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Neuropsychological Frameworks for Understanding Autism

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Abstract

Neuropsychological theories have traditionally attempted to provide a unifying account of the complex and diverse behavioral manifestations of autism in terms of their underlying psychological mechanisms and associated brain bases. This article reviews three competing neuropsychological theories of autism: the *executive dysfunction hypothesis*, the *weak central coherence hypothesis*, and the *limbic system hypothesis*. Each theory is evaluated critically with regard to the primary neuropsychological deficit hypothesized and the research findings that have been offered in support of it. In a concluding section, some of the metatheoretical assumptions informing attempts to identify a “core” neuropsychological impairment in autism are outlined and questioned, and new approaches to a neuropsychological understanding of autism are suggested.

Neuropsychological Frameworks for Understanding Autism

Although the current diagnostic criteria for autism (APA, 1994; WHO, 1994) represent an important consensus on how to identify this complex syndrome, they constitute a *descriptive* account of the disorder. A complete *explanatory* framework for understanding autism would ultimately involve at least four levels of analysis: (1) etiology, (2) brain structures and processes, (3) neuropsychology, and (4) symptoms or behaviors (Pennington, 1991; Pennington & Welsh, 1995). At the most basic level, *etiology* concerns the genetic and possible environmental factors that cause autism. These etiological factors are presumed to lead to abnormalities in the development of *brain structures and processes* that underlie the behaviors and symptoms that constitute autism. The *neuropsychological* level of explanation is intermediate between brain and behavior. It attempts to link these two levels of analysis by mapping relationships between brain structures and the psychological and behavioral functions they serve. As such, neuropsychological constructs potentially provide a unifying explanation for a range of behaviors and symptoms that may otherwise appear disparate and unrelated.

The neuropsychological and brain levels of analysis can ideally inform one another. In particular, it has been argued that research delineating the core psychological deficits of autistic individuals can help elucidate the brain bases of autism (Frith, 1989; Ozonoff, 1997a). Conversely, knowledge of what is or is not abnormal in the brain of autistic individuals can help to generate and constrain hypotheses about neuropsychological functioning (Bailey, Phillips, & Rutter, 1996; Pennington & Welsh, 1995). Yet, although there are a number of neurobiological findings concerning the brain bases of autism (see Bailey et al., 1996; Bauman & Kemper, 1994; Minshew, 1996; Minshew, Sweeney, & Bauman, 1997 for recent reviews), neurobiological and neuropsychological research on autism have proceeded largely independently of one another, and their “vertical integration” (Robbins, 1997) remains a promise of future research.

A major obstacle to this vertical integration has been the basic incompatibility between, on the one hand, the multiple brain abnormalities that have thus far been identified at the neuronal level of autism and, on the other hand, the search for focal and localizable psychological deficits that has guided most neuropsychological research on autism until now. On the basis of their comprehensive review of neuropathological and neuroimaging studies of autism, Minshew and her colleagues (Minshew, 1996; Minshew et al., 1997) have characterized the brain bases of autism as involving repeated and widespread disturbances in neuronal structure and organization, including truncated dendritic development in limbic structures, a reduced number of neurons and abnormal cell packing in the cerebellum, overgrowth of the posterior cortex, and indications of diffuse abnormalities in cortical connectivity. Findings of widespread structural abnormalities at the brain level of autism are not easily assimilated by neuropsychological paradigms seeking to isolate and dissociate the “impaired” from “spared” components of psychological functioning with the goal of providing a unifying explanation for autism’s complex behavioral manifestations. However, as will be suggested below, rather than leading to an impasse, the emerging findings from neurobiological research on autism, in conjunction with recent efforts to rethink explanatory frameworks for understanding developmental disorders (Karmiloff-Smith, 1998; Pennington, Rogers, Bennetto, Griffith, Reed, & Shyu, 1997), can help to reframe and redirect efforts to define the neuropsychological features of autism, which, in turn, may help to elucidate brain-behavior relationships in this complex developmental disorder.

This review will focus on three theories of autism, each of which postulates a core neuropsychological impairment that is argued to account for defining behavioral features of autism. The first of these is the *executive dysfunction hypothesis*, which views autism as a manifestation of primary deficits in executive control over behavior. The second, the *weak central coherence hypothesis*, posits that perceptual and cognitive biases favoring piecemeal over holistic information processing are responsible for many of the abnormal behaviors as well as the unusual pattern of cognitive strengths that characterize autism. The third, the *limbic system hypothesis*, proposes that impairment of psychological functions traditionally associated with the medial temporal lobes and limbic brain structures may best explain the profound social and communicative deficits that characterize autism. These theories will be reviewed critically with regard to the “core” or “primary” nature of the impairments they hypothesize.

The Executive Function Hypothesis

Executive function is a complex and still largely provisional concept that refers to those “mental operations which enable an individual to disengage from the immediate context in order to guide behavior by reference to mental models or future goals” (Hughes, Russell, & Robbins, 1994; see also Dennis, 1991; Robbins, 1996; Shallice, 1988). Executive function has been conceptualized more specifically as involving several overlapping but potentially dissociable mental operations, such as planning, working memory, maintenance and shifting of mental set, and inhibition of prepotent responses. At the same time, these functions are viewed as constituting a unified, higher-level system that is domain-general (i.e., not content-specific) and that is distinct from basic functions of sensation, perception, memory, and language (Pennington & Ozonoff, 1996). Executive function has been argued to be closely related to “fluid intelligence” and the ability to succeed at non-routine problem-solving tasks requiring flexible thinking and the generation of novel solution strategies (e.g., Duncan, 1995). Although executive functions were traditionally viewed as emerging only in later childhood (e.g., Golden, 1981), there is now a growing body of research documenting the development of executive control processes during infancy and the preschool years (e.g., Diamond & Doar, 1989; Diamond & Gilbert, 1989; Gerstadt, Hong, & Diamond, 1994; Hughes, 1998a, 1998b; Welsh, Pennington, & Groisser, 1991).

Deficits in executive control have classically been associated with acquired damage to the prefrontal cortex (Bianchi, 1922; Luria, 1966; Milner & Petrides, 1984), and particularly to the dorsolateral area (Fuster, 1997, Petrides, 1996). Yet, most researchers caution that findings of executive dysfunction in developmental disorders should not be assumed to reflect damage specifically to the frontal lobes, as it may arise from damage to interconnected cortical (e.g., parietal, temporal, and cingulate cortex) and subcortical (e.g., thalamus, striatum, hippocampus, amygdala) brain structures or from more diffuse brain damage (Bennetto, Pennington, & Rogers, 1996; Duncan, 1986; Pennington & Ozonoff, 1996; Robbins, 1996). Rather than “residing” in the prefrontal cortex, executive control abilities may depend on the integrated functioning of a number of overlapping but distinct neural structures (Robbins, 1996; 1997). Given these complexities, research investigating executive dysfunction in autism has thus far focused mainly on the neuropsychological and behavioral levels of analysis, with little reference to the underlying neuropathology.

The first study that explicitly investigated executive function in autism was conducted by Rumsey (1985). Guided by Damasio and Maurer’s (1978) hypothesis that the many shared symptoms of autism and frontal lobe injury might arise from similar neuropsychological deficits and reflect damage to the same underlying brain systems, Rumsey administered the Wisconsin Card Sorting Test (WCST) to high-functioning autistic individuals to see if they would show deficits comparable to those shown by frontal lobe patients. The WCST is a widely used clinical measure designed by Berg (1948) as a measure of abstraction and conceptual flexibility. The test consists of four target stimuli which vary on the dimensions of color, shape, and number (one red triangle, two green stars, three yellow crosses, and four blue circles). Participants are given a deck of sorting cards varying on the same dimensions as the targets (e.g., two red stars, one blue circle, and so on) and are asked to match each card to one of the four target cards. The examiner tells participants whether they have sorted the card correctly, but does not inform them of the sorting principle (which must be inferred from the examiner’s feedback) and does not bring participants’ attention to the dimensions on which the stimuli vary. After ten consecutive cards have been sorted correctly, the sorting principle changes without warning, making a previously correct sorting strategy incorrect. Using the WCST procedure, Rumsey (1985) found that autistic individuals were significantly impaired relative to controls on all key dependent measures, including number of sorts completed, total errors, and percentage of perseverative errors (i.e., sorting according to the previously correct principle despite negative feedback). These findings have proven fairly robust, with 7 out of 9 subsequent studies finding autistic deficits on the WCST (see Pennington & Ozonoff, 1996, for a review). Rumsey (1985) cogently described the potential relevance of these findings to autistic social deficits, observing that successful social functioning, like the card sort test, requires “integration and weighing of multiple contextual variables, selective attention to relevant aspects of the environment, and inductive logic” (p. 34). Thus, executive function deficits could potentially explain not only the inflexible and rigid behavior of autistic individuals, but also their impaired ability to engage in reciprocal social-communicative interactions, which require “on-line” evaluation of and selection of appropriate responses to a constant stream of subtle, multidimensional, and context-specific information (Bennetto et al., 1996).

At the same time as Rumsey was conducting her pioneering research on executive function in autism, a major competing theory of the autistic social impairment had begun to take form with a landmark study by Baron-Cohen, Leslie, and Frith (1985). These authors found that adults with autism were disproportionately impaired relative to controls on a now-classic false belief task developed by Wimmer & Perner (1983). In this task, participants were presented with a story in which a doll protagonist named Sally places her marble in a basket. In Sally’s absence, a second character, Anne, removes the marble and places it in a box. Upon Sally’s return, participants were asked, “Where will Sally look for her marble?” A correct answer thus required participants to infer Sally’s ignorance of the marble’s location and impute to her a belief that

was inconsistent with reality and what they knew to be true. Baron-Cohen et al.'s initial finding of a striking autistic impairment on this task was replicated and further supported by additional studies using different paradigms (see Baron-Cohen & Swettenham, 1988, for a review), leading to the theory of mind hypothesis of autism (Baron-Cohen, Tager-Flusberg, & Cohen, 1993). This hypothesis postulates that the social and communicative abnormalities of autism derive from a specific inability to understand other people's minds and to interpret behavior in terms of underlying mental states. Thus, from this point of view, the profound social abnormalities of autism arise from a domain-specific psychological deficit in social cognition (Baron-Cohen, 1990) and particularly the ability to "mentalize" the contents of another's mind.

The emergence of the theory of mind hypothesis of autism served as an important impetus for the executive dysfunction account of autism that has tended to view the limited appreciation of other people's minds in autism as reflecting a broader, domain-general deficit in executive control processes. These two hypotheses were first tested head-on in a study by Ozonoff, Pennington, and Rogers (1991), which included several theory of mind tasks as well as two measures of executive function, the WCST and the Tower of Hanoi (TOH; Borys, Spitz, & Dorans, 1982). Whereas the WCST is usually taken as a measure of set-shifting and cognitive flexibility, the TOH is typically viewed as a measure of planning. In this disk-transfer task, participants are presented with 3 or 4 disks placed in a specific configuration on three pegs, and are asked to re-arrange the pegs from the initial arrangement to a prescribed goal state, moving the pegs one at a time and using as few moves as possible. Possible moves on the TOH are further constrained by the rule that larger disks cannot be placed on top of smaller disks. Although Ozonoff et al. (1991) found that autistic individuals were impaired on both theory of mind and executive function tasks, the executive function deficits were the most widespread among autistic participants, and a composite score derived from WCST and TOH variables was the strongest discriminator of autism. These findings, in conjunction with the significant correlations found between theory of mind and executive function composite scores, led Ozonoff et al. to consider the possibility that executive dysfunction is the primary cognitive deficit in autism, and that the perseverative and inflexible problem-solving strategies that characterize executive dysfunction might account for the poor performance of autistic individuals on theory of mind tasks.

This possibility was tested more directly by Russell, Mauthner, Sharpe, & Tidswell (1991), who compared autistic children's performance on a location-change false belief task with their performance on the Windows task, a competitive candy-winning game. In this game, a piece of candy is placed in one of two boxes, each of which has a window that exposes the contents of the box to the participant but not to the experimenter-opponent. Participants then have to indicate to the opponent, by pointing, which of two boxes to look in to find the candy, and thus can win the candy by misleading the opponent and directing her to search in the empty box. Russell et al. found that autistic participants were less likely than controls to employ the deceptive strategy of misdirecting the opponent to the empty box, and in many cases perseveratively pointed to the baited box across all 20 test trials, despite the fact that they lost the candy to the opponent as a result. Further, deficient performance on the Windows task was associated with failure on the false belief task. Consequently, the authors proposed that autistic children's difficulties on these two tasks may have resulted from an impaired ability to inhibit responses to a salient stimulus (the true location of the object in the false belief task, the desired candy in the Windows task) rather than from a more specific deficit in mental state awareness. This proposal was supported in a critical follow-up study (Hughes & Russell, 1993) that demonstrated similar autistic difficulties in a no-opponent version of the Windows task that did not require deception and hence mental state awareness, but simply that participants point to an obviously empty box in order to obtain the candy that could be seen in the other box.

With a number of earlier studies having established the possibility that executive control deficits may account for the poor performance of autistic individuals on theory of mind tasks and, further, that executive dysfunction may be an underlying cause of the profound social impairment characteristic of autism (see also Ozonoff, Rogers, & Pennington, 1991; Ozonoff & McEvoy, 1994), more recent research efforts have turned toward better defining the specific executive function deficits in autism, especially given the global and underspecified nature of this construct and the omnibus quality of many of the measures that have been used to measure it. To illustrate, although the WCST has been characterized primarily as a measure of set-shifting or cognitive flexibility, successful performance on this measure depends upon a number of cognitive operations, not all of which are strictly executive in nature: conceptualization of the sort dimensions, deduction of the sorting principle from the feedback provided, maintenance of the sorting principle in working memory, selective attention to the relevant stimulus dimension, inhibition of the prepotent tendency to sort on the basis of the previously correct dimension, and shifting of cognitive set (Dehaene & Changeux, 1991; Ozonoff, 1995). Thus, standard clinical measures often do not permit identification of the specific executive control functions (and their potentially dissociable neural substrates) that are impaired and most closely linked to the behavioral manifestations of autism.

Further, findings of executive deficits in several different disorders calls into question the notion that executive dysfunction could have a specific causal role in the core symptomatology of autism, and raises the possibility that executive impairment is a general, non-specific correlate of many neuropathological conditions, including mental retardation, which occurs in the majority of autistic individuals (Ozonoff, 1997b; Pennington & Ozonoff, 1996). As Ozonoff (1997b) has suggested, although differences in the severity and in the timing of onset of the underlying neuropathology causing executive impairment may account for variations in its behavioral manifestations across disorders, it is also possible that a specific pattern of impairment in the components of executive function may distinguish autism from other disorders. Accordingly, more recent studies have turned away from using omnibus, clinical measures of executive functioning and have adopted information processing paradigms from experimental psychology and cognitive neuropsychology that are designed to dissociate the spared and impaired components of executive function (Feinberg & Farah, 1997; Friedrich & Rader, 1996; Ozonoff, 1997a). Component executive processes that have been investigated in autism include set-shifting and cognitive flexibility, inhibitory control, and working memory.

Recent research on cognitive flexibility in autism has focused on better defining the reasons for poor performance on the WCST, and particularly on differentiating what is an apparent weakness in the ability to shift cognitive set from other processes, such as a failure of inhibitory control or a tendency toward perseverative responding, that could also contribute to the autistic impairment on this task. Hughes et al. (1994) used the Intradimensional-Extradimensional (IDED) set-shifting task to investigate these issues. Unlike the WCST, the IDED task involves a hierarchy of increasingly challenging set-shifting demands that is designed to allow identification of the precise source(s) of difficulty. In the initial stages of the IDED task, Hughes et al. found no impairment in the ability of autistic individuals to learn to respond selectively to one of two simple patterns (pink shapes) or two compound patterns (pink shapes accompanied by irrelevant white lines), nor in their ability to respond to the previously incorrect pattern when the reinforcement contingencies were reversed. These findings ruled out specific problems with discrimination learning, establishing and maintaining a response set, attention to the relevant aspects of stimuli, and rule reversal. The key deficit for autistic individuals was revealed in the two final "transfer" stages of the IDED task. In the intradimensional shift, participants were required to transfer learning to a new set of exemplars while continuing to respond to the same dimension of the stimuli (pink shapes, not white lines). Finally, the extradimensional shift involved another change of exemplars, but success now required shifting response set to a new dimension of the stimuli (white lines instead of pink shapes).

Individuals with autism performed similarly to controls on the intradimensional shift, but were deficient relative to controls on the extradimensional shift. Thus, whereas autistic individuals had no particular problem with the conceptual requirements of the IDEED task in that they were able to identify the relevant response dimension and generalize it to a new set of exemplars, they appeared specifically impaired in their ability to break out of that particular cognitive set and adopt a new perspective on the task. Hughes et al.'s findings also confirmed that autistic deficits were of a specifically executive nature involving, in Sandson and Albert's (1984) terminology, "stuck-in-set" perseveration rather than a lower-level "recurrent" perseveration, which would have been evident in simple response repetition and set-shifting problems in the initial stages of the IDEED task.

Converging evidence for a specific autistic impairment in cognitive flexibility has been provided in a series of experiments conducted by Ozonoff and her colleagues (Ozonoff & Strayer, 1997; Ozonoff, Strayer, McMahon, & Filloux, 1994). These experiments demonstrated that autistic individuals were unimpaired when required to inhibit or withhold a simple response (e.g., pressing a button for circles, but not for squares), but that they had specific difficulty when required to shift from one response set to another (e.g., pressing a button for squares instead of circles). Further, these findings provided a partial dissociation of the executive function deficits of children with autism and those of children with attention disorders, whom have been shown to be impaired on the same inhibition measures on which autistic individuals were unimpaired (Ozonoff, 1997b).

Other studies have explored the possibility that executive dysfunction in autism may be driven by a core deficit in working memory. Working memory refers to the capacity to maintain in mind the transient, context-specific information that is needed to carry out ongoing or future actions. The question of whether autism involves a primary deficit in working memory capacity has been addressed by two recent studies (Bennetto, Pennington, & Rogers, 1996; Russell, Jarrold, & Henry, 1996), which have had conflicting results. Both of these studies used a similar set of working memory tasks, all of which required participants to respond to a series of items from a focal processing task while concurrently maintaining a mental record of all their responses. For example, in both studies, participants were administered a counting task (Case, Kurland, & Goldberg, 1982), in which they were asked to count the dots on each card in a set while keeping in mind the number of dots on each card in the order it was presented. However, whereas Bennetto et al. found that their relatively high-functioning autistic participants were significantly impaired relative to normal controls on such working memory tasks, Russell et al. found that the performance of a group of lower-functioning autistic children and adolescents, although inferior to a normal group, was similar to a mentally handicapped group. These findings led Russell et al. to argue that a deficit in working memory capacity, although perhaps characteristic of autism (as was also indicated by the findings of Bennetto et al.), is not specific to autism and is likely a manifestation of the broader neurological impairment common to autistic and mentally handicapped children.

The tasks used in the above studies are particularly informative in that they taxed working memory without taxing inhibitory control (Russell et al., 1996), and in doing so failed to isolate a specific autistic deficit in the working memory component of executive control. In addition, Ozonoff and colleagues did not find that problems with inhibition per se were fundamental to autism. These findings suggest that a model that takes into account the combined demands of working memory and inhibitory control may provide a useful heuristic for conceptualizing executive dysfunction in autism (Russell, 1997; Russell et al., 1996; Pennington et al., 1995). This model has been explicitly formulated by Roberts & Pennington (1996) as an "interactive framework," which views the resolution of response alternatives and the determination of appropriate action as involving both "working memory activations and inhibitory suppressions" (p. 106). From this perspective, the poor performance of autistic individuals on

a variety of executive tasks, often very different in their surface characteristics, may be best accounted for in terms of the simultaneous working memory and inhibitory demands posed by competing response alternatives. For example, the Tower of Hanoi disk-transfer task, on which autistic individuals consistently have been found to be impaired, can be conceptualized in terms of the dual requirements of maintaining a sequence of moves in mind and inhibiting the prepotent response to place the disk directly onto the goal peg (Roberts & Pennington, 1996; Russell et al., 1996).

Findings from studies using a variety of experimental measures have provided support for the idea that tasks requiring both working memory and inhibition of prepotent responses are particularly challenging for children with autism. Hughes (1996) found that children with autism were impaired relative to mentally handicapped and normal controls on a hand-game (Luria, Pribram, & Homskaya, 1964), in which they had to point a finger when the examiner made a fist, and vice versa. This task required children to maintain a response rule in working memory while inhibiting the prepotent response to copy the examiner. In another study, Hughes and Russell (1993) examined the performance of autistic children on two different conditions of a detour-reaching task. The goal of the detour-reaching task was to obtain a large marble that was located on a platform inside a box and in direct view of the child. In both conditions, a direct reach into the box would break an infrared beam and cause the marble to drop out of reach. In the "knob route" condition, the child was taught to turn a knob on the right side of the box connected to a paddle that would push the marble down a chute and out of the box. In the "switch route" condition, the child was taught to turn a switch on the left side of the box that would turn off the beam, thus enabling the child to reach into the box to obtain the marble. Thus, while there was a readily apparent mechanical relationship between turning the knob and obtaining the marble in the knob route condition, the connection between turning the switch and being able to retrieve the marble appeared completely arbitrary in the switch route condition. Hughes and Russell found that retrieving the marble on knob route trials posed no difficulty for their autistic participants, but that they were significantly impaired relative to controls in the switch route condition. Hence, the source of autistic difficulties could not be a simple lack of inhibitory control because autistic children were able to inhibit a direct reach to the marble in the knob route of the detour-reaching task. Rather, as Russell (1997) has argued in terms of the interactive model described above, the more arbitrary and obscure a mean-ends rule (e.g., turning an apparently unrelated switch to retrieve the marble in the detour-reaching task, pointing to the empty container to obtain the candy in the no-opponent Windows task), the more difficult it will be to maintain the rule in working memory, and thus to override the prepotent response bias (e.g., to reach directly for the desired object).

Drawing on Luria's classic ideas about the role of verbal processes in the development of self-control (Luria & Yudovich, 1971), Russell (1997) has elaborated on the interactive hypothesis, suggesting that autistic children may be impaired in the ability to use inner speech to maintain an arbitrary rule in mind (e.g., "turn switch, then reach for marble," or "when she points, I make a fist") and to guide behavior accordingly. In support of this hypothesis, Hughes (1996) found an association between children's success on the Luria hand game and their use of higher-level verbal self-control strategies in a delayed gratification task that was independent of their verbal comprehension level. Accordingly, the pragmatic language deficits in autism may extend beyond the appropriate use of language for communication to the use of language for self-regulation (Hughes, 1996).

Virtually all of the studies reviewed thus far have investigated executive function in older children, adolescents, and adults with autism. Although there has been a relative paucity of research on the executive functioning of younger children with autism, research with younger children is likely to prove particularly valuable in helping to distinguish between the core psychological deficits in autism and the secondary deficits and associated behaviors to which

they lead. The few studies that have been conducted thus far have produced inconsistent results. For example, McEvoy, Rogers, and Pennington (1993) examined both executive functions and basic social-communicative skills in older preschoolers with autism. On a Spatial Reversal executive functions measure, autistic children were found to have difficulty shifting set, and they made significantly more perseverative responses than controls. In the social-communicative domain, as has typically been found (e.g., Mundy, Sigman, Ungerer, & Sherman, 1986), autistic children exhibited deficits in joint attention behaviors, such as showing objects to the examiner as well as pointing and using shifts of gaze to direct the examiner's attention to an object of interest. Further, these social deficits were associated with perseveration on the Spatial Reversal task. Although correlational, McEvoy et al.'s findings suggested that an executive impairment in disengaging and flexibly shifting attention could contribute to a social impairment in coordinating shared attention between people and objects. Yet, although these findings were promising with regard to the possibility of establishing the causal precedence of executive functions deficits in autism, a subsequent study with younger autistic preschoolers conducted by Wehner and Rogers (1994; cited in Pennington et al., 1997) found no evidence of impairment on the Spatial Reversal task, even though the task appeared developmentally appropriate, with no floor or ceiling effects.

A more recent study of young children with autism has also yielded mixed results for the executive functions hypothesis. Dawson, Meltzoff, Osterling, and Rinaldi (1998) examined the performance of young autistic children on a delayed response task, a classic measure of executive functioning which is associated with dorsolateral prefrontal cortex (e.g., Diamond & Goldman-Rakic, 1989) and which taps both working memory and the ability to inhibit a previously rewarded response. In addition, children were administered a delayed nonmatching-to-sample (DNMS) task, a visual recognition memory test (described in detail below) traditionally associated with medial temporal lobe functioning. Dawson et al. found that autistic preschoolers were impaired relative to Down syndrome and normally developing children on both delayed response and DNMS. However, whereas autistic children's DNMS performance was correlated with a wide-range of measures assessing core autistic social deficits, delayed response performance was not. Although based on only two neuropsychological measures and in need of further investigation, these findings raise the possibility that although early executive deficits may be characteristic of autism, they are not primary in the sense of driving the core social and communicative abnormalities that are defining of the disorder.

In summary, a more finely delineated profile of executive function deficits in autism has begun to emerge from recent neuropsychological research. In particular, there is evidence of a specific impairment in cognitive flexibility or set-shifting in autism that is distinct from lower-level recurrent perseveration and motoric disinhibition, which may be more broadly associated with mental retardation. Further, although research indicates that there are not specific deficits in either inhibitory control or working memory, tasks that simultaneously tax both of these functions may be particularly challenging for individuals with autism. Executive functions deficits of this sort arguably account for the poor performance of autistic individuals on theory of mind tasks, which typically require participants to maintain information in mind while inhibiting a prepotent response. Although there has been considerable success in identifying the specific components of executive function that are impaired in autism and partially dissociating these executive deficits from those that are found in other childhood disorders such as attention disorder (Ozonoff, 1997), a remaining weakness of the executive dysfunction account of autism is that it has provided little evidence linking and specifying the relationship between executive control deficits and the autistic symptoms and behaviors they are presumed to underlie. One exception in this regard is a recent study by Turner (1997) that dissociated recurrent and stuck-in-set perseveration in the cognitive performance of autistic individuals, showing that the former was specifically associated with the presence of lower-level repetitive behaviors (e.g., stereotyped movements), whereas the latter was specifically associated with

the presence of higher-level repetitive behaviors (e.g., circumscribed interests). Yet, if the executive dysfunction theory is to provide a convincing alternative to the theory of mind view of autism, it needs to demonstrate similar associations between executive deficits and the impairments in social and communicative functioning that are so centrally defining of the disorder. Thus far, attempts to make such connections have not been replicated (Wehner and Rogers, 1994; cited in Pennington et al., 1997) or have suggested that executive deficits, although present, do not have a causal role in the social and communicative impairments (Dawson et al., 1998).

The Central Coherence Hypothesis

The weak central coherence hypothesis of autism (Frith, 1989; Frith & Happé, 1994; Happé, 1999) was explicitly formulated only in the last decade. Drawing upon assumptions from gestalt psychology (e.g., Koffka, 1935), this hypothesis posits that autism involves a failure of the “built-in propensity” in normal human information processing “to form coherence over as wide a range of stimuli as possible, and to generalize over as wide a range of contexts as possible” (Frith, 1989, p. 100). This failure is argued to result from a predominantly piecemeal processing style in which there is a tendency to perceive and construe complex stimuli as a disparate collection of parts rather than as coherent and meaningful wholes.

As has been argued by its main proponents (Frith & Happé, 1994, Happé, 1994), the notion of weak central coherence in autism provides an explanation of aspects of the disorder that have been relatively neglected by theories, including the theory of mind account, that have focused mainly on the social and communicative deficits of autism. Among these features are the restricted and repetitive interests and behaviors that constitute the third area of impairment defining of autism (APA, 1994). Although many of these behaviors have been more broadly associated with mental retardation and neurological impairment in general, they are nonetheless present in autistic individuals of normal and superior intelligence, and indeed are required for the diagnosis (Turner, 1997). These behaviors vary with developmental level and include, for example, a tendency to focus on the parts of play objects to the detriment of using them in more functional or conventional ways (e.g., spinning the wheels of a car, flicking the eyes of a doll); unusual and intense preoccupations with objects (e.g., push pins, paper clips) that would normally have little importance; extreme sensitivity and resistance to trivial changes in the environment (e.g., the position of a lamp on a table); and highly circumscribed interests (e.g., geographical or meteorological facts) that are not developed in relationship to a broader and more substantive body of knowledge (APA, 1994; Lord, Rutter, & Le Couteur, 1994). Such behaviors are argued to be manifestations of a weak “drive” for central coherence that ranges from the integration of lower-level perceptual inputs to the derivation of higher-level meaning.

Another unexplained aspect of autism addressed by the central coherence theory is the marked inconsistency that exists among different cognitive skills and, particularly, the “islets of ability” that often appear in the midst of otherwise limited intelligence. Among such splinter skills is the unusual ability demonstrated by individuals with autism on the Block Design subtest from the Wechsler and other intelligence scales (Elliott, Murray, & Pearson, 1979; Wechsler, 1991, 1997). A peak in Block Design performance relative to performance on other subtests has been found to be so reliable among both low- and high-functioning individuals with autism (see Happé, 1994, for a review; Yirmiya, Solomonica-Levi, Shulman, & Pilowsky, 1996) that it has been suggested as a possible marker for autism (Shah & Frith, 1993). The Block Design test, developed by Kohs (1923), requires an individual to mentally break down a geometric design into its component parts. These parts correspond to a set of blocks which must be used to reconstruct the design. Block design is generally viewed as a measure of visual-perceptual organization and nonverbal conceptualization ability (Lezak, 1995) and as a fairly good

indicator of general intelligence (Kaufman, 1979). However, Shah and Frith (1993) have provided evidence that paradoxically links autistic superiority in Block Design to an **abnormality** in information processing they describe as weak central coherence.

In their elegant experiment, Shah and Frith (1993) examined whether the autistic strength in Block Design derived from a tendency to see parts over wholes by comparing their ability to copy whole designs to their ability to copy segmented designs. They found that the autistic participants showed their usual advantage relative to controls in the whole condition, but that this advantage disappeared in the segmented condition as a result of the control participants' sharp improvement in performance. The lack of an improvement among the autistic individuals in the segmented condition thus suggested that their remarkable ability on the Block Design task is rooted in their superior segmenting skills and a weakening of the normal pull to see the design as a whole. Further, Shah and Frith presented participants with two other conditions in which task difficulty was increased by presenting the designs in rotated orientation and by increasing the number of oblique lines in the designs. They found that autistic participants demonstrated the same decrement in performance as controls in these two conditions, strengthening the conclusion that the autistic strength on Block Design derives from a facility with part-by-part processing rather than from more generally superior visual-spatial abilities.

In addition to Shah and Frith's (1993) intriguing findings, the notion of a weak drive for coherence in autism has received empirical support from a number of other experimental paradigms. This research includes experiments investigating coherence in visual-perceptual processes as well as studies that have examined central coherence in semantic processing and verbal inference. For example, in an investigation of the possibility of weak coherence at a relatively basic level of visual perception in autism, Happé (1997) examined children's judgment of well-known visual illusions, such as the Titchener circles and the Muller-Lyer figures. Following Gregory (1967), Happé conceptualized these standard illusions in terms of the normal operation of an "inducing" context on the misperceived part. For example, the inner circles in the Titchener circles illusion are easily perceived as the same size when they are presented alone, but are misperceived as different in size when one circle is presented in the context of surrounding small circles and the other in the context of surrounding large circles. Consistent with her predictions, Happé found that children with autism were significantly less susceptible than control participants to the illusions, suggesting that their perception of the target parts of the figures was less influenced by the overall gestalt. Moreover, Happé included a control condition similar to the segmentation condition in Shah and Frith's (1993) Block Design experiment in which the target parts of the black-and-white figures were represented with raised colored lines so as to make them stand out from the rest of the figure. She found that autistic children derived much less benefit than controls from this disembedding condition, adding support to her conclusion that there is a failure of normal coherence at this basic level of visual perception in autism. Happé (1999) cites similar evidence of local perceptual biases in autism from other recent studies, including findings that individuals with autism benefit less than controls from canonical die patterns in dot counting (Jarrod & Russell, 1997) and exhibit an unusually high occurrence of absolute pitch in their note-naming abilities (Takeuchi & Hulse, 1993).

Another paradigm for research on central coherence in autism has been provided by the Embedded Figures Test (EFT; Witkin, Oltman, Raskin, & Karp, 1971). Shah and Frith (1983) were the first to examine autistic children's performance on this test, using the children's version in which participants were asked to find simple shapes (a triangle or house shape) hidden in pictures of familiar, meaningful objects (e.g., a baby carriage, a rocking horse). They found that their low-functioning autistic participants were significantly superior to both mentally handicapped and normal controls in their ability to find the hidden shapes, consistent with the argument that autistic individuals are relatively insensitive to the broader context in

their information processing (Frith & Happé, 1994). Jolliffe and Baron-Cohen (1997) extended these findings by showing that high-functioning individuals with autism and with Asperger syndrome were superior to normal controls on the adult version of the EFT, in which participants were required to pick out target parts from abstract geometric designs. However, other studies (Ozonoff, Pennington, & Rogers, 1991; Brian & Bryson, 1996) have not found superior EFT performance among autistic individuals, and thus have failed to support the weak central coherence hypothesis.

The earliest experimental findings that have been garnered in support of a lack of central coherence in semantic processing in autism (Frith, 1989) are from among a series of seminal studies conducted by Hermelin and O'Connor that led these authors to conclude that autism involves a central deficit in the ability to "encode stimuli meaningfully" (Hermelin & O'Connor, 1970, p. 129). For example, in one experiment, Hermelin & O'Connor (1967) compared the ability to recall sentences with the ability to recall strings of unconnected words in autistic children and mentally handicapped controls matched on receptive vocabulary and digit span. Whereas the mentally handicapped group was able to recall much more from sentences than from the strings of random words, the autistic group did not benefit from the meaningful structure provided in the sentence condition. In a subsequent experiment designed to rule out the possibility that the relatively poor performance of autistic individuals on sentences was not due to deficits in syntactic processing, Tager-Flusberg (1991) demonstrated that autistic children, unlike controls, were no better at recalling a list of 12 nouns from the same semantic category (animals) than they were at recalling 12 semantically unrelated nouns.

Frith and Snowling (1983) further investigated weak coherence at the semantic level in autism by testing children's ability to use linguistic context to disambiguate homographs (words with the same spelling, but different pronunciation and meaning) in sentences they were asked to read aloud. In presenting children with sentences such as "He made a deep *bow*," Frith and Snowling found that autistic children were less likely than controls to use the sentence context to determine the meaning and hence the correct pronunciation of the word, but instead tended to use the more common pronunciation, as in, for example, "He had a pink *bow*." Happé (1997) subsequently replicated these findings in a larger and higher-functioning sample of autistic children and adults.

In her initial formulation, Frith (1989) suggested that autistic theory of mind deficits reflected a failure of central coherence in the sense that an understanding of the mental underpinnings of people's behaviors would require a "cohesive interpretive device [that] forces together complex information from totally disparate sources into a pattern which has *meaning*" (p. 174). However, this view was subsequently modified with the proposal that there are two distinct but "interacting" deficits in autism, namely, a specific modular impairment in the ability to mentalize, and a broader domain-general weakness in coherence (Frith & Happé, 1994; Happé, 1997; Happé, in press). This revision was motivated by evidence of weak central coherence even in the minority of autistic individuals who pass theory of mind tasks (Happé, 1994, 1997, in press). Interestingly, Happé (in press) has used these findings to explain the enduring social deficits of autistic individuals who exhibit a basic mentalism in the highly structured context of a false belief task, arguing that competence in real-life social interactions requires the integrative and inferential abilities that are defining of central coherence.

There has been little specification of the brain mechanisms potentially subserving central coherence. Happé (1999) has recently drawn a parallel between weak central coherence in autism and evidence of holistic-processing deficits for both verbal-semantic and visual-spatial information in patients with right-hemisphere damage (e.g., Benowitz, Moya, & Levine, 1990). However, at the same time, she raises doubts as to whether it makes sense to attribute an apparent failure of coherence across levels and domains of processing in autism to a unitary,

top-down brain mechanism such as a right-hemisphere hypothesis would imply. Instead, Happé proposes that the pervasive weakness in coherence and focus on detail that is found across levels of information processing is a result of more diffuse abnormalities of brain structure in autism. In support of this possibility, Happé cites recent evidence of brain overgrowth in autism (e.g., Piven, Arndt, Bailey, Havercamp, Andreasen, & Palmer, 1995), which may reflect a disruption of programmed cell death and neuronal pruning in autistic brain development. According to Happé (see also Cohen, 1994), the intriguing implication of these findings is that an excess of neurons may support piecemeal and exemplar-based processing and learning, but to the detriment of the development of more economic and efficient holistic processing strategies such as would be required, for example, to process verbal information for its gist rather than in a more strictly verbatim or literal manner.

In summary, the weak central coherence theory is perhaps most compelling in its ability to explain the remarkable skills as well as the profound deficits of autism in terms of a single underlying cognitive dysfunction or processing style (Happé, 1999; Shah & Frith, 1993). Yet, as Happé (1997) has pointed out, the central coherence account of autism remains “loosely defined and conceptualized.” One possible criticism is that it does not make sense on a neurological level to treat what are arguably highly modularized and encapsulated perceptual processes, such as are involved in visual illusions, as being subject to the same brain mechanisms as the higher-level processes involved in the interpretation of verbal information. However, as will be argued below, it may be misguided to constrain hypotheses about the functioning of developmentally disordered brains on the basis of what is known or thought about normally functioning brains. Nonetheless, there is as of yet no evidence that the abnormalities found across a range of tasks tapping visual-perceptual, visual-constructional, and verbal-semantic processing are, in fact, related to one another, as would be predicted on the basis of weak coherence, whether coherence is achieved via “centralized” or diffuse brain processes. Thus, in the very least, a remaining challenge for the weak coherence theory of autism is to determine if and how these wide-ranging processing abnormalities are related at the neuropsychological as well as the neuroanatomical level.

The Limbic System Hypothesis

What is here provisionally referred to as the limbic system hypothesis is less a coherent psychological theory than a collection of biological, neuropsychological, and behavioral findings that have connected this region of the brain to autism. The limbic system is comprised of the orbital frontal cortex as well as the medial temporal lobe, which includes the hippocampal formation and the amygdala. This region of the brain has been of particular interest to autism researchers because of the many ways in which it has been implicated in the social-emotional functioning of humans and primates (Brothers, 1990; Damasio, 1994; Dawson, 1996; Dawson et al., 1998; LeDoux, 1994). In addition, evidence of histoanatomic abnormalities in the medial temporal lobe structures of autistic individuals (e.g., Bauman & Kemper, 1985) has strongly contributed to interest in the potential role of this brain region in autism. Unlike the executive function and central coherence theories of autism, which have been elaborated primarily at the neuropsychological level of analysis, the limbic system hypothesis, as it names implies, has thus far been geared toward identifying a brain-level mechanism for autism. Its failure to specify the psychological functions and processes that may mediate between brain and behavior in autism, it will be argued, is the main weakness of this hypothesis.

Boucher and Warrington (1976) were among the first researchers to implicate dysfunction of the medial temporal lobe in autism, arguing that autism may be regarded as a “developmental form of the amnesic syndrome” (p. 85). They based this argument on a set of experiments investigating the memory skills of autistic children, in which they found deficits in delayed free recall and recognition abilities. Given prior findings of intact immediate recall abilities in

autism (e.g., Bartak, Rutter, & Cox, 1975), Boucher and Warrington argued that the selective impairment of long-term memory was similar to the pattern of memory impairment observed in amnesic adults with damage to the hippocampus.

However, several subsequent studies investigating memory functioning in autism have failed to support Boucher and Warrington's amnesic theory of autism. For example, a number of studies (Bennetto et al., 1996; Minschew & Goldstein, 1993; Minschew, Goldstein, & Siegal, 1997) investigating verbal memory in autism have failed to reveal autistic deficits in delayed recall or recognition of a list of words. Rather than an impairment in the storage and consolidation of verbal information, which would be expected in the case of an amnesic condition, these studies have demonstrated strategic or organizational weaknesses in verbal learning and memory that are more consistent with an executive function syndrome (Bennetto et al., 1996; Minschew & Goldstein, 1993). A similar pattern of findings has emerged from research on delayed recognition of visual stimuli in autism (Ameli, Courchesne, Lincoln, Kaufman, & Grillon, 1988; Bennetto et al., 1996). Thus, a relationship between temporal lobe amnesia and autism has not been supported.

Although there is little evidence to support the notion that autism is a temporal lobe amnesic syndrome, Bachevalier (1994) has nonetheless produced a convincing animal model of autism in monkeys with lesions to this region of the brain. To begin, Bachevalier examined the social-emotional functioning of rhesus monkeys who underwent removal of the entire medial temporal lobe (including the hippocampus, amygdala, entorhinal and perirhinal cortex, and parahippocampal gyrus) soon after birth. In comparing the lesioned monkeys to normal controls, she observed not only repetitive motor behaviors in the lesioned monkeys, but also striking, autistic-like social abnormalities that increased in severity between two and six months of age. These behaviors included passivity and failure to initiate interactions at younger ages, followed by active social avoidance with increasing age, and a relative paucity of eye contact and of facial and bodily expression (e.g., playful posturing). These social deficits increased in severity in the early months of life, and persisted in adulthood, and thus were consistent with the developmental course seen in humans with autism.

In an attempt to identify which structure was the most critical in autistic symptomatology, Bachevalier examined the behavior of monkeys who underwent selective ablation of either the hippocampal formation or of the amygdaloid complex in early infancy. The monkeys with selective amygdalar lesions displayed a pattern of socioemotional abnormalities that was similar to those of monkeys with removal of the entire medial temporal lobe, except that the behavioral disturbance was less severe. In contrast, monkeys with selective hippocampal lesions displayed only transient socioemotional disturbances that were no longer apparent at six months of age. Thus, Bachevalier's animal model associated autistic-like symptomatology specifically with damage to the amygdala. Overall, Bachevalier's findings were consistent with the neuropathological observations of Bauman and Kemper (1985), who found abnormalities in both the amygdala and hippocampus of severely affected autistic children.

Dawson et al. (1998) continued this line of investigation into the potential contribution of limbic system dysfunction to autism by comparing the performance of autistic preschoolers on a test of delayed response (described above), known to tap dorsolateral prefrontal cortex functioning in both humans and monkeys (e.g., Diamond & Goldman-Rakic, 1989), to their performance on a test of delayed nonmatching-to-sample (DNMS), which has been linked to the medial temporal and orbital frontal lobes (e.g., Diamond, Towle, & Boyer, 1994; Kowalska, Bachevalier, & Mishkin, 1991). In the version of the DNMS task used by Dawson et al., children were presented with a novel object. During this familiarization phase, children naturally reached to and displaced the object to find a reward underneath. The object was then removed for five seconds. During the test phase, the original object was presented again along

with a new object, under which a reward was hidden. Thus, to succeed at nonmatching to sample, children had to recognize the original object as familiar so as to be able to discriminate the novel object. In addition, over a series of trials, children had to learn the rule that choosing the novel object resulted in the receipt of a reward. In order to examine whether the two neural systems tapped by the delayed response and DNMS tasks are differentially implicated in autism, Dawson et al. compared children's performance on each of these tasks to their performance on a number of behavioral measures designed to assess key social and communicative deficits associated with early childhood autism. Behaviors assessed included children's level of social orienting, immediate and deferred imitation, joint attention, affective responsivity, and symbolic play. Dawson et al. found that autistic preschoolers were impaired relative to Down syndrome and normally developing children on both the delayed response and DNMS tasks, and in all of the social-communicative domains assessed. However, whereas autistic children's performance on the delayed response task was only associated with immediate imitation skills, their DNMS performance was strongly correlated with performance on all of the social-communicative measures that were administered.

An important consideration in evaluating Dawson et al.'s findings concerns the particular DNMS task that was used. As acknowledged by the authors, the brief five-second delay employed between familiarization and testing had the effect of emphasizing the rule-learning requirements over the memory demands of the DNMS task. In fact, there is evidence that DNMS does not tap medial temporal lobe functioning at delays less than 15 – 30 seconds (Diamond et al., 1994; Diamond et al., 1997). Further, mastery of the nonmatching to sample rule (and, particularly, the understanding that the novel stimulus is a "marker" or "symbol" of the reward's location; Diamond et al., 1997) appears to be dependent on the integrity of the ventral lateral region of the orbital frontal lobes (Kowalska et al., 1991). Accordingly, Dawson et al.'s findings appear to implicate, more specifically, orbital frontal dysfunction in the social-communicative deficits of autism, and thus converge with other evidence connecting orbital frontal cortex to social cognition (Damasio, 1994; see Stone, in press, for a review).

In summary, the available evidence does not support the amnesic theory of autism. Rather, the memory deficits that have been identified in autism appear to be of an executive nature, involving weaknesses in the use of organizational strategies to encode and recall information. Nonetheless, the neuropathological findings of Bauman and Kemper, Bachevalier's animal model, and the study recently conducted by Dawson and her colleagues continue to indicate that the limbic system and its associated functions are potentially important in development of autism, and suggest that this is an important area for future research. The Dawson study is particularly informative in that it demonstrates autistic deficits in both executive function (delayed response) and in what is arguably a limbic system function (short-delay DNMS), but shows that only the latter deficit is associated with core autistic symptoms. Yet, although the Dawson study is exemplary in providing a paradigm for evaluating the core neuropsychological deficits in autism, its main weakness is that it implicates limbic brain structures in autism only indirectly, by virtue of the connections that have been previously made between DNMS performance and this region of the brain. Thus, the exact nature of the neuropsychological impairments that are thought to drive autistic social deficits remains to be defined, which is the promise of future research into the potential role of medial temporal and limbic brain structures in autism.

Summary and Conclusions

As a complex developmental disorder of genetic and, more proximally, neurological origin, autism poses the challenge of explaining how widespread anomalies in brain structure and organization lead to such a distinct yet disparate pattern of symptoms and behavioral features. Each of the theories reviewed in this article hypothesizes and provides laboratory evidence in

support of a different core neuropsychological impairment that can arguably account for many if not all of the behavioral manifestations of autism and that can potentially map onto abnormalities at the brain level. The weak central coherence and executive dysfunction hypotheses are similar in that they posit broad, domain-general cognitive abnormalities in information processing or, in the case of the latter, in executive control over information processing and the planning of action. Neither of these theories makes strong claims regarding the neural systems underlying the cognitive impairments they postulate, but in both cases their proponents argue that the neural bases are more likely to be widely distributed than localizable to any specific region of the brain (e.g., Happé, 1999; Robbins, 1997). The opposite situation obtains in the case of the limbic system hypothesis, which connects autistic social deficits to brain-level dysfunction in medial temporal and limbic structures, but which fails to specify the precise psychological mechanisms that mediate between brain and behavior. Presumably, these functions involve such processes as assigning appropriate affective significance to stimuli (e.g., Dawson et al., 1998; DeLong, 1992), but these functions have not yet been operationalized or empirically evaluated in autism.

A weakness relevant to all the theories reviewed here is the lack of data connecting the neuropsychological deficits that have been identified in autism to the behavioral abnormalities they presumably underlie. The recent study conducted by Dawson and her colleagues (Dawson et al., 1998) is an exception in this regard. Future research investigating the relationship between the neuropsychological and behavioral levels of autism will be especially useful in differentiating between primary deficits and those that are secondary or that are more generally correlated with mental retardation and not necessarily specific to autism. Although difficult to conduct with rare populations, longitudinal studies can examine the causal relationships between hypothesized core neuropsychological deficits and measures of core autistic symptoms, such as were employed in the Dawson study. Longitudinal studies focusing on early development in autism are likely to be particularly informative for two reasons. First, the use of relatively simple tasks makes the component processes of neuropsychological functions easier to isolate and measure, and thus avoids confounding them with other processing demands (Hughes, 1998). Second, the primary versus secondary nature of a deficit (e.g., whether executive impairments lead to social skills deficits, or a failure to engage in social interactions impedes the development of executive control skills; Pennington et al., 1997) can be established by tracking the emergence of behaviors early in development.

Defining the connections between the neuropsychological impairments in autism and their underlying brain bases is an even greater challenge for future research. As was suggested in the introduction to this paper, evidence of multiple brain abnormalities in autism is not compatible with the metatheoretical assumptions that tend to inform most neuropsychological research on autism and other neurodevelopmental disorders. These assumptions derive from traditional neuropsychological approaches to studying the effects of acquired lesions on the functioning of normally developing brains, and are most closely associated with the classic method of *double dissociation*: a lesion to brain area *A* impairs function *a* but spares function *b*, whereas a lesion to brain area *B* spares function *a* but impairs function *b* (Heilman & Valenstein, 1985). The success of classic lesion studies in dissociating component cognitive functions and the brain regions subserving them has lent support to the view of the brain as organized into specialized modules that are differentially subject to damage in the case of brain insult or injury. From this perspective, the behavioral features of autism and other neurodevelopmental disorders are explainable in terms of a pattern of impaired and intact brain modules or neural circuits. However, as has been increasingly recognized by researchers working in the field of developmental disorders (e.g., Bailey et al., 1996; Kamiloff-Smith, 1997, 1998; Pennington, 1994; Pennington et al., 1997), extrapolating from the effects of late-acquired brain lesions on normally developing brains to the functioning of developmentally disordered brains is problematic. As Kamiloff-Smith (1997, 1998) has argued, the brain

develops differently from the start in a neurodevelopmental disorder, both in terms of the dynamics of genetic expression and the constraints that disordered development sets on environmental inputs. Thus, the same structure-function relations do not necessarily hold across normal and disordered brain development and, consequently, similar behaviors, whether aberrant or apparently normal, do not necessarily reflect the same brain bases. This has been illustrated by the weak central coherence account of autism, which argues that the apparently “intact” visual processing skills evidenced by autistic individuals on Block Design and other tasks are actually supported by an abnormal, local bias in information processing. Karmiloff-Smith (1997) makes a similar point with regard to claims of “spared” face recognition abilities in the midst of otherwise severely impaired visual-spatial skills in individuals with Williams syndrome, arguing that such a distinction obscures the likely possibility that these individuals depend excessively on exemplar-based learning of faces and are actually deficient in the higher-level configural processing abilities that are normally used to recognize and to discriminate unfamiliar faces.

A second, related and largely implicit assumption that informs most neuropsychological research on developmental disorders is the expectation that the behavioral manifestations of a disorder will be accountable in terms of one critical and unifying cognitive deficit (e.g., Morton & Frith, 1995). Thus, although pathological brain processes may have direct, unmediated effects on behavior, privilege tends to be accorded to the intervening cognitive or psychological level of explanation in providing a cohesive and unifying explanation of the core symptoms of a disorder. This is seen in the theory of mind, executive functions, and central coherence theories of autism, each of which postulates a critical cognitive impairment that can account for symptoms across the social, communicative, and restrictive and repetitive behavior domains of autism. However, as several investigators have recently pointed out (Bailey et al., 1996; Minshew et al., 1997; Pennington et al., 1997), the diverse behavioral manifestations of autism may only cohere, if at all, at the biological or genetic level. To illustrate this possibility, Pennington et al. (1997) propose that a dopaminergic deficit in autism could conceivably produce impairments in working memory, affective responsivity, and motor control by virtue of its effects on three independent neural systems – the prefrontal cortex, limbic cortex, and basal ganglia, respectively. Accordingly, executive function deficits, social aloofness, and motor stereotypes would co-exist in autism with no unifying explanation at the cognitive level. Instead, they would derive from a shared abnormality at the biological level (Pennington et al., 1997).

A reworking of these assumptions and biases is likely to foster the vertical integration of the brain, neuropsychological, and behavioral levels of analysis in neurodevelopmental disorders. Further, reassessment of modularity and localizing assumptions may allow for a greater “horizontal integration” of neuropsychological findings in autism, rather than continued adherence to the notion of a central unifying deficit. This direction may be particularly fruitful in light of emerging neurobiological findings suggesting multiple and diffuse rather than localized brain abnormalities in autism (e.g., Minshew et al, 1997). From this perspective, the competing neuropsychological theories of autism may prove to complement one another in explaining autism’s complex behavioral phenotype. Thus, for example, decreased connectivity among cortical regions and between the cortex and subcortex could lead to disturbances of the integrated functioning necessary for executive control as well as to disruptions of subcortical inputs crucial to appropriate social-affective behavior. At the same time, widespread aberrations in neuronal pruning and dendritic development could result in the piecemeal processing bias, exemplar-based learning, and failure to integrate information for the extraction of higher-level meaning that has been described as weak central coherence in autism. Of course, the danger of any perspective emphasizing the diffuse and generalized abnormalities of brain functioning in autism is that it will fail to reveal what is specific about autism and what sets it apart from other neurodevelopmental disorders. However, research that strives to define the

links between the core behavioral manifestations of autism and the wide-ranging brain and neuropsychological impairments that have thus far been identified promises to capture both the complexity and what is unique about this enigmatic developmental disorder.

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References

- Ameli R, Courchesne E, Lincoln A, Kaufman AS, Grillon C. Visual memory processes in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders* 18:601–615. [PubMed: 3215886]
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th Ed.). Washington, DC: American Psychiatric Association.
- Bachevalier J. Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia* 1994;32:627–648. [PubMed: 8084420]
- Bailey A, Phillips W, Rutter M. Autism: Towards an integration of clinical, genetic, neuropsychological, and neurobiological perspectives. *Journal of Child Psychology and Psychiatry* 1996;37:89–126. [PubMed: 8655659]
- Baron-Cohen S. Autism: A specific cognitive disorder of “mindblindness. *International Review of Psychiatry* 1990;2:79–88.
- Baron-Cohen S, Leslie AM, Frith U. Does the autistic child have a “theory of mind”? *Cognition* 1985;21:37–46. [PubMed: 2934210]
- Baron-Cohen, S., & Swettenham, J. (1998) Theory of mind in autism: Its relationship to executive function and central coherence. In D.J. Cohen & F.R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 880–893). New York: Wiley.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (1993). *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.
- Bartak L, Rutter M, Cox A. A comparative study of infantile autism and specific developmental language disorder: I. The children. *British Journal of Psychiatry* 1975;126:127–145. [PubMed: 1131465]
- Barth C, Fein D, Waterhouse L. Delayed match-to-sample performance in autistic children. *Developmental Neuropsychology* 1995;11:53–69.
- Bauman ML, Kemper TL. Histoanatomic observations of the brain in early infantile autism. *Neurology* 1985;35:866–874. [PubMed: 4000488]
- Bauman, M. L., & Kemper, T. L. (1994). *Neurobiology of autism*. Baltimore, MD: Johns Hopkins University Press.
- Benowitz LI, Moya KL, Levine DN. Impaired verbal reasoning and constructional apraxia in subjects with right hemisphere damage. *Neuropsychologia* 1990;23:231–241. [PubMed: 2325836]
- Bennetto L, Pennington B, Rogers S. Intact and impaired memory functions in autism. *Child Development* 1996;67:1816–1835. [PubMed: 8890510]
- Berg EA. A simple objective technique for measuring flexibility in thinking. *Journal of General Psychology* 1948;39:15–22.
- Bianchi, L. (1922). *The mechanism of the brain and the function of the frontal lobes*. Edinburgh: Livingstone.
- Borys SV, Spitz HH, Dorans BA. Tower of Hanoi performance of retarded young adults and nonretarded children as a function of solution length and goal state. *Journal of Experimental Child Psychology* 1982;33:87–110. [PubMed: 7057138]
- Boucher J, Warrington EK. Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology* 1976;67:73–87. [PubMed: 1268453]
- Bowler DM, Matthews NJ, Gardiner JM. Asperger’s syndrome and memory: Similarity to autism but not amnesia. *Neuropsychologia* 1997;35:65–70. [PubMed: 8981378]

- Brian JA, Bryson SE. Disembedding performance and recognition memory in autism/PDD. *Journal of Child Psychology and Psychiatry* 1996;37:865–872. [PubMed: 8923229]
- Brothers LA. The social brain: A project for integrating primate behavior and neurophysiology in a new domain. *Concepts in Neuroscience* 1990;1:27–51.
- Case R, Kurland DM, Goldberg J. Operational efficiency and the growth of short-term memory span. *Journal of Experimental Child Psychology* 1982;33:386–404.
- Cohen IL. An artificial neural network analogue of learning in autism. *Biological Psychiatry* 1994;36:5–20. [PubMed: 8080903]
- Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Avon Books.
- Damasio AR, Maurer RG. A neurological model for childhood autism. *Archives of Neurology* 1978;35:777–786. [PubMed: 718482]
- Dawson G. Neuropsychology of autism: A report on the state of the science. *Journal of Autism and Developmental Disorders* 1996;26:179–184. [PubMed: 8744481]
- Dawson G, Meltzoff AN, Osterling J, Rinaldi J. Neuropsychological correlates of early symptoms of autism. *Child Development* 1998;69:1276–1285. [PubMed: 9839415]
- Dehaene S, Changeux JP. The Wisconsin Card Sorting Test: Theoretical analysis and modeling in a neuronal network. *Cerebral Cortex* 1991;1:62–79. [PubMed: 1822726]
- DeLong GR. Autism, amnesia, hippocampus, and learning. *Neuroscience and Biobehavioral Reviews* 1992;16:63–70. [PubMed: 1553107]
- Dennis M. Frontal lobe function in childhood and adolescence: A heuristic for assessing attention regulation, executive control, and the intentional states important for social discourse. *Developmental Neuropsychology* 1991;7:327–358.
- Diamond A, Doar B. The performance of human infants on a measure of frontal cortex function, the delayed response task. *Developmental Psychobiology* 1989;22:271–294. [PubMed: 2707496]
- Diamond A, Gilbert J. Development as progressive inhibiting control of action: Retrieval of a contiguous object. *Cognitive Development* 1989;4:223–249.
- Diamond A, Goldman-Rakic PS. Comparison of human infants and rhesus monkeys on Piaget's AB task: Evidence for dependence on dorsolateral prefrontal cortex. *Experimental Brain Research* 1989;74:24–40.
- Diamond, A., Prevor, M. B., Callender, G., & Druin, D. P. (1997). Prefrontal cortex cognitive deficits in children treated early and continuously for PKU. *Monographs of the Society for Research in Child Development* (Vol. 62). Chicago: The University of Chicago Press.
- Diamond A, Towle C, Boyer K. Young children's performance on a task sensitive to the memory functions of the medial temporal lobe in adults – the delayed nonmatching-to-sample task – reveals problems that are due to non-memory-related task demands. *Behavioral Neuroscience* 1994;4:659–680. [PubMed: 7986361]
- Duncan J. Disorganisation of behavior after frontal lobe damage. *Cognitive Neuropsychology* 1986;3:271–290.
- Duncan, J. (1995). Attention, intelligence, and the frontal lobes. In M. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 721–733). Cambridge, MA: MIT Press.
- Elliott, C. D., Murray, D. J., & Pearson, L. S. (1979). *The British Ability Scales*. Windsor: NFER-Nelson Publishing Co., Ltd.
- Feinberg, T. E., & Farah, M. J. (1997). The development of modern behavioral neurology and neuropsychology. In T. Feinberg & M. Farah (Eds.), *Behavioral neurology and neuropsychology* (pp. 3–23). New York: McGraw-Hill.
- Friedrich, F. J., & Rader, S. (1996). Component process analysis in experimental and clinical neuropsychology. In M. Maruish & J. Moses (Eds.), *Theoretical foundations of clinical neuropsychology for clinical practitioners* (pp. 59–79). Hillsdale, NJ: Lawrence Erlbaum.
- Frith, U. (1989). *Autism: Explaining the Enigma*. Oxford: Blackwell.
- Frith U, Happe F. Autism: beyond "theory of mind. *Cognition* 1994;50:115–132. [PubMed: 8039356]
- Frith U, Snowling M. Reading for meaning and reading for sound in autistic and dyslexic children. *Journal of Developmental Psychology* 1983;1:329–342.

- Fuster, J. M. (1997). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe* (3rd ed.). New York: Raven Press.
- Gerstadt C, Hong Y, Diamond A. The relationship between cognition and action: Performance of 3 ½ – 7 year old children on a Stroop-like day-night test. *Cognition* 1994;53:129–153. [PubMed: 7805351]
- Golden, C. J. (1981). The Nebraska-Luria Children's Battery: Theory and formulation. In G. W. Hynd & G. E. Obrzut (Eds.), *Neuropsychological assessment and the school-aged child* (pp. 277–302). New York: Grune & Stratton.
- Gregory, R. L. (1967). *Eye and brain*. New York: World University Library.
- Happé FGE. Wechsler IQ profile and theory of mind in autism: A research note. *Journal of Child Psychology and Psychiatry* 1994;35:1461–1471. [PubMed: 7868640]
- Happé FGE. Studying weak central coherence at low levels: Children with autism do not succumb to visual illusions. A research note. *Journal of Child Psychology and Psychiatry* 1996;37:873–877. [PubMed: 8923230]
- Happé FGE. Central coherence and theory of mind in autism: Reading homographs in context. *British Journal of Developmental Psychology* 1997;15:1–12.
- Happé F. Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences* 1999;3:216–222. [PubMed: 10354574]
- Happé, F. (in press). Parts and wholes, meanings and minds: Central Coherence and its relation to theory of mind. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism and developmental cognitive neuroscience* (2nd ed.). Oxford: Oxford University Press.
- Heilman, K. M., & Valenstein, E. (1985). *Clinical Neuropsychology* (2nd ed.). Oxford: Oxford University Press.
- Hermelin B, O'Connor N. Remembering of words by psychotic and subnormal children. *British Journal of Psychology* 1967;58:213–218. [PubMed: 5588649]
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Oxford University Press.
- Hughes C. Control of action and thought: Normal development and dysfunction in autism: A research note. *Journal of Child Psychology and Psychiatry* 1996;37:229–236. [PubMed: 8682904]
- Hughes C. Executive function in preschoolers: Links with theory of mind and verbal ability. *British Journal of Developmental Psychology* 1998a;16:233–253.
- Hughes C. Finding your marbles: Does preschoolers' strategic behavior predict later understanding of the mind? *Developmental Psychology* 1998b;34:1326–1339. [PubMed: 9823515]
- Hughes C, Russell J. Autistic children's difficulty with mental disengagement from an object: Its implications for theories of autism. *Developmental Psychology*, 1993;29:498–510.
- Hughes C, Russell J, Robbins TW. Evidence for executive dysfunction in autism. *Neuropsychologia* 1994;32:477–492. [PubMed: 8047253]
- Jarrold C, Russell J. Counting abilities in autism: Possible implications for central coherence theory. *Journal of Autism and Developmental Disorders* 1997;27:25–37. [PubMed: 9018580]
- Jolliffe T, Baron-Cohen S. Are people with autism and Asperger syndrome faster than normal on the Embedded Figures Test? *Journal of Child Psychology and Psychiatry* 1997;38:527–534. [PubMed: 9255696]
- Karmiloff-Smith A. Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology* 1997;13:513–524.
- Karmiloff-Smith A. Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences* 1998;2:389–398.
- Kaufman, A. S. (1979). *Intelligent testing with the WISC-R*. New York: Wiley.
- Koffka, K. (1935). *Principles of gestalt psychology*. New York: Harcourt.
- Kohs, S. C. (1923). *Intelligence measurement*. New York: Macmillan.
- Kowalska D, Bachevalier J, Mishkin M. The role of the inferior prefrontal convexity in performance of delayed nonmatching-to-sample. *Neuropsychologia* 1991;29:583–600. [PubMed: 1944863]
- LeDoux JE. Emotion, memory and the brain. *Scientific American* 1994 June;:50–54. [PubMed: 8023118]
- Lezak, M. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

- Lord C, Rutter M, Le Couteur. Autism Diagnostic Interview – Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders* 1994;24:659–685. [PubMed: 7814313]
- Luria, A. R. (1966). *Higher cortical functions in man*. New York: Basic Books.
- Luria AR, Pribram KH, Homskaya ED. An experimental analysis of the behavioural disturbance produced by a left frontal arachnoidal endothelioma (meningioma). *Neuropsychologia* 1964;2:257–280.
- Luria, A. R., & Yudovich, F. (1971). *Speech and the development of mental processes in the child*. Harmondsworth: Penguin Books.
- McEvoy RE, Rogers SJ, Pennington BF. Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry* 1993;34:563–578. [PubMed: 7685360]
- Milner B, Petrides M. Behavioral effects of frontal-lobe lesions in man. *Trends in Neuroscience* 1984;7:403–407.
- Minshew NJ. Brief report: Brain mechanisms in autism: Functional and structural abnormalities. *Journal of Autism and Developmental Disorders* 1996;26:205–209. [PubMed: 8744486]
- Minshew NJ, Goldstein G. Is autism an amnesic disorder? Evidence from the California Verbal Learning Test. *Neuropsychology* 1993;7:209–216.
- Minshew NJ, Goldstein G, Siegel DJ. Neuropsychologic functioning in autism: Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society* 1997;3:303–316. [PubMed: 9260440]
- Minshew, N. J., Sweeney, J. A., & Bauman, M. L. (1997). Neurological aspects of autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 344–369). New York: Wiley.
- Morton, J., & Frith, U. (1995). Causal modelling: A structural approach to development psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Manual of developmental psychopathology*, Vol. 1 (pp. 357–390). New York: John Wiley.
- Moya KL, Benowitz LT, Levine DN, Finklestein S. Covariant defects in visuospatial abilities and recall of verbal narrative after right hemisphere stroke. *Cortex* 1986;22:381–397. [PubMed: 3769493]
- Mundy P, Sigman M, Ungerer J, Sherman T. Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry* 1986;27:657–669. [PubMed: 3771682]
- Ozonoff S. Reliability and validity of the Wisconsin Card Sorting Test in studies of autism. *Neuropsychology* 1995;9:491–500.
- Ozonoff, S. (1997a). Causal mechanisms of autism: Unifying perspectives from an information processing framework. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 868–879). New York: Wiley.
- Ozonoff, S. (1997b). In J. Russell (Ed.), *Autism as an executive disorder* (pp.179–211). Oxford: Oxford University Press.
- Ozonoff S, McEvoy RE. A longitudinal study of executive function and theory of mind development in autism. *Development and Psychopathology* 1994;6:415–431.
- Ozonoff S, Pennington BF, Rogers SJ. Are there emotion perception deficits in young autistic children? *Journal of Child Psychology and Psychiatry* 1990;31:343–361. [PubMed: 2318918]
- Ozonoff S, Pennington BF, Rogers SJ. Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry* 1991;32:1081–1105. [PubMed: 1787138]
- Ozonoff S, Rogers SJ, Pennington BF. Asperger’s syndrome: Evidence of an empirical distinction from high-functioning autism. *Journal of Child Psychology and Psychiatry* 1991;32:1107–1122. [PubMed: 1787139]
- Pennington BF. Genetics of learning disabilities. *Journal of Child Neurology* 1994;10:69–76.
- Pennington, B. F., Rogers, S. J., Bennetto, L., Griffith, E. M., Reed, D. T., & Shyu, V. (1997). Validity tests of the executive dysfunction hypothesis of autism. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 143–178). Oxford: Oxford University Press.
- Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry* 1996;37:51–87. [PubMed: 8655658]

- Pennington, B. F., & Welsh, M. (1995). Neuropsychology and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Manual of developmental psychopathology*, Vol. 1 (pp. 254–290). New York: John Wiley.
- Petrides M. Specialized systems for the processing of mnemonic information within the primate frontal cortex. *Philosophical Transactions of the Royal Society of London: Series B* 1996;351:1455–1462. [PubMed: 8941957]
- Piven J, Arndt S, Bailey J, Havercamp S, Andreasen NC, Palmer P. An MRI study of brain size in autism. *American Journal of Psychiatry* 1995;152:1145–1149. [PubMed: 7625461]
- Robbins TW. Dissociating executive functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London: Series B* 1996;351:1463–1471. [PubMed: 8941958]
- Robbins, T. W. (1997). Integrating the neurobiological and neuropsychological dimensions of autism. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 21–53). Oxford: Oxford University Press.
- Roberts RJ, Pennington BF. An interactive framework for examining prefrontal cognitive processes. *Developmental Neuropsychology* 1996;12:105–126.
- Rumsey JM. Conceptual problem-solving in highly verbal, nonretarded autistic men. *Journal of Autism and Developmental Disorders* 1985;15:23–36. [PubMed: 3980427]
- Russell, J. (1997). How executive disorders can bring about an inadequate ‘theory of mind.’ In J. Russell (Ed.), *Autism as an executive disorder* (pp. 256–304). Oxford: Oxford University Press.
- Russell J, Jarrold C, Henry L. Working memory in children with autism and with moderate learning difficulties. *Journal of Child Psychology and Psychiatry* 1996;37:673–686. [PubMed: 8894948]
- Russell, J., Jarrold, C., & Hood, B. (in press). Two intact executive capacities in children with autism: Implications for the core executive functions in the disorder. *Journal of Autism and Developmental Disorders*.
- Russell J, Mauthner N, Sharpe S, Tidswell T. The ‘windows task’ as a measure of strategic deception in preschoolers and autistic subjects. *British Journal of Developmental Psychology* 1991;9:331–349.
- Sandson J, Albert ML. Varieties of perseveration. *Neuropsychologia* 1984;22:715–732. [PubMed: 6084826]
- Shah A, Frith U. An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry* 1983;24:613–620. [PubMed: 6630333]
- Shah A, Frith U. Why do autistic individuals show superior performance on the Block Design task? *Journal of Child Psychology and Psychiatry* 1993;34:1351–1364. [PubMed: 8294523]
- Shallice T. Specific impairments of planning. *Philosophical Transactions of the Royal Society of London, Series B* 1982;298:199–209. [PubMed: 6125971]
- Shallice, T. (1988). *From neuropsychology to mental structure*. Cambridge, England: Cambridge University Press.
- Stone, V. E. (in press). The role of the frontal lobes and the amygdala in theory of mind. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism and developmental cognitive neuroscience* (2nd ed.). Oxford: Oxford University Press.
- Tager-Flusberg H. Semantic processing in the free recall of autistic children: Further evidence for a cognitive deficit. *British Journal of Developmental Psychology* 1991;9:417–430.
- Takeuchi AH, Hulse SH. Absolute pitch. *Psychological Bulletin* 1993;113:345–361. [PubMed: 8451339]
- Turner, M. (1997). Towards an executive dysfunction account of repetitive behavior in autism. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 57–100). Oxford: Oxford University Press.
- Wechsler, D. (1997). *Wechsler Adult Intelligence Scale – Third Edition*. San Antonio, TX: The Psychological Corporation.
- Wechsler, D. (1991). *Wechsler Intelligence Scale for Children – Third Edition*. San Antonio, TX: Psychological Corporation.
- Wehner, E., & Rogers, S. (1994). *Attachment relationships of autistic and developmentally delayed children* Paper presented at the bi-monthly meeting of the Developmental Psychobiology Research Group, Denver, CO.
- Welsh MC, Pennington BF, Groisser DB. A normative-developmental study of executive function: A window on prefrontal function in children. *Developmental Neuropsychology* 1991;7:131–149.

- Wimmer H, Perner J. Beliefs about beliefs: Representation and constraining function of wrong beliefs in children's understanding of deception. *Cognition* 1983;13:103–128. [PubMed: 6681741]
- Witkin, H. A., Oltman, P. K., Raskin, E., & Karp, S. A. (1971). *Manual for the Embedded Figures Test*. Palo Alto, CA: Consulting Psychologist Press, Inc.
- World Health Organization (1994). *International classification of diseases* (10th ed.). Geneva: World Health Organization.
- Yirmiya N, Solomonica-Levi D, Shulman C, Pilowsky T. Theory of mind abilities in individuals with autism, Down syndrome, and mental retardation of unknown etiology: The role of age and intelligence. *Journal of Child Psychology and Psychiatry* 1996;37:1003–1014. [PubMed: 9119934]