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CHAPTER 41

Theory of Mind in Autism: Its Relationship to Executive Function and Central Coherence

SIMON BARON-COHEN AND JOHN SWETTENHAM

Over the past decade, an important area of research in the psychology of autism has emerged, generating a considerable amount of scientific attention. This concerns the ability of children with autism (a) to appreciate their own and other people's mental states—such as their beliefs, desires, intentions, knowledge, pretense, and perception; and (b) to understand the links between mental states and action. This area is, for shorthand reasons, referred to as "theory of mind." This phrase was coined by Premack and Woodruff (1978). They suggested that the ability to reflect on mental states was theory-like because mental states are *unobservable* entities which we infer to be underlying people's actions; and because reference to mental states allows us to *explain and predict* other people's behavior with remarkable power. This gives it at least some of the properties of a theory.

In 1987, with the first edition of this book, work in theory of mind and autism had already begun. It did not surface there because of the time lag familiar in academic publishing. Thus, in reviewing this area, we will cite work from the mid-1980s. We begin with the review, and we then consider the relationship of this aspect of cognition in autism to two other cognitive processes: "executive function," and "central coherence." Like theory of mind, both of these have also emerged as important domains over the past decade of psychological research in autism.

DOES THE AUTISTIC CHILD HAVE A THEORY OF MIND?

This was the question—and the title of the paper—that opened this area (Baron-Cohen, Leslie, & Frith, 1985). The question was asked because of the interest that was developing concerning the normal child's understanding of mental states. Indeed, two years before this was asked of children with autism, the related question had been asked of normal 4-year-olds. To make this issue tractable, Wimmer and Perner (1983) had devised an elegant paradigm in which the child was presented with a short story, with the simplest of plots. The story essentially involved one character who was not present when an object was moved, and therefore did not *know* that the object was in a new location. The subject being tested is asked where the character thinks the object is. Wimmer and Perner called this the False Belief test: the focus was on the subject's ability to infer a story character's mistaken belief about a situation. These authors found that normal 4-year-olds could correctly infer that the character would think the object was where the character had last left it, rather than where it actually was. This was impressive evidence for the normal child's ability to distinguish between his or her own knowledge (about reality) and someone else's false belief (about reality).

The screening studies reported herein were supported by three sequential project grants from the Medical Research Council (MRC) (1988–1996).

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When this test was given to a sample of children with autism, with mild degrees of mental handicap, a large majority of them "failed" the test by indicating that the character would think the object was where it actually was (Baron-Cohen et al., 1985). That is, they appeared to disregard the important information that, by virtue of being *absent* during the critical moving, the character's mental state would be different from the child's own mental state. In contrast, a control group of children with Down syndrome, with moderate degrees of mental handicap, passed this test as easily as the normal children. The implication was that the ability to infer mental states may be an aspect of social intelligence that is relatively independent of general intelligence (Cosmides, 1989), and that children with autism might be specifically impaired in the theory-of-mind domain.

Simply failing one test would not necessarily mean that children with autism lacked a theory of mind. One swallow does not make a summer. There might be many reasons for failure on such a test. (Interestingly, control questions in the original procedure ruled out memory, or language difficulties, or inattention as possible causes of failure.) The conclusion that children with autism are indeed impaired in the development of a theory of mind only becomes possible because of the convergence of results from widely differing experimental paradigms. These are reviewed in detail in an edited volume (Baron-Cohen, Tager-Flusberg, & Cohen, 1993) and for that reason are only briefly summarized in the next section.

SUMMARY OF RESULTS ON AUTISM AND THEORY OF MIND[1]

The majority of children with autism have the following characteristics:

1. They are at chance on tests of the *mental–physical distinction* (Baron-Cohen, 1989a): that is, they do not show a clear understanding of how physical objects differ from *thoughts* about objects.
2. They have an appropriate understanding of the functions of the brain, but have a poor understanding of the functions of the mind (Baron-Cohen, 1989a); that is, they recognize that the brain's physical function is to make a person move and do things, but they do not spontaneously mention *the mind's mental function* (in thinking, dreaming, wishing, deceiving, and so on).
3. They also fail to make the *appearance–reality distinction* (Baron-Cohen, 1989a), meaning that, in their description of misleading objects (like a red candle in the shape of an apple), they do not distinguish between what the object *looks* like, and what they *know* it really is.
4. They fail a range of *first-order false belief* tasks, of the kind described in the previous section (Baron-Cohen et al., 1985, 1986; Leekam & Perner, 1991; Perner, Frith, Leslie, & Leekam, 1989; Reed & Peterson, 1990; Swettenham, 1996).
5. They also fail tests that assess whether they understand the principle that "seeing leads to knowing" (Baron-Cohen & Goodhart, 1994; Leslie & Frith, 1988). For example, when presented with two dolls, one of whom touches a box, and the other of whom *looks inside* the box, and when asked, "Which one *knows* what's inside the box?" they are at chance in their response. In contrast, normal children, at 3 to 4 years of age, correctly judge that the doll who looked is the one who knows what's in the box. (See Figure 41.1).
6. They are at chance in *recognizing mental state words* (like "think," "know," and "imagine") in a word list (Baron-Cohen et al., 1994).
7. They do not *produce* the same range of mental state words in their spontaneous speech (Baron-Cohen et al., 1986; Tager-Flusberg, 1992).
8. They are impaired in the production of *spontaneous pretend play* (Baron-Cohen, 1987; Lewis & Boucher, 1988; Wing, Gould, Yeates, & Brierley, 1977). Pretend play is relevant here simply because it is thought to involve understanding the mental state of *pretending*.
9. Although they can understand simple causes of emotion (such as situations and

[1] In the following list of studies, all of the tests mentioned are at the level of a normal 4-year-old child.

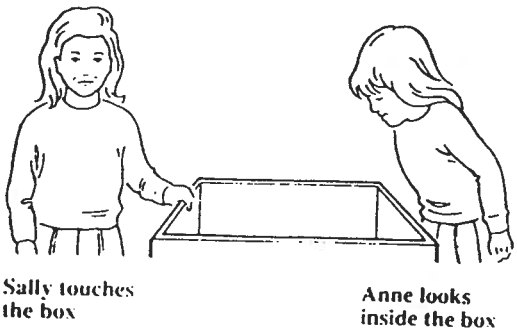

Figure 41.1 The "seeing-leads-to-knowing" test. After Baron-Cohen and Goodhart (1994). Adapted from Pratt and Bryant (1990).

desires), children with autism have difficulty understanding more *complex causes of emotion* (such as beliefs; Baron-Cohen, 1991a; Baron-Cohen, Spitz, & Cross, 1993). For example, they can understand that if Jane falls over and cuts her knee, she will feel sad, and that if John gets what he wants, he will feel happy. But they are poor at understanding that if John *thinks* he's getting what he wants (even if in reality he is not), he will feel happy.

10. They fail to recognize *the eye-region of the face* as indicating when a person is *thinking* and what a person might *want* (Baron-Cohen, Campbell, Karmiloff-Smith, Grant, & Walker, 1995; Baron-Cohen & Cross, 1992). For example, unlike normal 4-year-olds, they do not correctly judge which person is thinking in Figure 41.2, or which candy the cartoon character Charlie wants in Figure 41.3. Children and adults without autism use gaze to infer both of these mental states.
11. They fail to make the *accidental-intentional distinction* (Phillips, 1993); that is, they are poor at distinguishing whether someone "meant" to do something, or whether it simply happened accidentally.
12. They seem unable to *deceive* (Baron-Cohen, 1992; Sodian & Frith, 1992), a result that would be expected if they are unaware that people's beliefs can differ and therefore can be manipulated.
13. They fail tests of understanding metaphor, sarcasm, and irony, which are all *intentionally nonliteral statements* (Happe, 1994).
14. They fail to produce most aspects of *pragmatics* in their speech (reviewed in Baron-Cohen, 1988; see also Tager-Flusberg,

Figure 41.2 The "Which one is thinking?" test. From Baron-Cohen and Cross (1992).

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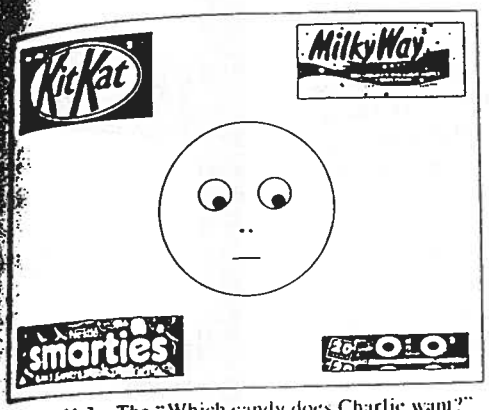

Figure 41.3 The "Which candy does Charlie want?" test. From Baron-Cohen, Campbell, Karmiloff-Smith, Grant, and Walker (1995).

1993), and fail to recognize violations of pragmatic rules, such as the Gricean Maxims of conversational cooperation (Surian, Baron-Cohen, & Van der Lely, 1996). For example, if a character replies to a question with an irrelevant answer, normal young children are very sensitive to this pragmatic failure, but most children with autism are not. Because many pragmatic rules involve tailoring one's speech to what the listener needs to *know*, or might be *interested* in, this can be seen as intrinsically linked to a theory of mind.

This long list of experiments provides strong evidence for there being a theory of mind deficit in autism. For this reason, autism can be conceptualized as involving degrees of ***mindblindness*** (Baron-Cohen, 1990, 1995). One possibility arising from these studies is that there may be a particular part of the brain which, in the normal case, is responsible for understanding mental states, and which is specifically impaired in autism. If this view is correct, the assumption is that this may be for genetic reasons, because autism appears to be strongly heritable (see Chapter 17). The idea that the development of theory of mind is under genetic/biological control in the normal case is consistent with evidence from cross-cultural studies: Normally developing children from markedly different cultures seem to pass tests of theory of mind at roughly the same ages (Avis & Harris, 1991). The exact part of the brain that might be involved in this is not yet clear, although candidate regions include right orbitofrontal cortex, which is active when subjects are thinking about mental state terms during functional imaging using single photon emission tomography (SPECT; Baron-Cohen et al., 1994); and left medial frontal cortex, which is active when subjects are drawing inferences about thoughts while being PET- (positron emission tomography) scanned (Fletcher et al., 1995; Goel, Grafman, Sadato, & Hallett, 1995). Other candidate regions include the superior temporal sulcus and the amygdala (for reasons explained below). These regions may form parts of a neural *circuit* supporting theory-of-mind processing (Baron-Cohen & Ring, 1994). Finally, it is possible that the development of a theory of mind involves input from lower-level social perception systems, some of which may be impaired in autism (Baron-Cohen, 1994; Klin & Volkmar, 1993).

It is important to mention data that do not easily fit the strong form of the theory-of-mind hypothesis. First, a small minority of children or adults with autism pass first-order false-belief tests. (First-order tests involve inferring what one person thinks.) However, these individuals often fail second-order false-belief tests (Baron-Cohen, 1989b), that is, tests of understanding what one character thinks another character thinks. Such second-order reasoning is usually understood by normal children of 5 to 6 years of age, and yet these tests are failed by individuals with autism with a mental age above this level. We can therefore interpret these results in terms of there being a *specific developmental delay* in theory of mind at a number of different points.

Some individuals with autism who are very high-functioning (in terms of IQ and language level), and who are usually adults, may pass even second-order tests (Bowler, 1992; Happé, 1993; Ozonoff, Pennington, & Rogers, 1991). Those who can pass second-order tests correspondingly may have difficulties understanding figurative language (Happé, 1993), suggesting they do not have a normal theory of mind. Appropriate adult tests of theory of mind reveal these persisting deficits (Baron-

Cohen, Jolliffe, Mortimore, & Robertson, in press; Baron-Cohen & Hammer, in press).

In summary, the theory-of-mind deficit in the majority of cases with autism is very severe. It has the potential to explain the social, communicative, and imaginative abnormalities that are diagnostic of the condition, because being able to reflect on one's own mental states (and those of others) would appear to be essential in all of these domains. The theory-of-mind deficit has been found to correlate with real-life social skills, as measured by a modified version of the Vineland Adaptive Behavior Scales (Frith, Happé, & Siddons, 1994). In the next section, we consider the developmental origins of this cognitive deficit.

DEVELOPMENTAL ORIGINS OF THEORY OF MIND

In an influential article, Leslie (1987) proposed that, in the normal case, the developmental origins of theory of mind lay in the capacity for pretense, and that, in the case of children with autism, the developmental origins of the theory-of-mind deficit lay in their inability to pretend. In his model, pretense was the crucible for theory of mind because both involved the same computational complexity. Thus, to understand that someone else might *think* "This banana is real," or might *pretend* "This banana is real," the child (according to Leslie) would need to be able to represent the agent's *mental attitude* toward the proposition. One idea, then, is that theory of mind is first evident from about 18 to 24 months of age, in the normal toddler's emerging pretend play.

However, there is some evidence that theory of mind might have even earlier developmental origins. Soon after the first demonstrations of a theory-of-mind impairment in autism—and again, too late for inclusion into the first edition of this *Handbook*—Marian Sigman and her colleagues at UCLA reported severe deficits in *joint attention* skills in children with autism (Sigman, Mundy, Ungerer, & Sherman, 1986). Joint attention skills are behaviors, produced by the child, which involve monitoring or directing the target of attention of another person, so as to coordinate the child's own attention with that of somebody else (Bruner, 1983). Such behaviors include the pointing gesture, gaze monitoring, and showing gestures, most of which are absent in most children with autism. This was an important discovery because joint attention behaviors are normally fully developed by about 14 months of age (Butterworth, 1991; Scaife & Bruner, 1975), so their absence in autism signifies a very early-occurring deficit. This was also important because the traditional theory-of-mind skills referred to above are mostly those one would expect to see in a 3- to 4-year-old normal child. Deficits in these areas cannot therefore be the developmentally earliest signs of autism, because we know that autism is present from at least the second year of life if not earlier.

Implicit in the idea of joint attention deficits in autism was the notion that these might relate to a failure to appreciate other people's point of view (Sigman et al., 1986). Bretherton, McNew, and Beeghly-Smith (1981) had also suggested that joint attention should be understood as an implicit theory of mind. Baron-Cohen (1989c, 1989d, 1991b) explicitly argued that the joint attention and theory-of-mind deficits in autism were no coincidence, and proposed that joint attention was a *precursor* to the development of a theory of mind. In that study (Baron-Cohen, 1989c), young children (under 5 years old) with autism were shown to produce one form of the pointing gesture (imperative pointing, or pointing to request) while failing to produce another form of pointing (declarative pointing, or pointing to share interest). This dissociation was interpreted in terms of the declarative form of pointing being an indicator of the child's monitoring of another person's mental state—in this case, the mental state of "interest," or "attention." More recent laboratory studies have confirmed the lack of spontaneous gaze-monitoring (Leekam, Baron-Cohen, Brown, Perrett, & Milders, 1997; Phillips, Baron-Cohen, & Rutter, 1992). The demonstration of a joint attention deficit in autism and of the role that the superior temporal sulcus in the monkey brain plays in the monitoring of gaze direction (Perrett et al., 1985) has led to the idea that the superior temporal sulcus may be involved in the development of a theory of mind

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AN APPLICATION TO THE EARLY DIAGNOSIS OF AUTISM

The work on the developmental origins of theory of mind is important not only for its theoretical interest (in terms of understanding normal development), but also for its value as a tool in lowering, into infancy, the age of diagnosis of autism. Thus, at 18 months of age, absence of joint attention, in combination with an absence of pretend play, is a very strong predictor of autism, both in a high-risk study of siblings who were undiagnosed (Baron-Cohen, Allen, & Gillberg, 1992), and in a random population study (Baron-Cohen, Cox, et al., 1996). In the latter study, 16,000 children were screened by their health visitors for these behaviors, using the Checklist for Autism in Toddlers (CHAT). Just 12 children out of the total population lacked joint attention and pretend play, of whom 10 were discovered to have clear autism. (The other two cases were not normal, but did not meet research criteria for autism.)

THEORY OF MIND, EXECUTIVE FUNCTION, AND CENTRAL COHERENCE

While there is now considerable evidence for the theory-of-mind deficit in autism, it is also clear that this is not the only cognitive deficit in autism. Two others have emerged as important in the past 5 years: (a) children with autism fail tests of "executive function," and (b) they also fail tests of "central coherence." Each of these is reviewed briefly below (see also Chapter 40). These additional deficits are important because although the theory-of-mind deficits may account for aspects of the social, communicative, and imaginative abnormalities, there are other symptoms (such as repetitive behavior, and unusual perception) that are not easily explained by the theory-of-mind deficits.

Executive Function and Autism

Executive function is the postulated mechanism that enables the normal person to shift attention flexibly, inhibit prepotent responses, generate goal-directed behavior, and solve problems in a planful, strategic way (see Baddeley, 1991; Shallice, 1988). The basic idea, developed by Norman and Shallice (1980), is that without a "central executive," or a "Supervisory Attentional System" (SAS) as it is also called, actions are controlled by the environment, and the organism simply responds to cues that elicit behavior. Without an SAS, action schemas or motor programs "contend" between themselves for execution. This takes place in a system known as the Contention Scheduling System (CSS). Shallice's notion is that the CSS is broadly a basal-ganglia function, and the SAS is basically a frontal lobe function. The SAS allows inhibition of routine (CSS) actions. The claim that the SAS is a frontal lobe function derives from the evidence that patients with frontal lobe damage fail tests of SAS (or executive) function.

Tests of executive function include the following:

1. The Wisconsin Card Sorting Test (Milner, 1964), in which the subject has to shift card-sorting strategies flexibly.
2. The Tower of Hanoi (and its modified version, the Tower of London; Shallice, 1982), in which the subject has to solve problems by planning before acting.
3. The Verbal Fluency Test (or F-A-S test; see Perret, 1974), in which the subject has to generate, in a fixed time period, novel examples of words beginning with a given letter.
4. The Detour Reaching Test (Diamond, 1991), in which the subject has to inhibit reaching straight for a visible goal, and must take a detour route to the goal instead.

Patients with frontal lobe damage fail on these tasks (reviewed in Shallice, 1988), and so do patients with autism (Hughes & Russell, 1993; Hughes, Russell, & Robbins, 1994; Ozonoff, Pennington, & Rogers, 1991; Prior & Hoffman, 1990; Rumsey & Hamburger, 1988). This observation has led to the conclusion that children with autism might have frontal lobe damage. Hughes and Russell (1993) have

suggested that they might fail theory-of-mind tests listed earlier because they cannot "disengage from the salience of reality."

There seems little doubt that there is an executive dysfunction in autism, and that this is likely to be a sign of frontal pathology. However, it is important to note that executive dysfunction occurs in a large number of clinical disorders, and in this respect it is not specific to autism. Thus, the following eight patient groups all show impairments on different tests of executive function:

1. Schizophrenia (Elliot, McKenna, Robbins, & Sahakian, 1995; Frith, 1992; see Elliot & Sahakian, 1995, for a review).
2. Treated patients with PKU (Diamond, 1994; Pennington, van Doorninck, McCabe, & McCabe, 1985; Welsh, Pennington, Ozonoff, Rouse, & McCabe, 1990).
3. Obsessive-Compulsive Disorder (Christensen, Kim, Dysken, & Hoover, 1992; Head, Bolton, & Hymas, 1989; Zelinski, Taylor, & Juzwin, 1991).
4. Tourette's Syndrome (Baron-Cohen, Moriarty, Mortimore, & Robertson, 1995; Baron-Cohen & Robertson, 1995; Bornstein, 1990, 1991).
5. Attention Deficit with Hyperactivity Disorder (ADHD) (Chelune, Ferguson, Koon, & Dickey, 1986; Gorenstein, Mammato, & Sandy, 1989; Grodzinsky & Diamond, 1992; Loge, Staton, & Beatty, 1990).
6. Parkinson's disease (Downes et al., 1989).
7. Frontal lobe syndrome (Owen, Roberts, Polkey, Sahakian, & Robbins (1991).
8. Children and adults with mental handicap (Borys, Spitz, & Dorans, 1982).

This list implies that there is no specific mapping between psychiatric classification and the concept of what Baddeley and Wilson (1988) call a "dysexecutive syndrome" (Baron-Cohen & Moriarty, 1995). Because all of these conditions involve an executive impairment, and yet do not lead to autism, it follows that, by itself, an impairment in executive function cannot explain autism. In addition, because some studies now show a dissociation between executive function and theory of mind in some disorders (e.g., Tourette's Syndrome; Baron-Cohen, Moriarty, et al., 1995), this means that they may be relatively independent processes.[2]

As presently construed, the concept of executive function may be too broad a level of analysis. The model suggests the presence of several component processes (generativity, attention shifting, disengaging, and so on), and perhaps specificity of deficit will be more apparent at this more fine-grained level of analysis. One example of a component process hypothesis is that, in autism, there is a deficit in "disengaging from the salience of reality." However, this cannot be correct in its strong form because, in a number of studies, subjects have to do just this, and yet children with autism *pass* these tests. The tests include:

1. Visual perspective taking (Baron-Cohen, 1989c, 1991c; Hobson, 1984; Tan & Harris, 1991). In these tasks, the child has to infer what someone else can see from his or her spatial position, even if this view is different from what the child currently sees.
2. False photograph tests (Leekam & Perner, 1991; Leslie & Thaiss, 1992; Swettenham, Baron-Cohen, Gomez, & Walsh, 1996). In these tasks, the child has to infer where something will be in an outdated photograph of reality, when the child knows that reality has been changed and the object is actually in a new position.
3. False map tests (Leekam & Perner, 1991; Leslie & Thaiss, 1992). This test uses a map instead of a photograph to measure the same ability as in the false photograph task.
4. False drawing tests (Charman & Baron-Cohen, 1992). These tasks test the same ability as the false photograph task, but a drawing is used instead of a photograph.
5. False model tests (Charman & Baron-Cohen, 1995). These tasks test the same ability as the false photograph task, but a model is used instead of a photograph.
6. Intellectual realism tests in drawing (Charman & Baron-Cohen, 1993). In these tasks,

[2] A further confound within the field of autism research is that many tests of theory of mind involve some attention shifting, and many tests of executive function involve taking into account one's own mental states, such as one's plans and thoughts.

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the subject is asked to draw an object that is partially occluded—for example, a coffee mug whose handle is out of view. Children with autism show "intellectual realism" at the same mental age as do children without autism (i.e., below a mental age of about 6 years); they include the occluded part or object even though it is out of view. For example, they draw the handle of the coffee mug, even when it is not visible. (Not until after a mental age of about 6 years has been achieved will subjects (with or without autism) show "visual realism," drawing only what they see, not what they know about.) This task is relevant in that if children with autism were "prisoners" of reality, they would show precocious visual realism, which they do not.

For these reasons, it is likely that theory of mind is not reducible to executive function. Rather, executive function deficits in autism may cooccur with theory-of-mind deficits because of their shared frontal origin in the brain. Despite these provisos, the executive hypothesis of autism is important because of its potential to explain the perseverative, repetitive behaviors in this condition, which are not accounted for by the theory of mind hypothesis. Perseveration and repetitive behaviors are symptomatic of frontal lobe syndrome, in which executive dysfunction is also seen (Shallice, 1988). In this view, the two cognitive deficits may be separately responsible for different types of abnormal behavior.

Central Coherence and Autism

The third and last area of cognitive deficit in autism that is reviewed here is in what Frith (1989) calls "central coherence." This is a slippery notion to define; the essence of it is the normal drive to integrate information into context, gist, gestalt, and meaning. Frith argues that the autistic person's superior ability on the Embedded Figures Test (Shah & Frith, 1983; Jolliffe & Baron-Cohen, in press) and on an unsegmented version of the Block Design subtest in the Wechsler Intelligence Scale for Children (WISC) and Wechsler Adult Intelligence Scale (WAIS) (Shah & Frith, 1993) arises because of a relative immunity to context effects in autism. Happé (in press) also reports a failure, by people with autism, to use context in reading, such that homophones are mispronounced (e.g., "There was a *tear* in her eye" might be misread so as to sound like "There was a *tear* in her dress"). A recent study has shown that children with autism are equally good at judging the identity of familiar faces in photographs, whether they are given the whole face or just part of the face. Nonautistic controls show a "global advantage" on such a test, performing significantly better when given the whole face, not just the parts of the face (Campbell, Baron-Cohen, & Walker, 1995). The central coherence account of autism is attractive in having the potential to explain the nonholistic, piecemeal, perceptual style characteristic of autism, and the unusual cognitive profile seen in this condition (including the islets of ability).

As with the other two theories, it appears that a strong version of the central coherence account cannot be correct because children with autism perform in line with their mental age on a range of tasks that would seem to involve integration across context. These include: (a) *transitive inference* tests (Scott & Baron-Cohen, 1996); (b) *analogical reasoning* tests (Scott & Baron-Cohen, 1996); and (c) *counterfactual syllogistic reasoning* tests (Scott, Baron-Cohen, & Leslie, 1995).

Happé (in press) reports that some high-functioning patients with autism who pass second-order theory-of-mind tasks nevertheless fail tasks of central coherence, such as the homophone task mentioned earlier. This dissociation implies that theory of mind and central coherence may also be relatively independent processes (Frith & Happé, 1994). In sum, a strong version of the central coherence theory would suggest that individuals with autism should be unable to recognize whole objects, and only perceive their parts, which we know does not occur. Instead, a weak form of central coherence theory seems likely to be correct, disabling individuals with autism from making full use of context. Whether this can account for islets of ability in autism (and even in Idiot Savant Syndrome) remains to be investigated in detail.

SUMMARY AND FUTURE DIRECTIONS

In this chapter, we have reviewed evidence for a theory-of-mind deficit in autism. We have also looked at two other cognitive deficits, in executive function and central coherence and have concluded that the theory of mind, executive function, and central coherence deficits are relatively independent of one another. Given this independence, one possibility is that there is a specific theory-of-mind mechanism (ToMM; Leslie, 1987, 1991; Leslie & Roth, 1993) and it is specifically damaged in autism. Leslie suggests that the function of such a mechanism is to represent information in a data structure, as shown in the following example:

> Agent—Attitude—"Proposition"
>
> Fred thinks "the safe is behind the Picasso."

Such a proposal is sufficient to allow representation of the full range of mental states in the Attitude slot. Explaining exactly how the brain is able to implement such a process will be important for future research, not only in relation to understanding the normal brain, but also in relation to autism. Circumventing theory-of-mind deficits through the use of carefully designed teaching methods will also be an important goal for applied research in this area. This research is already underway, with some promising results (Hadwin, Baron-Cohen, Howlin, & Hill, 1995; Swettenham, 1996; Swettenham et al., 1996). Ultimately, any biological theory of autism will have to account not only for specific genetic abnormalities, but also for how such abnormalities cause brain damage of the type that causes the specific cognitive deficits reviewed above.

We close with four questions for future research.

1. If, in autism, there are cognitive deficits in all three domains reviewed here (theory of mind, executive function, and central coherence), which of these are *necessary and sufficient* for the development (and diagnosis) of autism? We can clarify this question using the Venn diagram method in Figure 41.4. In which regions of the diagram do individuals with autism fall? Only in regions A, D, F, and G? Are these different subtypes of autism?

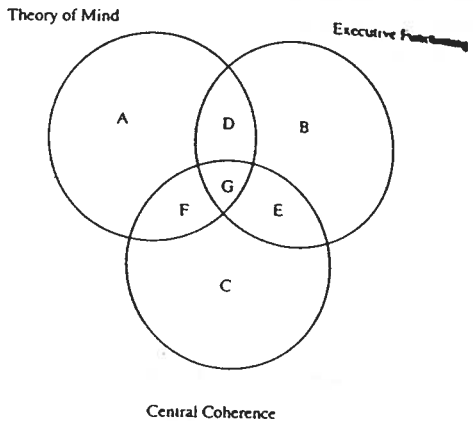

Figure 41.4 A Venn diagram of the possible relationship among deficits in theory of mind, executive function, and central coherence. Different diagnostic groups and subgroups may correspond to the different regions in the diagram. For example, autism may only occur in regions A, D, F and G, and these may reflect subtly different subgroups. See text for details.

2. Related to the first question, how do cognitive deficits in each of these three areas map on to areas of abnormal behavior? Do the three cognitive domains correlate with the three behavioral domains to which they have been theoretically tied? (To recap, the theory-of-mind deficit has been theoretically tied to the abnormal social, communicative, and imaginative development; the central coherence deficit has been theoretically tied to the abnormalities in perception and in processing contextual information; and the executive function deficit has been theoretically tied to the presence of repetitive behavior and cognitive inflexibility.) Do correlational studies bear out these mappings between cognition and behavior?

3. Which other psychiatric conditions might correspond to the "pure" or combined forms of executive function, central coherence, or theory-of-mind deficits? Can one derive any specificity between diagnosis and type of cognitive deficit, for each of the lettered regions in Figure 41.4?

4. Are these three domains of cognition really independent of each other, as the Venn

diagram implies? Double dissociations should be possible among all of these, if they are truly independent processes. If not, how might they relate to one another? We hope that such questions will be answerable in the next edition of the *Handbook*.

Cross-References

Aspects of social development in autism and a critique of theory of mind are provided in Chapters 8 and 43. Cognitive aspects of autism are discussed in Chapters 11 and 19. Executive function studies are reviewed in Chapter 40, and language aspects of theory of mind are discussed in Chapter 42.

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