

LITERATURE REVIEW

Disrupted Cortical Connectivity as an Explanatory Model for Autism Spectrum Disorder

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The aim of this article is to explain the theory of Disrupted Cortical Connectivity and discuss whether or not it can integrate the following three theories: Theory of Mind, Executive Functioning, and Weak Central Coherence that dominate the field of autism spectrum disorder research. Due to a lack of existing literature discussing this potential integration, we have consequentially undertaken such an endeavour. In our opinion, integration appears to be possible since this explanatory model can account for difficulties in both social cognition and executive functioning commonly found in autism spectrum disorder. Moreover, the theory of Disrupted Cortical Connectivity could be described as an extension of the theory of Weak Central Coherence.

Keywords: Autism; ASD; Review; Brain connectivity; Theory of Disrupted Cortical Connectivity; Theory of Mind; Executive Functioning; Weak Central Coherence

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterised by impairment in social communication and restrictive or repetitive behaviours, interests and activities (APA, 2013). Many contesting theories exist within the field of autism research, each individually attempting to explain the basis for ASD (Happé & Ronald, 2008). One theory argues that ASD results from an impaired Theory of Mind. This perspective focuses on deficits in social cognition and suggests that an inability to ascribe mental states to oneself and others represents the central issue in ASD (Baron-Cohen, Leslie, & Frith, 1985). Another theory points to impairment in Executive Functioning as a primary explanation. Executive Functioning describes the higher-level cognitive processes that regulate, control, and manage other cognitive functions. Thus, explanations of ASD based on this theory suggest that executive dysfunction underlies the observable cognitive and social deficits associated with the disorder (Ozonoff, 1997). Finally, a third dominant theory argues that Weak Central Coherence can be used as a basis of explanation. The theory of Weak Central Coherence suggests that a limited ability to integrate representations of stimuli, resulting in a fragmented information-processing style, underlies this neurodevelopmental disorder (Frith, 1989).

Although an extensive number of explanatory theories exist, Theory of Mind, Executive Functioning and Weak Central Coherence are typically the most prevalent in reference books on the subject. However, taken in isolation, none of these can fully account for the entire spectrum

of both social-communicative and non-socially restrictive and repetitive patterns of behaviour seen in individuals with ASD. This has led to Happé and Ronald (2008) questioning whether it is advisable to look at ASD as a unified disorder composed of the core symptoms outlined in the DSM-IV. They argue that the complexity of this neurodevelopmental disorder far exceeds our ability to describe it through a single model. Instead, they suggest that it is caused by numerous isolated factors, which researchers in this area have, essentially, arbitrarily chosen to label as ASD (Happé & Ronald, 2008).

We conceive this problematisation of ASD as a unified disorder as exceptionally interesting, particularly with regards to the changes in the autism diagnosis made in the new DSM-V. Here, Asperger's syndrome, childhood disintegrative disorder, autistic disorder and Pervasive Developmental Disorder Not Otherwise Specified have been replaced by the simple umbrella diagnosis 'autism spectrum disorder' to reflect the state of knowledge about autism (Lord & Jones, 2012; APA, 2013).

Furthermore, within the field of autism research, there have long been attempts to localise the dysfunctions of ASD to specific neural structures. However, no convincing candidate region has been found (Anagnostou & Taylor, 2011). Arguably, such attempts are also quite optimistic considering the complexity of ASD symptoms.

Moreover, a relevant distinction between high and low functioning individuals with ASD is often neglected in the empirical literature. The majority of research has involved experiments conducted with high functioning individuals since difficulties with, for example, compliance and task comprehension may arise when experiments are conducted with low functioning participants. However,

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such findings are often generalised to the whole population of people experiencing ASD symptoms (Stefanatos & Joe, 2008).

A relatively new approach in investigating the neural basis for ASD has been to consider the brain as a system of coordinated components. From this perspective, ASD is not explained as a dysfunction in certain neural structures but as a system-level disorder. This approach was already introduced by Horwitz, Rumsey, Grady, and Rapoport (1988) about twenty-five years ago. It is only with recent improvements in advanced scanning techniques, however, that researchers have been able to provide a more solid basis for the theory of Disrupted Cortical Connectivity as an explanatory model for ASD.

Additionally, this theory strives to incorporate the three, previously mentioned, theories (Just, Cherkassky, Keller, Kana, & Minshew, 2007; Just, Keller, Malave, Kana, & Varma, 2012). In line with Happé and Ronald's (2008) critique of perceiving ASD as a unified disorder, and unlike the earlier mentioned theories which appear insufficient in their ability to solely account for the whole spectrum of ASD, the theory of Disrupted Cortical Connectivity could be considered a potentially unifying, explanatory model.

It must be noted that the term 'Disrupted Cortical Connectivity' was recently introduced by Kana, Libero, and Moore (2011) to replace the term 'under-connectivity', which is otherwise used in the majority of the current, reviewed literature. Changing this term is considered important as it indicates that this new theory includes both neural under- and over-connectivity. Thus, we have also chosen to use this term in our article. Finally, it is important to highlight that recent research, upon which the theory of Disrupted Cortical Connectivity is based, like most research in this area, has been generalised to the wider ASD population from empirical findings using primarily high functioning participants.

The Theory of Disrupted Cortical Connectivity

It has been well established that the execution of any task depends on the activation of multiple cortical and sub-cortical regions of the brain. Effective problem solving requires the integration of neural inputs and outputs to and from these different areas. It is this integrative function, referring to the connectivity between different neural regions, that, according to the Theory of Disrupted Cortical Connectivity, is compromised in individuals with ASD (Just et al. 2007; Vissers, Cohen, & Geurts, 2012).

From a developmental point of view, the disrupted connectivity in ASD does not result from a reduction in the connectivity of previously connected regions as seen in traditional disconnection syndromes, such as conduction aphasia, visual agnosia and pure alexia (Catani & ffytche, 2005). Instead, the problem occurs during early development. These connections are a determining factor for both the normal and continuous development and reorganisation of further connections (Geschwind & Levitt, 2007). Such disrupted connectivity typically manifests itself in under-connectivity across neural systems that involve a more complex level of co-operation. This may be the case, in particular, when long distance connections,

such as the communication between frontal and posterior brain areas, appears reduced (Just et al., 2012). Conversely, within both the frontal and posterior parts of the brain, an abnormally high level of connectivity has been observed. Within the frontal regions of the brain, this high level of connectivity can be unorganised and responsible for reduced integration. Meanwhile, within the posterior parts of the brain, the functional coordination may be intact, if not working at an even higher level than is seen in neurotypical participants (Kana et al., 2011). This well organised over-connectivity in individuals with ASD is notably observed in the visuospatial and visual areas of the brain (Mizuno, Villalobos, Davies, Dahl, & Müller, 2006). Thus, individuals with ASD are assumed to rely on an information processing style which is less reliant on frontal input and more dependent on posterior processing (Kana et al., 2011).

At a Microscopic Level

Advances in brain scanning methods and technology have paved the way for the theory of Disrupted Cortical Connectivity. At a microscopic or cellular level, a number of structural studies have collectively suggested that there are differences in the neural development of individuals with ASD as compared with individuals without an ASD diagnosis (Stefanatos & Joe, 2008). Possible explanations for these variations include abnormalities in neurogenesis, neuronal migration, axon pathfinding and deficient apoptosis and pruning (Anagnostou & Taylor, 2011; Geschwind & Levitt, 2007).

In studies of connectivity in individuals with ASD, special attention is often paid to the observed atypical distribution of white matter. This refers to myelinated axons that are white in appearance. In contrast, grey matter primarily consists of neuronal cell bodies and capillary blood vessels that are grey in appearance. The main function of myelination, also known as 'white matter tracts', is to increase the speed at which electrical impulses are able to travel along the axon. Thus, connectivity both within, and between, different neural regions is greatly affected by the extent of axonal myelination (Just et al., 2012).

Diffusion Tensor Imaging (DTI) studies are particularly beneficial when investigating white matter tracts and the connections between different brain areas. DTI studies can provide a measure of the directional dependency of water diffusion in white matter tracts, referred to as Fractional Anisotropy (FA). An FA value of zero (values range from zero to one) indicates that the diffusion is equal in all directions, whereas a value of one means that diffusion occurs only along one axis. The assumption is that a more directionally dependent water flow, which would represent a relatively linear diffusion and an FA value close to one, is indicative of the presence of more myelinated axons and a greater level of structural connectivity (Vissers et al., 2012).

Finally, it has been suggested that an atypical development of the frontal and temporal minicolumns (vertical columns through the cortical layers of the brain) could account for any local over-connectivity, and decreased long distance connectivity, observed in individuals with

ASD (Casanova, 2005; Courchesne & Pierce, 2005; Just et al., 2012). This supports previous claims about unorganised connectivity resulting in reduced integration within the frontal areas of the brain.

At a Macroscopic Level

At the macroscopic level, which in this case refers to the connectivity between different neural areas, a series of research studies have identified disrupted connectivity as a factor in sentence comprehension (Just, Cherkassky, Keller, & Minshew, 2004), verbal working memory (Koshino et al., 2005) and visuomotor coordination (Mizuno et al., 2006). Disrupted connectivity has also been observed in studies involving Theory of Mind and Executive Functions (Just et al., 2007; Kana, Keller, Cherkassky, Minshew, & Just, 2009; Kana, Libero, Hu, Deshpande, & Colburn, in press). Synchronisation between brain regions in such studies is typically measured by calculating the co-variance between the fluctuations in blood flow between functionally related neural areas (Just et al., 2007). In the following section, we will primarily refer to two of these studies – one concerning Theory of Mind and the other in relation to Executive Function.

Empirical Studies

Theory of Mind

Throughout the literature, different concepts such as social cognition, mentalisation and Theory of Mind are often used interchangeably when describing how we, as social agents, are able to read and attribute emotional and cognitive mental states to ourselves and others (Gade, 2009). In relation to Theory of Mind, a Positron Emission Tomography (PET) study by Castelli, Frith, Happé, and Frith (2002) found that individuals with ASD do not experience activation of the superior temporal sulcus, the temporo parietal junction and the medial prefrontal cortex to the same extent as controls when asked to attribute mental states to animated geometric figures. These particular brain areas are arguably some of the most important regarding social cognition, which is why they were initially investigated. Kana et al. (2009) decided to replicate these findings by conducting a similar study using fMRI scanning techniques. In keeping with the previous research, a reduced activation of these neural regions was identified. The two studies differed in their conclusions however. Whereas Castelli et al. (2002) only theorised about a possible problematic feedback loop between the implicated neural areas, Kana et al. (2009) empirically revealed a decreased level of frontal-posterior connectivity in the mentalisation system among individuals with ASD (Kana et al., 2009). Consequently, as the theory of Disrupted Cortical Connectivity suggests connectivity issues between different brain areas, this could be useful when applied to explaining the impaired Theory of Mind abilities in individuals with ASD.

Executive Function

Executive Functioning refers to the mental activity involved in the planning, initiation, inhibition, regulation and flexible adaption of behaviour to external and

internal circumstances. These functions all have different neural sub-components, both cortically and sub-cortically. Thus, they are not limited to any one single area of the brain, but rather are all dependent on the prefrontal cortex for integration and supervision (Gade, 2009). In a functional Magnetic Resonance Imaging (fMRI) study by Just et al. (2007), cerebral activation while planning was examined during the performance of the 'Tower of London' test. During this, participants are typically presented with a model board complete with three rods and differently coloured beads strategically positioned on them. The participants are then asked to manipulate the beads on a similar problem-solving board to match these positions. The findings of this study revealed lower levels of synchronisation between frontal and parietal cortical areas in the group with ASD when compared to controls (Just et al., 2007). In line with the theory of Disrupted Cortical Connectivity, the planning deficits typically found in individuals with ASD could potentially be understood as a result of this inferior integration of information across frontal and parietal areas due to reduced connectivity.

The evidence reviewed thus far suggests that communication across certain cortical areas involved in particular types of social and executive functioning is less effective in individuals with ASD (Just et al. 2007; Kana et al., 2009). This lack of efficient connectivity is likely to have an impairing influence on more complex and higher order functions, such as social, linguistic and problem-solving abilities, which are all crucially dependent on the cooperation between numerous different brain areas (Kana et al., 2011). More complex and higher order functions require the simultaneous coordination of many different types of information processing. This explains why otherwise dissimilar symptoms may often co-exist in individuals with ASD (Just et al., 2007; Just et al., 2012).

Studies have also been conducted to examine the unique behavioural patterns often observed in individuals with ASD. For example, Monk et al. (2009) found that restrictive and repetitive behaviour among individuals with ASD was correlated with over-connectivity between the posterior gyrus cingularis and the right parahippocampal gyrus in a resting state (i.e. the level of brain activity wherein an individual is unaffected by external stimuli). These correlations do not, however, reveal whether this over-connectivity should be regarded as the cause of the restrictive and repetitive behaviours, or if it should be seen as the result of them. The authors proposed that increased connectivity may also be a neural manifestation of a constant effort to control these behaviours. Any interpretation of these results, in terms of cause and effect, can therefore only be a tentative one. Further empirical investigations are required to fully appreciate and understand these findings (Monk et al., 2009).

It is thus possible to argue that the studies reviewed can provide an explanation for the impairments related to both Theory of Mind and Executive Function. The theory of Disrupted Cortical Connectivity also agrees with, or can even be defined as, a more precise and neurologically detailed expansion of the theory of Weak Central Coherence (Just et al., 2007). As previously stated, individuals

with ASD have difficulties integrating representations resulting in a fragmented information-processing style (Frith, 1989). The theory of Disrupted Cortical Connectivity specifies the neural mechanisms underlying these difficulties and expands the decreased integration to include complex, cortically mediated functions in general (Just et al., 2007).

Disrupted Cortical Connectivity as an Explanatory Model for ASD

The Theory of Disrupted Cortical Connectivity can account for the deficits typically observed across the autism spectrum. Furthermore, the theory can be considered as a uniquely unifying explanatory model, integrating the three predominant theories within autism research. Moreover, the theory is strengthened by its foundation in advanced neuro-imaging methods which have facilitated a more extensive examination of this neurodevelopmental disorder at system-level. This has greatly advanced autism research, as the majority of prior studies have been conducted without a strong neuropsychological foundation, or have attempted to localise the dysfunctions of ASD to specific neural structures.

Certain issues must be considered in relation to the Theory of Disrupted Cortical Connectivity however. First, this theory has not been evaluated among low functioning individuals with autism, even though this group represents a major portion of the ASD population (Stefanatos & Joe, 2008). Testing these hypotheses among low functioning individuals with ASD could prove remarkably useful however, and reveal whether there is a correlation between disrupted cortical connectivity and symptom severity, potentially strengthening the theory. An additional area to investigate further, in relation to the theory of Disrupted Cortical Connectivity, could also involve evaluating the potential of early intervention and behavioural training in altering the course of brain development and connectivity in young children with ASD (Dawson, 2008; Keller & Just, 2009).

Additional criticisms have been recently proposed by Vissers et al. (2012). In this article, the emphasis placed on the frontal lobe areas regarding over-connectivity is called into question, as findings on this matter have not been conclusive. Nonetheless, the most consistent finding across DTI studies, when comparing neurotypical individuals and those with ASD, is a volumetric reduction of white matter tracts in the corpus callosum of participants with ASD (Stefanatos & Joe, 2008, Vissers et al., 2012). Similar volumetric reductions of white matter tracts have been found in other brain areas. However, these results are relatively inconsistent, and as such their importance remains undetermined (Vissers et al., 2012). Additionally, across studies, a consensus has not yet been reached as to how one should define local connectivity and long distance connectivity respectively, rendering it difficult to draw comparisons across studies that have been carried out thus far (Vissers et al., 2012). Furthermore, it is our opinion that the frequent co-morbidity of other disorders in individuals with ASD has been overlooked in the theory

of Disrupted Cortical Connectivity thus far. This is problematic, in that it can then prove difficult to determine whether findings are caused by ASD or are expressions of co-morbid disorders, such as epilepsy or intellectual impairment (Casanova, 2005).

The theory of Disrupted Cortical Connectivity faces other additional challenges, as disrupted cortical connectivity can also be observed in other distinct disorders, such as schizophrenia and dyslexia (Catani & ffytche, 2005; Just et al., 2007). A recent study, for example, has shown that children and adolescents at risk for developing psychosis, or who experience psychotic symptoms, show similar structural and functional abnormalities as individuals with ASD (Jacobson et al., 2010). Interestingly, there is a resemblance between the structural and functional deficits of ASD and Attention Deficit Hyperactivity Disorder (ADHD). Co-morbidity between these two neurodevelopmental disorders is often discussed in clinical settings and trials (Gargaro, Rinehart, Bradshaw, Tonge & Sheppard, 2011). In addition, disrupted cortical connectivity is also found among individuals with ADHD (Corbett, Constantine, Hendre, Rocke & Ozonoff, 2010; Gargaro et al., 2011; Tomasi & Volkow, 2012, Liston, Cohen, Theresa & Casey, 2011; Uekermann et al., 2010). Individuals with ASD generally have more severe executive difficulties relative to individuals with ADHD, but the structural and functional resemblances are unmistakable (Corbett et al., 2010; Gargaro et al., 2011). From these findings, it could be argued that disrupted cortical connectivity is merely a predictor for different kinds of psychopathology and not necessarily a predictor of ASD as such.

Conclusion

The purpose of this article was to explain the theory of Disrupted Cortical Connectivity, and discuss whether or not it could integrate three of the predominant theories in autism research: Theory of Mind, Executive Functioning and Weak Central Coherence. We have also discussed the strengths and limitations of this theory and approach. We have shown how the theory of Disrupted Cortical Connectivity can account for social cognitive and executive difficulties in high functioning individuals with ASD. As such, we suggest that the theory of Disrupted Cortical Connectivity as an explanatory model for ASD is capable of synthesising both Theory of Mind and Executive Functioning based explanations. Furthermore, the theory of Disrupted Cortical Connectivity corresponds with and extends the theory of Weak Central Coherence by specifying the underlying neural mechanisms of ASD. Consequently, the theory of Disrupted Cortical Connectivity appears capable of integrating all three theories. In addition, it can account for the otherwise dissimilar symptoms such as social, linguistic and problem-solving difficulties that co-exist in individuals with ASD, by proposing that such complex and higher order functions are crucially dependent on the cooperation between different brain areas. As a relatively novel theory, much empirical work still remains to be done before it can be accepted as an inclusive explanatory

model for ASD. Despite this, we remain optimistic about its potential to offer a more comprehensive explanation of ASD and a number of other neurological and neurodevelopmental disorders.

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
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