Aging and Autism Spectrum Disorder: Evidence from the Broad Autism Phenotype

# Gregory L. Wallace<sup>1</sup>, Jessica Budgett<sup>2</sup>, & Rebecca A. Charlton<sup>2</sup>\*

<sup>1</sup>Department of Speech and Hearing Sciences, The George Washington University,

Washington, DC, USA; <sup>2</sup>Department of Psychology, Goldsmiths University of London,

London, UK

\*Correspondence: Rebecca A. Charlton, Department of Psychology, Goldsmiths University of London, London, SE14 6NW, UK. Tel: +44 (0) 20 7919 7870; Fax: +44 (0) 20 7919 7873. Email: <u>r.charlton@gold.ac.uk</u>

Running Head: BAP and Aging

Number of Text Pages: 40; Number of Tables: 4; Number of Figures: o

Acknowledgments: We would like to thank the individuals who volunteered their time to contribute to this research. The authors have no conflicts of interest to declare.

### Lay Abstract

This study investigated for the first time the Broad Autism Phenotype (BAP), or the milder expression of characteristics of autism often found in relatives of people with the diagnosis, in the context of older adulthood. The current study also examined the association between the BAP and real world executive function, social support, and both depression and anxiety symptomatology. Based on self-ratings of autistic traits, 66 older adults (60+ years old, range=61-88) were split into BAP (n=20) and control (n=46) groups. Individuals in the BAP group, even after controlling for age, education level, sex, and health problems, exhibited more real world executive function problems in multiple domains, reported lower levels of social support, and self-rated increased depression and anxiety symptomatology compared to the control group. Level of social support was the strongest predictor of BAP traits across both groups, although real world executive function problems and depression symptomatology were also significant predictors. Moreover, when predicting anxiety and depression symptomatology, BAP traits were the strongest predictors above and beyond the effects of demographic factors, real world executive function problems, and social support levels. These findings suggest that the BAP in older adulthood imparts additional risks to areas of functioning that are known to be crucial to aging-related outcomes in the context of typical development. These results might in turn inform aging in autism spectrum disorder, which has been largely unexplored to date.

#### Abstract

This study investigated for the first time the Broad Autism Phenotype (BAP) in the context of older adulthood and its associations with real world executive function, social support, and both depression and anxiety symptomatology. Based on self-ratings of autistic traits, 66 older adults (60+ years old, range=61-88) were split into BAP (n=20) and control (n=46) groups. Individuals in the BAP group, even after controlling for age, education level, sex, and health problems, exhibited more real world executive function problems in multiple domains, reported lower levels of social support, and self-rated increased depression and anxiety symptomatology compared to the control group. Regression analysis revealed that level of social support was the strongest predictor of BAP traits across both groups, although real world executive function problems and depression symptomatology were also significant predictors. Moreover, when predicting anxiety and depression symptomatology, BAP traits were the strongest predictors above and beyond the effects of demographic factors, real world executive function problems, and social support levels. These findings suggest that the BAP in older adulthood imparts additional risks to areas of functioning that are known to be crucial to aging-related outcomes in the context of typical development. These results might in turn inform aging in autism spectrum disorder, which has been largely unexplored to date.

Keywords: broad autism phenotype, autism, aging, older adulthood, executive

function, anxiety, depression, social support

# Introduction

The Broad Autism Phenotype (BAP) is a set of subclinical characteristics associated with autism spectrum disorder (ASD). Atypical social-communication and restricted and repetitive patterns of behavior, which are pathognomonic with ASD, are now conceptualized as dimensional, varying from subclinical to clinical (i.e., ASD) levels of expression, throughout the general population (Constantino & Todd, 2003; Ronald et al., 2005; Ruzich et al., 2015; Skuse et al., 2005). These ASD features are more prevalent in unaffected first-degree family members of individuals with an ASD diagnosis than found in the general population (Sasson et al., 2013a; Wheelwright et al., 2010). This overrepresentation of BAP characteristics is likely due to the high heritability of ASD as evidenced by numerous twin and family studies (for review, see Ronald & Hoekstra, 2011). Thus, there is susceptibility for ASD to be inherited as mild traits, which are qualitatively similar to the defining characteristics of ASD (Hurley et al., 2007). This milder expression of autistic traits, known as the BAP, has been noted since Kanner's (1943) original description of the disorder. For example, he suggested that obsessiveness was found in the family backgrounds of many these children. Prevalence rates of the BAP vary from study to study but ~20-50% of family members of an individual with ASD have been reported to display at least one BAP feature (Dawson et al., 2007).

Early studies of the BAP primarily utilized interviews and similar assessment tools (Bolton et al., 1994; Landa et al., 1992; Piven et al., 1990); however, these instruments can be somewhat laborious to administer and require training, which limits their usage. The Broad Autism Phenotype Questionnaire (BAPQ; Hurley et al., 2007) was developed to provide a quick and easily delivered assessment of the BAP. The BAPQ assesses three key ASD diagnostic domains: social problems (aloof personality), pragmatic language difficulties, and rigid personality (Hurley at al., 2007). The BAPQ has shown good sensitivity and specificity (both >70%) for identifying the BAP. Parents of children with ASD (mean age in their 40s) scored significantly higher across all three scales than parents of typically developing children (Hurley et al., 2007; Sasson et al., 2013b). Thus far, there has been little work examining lifespan changes across the BAP (or ASD) that extends beyond young to middle adulthood.

Only recently has there been a focus on adulthood and aging in ASD. The knowledge base on aging in ASD is woefully small (though see case series by: James et al., 2006; van Niekerk et al., 2011 and group studies by: Lever & Geurts, 2015; van Heijst & Geurts, 2015) compared to childhood and even adolescence, which have garnered the lion's share of research attention and funding (Mukaetova-Ladinska, et al., 2012). The first individuals diagnosed with ASD in the 1940s are only now reaching old age meaning that opportunities for studying aging in ASD to date have been limited. A combination of a rapidly growing elderly population and increases in adulthood ASD diagnoses poses a growing social and financial challenge for society (Happé & Charlton, 2011; Piven et al., 2011). Although studies are now beginning to investigate age-related changes in ASD across the lifespan, conclusions of cross-sectional studies are stymied by changes in diagnostic criteria and awareness of ASD over the past 75 years, as well as current difficulties recruiting older individuals with ASD to research studies (Stuart-

Hamilton et al., 2009). Studies utilizing the BAP to examine ASD traits across the lifespan, including older adulthood, may be less impeded by the difficulties inherent to studies of clinical populations.

The majority of BAP studies have focused on school aged siblings and parents of children with ASD (Gerdts & Bernier, 2011). Investigating the BAP among older adults might provide important clues about the aging process in ASD. Moreover, investigating aging in the BAP is also important to identify risk factors that might affect the management and planning for appropriate services for this elderly population. The BAP is likely associated with other important areas of cognitive, behavioral, and social functioning previously shown to impact outcomes in aging populations. One domain repeatedly implicated is executive function (EF), which is an umbrella term referring to a number of skills involving higher-order cognitive processes (e.g., inhibitory control, working memory, flexibility/shifting) that ultimately aid goal-directed behavior and problem-solving. EF deficits are a central part of normal age-related cognitive decline with different components of EF (e.g., set-shifting, working memory) declining at different rates across adult development (Jurado & Rosselli, 2007). Similarly, EF impairments particularly in flexibility/shifting (using both cognitive tasks like the Wisconsin Card Sorting Test and real-world measures) are well established in the ASD literature among both children and adults (for review, see Hill, 2004; Kenworthy et al., 2008). However, findings for the link between EF deficits and the BAP have been mixed with some studies documenting EF deficits (particularly in the areas of planning and cognitive flexibility) among parents of children with ASD in comparison to parents of

typically developing children (e.g., Delorme et al., 2007; Hughes et al., 1997; Piven & Palmer, 1997), while others do not (e.g., Boltë & Poustka, 2006; Losh et al., 2009).

The social environment in which older adults live has been well studied in terms of being a modifiable factor that may help protect cognitive function (Shankar at al., 2013) and improve health (Hawkley et al., 2009) and wellbeing (Liu et al., 2014). Older people are vulnerable to both social isolation and loneliness due to loss of loved ones and decreased mobility or income. These factors have a significant negative effect on quality of life in older adults, including associations with depression (Isaac et al., 2009; Liu et al., 2014), disability (Lund et al., 2010), increased mortality risk (Shiovitz-Ezra, 2010), and deficits in cognition including EF (Shanker et al., 2013; Tun et al., 2013). A survey conducted by the National Autistic Society (NAS) in 2008 of 1,400 individuals with ASD and their caregivers showed that of those aged 65 and older living independently in the UK, 46% had most support provided by family and only 8% had most support via professionals (Rosenblatt, 2008). A later NAS survey of 1,412 adults aged over 18 years old completed in 2012 found that 73% of respondents with ASD aged 55 or older had 3 friends or fewer and 65% reported that their main friends were family members or caregivers (Bancroft et al., 2012). This poses a potential problem for the aging ASD population as they start to lose family members, which could in turn, result in more severe and devastating effects in terms of isolation, loneliness and depression in old age (Happé & Charlton, 2011). Greater levels of BAP characteristics in young adults/university students are associated with more interpersonal problems and fewer, shorter, and less satisfying friendships (Wainer et al., 2013). It is therefore not

surprising that BAP characteristics are also associated with higher levels of loneliness and social isolation (Jobe & White, 2007). Whether and how strongly loneliness and social isolation are associated with BAP traits in older adults remains unexplored.

Depression and anxiety during older adulthood can result in reduced quality of life, such as increased risk for dementia, and onset of physical and social impairments (Stuart-Hamilton, 2012). Adults with ASD are more likely to experience comorbid depression and anxiety, compared with their same age (young and mid-life adult) neurotypical peers (Croen et al., 2015). Compared to base rates found in the general population, higher rates of depression and anxiety have also been documented in family members of individuals with ASD (Ingersoll et al., 2011; Wilcox et al., 2003) and in individuals from the general population with high levels of BAP traits (Wainer et al., 2013). Risk factors such as social isolation or exclusion, smaller social networks, difficulties in social problem solving, and experiences of bullying may predispose individuals with elevated BAP traits to develop depression (Rosbrook & Whittingham, 2010). Therefore, we examined whether BAP traits (along with other factors linked with depression and anxiety symptomatology [e.g., EF, social support, etc.] in [Wallace et al., in press] and out of the context of ASD), predict either anxiety or depression symptomatology, given their potentially exacerbating influence.

This study investigates BAP traits among older adults aged 60 years and older and whether elevated BAP traits are associated with EF problems, limited social support, and greater depression and/or anxiety symptomatology. These domains of functioning are affected not only in ASD and the BAP (in childhood, adolescence,

and/or young adulthood), but also in typical age-related decline. Elderly adults with elevated BAP traits could experience additional risk in which a faster deterioration or greater prevalence of problems in these domains occurs. It is predicted that: 1) older adults with elevated BAP traits will show greater EF deficits than those with lower levels of BAP traits; 2) elevated BAPQ scores will be associated with lower levels of social support; and 3) elevated BAPQ scores will be correlated with increased depression and anxiety symptomatology.

## Methods

#### <u>Participants</u>

Adults aged 60 years and older (range 61-88 years old) were recruited to participate in the study. Individuals were recruited via community outreach including the University of the Third Age in Ealing, two general practice surgeries in West London, and by posting information about the study on social media sites for older adults. The study was also advertised on online forums for relatives of those with an ASD diagnosis (such as <u>http://www.talkaboutautism.org.uk</u> and

<u>http://www.autismspeaks.org</u>) in order to oversample for the presence of BAP traits.

Sixty-seven individuals completed the study, however one individual (female, aged 75 years) did not complete the BAPQ and is excluded from the analyses. The final sample includes 66 individuals (33 males, 33 females) aged 61-88 years old (see Table 1 for demographic details). Twenty individuals meet criteria for inclusion in the BAP group due to scoring above cut-off on the BAPQ, of these 20 individuals seven had a relative with ASD. The remaining 46 individuals were categorized as control older adults (COA); four had a relative with an ASD diagnosis. Means, standard deviations, and ranges for the Total BAPQ and its subscales are presented for each of the groups in Table 2.

# <u>Materials</u>

The study was approved by the Goldsmiths University Ethics Committee, and all research was carried out according to the Declaration of Helsinki and individuals provided informed consent. Questionnaires were administered either online using 'Unipark Questback' software (<u>http://www.unipark.info</u>) or in a paper format distributed as described above. Forty participants completed the questionnaire online, and 26 participants completed the paper questionnaire. In both online and paper formats, an information sheet described the study and individuals were required to indicate consent by answering a question (online format) or sign a consent form (paper format).

Demographic information was recorded including age and sex. Marital status was coded as single, married or in a significant relationship, or divorced. Participants were asked whether they had a relative with ASD (other neurological disorders were noted), and also asked if they suspected ASD in themselves. Five individuals who met criteria for BAP suspected that they may themselves have an ASD, but none had or were seeking a diagnosis. Highest education level achieved was recorded (see Table 1).

Participants completed additional guestionnaires based on self-rating of their behavior and abilities. The BAPQ (Hurley et al., 2007) was used to rate autistic-type traits; the Behavior Rating Inventory of Executive Function – Adult version (BRIEF-A; Roth et al., 2005) measured EF problems (with the Behavior Regulation Index [BRI] and Metacognition Index [MCI] utilized as the variables of interest rather than individual scales in order to limit the number of statistical comparisons), and the Duke Social Support Index (DSSI; Koenig et al., 1993) measured amount of perceived social support. Depression and anxiety symptomatology were assessed using the Geriatric Depression Scale (GDS; Yesavage et al., 1983) and the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). One participant failed to complete the GDS and four participants failed to complete the BAI online. Experimenter error resulted in systematic omission (i.e., across all participants) of four items from the BRIEF-A. Prorated scores were substituted (by taking the average of the other items composing that scale) for these items in order to derive age-norm-referenced T scores. Note that use of raw vs. T scores from the BRIEF-A did not alter the pattern of findings reported below.

# Statistical Analysis

All statistical analyses were performed in SPSS (version 22.0; IBM Corp., 2013). Differences between the BAP and COA groups were assessed using ANOVA and Chi-Square, as appropriate. Associations between BAP traits and other variables of interest were also examined using correlational analyses. Two sets of regression analyses were performed. The first examines how well demographic variables (age, marital status, highest education level, having a relative with an ASD diagnosis) and clinical ratings scales (i.e., the raw BRI and MCI scores from the BRIEF-A, GDS, BAI, and DSSI scores) predict BAP traits. The second set examines the variables (including demographic characteristics as well as BAPQ, BRIEF-A [MCI and BRI raw scores], DSSI, and GDS or BAI scores) that best explain depression and anxiety symptomatology, respectively.

# Results

# <u>Demographics</u>

Individuals in the BAP group were significantly older than those in the COA group (see Table 1). There were no differences between the groups in terms of sex ratio, marital status, highest education level, or medical problems.

# <Table 1 here>

# Group differences

Group differences were observed for each of the scales measuring self-report EF problems, social support, and both depression and anxiety symptomatology. Individuals in the BAP group had scores indicating more EF problems, less social support, and more depression and anxiety symptomatology than the COA group (see Table 3). As the BAP group was slightly older than the COA group, the analysis was

repeated controlling for age: all group differences remained significant with or without age entered as a covariate.

For the GDS and BAI, scores can be categorized as "normal," "mild," or "severe." For depression scores (GDS) 40 individuals had normal (BAP=3, COA=37), 16 had mild (BAP=8, COA=8), and nine had severe (BAP=9) levels of depression, which differed significantly between the BAP and COA groups ( $X^2$ =26.44, p<.001). For anxiety scores (BAI), 50 individuals were categorized as having low levels of anxiety (BAP=10, COA=40), 10 were classified as having moderate anxiety (BAP=8, COA=2), and two were classified as having high levels of anxiety (BAP=8, COA=2), and two were classified as having bigh levels of anxiety (BAP=2), which again was significantly different in the BAP and COA groups ( $X^2$ =17.76, p<.001). For the BRIEF-A, a T-score of  $\geq$ 65 can be considered clinically elevated EF problems. Fourteen individuals met criteria for clinically elevated BRI scores (BAP, n=11; COA, n=3;  $X^2$ =19.60, p<.001) and 12 individuals were above this cut-off on the MI (BAP, n=7; COA, n=5;  $X^2$ =5.46, p=.02), both of which differed significantly between the BAP and COA groups.

<Tables 2 & 3 here>

### **Correlations**

Correlations between each self-rated scale and age were performed. Age was positively correlated with metacognitive problems (MCI from the BRIEF-A) and depression symptomatology (GDS). No other significant correlations with age were observed. See Table 4. Significant correlations were observed between scores on the BAPQ, and all of the following: the BRIEF-A BRI and MCI scores, the GDS and BAI scores, and the DSSI social support score (*p*s<.001). See Table 4.

Correlations were repeated for each group separately. Although the sample size in the BAP group was smaller than the COA group, correlations between variables were generally more robust, possibly due to the larger variance within the BAP group. Fisher's r to z transformations indicated that the correlation was significantly greater in the BAP group compared to the COA group for the associations between BAPQ and both BRI. See Table 4.

<Table 4 here>

# Stepwise Multiple Regression

Regression analysis was performed to explore which variables best predicted BAPQ ratings. Included as independent variables were the demographic variables age, marital status, highest education level, and whether a relative had ASD; as well as BRI and MCI from the BRIEF-A, GDS, BAI, and social support (DSSI) scores. The model significantly explained 78.7% of the variance in BAPQ scores (F(4,54)=49.75, p<.001). Social support accounted for 63.5% of the variance (Beta=-.40, p<.001), with both subscales from the BRIEF-A contributing to the model (BRI=10.3%, Beta=.45, p<.001; MCI=2.1%, Beta=-.24, p=.03) and GDS scores also explaining a significant portion of the variance (GDS= 2.8%, Beta=.34, p=.002) in BAPQ scores. If a group-by-age interaction

term is also included, the results do not change and this term does not contribute significantly to the model.

Regression analyses were performed to explore which variables best predicted, in turn, depression (GDS) and anxiety (BAI) ratings. Included as independent variables were the demographic variables age, marital status, highest education level, and whether a relative had ASD; as well as BAPQ ratings, BRI and MCI scores from the BRIEF-A, social support (DSSI) scores and either GDS or BAI ratings (depending on analysis).

For GDS scores, the model significantly explained 68.5% of the variance (F(2,58)=63.11, p<.001). BAPQ scores accounted for 59% of the variance (Beta=.57, p<.001), with MCI from the BRIEF-A explaining an additional 9.5% of the variance (Beta=.37, p<.001) in GDS scores.

For BAI scores, the model significantly explained 52.8% of the variance (F(2,58)=32.47, p<.001). BAPQ scores accounted for 47.4% of the variance (Beta=.41, p=.005), with GDS scores explaining an additional 5.5% of the variance (Beta=.37, p=.012) in BAI scores.

# Alternative cut-off for BAP

In order to insure that results were robust, the analyses examining group differences and correlations for each group were repeated using the BAPQ cut-off described by Sasson et al. (2013b), rather than the original criterion (Hurley at al., 2007). Using a cut-off of >3.55 for males and >3.17 for females, the BAP group was reduced in

size to n=16, with the COA group n=50. Using these alternative cut-off scores had no impact on the results. All group differences remained significant. The patterns of correlational results showed some changes with the different sample. Age correlated with BAPQ score for the BAP group only, although according to Fisher's r to z transformations the correlation was not significantly different from that found in the COA group. Within the BAP group, the association between BAPQ and GDS was no longer statistically significant (r=.42, p=.11); and within the COA group the association between BAPQ and BAI became statistically significant (r=.33, p=.03).

# Discussion

The aim of this study was to examine the presence of BAP traits in an oversampled elderly population, and the association between these traits and self-rated EF problems, depression and anxiety symptomatology, and levels of social support. To our knowledge BAP traits have not previously been examined in a population of older adults. Within the sample described here, of the 20 individuals who were included in the BAP group, seven not only scored above cut-off on the BAPQ but also had a relative with ASD. This supports BAP traits existing across a continuum during late life, consistent with numerous studies documenting variance in these traits in childhood and adolescence (e.g., Constantino & Todd, 2003; Ronald et al., 2005; Skuse et al., 2005). Importantly, level of BAP traits did not correlate with age, suggesting that BAP traits are stable across older adulthood. Within this sample, similar proportions of males and females were present in the BAP and COA groups. This is also

consistent with previous findings suggesting that unlike ASD, which affects considerably more males than females, BAP traits are expressed comparably in males and females (Klusek et al., 2014; Piven et al., 2007).

On both subscales of the BRIEF-A (BRI and MCI), the BAP group demonstrated more self-reported EF problems than the COA group, and these differences were independent of age. These findings are in keeping with previous studies of parents of children with ASD that have demonstrated subtle deficits in aspects of EF, particularly cognitive flexibility (e.g., Piven & Palmer, 2007). Results suggest that older adults with more BAP traits show more real world EF problems than COA in multiple domains, which could have clinical implications. The emergence of EF difficulties is common in neurotypical aging (Jurado & Rosselli, 2007); thus, elevated EF problems among older adults with BAP traits (compared to COA adults) suggests the possibility of additive impacts that might impart accelerated cognitive decline (Geurts & Vissers, 2012; though Lever & Geurts, 2015 failed to replicate these initial cross-sectional findings). Correlational analyses also revealed a link between BAP traits and behavior regulation difficulties in particular for the BAP group, but not the COA group. Finding this correlational pattern is perhaps unsurprising given that the Behavior Regulation Index of the BRIEF includes the Shift subscale (i.e., a measure of cognitive and behavioral (in)flexibility), which is the peak difficulty in prior studies of both children (Granader et al., 2014) and adults (Wallace et al., in press) with ASD. BRIEF-A BRI scores in particular, as well as MCI scores to a lesser degree, contributed unique variance in predicting BAP-

Q scores in the context of regression analyses, further underlining the associations between real-world EF and BAP traits in this sample of older adults.

Older adults in the BAP group were more likely to have smaller social network sizes, including reduced frequency of social interactions and less satisfaction with quality of social contacts, than those in the COA group. Moreover, regression analyses indicated that level of social support was the strongest predictor of BAP trait scores. These results are in line with previous studies of young adults where BAP characteristics were associated with social isolation, loneliness, and less social motivation (Jobe & White, 2007; Wainer et al., 2013). Since neurotypical older adults are also at greater risk of both social isolation and loneliness due to multiple risk factors (such as bereavement, ill health, decreased mobility, and less income), aging, BAP individuals could be experiencing exacerbated vulnerability. Individuals with elevated BAP traits are likely to already have increased social difficulties (Wheelwright et al., 2010) and fewer and lower quality friendships (Losh et al., 2008); therefore, they may experience increased isolation and loneliness as they transition to old age and experience its concomitant risk factors. Extrapolating to individuals with ASD, they may have increased reliance on a few close family members or friends so that when these support contacts are lost during old age, it may have a more devastating effect, leading to increased isolation and feelings of loneliness (Happé & Charlton, 2011; Bancroft et al., 2012).

The BAP group also endorsed greater levels of depression and anxiety symptomatology than the COA group. Elevated rates of depression and anxiety have been documented among family members of individuals with ASD (Ingersoll & Wainer,

2011; Wilcox et al., 2003). Although there is a lack of research investigating mood in later life in ASD or the BAP, our results suggest that these elevated rates of depression and anxiety continue into old age. There is also a significant association between BAP traits and both depression and anxiety symptomatology in the general population. At least one study of young adults suggests that the relationship between BAP traits and both depression and anxiety symptoms is mediated by social problem-solving abilities and teasing/bullying history (Rosbrook & Whittingham, 2010). Neurotypical aging in and of itself presents a number of additional risk factors for depression, such as social isolation, loneliness (Taylor & Lynch, 2004), increased likelihood of bereavement, and reduced sense of purpose (Anstey et al., 2007). Increased fears in old age have been associated with elevated levels of anxiety as well (Woods, 1999). Taken together, these studies suggest another potential additive effect in which BAP traits and aging together compound one's risk of developing depression and/or anxiety.

Finally, regression analyses revealed that BAPQ scores explained the most variance in depression or anxiety ratings, respectively, over social support levels, EF problems, and the other form of internalizing psychopathology (i.e., anxiety or depression symptomatology). This is particularly striking given the well-established links between social support and mood, for example, among older adult populations. These findings only serve to underscore the potential importance of ASD traits to critical outcomes, such as co-morbid psychopathology, in older adulthood. While BAP traits were the largest contributors to depression and anxiety ratings, respectively, they were not the only ones. Metacognitive EF problems also contributed to variance in

depression symptomatology scores, which is highly consistent with findings from a recent study showing this same specific association in a sample of 35 young adults with ASD (Wallace et al., in press). Depression ratings contributed significant variance to anxiety scores, which is a well-replicated finding in the general population, including samples of older adults (e.g., Spinhoven et al., 1997).

Given the paucity of evidence to date, this study of BAP traits during older adulthood provides potentially important clues to areas of concern and their functional associations during aging in ASD. However, extending these and other types of studies to older adults with ASD diagnoses is much needed given the current dearth of research on aging in ASD. The present study has several limitations to consider. For example, it cannot be ruled out that lowered levels of social support and elevations in EF problems, anxiety and depression symptomatology, all concordant with elevated BAP traits, don't represent broader adjustment related issues, particularly given this study's reliance on self-report methods alone. Moreover, the sample size is relatively small; thus, somewhat limiting statistical power to detect potentially subtle group differences or relationships among key variables. Finally, questions concerning aging in particular and efforts to address the directionality of influences between the BAP and areas important to aging-related outcomes, might be best answered utilizing longitudinal designs, which future research should endeavor to address. Nevertheless, in spite of these shortcomings, this investigation represents a first step toward attempting to tackle the challenge of better understanding aging and its (a)typicality in the BAP and in ASD.

#### References

Anstey, K. J., von Sanden, C., Sargent-Cox, K., & Luszcz, M. A. (2007). Prevalence and risk factors for depression in a longitudinal, population-based study including individuals in the community and residential care. *American Journal of Geriatric Psychiatry*, 15, 497-505.

Bancroft, K., Batten, A., Lambert, S., & Madders, T. (2012). *The way we are: Autism in 2012*. London, UK: The National Autistic Society.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, 56, 893-897.

Bolton, P., Macdonald, H., Pickles, A., Rios, P., Goode, S., Crowson, M., et al. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry*, 35, 877-900.

Constantino, J. N., & Todd, R. D. (2003). Autistic traits in the general population: a twin study. *Archives of General Psychiatry*, *60*, 524-530.

Croen, L. A., Zerbo, O., Qian, Y., Massolo, M. L., Rich, S., Sidney, S., & Kripke, C.

(2015). The health status of adults on the autism spectrum. Autism.

Dawson, G., Estes, A., Munson, J., Schellenberg, G. D., Bernier, R., Abbott, R., et al. (2007). Quantitative assessment of autism symptoms in autism probands and parents: broader phenotype autism symptom scale. *Journal of Autism and Developmental Disorders*, 37, 523-536. Delorme, R., Goussé, V., Roy, I., Trandafir, A., Mathieu, F., Mouren-Siméoni, M.

C., . . . Leboyer, M. (2007). Shared executive dysfunctions in unaffected relatives of patients with autism and obsessive compulsive disorder. *European Psychiatry*, *22*, 32-38.

Gerdts, J., & Bernier, R. (2011). The broader autism phenotype and its implications on the etiology and treatment of autism spectrum disorders. *Autism Research and Treatment*, Article ID 545901. doi:10.1155/2011/545901

Geurts, H. M., & Vissers, M. E. (2012). Elderly with autism: executive functions and memory. *Journal of Autism and Developmental Disorders*, 42, 665-675.

Granader, Y., Wallace, G. L., Hardy, K. K., Yerys, B. E., Lawson, R. A., Rosenthal, M., Wills, M. C., Dixon, E., Pandey, J., Penna, R., Schultz, R. T., & Kenworthy, L. (2014). Characterizing the factor structure of parent reported executive function in autism spectrum disorders: the impact of cognitive inflexibility. *Journal of Autism and Developmental Disorders*, 44, 3056-3062.

Happé, F., & Charlton, R. A. (2011). Ageing in Autism Spectrum Disorders: A Mini-Review. *Gerontology*, *58*, 70-78.

Hawkley, L. C., Thisted, R. A., Masi, C. M., & Cacioppo, J. T. (2010). Loneliness predicts increased blood pressure: Five-year cross-lagged analyses in middle-aged and older adults. *Psychology & Aging*, 25, 132-141.

Hill, E. L. (2004). Executive dysfunction in autism. *Trends Cogn Sci*, 8, 26–32. Hughes, C., Leboyer, M., & Bouvard, M. (1997). Executive function in parents of children with autism. *Psychological Medicine*, 27, 209-220. Hurley, R. S. E., Losh, M., Parlier, M., Reznick, J. S., & Piven, J. (2007). The broad autism phenotype questionnaire. *Journal of Autism and Developmental Disorders*, 37, 1679-1690.

Ingersoll, B., Meyer, K., & Becker, M. W. (2011). Increased rates of depressed mood in mothers of children with ASD associated with the presence of the broader autism phenotype. *Autism Research*, *4*, 143-148.

Isaac, V., Stewart, R., Artero, S., Ancelin, M.L., & Ritchie, K. (2009). Social activity and improvement in depressive symptoms in older people: a prospective community cohort study. *American Journal of Geriatric Psychiatry*, 17, 688-696.

James, I. A., Mukaetova-Ladinska, E., Reichelt, F. K., Briel, R., & Scully, A.

(2006). Diagnosing Aspergers syndrome in the elderly: A series of case presentations. International Journal of Geriatric Psychiatry, 21, 951-960.

Jobe, L. E., & White, S. W. (2007). Loneliness, social relationships, and a broader autism phenotype in college students. *Personality and Individual Differences*, 42, 1479-1489.

Jurado, M. B., & Rosselli, M. (2007). The Elusive Nature of Executive Functions: A Review of our Current Understanding. *Neuropsychology Review*, 17, 213-233.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217-249.

Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, *18*, 320-338. Klusek, J., Losh, M., & Martin, G. E. (2014). Sex differences and within-family associations in the broad autism phenotype. *Autism*, 18, 106-166.

Koenig, H. G., Westlund, R. E, George, L. K, Hughes, D. C., Blazer, D. G., & Hybels, M. P. H. (1993). Abbreviating the Duke Social Support Index for Use in Chronically III Elderly Individuals. *Psychosomatics*, *34*, 61-69.

Landa, R., Piven, J., Wzorek, M.M., Gayle, J.O., Chase, G.A., & Folstein, S.E. (1992). Social language use in parents of autistic individuals. *Psychological Medicine*, *22*, 245-254.

Lever, A. G., & Geurts, H. M. (2015). Age-related differences in cognition across the adult lifespan in autism spectrum disorder. *Autism Research*.

Liu, L., Gou, Z., & Zuo, J. (2014). Social support mediates loneliness and depression in elderly people. *Journal of Health Psychology*.

Losh, M., Adolphs, R., Poe, M. D., Couture, S., Penn, D., Baranek, G. T., & Piven, J. (2009). Neuropsychological profile of autism and the broad autism phenotype. *Archives of General Psychiatry*, 66, 518-526

Losh, M., Childress, D., Lam, K., & Piven, J. (2008). Defining key features of the broad autism phenotype: A comparison across parents of multiple- and singleincidence autism families. *American Journal of Medical Genetics, Part B*,

Neuropsychiatric Genetics, 147, 424-433.

Luijendijk, H., et al. (2008). Incidence and recurrence of late-life depression. Archive of General Psychiatry, 65, 1394-1401. Lund, R., Nilsson, C. J., & Avlund, K. (2010). Can the higher risk of disability onset among older people who live alone be alleviated by strong social relations? A longitudinal study of non-disabled men and women. *Age & Ageing*, 39, 319-326.

Mukaetova-Ladinska, E. B., Perry, E., Baron, M., Povey, C., et al. (2012). Ageing in people with autistic spectrum disorder. *International Journal of Geriatric Psychiatry*, 27, 109-118.

Piven, J., Rabins, P., et al. (2011). Autism spectrum disorders in older adults: Toward defining a research agenda. *Journal of the American Geriatrics Society*, *59*, 2151-2155.

Piven, J., Gayle, J., Chase, G. A., et al. (1990). A family history study of neuropsychiatric disorders in the adult siblings of autistic individuals. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 177-183.

Piven, J., Palmer, P., Landa, R., Santangelo, S., Jacobi, D., & Childress, D. (1997). Personality and language characteristics in parents from multiple-incidence autism families. *American Journal of Medical Genetics*, 74, 398-411.

Piven, J., & Palmer, P. (1997). Cognitive deficits in parents from multipleincidence autism families. *Journal of Child Psychology and Psychiatry*, 38(8), 1011–1021.

Robinson, E., Koenen, K. C., McCormick, M. C., Munir, K., Hallett, V., Happé, F., et al. (2011). Evidence that autistic traits show the same etiology in the general population and at the quantitative extremes (5%, 2.5% and 1%). *Archives of General Psychiatry*, 68, 1113-1121. Ronald, A., Happé, F., & Plomin, R. (2005). The genetic relationship between individual differences in social and nonsocial behaviours characteristic of autism. *Developmental Science*, *8*, 444-458.

Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: a decade of new twin studies. *American Journal of Medical Genetics, Part B, Neuropsychiatric Genetics*, 156, 255-274.

Rosbrook, A., & Whittingham, K. (2010). Autistic traits in the general population: What mediates the link with depressive and anxious symptomology? *Research in Autism Spectrum Disorders*, 4, 415-424.

Rosenblatt, M. (2008). *I Exist: The message from adults with autism in England*. London, UK: The National Autistic Society.

Roth, R. M., Isquith, P.K., & Gioia, G. A. (2005). Behavioral Rating Inventory of

Executive Function-Adult version. *Psychological Assessment Resources*, Inc., Lutz, FL.

Ruzich, E., Allison, C., Smith, P., Watson, P., Auyeung, B., Ring, H., & Baron-Cohen, S. (2015). Measuring autistic traits in the general population: a systematic review of the Autism-Spectrum Quotient (AQ) in a nonclinical population of 6,900 typical adult males and females. *Molecular Autism*, 6.

Sasson, N. J., Lam, K. S. L., Childress, D., Parlier, M., Daniels, J. L., & Piven, J. (2013b). The broad autism phenotype questionnaire: prevalence and diagnostic classification. *Autism Research*, *6*, 134-143.

Sasson, N. J., Lam, K. S., Parlier, M., Daniels, J. L., & Piven, J. (2013a). Autism and the broad autism phenotype: familial patterns and intergenerational transmission. *Journal of Neurodevelopmental Disorders*, *5*, 11.

Shankar, A., Hamer, M., McMunn, A., & Steptoe, A. (2013). Social isolation and Ioneliness: Relationships with cognitive function during 4 years of follow-up in the English Longitudinal Study of Ageing. *Psychosomatic Medicine*, 75, 161-170.

Shiovitz-Ezra, S., & Ayalon, L. (2010). Situational versus chronic loneliness as risk factors for all-cause mortality. *International Psychogeriatrics*, *22*, 455-462.

Skuse, D. H., Mandy, W. P., & Scourfield, J. (2005). Measuring autistic traits: heritability, reliability and validity of the Social and Communication Disorders Checklist. *British Journal of Psychiatry*, *18*7, 568-572.

Spinhoven, P., Ormel, J., Sloekers, P. P. A., Kempen, G. I. J. M., Speckens, A. E. M., & Van Hemert, A. M. (1997). A validation study of the Hospital Anxiety and Depression Scale (HADS) in different groups of Dutch subjects. *Psychological Medicine*, *27*, 363-370.

Stuart-Hamilton, I., Griffith, G., Totsika, V., Nash, S., Hastings, R. P., Felce, D., & Kerr, M. (2009). The circumstances and support needs of older people with Autism. Report for the Welsh Assembly Government. Cardiff, Welsh Assembly.

http://wales.gov.uk/docs/dhss/report1006220lderpeoplewithautismreporten.pdf

Taylor, M. G., & Lynch, S. M. (2004). Trajectories of impairment, social support and depressive symptoms in later life. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 59, 238-46. Tun, P. A., Miller-Martinez, D., Lachman, M. E., & Seeman, T. (2013). Social strain and executive function across the lifespan: The dark (and light) sides of social engagement. *Neuropsychology, Development, and Cognition, Section B, Aging, Neuropsychology, and Cognition, 20*, 320-338.

van Heijst, B. F. C., & Geurts, H. M. (2015). Quality of life in autism across the lifespan: A meta-analysis. *Autism*, 19, 158-167.

van Niekerk, M. E., Groen, W., Vissers, C. T., van Driel-de Jong, D., Kan, C. C., & Oude Voshaar, R. C. (2011). Diagnosing autism spectrum disorders in elderly people. *International Psychogeriatrics*, 23, 700-710.

Wainer, A. L., Block, N., Donnellan, M. B., & Ingersoll, B. (2013). The broader autism phenotype and friendships in non-clinical dyads. *Journal of Autism and Developmental Disorders*, 43, 2418-2425.

Wallace, G. L., Kenworthy, L., Pugliese, C. E., Popal, H. S., White, E. I., Brodsky, E., & Martin, A. (*in press*). Real-world executive functions in adults with autism spectrum disorder: Profiles of impairment and associations with adaptive functioning and co-morbid anxiety and depression. *Journal of Autism and Developmental Disorders*.

Wheelwright, S., Auyeung, B., Allison, C., & Baron-Cohen, S. (2010). Defining the broader, medium and narrow autism phenotype among parents using the Autism Spectrum Quotient (AQ). *Molecular Autism*, 1, 1–10.

Wilcox, J. A., Tsuang, M. T., Schnurr, T., & Baida-Fragoso, N. (2003). Casecontrol family study of lesser variant traits in autism. *Neuropsychobiology*, 47, 171-177. Woods, R. T. (1999). Mental health problems in later life. In R.T. Woods (ed.)

*Psychological Problems of Ageing*. Chichester: Wiley, 73-110.

Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V., Adey, M., & Leirer,

V. O. (1983). Development and validation of a geriatric depression screening scale: a preliminary report. *Journal of Psychiatric Research*, 17, 37-49.

**Table 1**. Demographic characteristics of the broad autism phenotype and control

groups.

		Whole	BAP	COA	Group differences
		Sample	(n =20)	(n=46)	
		(n=66)			
Age, M (SD), years		70.80	73.65	69.57	<i>F</i> (1,64)=5.17, <i>p</i> =.03
		(6.92)	(7.34)	(6.42)	
Sex, (Male:Female)		33:33	11:9	22:24	X²=0.29, p=.59
Family member	ASD	11	7	4	ASD yes/no:
diagnosis	Psychiatric	8	2	6	<i>X</i> <sup>2</sup> =6.94,
	Neurological	4	0	4	<i>p</i> =.008
Marital Status	Single	16	8	8	X <sup>2</sup> =5.86,
	Married or	44	10	34	<i>p</i> =.12
	relationship				
	Divorced	6	2	4	
Highest Education	None	2	1	1	X <sup>2</sup> =8.37,
	School to 16	17	6	11	<i>p</i> =.14
	School to 18	6	1	5	
	Undergraduate	18	3	15	
	Postgraduate	21	9	12	
Personal Health					
	High Blood Pressure	25	11	14	<i>X</i> <sup>2</sup> =8.58,
	Diabetes	3	2	1	<i>p</i> =.07
	Other	6	1	5	
Other affectir	ng activity or cognition	2	1	1	

BAP - Broad Autism Phenotype; COA - Control Older Adults

**Table 2.** Mean (standard deviation) and range for the Broad Autism Phenotype

Questionnaire (BAPQ) subscales and total score.

BAPQ Subscale	BAP	COA
	(n=20)	(n=46)
Aloof	4.15 (0.80)	2.61 (0.54)
	2.58-5.33	1.42-3.83
Rigid	4.48 (0.59)	2.61 (0.57)
	3.17-5.08	1.33-4.08
Pragmatic Language	4.20 (0.92)	2.16 (0.39)
	2.50-5.50	1.25-3.17
Total	4.27 (0.69)	2.46 (0.37)
	3.17-5.19	1.67-3.11

BAPQ - Broad Autism Phenotype Questionnaire; BAP - Broad Autism Phenotype; COA

- Control Older Adults

Table 3.	Means and	standard	deviations	for self-	rated be	ehaviors l	by gr	oup	).

	BAP	COA	Group difference
BRIEF-A			
BRI raw scores	53.05 (6.82)	39.74 (6.51)	<i>F</i> =(1,64) 56.62, <i>p</i> <.001
MCI raw scores	69.55 (12.97)	56.96 (8.58)	<i>F</i> =(1,64) 21.73, <i>p</i> <.001
BRI T scores	65.70 (8.26)	48.93 (7.77)	<i>F</i> =(1,64) 62.45, <i>p</i> <.001
MCI T scores	64.35 (11.73)	52.59 (7.95)	<i>F</i> =(1,64) 22.61, <i>p</i> <.001
Mood scales <sup>1</sup>			
GDS	16.85 (6.90)	5.53 (4.45)	<i>F</i> =(1,63) 62.84, <i>p</i> <.001
BAI	18.05 (5.76)	4.95 (4.68)	<i>F</i> =(1,60) 91.33, <i>p</i> <.001
Social Support			
DSSI	20.55 (4.64)	27.87 (2.74)	<i>F</i> =(1,64) 64.06, <i>p</i> <.001

<sup>1</sup>GDS - COA n=45; BAI COA n=42

BAP - Broad Autism Phenotype; COA - Control Older Adults; BRIEF-A - Behavior Rating Inventory of Executive Function-Adult version; BRI - Behavior Regulation Index; MCI -Metacognition Index; GDS - Geriatric Depression Scale; BAI - Beck Anxiety Inventory; DSSI - Duke Social Support Index

	BAPQ Total	BRI	MCI	GDS	BAI	DSSI
Full Sample						
Age	<i>r</i> =.16, <i>p</i> =.21	<i>r</i> =.05, <i>p</i> =.72	<i>r</i> =.26 <b>,</b> <i>p</i> =.03*	<i>r</i> =.27, <i>p</i> =.03*	<i>r</i> =.20, <i>p</i> =.13	<i>r</i> =04, <i>p</i> =.78
BAPQ Total		<i>r</i> =.73, <i>p</i> <.001 <sup>±</sup>	<i>r</i> =.55, <i>p</i> <.001 <sup>±</sup>	<i>r</i> =.78, <i>p</i> <.001 <sup>±</sup>	<i>r</i> =.69, <i>p</i> <.001 <sup>±</sup>	<i>r</i> =80, <i>p</i> <.001 <sup>±</sup>
BAP						
Age	<i>r</i> =28, <i>p</i> =.23	r=-23, p=.33	<i>r</i> =.23, <i>p</i> =.34	<i>r</i> =.06, <i>p</i> =.81	<i>r</i> =.004, <i>p</i> =.99	<i>r</i> =.26, <i>p</i> =.28
BAPQ Total		<i>r</i> =.73, <i>p</i> <.001 <sup>±,a</sup>	<i>r</i> =.32, <i>p</i> =.17	<i>r</i> =.55, <i>p</i> =.01*	<i>r</i> =.04, <i>p</i> =.85	<i>r</i> =65, <i>p</i> =.002**
<u>AOT</u>						
Age	<i>r</i> =08, <i>p</i> =.61	<i>r</i> =19, <i>p</i> =.20	<i>r</i> =.10, <i>p</i> =.51	r=.12, p=.44	<i>r</i> =15, <i>p</i> =.92	<i>r</i> =.22, <i>p</i> =.14
BAPQ Total		$r=.12, p=.42^{a}$	r=.22, p=.15	r=.39, p=.008**	r=.07, p=.67	r=35, p=.02*

**Table 4.** Correlations (by group) between age or broad autism phenotype traits and self-rated executive function, depression and anxiety symptomatology, and levels of social support.

# \**p*<.05; \*\**p*<.01; <sup>±</sup>*p*<.001

Differences between correlations for BAP and COA groups were calculated using Fisher's r to z transformation; a significant group difference was noted (BAP>COA) for the correlations between <sup>a</sup>BAPQ Total and BRI scores: *z*=2.82, *p*=.005

BAPQ - Broad Autism Phenotype Questionnaire; BRI - Behavior Regulation Index; MCI - Metacognition Index; GDS - Geriatric Depression Scale; BAI - Beck Anxiety Inventory; DSSI - Duke Social Support Index; BAP - Broad Autism Phenotype; COA - Control Older Adults