Invited Paper

Review

The Primacy of Social and Language Deficits in Autism*

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Current theories concerning psychological deficits in children with autism were reviewed. The author argues that, to the extent that there is a single area of primary impairment, it must lie in the domain of social attachment, including the impetus to communicate. Some recent data support this position. The findings from research with animals and humans suggest that the amygdala might be crucially implicated in the social deficits in autism. Human communication represents the merging of two evolutionary lines of development, that of mammalian communication systems and that of the primate capacity for internal representation. Such a view has direct implications for the nature of the communication deficit in autism.

Key Words: autism, neuropsychology, social attachment, communication, amygdala

There are many current neuropsychological theories that attempt to identify a primary psychological deficit in autism and to relate it to a particular brain structure or system. Such neuropsychological theories are not usually etiological theories. A particular neuropsychological deficit could be consistent with a variety of causal agents. Current etiological theories and research focus heavily on genetics. For those who are interested in the genetics of autism, the autumn 1998 issue of the *Journal of Autism and Developmental Disorders* is devoted to this topic and presents several reviews of the latest genetic findings. In addition to genetic findings, there is also a small but consistent effect of season of birth (Stevens & Fein, in press), with a peak of birthdays in March, which is hard to reconcile with a purely genetic causation and raises the possibility of viral or other environmental insult in the etiology of autism. Gillberg and Coleman (1992) and other researchers have found evidence for viral insults as

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D. Fein

TABLE 1 Leading neuropsychological theories of autism

Theory	Theorists	Brain Areas	Assessment	Treatment
Language	Rutter, Prior	Left hemisphere	Focus on assessing language	Language therapy
Attention/arousal	Courchesne Dawson, Kinsbourne	Cerebellum, parietal cortex Right hemisphere Brainstem-cortical input	Baseline arousal, sustained attention, switching attention	Decrease arousing input, give time for attention switching
Complex information processing	Minshew and Goldstein	Association cortex	Rote tasks should be spared, complex tasks impaired	Build complex skills slowly from simple input
Executive functions	Ozonoff, Pennington, and Rogers	Prefrontal cortex	Executive tasks (e.g., Wisconsin Card Sorting)	External structure needed
Social emotions	Fein & Waterhouse, Hobson	Limbic system, esp. amygdala	Social cognitive tasks, behavioral observation	Make interaction positive, use external reinforcers
Theory of mind	Baron-Cohen, Leslie, Frith, Brothers, Tager-Flusberg	Prefrontal or temporal lobe/amygdala	Assess theory of mind	Teach theory of mind

possibly causing some cases of autism.

I would like to briefly review the major current theories to provide a framework. Then I would like to focus on the language and social/emotional ideas in more detail, and present some findings and ideas that appear to support the social/emotional point of view. I will end, however, with some clinical observations that have led me to reconsider the language point of view about autism.

Some of the key current theories are listed in Table 1. The implications for assessment and treatment are my own; most of the theorists do not draw these out explicitly, and I am sure some might disagree with my conclusions about how their ideas would lead to specific assessments or treatments.

1. Language

One of the oldest neuropsychological theories of autism, dating back to the 1960's and 1970's, holds that autism is fundamentally a disorder of language, and that the additional symptoms of autism, including the social impairments, are secondary to the autistic child's inability to communicate (Prior, 1979; Rutter, 1968). This would seem to imply that the most important areas to focus on in assessment

The Primary of Social and Language Deficits in Autism

and intervention are specific language functions such as vocabulary and grammar. I will return to this view later.

2. Attention and Arousal

Three theorists present arguments related to each other in the realm of attention and arousal: Courchesne (Courchesne et al., 1994) argues that autistic individuals are deficient in making rapid and accurate shifts of attention, which in turn undermines social and cognitive development. Courchesne relates this deficient shift of attention in particular to the lack of development of joint attention behavior. He argues that if children with autism cannot rapidly shift their attention back and forth between a parent and an interesting object, then they will not be able to develop to joint attention behaviors such as pointing to objects of interest or bringing such objects to show the parent. This theory has received widespread attention, and is the subject of a recent study by Pascualvaca et al, from Mirsky's laboratory at the National Institutes of Health (Pascualvaca et al., 1998). In their article, they investigated the ability of children with autism to shift attention between stimulus attributes; they found that when the information feedback was provided by an adult, there was an attention-switching deficiency, but that when the feedback was provided by a computer, there was no deficiency compared to mental-age-matched controls. This suggests that the motivation or incentive conditions may be responsible for at least some of the reported attention problems in children with autism. Garretson et al. found something very similar (Garretson, Fein, & Waterhouse, 1990). They tested autistic children's ability to sustain their attention on a long signal detection task. To their surprise, they found that the main variable of importance was the reinforcer: when social reinforcement was used, the autistic children's attention rapidly waned, but when tangible reinforcers such as candy were used, their sustained attention performance was equal to that of mental-age-matched normal controls.

The second attention theory is that of Kinsbourne (1987); he argues that the higher cognitive deficiencies in autism are secondary to impairments of ascending activation from brainstem to cortex, and that some autistic symptoms can be viewed as secondary to overarousal or to unstable arousal. In this view, the child tries to contain uncomfortable arousal by restricting environmental input, especially social input, which is highly arousing. Similarly, Dawson (1991) argues that right hemisphere dysfunction leads to a low threshold for detecting novelty and therefore a low threshold for overarousal. In other words, the children judge even mildly novel stimuli as quite novel and arousing. Social stimuli, being inherently unpredictable and arousing, are therefore found aversive by these children.

All of these attention/arousal theories hold that the social, language, and other cognitive deficits of autism are secondary to primary deficits in modulating arousal, responding to novelty, or shifting attention. These aspects of attention would therefore be the focus of neuropsychological assessment, and the implications for treatment might be that one should accommodate to these attention problems by giving the children lots of time to switch their attention and to decrease arousing input.

D. Fein

3. Deficits in Complex Problem Solving

Another approach is taken by Minshew and Goldstein (Minshew, Goldstein, and Siegel, 1997). They suggest that, because of dysfunction in association cortex but not primary sensory and motor cortex, autistic individuals can perform simple rote cognitive tasks in all domains, but are seriously impaired in the performance of complex or abstract tasks. One would therefore tend to expect deficiencies on all assessment tasks that have demands for processing complex or abstract information, whether they are language-based or not. In teaching the children, this theory would stress the need for teaching very concrete and rote tasks, and trying build abstract or complex skills slowly by assembling sets of simple or rote skills.

4. Executive Dysfunction

Theorists such as Ozonoff, Pennington, and Rogers (1991) suggest that the primary impairment in autism is in the domain of executive functioning, secondary to dysfunction in prefrontal cortex. Such deficits would include problems with organization, planning, behavioral inhibition, self-monitoring and self-correction, and working memory. They believe that deficits in such domains as understanding the mental events of others and social functioning can be attributed to executive functioning demands in the tasks used. Autistic children's deficits should be most pronounced on assessment tools such as the Wisconsin Card Sorting Task, on which the individual has to sort cards by one of several properties of sample cards (such as color, shape, and number of objects), and then flexibly switch the sorting principle when the examiner starts to say that the individual is wrong. In educating the children, one would not expect them to be able to self-organize, effectively plan, or inhibit impulsive responding, and the teacher would have to provide the structure that the children's frontal lobes are not providing.

5. Social/Emotional Dysfunction and Theory of Mind

Other theorists postulate that the primary deficit in autism is in the domain of social behavior, attachment, and processing of social information, and that at least some of the remaining symptoms flow from this deficit. Some of the arguments for this point of view are (1) the earliest symptoms of autism are mostly in the realm of reduced social attention, reciprocity, and responsiveness, (2) most of the cognitive deficits proposed such as language, theory of mind, or executive function deficits occur too late in development to explain early abnormalities such as those that sometimes occur in the first year of life, (3) when cognitive tasks involve social vs. nonsocial material, the autistic children have relative deficiencies on the social tasks, (4) some of the autistic deficits disappear, at least temporarily, when strong incentives are given, suggesting a motivational component to their deficit, (5) there are many anecdotal accounts of autistic children who are mute, but utter a few words or even a sentence on rare occasions when the child perceives an emergency situation, and (6) cognitive deficits such as deficits in executive functioning are usually found in only a subset of autistic children, and are also found in other clinical groups who do not

The Primary of Social and Language Deficits in Autism

show the autistic social impairment.

This approach (Fein, Pennington, Markowitz, Braverman and Waterhouse, 1986; Waterhouse, Fein, and Modahl, 1996; Hobson and Lee, 1998; Hobson, 1991) regards the primary deficit in autism as emotional and motivational, that is, that the natural immediate appeal of social stimuli is reduced or absent. The autistic child's deficit should appear most clearly on social cognitive tasks and in behavioral observations, and the implications for treatment are that one may need to rely on external reinforcers to promote good behavior and learning, and that one should try to make social interaction as reinforcing as possible.

A variant of this idea is that autistic deficits are primarily social, but that they are really in the cognitive or perceptual realm, that is, that autistic children cannot decode facial expressions of emotion, perceive others' direction of gaze, or understand others' mental states (e.g., Fein, Lucci, Braverman, & Waterhouse, 1992). These theories are represented by Brothers (1995) Baron-Cohen (Baron-Cohen, Leslie, & Frith, 1985; Baron-Cohen, Baldwin, & Crowson, 1997), and Tager-Flusberg (Tager-Flusberg & Sullivan, 1994a, 1994b). Some of these theorists have begun to develop tools for assessing and teaching theory-of-mind skills.

Now I would like to examine the language theory more closely. In 1984, I was honored to have my neuropsychology professor and mentor, Dr. Edith Kaplan, collaborate with me on a paper in which we examined the theory that autism is a disorder of language, and more specifically, a disorder of the left cerebral hemisphere. We presented several arguments against this idea (Fein, Humes, Kaplan, Lucci, & Waterhouse, 1984).

First, leaving language aside for the moment, if one examines the drawings of autistic children, one sometimes sees pronounced segmentation or fragmentation of designs and even human figures. To the extent that one can draw an analogy between children with developmental disorders and adults with acquired brain lesions, these drawings more closely resemble those of adults with right hemisphere lesions rather than left hemisphere lesions.

Second, researchers have failed to show a consistent tendency to right-sided sensory or motor deficits, as might be expected with consistent left hemisphere abnormality.

Third, autistic individuals show an elevated incidence of left-handedness, which has been cited as evidence for left-hemisphere impairment. However, most clinical groups such as children with learning disorders show elevated rates of left-handedness compared to normal populations, similar to the rates in autism, and Satz (1973) has shown how this may be a statistical artifact of the low base rates of left-handedness in the normal population and not reflect differential involvement in the left hemisphere.

Turning to the language issues, the fourth point is that not all individuals with autism have language impairment relative to visuo-spatial impairment. Some have the opposite pattern, in which perceptual and motor impairments are seen but language is strong.

D. Fein

Fifth, children with mental retardation or specific language handicaps may have language that is as impaired as that of some autistic children but do not show the social deficits. If the language impairments were causing the social deficits, one would not expect to see this pattern. Sixth, within language functions, syntax and phonology, which are probably most dependent of all language functions on intact left hemisphere functioning, tend to be relatively spared.

Finally, it is hard to imagine how impairments in language could account for abnormalities in infancy, which appear in some autistic children, or for such features of autism as abnormal orienting and startle responses, abnormal sleep, and abnormal responses to pain.

If autism is not primarily a disorder of language, what is the primary area of difficulty? Of course there is not necessarily one psychological domain that represents the primary area of difficulty for all autistic children. However, it perhaps does make sense to ask if there is one domain that is invariably disturbed early in the course of the disorder. If one does ask that question, it seems the answer has to be the domain of social attachment, including the drive to communicate with others.

The earliest abnormal behaviors, seen in autistic children in the first year or two of life, include poor or absent eye contact, reduced understanding of others' emotions, and less expression of positive emotions such as laughing and smiling to others, poor response to their name, poor social interaction such as in turn-taking games and listening to others, delayed or absent joint attention (such as pointing at objects of interest, showing objects to caregivers, looking at what parents are looking at), reduced or absent social referencing (that is, looking at the parent's face to judge their reaction when faced with an uncertain stimulus), reduced social smile, reduced or absent imitation, reduced use of gesture to communicate, and poor attention to others' language (Cox, Charman, Baron-Cohen, Drew, Klein, Baird, Swettenham, & Wheelwright, 1999). Often when I question parents about gesture, they will report that their child does communicate through gesture, but when questioned further, it appears that the gesture is not truly symbolic but what Prizant calls "re-enactment" gestures—that is, not really intended to communicate symbolically, but intended to bring about the desired result by re-enacting a successful experience. For example, instead of nodding or shaking the head to indicate "yes" or "no", the child may push the object away or reach for it. Instead of pointing to a desired object, the child leads the parent by the hand to that object in order to obtain it.

It can be seen that most or all of these early symptoms pertain to emotional relatedness to others, including the drive to communicate. One recent finding concerning oxytocin (OT) in autism lends some support to this notion. OT is a hormone and a neurotransmitter that has been implicated in a range of social behaviors. It is present in limbic areas of the brain (see review in Waterhouse, Fein, & Modahl, 1996). In our autism study (Modahl, Green, Fein, Morris, Waterhouse, Feinstein, & Levin, 1998), we examined OT levels in plasma from 30 children with autism and 30 age-matched controls. The autistic group showed significantly lower average levels of OT, although the distributions of OT in the two groups overlapped.

The Primary of Social and Language Deficits in Autism

For the children in the control group, OT increased with age and was higher in the children with more advanced social skills. For the autistic children, OT was unrelated to age and was associated with lower social functioning.

Furthermore, a more recent result, not yet published, indicates a possible mechanism for this finding. Blood samples from the same children were analyzed for OT-X, the precursor of OT, and it was found that while OT was significantly lower in the autistic children, OT-X was significantly higher. While OT was correlated with age for the control-group children but not the autistic children, OT-X was correlated with age for the autistic children, but not for the other children. Therefore, it is possible that OT-X is normally produced in children with autism, but that it is not normally cleaved into OT.

One of the areas in which OT receptors are found, and which has been directly linked to a variety of social behaviors, is the amygdala. Abnormalities in cell morphology have in fact been found in the amygdala in all cases studied so far by Bauman and Kemper (1985), who have done the most thorough postmortem examinations of the greatest number of autistic brains. There are several behavioral aspects of amygdala functioning, found in humans and animals, that relate directly to autistic symptomatology and that suggest that amygdala dysfunction may underlie some of the key behaviors in autism.

Changes in social behavior, including social isolation, have been demonstrated in animal and human lesion studies (Kling & Brothers, 1992). Bachevalier and Miskhkin (Bachevalier, 1991) have created the only reasonably convincing model of autistic social isolation by making bilateral medial temporal lesions in young monkeys, including lesions of the amygdala.

Processing of social information, in particular decoding facial expressions of emotion, has been shown to be deficient in adult neurological patients with amygdala lesions (Aggleton, 1992), and experimental work with autistic children demonstrates the same deficiency (Braverman, Fein, Lucci, & Waterhouse, 1989; Fein, Lucci, Braverman, & Waterhouse, 1992).

Detecting the direction of another's gaze, while not widely studied, has been shown to be dependent on amygdala functioning in humans (Kawashima, Sugiura, Kato, Nakamura, Hatano, Ito, Fukuda, Kojima, & Nakamura, 1999), and ignoring the direction of another's gaze is one of the earliest signs of autism.

Amygdala neurons are selectively responsive to novelty and participate in the orienting response (Halgren, 1992), which is abnormal in many autistic children (Harris, Courchesne, Townsend, Carper, & Lord, 1999; Wainwright-Sharp & Bryson, 1993).

The amygdala also plays a role in the formation of associations between stimuli and reinforcement (Ono & Nishijo, 1992) and, perhaps with inhibitory input from the frontal cortex, may play a role in the reversal or extinction of such associations. LeDoux, Romanski and Xagoraris (1989) showed that when frontal lobe input to the amygdala was severed in rats, the rats could not unlearn or extinguish an emotional reaction to a stimulus, despite numerous extinction trials. One aspect of autistic

D. Fein

symptomatology that seems to relate to this is the occasional cementing of an association between a drive state and a particular stimulus, with no room for flexibility, sometimes for years at a time. For example, some autistic children will eat only foods of a particular visual appearance; Rimland (1964) gives the example of a child who ate only white sandwiches cut into squares, for several years. Another example perhaps is the clinical observation that many young autistic children show apparently normal attachment to their mothers, but make no emotional connection to anyone else until comparatively late in their development. They may seek their mother out, make eye contact with her, go to her for comfort when hurt or tired, and want to be hugged by her, but be oblivious to other people. One child who had a very early and intense attachment to his mother had a very involved and loving father, who told me that he knew exactly the first time his son recognized him: the child was four years old, the father had taken all the children to a swimming pool, which the child loved, and when the child climbed out of the pool in a pleasurable excited state, he looked directly at his father for the first time in his life.

Another similarity between the symptoms of autism and what is known about amygdala functioning relates to the developmental course of autism: As many as one-third or even more of autistic children show apparently normal development until some time in the second year of life, most typically 15-18 months, at which time they lose their language skills and social responsiveness (Klein, Tuchman, & Rapin, 2000; Rapin & Katzman, 1998). Lesion studies of nonhuman primates and memory studies of human babies suggest that the amygdala may be making a developmental leap in influencing behavior at around this time, so that the child may behaviorally grow into a preexisting lesion (Bachevalier, 1990). That is, the amygdala may be malformed or malfunctioning, but may not show much direct influence on behavior until the second year, at which time the abnormality becomes evident.

Poor memory for faces also seems strongly related to bilateral amygdala dysfunction. Patients with bilaterally impaired amygdalae are reported to show poor recognition memory for unfamiliar faces (Jacobson, 1986; Young, Aggleton, Hellawell, Johnson, Brooks, & Hanley, 1995; Young, Hellawell, Van De Wal, & Johnson, 1996). A recent study (Hauck, Fein, Maltby, Waterhouse, & Feinstein, 1998) demonstrated the same finding in autistic children. They were given four tasks: face and object matching, and face and object memory. The autistic children showed a small but nonsignificant difficulty in matching faces, and a highly significant difficulty with recognition memory for faces, consistent with the adult amygdala reports. Furthermore, only the face memory task showed strong correlations with independent measures of social development and relatedness. One interesting thing to note about these results is that the autistic children did not do badly on face memory, relative to object memory—they just did not do any better, while the normal children showed extraordinary memory for faces. We speculated that the autistic children performed the face memory task using the same strategies or systems as they used for the object memory task, while the normal children used brain systems specialized for face recognition. This speculation has received support in a recent fMRI study (Schultz,

The Primary of Social and Language Deficits in Autism

1998; Schultz, Romanski, & Tsatsanis, 2000).

Given that there is reason to connect the primary symptoms of autism to dysfunction of the amygdala and given that there is in fact postmortem evidence of malformation of limbic areas, including amygdala and anterior cingulate, but that there is no consistent or convincing evidence of malformation or damage to lateral cortical language areas, what can be speculated about the nature of the autistic communication deficit?

Evidence that bears on this question comes from communication studies in nonhuman primates and other mammals, and from communication deficits in humans with acquired lesions.

Monkeys and apes have a rich repertoire of species-specific vocal and gestural signals. Such signals serve to communicate the animal's motivational state and intentions, and also to communicate specific information about the environment. For example, vervet monkeys studied by Cheney and Seyfarth had distinctive calls for three kinds of environmental threat requiring different actions, such as a snake and an eagle. They also communicate information about specific emotional states, such as fear, aggression, and friendliness (Seyfarth & Cheney, 1980; Seyfarth, Cheney, & Marler, 1980). A phylogenetic neural model of primate communication was suggested by Jurgens and Ploog (Jurgens & Ploog, 1976; Ploog, 1979). In this hierarchical model, Level I represents the motor nuclei having direct control over output. Level II represents the midbrain and pontine central grey where the oldest reptilian species-specific vocalizations can be elicited. Level III represents the mammalian level, where vocalizations represent motivational states, and is comprised of the amygdala, and thalamic and hypothalamic nuclei. Level IV is the anterior cingulate and represents, in the nonhuman primate, the only known site for voluntary control of vocalization. Note that there is no lateral cortical site for primate vocalization corresponding to human cortical speech areas such as Broca's area or Wernicke's area.

The extent to which chimpanzees and other apes can master the attributes of true language in the laboratory is still a matter of debate, but their natural communications lack key features of language (Deacon, 1997; Pinker, 1994). In particular, their communications form a closed class; they do not learn new communications for new events, and they do not combine their communications into novel or complex ones. Nor can they apparently communicate about the past or future, but only about what is immediately present. On the other hand, apes show evidence of a sophisticated capacity for conceptual categorization, planning, and working memory; they have the capacity to perform high level cognitive analyses of stimulus input, form internal representations of such input, and mentally manipulate these representations (Russon, Bard, & Parker, 1996). For example, some nonhuman primates, especially chimps, seem able to recognize themselves in a mirror, attribute mental states to others (Povinelli, 1993), and are capable of deliberate deceit (such as deliberately looking for food in the wrong place when others are watching them) (Savage-Rumbaugh & McDonald, 1988). Some of them can understand photographs (Daven-

D. Fein

port & Rogers, 1971). Kojima & Goldman-Rakic (1984) have shown that monkeys have neurons in the dorsolateral frontal lobe that operate as working memory elements to preserve information during a delay period.

Thus, on the one hand, these primates have sophisticated internal representations of reality and can mentally manipulate these representations, and they have a repertoire of vocal and gestural signals with which to communicate information about themselves and the world. What they cannot do is make public the results of their cognitive analyses of the world around them; their communication is restricted to a predetermined set of codes for specific events. Unlike humans, they cannot communicate the plans, intentions, or observations that they may be making. At some point in human evolution, the primate communication system gained access to a phonological and syntactic code for the products of cognitive analysis. Some language evolution theorists hold that human language evolved slowly and progressively directly from primate vocalization (reviewed in Deacon, 1997; Pinker, 1994). Another group of theorists (Burling, 1993) suggest that human language is a direct descendant of primate cognition, while primate communication gave rise to human nonverbal communication, including emotional expressions of face, hands and body, and tone of voice and intonation patterns. In this view, nonverbal emotional communication and propositional language are quite separate; in fact, Burling, one of the major theorists in this school, suggests that tone of voice is an “invasion of language by something quite alien”. The nonverbal aspects of communication largely transmit emotional information, while the linguistic aspects (derived from primate higher cognitive abilities) largely transmit propositional information.

It is obvious, however, that these two systems work together seamlessly in the production of normal language, and we are not usually aware of these aspects of language as separate and distinct. In normal communication, the drive to initiate communication, the communication of emotional information, and the communication of propositional information are inseparably intertwined. However, given their possibly different evolutionary roots, they may rest on separable neural systems that can be dissociated by pathology.

Such dissociated conditions can be seen in adult humans with acquired lesions. A variety of human patients with bilateral lesions to the anterior cingulate and supplementary motor area demonstrate that in severe cases, there is complete mutism and lack of initiation of nonverbal communication. In milder or recovered cases, speech may be sparse and lack the normal melody of speech (Mesulam, 2000). On the other hand, anyone who has worked with patients with Broca’s aphasia has seen them struggling to find words to express what they wish to communicate, and occasionally emitting purely emotional utterances, such as curses. One patient seen by my professor, Dr. Kaplan, had this type of clinical picture. She asked him what “D-A-M-N” spelled. He struggled and struggled to produce the word, coming out with “D-D-” and finally gave up and exclaimed “Oh, damn!” The impaired lateral cortical language system could not produce the word as an answer to a simple spelling question, but the unimpaired emotional communication system emitted it as

The Primary of Social and Language Deficits in Autism

an exclamation of frustration.

Does the profile of language in high-functioning autism resemble one of these clinical pictures? If one tries to summarize the results of many research studies on autistic language (Fein, Green, Joy, & Waterhouse, 1996; Green, Fein, Joy, & Waterhouse, 1995), it seems that in general, phonological development, the discrimination and production of the sounds of the language is delayed consistent with overall verbal mental age, but not specifically abnormal except in individual cases. Similarly semantic development, the understanding and production of word meaning, is delayed consistent with verbal mental age, but also not systematically abnormal except in individual cases. Syntactic development, the production and understanding of grammatical forms and word order, seems also to be delayed but not specifically abnormal in most individuals with autism. On the other hand, several areas of language functioning are usually relatively impaired in such individuals. Verbal memory is often impaired relative to other aspects of language, and this is particularly marked when there is a high level of organization to the material, such as a story relative to a sentence, or a sentence relative to unrelated strings of words (Fein, Dunn, Allen, Aram, Hall, Morris, & Wilson, 1996). Even more marked are deficits in prosody and pragmatics (Rapin & Dunn, 1997). Prosody is the normal melody of speech, and the autistic individuals often ignores others' intonations or emphases, and produces language that is stereotyped or flat in its prosody. Pragmatics is the social use of language, and autistic individuals are particularly abnormal in the social uses to which they put their language and the degree to which they initiate communications with others.

Thus, to the extent that phonology, semantics, and syntax are relatively spared, while prosody and pragmatics are impaired, autism appears to be one of the conditions in which the lateral cortical systems are relatively spared, while there is impairment in the subcortical and medial fronto-temporal systems, in other words, the primate component of communication—the amygdala-cingulate-supplementary motor area axis that contributes crucially to one's drive to communicate, prosody, pragmatics, and the communication of emotion.

Are there any predictions that such a view would make about treatments that might be particularly effective for helping autistic children develop better language? In its purest form, this view holds that the language disturbances of autism are secondary to impaired attention to language and impaired motivation to communicate. Therefore, if one could force the child to attend to language and force the child to learn and perform expressive and receptive language tasks, then emerging language competence might unfold normally. Furthermore, the increasing language and social interaction skills that have been specifically taught might feed back and stimulate the motivational system. One treatment that meets this description is intensive early intervention of the applied behavior analysis type, in which the child works initially for extrinsic reinforcers, in order to master language tasks of increasing difficulty and complexity (Maurice, Green, & Luce, 1996). I have now seen approximately 150 such children in the last 3 to 4 years, many of them for more than one

D. Fein

evaluation. I am in the process of examining these patient's files to assess the number of such children whose language catches up to the level, or close to the level, of their peers in basic areas of language competence, and assessing the most-recovered among them for residual subtle language deficits. While I do not yet have data to answer this question, I have formed some strong overall clinical impressions. One is that while all children in these intensive intervention programs make significant progress, approximately one-third make gains that place them at or near the level of their age peers in language skills. The children who make these dramatic gains all began their treatments before the age of 4. Another impression is that nonverbal intelligence in the normal range is a necessary but not sufficient condition for such rapid language progress. Some children who do not make dramatic language gains appear to be globally retarded, but others do not.

What bars these remaining children from making the dramatic language progress of the most successful children? It appears that there are at least two types of children who fail to make the rapid gains of the most successful group. One is the children whose desire for self-stimulatory behavior, and whose basic indifference to most of the incentives that we can provide, is so strong that it prevents full involvement with the educational program. They will work for primary reinforcers such as food, but the motive for mastery of the material or to please the adults never seems to take over, as it does in the more successful children. In other words, the motivational abnormality is of a type or intensity which is beyond the therapists' ability to bypass. The other type of children with limited success in language gains is the children who appears to be engaged with the tasks and trying their best, but who simply cannot seem to master the basic elements of language such as discriminating, imitating, or combining words. In other words, these children, some but not all of whom have general retardation, also seem to have a dense language impairment.

How do these types of children relate to the view of autism as fundamentally a disorder of motivation and emotional relatedness based on limbic system dysfunction? For the children who perform very successfully in intense behavioral programs, one could say they had a motivational deficit which was to some degree overcome. For those who simply desire to engage in self-stimulatory behavior, one could say that they might have a motivational abnormality which it was not possible to overcome. For the children with general retardation, there must be additional CNS dysfunction; that is, they do not have "simple autism". The most problematic children, however, are those with normal nonverbal intelligence, but severely impaired ability to learn language. For these children, the arguments presented earlier really do not suffice, and one would have to broaden the search for their underlying pathology to include sources of specific language impairment.

Therefore, I must conclude that the arguments Dr. Kaplan and I made in our 1984 paper, and the limbic system approach that grew out of that viewpoint, may be correct for some of the children. It also appears, however, that autism is a highly complex set of possibly different disorders and that explaining the deficits shown by some of the children will require a different theory, possibly one that invokes a

The Primary of Social and Language Deficits in Autism

specific language impairment as a primary feature.

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D. Fein

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D. Fein

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