similar to ideas proposed by other authors (e.g., Frith & Frith, in press; Goldberg & Bilder, 1987) concerning the vulnerability of prefrontal integrative functions to deficient input from other brain regions. If applied to autism, this view could account for the ability to form some social representations (i.e., the understanding of another’s visual perspective) concomitant with the inability to form other kinds of social representation, for instance, another’s cognitive or affective perspective. It could also account for autistic children’s ability to pass some executive-function tasks, such as the A not B search task, while failing other tasks that appear to require executive function, such as joint attention (Butterworth & Grover, 1988). We would also predict that there might be deficits in some autistic people in the formation of such representations, and in others only in the use of those representations to generate future plans. Additional predictions are implicit in this line of thought: (a) the development of differing aspects of social functioning can be disrupted by a number of different lesions to different parts of this neural system, (b) that only some of these will cause autism, and (c) that there may be neurologically distinct subtypes of autism.

A fourth potential problem in the application of executive-function deficits to autism is the affective deficit in autism, which would appear to involve the limbic system rather than the prefrontal cortex. However, as previously stated, Nauta (1971) suggested that the frontal lobes are the main neocortical projection of the limbic system. There are areas of the prefrontal cortex, such as the orbital area, that have extensive reciprocal connections with the limbic system, and these connections might specialize in the representation of affective, motivational, or social-affective information. Dysfunction in this circuitry could disrupt this type of affective information processing.

A fifth potential problem involves suggesting one common locus of deficit in autism when it seems clear that a variety of biologic conditions appear to underlie the disorder (Coleman & Gillberg, 1985). However, different etiologies may all lead to pathophysiology in the same critical frontal-limbic circuit and, nonetheless, cause variable deficits in other systems. It appears to us that in addition to the hypothesized frontal-limbic deficit, other kinds of neuropsychological impairments are probably also present in most autistic people and account for the correlated symptom that they share with other clinical groups including children with retardation, attention disorders, stereotypes, and sensory perceptual dysfunction. We are suggesting that in autism, a final common neurological pathway of these various biologic conditions may reflect the result of dysfunct
tion of specific limbic-frontal circuitry sustained very early in life, and that this dysfunction accounts for the commonality and specificity of social deficits seen in autism.

Summary

There are three early social capacities that seem to be primarily and specifically deficient in autism—imitation of another’s body movements, emotion sharing, and theory of mind. We suggest that these abilities involve forming and coordinating social representations of self and other at increasingly complex levels via amodal or cross-modal representational processes that extract patterns of similarity between self and other.

Stern’s interpersonal theory delineates interrelationships among these three social capacities, and views subjective knowledge of self and other as starting with the physical self, shared through imitation, moving to the affective self, shared through mutual emotional exchanges, then to the intersubjective self, shared through a theory of interfacing minds and the concomitant developments in referential communication and shared meanings, and finally to the symbolic self, in which one’s experience is shared through symbolic play and language. We posit that autism involves deficits in early development of the coordination of self/other representations at each of these levels.

As a secondary part of this article, we have suggested that a neuropsychological model of this kind of interpersonal coordination suggests a particular kind of representational mapping for which the prefrontal cortex appears to be particularly well suited because of its connections with both posterior cortex and limbic system. In applying such a model to autism, we are suggesting that certain connections to and within the prefrontal cortex do not function normally and do not allow for the mapping between representations of self and other necessary for the specific physical, affective, or mental coordinations seen in imitation, affect sharing, and theory of other minds. We suggest, however, that other connections in prefrontal cortex, as well as the functions of the posterior cortex and reciprocal connections with the limbic memory systems, are preserved. This allows the autistic person to develop complex primary representations (as opposed to metarepresentations), to make interconnections among them, and to store them in long-term memory. These preserved abilities account for the presence of social and nonsocial capacities and, in a few autistic people, remarkably advanced skills. This neuropsychological model is not necessary to the developmental model of autism presented in this article, but rather presents a possible biologic extension that is congruent with the developmental model. It does, however, generate some testable neuropsychological hypotheses concerning autism.

Several issues remain unaccounted for by the present model. One is the need for a detailed model of the formal structure of intersubjective representational mappings and how the brain might compute them. Our theory would suggest that there would be deficiencies in the formation of some human representations, particularly in terms of representation of affective and other “mentalistic” aspects of human beings, as well as in the intercoordination of some self and other mappings.

A second problem is the emphasis on the imitation deficit, given the presence of echolalia in some autistic people. There are two aspects to consider. First, we are not hypothesizing that deficits are lifelong. We would suggest that an imitation deficit may be most central and most handicapping early in life. An autistic child may well slowly discover the similarity of self and other’s bodies and may master body imitation later. Thus, we would predict the greatest imitation deficit in autistic persons of young chronological or mental age. Second, as stated earlier, we postulate different loci and circuits in the brain corresponding to different representational systems. The neural circuit involved in immediate and delayed verbal repetition in-
volves the peri-Sylvian language structures, including Wernicke's area, the arcuate fasciculus, and Broca's area. This circuit can operate autonomously, with little reference to meaning. Echolalia may represent the preserved function of this verbal-repetition circuit. Thus, we would predict some dissociation between body imitations and verbal imitations. We need imitation studies of echolalic autistic subjects to assess the relative universality of motor imitation deficits in autism.

A third problem that has not been addressed is late onset of autism. If autism can begin after the first 12 to 18 months of life when intersubjectivity should be well begun, how might this theory account for it? We would suggest that late onset might be understandable in two ways (Rogers & DiLalla, 1990). The first explanation involves late detection of symptoms. We would hypothesize the first symptoms of autism in infancy to be subtle "negative" symptoms in infants—lack of imitation, affective mutuality, and joint-attention behaviors—behaviors that are not as compelling to new parents as motor milestones and would be easy to miss in the presence of an otherwise thriving child. Second, we would suggest that some cases of late onset involve later-occurring, acquired neurological damage, due to clinical or subclinical causes, that disrupts the prefrontal connections necessary for intersubjectivity as well as for emotion expression/perception, thus resulting in the loss of earlier developed language, social, and play skills that is sometimes reported by parents of children with late-onset autism.

A final problem to be addressed here is the lack of empirical data demonstrating the hypothesized relationships among the main deficit areas. From this theoretical understanding of autism some novel hypotheses can be made that differ from Hobson's or Baron-Cohen's predictions. We would predict that in a longitudinal study of normally developing infants there would be positive, predictive correlations between imitation, emotion sharing, joint attention, and later performance on theory of mind tasks. In addition, we would predict significant positive correlations among these domains in both longitudinal and cross-sectional studies of young autistic children. We would also predict a pattern of profile differences in young autistic children as compared with appropriate comparison groups—a profile involving related deficits in imitation, affect sharing, joint attention, and symbolic play. In older autistic people, we would predict a specific profile involving related deficits in theory of mind, affect praxis, and pragmatics of language. Moreover, across these kinds of studies we would expect relations between all these deficits and executive-function tasks. The studies hypothesized here would provide some of the critical tests needed to decide among the alternate theories of autism described in this article.

References


Melzoff, A. N. (1985). The roots of social and cogni-
Theoretical approach to infantile autism

disabilities of early frontal lobe damage. Unpublished manuscript.
Sigman, M., Mundy, P., Sherman, T., & Ungerer, J. (1986). Social interactions of autistic, mentally

Theoretical approach to infantile autism

disabilities of early frontal lobe damage. Unpublished manuscript.
Sigman, M., Mundy, P., Sherman, T., & Ungerer, J. (1986). Social interactions of autistic, mentally
retarded, and normal children and their caregivers. 


