**INVESTIGATING THE CONTINUUM OF AUTISM: EVIDENCE FROM A NON-
CLINICAL SAMPLE**

A thesis submitted to
the Faculty of Graduate and Postdoctoral Affairs
in Partial Fulfillment of the requirements for the degree
Master of Arts
in
Psychology

Carleton University
Ottawa, Canada

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ISBN: 978-0-494-91568-4

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ISBN: 978-0-494-91568-4

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Abstract

Autism is argued to represent the extreme version of traits continuously distributed within the general population, including among relatives of those diagnosed with the disorder (Hoekstra, 2007). Considerable debate exists on whether the Autism Spectrum Quotient (AQ) (Baron-Cohen et al., 2001) is a reliable measure of autistic trait endorsement in non-clinical populations. To address this debate, the present study examined a continuum of autism. Towards this end, there were three objectives: (1) assess the factor structure of the AQ; (2) provide evidence of autistic traits in healthy undergraduates and (3) among relatives of individuals diagnosed with clinical autism. Using an archival data set ($N = 1391$), factor analysis and group differences on the AQ were assessed. Of the factor models assessed, none provided a good fit to the current data. As predicted men scored significantly higher than women on the AQ as did those with a family history of autism. Results provide a novel contribution to the literature regarding the continuum of autism and the psychometric assessment of non-clinical samples. Limitations and proposed research directions are discussed.

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Investigating the Continuum of Autism: Evidence from a Non-Clinical Sample

Autism (ASD) is a family of pervasive development disorders clinically defined by the presence of varying degrees of deficits in three domains: social interaction, communication, and behavior (Boucher et al, 2005; Faras, Al Ateeqi, & Tidmarsh, 2010; Myhr, 1998). Typically, these disorders are conceptualized as lying on a spectrum ranging from severe disabilities in all domains (i.e., autistic disorder) to less severe disabilities (i.e., high functioning autism). Although the exact cause of autism is not known, recent evidence supports Baron-Cohen's (2002) extreme male brain theory of autism. Specifically, the *extreme male brain theory of autism* posits that autism exaggerates what occurs in the typical 'male' brain and thus, reflects an exaggeration of 'male' personality characteristics. Supporting evidence of this theory comes from the fact that milder forms of deficits in ASD are found in non-clinical groups (e.g., broad autism phenotype; Folstein & Rutter, 1977; Hurley, Losh, Parlier, Reznick, & Piven, 2007; Losh et al., 2009).

Given the evidence for the *extreme male brain theory of autism*, some researchers argue that ASD should be reframed as a continuum whereby the characteristic deficits overlap with 'normal' functioning in the general population (Ingersoll, 2010; Hurst et al., 2007; Rajendran, & Mitchell, 2007). To date, this issue has not been thoroughly examined. Additionally, there is little evidence for the reliable measurement of ASD deficits in the general population. Thus, the current study seeks to address the issue of whether the social, communicative, and behavioral deficits associated with ASD occur in an undergraduate sample. Towards this end, there are three goals: (1) to assess the

factor structure of a questionnaire used for the assessment of ASD symptoms in non-clinical samples; (2) to provide evidence that ASD deficits differ between men and women, and (3) to provide evidence that ASD deficits differ between those with and without a family history of ASD. To address the research questions, a review of the literature is presented on the: (1) core deficits in ASD (2) genetics and neuroanatomy of autism (3) extreme male brain theory of autism (4) broad autism phenotype, and (5) measurement of ASD deficits.

Core Deficits in ASD

Autism is diagnosed as a spectrum of disorders with symptoms apparent in early childhood and continuing throughout the life cycle (Faras, Al Ateeqi, & Tidmarsh, 2010; Ogle, 2009). The prevalence rate in Canada is approximately one in 165 children, resulting in an estimated 220,000 Canadians living with the disorder (Autism Society Canada, 2007). Although in its most severe form ASD includes deficits in three domains (social interaction, communication, behavior), deficits ranging from mild to severe in one or more of these areas are viewed as problematic (American Psychiatric Association, 2000). What follows is a review of the three core features in ASD.

Social Interaction. Social interaction refers to interacting socially in interpersonal situations and in larger social groups (Baron-Cohen et al., 1999). Perhaps the importance of social understanding is best highlighted by examining atypical social interaction as it occurs in autism. Specifically, individuals with ASD demonstrate impairments in making appropriate social responses and in forming reciprocal relationships (Boucher et al, 2005; Hill, & Frith, 2003; Shultz, 2005). As a result, social interaction deficits in ASD are expressed as a preference for isolation, limited motivation

for peer relations, and difficulties fitting in with peers (Frith & Frith, 2007; Ogle, 2009; Rajendran, & Mitchell, 2007). Particularly important is the inability of individuals with ASD to recognize and interpret another individual's emotions.

Research examining emotion recognition often employs visual discriminating, sorting and matching tasks whereby individuals are asked to match a particular emotion with a facial expression (e.g., happy with smiling face) or to identify the specific emotion being expressed. One study that demonstrates social impairments in ASD found that when asked to recognize and infer emotional states from still images of the eye region, high functioning autistic participants made more mistakes in comparison to normal controls (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997). In addition, ASD participants demonstrate a hypo-activation in the amygdala (a brain structure important for facial emotion processing) when judging mental and emotional states from the images; a lack of activation that was not seen in normal controls (Baron-Cohen et al., 1999).

A second study that demonstrates social deficits in ASD found that, unlike normal control participants, high functioning autistic participants showed hypo-activation of the fusiform gyrus (a cortical area important for facial emotion processing) when asked to recognize facial expressions of emotions (happy, sad versus neutral). Wang et al., (2004) showed that adolescent high functioning autistics demonstrated a lack of activation of the fusiform gyrus when instructed to match facial expressions of emotions to specific emotions (e.g., anger with scowling face). Thus, deficits in ASD for emotion recognition and social interaction are likely tied to differences in the amygdala and its associated structures (e.g., fusiform gyrus) between typically developed individuals and those with

ASD (Baron-Cohen et al. 1999:2000; Schultz et al., 2003; Shultz, 2005). What follows is a review of communication deficits as they occur in autism.

Communication. Successful social interactions also rely on the ability to communicate and existing evidence demonstrates that individuals with ASD have deficits in communication (Bara, Bucciarelli, & Colle, 2001; Klin, Jones, Shultz, & Volkmar, 2003). Although autism is characterized by impairments in many areas of communication, research has primarily focused on the use and interpretation of cues from the eyes (Farroni et al., 2002; Kawashima et al. 1999; Pelphrey et al. 2002). Because the ability to effectively interpret eye contact in social settings is critical for social relationships, it is not surprising that individuals with ASD demonstrate impairments in the processing of eye gaze (Senju et al., 2003; 2005). Indeed, some researchers suggest that communication deficits in ASD are the result of a basic impairment in detecting gaze cues (Pelphrey et al. 2002; Redcay, 2008; Redcay et al., 2010). What follows is a presentation of research examining the role of eye gaze in both communication and social interaction.

The eye region expresses information about the emotional and physical state of an individual and is considered a salient form of nonverbal communication (Adams, & Kleck, 2003; Pelphrey, Morris, & McCarthy, 2005; Senju, Tojo, Yaguchi, & Hasegawa, 2005). As a result, the ability to engage in reciprocal eye contact and detect social cues from the eyes is crucial to social interactions and communication (Leeb & Rejskind, 2004). The evidence that individuals with ASD are unable to effectively process gaze cues is illustrated in studies examining the detection of the social importance of mutual eye gaze.

One study by Senju, Yaguchi, Tojo, and Hasegawa (2003) showed that children with ASD are slower to react compared to children without ASD when instructed to determine which way the eyes are looking on still images of non-emotive faces. For example, a slower reaction time in ASD participants was evident when asked to detect direct gaze (are the eyes looking at you?) or averted gaze (are the eyes looking away from you?). Also, when asked to indicate the direction of gaze by pressing a corresponding computer key, ASD participants made more errors compared to controls and failed to detect the importance of direct gaze on an eye gaze detection task. In other words, unlike in age-matched controls, the saliency of direct gaze did not speed up or aid the detection in children with ASD. As a result, the ability to detect an important social cue in normal controls (e.g., mutual eye contact) is not evident in ASD. The presented evidence demonstrates that the social importance of recognizing emotions and interpreting cues from the eye region is somehow impaired in ASD (Pelphrey, Morris, & McCarthy, 2005; Itier, & Batty, 2009). What follows is a review of the literature examining the specific role of attention in behavioral impairments as they occur in autism.

Atypical Behavior. The third core feature of ASD is manifested as repetitive use of language or gestures, adherence to unusual rituals and routines, or abnormal preoccupations with component parts (American Psychiatric Association, 2000). As a result of these behavioral impairments, individuals with ASD often express social awkwardness, difficulty attending to social cues, and a preference for isolation in social settings (Elfenbein, Marsh, & Ambady, 2002; Low, Goddard, & Melser, 2009). Researchers assessing the attentional impairments in ASD identify difficulties in joint attention as the possible source of atypical behaviors (Stahl, & Pry, 2002). More

specifically, joint attention refers to the selective use of attention with the attentional focus of another individual (Dawson et al., 2004; Leekam, Lopez, & Moore, 2000; Morgan, Mayberry, & Durkin, 2003). One example of joint attention in social communication is identifying that another person's eye contact has shifted to another person in the room and requires the individual to then shift attention to follow. Thus, joint attention involves engaging in a common attention focus using nonverbal cues (Delinicolas, & Young, 2007; Morgan, Mayberry, & Durkin, 2003; Redcay, 2008).

One study examining social-communication and attention in ASD showed that children with ASD do not jointly attend as frequently as age-matched controls when asked to attend to an object after someone pointed or gestured towards the object (Colombi et al., 2009). Similarly, difficulties are evident in ASD when participants are asked to orient attention to visual social stimuli (e.g., still images of the eye region), indicating an avoidance to selectively attend to social stimuli (Allen, & Courchesne, 2001). Several related studies show that unlike typically developed individuals, impairments in ASD are evident for shifts of attention, maintaining focus for social cues, and motivation for acquiring joint attention (Allen, & Courchesne; Charman, 2003; Delinicolas, & Young, 2007; Morgan, Mayberry, & Durkin, 2003).

Overall, individuals with ASD vary in the extent of difficulties for attention. However, it is theoretically argued that attentional impairments in combination with social-communicative deficits may result in difficulties for initiating social and emotional interactions (Bormann-Kischkel, Vilsmeier & Baude, 1995; Delinicolas, & Young, 2007; Naber et al., 2007). Furthermore, individuals with ASD have been said to have what some call "sticky attention" or in other words, a difficulty in switching attention (Morgan

et al., 2003). Given the evidence, researchers identify difficulties in attention as a possible source of atypical social behaviors (i.e., social awkwardness), unusual adherence to rituals (i.e., compulsive tapping or rocking), and sticky interest patterns (Dawson et al., 2004; Redcay, 2008; Stahl & Pry, 2002).

In summary, persons with ASD demonstrate some degree of impairment in recognizing emotions, interpreting the social importance of eye gaze, and attention that appears to underlie the three core deficits of (1) social interaction, (2) communication, and (3) atypical behavior. Indeed, many proposed theories and contributing factors have been put forward to address the possible causes of ASD; however, to date the disorder is not attributed to any one cause.

Genetics and the Neuroanatomy of Autism

Research findings from family and twin studies indicate that the expression of ASD deficits is influenced by genetics (Hoekstra, Bartels, Verweij, & Boomsma, 2007). For example, the prevalence rate in siblings of individuals with ASD is estimated at 4.5% to 15% (Bailey et al, 1995; Ronald et al., 2002: 2008; Yang & Gill, 2007). Furthermore, a higher overall incidence of autistic “like” symptoms are found in relatives of individuals diagnosed with ASD (Bolton et al, 1994; Le Couteur et al, 1996; Piven et al, 1997; 2001). Although no single genetic link is evident, researchers suggest that multiple genes are involved in the disorder (Geier, Kern, & Geier, 2010; Ronald et al., 2006; 2008). Given that autism is diagnosed as a triad of deficits, it is possible that not only are different genes involved with the presentation of symptoms but that environmental factors may also play a role at different stages of development (Le Couteur et al, 1996; Muhle, 2004).

Recent studies have investigated the neuroanatomy of autism. For example, Geier, Kern, and Geier (2010) suggest that cell migration and division in the ASD brain is slowed resulting in degenerative cell death. Further, that functional connectivity is thought to be diminished by decreases in the retention of neural networks (Amaral, Schumann, & Nordahl, 2008; Lewis, & Elman, 2008; Stanfield et al., 2008). A potential result of degeneration is the occurrence of non-specific neurological, motor, and behavioral dysfunctions (Geier, Kern, & Geier; Lewis & Elman, 2008). In other words, neuroanatomical changes in ASD individuals as well as the genetic markers responsible for deficits have not been linked to a specific cause; rather, several possible non-specific symptoms (Stanfield). As such, no clear pathology has been uncovered. One theory that holistically addresses the multiple deficits apparent in ASD and incorporates existing evidence is Baron-Cohen's (2002) extreme male brain theory of autism. What follows is a review of the *extreme male brain theory of autism*.

The Extreme Male Brain Theory of Autism

The theory is based on three known observations about ASD. First, autism is characterized by a strong attention to detail, social awkwardness, and patterns of restricted interests (Baron-Cohen, 2002:2004; Delinicolas, & Young, 2007). Second, testosterone, a masculinizing hormone, is linked to social, communicative, and behavioral deficits in ASD for both males and females (Baron-Cohen, 2002; Knickmeyer, Baron-Cohen, Raggatt, & Taylor 2005; Silani, Bird, Brindley, Singer, Frith, & Frith, 2008). Third, the prevalence rate of ASD in males is higher than that of females. The theory posits that autism is the extreme form of the typical male brain. What follows is a review of the key findings supporting the *extreme male brain theory of autism*.

Systemizing. Systemizing refers to the drive to analyze, construct, and identify the rules of systems to predict how systems will behave (Baron-Cohen, 2002; Baron-Cohen & Wheelwright, 2004). Specifically, individuals with a strong systemizing ability tend to perform well on rule based tasks relating to a high attention to detail, repetition, and replicating patterns (Baron-Cohen & Wheelwright). Baron-Cohen and Wheelwright (2004) argue that male and female brains possess innate differences and that the male brain has a higher propensity towards *systemizing*. Evidence for the *extreme male brain theory of autism* is supported by neurodevelopment and cognitive differences found between men and women on measures designed to assess systemizing ability.

Sex Differences. Baron-Cohen, Richler, Bisarya, Gurunathan, and Wheelwright (2003) examined scores on self-report measures designed to test systemizing ability. The findings confirmed that as predicted by the extreme male brain theory, men scored significantly higher than women on systemizing (Baron-Cohen et al., 2003). Thus, the findings indicate that a sex difference is present in the normal population on a measure representing autistic-like characteristics. Given that men and women differ on systemizing ability, research has focused on factors that lead to these differences (Wakabayashi, Baron-Cohen, & Wheelwright, 2006). One possible factor concerns early exposure to hormones on the developing brain that remain apparent in adulthood (Knickmeyer & Baron-Cohen, 2006).

Androgen Exposure. One study examining fetal testosterone found that high levels were associated with deficits in social communication similar to those found in autism (Knickmeyer & Baron-Cohen, 2006a). Specifically, high levels of pre-natal exposure (testosterone measured by amniocentesis in mothers) in both male and female

children resulted in a poorer quality of social relationships, increased restricted patterns of interest, and difficulty in attention switching (Knickermeier, Baron-Cohen, Raggatt, & Taylor, 2005). Thus, the evidence suggests that a strong positive relationship exists between high levels of fetal testosterone and the characteristic deficits found in ASD in both males and females (Auyeung et al., 2009; Arndt, Stodgell, & Rodier, 2005; Szatmari, 2003). Given that similar ‘autistic-like’ deficit patterns in non-autistic children are linked to high levels of a “male” brain-shaping hormone, researchers argue that autism can be framed as an extreme of the typical male brain (Auyeung, et al., 2009; Knickmeyer, & Baron-Cohen; Knickermeier, Baron-Cohen, Raggatt, & Taylor). To further demonstrate, researchers examined systemizing ability in persons with ASD.

Systemizing in ASD. Baron-Cohen, Richler, Bisarya, Gurunathan, and Wheelwright (2003) tested high functioning autism and Asperger syndrome participants on systemizing and as predicted, high functioning ASD adults scored significantly higher on systemizing than control participants. Specifically, ASD participants demonstrated more interest in the organization and structure of systems (e.g., weather patterns, timetables) than matched normal controls (Baron-Cohen et al., 2003). In a related study testing systemizing in ASD, Lawson, Baron-Cohen, and Wheelwright (2004) found that, unlike typically developed individuals, high functioning ASD participants demonstrated an advanced systemizing ability. Overall, the evidence suggests that there is a strong preference to systemize in individuals with ASD. Of particular interest, researchers found that male persons with ASD show a higher propensity towards systemizing than do female persons with ASD, and both male and female normal controls (Lawson, Baron-Cohen, & Wheelwright). Given the evidence for commonalities amongst high

systemizing, sex differences in the prevalence rates of ASD, and the characteristics deficits, some researchers argue that autism can be thought of as a variant of the typical male brain.

The *extreme male brain theory of autism* suggests that autism exaggerates what occurs in the typical 'male' brain and thus reflects an extreme version of male personality and cognitive characteristics. Supporting evidence of this theory also comes from the fact that milder forms of deficits in ASD are found in non-clinical groups (Piven, Palmer, Jacobi, Childress, & Arndt, 1997; Yang & Gill, 2007). What follows is a review of a population with a genetic susceptibility to ASD who demonstrate non-clinical forms of the disorder.

Broad Autism Phenotype

The term, broad autism phenotype (Folstein & Rutter, 1977) was coined to describe the presence of 'autistic-like' characteristics among relatives of individuals diagnosed with ASD. For example, the broad autism phenotype in parents of children with autism has been shown to increase the likelihood of endorsing milder forms of deficits found in clinical ASD. The group of 'autistic-like' impairments resembles those in clinical ASD however, the severity is milder and the frequency of occurrence is lessened (Dawson et al. 2002; Hurley, et al. 2007).

Core Deficits in the Broad Autism Phenotype. As in clinical ASD, impairments in the broad autism phenotype occur in three domains: (1) social interaction, (2) communication, and (3) atypical behavior (Bailey et al., 1995:1998; Ronald et al., 2006; Yang & Gill, 2007; Yirmiya & Shaked 2005). First, research has shown that individuals with the broad autism phenotype have difficulties in social interaction. For

example, individuals without a clinical diagnosis but who have a family history of autism show impairments in the creation and maintenance of intimate, reciprocal, and confiding friendships (Piven et al., 1997; Jobe & Williams-White, 2007). Second, researchers have found ‘autistic-like’ communication impairments in broad autism phenotype individuals. For example, relatives of individuals with ASD demonstrate inappropriate semantic understanding (e.g., misattributing meaning) and awkward use of language (e.g., verbal intonations) in social situations (Landa, Piven, Wzorek, Gayle, Chase, & Folstein, 1992). Third, unusual adherence to specific interests (e.g., fascination with dates or maps) and aloof behavior (e.g., detached or unfriendly) are also evident in broad autism phenotype individuals (Bishop, Mayberry, Maley, Wong, Hill, & Hallmayer, 2004; Losh, & Piven, 2007; Szatmari et al., 2000).

The evidence thus suggests that non-autistic relatives demonstrate a phenotype similar to that of ASD. Specifically, as seen in clinical ASD, non-autistic relatives show some mild degree of impairment in the core deficits of (1) social, (2) communication, and (3) atypical behavior. The presented evidence demonstrates that characteristic deficits associated with clinical autism are found in sub-clinical populations (e.g., broad autism phenotype). Furthermore, these findings point to the need for examining what factors, if any, are involved outside of the limits of a clinical ASD spectrum (Ingersoll, 2010; Losh et al, 2009; Ogle, 2009).

Investigating Non-clinical Populations

Traditionally, autism is considered to be two distinct boundaries on each end of clinical diagnoses (i.e., classic autism, and high functioning autism. Given the evidence for the *extreme male brain theory of autism* and that milder forms of deficits in ASD are

found in non-clinical groups (e.g., broad autism phenotype), researchers argue that classifying markers of the disorder in the general population may clarify what constitutes “milder” forms of autism (Leboyer, 2003; Wheelwright, Auyeung, Allison, Baron-Cohen, 2010).

The Autism Spectrum Quotient (Baron Cohen et al., 2001)

The Autism Spectrum Quotient (AQ) was designed as a screening measure of autistic traits in non-autistic individuals (Woodbury-Smith, Robinson, Wheelwright, & Baron-Cohen, 2005). The five subscales of the AQ (Social Skill, Communication, Attention Switching, Imagination, and Attention to Detail) are derived from the three core deficit features found in clinical ASD. However, based on clinical observation, Baron-Cohen and colleagues (2001) included additional features (Imagination, and Attention) that are not a part of the DSM-IV criteria. The measure has been reported to be sensitive enough to identify ASD traits in intellectually normal adults (Hurst, Mitchell, Kimbrel, Kwapi, & Nelson-Gray, 2007) and demonstrates moderate reliability (Cronbach's $\alpha = 0.78$) (Baron-Cohen et al., 2001; Wakabayashi et al., 2006).

Factor Structure of the Autism Quotient. The 5-structure model proposed by Baron Cohen et al., (2001) has little empirical testing and as such, further research is needed to merit the psychometric properties of the AQ. Specifically, three and four factor models have been argued to provide a better fit compared to the original structure of the AQ.

To date, three studies have examined the factor structure of the AQ suggesting various three and four subscale models (Austin, 2005; Hurst et al. 2007; Stewart & Austin, 2009). In the first study, using a considerably small sample, Austin (2005)

examined the factor structure of the AQ using 201 undergraduate students and provided evidence for three factors (Communication/mind-reading, Social Skill, and Details/patterns). In the second study, Hurst et al. (2007) conducted a principal component analysis on the AQ using 1005 undergraduate students, providing evidence for a similar 3-factor to Austin's (2005) study. In the third study, Stewart and Austin (2009) examined the factor structure of the AQ using 536 students and provided support for a 4-factor model. The model proposed by Stewart and Austin is in accordance with previous factor analytic studies suggesting a socialness, attention, and understanding of others factor, however, a fourth imagination factor was identified.

Thus, various models that include social skill, communication, and patterns are argued to provide a better goodness of fit than the original 5-factor model. Of particular relevance to the current study, the AQ is used as an assessment measure of autistic trait endorsement in adults and in undergraduate samples. Given its potential utility in researching sub-clinical forms of autism, it is critical that the psychometric properties of the AQ be evaluated. What follows is a review of the research goals for the current thesis study.

Research Goals

The current study is designed to examine whether the characteristic deficits associated with clinical autism occur as theoretically expected in an undergraduate sample. To accomplish the research objective, there are three goals. The first and primary goal is to test the factor structure of the AQ in a large non-clinical sample by examining the 5-structure model proposed by Baron Cohen et al., (2001) and alternative models. The second goal is to provide evidence that mild forms of ASD deficits as

measured by the AQ differ between men and women. The third goal is to provide evidence that ASD core deficits differ between those with and without a family history of ASD.

To address the first research goal, two exploratory factor analyses and three confirmatory factor analyses of the AQ will be completed using an archival data set. To address the second goal, a two (sex; men, women) x six (Total and Subscale) MANOVA will be conducted. To address the third goal, a two (family history of ASD; yes, no) x six (Total and Subscale) MANOVA will be conducted. We predict the following:

Hypotheses (1) - Autism Spectrum Quotient. Given the evidence for reduced factor models, it is likely that combining certain domains will result in a better fit to the data (Austin, 2005). Thus, we predict that a 3-factor model will account for more of the variability than a 5-factor model. Specifically, it is expected that the factor analyses will provide three reliable factors that center around Social Communication, Interest Patterns, and Attention.

Hypotheses (2) - Sex Differences. In line with the *extreme male brain theory of autism*, it is expected that men and women will differ on scores from the AQ (Austin, 2005; Baron-Cohen et al. 2001; Wakabayashi et al., 2006). Specifically it is hypothesized that mean scores for the total and subscale scores will be significantly higher for men than for women.

Hypotheses (3)-Heritability of ASD. Non-autistic relatives show milder impairments in the core deficits associated with clinical autism (Jobe & Williams-White, 2007; Losh, & Piven, 2007; Yang & Gill, 2007). Given these findings, it is expected that

mean scores on the AQ for participants with a family history of autism will be significantly higher than participants without a family history of the disorder.

Method

Participants

Participants for the study were 1391 students ranging in age from 17 to 53 years ($M = 20.38$, $SD = 4.89$) recruited from an undergraduate participant pool in a medium size Canadian university. Volunteer participants were enrolled in various student degree programs (e.g., science, engineering, and arts). There were 442 men, ranging in age from 17 to 53 years ($M = 20.49$, $SD = 5.12$) and 949 women, ranging in age from 18 to 52 years ($M = 20.33$, $SD = 4.79$).

Materials and Procedure

A general screening questionnaire (Parlow, 2009) was administered that asked participants to indicate in open-ended response, their age in years and gender. The second section asked (Yes / No) whether or not participants had an immediate family history of autism. Participants then completed the AQ.

The AQ (Baron-Cohen et al., 2001) is a 50-item pen and paper questionnaire that measures autistic trait endorsement using five scales; Social Skill, Communication, Attention Switching, Imagination, and Attention to detail. The questionnaire consists of ten questions for each of the five subscales. Participant's rate the extent they agree or disagree with each statement. Example questions include *social skills* (e.g., "I find social situations easy"), *attention switching* (e.g., "I prefer to do things the same way over and over again"), *imagination* (e.g., "I find making up stories easy"), *communication* (e.g., "I enjoy social chit-chat"), and *attention to detail* (e.g., "I notice patterns in things all the time").

Scoring the AQ. Dichotomous scaling can result in decreases in reliability when used in factor analysis (Shapiro, Lasarev, & McCauley, 2002; Waller, Tellegen, McDonald, & Lykken, 1996). To correct for this and to facilitate planned analyses, items in the present study were scored on a continuous Likert scale. Specifically, responses were coded on a four-point scale (definitely agree, slightly agree, slightly disagree, definitely disagree) and recoded as necessary. A total AQ score is calculated as the sum of scores for all items, resulting in a minimum score of 50 and a maximum score of 200. A higher overall score represents a higher degree to which an individual endorses traits (Baron-Cohen et al.,).

Results

Preliminary Analysis

Two preliminary analyses were conducted on the data set prior to the factor analyses. The first analysis was used to exclude participants with missing data. For the factor analysis only those participants who completed all of the items on the AQ were selected. The resulting sample ($n = 1251$) was randomly divided into two groups to allow for exploratory and confirmatory analyses to be conducted on different samples from the same population. The second preliminary analysis was conducted to verify that the two randomly divided groups (**sample A**, $n = 625$; **sample B**, $n = 626$) did not differ on specific characteristics; age, total score and subscales.

Given the number of comparisons completed, a conservative alpha level was set at 0.01. The two groups did not significantly differ in age, $t(1234) = -1.50, p = .14$ or total score, $t(1251) = 1.41, p = .16$. Also, the two randomly divided groups did not differ on subscale scores; Social Skills, $t(1251) = -2.34, p = .02$, Attention Switching, $t(1251) =$

1.52, $p = .13$, Attention to Detail, $t(1251) = -.89$, $p = .38$, Communication, $t(1251) = .19$, $p = .85$, or Imagination, $t(1251) = 1.02$, $p = .31$.

Because there are additional statistical requirements for factor analysis, several other parameters were also examined. First, it was determined that both samples A and B exceed the minimum size of 100 to 200 participants that is recommended for completing a factor analysis (MacCallum, Widaman, Preacher, & Hong, 2001). Secondly, to address the issue of subject to item ratio, the ratio for both samples meets the minimum requirement of (5:1) for the stability and accuracy of estimates for the factor structure (Costello & Osborne, 2005; Widaman, 1993). Finally, two additional statistics were calculated to assess the suitability of the data for factor analysis with respect to the strength of inter-correlations (Dziuban & Shirkey, 1974; Weiss, 1971). Bartlett's test of sphericity was used to test the strength of the relationships among variables. A significant value indicated strong relationships among variables, $\chi^2 = 7442.34$, $df = 1225$, $p < 0.001$. Additionally, a Kaiser-Meyer Olkin test was used to assess the sampling adequacy for a satisfactory factor analysis. The current value of .82 indicated a reliable factorability of the data.

Exploratory Factor Analysis of Sample A

To examine the structure of the five-factor model that includes the subscales of ((1) Social Skill, (2) Communication, (3) Attention Switching, (4) Attention to Detail, and (5) Imagination), an Exploratory FA was conducted. A principal axis factor analysis was performed using a rotation of loadings to allow for the clearest pattern of loadings to be interpreted (Costello & Osborne, 2005; Hurst et al., 2007). Given that items were

expected to be related based on the high number of correlations above .3, a Direct Oblimin rotation was selected.

Using the Kaiser criterion (Kaiser, 1960) for retention of components with eigenvalues higher than 1, five factors accounting for 11.49, 5.86, 4.31, 3.30, and 2.45 % of the variance respectively were found. To aid in the examination of the factor structure and visualize the comparative importance of the factors, a Scree plot was used to determine the number of factors to retain. The results indicated that all five factors should be extracted. Descriptive statistics for the five factors are presented in Table 1.

The analysis resulted in a 5-factor model of the AQ (see Appendix A for loadings, pg. 49). The factor labels for the current study were constructed from Baron-Cohen et al. (2001). Nine items loaded onto Factor 1 that center on social skills and communication. Factor 1 was labeled Social Skills. A total of five items loaded onto Factor 2, labeled Attention to Detail. Four items loaded onto Factor 3, labeled Communication. Seven items loaded onto Factor 4, labeled Imagination. The fifth factor had two items that loaded onto it, labeled Attention Switching.

Table 1

Descriptive statistics for the five-factor model of the Autism Spectrum Quotient.

	Number of Items	<i>M (SD)</i>	Skewness	Kurtosis
Social Skills	9	18.194 (5.261)	.363	-.358
Attention to detail	5	12.59 (2.297)	.03	-.097
Communication	4	8.292 (2.055)	.18	-.433
Imagination	7	13.296 (3.363)	.18	-.302
Attention switching	2	5.151 (1.268)	.024	-.31

Internal reliabilities were examined using Cronbach's alpha for the five factors and indicated low to moderate values for total ($\alpha = .74$), Social Skills ($\alpha = .81$), Attention to Detail ($\alpha = .39$), Communication ($\alpha = .51$), Imagination ($\alpha = .63$), and Attention Switching ($\alpha = .32$). Low alpha values indicated that the items on the subscales of attention, communication, and imagination are not consistently measuring the assessed construct. Given that previous research findings supported a three-domain model an additional analysis using a fixed number of factors was then conducted using data from 625 (sample A) participants.

Reduced model of Sample A

As in the first analyses, the data were first subjected to a principal axis factor extraction and a Direct Oblimin rotation was used. Item loadings on the three factors with values less than .4 were excluded. An Exploratory FA was undertaken that forced items assessing social-communication, attention, and understanding others to create three factors as per (Austin, 2005; Hurst et al., 2007; Stewart & Austin, 2009). The three-factor structure obtained using EFA explained 20.68 % of the variance (see Appendix B for loadings, pg. 50). The three factors accounted for 11.24, 5.52, and 3.94 % of the variance respectively. Descriptive statistics for the factor loadings are shown in Table 2.

Factor labels for the model were adopted from previous factor analytic studies (Austin; Stewart & Austin). Ten items loaded onto Factor 1 that centered on social skills and communication. Factor 1 was labeled Social Communication. A total of five items loaded onto Factor 2. Items from the second factor centered on restricted patterns of attention; the factor was labeled Attention to Detail. Three factors loaded onto the third factor that centered on the intentions of others; Factor 3 was labeled Understanding

Intentions of Others. Internal reliabilities were examined using Cronbach's alpha for the three factors and indicated moderate to high values for total ($\alpha = .75$), Social Communication ($\alpha = .87$), Attention to Detail ($\alpha = .66$), and Understanding Intentions of Others ($\alpha = .60$).

Table 2

Descriptive statistics for the three-factor model of the Autism Spectrum Quotient.

	Number of Items	<i>M (SD)</i>	Skewness	Kurtosis
Social communication	10	20.961 (5.675)	.32	-.33
Attention to detail	5	12.483 (2.831)	.07	-.12
Understanding intentions	3	6.243 (1.613)	.05	-.09

Confirmatory Factor Analyses of Sample B

To confirm the structure of the AQ, three confirmatory factor analyses were conducted. The fit of the 5-factor and 3-factor models from the exploratory analysis was examined using data collected from 626 (sample B). To test the best fitting model and estimate the relationships between variables, confirmatory factor analysis was completed using the 5-factor structure of the AQ proposed by Baron-Cohen and colleagues (2001). Along with chi-square, other model fit indices were evaluated in the current study. The Comparative Fit Index (CFI) and Root Mean Square Error of Approximation (RMSEA) were selected as non-centrality based transformations of the overall chi-square (Macdonald, Marsh, & Herbert, 1990). The Adjusted Goodness of Fit Index (AGFI) and

the Standardized RMR (SRMR) were selected as goodness of fit indices based on residuals (Byrne, 2001).

Confirmatory FA: 5 factor model. The model of the AQ (Social Skill, Communication, Attention Switching, Attention to Detail, and Imagination) derived from the initial exploratory analysis was tested using confirmatory FA procedure in AMOS. Results showed that the 5-factor model did not fit well statistically, $\chi^2 = 1322.601$, $p < .001$. In addition, the assessed model fit indices did not meet the required goodness of fit thresholds (CMIN/df = 4.212, AGFI = .825, CFI = .749, RMSEA = .072, SRMR = .083) (Byrne; Macdonald, Marsh, & Herbert, 1990). Specifically, non-centrality based fit and goodness of fit indices were above the threshold which, for the current study, is indicative of poor model fit (Byrne; Macdonald, Marsh, & Herbert). For a path diagram of the results see Appendix C (pg. 51).

Confirmatory FA: 3 factor model. The model of the AQ (Social Communication, Attention to Detail, and Understanding Others) derived from the exploratory analysis was also tested using confirmatory FA procedure in AMOS. Results showed that the 3-factor model did not fit well statistically, $\chi^2 = 641.56$, $p < .001$. Model fit indices were below the required goodness of fit thresholds (CMIN/df = 4.86, AGFI = .853, CFI = .834, RMSEA = .068, SRMR = .071). Specifically, goodness of fit indices and the selected non-centrality based fit indices were above the threshold which is indicative of poor model fit (Byrne). For a path diagram of the results see Appendix D (pg. 52).

Confirmatory FA: Original model of the AQ. Confirmatory FA was conducted using the factor model originally proposed by Baron-Cohen and colleagues in (2001).

Results showed that the model did not fit well statistically, $\chi^2 = 3938.46$, $p < .001$. Also, the model fit indices did not meet the required goodness of fit thresholds (CMIN/df = 3.41, AGFI = .734, CFI = .569, RMSEA = .062, SRMR = .082). The goodness of fit and non-centrality based fit indices were above the threshold which indicates poor model fit. For a path diagram of the results see Appendix E (pg.53).

Comparison of Models. Overall, model fit for all three models did not meet the goodness of fit criteria specified in the current study. Based on the fit indices, the 3-factor model that included *social communication*, *attention to detail*, and *understanding intentions* provided a better fit to the current data in comparison to the other proposed 5-factor models. This is evidenced in the stronger AGFI, CFI, SRMR, and the lowest expected cross-validation index (ECVI). However, none of the models assessed in the current study provided a good fit to the data. Fit statistics are presented in Table 3 for all three models compared.

Table 3

Fit statistics for three confirmatory factor analyses

	CMIN/df	AGFI	CFI	RMSEA	SRMR	ECVI
Factor model (Baron-Cohen et al.)	3.381	.734	.569	.062	.082	6.263
5-factor model (from EFA)	4.212	.825	.749	.072	.083	2.321
3-factor model (from EFA)	4.86	.853	.834	.068	.071	.947

Sex Differences

In line with the *extreme male brain theory of autism*, the second hypothesis predicted that scores for the total AQ would be significantly higher for men than for women. Only those participants who indicated no family history of autism on the screening questionnaire were selected for the analyses in order to dissociate gender and family history (Parlow, 2009). To test the hypothesis, a one-way ANOVA was conducted with sex as the independent variable and the total AQ score as the dependent variable. As expected men ($M = 111.565$, $SD = 10.191$) scored significantly higher than women ($M = 108.181$, $SD = 11.434$), $F(1, 1204) = 15.363$, $p < .001$.

Also in line with the *extreme male brain theory of autism*, the second hypothesis predicted that mean scores for each of the AQ subscales would be significantly higher for men than for women. Again, only those participants who indicated no family history of autism were selected for the analysis. To test the hypotheses, a MANOVA was conducted to examine sex differences on each subscale score. The independent variable for the analyses was sex and the dependent variables were the subscale scores.

A significant multivariate main effect for sex was found, Hotelling's $T^2 = .047$, $F(5, 1198) = 11.426$, $p < .001$. Given the significance of the overall test, the univariate effects were examined. Results showed that men scored significantly higher than women on the subscales of *social skills*, $F(1, 1204) = 7.916$, $p < .001$; *communication*, $F(1, 1204) = 18.297$, $p < .001$ and *imagination*, $F(1, 1204) = 37.346$, $p < .001$. However, counter to initial predictions, men and women did not significantly differ on scores from the two attention-based subscales of *attention to detail*, $F(1, 1204) = .483$, $p = .487$ or *attention switching*, $F(1, 1201) = 1.984$, $p = .159$.

Descriptive statistics for the subscale scores for men and women are presented in Table 4.

Overall, results examining sex differences on total and subscale scores provide partial support for the hypothesis that men and women differ in autistic trait endorsement as measured by the AQ.

Table 4

Descriptive statistics for men (n = 394) and women (n = 810) for subscale scores

	Gender	
	Men	Women
	M (SD)	M (SD)
Social Skills	20.53 (4.22)	19.79 (4.46)
Communication	20.67 (3.95)	19.68 (3.71)
Attention Switching	24.40 (3.23)	24.64 (3.39)
Attention to Detail	25.55 (3.97)	25.73 (4.24)
Imagination	20.40 (3.74)	19.02 (3.73)

Heritability

The third hypothesis predicted that scores on the AQ for participants with an immediate family history of ASD would be significantly higher than for participants without a family history. Of the total sample, 52 (4.4%) indicated that they had an immediate member of their family diagnosed with autism. A random number generator was used in Microsoft Excel to randomly select 52 (12 men, 40 women) out of a possible 1204 participants without a family history. The randomly drawn participants were then

compared to 52 (12 men, 40 women) participants who indicated an immediate family history of ASD.

To test the hypothesis, a one-way ANOVA was conducted using data from this sample of 104 participants, with sex as the independent variable and the total AQ score as the dependent variable. The results showed that those with an immediate family of ASD ($M = 110.40$, $SD = 12.043$) scored significantly higher on the total AQ score than those without an immediate family history ($M = 105.46$, $SD = 9.28$), $F(1,103) = 5.94$, $p = .021$.

A MANOVA was also conducted to examine the subscale scores. The independent variable for the analysis was immediate family history of autism and the dependent variables were the AQ subscale scores. A significant multivariate main effect for heritability was found, Hotelling's $T^2 = .142$, $F(5, 98) = 2.785$, $p = .021$. Given the significance of the overall test, univariate effects were then examined. In line with predictions, those with a family history scored higher than those without for two of the subscales; *social skills*, $F(1,103) = 8.84$, $p = .004$ and *communication*, $F(1,103) = 8.327$, $p = .005$.

Univariate results showed no significant differences for the subscales of *attention switching* $F(1,103) = 2.876$, $p = .093$, *attention to detail*, $F(1,103) = .968$, $p = .327$, and *imagination*, $F(1,103) = 1.074$, $p = .303$. Overall, weak partial support was found for the hypothesis that those with a family history of ASD differ from those without a family history in their endorsement of autistic traits as measured by the AQ. Descriptive statistics for the subscale scores are presented in Table 5.

Table 5

Descriptive statistics for subscale scores of the Autism Spectrum Quotient

	Family history of ASD	
	Yes M (SD)	No M (SD)
Social Skills	20.19 (4.88)	17.77 (3.26)
Communication	20.46 (3.38)	18.63 (3.05)
Attention Switching	24.88 (3.41)	23.73 (3.53)
Attention to Detail	25.88 (4.23)	25.04 (4.54)
Imagination	18.98 (3.69)	18.21 (3.87)

Discussion

The purpose of the present study was to assess whether the characteristic deficits associated with clinical autism measured by the Autism Quotient (AQ) occur as theoretically expected in an undergraduate sample. The first hypothesis predicted that a 3-factor model would provide a better fit to the data than a 5-factor as in (Austin, 2005; Hurst et al., 2007). This hypothesis was not supported by the results. Specifically, a reduced factor model did not provide a more parsimonious assessment of quantitative autistic traits. The second hypothesis predicted that that men and women would differ on scores from the AQ. This hypothesis was partially supported. Results showed that men scored significantly higher than women on the total and subscales of *social skills*, *communication*, and *imagination*. The third hypothesis predicted that participants with a family history of ASD would score significantly higher than participants without a family

history of ASD. Partial support for the hypothesis was found as shown in the difference in scores between those with and without a family history of autism.

In the following discussion, the factor structure of the AQ will be considered first. Given previous findings, we expected that a 3-factor model would better account for the variability in AQ scores than Baron-Cohen et al.'s 5-factor model (Austin, 2005; Hurst et al., 2007; Stewart & Austin, 2009). We also considered an alternative 5-factor model emerging from the initial exploratory analysis which did not correspond to Baron-Cohen et al.'s model. Counter to initial predictions, our findings did not support any of the three models, although the 3-factor model was better than either of the 5-factor models.

Both 5-factor models assessed in the current study did not fit well to the data. Fit indices for the 3-factor model were approaching acceptable levels; however, each fell below the goodness of fit criteria. One possible reason for the lack of fit is the strong interrelation among items and considerable cross-loadings onto factors assessing social and communication deficits. Given the lack of findings for multiple independent factors, it is argued that the present findings provide weak support for a reduced structure rather than being highly fractionable (Dworzynski, Happé, Bolton, & Ronald, 2009). Implications for fractionation will be considered next.

The Fractioning of Autism

Some researchers examining the phenotypic relationship among the triad of impairments argue that social, communicative, and atypical behaviors associated with ASD are independent and considered fractionable (Happé & Ronald, 2008). That is, each factor in the ASD triad is to some degree genetically independent and cognitively homogeneous (Ronald et al. 2006). Contrary to this notion, the 3-factor model from the

present study indicated two factors measuring autistic traits in a social domain (*social communication, and understanding others*), and one factor assessing non-social traits (*attention to detail*). Of particular relevance to non-fractioning, participants in the current study who scored high on social subscales also tended to score higher on the non-social subscale. Therefore, it is argued that difficulties in verbal and non-verbal social-communication may impede one's ability for effectual social communication (Austin, 2005). Thus, a one-factor model based on sociability may account for a single underlying factor that represents a large range of communicative, perspective taking, and socio-emotional behaviors (Hoekstra et al., 2007). This would have implications for non-fractionation.

Another line of evidence towards non-fractioning amid social factors can be found in the diagnosis of ASD (Hurst et al. 2007). Currently, the DSM-IV specifies a triadic diagnostic that involves (1) social, (2) communicative, and (3) atypical behaviors. Given that deficits in social-communication necessitate at some level, a degree of overlap in the presented symptoms of ASD, the homogeneity of the triad of impairments has come under question. We have argued that one possible solution is a unidimensional approach that includes a heterogeneous range of ASD symptoms along a continuum of autism. Indeed, a proposed change to the DSM-V that merges dimensional categories (e.g. social and communicative aspects into one category) is argued to possibly minimize the overlap of symptoms in the current triadic view (Happé & Ronald, 2008). In line with the future diagnostic direction, findings from the present factor analyses provide support for the unidimensional combination of social and communicative domains (Hoekstra et al., 2010; Mandy & Skuse, 2008). Also, results from the current study can be interpreted as

providing weak evidence for a possible non-fractionation of ASD traits in non-autistic individuals.

Findings from the current non-autistic sample may not generalize to individuals on the clinical spectrum of ASD. More specifically, autistic traits in the general population may become more fractionable for those who are clinically diagnosed (Ronald et al., 2006). However, given the proposed reconsiderations of ASD in the DSM-V, future research would benefit from re-evaluating a reduced factor model. Specifically, we argued that a model of the AQ that includes a reduced factor structure (e.g. 2-factor or a single structure) could reinforce the notional link between the AQ and the relevant DSM criteria at varying levels of the disorder.

Sex Differences

The *extreme male brain theory of autism* posits that autism is an extreme version of male personality and cognitive characteristics. Evidence for this proposition comes from research examining the characteristics of ASD and the link to extremes of certain male-typical behaviors (Baron-Cohen, 2002). In line with the *extreme male brain theory*, men did score significantly higher than women on the total AQ score and on the three subscales of *social skills*, *communication*, and *imagination*. Counter to initial predictions, men and women did not differ on scores from the two subscales of *Attention Switching* and *Attention to Detail*.

One possible explanation as to why the proposed sex difference was not found for items assessing attention could be that the construct of attention as assessed by the AQ does not reflect the current DSM criteria. It is possible that attentional preference measures in the AQ may not directly map onto those found in clinical samples.

Specifically, difficulties in attention for clinical samples (e.g., joint attention, or “sticky” attention) may manifest differently than those found in non-clinical samples (Morgan et al., 2003; Redcay, 2008). Findings from the current thesis provide partial support to the extreme male brain theory as demonstrated by the significant difference between men and women with respect to scores on the total, social, communicative and imagination subscales.

Heritability

Research on the autism phenotype has uncovered milder deficits in phenotype individuals for social interaction, communication, and atypical behavior (Bishop et al., 2004; Yang & Gill, 2007). The present study supports findings that mild deficits in the autistic triad are observed among relatives. Consistent with initial predictions, the results showed that those with an immediate family of ASD endorsed more symptoms of autism on average than those without an immediate family history. When the individual subscales were examined, participants with a family history scored significantly higher than those without on the subscales of *social skills* and *communication* as predicted. However, the two groups did not differ on the *attention to detail*, *attention switching*, or *imagination* subscales.

It is possible that the broad autism phenotype represents a distinct high functioning group on the continuum and that only in the social realm are impairments evident given the deficit is primarily a social one. In other words, the autistic phenotype in high functioning groups shows only subtle indicators of the genetic expression of the disorder. Findings for the heritability of ASD provide partial evidence that milder, non-

clinical traits associated with autism are evident in an undergraduate sample (Wheelwright, Auyeung, Allison, & Baron-Cohen, 2010).

The results of the factor analyses are proposed to be weak evidence towards the ongoing debate concerning the non-fractionation of ASD deficits. Given the interrelations and overlap among social-communicative and behavioral deficits in ASD among non-clinical groups, it is likely that a degree of heterogeneity exists along a continuum of autism (Bishop et al., 2004; Ogle, 2009; Yang & Gill, 2007). As no model assessed in the current study provided significant parsimony, the AQ's dimensional structure in non-clinical adults remains to be fully explored. Also, using a large non-clinical sample, results provided partial support for *the extreme male brain theory of autism* and partial support for the broad autism phenotype.

Limitations

There are several important limitations pertaining to sample characteristics and the measurement of ASD traits. These limitations are discussed in the next section, followed by suggestions for future research.

One limitation concerning the sample tested was that due to the time constraints of using a large recruitment procedure, participants were not screened for the presence of other psychiatric disorders. It is possible that participants had a pre-existing psychiatric or neurodevelopment disorder. Consequently, co-morbidity with other relevant disorders may have impacted the ability to generalize the current results. Specifically, other pervasive development disorders (e.g., anxiety, ADHD) may have similar attentional and social-communicative deficits and as such, may have confounded the measures used to assess ASD traits. The presence of this confound may have affected the analyses in

unknown ways. To attend to this limitation, future research could pre-screen participants for co-morbid disorders (e.g. depression, social communication disorder). Alternatively, the sample could be restricted to only those with high scores on the AQ or who have a clinical diagnosis. If disorders that share similar characteristic symptoms are controlled for, it is expected that a clearer picture of how ASD manifests sub-clinically would become apparent. Specifically, it is predicted that each characteristic deficit (social and non-social) associated with clinical autism would be clearly identified in non-clinical samples.

A second limitation relating to sample characteristics was that only a relatively small sample of participants reported having an immediate family history of autism. Differences in scores for the AQ were all in the predicted direction however, it is possible that the sample of 52 (4%) individuals was not large enough to capture the hypothesized significant differences. It is likely that the sample size is an underestimation based on undergraduates in the current study having either a limited knowledge of family history, or medical diagnosis information from family. Thus, it is likely that members of the “non-related” group were actually relatives of those with autism and as a result, the difference between the two groups may have overlapped. One possible solution would be to use a more detailed family history screening including parental reports. Given that a non-significant trend was found in the predicted direction, it is expected that using a more stringent screening method would result in a significant difference on the AQ between those with and without a family history.

A third limitation concerns the method of assessing ASD traits in non-clinical samples. There is currently no universally agreed method for assessing autistic trait

endorsement in high functioning adults outside the clinical limits of the disorder. In particular, as symptoms no longer impair everyday functioning (e.g. undergraduate students) they become more subtle and difficult to quantify (Wheelwright, Auyeung, Allison, Baron-Cohen, 2010). It is therefore possible that findings from the current study may not be replicable in a clinical sample and it is less likely that findings apply to those with severe autism. To correct for this, other measures that assess the phenotype, clinical ASD, and non-clinical traits could be included using a clinically referred sample. Specifically, using measures that include parental reports and a comprehensive medical history would provide a clearer picture of the genetic susceptibility to autism and what role milder forms of autistic traits serve (Hurley et al., 2007).

The present study was designed to be part of a larger program of research examining a continuum of autism. This program consists of three parts. The first step was to assess the factor structure of the AQ as a measurement of autistic traits in non-clinical ASD samples. Although our 3-factor model of the AQ provided improvement to the other considered models, the goodness of fit, measures of internal consistency, and number of defined factors were all below the thresholds required. Findings are argued to be suggestive of possible AQ revisions for items assessing attention and re-conceptualizing items that relate closely together in a reduced model of the AQ. If addressed, it is also argued that the AQ would serve as a suitable measure for assessing autism phenotypes in non-clinical samples.

The second step in the program of research was to examine whether the characteristic deficits (social, communicative, and behavioral) associated with clinical autism occur as theoretically expected in an undergraduate sample. In line with

predictions, results from the present study provide evidence that mild forms of ASD deficits as measured by the AQ differ between men and women and between those with and without a family history of ASD. The findings provide supporting evidence for a continuum of autism that includes “autistic like” tendencies in the general population (e.g., broad autism phenotype).

The third step, to be completed, involves verifying the factor structure and psychometric properties of a revised AQ. Given the considerable debate regarding the AQ’s dimensional structure, a reliable and valid reduced factor model remains to be fully explored. Thus, the third step in the program of research would be to assess and utilize a revised version of the AQ. The goal is to demonstrate whether or not assessments of ASD traits share a similar representation when using the AQ as a quantitative measure for those who may lie outside a clinical diagnosis. Upon obtaining a stable factor structure, the third step is predicted to clarify how ASD traits manifest in sub-clinical groups (e.g. phenotype) and in the general population; representing a continuum of autism as measured by the AQ.

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Appendix A

Item loadings of the 5-factor solution of the Autism Spectrum Quotient

	Loadings
Factor 1: <i>Social skills</i>	
Q11: I find social situations easy.	.75
Q13: I would rather go to a library than a party.	.43
Q15: I find myself drawn more strongly to people than to things.	.54
Q17: I enjoy social chit-chat.	.72
Q22: I find it hard to make new friends.	.61
Q26: I frequently find that I don't know how to keep a conversation going.	.64
Q38: I am good at social chit-chat.	.80
Q44: I enjoy social occasions.	.72
Q47: I enjoy meeting new people.	.67
Factor 2: <i>Attention to detail</i>	
Q6: I usually notice car number plates or similar strings of information.	.58
Q10: In social group, I easily track several different people's conversations.	-.45
Q12: I tend to notice details that others do not.	.58
Q19: I am fascinated by numbers.	.43
Q23: I notice patterns in things all the time.	.53
Factor 3: <i>Communication</i>	
Q7: People frequently tell me what I've said is impolite, even though I think it is	.40
Q18: When I talk, it isn't always easy for others to get a word in edgeways.	.40
Q39: People often tell me that I keep going on and on about the same thing.	.46
Q45: I find it difficult to work out people's intentions.	.44
Factor 4: <i>Imagination</i>	
Q3: If I try to imagine something, it's very easy to create a picture in my mind.	.46
Q8: When I'm reading a story, I easily imagine what the characters look like.	.57
Q14: I find making up stories easy.	.43
Q20: When I'm reading a story, it's difficult to work out characters' intentions.	.52
Q21: I don't particularly enjoy reading fiction.	.47
Q40: When I was young, I enjoyed playing games pretending with other children.	.45
Q50: I find it very easy to play games with children that involve pretending.	.41
Factor 5: <i>Attention switching</i>	
Q25: It does not upset me if my daily routine is disturbed.	.53
Q46: New situations make me anxious.	.42

Appendix B

Item loadings of the three-factor solution of the Autism Spectrum Quotient

	Loadings
Factor 1: <i>Social communication</i>	
Q11: I find social situations easy.	.80
Q13: I would rather go to a library than a party.	.42
Q15: I find myself drawn more strongly to people than to things.	.52
Q17: I enjoy social chit-chat.	.67
Q22: I find it hard to make new friends.	.64
Q26: I frequently find that I don't know how to keep a conversation going.	.66
Q38: I am good at social chit-chat.	.80
Q44: I enjoy social occasions.	.72
Q46: New situations make me anxious.	.41
Q47: I enjoy meeting new people.	.68
Factor 2: <i>Attention to detail</i>	
Q6: I usually notice car number plates or similar strings of information.	.55
Q9: I am fascinated by dates.	.44
Q12: I tend to notice details that others do not.	.50
Q19: I am fascinated by numbers.	.40
Q23: I notice patterns in things all the time.	.57
Factor 3: <i>Understanding intentions</i>	
Q20: When reading a story, it's difficult to work out the characters' intentions.	.53
Q27: I find it easy to "read between the lines" when someone is talking to me.	.45
Q45: I find it difficult to work out people's intentions.	.56

Appendix C

5-Factor Model of the Autism Spectrum Quotient

Figure 2. 5-factor structure including factor loadings and estimated correlations. “e” refers to the error term associated with each item and “Q” refers to the question number found in the AQ questionnaire.

Appendix D

3-Factor Model of the Autism Spectrum Quotient

Figure 1. Factor structure of the 3-factor model including factor loadings and estimated correlations. “e” refers to the error term associated with each item and “Q” refers to the question number found in the AQ questionnaire.

Appendix E

Factor Model of the Autism Spectrum Quotient (Baron-Cohen et al. 2001)

Figure 3. Factor structure including factor loadings and estimated correlations. “e” refers to the error term associated with each item and “Q” refers to the question number found in the AQ questionnaire.