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Is there a bit of autism in all of us? Autism spectrum traits are related to cortical thickness differences in both autism and typical development

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ABSTRACT

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by impairments in communication and social interaction, as well as repetitive behaviors and interests. However, these traits are highly variable across individuals with ASD and are also present in the typically developing population. Brain structural correlates of ASD are also heterogeneous. Recent findings have indicated that ASD traits as measured by the autism quotient (AQ) are reflected in white matter structural differences in a continuous way across both typically-developed and ASD individuals. Here, we tested for the first time, how ASD traits are related to gray matter structural differences (and particularly cortical thickness) in both ASD and typically developing adults. The present results show that ASD traits are primarily correlated with reductions in cortical thickness in a continuous fashion across ASD and typically developing adults in social brain areas and the default mode network including the orbitofrontal cortex, postcentral gyrus, and lingual gyrus. These findings provide new evidence that ASD traits are primarily reflected in neural structure that exists along a continuum extending into the typically developing population.

1. Introduction

Autism spectrum disorder (ASD) is a heterogeneous disorder in terms of both behavioral profiles and brain correlates (Amaral, Schumann, & Nordahl, 2008; APA, 2000; Geschwind & Levitt, 2007). Behaviorally, an ASD diagnosis is based on impaired communication and social interaction, along with repetitive behaviors and interests. Studies on the neural basis of ASD have yielded variable results but generally converge to show an early brain overgrowth as well as atypical brain connectivity (see Anagnostou & Taylor, 2011 for a review). Brain structural studies in ASD have revealed both white matter and gray matter abnormalities in distributed brain regions that are implicated in the core features of ASD such as the social

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brain network, the mirror neuron system and the default mode network (Amaral et al., 2008; Anagnostou & Taylor, 2011; Kennedy, Redcay, & Courchesne, 2006; Philip et al., 2012).

Importantly, ASD traits are also present in the typically developing (TD) population, and ASD may represent the outer end of a continuum of social and communicational abilities extending into TD (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Frith, Morton, & Leslie, 1991). A key question is therefore whether brain structural differences associated with ASD also exhibit a continuum extending into TD. One way to quantify ASD traits in TD and ASD individuals is by using the autism spectrum quotient (AQ) questionnaire (Baron-Cohen, Wheelwright, Skinner, et al., 2001). The AQ gives a total score, and five subscores: social impairment, attention to detail, attention switching, impaired imagination and communication. ASD traits measured with the AQ are relatively stable across the lifespan (Whitehouse, Hickey, & Ronald, 2011) and are strongly heritable (Hoekstra, Bartels, Verweij, & Boomsma, 2007). Moreover, the AQ predicts impaired performance by TD adults on social cognitive tasks (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001; Bayliss & Tipper, 2005; Hermans, van Wingen, Bos, Putman, & van Honk, 2009; Hudson, Nijboer, & Jellema, 2012; Lombardo, Barnes, Wheelwright, & Baron-Cohen, 2007; Nummenmaa, Engell, von dem Hagen, Henson, & Calder, 2012; Poljac, Poljac, & Wagemans, 2012; Valla, Maendel, Ganzel, Barsky, & Belmonte, 2013), as well as enhanced performance on low-level perceptual tasks, such as auditory pitch discrimination (Dohn, Garza-Villarreal, Heaton, & Vuust, 2012) and visual processing (Almeida, Dickinson, Maybery, Badcock, & Badcock, 2012; Grinter et al., 2009; Skewes, Jegindø, & Gebauer, 2014; Stewart, Watson, Allcock, & Yaqoob, 2009; Takahashi & Gyoba, 2012).

Few studies have investigated the neural correlates of performance on the AQ test. Functional MRI studies in TD individuals have found positive correlations between higher AQ scores (more ASD traits) and atypical brain activation in social brain areas such as superior temporal gyrus, medial prefrontal cortex, temporal parietal junction, amygdala and fusiform gyrus during eye gazing and face processing tasks (Dalton, Nacewicz, Alexander, & Davidson, 2007; Hasegawa et al., 2013; Nummenmaa et al., 2012). In addition, performance on the AQ has been correlated with reduced activity in primary visual cortex during visual local/global tasks in TD individuals (Sutherland & Crewther, 2010). Most recently, Jung et al. (2014) found that the strength of resting state functional connectivities in the default mode network (and specifically the anterior medial prefrontal cortex) was associated with ASD traits in TD and ASD individuals, supporting the view that ASD traits lie on a continuum.

Only three studies have examined the association between performance on the AQ and brain structure. Using voxel-based morphometry (VBM), von dem Hagen et al. (2011) found inverse correlations between total AQ score and white matter volume in TD individuals in the posterior superior temporal sulcus, and between the AQ 'social impairments' subscore and white matter volume in both the anterior cingulate cortex and medial prefrontal cortex. They also found inverse correlations between total AQ score and gray matter volume in left superior frontal sulcus in TD individuals. In a recent diffusion tensor imaging (DTI) study, Iidaka, Miyakoshi, Harada, & Nakai (2012) found a positive correlation between the AQ 'imagination' subscore and white matter connectivity in the superior temporal sulcus and the amygdala in TD individuals. However, both the von dem Hagen et al. (2011) and Iidaka et al. (2012) studies only considered TD individuals, and did not include ASD participants, thus precluding the ability to test whether both groups show similar correlations between AQ score and brain structure.

To answer this question, one recent DTI study did examine the relationship between white matter structure and AQ scores in both TD and ASD individuals (Gibbard et al., 2013). They found reduced white matter fractional anisotropy to correlate with total AQ score in a similar way across both the TD and the clinical ASD sample supporting the concept of a dimensional relationship between WM microstructure and ASD symptomatology in young adults. However, no one has investigated whether similar continuous relations exist between AQ scores and gray matter brain structure across both TD and ASD participants.

To this aim, here we used cortical thickness to examine gray matter structure and its relationship to performance on the AQ in both TD and ASD adults. As a surface-based metric, cortical thickness measures avoids ambiguities in shape and position of the cortical mantle commonly associated with volumetric approaches such as VBM (Hyde, Samson, Evans, & Mottron, 2010). Previous studies have found cortical thickness differences between ASD and TD individuals mainly in brain areas implicated in social cognition, including the mirror neuron system (e.g., Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006; Hyde et al., 2010). Moreover, reduced cortical thickness has been correlated with more severe social symptoms of ASD mostly in frontal brain areas (Hadjikhani et al., 2006; Hardan, Libove, Keshavan, Melhem, & Minshew, 2009), and in particular the orbitofrontal gyrus (Doyle-Thomas et al., 2013). Based on the previous studies reviewed above, we expected to find continuous correlations between gray matter structure and performance on the AQ across both ASD and TD individuals.

2. Material and methods

2.1. Participants

Two groups of adults participated in the present study: 25 individuals with a formal diagnosis of ASD, and 26 TD adults. All participants were right-handed, and groups were matched on gender and full-scale IQ (Wechsler, 1997). The characteristics of the participants are shown in Table 1. Participants with ASD were recruited through the National Autism and Asperger's Association, assisted living services for young people with ASD, and specialized educational facilities. ASD participants were diagnosed using a standard diagnostic test (ADOS-G (Lord et al., 2000)) supported by expert clinical