Autism is defined as a developmental disorder that includes three primary features (American Psychiatric Association, 1994). First, there is social impairment, such as lacking eye-to-eye gaze, or lack of social-emotional reciprocity. Second, there is communicative impairment, such as delayed language development, or poor ability to initiate or sustain conversation. Finally, there are restricted repetitive and stereotypic patterns of behavior, such as inflexible adherence to specific nonfunctional rituals. Autism is evident before 3 years of age, when children show delayed or abnormal social interaction, language, or symbolic imaginative play. Many theories have been advanced regarding its etiology. In recent years, scholars have become excited by theories suggesting that a deficit in a Theory of Mind module, or in cognitive executive functioning, or in social-orienting skills may underlie this mysterious disorder.

The authors of this article propose that “a developmental and dynamic systems perspective” may be important in understanding the social and communicative deficits in autism. They emphasize throughout the article that the functioning of the developing brain underlies the child’s ability to interact with the social environment and is in turn shaped by those interchanges; that these reciprocally influence one another. By observing the deficits that appear when the system breaks down, we can learn about how it normally develops.

**Overview Questions**

1. What communicative and language skills are impaired in children with autism? What areas of the brain appear to be focal points for these types of social-communicative impairments?

2. What are Theory of Mind and executive functioning? Explain in your own words why some people believe that each of these is responsible for the social and communicative deficits of autism.

3. Explain in your own words how an early disturbance of social orienting could influence the development of a) Theory of Mind and b) executive functioning. With these in mind, explain/give an example in your own words of how “the dynamic interplay between initial biological insult and subsequent transactions with the environment may be crucial to an understanding of autism.”

**Focus Questions**

1. What is metarepresentation, and how is it thought to be responsible for the social deficits of autism? Briefly describe a false belief test and explain why this involves metarepresentation. Give three reasons why the social deficits of autism might be due to a modular (i.e., specialized, neurologically-based) social-cognitive process.

2. Executive functions enable people to do what three things? Why do some people believe that difficulties people with autism have on false belief tasks are due to executive function problems? The left medial frontal gyrus has been linked to what Theory of Mind task and what executive functioning task? What are four problems with executive functioning as an explanation of the social-communicative problems of autism?

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**On the Nature of Communication and Language Impairment in Autism**

Peter Mundy and Jessica Markus

The social and communication disturbance of autism is characterized by a syndrome-specific pattern of strengths and weaknesses, rather than a pervasive lack of responsiveness to others. In children with language, this pattern is manifest as relatively well-developed phonological, syntactic, and semantic facilities, but impaired or deviant pragmatic capacities. In preverbal children, communication for instrumental or attachment functions may be observed, but joint attention, as well as other more purely socially oriented bids, are often lacking. Three neuropsychological models have been proposed that explicitly address elements of this pattern of social communication disturbance in autism. These models differ in the mechanisms of impairment proposed to explain the social-communication disturbance of autism. Nevertheless, these models converge to suggest that the specific pattern of social communication disturbance displayed in autism results from a dysfunction that involves frontal neurological processes. A discussion of the similarities and differences among these models is presented. In the final analysis, this discussion leads to two conclusions. First, it may be necessary to adopt a developmental and dynamic systems perspective to gain a complete understanding of the complexities of the social-communication pathology of autism. Second, the study of autism raises many important observations and hypotheses regarding the ontogeny of the quintessential human capacity for communication and social cognition.

Autism is a pernicious and biologically based disorder that is characterized by impaired social development [Kanner, 1943; Bailey et al., 1996]. It may be more prevalent than once thought, occurring at a rate of 1:1000 [Bryson, 1996]. Also, rather than displaying a “pervasive lack of responsiveness to others” [APA, 1980], it is now understood that people with autism display a pattern of strengths and weaknesses in the acquisition of social and communication skills, which changes with development [Mundy and Sigman, 1989a].

In preverbal children, the communication disturbance of autism is exemplified by a robust failure to adequately develop joint attention skills. These skills involve the tendency to use eye contact, affect, and gestures for the singularly social purpose of sharing experiences with others. Prototypical of joint attention behavior is the act of pointing or showing to share one’s pleasure in a toy. Alternatively, less impaired is the use of eye contact and gestures to regulate the behavior of others is more instrumental. These behaviors include requesting aid in obtaining objects, or even displaying attachment-reunion behaviors after a caregiver separation [Curcio, 1978; Loveland and Landry, 1986; Mundy et al., 1986; Sigman and Mundy, 1989; Wetherby and Prutting, 1984].
In children with functional speech skills, the processes that enable adequate phonological, syntactic, and semantic usage of language may only be mildly impaired [Volden and Lord, 1991]. Alternatively, a disturbance of the pragmatics of language usage is a prominent feature of autism [Eales, 1993; Happe, 1993; Surian et al., 1996; Tager-Flusberg, 1993]. The pragmatics of language refers to a broad array of skills involved in prosody, appropriate turn-taking, politeness, and topic maintenance in conversation. Pragmatics also involves the critical ability to signal and interpret unspoken premises, as with figures of speech (e.g., metaphor), or with nonverbal behaviors or by relying on the context of a communicative interaction.

This brief synopsis indicates that a comprehensive model of autism will ultimately need to address both linguistic pragmatic difficulties and the very early onset of preverbal social-communication deficits that are characteristic of the syndrome. In the course of debate necessary to develop this model, fundamental issues must be raised and examined. These issues concern not only autism but also the nature of the ontogeny of the human capacity for complex social-communication. Two of these issues will be highlighted in this review.

The first issue concerns the nature of the neurological disturbance that leads to the social-communication disturbance of autism. The current status of the neuroscience of social behavior is not sufficiently well articulated to allow anything but an oversimplified modeling of this neurological impairment. Nevertheless, animal and human lesion studies suggest that neurological functions associated primarily with frontal cortical and medial temporal systems are likely foci for the types of social-communication disturbance displayed by individuals with autism [e.g., Damasio and Maurer, 1978; Bachevalier, 1994; Dawson, 1996; Pennington and Osonoff, 1996; Minshew, 1996; Mundy, 1995]. Of course, there is considerable debate as to whether functional disturbances of these systems are primary in the etiology of autism, or secondary upstream effects of impaired functions in more caudal brain systems [Courchesne et al., 1994; Damasio and Maurer, 1978].

The second issue concerns the functional, or psychological, nature of the neurological disturbance in autism. Three prominent models propose to account for the social-communication disturbance of autism, while sharing a common focus on frontal processes. These include the Theory of Mind model, the Executive Function model, and the Social Orientation model. The Theory of Mind (ToM) model suggests that one aspect of the frontal process may be dedicated to a facility for social cognition necessary to estimating the psychological status of others, such as their beliefs. According to this model, the social-communication disturbance of autism may be understood in terms of an impairment in the functions of this system [Baron-Cohen et al., 1994].

The Executive Function model suggests that social-cognitive and social-communication impairment in autism does not derive from a system dedicated to social cognition. Rather, the social-communication disturbance of autism is viewed as one manifestation of an impairment in frontally mediated executive cognitive structures. The fundamental function of these is to select appropriate goal-directed actions from an array of competing action potentials [Pennington and Ozonoff, 1996].

A critical assumption of a third model is that the earliest forms of social-communication impairment of autism may not be completely understood in terms of either of the cognitive mechanisms espoused in the Theory of Mind or Executive Function models. The Social Orienting model suggests that, prior to the emergence of cognition as the primary regulator of behavior, frontally mediated neuroaffective motivation systems serve to prioritize social information processing in human development. A deficit in these systems is thought to contribute to initial, as well as subsequent, social and cognitive disturbances in autism [e.g., Dawson and Lewy, 1989; Fotheringham, 1991; Hobson, 1993; Mundy, 1995].

Each model provides a different and valuable perspective on the nature of autism, and on social-communication skill acquisition more generally. In the remainder of this essay we will provide an overview of the similarities and differences...
among these models. We also note that a collective view of these models gives rise to a consideration of how related neurological processes may serve different functions at different stages in the development of autism.

Theory of Mind and Social-Communication Disturbance in Autism

Consider a possible newspaper headline, “IRAQI HEAD SEeks ARMS” [Pinker, 1994]. Language development alone does not allow for the correct interpretation of this statement. Rather, some cognitive, pragmatic facility that goes beyond neural systems specific to the grammar of language development is assumed to play a role in correctly conveying and recognizing the ambiguous communicative intentions that are frequently embedded in language [Pinker, 1994].

Children with autism display a poor facility for the pragmatics of language [Frith, 1989]. To understand this feature of autism, some have turned to theory and research on the nature of social cognition. The capacity for social cognition may be an important, if not defining, feature of primate, and especially human, neurobehavioral evolution [Cosmides, 1989; Whiten and Byrne, 1988]. In keeping with this evolutionary psychological view, a modular perspective on cognition has been adapted to suggest that the capacity to understand mental states in others follows its own proprietary developmental course, with brain mechanisms responsible for apprehending mental states separate from brain mechanisms related to non-social cognition [e.g., Leslie and Thaiss, 1992; Baron-Cohen, 1995]. This dedicated socioneurocognitive mechanism has various descriptions [Baron-Cohen, 1995], but will be referred to here as the Theory of Mind (ToM) module [Leslie, 1987].

Hypothetically, the ToM module employs a special type of cognition called metarepresentation. Metarepresentational ability allows one to mentally depict the psychological status of others, such as their thoughts and beliefs. It is called metarepresentation because it involves the capacity to cognitively represent the mental representations of others. Metarepresentation also involves a critical “decoupling” mechanism that enables the child to keep cognitive representations organized so that their own thoughts and feelings can be easily distinguished from representations of others’ thoughts and feelings [see Leslie, 1987, 1993 for details]. According to the ToM model, a disturbance in this type of representational thought process gives rise to the social and pragmatic deficits of people with autism [Baron-Cohen, 1995; Frith, 1989; Leslie, 1987; Tager-Flusberg, 1993]. The logic here is that, if children with autism have difficulty thinking about others’ psychological status, correctly identifying the communicative intent of the author of the headline “Iraqi Head Seeks Arms” would be unlikely. A host of other types of pragmatic errors would also be evident, such as difficulties with understanding figures of speech (e.g., irony), or difficulty in gauging the timing constraints of discourse, or difficulty in gauging the informational needs of others, as well as conventions of topic maintenance. According to the ToM, most if not all of the social deficits of autism may be understood in terms of this type of social-cognitive disturbance [Baron-Cohen, 1995].

Numerous experimental studies support the hypothesis that children with autism have difficulty on theory of mind measures [Baron-Cohen, 1995]. In the prototypical “false-belief paradigm,” a child is asked to watch an agent (“Sally”) hide an object in one of two hiding places (Place 1 vs. Place 2). Sally then leaves the room and another agent (“Anne”) moves the object from Place 1 to Place 2. When Sally returns, the child must answer the question, “Where will Sally look for the object?” To answer this question correctly the child must disregard his/her own knowledge of where the object really is (Place 2) and think about where Sally thinks the object is (the putative metarepresentational component of this task has been italicized). Children typically
develop the ability to solve this type of problem between 3–5 years of age. However, people with autism manifest robust difficulty with false-belief and related ToM tasks relative to language- and IQ-matched controls. Theoretically, this is because they lack the requisite metarepresentational cognitive functions required to think about others’ thoughts [Leslie, 1987].

The argument for the modularity, or the dedicated nature of this type of social-cognitive process, has been made on numerous grounds [Leslie, 1993]. The most important observation may be that representational deficits are more likely to be manifest by children with autism on social-cognitive tasks, rather than analogous nonsocial-cognitive tasks [e.g., Leekam and Perner, 1991; Leslie and Thaiss, 1992; Scott and Baron-Cohen, 1996]. Two other findings are of critical importance. First, ToM-related ability has been directly linked to the degree to which pragmatic skill deficits are displayed among people with autism [Happe, 1993; Surian et al., 1996]. Second, specific neural subsystems may be involved in thinking about the thoughts, beliefs, and feelings of others [Baron-Cohen et al., 1994; Fletcher et al., 1995].

The first of these studies utilized single photon emission computerized tomography (SPECT) data and suggested that the right orbital frontal region may be involved in the processing of mental state turns [Baron-Cohen et al., 1994]. The second study presented actual ToM tasks and employed functional neuroimaging during task engagement [Fletcher et al., 1995]. In this study, six male volunteers were presented with ToM stories in which they were asked to think of the answer to questions about the internal motivations, beliefs, or thoughts of the protagonists. They were also presented with stories that required them to think of the answer to questions about causality of physical events and to answer questions about sequences of unrelated sentences. The results indicated that thinking about the answers to the ToM vignettes involved cortical activity in the left medial frontal gyrus (Brodmann’s area 8) to a significantly greater extent than did thinking about the answers to the physical stories or unrelated sentence questions [Fletcher et al., 1995]. Interestingly, the authors observed that this area had been linked in comparative and human studies to facility with conditional learning [Petrides, 1990]. The potential importance of the latter observation will be made clear in the discussion of the Executive Function model.

The ToM model is seminal to the current understanding of language and communication disturbance in autism. It has been directly linked to the significant phenomenon of pragmatic communication disturbance in this syndrome. Moreover, preliminary data on the brain mechanisms that may be specific to ToM functions has also been presented. However, several problems arise with this model. Recent research suggests that ToM tasks deficits may not be as specific to autism as once thought [Peterson and Siegal, 1995; Yirmiya and Shulman, 1996]. It is also debatable whether or not the ToM model can explain deficits in the early forms of social-communication disturbance displayed by children with autism [Leslie and Happe, 1989; Mundy and Sigman, 1989b]. Furthermore, it is not clear if the ToM model can explain a class of phenomena referred to as executive function deficits in autism [Bishop, 1993; Pennington and Ozonoff, 1996]. Alternatively, executive function deficits may contribute to an explanation of difficulties in ToM functions, as well as pragmatic communication disturbance, in people with autism [Hughes and Russell, 1993; Ozonoff, 1995; Pennington and Ozonoff, 1996].

**Executive Functions and Social-Communication Disturbance**

A critical difference between the Executive Function model and the ToM model is that the fundamental disturbance of autism is not considered to be specific to a neurologically dedicated system for social cognition. Rather, a more general cognitive
disturbance in so-called executive functions is viewed as central to autism. Executive functions are thought to involve a system of frontal neurological processes that are behaviorally manifest in the related capacities to: 1) initiate behaviors while inhibiting competing responses which may interfere with effective problem solving; 2) regulate attention in order to filter distractions during problem solving and shift attention across relevant stimulus components; and 3) upload and manipulate mental representations to bring them to bear in a task-effective fashion [Pennington and Ozonoff, 1996; Ozonoff, 1995]. Central to the executive functions is the notion of appropriate action selection in the face of competing, but context-inappropriate responses. Action selection is thought to be dependent on the integration of behavioral constraint and activation parameters that flow from memory, perception, and affective or motivation systems [Pennington and Ozonoff, 1996].

Studies indicate that people with autism display difficulties with appropriate action selection in the face of competing response potentials [Hughes and Russell, 1993; see Pennington and Ozonoff, 1996, for review]. Moreover, several researchers have argued that, instead of a disturbance in a ToM module, autistic difficulties on false belief and related social-cognitive tasks may be explained in terms of this type of more general executive function difficulty [Pennington and Ozonoff, 1996; Hughes and Russell, 1993; Frye et al., 1995].

For example, recall that to solve the Sally–Anne false–belief task the child must disregard their own knowledge of where the object really is (Place 2), and think about where Sally thinks the object is (notice that we have now italicized two operations in this task sequence, the first associated with inhibiting a competing response, and the second associated with metarepresentation). The Executive Function model suggests that children with autism have difficulty with the former, and fail false belief and related social-cognitive tasks because of this difficulty. Similarly, they may be unable to correctly interpret the statement “Iraqi Head Seeks Arms” because they cannot disregard the false literal meaning in favor of the correct nonliteral inference. A key diagnostic feature of autism that involves the singular pursuit of a limited, idiosyncratic set of interests may also be explained in terms of the executive function disturbance of this model [Hughes and Russell, 1993; Ozonoff, 1995].

A recent study has, in fact, suggested that the normal course of ToM development is associated with executive function development. Frye et al. [1995] demonstrated that, in normal 3–5-year-olds, performance on a nonsocial sorting task that measured the ability to select appropriate actions in the face of competing responses was significantly correlated with the development of ToM in the form of false-belief task performance. In addition to being consistent with the Executive Function model, this observation is intriguing for another reason. A link exists between the type of executive function isolated in the work of Frye et al. [1995] and the neurological concomitants of ToM performance. Recall that ToM Performance was linked to activity in left medial frontal circuits (Brodmann’s area 8; [Fletcher et al., 1995]). Fletcher et al. [1995] also noted that Petrides [1990] had connected activity in this subsystem to conditional associate learning. The primary task demand of this type of learning is the capacity to inhibit competing responses in order to efficiently solve a problem. Indeed, the task used by Petrides [1990] was very similar to the task employed by Frye et al. [1995]. Thus, the neurological linkage between frontal processes and ToM performance observed by Fletcher et al. [1995] may overlap with frontal correlates of the types of processes that are central to the Executive Function model.

The Executive Function model poses a reasonable alternative to the ToM model of autistic social-cognitive and social-communication pathology. This model, however, may have difficulty explaining the observation that children with autism manage nonsocial representational tasks better than analogous false-belief tasks.
[Leslie, 1993]. It may also have difficulty explaining why children with autism display even more basic social-cognitive difficulties, such as more difficulty using mental as opposed to physical state words [Baron-Cohen et al., 1994; Tager-Flusberg, 1993]. Furthermore, while executive function tasks may be correlated with ToM tasks, they do not explain all of the variability in the latter [Frye et al., 1995]. Also, some people with pervasive developmental disorders may display executive function disturbance, but not ToM disturbance [Ozonoff, 1995]. These observations suggest that an executive function disturbance and a ToM impairment may have partially independent paths of effects on the poor social-communication skills of autism. Thus, a combination of the Executive Function and ToM models provides a clearer picture of the social-communication disturbance of autism. It is unlikely, though, that even a combination of these compelling models may provide a complete explanation of the earliest forms of social-communication disturbance observed in autism.

**Social Orienting and Social-Communication Disturbance in Autism**

Recall that preverbal children with autism display a deficit in joint attention skills, but not in more instrumental social-communication behaviors, such as those involved in requesting or attachment. For example, children with autism will rarely use eye contact and gestures such as showing or pointing to share attention regarding an active wind-up toy. If the toy is moved out of reach, though, they will be as likely to use eye contact and pointing to elicit aid obtaining the object as will comparison children [e.g., Mundy et al., 1994].

This seemingly simple observation may be fundamental to an understanding of autism. Joint attention behaviors reflect the tendency of children to socially orient while engaged in observing an object or event in order to share their experience of the object or event with others [Mundy, 1995]. This capacity normally emerges between 6–12 months. Thus, observations of joint attention impairment in autism suggest that the pathological processes fundamental to this disorder may be manifestly active in the first year of life [Mundy and Sigman, 1989b]. Consistent with this notion, first birthday videotape data suggests that 12-month-old children with autism display evidence of a disturbance in joint attention and social orienting [Osterling and Dawson, 1994]. Measures of joint attention skills have also contributed to the very early identification of autism at 18 months to a sample of 16,000 children [Baron-Cohen et al., 1996].

Several other observations attest to the importance of joint attention deficits. These deficits are observed in young children regardless of IQ, and are related to parents’ reports of symptom intensity [Mundy et al., 1994]. Individual differences in joint attention skill development also appear to be singularly powerful in predicting language development among these children [Mundy et al., 1990; Sigman and Ruskin, 1997]. Indeed, joint attention skill development is considered to be integral to language, social, and cognitive development among all children [Tomasello, 1995], not just children with autism. For example, joint attention skills have been observed to predict language development from as early as 6 months of age [Morales et al., 1997], and to predict individual differences in childhood IQ from 13 months of age [Ulvund and Smith, 1996].

Attempts have been made to explain joint attention disturbance in terms of ToM dysfunction [Baron-Cohen, 1995; Leslie and Happe, 1989], or cognitive executive functions [McEvoy et al., 1993]. However, with evidence of the emergence of this domain in the first year of life, it may be less than parsimonious to explain impairments in such an early emerging facet of behavior solely in terms of later developing, complex
cognitive functions. Alternatively, several researchers have suggested that joint attention deficits may be understood as part of a fundamental social-approach, or social-orienting impairment [Dawson and Lewy, 1989; Hobson, 1993; Fotheringham, 1991; Mundy, 1995]. These models vary in the hypothesized mechanisms of impairment. Yet they agree that a primary social-orienting impairment may have enormous ramifications for the subsequent development of social, cognitive, and even neurological disturbance in autism. To illustrate this point, consider the basic assumptions of one of these social-orienting models [Mundy, 1995; Mundy and Crowson, in press; Mundy et al., 1993].

Like the ToM and Executive Function models, it is assumed that the social communication pathology of autism, including joint attention disturbance, derives in part from neurological impairment that involves frontal cortical processes. Three studies, using EEG [Card et al., 1997], PET imaging [Caplan et al., 1993], and behavioral measures [McEvoy et al., 1993] have directly linked joint attention development to frontal processes. It is also assumed, though, that frontal systems may play different functional roles at different points in development. In particular, the types of functions proposed by the ToM and Executive Function models are thought to require a degree of cognitive maturation and information acquisition that is not likely to be available in the first 12 to 18 months of life.

Alternatively, in the terminology of the Executive Function model [Pennington and Rogers, 1996], early in life, frontal action selection functions may be more dependent on behavioral constraint and activation parameters that flow from affective and motivational parameters, as opposed to cognitive-memory parameters. That is, prior to the effects of more cognitive constraint parameters, frontally mediated action selection is constrained by a motivational executive system that serves to prioritize perceptual inputs that are most significant to the development of the infant [Derryberry and Reed, 1996; Mundy, 1995; Tucker, 1992]. In particular, this system prioritizes social perceptual input and social information processing, via social orienting, from early on in the development of the child. One mechanism of this prioritization may involve the attribution of positive valence to the perception of social information, possibly by way of temporal/midbrain systems involving the amygdala [LeDoux, 1989], as well as brainstem nuclei (e.g. nucleus ambiguous [Derryberry and Reed, 1996]).

Several studies provide evidence for a basic social-orienting disturbance in autism. Klin [1991] has reported that the typical preference for speech and speech-like sounds, which is usually displayed by infants in the first months of life, was not present in any of the children with autism he observed. It was, however, present in all of the developmentally delayed matched controls observed in this study.

In an even more intriguing study, Dawson et al. [1995a] examined the degree to which children with autism, Down syndrome, or normal development oriented (displayed a head turn) toward social stimuli (clapping hands or calling the child’s name) and two nonsocial stimuli (playing a musical jack-in-the-box or shaking a rattle). The results indicated that the children with autism more often failed to orient to both types of stimuli. Their failure to orient to social stimuli, however, was significantly more extreme than their impaired orienting to nonsocial stimuli. Furthermore, individual differences in difficulty with social orienting, but not object-orienting, were significantly related to a measure of joint attention among the children with autism. A disturbance in social orienting among children with autism has also been observed in first birthday videotape data [Osterling and Dawson, 1994]. Finally, individual differences in social orienting have long-term stability and predict the degree to which children with autism process the nonverbal affective information presented by others [Dissanayake et al., 1996]. In addition to providing some support for a social-orienting impairment model, these observations may not be easily ex-
plained in terms of current ToM or Executive Function models [see Baron-Cohen, 1995 for an alternative view].

What might the ramifications of an early social-orienting disturbance be? Another assumption of the model is that experience drives a substantial portion of postnatal brain development. This occurs through the competitive enhancement of active neural connections and the culling of less active connections [Huttenlocher, 1995]. We also assume that, to some degree, the human neural behavioral system is self-organizing. One component of this self-organizing system is the aforementioned prioritization of social information processing. This drives the early developing neuroarchitecture along paths that normally emphasize social-cognitive development [Cosmides, 1989]. In the child with autism, however, the lack of this self-organizing feature leads to increasingly deviant development of neurobehavioral systems over time. Thus, autism may be characterized by primary neurobiological deficits, which lead to less than optimal behavioral proclivities in the first months of life (e.g., a lack of early social orienting). These, in turn, lead to a secondary neurological disturbance via a negative feedback system. In this system, the lack of social orienting and social information processing contributes to a dynamic alteration of the typical, experience-driven mechanisms of neural activation and culling. Hence, the lack of early social orienting and processing contributes to subsequent disruptions of neurobehavioral development [Mundy and Crowson, in press].

In this model, joint attention skill measures are viewed as a sensitive index of social-orienting disturbance. Hence, one aspect of autistic social-communication pathology (i.e., joint attention deficits) may be traced directly to this hypothesized social-orienting disturbance [Mundy, 1995]. It is not clear, though, whether all subsequent executive function, social-cognitive, and social-communication deficits flow directly from this early source. Nevertheless, some interesting hypotheses follow from this model.

Frontally mediated social-orienting processes may contribute to the observation that autistic children vary from aloof to active but odd in their social communication style. Dawson et al. [1995b] have reported that variability in frontal activity (EEG power) was related to these social style differences among people with autism. Another observation that the social-orienting disturbance in autism is most severe in the preschool years may be linked to the observation of a transient component of frontal metabolic activity disturbance, which improves between the ages of 6–7 in children with autism [Zilbovicius et al., 1995]. Hence, there may be an early critical period for the process involved in social orienting impairment in autism. This possibility may be important in considering recent reports of positive early intervention effects with autism [Mundy and Crowson, in press].

It is also likely that an early disturbance of social orienting could influence executive function and ToM development. With regard to the former, perhaps the frontal executive motivation system that prioritizes orienting to social stimuli primes the development of the more general executive capacity to engage action selection in the face of competing response potentials. Presumably, early in life infants are frequently confronted with a choice between attending to competing social exteroceptive stimuli, nonsocial exteroceptive, and/or proprioceptive stimuli. It may be that the activation of an intrinsically motivated social-orienting system yields, as an important by-product, early practice (hence neuro-organization) associated with selecting an action (social orienting) in the face of exteroceptive and proprioceptive stimuli that compete for attention. Without such practice contributing to adequate neural self-organization, the later-emerging cognitive executive functions of the frontal system may not develop normally in children with autism [see Hughes and Russell, 1993, for an alternative hypothesis].

Similarly, it may be that a relative failure to process social information early on gives rise to a cognitive system that has insufficient information and experience to
develop facility with ToM functions and social cognition [Mundy et al., 1993; Mundy, 1995]. Indeed, recent research with sensory-impaired children strongly suggests that sufficient social input is required for typical ToM development as measured on false-belief tasks [Peterson and Siegal, 1995]. Moreover, a disturbance of joint attention development, secondary to a social-orienting impairment, may deprive children with autism of early, critical social interactive experiences. Theoretically, the negative feedback of such a loss during a critical period of cognitive development may distort typical symbolic and social cognitive development [Mundy et al., 1993], as well as contribute to the language delays symptomatic of this syndrome [Mundy et al., 1990; Sigman and Ruskin, 1997]. Finally, we have hypothesized that attenuated social orienting may be associated with a reciprocal augmentation of nonsocial information processing in autism. Such a reciprocal function may assist in explaining the relative success of people with autism in nonsocial problem-solving situations [Mundy, 1995].

Conclusions

Researchers in psychopathology typically agree that models that identify a single cause for abnormal behavior are likely to be incomplete [Cicchetti, 1993]. This is not to say that single-factor models are not valuable. The value of single-factor models, however, may only be truly realized when they are synthesized to yield a more divergent, rather than convergent, perspective on the processes involved in the ontogeny of pathology. This is clearly the case with respect to the state of research on autism. It is abundantly clear that higher-order cognitive dysfunctions play a critical role in autism [Minshew, 1996]. In particular, at this time both an executive function disturbance and a form of higher-order representational impairment appear to be linked to the social, pragmatic disturbance of communication that is characteristic of the older child with autism [Leslie, 1993; Ozonoff, 1995]. Furthermore, these impairments may be linked to functions of the frontal systems [Baron-Cohen et al., 1994; Fletcher et al., 1995]. A complete understanding of the nature of this linkage, however, may not be clear unless the earliest manifest forms of social-communication disturbance in autism are considered. These joint attention and social-orienting difficulties may also be related to an impairment involving frontal systems. It is unlikely, though, that these ontogenetically primary impairments may be explained simply by way of recourse to the constructs used to explain later-emerging cognitive deficits in autism. Instead, these early-emerging deficits challenge researchers to adopt a more developmental and dynamic systems approach to understanding the nature of autism. Such a perspective reminds us that the behavioral function of a neurological subsystem may change over development. Moreover, a sufficiently powerful disturbance of early behavior may in and of itself lead to subsequent disturbance in neurological and neurobehavoiar development. Thus, a consideration of the dynamic interplay between initial biological insult and subsequent transactions with the environment may be crucial to an understanding of autism. It may also be that an understanding of autism will play a critical role in acquiring a better understanding of the complex dance that occurs between neural development and environmental constraint in the ontogeny of the quintessential human capacity for social communication and cognition.

References

Reading 6  ■  On the Nature of Communication in Autism


