The autistic mind in the light of neuropsychological studies

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The underlying causes of autism spectrum disorders have not yet been identified. There is a group of theories which attempt to explain the source of difficulties experienced by people with these disorders through neurocognitive deficits. The most popular among them refer to deficits in theory of mind, executive dysfunction and weak central coherence. The results of over 20 years of research show that although these deficits may at least partially explain variations in the course of social development, communication difficulties and the formation of rigid patterns of behavior and interests in individuals with autism, none of them is sufficient on its own to comprehensively explain the complex picture of autistic disorders. Information about the neurobiological correlates of these deficits complement our knowledge about their mutual relationships by showing that multiple neural networks are involved in the emergence of autism’s signs and symptoms. Further investigation of various aspects of these cognitive deficits and the relationships between them may significantly improve our understanding of processes involved in the development of autism.

Key words: autism, theory of mind, weak central coherence, executive function

INTRODUCTION

Diagnosis of autism is still based on behavioral criteria, since its biological causes are yet to be determined. The autistic triad of impairments comprises social development disorders, limited verbal and non-verbal communication skills, and repetitive behaviors and restricted interests (WHO 1992, APA 2000). The most characteristic core problems are difficulties with social communication, initiating and sustaining interaction (Volkmar et al. 2004). Individuals with autism spectrum disorders do not use eye contact, facial expressions or gestures to control interactions with others. They are incapable of sharing their interests and feelings, and do not form age-appropriate social relationships. They also experience speech development problems, with many of them not speaking at all (Tager-Flusberg et al. 2005). Typically affected areas of speech include modulation, rate, rhythm, grammar, restricted and unevenly developed vocabulary, and echolalia.

A number of individuals with autism are diagnosed with intellectual disability (see Fombonne 2003), even though deficits in social functioning render an accurate assessment of their intelligence difficult. Some authors have posited the existence of a unique intellectual profile associated with autism, with non-verbal skills developed better than verbal communication (Barnhill et al. 2000, Joseph et al. 2002), a relatively good development of visuo-spatial skills and poor grasp of social situations (Happé 1994, Goldstein et al. 2001). Their claims, however, are undermined by significant individual differences in the development of various skills in individuals diagnosed with autism. Research on the subject has brought relatively few findings which could be applied to support individuals with autism. Of much more value in clinical practice is information about cognitive deficits contributing to the emergence of certain functional difficulties. Some models refer to such deficits to explain difficulties with participation in social interactions and forming relationships with other characteristics for autism. The most important of those deficits affect theory of mind, executive function and central coherence.

THEORY OF MIND DEFICITS

According to the concept of deficits in theory of mind, the difficulty experienced by individuals with autism in social interactions and communication are
closely related to problems of neurocognitive origin which impair intuitive grasp of phenomena occurring in the mind (Hill and Frith 2003). The term "theory of mind" (Premack and Woodruff 1978) refers to the ability to explain and predict behavior by hypothesizing on the thoughts, feelings and goals of partners in social interaction. Individuals with autism are unable to correctly recognize mental states (e.g. the state of knowledge, intentions, desires and beliefs) and, consequently, cannot use that information to predict other people’s behavior and motives. Their ability to use this knowledge to interpret social situations is also compromised. These deficits are associated with the ability to form mental representations and metarepresentations (Egget and Kurzban 2008), although there is no consensus on the nature and scope of this relationship (e.g. Stone and Gerrans 2006).

In one of the first publications on the subject, Baron-Cohen and coauthors (1985) suggested that theory of mind deficits were caused by a defective innate cognitive mechanism which makes it possible to imagine what goes on in the mind. A classic example of a diagnostic tool used in this domain is the Sally-Anne Test (Wimmer and Perner 1983). Participants are presented with the following story: Sally, one of two dolls, has a ball, which she puts it in the basket and covers, and then goes away. While she is away, the other doll, Anne, moves the ball from the basket to a box. Participants are asked to predict where Sally will look for her ball when she comes back. The correct solution requires the participant to take into account what Sally knows. Therefore the participant has to assume the perspective of one of the dolls and ignore his/her own knowledge about the actual location of the ball. Although most typically developing children with mental age of 4 to 6 years can solve this problem, as few as 20% of children with autism with mental age of 5.5 years give the correct response (Baron-Cohen et al. 1985).

The hypothesis of theory of mind mind deficits with autism and their relationship with difficulties in social interactions and communication has been tested in a number of creative experiments (see Frith 2003). They found the presence of difficulties in reading mental states (e.g. Baron-Cohen et al. 1999, Castelli et al. 2002, Beaumont and Sofronoff 2008, Kaland et al. 2008).

Theory of mind deficiencies are probably compounded by low preference of individuals with autism for social stimuli, including the human face (Dawson et al. 2004). Eye tracking studies (Speer et al. 2007) have demonstrated that individuals with autism watching a conversation between several people tend to look less frequently at their eyes, focusing on the movements of mouths and hands. Similar differences were found between 2-year-old children with autism, their typically developing peers and children with developmental delay (Jones et al. 2008). In this study, children watched a video showing a close-up of the face of an actress trying to involve them in conversation. There was a negative correlation between the time children looked into her eyes and the extent of their social development deficits: the greater the problems, the shorter the time of looking to the eyes. Psychophysiological data have also confirmed the atypical functioning of individuals with autism in terms of face processing processes (Critchley et al. 2000, Schultz et al. 2000, Pierce et al. 2001). During this type of behavior, their brains showed no activity in the areas associated with face-specific information processing, i.e. the face area of the fusiform gyrus. However, Rutherford and Towns (2008) found no differences between adults with autism and adults functioning typically in terms of the amount of time spent looking to the eyes and at the faces of people on photographs. Thus, the results of research in this field are inconclusive and certainly influenced, for example, by the type of stimulation used (e.g. dynamic vs. static).

We should mention here interesting reports on biological motion tracking in small children with autism (Klin et al. 2009). They were unable to follow moving points of light mimicking the movements of living creatures, while being highly sensitive to physical stimuli produced accidentally during this simulation. Since neural mechanisms underlying the perception of biological motion involve the same brain regions that are active in the perception of basic social cues, e.g. facial expression and eye tracking, this finding could be of vital importance for the understanding of difficulties experienced by individuals with autism. Directing one’s attention towards biologically significant movement is considered to be the prerequisite for the ability to recognize other people’s intentions, and consequently, the development of theory of mind.

The issue of human face perception is associated with problems recognizing emotions. The majority of individuals with autism can recognize basic emotions: happiness, anger, sadness, fear, disgust, and surprise (Castelli 2005), but they fare much worse with complex
states, e.g. shame or jealousy (Bauminger 2004). This is due to the fact that to interpret such emotions one needs to use knowledge about social context and the resulting mental state of the observed person. Decoding that information, especially taking context into account, is impossible for most individuals with autism. These problems are not limited to recognition of emotions. Even if children with autism are able to identify emotions, they fail to use this information spontaneously to interpret (Begeer et al. 2006) and anticipate the behavior of others (e.g. Ruffman et al. 2001).

In addition, individuals with autism find it difficult to identify and name their own emotions. This information comes from studies using alexithymia and empathy questionnaires (Hill et al. 2004), as well as functional magnetic resonance imaging (fMRI) during participants’ introspection of their own emotional state (Silani et al. 2008). In the assessment of one’s feelings arising when looking at unpleasant pictures (e.g. showing a dog bearing its teeth menacingly), there is a strong association between the ability to read emotions and brain activity in the anterior insula region. Activity in this region is associated with the induction of positive and negative feelings, e.g. through unpleasant or painful stimulation (cf. Craig et al. 2000). Introspection activates the same brain regions in individuals with autism as in typically functioning individuals during self-reflection and attribution of mental states, but the activity is significantly weaker (Silani et al. 2008). These low-activity regions were: anterior cingulate cortices, frontal inferior orbital cortex, medial parieto-frontal cortex, precuneus, left temporal pole and cerebellum. On the other hand, posterior regions, especially the parietal and occipital cortex, were activated to a much larger extent.

It should be pointed out, however, that results of some recent studies have demonstrated that the development of the ability to identify and categorize emotions in individuals with autism may be much better than earlier reports suggested (e.g. Homer and Rutherford 2008). The latest findings show that they process face-specific information in a comprehensive manner, without focusing on the analysis of a selected part of the face. These findings present a challenge to the well-established notion of poorer global processing of information discussed further in the text and should certainly be followed up in further research.

Theory of mind deficiencies may be closely related to the difficulties in establishing social relations and in communication experienced by individuals with autism, especially with pragmatic impairments of language and communication (Miller 2006, Peterson et al. 2007, Colle et al. 2008). However, there is a lot of variation among individuals with autism in terms of the level of development of skills needed to solve theory of mind tasks. It depends on mental and language development (Colle et al. 2007), as well as a number of other factors (Ronald et al. 2006). Theory of mind develops at a slower rate in individuals with autism, and a significant proportion of them are unable to solve tasks which present no problems to their typically developing peers of the same mental age (Happé 1995). Difficulties in this area are also experienced by adults with autism, who, for example, perform much worse than typically functioning individuals in attributing mental states based on vocalization (Rutherford et al. 2002, Golan et al. 2007). Moreover, even those who correctly solve some tasks requiring the use of theory of mind experience significant problems in understanding social phenomena and developing skills necessary in social interaction. On the other hand, these difficulties do not apply to all tasks involving knowledge about mental states. Peterson (2005) demonstrated that the majority of high-functioning children with autism aged 6–13 years were able to correctly describe the functions of the brain and mind. They referred to the psychological and physiological functions of the brain, e.g. “brain is for thinking” or “brain sends nerve messages around the body” (p. 491). In a review of research, C. Hamilton (2009) concluded that the results of some studies suggest that children with autism are quite capable of understanding intentions and goals of behavior. Besides, theory of mind disorders cannot account for the emergence of restricted patterns of behavior and interests, obsessive preference for sameness, as well as preoccupation with parts of objects (Frith and Happé 1994, Levy 2007).

Researchers conducting neuroimaging studies on normally functioning individuals were able to identify regions of the brain activated when subjects were solving tasks related to attributing mental states. These include the medial prefrontal cortex (anterior paracingulate cortex), the temporal–parietal junction and the temporal poles (Brunet et al. 2000, Castelli et al. 2000, Gallagher et al. 2000, Sabbagh and Taylor 2000, Vogeley et al. 2001). Studies on deception detected that attempted deception is associated with activation in the prefrontal and anterior cingulate cor-
tices, probably attributable to the fact that deception was a purposeful and intentional act in these studies, regardless of various methods of experimental protocols (Spence et al. 2004). While performing tasks involving inference of mental states, individuals with autism showed less extensive activation in frontal regions and in the amygdala (Baron-Cohen et al. 2000, Frith 2003), i.e. in those regions that are activated in typically developing people. Some studies have also indicated that the skills involved in the inference of mental states are not only associated with lower activation in frontal regions (lateral inferior frontal cortex), but also with increased activity in the temporal region (Happé et al. 1996, Frith 2003). Frith and Frith (2008) found that during a game in which participants could make decisions potentially enhancing or lowering their reputation in the eyes of their partner, individuals with autism lacked the expected brain activation in the mid cingulate cortex region, which could suggest that they lacked awareness of provoking a particular opinion in a partner.

As suggested by Sabbagh (2004) in a comprehensive review, the ability to make inferences about mental states is likely to be related to several anatomically and functionally separate neural circuits. The ability to recognize mental states based on observable cues, e.g. facial expression, is associated with the operation of the orbitofrontal/medial temporal circuit within the right hemisphere, while inferring about others’ mental states is associated with left medial frontal regions. Walenski and others (2006), have noted that the pattern frequently described in research studies, involving decreased activity in Broca’s area I and increased activity in posterior superior temporal/temporo-parietal cortex is similar to the results obtained in neuroimaging studies on the use of language in individuals with autism. This could validate the search for a common basis for social difficulties and use of language impairments in the neurocognitive deficits related to the theory of mind.

Scholz and colleagues (2009) tested, if the theory of mind and orienting attention engage the same cortical region in the right temporo-parietal junction (RTPJ). They found, utilizing advanced methodology, that these processes recruit neighboring but distinct cortical regions near the RTPJ.

Some authors have indicated that inhibitory control disorders related to data selection skills could be a factor related to poor performance of individuals with autism in standard false-belief tasks, e.g. Sally-Ann test (Leslie et al. 2004). Yang and coworkers (2009) found that the scores obtained in theory of mind tasks were correlated with performance in executive function tasks, especially those involving inhibitory control. Any defects in these mechanisms may have a significant effect on the process of social learning, inhibiting identification of socially salient stimuli in the background context. With this interpretation our attention is shifted towards cognitive processes, which are at the core of the two hypotheses discussed below.

EXECUTIVE DYSFUNCTION

The theory of mind deficits hypothesis assumes that the underlying cause of many problems of individuals with autism are cognitive deficits directly involved in social functioning. There is an alternative explanation, which points to executive function deficits as the source of various difficulties experienced by individuals with autism. This term refers to a broad spectrum of neuropsychological processes underlying physical, cognitive and emotional self-control (Corbett et al. 2009). It covers a complex set of brain functions, including action planning, maintaining the level of cognitive stimulation, focusing on tasks and shifting attention, monitoring performance level, using feedback, and ignoring external context and flexibility in adjusting activity to changes in setting.

People with autism, similarly to individuals with prefrontal cortex damage, were found to have difficulty with goal-directed behavior planning and execution resulting from lack of flexibility in thinking and behavior, a tendency to perseverate and focus on details while ignoring context, impulsiveness, difficulty in switching to a new task and ignoring stimulation unrelated to solving a particular problem (Ozonoff 1997). Results have found disorders in this group involving response inhibition, maintaining and shifting attention and in working memory, all of which are key components of performance control (Luna et al. 2007, Sanders et al. 2008, Robinson et al. 2009). There are also reports of abnormalities in state regulation (Raymaekers et al. 2004).

Some development of executive function in children with autism occurs between childhood and adolescence (Luna et al. 2007, O’Hearn et al. 2008). Behavioral plasticity improves, but differences in the level of functioning compared to typically developing individuals are present at all ages.
Attention shift and response inhibition problems are probably associated with ritualized and rigid behavior (Pietrefesa and Evans 2007). Knowledge about the type of problems experienced by individuals with autism due to executive function deficits is helpful in setting up a suitable living environment and selecting support strategies. We know that these individuals function better in clearly structured situations, which follow a predefined pattern and occur in a specific place and at a specific time. Explaining the relationship between these deficits and problems in social interactions presents a much tougher challenge.

In all likelihood, executive function are associated with large brain networks that comprise multiple structures, which lead to integration of processes responsible for processing complex information. As pointed out by Sanders and others (2008), there have been relatively few studies investigating neural correlates of attention focusing/shifting and response inhibition in individuals with autism. Research into the integrity of brain systems has found many grey and white matter irregularities in people with autism which have adverse effects on functional integration (O’Hearn et al. 2008). In inhibition tasks, they demonstrate less brain activity in anterior cingulate cortex (Kana et al. 2007). It should be mentioned that individuals with autism have also been diagnosed with frontal cortex abnormalities (Just et al. 2004). They were shown to exhibit greater extent of activation in premotor areas in inhibition and working memory tasks (Kana et al. 2007). Apart from differences in activation, they also have poorer synchronization of the inhibition network (anterior cingulate gyrus, middle cingulate gyrus, and insula) and frontal and parietal regions. Planning and working memory abnormalities may also be related to dysfunctions in the corpus callosum, which is smaller in individuals with autism than in typically developing people (Keary et al. 2009).

There are still gaps in our understanding of executive function deficits in individuals with autism. One particularly controversial issue concerns reaction inhibition skills. Some studies suggest that high-functioning children with autism have difficulties with planning and thinking fluently, but perform better than children with ADHD when it comes to inhibition (Happé et al. 2006). A comparison between children with autism, children with specific language impairments and children with pragmatic language disorders also found no clear relationship between inhibition deficits and autism symptoms (Bishop and Norbury 2005). These deficits were rather associated with poor verbal skills (often found in children with autism, but not exclusive to them) and attention deficits. From these findings we could conclude that inhibition disorders are associated with relatively mildly affected executive functions in individuals with autism (Ozonoff and Strayer 1997). However, this conclusion is put into question by other reports. For example, Geurts and colleagues (2004) found significant impairments of response inhibition in children with autism. Inhibition studies using the Wisconsin Card Sorting Test have demonstrated that individuals with autism are able to switch to a new card sorting procedure, but find it difficult to retain a given sorting strategy in memory for a whole session (Kaland et al. 2008). Motivation also plays its part in solving tasks involving executive functions. In a classic “go/no go” study conducted to investigate response inhibition, performance of children with autism improved significantly when money was used as a reward (Kohls et al. 2009).

A popular opinion is that executive function deficits could have underlying genetic causes, responsible for the presence of similar difficulties (although not directly related to autistic symptoms) in close relatives of individuals with autism. This is supported by the results obtained by Delorme and coauthors (2007), who found difficulties in planning and working memory in parents and siblings of people with autism.

In terms of executive function deficits, children with Autism Spectrum Disorders share many features with children with ADHD (Raymaekers et al. 2007, Corbett et al. 2009). Analyses of performance in various cognitive tasks have demonstrated that although both groups have attention and performance control deficits, they are not identical (e.g. Johnson et al. 2007). However, it would be difficult to pinpoint the exact pattern of those disorders typical for autism, especially since some studies found no deficits in terms of planning, attention shifting and inhibition in high-functioning adults with autism (Bogte et al. 2008).

We can therefore conclude that the presence of executive function deficits in individuals suffering from disorders other than autism (e.g. ADHD) calls into question their unique role in the emergence of social functioning and communication difficulties typical for autism. It should be remembered, though, that the term “executive functions” and the set of
some results (e.g. López et al. 2008) show that the term “central coherence” may comprise a whole host of complex and heterogeneous phenomena. Global processing skills vary among individuals with autism spectrum disorders (Ropar and Mitchell 2001). Not all individuals with autism have problems in this area, although they are more prevalent in this population than in typically developing individuals (Teunisse et al. 2001). Another finding has been that individuals with autism are capable of processing information globally when properly instructed, but without such direction their processing is predominantly local (Rinehart et al. 2000). Their performance is largely dependent on the type of task (e.g. Molesworth et al. 2005).

The role of problems in global processing of information in the development of social difficulties is unclear. Some studies found no relationship between weak central coherence and severity of autism’s signs and symptoms, understanding of social situations and social competence (Teunisse et al. 2001). Burnette and colleagues (2005) investigated the links between central cohesion and theory of mind. Although the results showed a weak correlation between the two variables, there was no relationship between central coherence and social and emotional functioning of individuals with autism. Findings of studies on the relationship between central coherence and rigid behavior patterns are also inconsistent. Some authors found no such relationship (South et al. 2007), while others reported a relationship between weak central coherence and propensity for repetitive, ritualistic behavior and persistent interests (Chen et al. 2009). At the same time, there is no doubt that impaired global processing of information, particularly in terms of using context to decode meanings, may have a significant impact on communication (e.g. Noens and van Berckelaer-Onnes 2004).

Neural mechanisms underlying weak central coherence in individuals with autism are not yet known. The poor performance of these people in global processing tasks has been explained either by reference to abnormalities in regions of the brain specialised in processing specific stimulation used in a given task or, more commonly, by positing the existence of diffuse changes in neural connectivity (Happé and Frith 2006). It is supposed that poorly developed global processing of information may be associated with reduced (weakened) structural or functional connectivity between various regions of the brain, enabling
affected individuals to perform well when processing focused information, but restricting their ability to generalize (White et al. 2009). Another hypothesis points to reduced integration of specialized neural networks caused by a deficit in temporal binding (Brock et al. 2002). Its authors assume that the anomalies in visual perception associated with central coherence may be caused by reduced synchronization of high-frequency gamma activity between local networks processing local features. The right hemisphere is the brain part implicated in global processing and integration of information (Happé and Frith 2006). Mevorach and others (2005) demonstrated that the right posterior parietal lobe plays an important role in the perception of global aspects of hierarchical figures in right-handed people. There is still little known however, about specific right hemisphere abnormalities in individuals with autism (e.g. Waiter et al. 2005).

Happé and Frith (2006) note that preference for local vs. global processing of information may be determined genetically. Parents of children with autism perform better than controls in tasks requiring participants to find an embedded figure (Bölte and Poustka 2006). It was also found that fathers, similarly to their children with autism, demonstrate a local style of information processing (Happé et al. 2001). This could confirm the hypothesis that the development of cognitive profiles associated with autism is genetically predisposed. Reduced central coherence is seen not only as a deficit, but also as a feature of information processing (see: Happé and Frith 2006). Individuals with the so-called weak coherence (e.g. individuals with autism and some of their relatives) are at one extreme of the continuum, characterized by the detail-focused perception style, with the other extreme occupied by people with strong coherence, whose cognitive style is dominated by global, comprehensive processing of information.

It should be noted that there is much inconsistency in the results of studies on the abilities of individuals with autism. Not all reports have confirmed, for example, superior development of visuospatial skills, i.e. a potential argument for the weak central coherence hypothesis, in high-functioning children with autism or Asperger’s syndrome compared with their typically developing peers (Kaland et al. 2007).

**CONCLUSIONS**

Cognitive deficits discussed in this paper may offer clues to a better understanding of difficulties experienced by individuals with autism. None of them, however, can answer the question why disorders in social development and communication coincide with rigid behavior patterns in autism.

The role of these deficits in the emergence of the signs and symptoms of autism is also unclear. The results of studies on the relationship between cognitive characteristics (theory of mind, executive function and central coherence) and the autistic triad of impairments show that these characteristics may be predictors of behavioral signs of autism, such as social impairments and communication deficiencies, and rigid patterns of behavior and interests (e.g. Best et al. 2008). Thus, they may suggest that mild autistic features, more prevalent in the general population than the number of people diagnosed with autism would suggest, may have the same cognitive basis as the problems experienced by individuals with autism. Although individual differences in social functioning and communication among people with autism may be explained, at least to some extent, by the severity of deficits in the theory of mind, still, as noted by Tager-Flusberg (2007), after over 20 years of research on the subject we know that social and emotional difficulties of these people are by no means limited to the development of theory of mind. The precursors of theory of mind include joint-attention and executive functions (Belmonte 2009). The extent of impairments in these domains may determine the scope and severity of difficulties in mental state attribution. However, precise determination of relationships between these deficits (and central coherence) is particularly challenging. As pointed out by Happé and Frith (2006), the term “executive functions” may encompass, at least to some extent, the skills associated with weak central coherence, and the relationship between theory of mind and weak central coherence is unclear (Jarrold et al. 2000, Beaumont and Newcombe 2006).

An important question to ask is whether autism can develop in a person with no theory of mind, executive function or central coherence impairments. While the answer could be affirmative for each deficit independently (especially if we take into account their selected features), there is no readily available data to suggest that some individuals with autism demonstrate typical devel-
opment in all these domains. Thus, although the contribution of cognitive deficits in the development of social difficulties in individuals with autism may sometimes be overstated, there is no doubt that the investigation of their various aspects and the relationships between them may offer new insights into the processes involved in the emergence of autism. We still need to find out whether individual differences in the scope and severity of these deficits may be treated as a criterion for identifying subtypes in autism spectrum disorders, as well as to determine the role their neural correlates could play in this process.

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