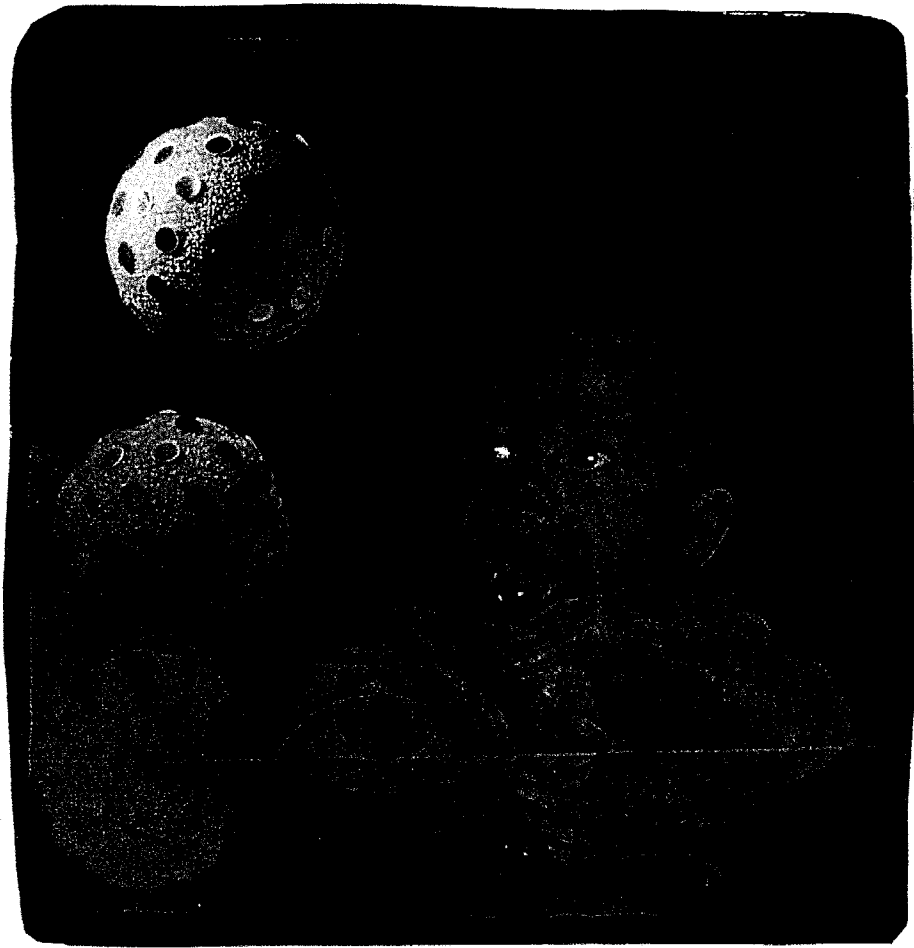


# THE DEVELOPMENT OF MIND:

Perspectives from Autistic Disorder



Gül Dölen

## INTRODUCTION

When we normally hear of autism we inevitably conjure up images of Dustin Hoffman's portrayal of the disease in the Barry Levinson film, Rain Man. We think of exceptional abilities that range from photographic memory to computational miracles (including the ability to count, in an instant, the number of toothpicks that Tom Cruise drops on the floor). Exceptional abilities in music, drawing, and calculation are indeed characteristic of autistic savants (see drawings below); however, only one in every ten individuals with autism show savant abilities. Autism afflicts some 80,000 American children, about one in every 2,500 children. Furthermore, of these, approximately 75% of are mentally retarded (IQ 35-50). Perhaps a more typical understanding can be gained by the following description:

"The boy is five years old. When spoken to he turns his head away. Sometimes he mumbles unintelligibly. He is neither toilet trained nor able to feed himself. He actively resists being touched. He dislikes sounds. He cannot relate to others and avoids looking anyone in the eye. He often engages in routine manipulative activities, such as dropping an object, picking it up, and dropping it again. While seated, he often rocks back and forth in a rhythmic motion for hours. Any change in routine is highly upsetting to him. He is in a school for severely psychotic children at UCLA. His diagnosis is childhood autism (Coleman, et. al., 562).

Autism was first described in the literature in a paper, "Autistic disturbances of affective contact," written in 1943, by L. Kanner (Happe, p 10). In 1944 Hans Asperger published another paper on the topic, independently and apparently without knowledge of Kanner's work. Amazingly, the two independent authors chose the same name for the disorder they were describing; the term 'autistic' comes from Bleuler's use of it in 1908 to describe schizophrenic's withdrawal from society as adults (derived from the Greek "autos" meaning "self"). This coincidence indicates the authors' belief that the primary and characteristic feature of this disease is the child's inability to socialize. Though the authors

disagreed on some of the features of the disease (such as the extent of language and motor disabilities), the current definition of autism reflects, to a large extent, their original descriptions (Frith, 1991). The DSM-IV criteria for autism are as follows:

A) A total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3):

(1) qualitative impairment in social interaction, as manifested by at least two of the following:

- a) marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
- b) failure to develop peer relationships appropriate to developmental level
- c) a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by lack of showing, bringing, or pointing out objects of interest)
- d) lack of social or emotional reciprocity

(2) qualitative impairments in communication as manifested by at least one of the following:

- a) delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
- b) in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
- c) stereotyped and repetitive use of language or idiosyncratic language
- d) lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level

(3) restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:

- a) encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
- b) apparently inflexible adherence to specific, nonfunctional routines or rituals
- c) stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
- d) persistent preoccupation with parts of objects

B) Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.

Those patients who have some of these features, but do not present with the full syndrome may be diagnosed with Asperger's Disorder or Pervasive Developmental Disorder Not Otherwise Specified. The disease is distinct from schizophrenia for its early onset, lack of hallucinations, and improvement with age (DSM-IV). Behavioral symptoms of autism include hyperactivity, short attention span, impulsivity, aggressiveness, self-injurious behaviors, and temper tantrums. Patients may be oversensitive to visual, auditory, olfactory, and tactile stimulation which result in extreme reactions to these stimuli. Autistics also bang their heads, spin, and rock, in a characteristically repetitive way. These behaviors and symptoms were originally thought to be caused by a cold and unresponsive mother whose attitude towards the child resulted in social withdrawal and isolation. However, this early hypothesis, posited by Bettelheim as well as Kanner, has more recently been rejected based on evidence that suggest that parents of autistics are no more likely to be 'emotional refrigerators' than parents of normal children. In fact, the causal role has since then been reversed; it has been suggested that "mothers are rendered autistic because of an inborn inability of their infants to respond affectionately to them" (Coleman, et. al., 565)

The problem of autism is complicated by the difficulty in identifying which are its primary symptoms, and which are its secondary symptoms (those which are the consequence of the primary symptoms); for these there are three candidates: disorder of linguistic competence, faulty sensory input and modulation, or an inborn error of relating. While it has been accepted autistic disorder is indeed an inborn one, biological evidence to support this view has been indistinct such that no one model can adequately account for the various symptoms and behaviors associated with the disease (including the problem of identifying its primary symptoms, as mentioned above). In the next section, these

biological models, including neurophysiology, neurochemistry and genetics will be explored. Still the most promising area of investigation concerning autism, continues to be cognitive psychology; a discussion of its models, specifically the theory of mind model, will therefore follow the biological. Finally some digression will be made into philosophy, not only to discuss the interesting philosophical problems that autism may shed light on, but also to answer some questions about the normal mind, specifically what it is, how it is developed, and how we should think about it in terms of physicalism.

## **NEUROBIOLOGY**

The neurological symptoms associated with autism include primitive reflexes, delayed development of hand dominance, and in some cases (25%) seizures. These symptoms, along with the broad range of psychological dysfunctions (described above) have lead researchers to look for a disrupted brain region to explain the disease. Unlike highly successful attempts to pinpoint a psychological disease to a specific brain area (like Broca's aphasia, for example) neurobiological research seems to suggest that autism is a dysfunction of a system of brain areas rather than a specific, localized anatomical region (Reichler, p 23). Nonetheless, a number of brain regions have been targeted based on existing information about their function and the possible psychological correlates between a disruption of those functions (caused by lesion) and the symptoms found in autism. Assuming that such a brain region could be targeted, it would follow that the disruption of these functions would produce the primary symptoms of autism, and the rest could be explained in terms of connectivity to other regions of the brain (whose disconnection from lesioned areas would produce secondary symptoms). For example, a study by Ornitz (1985) cited by Reichler, suggests that the problems associated with autism are caused by an insufficient modulation of sensory input by the brainstem- vestibular system which sends its projections to the thalamus. The variety of sensory processing problems associated with autism, like hyperactivity to auditory stimuli, visual ignoring, diminished

response to tactile stimuli and stereotyped repetitive movement, support this hypothesis. While these postulates do not directly account for language dysfunction and inability to properly relate to objects and people, it is presumed that the interaction between this and other systems in the brain, could account for these deficiencies (Reichler, p22-23). Possible brain areas that may modulate functions that are impaired in autistic patients include: Frontal lobes (formation of memory and modulation of emotional expression), Temporal lobes (modulation of language, hearing, vestibular function and motor activity), Amygdala (modulation of a broad spectrum of emotional reactions) and the Hippocampus (modulation of motor behavior and memory formation) (Reichler, p24-28). The cerebellum has also been implicated as a result of autopsy studies which indicate significant reductions in Purkinje cell density (cerebellum) compared with normal controls (Happe, p 31). Still, no neurobiological model has been able (either through lesion studies or otherwise) to predict the broad range of deficits associated with the disease.

### **NEUROCHEMISTRY**

The broad range of possibly damaged areas that could perhaps account for autistic deficits might be better explained in terms of a neurochemical problem, since a single neurotransmitter can act on more than one region of the brain. Once again, however, unlike research which has correlated psychological disease to neurochemical imbalance (like Parkinson's disease and dopamine) a neurochemical basis for autism has yet to find any causally sufficient imbalance. Part of the problem lies in the fact that in humans we are limited to neurochemical investigation by cerebrospinal fluid assay. However, research on other diseases also suffer from this setback, and it may simply be that autism will not ultimately be explained neurochemically. Still, it might be a matter of luck; we may someday find a drug that cures the disease (or key symptoms of it) that may shed light on an underlying neurochemical pathway for autism. There is some evidence for hyperserotonemia (elevated blood levels of Serotonin) in autistics: elevated blood Serotonin concentration, antiserotonin antibodies, and some behavioral response to drugs that affect

the serotonergic system. But these findings are neither consistent among all autistics nor are they limited only to autistics, which places some doubt on their causal relation to the disease (Yuwiler, 265-69). Dopamine, or rather the metabolites of dopamine found in the blood, levels are somewhat higher in autistic children, though again the inconsistent detection --neither all nor only autistics have been shown to have increased levels-- makes the evidence less significant. Still, dopamine receptor blockers, also used in treatment of Schizophrenia, do help mediate some of the common behaviors--but not the core signs or symptoms -- such as hyperactivity, stereotyped behaviors, self mutilations, and aggressive outbursts of autistics (Yuwiler, 273). Noradrenergic systems have not been significantly correlated to autism. Opioid systems may play a role in the play behavior children; some evidence purports lower concentrations of endorphin-like substances in autistic children in comparison to schizophrenic children, but evidence is limited and it has not been established how this circulating compound would affect opioid activity in the brain (Elliot, 253-54).

## **GENETICS**

Though the specific and critical pathway or brain region that is damaged in autism is not known, nor is there a strong correlation between autism and a neurochemical disruption, there is some genetic evidence -- although the exact role of genetic component is not clear -- that there is a biological cause of autism. First of all, autism is more than twice as common in boys as in girls (some studies place the ratio at 5:1); which may predict a chromosomal correlate. Autism is also 50 times more frequent in the siblings of autistics; even in those siblings who are not themselves autistic, there is an increased incidence of other cognitive, language and social, disorders (Happé p 29). Twin studies also point to a genetic predisposition for autism: identical twins show a far greater concordance for autism (95.7%) than fraternal twins (23.5%) (Schopler p 32). The genetic theory is also supported by the increased incidence of autism in patients with Fragile X syndrome, phenylketonouria, and tuberous sclerosis, all of which are genetically caused, though the

common pathway is unknown (Happe p 30). Another hypothesis that has been explored is that parents of autistic children may share more human leukocyte antigens. This increased similarity between the parents may lead to autistic symptoms caused by damage to the fetus done by the mothers' immune system, which may account for the increased incidence of preeclampsia and spontaneous abortions in mothers of autistic children (Shopler, p 32). Still much more research must be done to understand the genetic, and indeed biologic, component of autism.

### **COGNITIVE PSYCHOLOGY**

Because of the limited solutions that biology has provided in relation to this disease, presumably because of the complex functions the disease disrupts, much of the explanatory work in the area has come from cognitive psychology. The three symptoms, problems with socialization, communication, and imagination, that are most often associated with autism in cognitive psychology have been best predicted and explained by the concept of mind theory of autism proposed by Alan Leslie, Uta Frith, and Simon Baron-Cohen. This theory suggests that the deficits associated with autism stem from an inability to "mind-read." According to this explanation, normal people develop a concept of their own minds by an increased complexity of interaction with the world that surrounds them. By age four normal children understand the difference between persons and objects, and make the distinction between their own perspectives and those of other persons. Also, there is an implicit understanding in normal children that it is a person's beliefs and desires about the world -- which are mental states distinct from the physical world -- that govern a person's behavior. (Happe, pg. 38) Fundamentally problems with "mind-reading" have to do with an inability to metarepresent on the part of the autistic child. Failure to metarepresent has to do with an inability to think about mental states.

The theory of mind hypothesis of autism began with the observation that autistic children do not engage in pretend play. The inability to mentalize by autistic children was

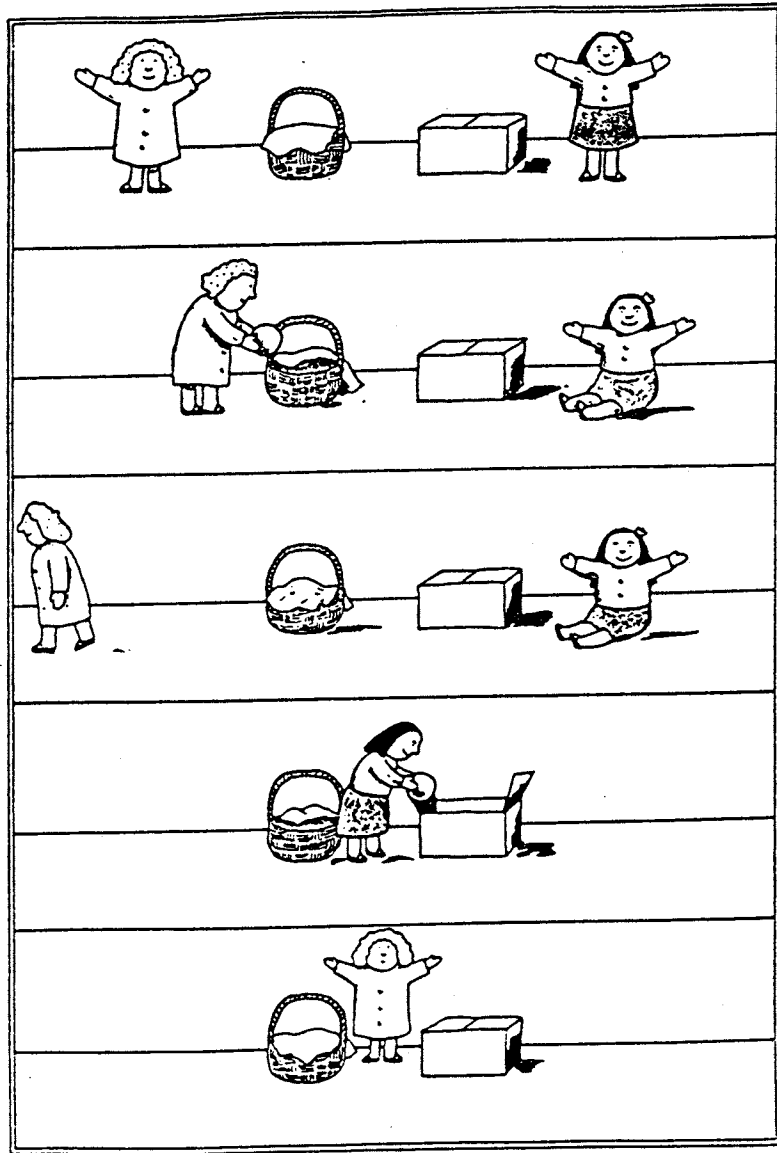


tested by Baron Cohen in the Sally-Ann task: Sally has a basket, Ann has a box. Sally puts a ball in her basket, then she leaves the room. Then Ann moves the ball from Sally's basket into her own box. Sally returns. The subjects are asked to indicate where the ball actually is; then, they are asked to indicate where Sally would think the ball is (see illustrated diagram of task, below). Normal children, as well as Down's syndrome children were able to successfully answer these questions over 86% of the time. On the other hand, 80% of the autistic children responded incorrectly to the second question. From such a false belief task, we learn that autistic children have a specific problem, independent of their mental retardation - evidenced by the down's syndrome patients' relative competence, understanding "that people have mental states which can be different from the state of the real world and different from the autistic person's own mental state" (Happe pg. 42). This inability to mentalize, presumably makes the world a very confusing and frustrating place for an autistic child and limits the patients' ability to communicate, interact socially, and have an active imagination. These deficits are reinforced with experience and development, and lead to the symptomatic problems which are associated with autism (see clinical description, above). Savant abilities do not violate the "theory of mind" explanation of autistic dysfunction; rather, they may actually be caused by these dysfunctions. The theory of mind "predicts that any skill which requires only primary representations should be unimpaired in autism - thus allowing for the islets of ability, good rote memory, savant abilities and above average IQ sometimes seen in autism" (Happe, 43-44). Excellent drawing ability, for example, may be characterized by a relatively piecemeal drawing style; whereas normal subjects start by constructing outlines and then proceed to parts, the autistic patient shows "no privileged status of the global form ... but rather a construction by local progression" (Happe 123) (see picture below). Uta Frith has constructed a causal diagram of the disease, which elucidates the connections between the biological factors affecting autism and the symptomatic characteristics of the disease.



Coleman

*Nadia, an autistic girl from Britain, drew the sketch shown at top left when she was only four years old. Her drawings—done in ballpoint pen—show a remarkably precocious understanding of the principles of perspective, foreshortening, and movement. At the age of seven and a half, Nadia entered a school for autistic children, where her learning skills improved while her artistic abilities suffered a decline. The man on the horse (top right) was drawn when Nadia was five (before her enrollment in the school). The drawing shown at left, done when Nadia was about eight, is a cruder treatment of the same subject.*



**Figure 5.1** The Sally-Anne task (by kind permission of the artist, Axel Scheffler).  
Reprinted from Frith 1989a.

Happe.

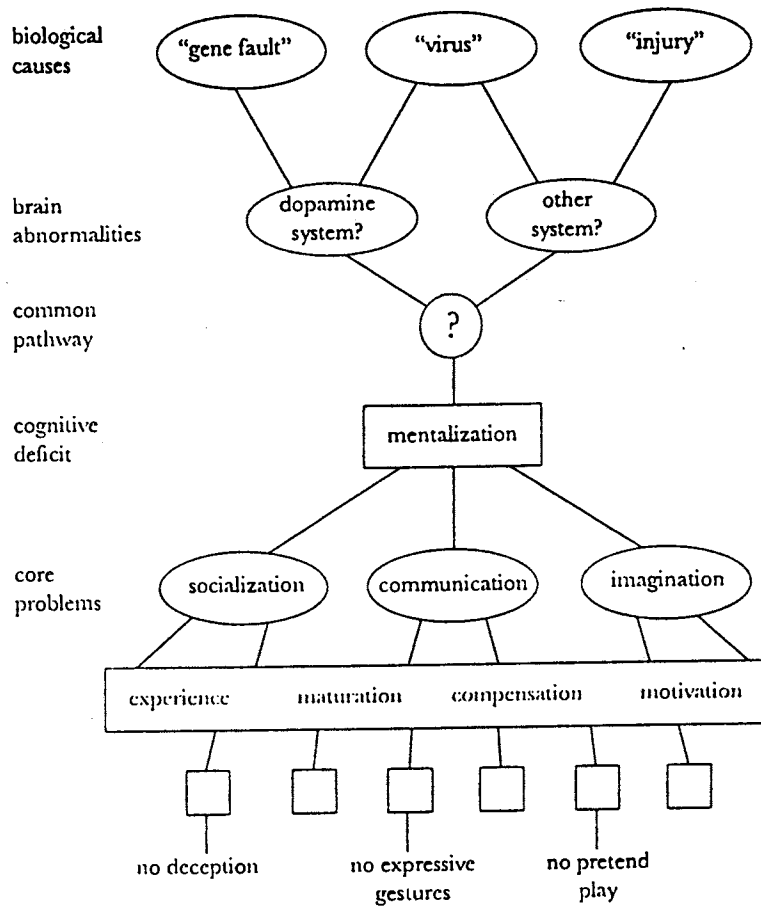


Figure 5.2 A causal model of the theory of mind account of autism (from Frith 1992, by kind permission of the author).

Happe.

## PHILOSOPHY

The problem of other minds, is one that philosophers have been struggling with at least as long as Descartes first proposed the skeptical problem. It poses the question -- how are we able to attribute consciousness to other minds, when, skeptically speaking, it is possible that no minds other than my own, really exist? Admittedly this is the sort of question that gives philosophers a bad name since it seems ridiculous in light of our common sense view of the world, specifically that other minds do exist. The question is interesting because it helps us to better formulate and understand what it is we are talking about when we ordinarily attribute mindfulness to other people -- what are the kinds of things a person must have in order to be counted as conscious. The question becomes particularly interesting in light of a disease like autism, which seems to be linked to the pervasive skepticism about other minds exhibited by the autistic child, and might be an instance where we might not want to attribute full consciousness. The use of "pervasive skepticism" is metaphorical here; autism is a developmental problem, its symptoms arise from some sort of error in processing and understanding information, rather than a rational (skeptical) conclusion about epistemology.

The problem of other minds, as proposed by Descartes, is that I am immediately and internally aware of my own mind, though my knowledge of others depends on external speculation and cannot therefore be supposed. There is an argument, which Jerry Samet ( pg. 431) calls the mediated access view, which attempts to solve the problem of other minds by attacking the first premise -- that we are immediately aware of our own minds -- which leads to skepticism about other minds. It posits that we only become fully aware of our own minds when we become aware of the existence of other minds. This position fundamentally rejects the dualist claim that we have a mind, independent of other beings and our own bodies -- it is in fact through our bodies and other minds that we become fully aware of ourselves. This is not to say that we only know our own minds the way we know

other minds, we still have first person access to our own minds. It does nonetheless call into question whether we have direct, immediate awareness. It suggests that even consciousness relies on external evidence to prove this first person awareness.

The philosophical point can perhaps be better understood by the following exercise. Although it may seem that we have a rough notion of I, independent of other, even this linguistic concept depends on the concept that *I* is separate from *other*. Without the concept *other*, the concept *I* has no meaning. If you believed that yours was the only mind that exists, you would also have to believe that all other consciousness are to be attributed to your own mind, in which case you could not even make the denotation of "me" which is the thing you are supposing, by taking this skeptical position, in the first place. Thus, you would have believe that at least one other mind exists, be it God's mind or whatever. Even Descartes' own brain in a vat example denotes the existence of one other mind -- that of the evil genius (Graham, p 37).

Samet hypothesizes that while normal children have an intuitive and introspective ability to grasp this conceptual distinction between self and other , a concept which must nonetheless be developed through experience, autistic children develop it more indirectly, "as a theoretical postulate" (Samet, p. 433). This difference, between concept and theory, normal and autistic, is comparable, Samet suggests, to the difference between the language acquisition of a native speaker and that of non-native speaker. Thus, in a sense, "mind-understanding" is rather like a second language for an autistic child. (The differences between the concept-mediated approach versus the theory-mediated approach is a topic of some controversy; however, for the purposes of this paper, the distinctions that make the controversy viable will not be pursued; for more on the topic, see Samet p.434 ). As a result, the psychology suggests, autistic children do not recognize the presence of other people, for example, *because* they are unable to attribute mindfulness to other people. They are also consequently unable to understand consciousness even in themselves -- they are neither aware of their minds nor their bodies. Examples of behaviors that support these

claims about mind attribution include autistics' inability to form meaningful emotional bonds with other people and their self-destructive behaviors, like repetitive head banging -- which seem to stem from a lack of self awareness, rather than anger or frustration. Of course the skeptic would say that we can never know what it is like to be in the mind of an autistic person, but the theory of mind explanation of autism says that neither does the autistic person. Thus, the rejection of the dualists' skeptical claim by the mediated access view seems to be validated by the cognitive psychology surrounding autism. It may be said that we are "begging the question" by rejecting the skeptical problem in light of a theory of mind which is itself based on a theory that rejects the skeptical problem. But, at some point we have to trust the research done in cognitive psychology; although the questions we are asking come from a theory of mind, they nonetheless lend credence to it, independent of the theory -- the theory allows us to make predictions about states of consciousness, predictions that can be, and have been, tested.

## REFERENCES

- Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) American Psychiatric Association, Washington, D.C. 1994.
- Bettelheim, Bruno. The empty Fortress, Infantile Autism and the Birth of the Self. The Free Press, New York, New York. 1967
- Coleman, J.C.; Butcher, J.N.; Carson, R.C. Abnormal Psychology and Modern Life. Seventh edition. Scott, Foresman and Company. Glenview, Illinois. Chapter 14.
- Elliot, G.R. and Ciaranello, R.D. "Neurochemical Hypothesis of Childhood Psychoses." Neurobiological Issues in Autism. Edited by Schopler, E. and Mesibov, G.B. Plenum Press, New York, New York, 1987
- Frith, U. Autism and Asperger syndrome. Cambridge University Press, Cambridge, 1991.
- Gopnik, A. 'Developing the idea of intentionality: Children's theories of Mind,' Canadian Journal of Philosophy, 20 (1990).
- Graham, G. Philosophy of Mind: An Introduction, Blackwell, 1993
- Happe, F. Autism, an Introduction to Psychological Theory. Harvard University Press. Cambridge, Massachusetts, 1995.
- Reichler, R.J. and Lee, E.M. "Overview of Biomedical Issues in Autism." Neurobiological Issues in Autism. Edited by Schopler, E. and Mesibov, G.B. Plenum Press, New York, New York, 1987
- Samet, J. "Autism and Theory of Mind: some philosophical perspectives." Understanding Other minds, Perspectives from Autism. Edited by Baron-Cohen, H. Tager-flusberg, D. Cohen. Oxford University Press, New York, New York, 1994.
- Yuwiler, A. and Freedman, D.X. "Neurotransmitter research in Autism" Neurobiological Issues in Autism. Edited by Schopler, E. and Mesibov, G.B. Plenum Press, New York, New York, 1987