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BRIEF REPORT

Brief Report: Atypical Social Cognition and Social Behaviours in Autism Spectrum Disorder: A Different Way of Processing Rather than an Impairment

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Abstract A central question to autism research is whether autism is largely the result of an impairment in social cognition and/or motivation or the result of a more general processing difference. This review discusses problems with the "social deficit" model of autism spectrum disorder (ASD). It is suggested that superior attention to low-level perceptual information potentially coupled with decreased attention to global information may provide a more comprehensive explanation for atypical social behaviours in ASD. This processing style may reflect increased activation of occipital-temporal regions and reduced functional (and possibly anatomical) connectivity. It is concluded that atypical social behaviours in ASD are more likely to be a consequence reflective of a general processing difference than impairment in social cognition and/or motivation.

Keywords Autism · Asperger's syndrome · Low-level perceptual processing · Central coherence · Social cognition

Introduction

The Social Deficit Hypothesis of Autism Spectrum Disorders (ASD)

When meeting a person with ASD for the first time one typically notices differences in their social behaviour: an unusual eye gaze (which may be reduced or over-extended), a limited use of facial expression and gesture and when present, atypical speech which tends to lack expression, have an unusual rhythm and may appear slightly stilted or 'robotic' (Asperger 1944; Attwood 1998; Kanner 1943).

Some researchers have suggested that autism may result primarily from a deficit and/or impairment in social motivation and/or cognition (Dawson et al. 2005; Klin et al. 2003; Schultz 2005). They suggest that investigators should be focussing on dysfunction in brain regions implicated in processing social information to reveal the underlying pathology of ASD. Several explanations have been postulated as support for this theory.

Firstly, in comparison to other conditions, multiple and specific impairments in social cognition and reciprocity are required for diagnosis of an Autistic Spectrum Disorder (APA 1994; WHO 1992). Other features of ASD such as repetitive behaviours and sensory sensitivities have been observed in individuals with severe mental retardation (Bodfish et al. 2000) and institutionally deprived children (Beckett et al. 2002; O'Connor et al. 2000). Moreover, delayed language onset is not only observed in Autistic Disorder, but also in William's syndrome (Singer-Harris et al. 1997) and individuals with specific language impairment (Tager-Flusberg and Cooper 1999).

Secondly, differences in social behaviour are generally observed in ASD prior to the emergence of other autistic symptoms. For example, longitudinal studies have shown that joint attention and other measures of reciprocal behaviour are reduced in individuals with ASD prior to age two when language typically starts to develop (Mundy and Gomes 1998; Mundy et al. 1990; Siller and Sigman 2002). Similarly, social differences in ASD are usually observed before the appearance of repetitive behaviours (Schultz 2005).

However, although early differences in social behaviour appear to be a key component of ASD there are several problems with primarily using a social deficit model to

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explain the underlying behavioural, pathological, anatomical and physiological basis of autism. This paper will examine some of the difficulties that have arisen from this hypothesis and will discuss alternative explanations for social differences and/or difficulties in ASD.

Problems With the Social Deficit Hypothesis

The present autism literature suggests that there is currently not enough support for the theory that ASD is primarily a disorder of impaired social cognition and/or motivation.

Firstly, many studies have found that individuals with ASD differ from typically developing controls in the way they process non-social stimuli. This shows that the symptoms of ASD are much broader than can be explained by a social deficit model. For example, both adults and children with ASD tend to perform better than controls on the Embedded Figures Task (EFT) which requires identification of a target within a complex pattern (Jolliffe and Baron-Cohen 1997; Morgan et al. 2003; Ropar and Mitchell 2001; Shah and Frith 1983). Subjects with ASD also tend to be more proficient at arranging blocks to reconstruct an original design in the Block Design Task (Ropar and Mitchell 2001; Shah and Frith 1993) and at detecting local relative global information when attention is not directed to hierarchical stimuli in the Navon Figures Task (Plaisted et al. 1999). A similar superiority for processing local information in ASD has been found in the auditory domain (Mottron et al. 2000). In addition to these findings, individuals with ASD often have an excellent (and sometimes exceptional) memory for facts and are more likely to excel at rote-learning than typically developing individuals (Attwood 1998; Frith 1989; Grandin 1995; Vermeulen 2001).

Secondly, it is difficult to use neuroimaging data obtained from individuals with ASD during participation in face processing tasks as evidence for a social impairment. Although a number of fMRI, MEG and EEG studies have found evidence for face processing differences between individuals with ASD relative to typically developing controls (Bailey et al. 2005; Dalton et al. 2005; Hubl et al. 2003; McPartland et al. 2004; O'Connor et al. 2005; Pierce et al. 2001; Schultz et al. 2000; see Jemel et al. 2006 for reviews), relatively few neuroimaging and neurophysiological studies have compared processing of faces and non-social stimuli (such as objects) in the same study. Using MEG, Bailey et al. (2005) found adults with ASD to exhibit smaller M170 amplitudes over right occipito-temporal regions to faces relative to controls. This difference was not observed to geometric patterns or pictures of mugs. McPartland et al. (2004) found adults with ASD to elicit delayed N170 latencies to faces but not to photographs of furniture relative to control subjects in an ERP study. These researchers did not observe any between group differences for N170 amplitudes elicited to faces or furniture. Unlike Bailey et al. (2005), however, the object stimuli used by McPartland et al. (2004) differed from face stimuli in that they were unable to be discriminated at the same subordinate level of categorization. Using fMRI, Schultz et al. (2000) found 14 adults with ASD to exhibit lower activation in the right fusiform gyrus and greater activation in the inferior temporal gyrus to faces in comparison to typically developing controls. Both groups exhibited increased inferior temporal activation to objects. However, although subjects were required to make discriminations between pairs of objects (or faces) at the same subordinate level of categorization in each trial, different types of objects (cars, birds, bottles etc.) were used across trials. Together these findings suggest that more controlled studies are needed to determine if past neurophysiological and neuroimaging differences between ASD and control subjects are specific for face stimuli or are reflective of a more general processing difference.

Thirdly, although past retrospective studies in infants later diagnosed with autism have reported reduced joint attention behaviours and abnormal eye contact (Adrien et al. 1993; Baron-Cohen et al. 1996; Maestro et al. 2002; Osterling and Dawson 1994), it is difficult to know whether these impairments are primarily social since most infant research is based on observation rather than more rigorous means of testing. Given the number of autobiographical accounts written by individuals with ASD which often describe their tendency to be distracted by background sensory information (Birch 2003; Cesaroni and Garber 1991; Grandin 1995; Miller 2003; Sainsbury 2000; Williams 1995), it is plausible that these early differences in social behaviour may serve as a compensatory strategy to filter out excessive sensory information. With the advance of more sophisticated eye-tracking, neurophysiological and neuroimaging techniques, it is hoped that more comprehensive explanations for early social differences in ASD will soon be revealed.

Fourthly, various experiments suggest that individuals with ASD are able to understand social information and the feelings of others. For example, individuals with ASD can solve social reasoning or theory of mind (ToM) tasks to relatively high levels (Bowler 1992; Dahlgren and Trillingsgaard 1996; Happe 1994) and very young children with autism have been found to be able to imitate the intentions of others (Aldridge et al. 2000). Furthermore, a number of research groups have found individuals with ASD can recognize basic facial expressions as accurately as typically developing controls (Adolphs et al. 2001; Baron-Cohen et al. 1997; Braverman et al. 1989; Castelli 2005; Grossman et al. 2000; Ozonoff et al. 1990; Wang et al. 2004). A recent study found toddlers with ASD were as proficient as typically developing controls at recognizing human faces (Chawarska and Volkmar 2007), while Lahaie et al. (2006) found evidence for superior recognition of facial features in adults

with ASD relative to controls. There are also autobiographical accounts from a number of higher-functioning individuals on the autistic spectrum currently working successfully in various professions such as occupational therapy, nursing, general medical practice, teaching and caregiving which require a certain degree of empathy and social understanding (Miller 2003).

Finally, some researchers have suggested that a lack of social motivation may explain abnormal social behaviour in ASD (Dawson et al. 2005). However, this explanation fails to explain why individuals with higher-functioning autism and Asperger's Syndrome (AS) have described a strong desire to make friends and expressed feelings of loneliness in their writings (Birch 2003; Jones et al. 2001; Miller 2003; Sainsbury 2000). In his original document Asperger suggested that individuals with AS are motivated to process social information as indicated by the following statement:

> Just as these children observe themselves to a high degree, so they also often have surprisingly accurate and mature observations about people in their environment (Asperger 1944).

Together, the above arguments suggest that the social deficit hypothesis does not adequately describe the symptomatology of ASD and raises doubts as to the validity of this hypothesis to explain the underlying basis of autism. Moreover, it is difficult to use experimental studies which have not included a non-social control condition as evidence for a social impairment model of ASD. Other hypotheses, such as the idea of enhanced perceptual functioning (Mottron and Burack 2001, Mottron et al. 2006; Plaisted et al. 1998) potentially coupled with weak central coherence (Frith 1989; Happe and Frith 2006) may provide a more accurate means of explaining social problems in ASD.

An Alternative Explanation for Social Difficulties in ASD

The Enhanced Perceptual Functioning theory suggests that ASD may result from enhanced perception of low-level perceptual information and attention to detail. In contrast, the Weak Central Coherence hypothesis proposes that increased attention to detail in ASD is the result of impaired global processing. However, both these theories share the idea that individuals with ASD are superior relative to typically developing subjects at processing detailed information. This idea offers a potential explanation for why people with ASD often perform better than typically developing subjects on cognitive tasks that are solved more efficiently when attention is focussed at the local-level (Happe and Frith 2006). Superior processing of details may explain other facets of ASD such as literal interpretation of events, narrow interests, savant skills, and analytic thinking (Mottron et al. 2006; Vermeulen 2001).

It is possible that enhanced perception of details in ASD may result in reduced attention to global information, thus resulting in a decreased tendency to process information in context. This idea is consistent with several past studies which have suggested individuals with autism may have difficulty shifting attention from a local to a global level (Plaisted et al. 1999; Rinehart et al. 2001). Given that successful face-to-face communication requires the ability to simultaneously integrate information from multiple sensory modalities (gestures, facial expressions, gaze, speech, tone of voice, posture etc.) over time, less attention to global information would be particularly detrimental to interpreting social interactions. Evidence for decreased integration of face and voice expressions in ASD has been observed in several studies (Hall et al. 2003; Hobson 1986; Hobson et al. 1988; Loveland et al. 1995; O'Connor et al. 2007). Similarly, the tendency for individuals with ASD to interpret written and verbal language literally rather than according to the context in which it is used (Minshew et al. 1995; Ozonoff and Miller 1996; Vermeulen 2001), may reflect decreased attention to global information. It has even been suggested that the difficulty individuals with ASD appear to have in understanding and using irony (Happe 1994; Leekman and Prior 1994; Martin and McDonald 2004; Wang et al. 2006) may relate to abnormal processing of contextual information rather than an underlying impairment in social cognition (Happe 1994; Wang et al. 2006).

Moreover, enhanced perception of low-level information may explain why individuals with ASD tend to focus more often on insignificant details in the environment that most typically developing individuals do not notice or are able to filter out. Superior attention to detail would make both social and non-social information difficult to process. Past autobiographical accounts written by individuals with ASD frequently describe being distracted by background noise, fluorescent lights, shiny jewellery, bodily movement and odours (Birch 2003; Grandin 1995; Miller 2003; Sainsbury 2000). Moreover, sensory questionnaires completed by parents have revealed atypical sensory responses (including hyper-responsiveness) to sensory stimuli in toddlers (Rogers et al. 2003) and children (Talay-Ongan and Wood 2000; Watling et al. 2001) with ASD relative to typically developing controls. Other studies have found that in comparison to typically developing children, people with ASD tend to match faces according to irrelevant details such as accessories and external features rather than facial expression (Davies et al. 1994; Weeks and Hobson 1987). The tendency to focus on irrelevant information would also be particularly detrimental to listening and communicating with others and may explain why individuals with ASD tend to find it easier to interact with others on a one-to-one basis, in a quiet environment and/or via email (Birch 2003; Sainsbury 2000).

It is possible that atypical social behaviours in ASD may develop as a means to reduce excessive quantities of sensory information. For example several research groups have observed a tendency for individuals with ASD to look less at the eye region of faces (Dalton et al. 2005; Klin et al. 2002; Pelphrey et al. 2002). Past researchers have suggested that this may result from decreased understanding of eye expressions perhaps resulting from a more general impairment in social cognition (Baron-Cohen et al. 2001; Klin et al. 2003). However, the eye region is also very complex due to the fact this region is constantly in motion (blinking, saccadic movements, squinting, upward and downwards movements etc.) and can depict many different expressions, often of very limited duration. The quantity of information transmitted from the eyes may be over-whelming to an individual with ASD, especially in the context of a social situation when individuals are exposed to a wide range of sensory information. This could explain why some individuals with ASD have reported that they find the eye region 'confusing' and/or 'frightening' and that they find it easier to listen to others without making eye contact (Attwood 1998; Birch 2003; Joliffe 1992; Miller 2003; Sainsbury 2000). It is possible that over time this may become a more automatic or 'learned' response so that individuals with ASD naturally look less at the eye region of faces than typically developing subjects, even when face stimuli are static as in a number of past computerized tasks (Dalton et al. 2005; Pelphrey et al. 2002; Spezio et al. 2007).

In summary, although enhanced perception of low-level perceptual information may have its advantages (i.e., savant skills, development of a special interest etc.), this ability may actually become more of a 'disability' when trying to process information that is dependent on global processing. Superior processing of details would potentially result in reduced attention to global information, resulting in a reduced ability to interpret social situations. Moreover, this processing style could result in accumulation of details which would make both social and non-social information difficult to process and could potentially result in 'sensory overload'. Finally, it is possible that behaviours such as reduced eye contact in ASD that appear to be anti-social to an observer are actually a means to reduce the level of available perceptual information in order to facilitate information processing.

A Potential Neuroanatomical Explanation for Social and Non-social Difficulties in ASD

A number of neuroimaging studies have observed enhanced activation of brain regions implicated in processing visual perceptual information (the occipital and occipito-temporal regions) when individuals with ASD process both social and non-social information (see Mottron et al. 2006 for a comprehensive review). In brief, hyperactivation or activation of various occipital and temporal regions has been observed in individuals with ASD relative to typically developing controls during processing of faces (Hadjikhani et al. 2004; Hubl et al. 2003), facial expressions (Critchley et al. 2000), expressive faces and voices (Hall et al. 2003) and mentalizing tasks (Baron-Cohen et al. 1999). Similar findings have been observed in a range of non-social tasks such as an n-back working memory task (Koshino et al. 2005), the Embedded Figures Task (Ring et al. 1999), a visual spatial attention task (Belmonte and Yurgelun-Todd 2003) and during a visual-motor (Müller et al 2003) and a word learning task (Hazlett et al. 2004). An exception to this pattern is the fusiform face area, a region that is especially sensitive to faces which tends to exhibit reduced (Critchley et al. 2000; Hubl et al. 2003; Schultz et al. 2000, 2005; Pierce et al. 2001; Wang et al. 2004) or similar (Hadjikhani et al. 2004; Pierce et al. 2004) activation to faces in individuals with ASD relative to typically developing controls. In comparison to subjects with ASD, typically developing individuals tend to activate more frontal regions and/or exhibit increased frontal activation in a number of social tasks (Baron-Cohen et al. 1999; Castelli et al. 2002; Dapretto et al. 2005; Happe et al. 1996; Nishitani et al. 2004; Ogai et al. 2003) as well as in various tasks which do not require processing of social information (Luna et al. 2002; Ring et al. 1999). Together, these findings suggest that individuals with ASD may depend more on brain regions implicated in processing low-level perceptual information to process both social and non-social information, such as temporal-occipital regions, rather than regions implicated in higher-order processing.

Several researchers suggest that autism may result from an increased number of short-distance, local connections in the brain (required for the processing of detailed information) coupled by a reduced number of long-range or global connections necessary for higher level processes (Belmonte et al. 2004; Courchesne 2004; Courchesne and Pierce 2005). Other researchers have suggested that the possibility of increased local connectivity in the brains of individuals with ASD may be a consequence of reduced or aberrant long-distance connections (Frith 2003; Just et al. 2004). This proposal, which is sometimes referred to as the 'underconnectivity hypothesis' predicts that a processing centre which does not develop adequate connections with a centre that it would typically associate with may start to develop independently, thus having the potential to become hyperspecialized (Just et al. 2004). The idea of increased local connectivity in people with ASD is consistent with evidence for superior low-level perception and increased attention to detail in individuals on the autistic spectrum and may also explain the savant abilities observed in a small percentage of individuals with ASD.

In support of 'underconnectivity' in ASD, several recent functional neuroimaging studies have found evidence for reduced co-ordination among different activated brain regions in individuals with ASD during various cognitive tasks. These studies analysed 'functional connectivity' a measure of the correlation between two brain regions activated over the same time period. One of the first published studies in this area examined functional connectivity using data obtained from PET. These researchers discovered reduced functional connectivity between the extrastriate cortex and superior temporal sulcus in adults with ASD relative to typically developing controls in a task which required attribution of mental states to animated shapes (Castelli et al. 2002). Most research since this time has analysed data obtained from fMRI to examine functional connectivity in autism. Just et al. (2004) found decreased functional connectivity between various brain regions in individuals with ASD relative to typically developing controls during a sentence comprehension task, particularly between fronto-temporal and fronto-parietal regions.

Another fMRI study observed lower functional connectivity between area VI and the inferior frontal cortex in adults with autism during a visuomotor co-ordination task which involved imitating another individual's hand movements (Villalobos et al. 2005). Meanwhile, Koshino et al. (2005) found adults with ASD to exhibit lower functional connectivity between the left inferior parietal and various frontal-parietal regions during an n-back working memory task with letters. An elegant study by Kana et al. (2006a) observed lower functional connectivity between regions of the anterior cingulate cortex and the right inferior parietal, frontal and right middle frontal regions in individuals with autism during a simple response inhibition task. Decreased functional connectivity between frontal and posterior brain regions has also been observed in adults with ASD when processing sentences of high and low imagery content (Kana et al. 2006b), in the Tower of London task of executive functioning (Just et al. 2006) and even during a baseline resting state (Cherkassky et al. 2006). The latter three studies also observed a correlation between the size of the anterior corpus callosum (genu) and connectivity, with smaller genu size related to lower frontal-parietal functional connectivity in individuals with ASD but not typically developing subjects. This suggests that anatomical abnormalities in the corpus callosum may contribute to reduced functional connectivity in ASD.

Interestingly, performance did not differ between groups in several of the above tasks (Kana et al. 2006b; Koshino et al. 2005) and Just et al. (2004) even found ASD subjects to exhibit faster reaction times relative to controls. This suggests that greater connectivity between various brain regions is not necessary to obtain high performance on these tasks. What these experiments do suggest however, is that individuals with ASD often use a different strategy to solve tasks relative to typically developing subjects that depends less on connectivity between brain regions.

Evidence for reduced connectivity in ASD has also been observed at the anatomical level in studies which have examined white matter. Several of these studies used whole brain voxel-based morphometry (VBM), a technique which treats all anatomical regions the same and facilitates the detection of subtle differences between groups. Using this technique, past research has found evidence for reduced white matter in the corpus callosum (Chung et al. 2004) (Waiter et al. 2005) and fronto-temporal regions (Waiter et al. 2005) of adolescents with autism and in the cerebellum (Boddaert et al. 2004; McAlonan et al. 2005), temporal pole (Boddaert et al. 2004) and internal capsule (McAlonan et al. 2005) of children with autism. Other studies have examined fractional anisotropy (FA) data in individuals with ASD. This is a measure obtained from diffusion-tensor imaging (DTI), a magnetic-resonance based method. FA values provide information on the structural integrity of white matter and are determined by variables such as axonal fibre diameter and density, the degree of myelination and the level of fibre tract coherence within a voxel (Basser 1995; Beaulieu 2002). Reduced FA values have been observed in white matter within and near the corpus callosum (Barnea-Goraly et al. 2004; Keller et al. 2006), the pre-frontal cortices, various temporoparietal, temporal and occipitotemporal regions (Barnea-Goraly et al. 2004) and in the internal capsule (Keller et al. 2006) of individuals with ASD relative to typically developing controls. Together, these results provide evidence for disruption of white matter tracts in ASD and anatomical connectivity. It is possible that aberrant white matter tracts may contribute to reduced functional connectivity in ASD, however further research is needed to investigate this.

Courchesne and Pierce (2005) have hypothesized that ASD may result from disrupted maturation of large cortical and cerebellar neurons that take many years to develop to their full structural and functional capacity. This prediction is consistent with evidence for early abnormal cerebral and cerebellar growth in autism (Courchesne et al. 2001, 2003; Carper et al. 2002; Dementieva et al. 2005) and underdeveloped cortical minicolumns in post-mortem autistic brain tissue (Cassanova et al. 2002a, b). Courchesne and Pierce predict that impaired development of large integrative and projecting neurons which project to distant regions of cortex would lead to reduced long-distance connectivity and difficulty integrating information from multiple modalities (sensory, autonomic, emotional, memorial etc.) in order to understand higher-order meaning. This would be especially detrimental to interpreting social situations which require the ability to integrate large quantities of information. However, impaired development

of large inhibitory interneurons would result in decreased inhibition, thus increasing local connectivity. At a cognitive level this may explain why individuals with ASD favour a more analytical processing style, which is biased towards processing details. Further post-mortem research is needed to investigate this hypothesis.

In summary, these findings provide a pattern for heightened activation of occipito-temporal regions, lower activation of frontal regions and reduced functional and anatomical connectivity in ASD. However, the reason for these differences is still a mystery and many questions remain unanswered. For example, is reduced functional connectivity in ASD related to decreased anatomical connectivity? Is reduced functional and/or anatomical connectivity of long-range connections in ASD associated with increased local connectivity or 'hyperspecialization' of neural regions? How does the Courschesne and Pierce (2005) hypothesis relate to connectivity differences between autistic and typically developing subjects and the increased occipital/decreased frontal activation pattern observed in ASD? What is the cognitive basis for these neuroanatomical differences? It is hoped that more detailed neuroanatomical examination of the brains of individuals with ASD will one day provide answers to some of these questions.

Summary

In conclusion, this review argues that, at present, there is limited behavioural and neurological evidence in support of the view that ASD is largely the result of a social deficit. Other hypotheses, such as the Enhanced Perceptual Functioning theory coupled with the Weak Central Coherence theory may provide a better explanation for atypical social behaviour in ASD. Superior attention to low-level perceptual information may potentially result in less attention to global information, accumulation of irrelevant details and sensory overload, all of which would make social interactions particularly difficult to interpret. Atypical social behaviours may serve as a means to decrease excessive sensory information. At a neurological level, increased attention to low-level perceptual information may reflect increased activation of occipital-temporal regions while less attention to global information may reflect reduced functional (and possibly anatomical) connectivity and possibly even dysfunction of large integrative neurons. Further research is needed to examine how these neurological differences relate to social cognition and behaviour in ASD. Taken together, these findings suggest that although more research is needed to examine the cognitive and neurological correlates underlying social functioning in autism, atypical social behaviour in ASD is more likely to be the consequence of a processing difference rather than a social deficit.

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