COGNITIVE AGING IN AUTISM SPECTRUM DISORDERS

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ABSTRACT

Patrick Snow Powell: Cognitive Aging in Autism Spectrum Disorders
(Under the direction of Mark R. Klinger)

Little is known about the effects of age on cognitive functioning in adults with autism spectrum disorder (ASD). However, previous aging studies in individuals with Down syndrome, Fragile X, and William's syndrome suggest accelerated cognitive decline with age. The current study used a cross-sectional design to examine age-related cognitive changes in adults with ASD (ages 30 to 67) compared to adults with typical development (ages 30-65). To examine whether ASD is associated with atypical aging, performance assessed through measures of effortful cognitive processing (known to decline with age) and measures of automatic processing (thought to be relatively age-invariant) were examined. Results indicated that diagnosis was related to poorer cognitive performance. However, aging in ASD was associated with three different patterns of cognitive decline compared to adults with typical development.

Adults with ASD exhibited greater age-related decline across three measures designed to assess mild cognitive impairment (e.g., the MoCA), cognitive flexibility [e.g., Trail Making Test (TMT) number-letter switching], and associative learning (e.g., classical conditioning). There was also evidence of similar age-related decline, as compared to controls, on measures of explicit free recall (e.g., RAVLT), visual search (e.g., TMT visual scanning), and processing speed (e.g., TMT number/letter sequencing subtests). Finally, no age-related decline was observed on measures of recognition memory (e.g. RAVLT recognition test), explicit category learning

(Woodcock-Johnson Concept Formation), and implicit category learning (e.g., prototype formation).

Given different patterns of age-related change observed in adults with ASD, a final multivariate analysis examined overall cognitive performance, including measures of processing speed, cognitive flexibility, executive functioning, explicit category learning, and free recall. Results indicated that when the overall pattern of age-relate cognitive change was considered, age had a disproportionately negative impact on cognitive performance in adults with ASD compared to adults with typical development.

These findings suggest aging in ASD may be characterized by greater age-related declines in cognitive functioning, including a particular disruption of executive functions. Theoretical insights are provided by the Processing Resources and Processing Speed theories of cognitive aging, and clinical implications regarding a higher risk for mild cognitive impairment and disruption of pre-frontal cortex are discussed.

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INTRODUCTION: COGNITIVE AGING IN AUTISM SPECTRUM DISORDER

Autism spectrum disorder (ASD) is a developmental disorder with core symptoms consisting of impairments in social communication and the presence of restrictive interests and repetitive behaviors (American Psychiatric Association, 2013). Often considered a disorder of childhood, researchers are now developing theoretical frameworks to understand the developmental trajectory of individuals with ASD across the lifespan. Unfortunately, little is known about this developmental trajectory as individuals leave young adulthood. With recent prevalence rates of 1 in 68 (Christensen et al., 2016), there will be a significant increase in the population of older individuals with ASD in the near future. Thus, the study of how aging impacts individuals with ASD is in dire need of investigation.

Although it is well known that cognitive changes occur with normal aging, very few studies have investigated how aging influences individuals with ASD. Individuals with ASD demonstrate a variety of cognitive impairments in childhood and young adulthood ranging from communication and social impairments to more fundamental cognitive impairments in executive functioning, working memory, and implicit and explicit forms of memory and learning (Brunsdon et al., 2015; Brunsdon & Happé, 2014). Yet little is known about how these impairments change with age. To date, only two studies have examined the cognitive profile of older adults with ASD compared to adults with typical development (Geurts & Visser, 2012; Lever & Geurts, 2015). The first study by Geurts and Visser (2012) found overall poorer performance in older adults with ASD in sustained attention, working memory, and verbal

fluency, while showing equivalent cognitive ability in processing speed, cognitive flexibility, and visual and verbal memory compared to older adults with typical development. Unfortunately, this study did not include samples of younger adults with ASD and typical development making it impossible to tell whether these differences in cognitive performance reflect global diagnostic differences or the interaction of aging and diagnosis (e.g., whether aging differentially impacts adults with ASD). Therefore, a subsequent study by Lever and Geurts (2015) examined performance on various cognitive tasks in a larger cross-sectional sample of younger and older adults with ASD (n=118) and typical development (n=118; ages 20 – 79). This study found no evidence of greater or accelerated cognitive decline in older adults with ASD. Instead, age-related changes in ASD were either reduced or similar to those observed for individuals with typical development. Overall, Geurts' research suggests that individuals with ASD may age similarly to those with typical development or may be partially protected against age-related declines in cognitive functioning. Although these findings are certainly strengthened by the large sample of adults with ASD included in this study, additional research is needed to confirm these results and to examine the impact of other moderating variables on these findings (e.g., diagnostic severity, intelligence). In Lever & Geurts (2015) study, the majority of participants in this study were diagnosed in adulthood and represented relatively mild cases of ASD (e.g., Asperger's syndrome or Pervasive Developmental Disorder – Not Otherwise Specified, PDD-NOS), which Lever and Geurts have suggested may be more common in adults with average to above average intelligence whose cognitive abilities can be used to compensate for ASD-related difficulties. Indeed, studies of normal aging have also indicated that age-related effects may be masked in individuals with higher verbal intelligence or high educational levels (Bolla, Lindgren, Bonaccorsy, & Bleecker, 1990; Tombaugh et al., 1999). Hence, it is possible that Lever and

Geurts' results may have been impacted by intellectual functioning of the participants which may have limited the ability to detect diagnostic and age-related changes in adults with ASD. In light of this possibility, the purpose of the present study was to explore age-related changes in cognitive ability across younger, middle-aged, and older adults with ASD compared to younger, middle-aged, and older adults with typical development while controlling for differences in intellectual functioning.

To begin this investigation of age-related cognitive changes in ASD, I will review what is known about changes in cognition in normal aging and discuss what is known about cognitive changes in older adults with other developmental disorders (DDs). This review should provide a theoretical framework to understand age-related cognitive changes in ASD and to develop predictions about these changes.

Normal Aging

A central focus of cognitive aging research is to examine both the trajectory of age-related changes in cognition as well as the specific cognitive functions negatively impacted by aging. Numerous cross-sectional and longitudinal studies of normal aging have indicated that age is associated with a pattern of linear decline in cognitive functioning such that by the time adults reach their eighties, the average level of cognitive performance is a full standard deviation below that of young adults (Salthouse, 2009; Schroeder & Salthouse, 2004). Salthouse and colleagues have argued that this difference between younger and older adults is due to declines in cognitive function that begin in mid-adulthood and continue into older adulthood. Due to this linear pattern of cognitive decline, researchers have investigated whether age has a global or more specific impact on cognitive processes. To investigate specific patterns of cognitive decline, studies of normal aging often differentiate between measures of effortful processes (i.e., processes that

require substantial awareness and cognitive effort) and measures of automatic processes (i.e., processes that are relatively unintentional, involuntary, effortless, or occur outside awareness; Reber, 1989). Therefore, the following sections briefly review studies of effortful and automatic processing in normal aging.

Effortful processing in normal aging. Measures of effortful processing typically include measures of working memory, executive functioning, explicit memory, processing speed, concept formation, and controlled aspects of attention. These measures consistently show poorer performance in older adults than younger adults (Buckner, 2004; Craik & Byrd, 1982; Craik, 1986; Craik & McDowd, 1987; Hess & Blanchard-Fields, 1996; Kramer & Madden, 2008; Salthouse, 1991, 1996; Salthouse, Atkinson, & Berish, 2003; Stoltzfus, Hasher, & Zacks, 1996). By far the most thoroughly researched area in cognitive aging is explicit memory, specifically episodic memory (Craik, 2002; Craik & Byrd, 1982; Craik & McDowd, 1987; Light, Prull, La Voie, & Healy 2000; Salthouse, 2004; 2009). Several meta-analyses have assimilated these findings to show that age-related differences between young adults and older adults are mediated by the way that episodic memory is assessed (Spencer & Raz, 1995; Verhaeghen & Salthouse, 1997). That is, compared to younger adults, older adults typically show more impairment on tests of free recall and cued recall, and less impairment on tests of recognition. For instance, Spencer and Raz (1995) found that the average effect size associated with age was greater when memory was tested by free or cued recall (d = 1.01) compared to recognition (d = .57). One explanation of these findings is that, tests of free recall provide participants with little information that could aid in retrieval (i.e., little environmental support) aside from the participant's own memory of the studied material. Thus, greater cognitive effort and strategic control is required during retrieval. In contrast, tests of recognition present the previously studied material to the participant which in

turn facilitates retrieval. Other studies have suggested that impaired free recall may be due to both a difficulty in retrieval as well as use of less effective encoding strategies. For instance, when free recall involves retrieval of semantically-related items, young adults often recall words from the same category, indicating that they used the categories to organize the information. However, older adults are less likely to spontaneously engage in this type of organizational strategy and instead tend to engage in more item-specific processing (i.e., remembering individual words) unless provided with an explicit strategy designed to highlight the semantic relationship between items. For instance, Woo and Schmitter-Edgecombe (2009) provided older adults (age range: 60-88 years) with semantic cues or no semantic cue (i.e., control group) followed by a free recall test. Results indicated that older adults provided with semantic cues at encoding demonstrated greater semantic clustering compared to participants who did not receive the semantic encoding cue. Therefore, when provided with an organizational strategy, older adults are more likely to engage greater relational processing (i.e., generating similarities among items). These findings, coupled with a general reduction in effortful processing resources, suggest that tasks requiring substantial cognitive effort (e.g., free recall) are often impaired in older adults

Automatic Processing in Normal Aging. In contrast, studies examining more automatic processes in normal aging such as implicit memory (Gopie, Craik, & Hasher, 2011; Light & Singh, 1987; Light, Singh, & Capps, 1986), implicit learning (Fera et al., 2005; Glass et al., 2012; Hess, 1986; Labar, Cook, Torpey, & Welsh-Bohmer, 2004), and automatic aspects of attention shifting (Colcombe et al., 2003; Kramer, Hahn, Irwin, & Theeuwes, 1999; Zanto & Gazzaley, 2014) typically show relatively preserved processing in older adults. Previous studies have demonstrated that these processes are largely independent of age (Huang-Pollock, Maddox,

& Karaluns, 2011; Weinert, 2009) and intelligence (Atwell, Conners, & Merrill, 2003). Presumably the relative preservation of automatic processing in normal aging occurs because these processes require less cognitive effort, and, therefore, rely less on older adults' limited, declining pool of effortful cognitive resources. Experience is another way in which automatic processes may be maintained in older adults. For example, Salthouse (1984) compared typing speed between younger adults and older adults. Older adults with more years of experience demonstrated faster typing speed compared to less experienced older adults and comparable speed to younger adults. Similarly, Parbery-Clark, Strait, Anderson, Hittner, and Kraus (2011) found that older adults with extensive musical training maintained similar auditory perception as compared to younger adults. Interestingly, years of musical training also helped maintain effortful processes such as auditory working memory, suggesting that experience not only maintains automatic processes, but may also mitigate declines in some effortful processes.

However, not all automatic processes are preserved in normal aging. For example, in some studies classical conditioning is seen to decline with age, such that older adults show weaker conditioned responses compared to younger adults (Bellebaum & Daum, 2004; Cheng, Faulkner, Disterhoft, & Desmond, 2010; Woodruff-Pak & Jaeger, 1998). This pattern of impairment has been attributed to the significant age-related functional and structural changes in the hippocampus and cerebellum which play roles in classical conditioning. Expectedly, when these structures are further compromised by neuro-degenerative disease, such as in patients with Alzheimer's disease, even poorer conditioning is found relative to healthy older adults. Given the role of the hippocampus and evidence of poorer conditioning in Alzheimer's disease, these results suggest that classical conditioning paradigms may involve both automatic and effortful processing. Further support for this notion comes from a study by Labar, Cook, Torpey, and

Welsh-Bohmer (2004) which found that increased age was associated with poorer conditioning and poorer explicit awareness of the learning contingencies (i.e., the CS-UCS relationship). However, when awareness was taken into consideration, there were no effects of age on conditioning. That is, older adults demonstrated comparable conditioning to younger adults when aware of the relationship between the CS and the UCR, but when older adults were unaware of this relationship, conditioning was impaired. Though classical conditioning is traditionally considered a non-declarative or automatic form of learning, these findings suggest that older adults may be capable of compensating for impairments in the automatic aspects of conditioned learning by using more effortful or explicit processes.

Theoretical Models of Cognitive Aging

Although several theoretical models have been proposed to account for patterns of age-related changes in cognition, the primary focus of the current study is to examine patterns of performance on effortful and automatic processing tasks in older adults with ASD. Therefore, the following section will discuss theories of effortful and automatic processing in older adults including Craik and colleagues' extension of the Dual-process theory (Craik & Byrd, 1982; Craik, 1983; 1986; Craik & Rose, 2012; Light et al., 2000), and Salthouse and colleagues' Processing Speed theory (Salthouse, 1996).

Processing resources. The Dual-process theory is a well-known theory in cognitive psychology that suggests cognitive processes largely fall into two independent, distinct systems. One system consists of a limited pool of effortful or explicit processes that operate under conscious control, whereas the other system involves automatic or implicit processes that operate relatively outside of conscious awareness. As previously mentioned, studies of normal aging have shown that automatic and effortful processes are differentially affected by age (Light et al.,

2000). That is, studies of automatic processing, such as implicit memory and learning, reveal minimal age effects, whereas studies of effortful processing, such as working memory or episodic memory, show substantial declines in older adults (Craik & Rose, 2012; Light et al., 2000). However, tests designed to assess effortful processes such as explicit memory are not "process pure" and, in fact, require varying amounts of effortful and automatic processes. For example, tests of free recall assess a participant's ability to explicitly retrieve previously studied information. As a consequence, free recall heavily depends on self-initiated, controlled processes. In contrast, recognition memory involves retrieval of specific details associated with previously studied information (i.e., recollection), as well as a subjective experience of having previously studied the information, but may often have an inability to recall specific details (i.e., familiarity). Thus, recognition can involve both effortful recollective processes, as well as the automatic processes associated with familiarity (Yonelinas, 2002). This idea led Craik and colleagues to propose the Processing Resources account of cognitive aging.

The Processing Resource account suggests that cognitive functioning in older adults is characterized by declines in effortful processing resources. Tasks with a greater degree of effortful processing requirements are hypothesized to show greater age-related declines. As previously mentioned, studies of explicit memory in normal aging indicate a disproportionately large age effect on free recall compared to recognition. According to the Processing Resource account, free recall is more dependent on effortful processes, therefore age-related declines in effortful processing resources ought to more adversely affect this type of test. In contrast, performance on tests of recognition is less impaired because automatic processes are used to compensate for declining effortful processing resources. The Processing Resource account has also tested the prediction that if aging is associated with a reduction in effortful processing

resources, then the pattern of memory performance in older adults may be mimicked in young adults whose available resources have been reduced by providing a secondary task to perform simultaneously with the primary memory task (Anderson, Craik, & Naveh-Benjamin, 1998; Craik & Rose, 2012). Anderson et al., (1998) provided support for this notion, showing that young adults whose attention was divided demonstrated behavioral results that replicated the pattern seen in older adults when performing a memory task with full attention. Finally, this account can explain the pattern of age-related changes in studies of classical conditioning. As previously mentioned, Labar et al. (2004) demonstrated a relationship between explicit awareness and classical conditioning in older adults. This result is consistent with the claim that classical conditioning is not a pure measure of automatic processing, but, in fact, draws upon both automatic and effortful processes. If classical conditioning is only associated with automatic processes, then it should be relatively unaffected by neuro-degenerative diseases (e.g., Alzheimer's) or medial temporal amnesia that specifically disrupt effortful processes such as explicit learning. However, studies of classical conditioning have indicated greater explicit learning is sometimes associated with stronger classical conditioning. Additionally, poorer performance in patients with Alzheimer's Disease (Hoefer et al., 2008; Woodruff-Pak, Finkbiner, & Sasse, 1990) and amnesia (Fortier et al., 2003; Meyers et al., 2001) compared to healthy older adults suggests that greater impairment in effortful processing is related to impairment in conditioning.

Processing speed. Another theory explaining age-related cognitive declines stems from the work of Salthouse and colleagues. These authors have proposed the Processing Speed theory of aging that argues that cognitive declines in older adults is specifically related to age-related declines in the speed of information processing (Salthouse, 1991; 1996; 2004; 2009; Verhaeghen

& Salthouse, 1997). This theory has received support from several meta-analyses indicating a substantial portion of age-related cognitive decline is accounted for by processing speed (Salthouse, 1996; Verhaeghen & Salthouse, 1997). For instance, through a meta-analysis of cross-sectional studies, Verhaeghen and Salthouse (1997) found that processing speed accounts for more than 70% of the age-related decline on tests of explicit memory. In contrast to the Processing Resource account, the Processing Speed theory suggests that declines in processing speed may represent a more fundamental cognitive change, and the impairment of effortful processes is simply a consequence of this change. To illustrate, processing speed corresponds to the speed of initial information processing such as early visual or auditory perception. Thus, declines in processing speed reflect slower initial processing. Information from these initial processes is simultaneously coordinated by more complex and effortful secondary processes such as working memory. Therefore, significant disruption occurs when secondary processes begin before slower initial processes are completed. In contrast, automatic tasks may require less information, and, therefore, less coordination among processes. This may explain why automatic processes are less disrupted by slower processing speed.

Age-related Changes in Cognition in Developmental Disabilities

Although these theoretical frameworks serve as guides for predictions of age-related cognitive changes in the current study, it is important to further support these predictions by incorporating what is known about age-related changes in cognitive functioning in individuals with other DDs. The following sections will discuss studies of effortful and automatic processing in older adults with several other DDs, specifically Down syndrome, William's syndrome, and Fragile X, and will end with a discussion of the current findings from studies of young adults with ASD.

Several studies of aging in developmental disorders such as Down syndrome (DS), Williams syndrome, and Fragile X have indicated that cognitive declines emerge much earlier in these DDs than in the general population (Devenny, Krinsky-McHale, Sersen, & Silverman, 2000; Krinsky-McHale, Kittler, Brown, Jenkins, & Devenny, 2005; Oliver, Crayton, Holland, Hall, & Bradbury, 1998; Woodruff-Pak, Papka, & Simon, 1994). For example, Oliver et al. (1998) conducted a prospective longitudinal study of adults with DS across four years. Their findings indicated that a pattern of decline in explicit memory and learning in young adulthood (20 to 39 year olds) preceded dementia-related symptoms of aphasia, agnosia, and apraxia. These authors argued that early declines in memory and learning may be related to the higher prevalence of neuro-degenerative disease (e.g., Alzheimer's disease) in adults with DS compared to the general population (Holland, Hon, Huppert, & Stevens, 2000; Lai & Williams, 1989; Wisniewski, Wisniewski, & Wen, 1985).

However, there are two important considerations that should be stated before interpreting this pattern of decline in DS. The first consideration is how the conventional concept of general intelligence can be broken down into two components referred to as crystalized (g*C*) and fluid intelligence (g*F*). Crystalized intelligence refers to knowledge acquired through education and experience and is relatively stable across age, with the exception of decline due to neuro-degenerative disease. Fluid intelligence refers to more abstract reasoning and problem-solving abilities allowing individuals to adapt to a changing and complex environment (Cattell, 1963). These skills typically begin to decline in the early 20's (Salthouse, 2009). Due to this pattern of decline, most of the age-related variance in cognitive decline in normal aging is accounted for by changes in g*F*, which is not surprising given that g*F* relates to more effortful processes, whereas g*C* may be related more to automatic processes. In light of this consideration it should be noted

that the pattern of age-related cognitive decline in adults with DS found by Oliver et al. (1998) was largely attributed to declines in g*F*, whereas declines in g*C* were present in only the most cognitively impaired individuals who were likely exhibiting advanced signs of dementia.

The second important consideration is that not all individuals with DS develop neuro-degenerative disease (Devenny et al., 2000). Thus, it is unclear whether early cognitive decline is only demonstrated by those individuals who go on to develop a neuro-degenerative disease or whether it is a specific characteristic of the aging process in DS. To better understand which factors contribute to this pattern of decline, Crayton et al. (1998) examined data from Oliver et al. (1998) but excluded individuals with advanced dementia. Using these exclusion criteria, Crayton and colleagues examined performance on measures of g*F* including pattern recognition, spatial recognition, and matching-to-sample in older adults with DS (50 to 58 years old), middle-aged adults with DS (40 to 49 years old), and younger adults with DS (20 to 40 years old). Findings showed significantly worse performance in older adults with DS compared to younger adults with DS. In contrast, performance on measures of g*C* (e.g., picture vocabulary tests) did not differ between age groups, suggesting that early cognitive decline in DS was specific to declines in g*F*. Given the exclusion criteria, it is unlikely that performance in older adults with DS was due to the presence of dementia. Nevertheless, greater declines were seen in non-demented adults with DS in their 50's suggesting that early cognitive decline may be an important characteristic of the aging process in DS.

Other studies have indicated a similar pattern of decline in William's syndrome. For instance, Krinsky-McHale and colleagues (Krinsky-McHale et al., 2005) examined performance on tests of explicit (free recall) and implicit memory (repetition priming) for adults with WS (age range: 32 to 77 years old) compared to age and IQ-matched adults with DS and unspecified

intellectual disability (ID). Their findings showed that both adults with WS and adults with DS demonstrated a similar rate of age-related decline in free recall (e.g., a measure of explicit memory). However, this pattern was not observed in the sample of individuals with ID. The pattern of performance on repetition priming (e.g., a measure of implicit memory) revealed no diagnostic differences and no significant interaction between age and diagnostic group, suggesting that this form of implicit memory may be unaffected by age in DS, WS, and ID.

In contrast to these findings, studies investigating other forms of implicit or automatic learning, such as classical conditioning, have demonstrated poorer performance in older adults with DS compared to age and IQ-matched individuals with non-specific intellectual disability (ID). Classical conditioning paradigms are well suited for populations that have co-morbid intellectual disability because a little comprehension is required to perform the task. Woodruff-Pak, Papka, and Simon (1994) examined classical conditioning in samples of younger (less than 35 years old) and older adults (greater than 35 years old) with DS, Fragile X (FX), and age-matched controls with typical development. Participants were presented with a simple eye-blink classical conditioning paradigm (EBCC) wherein a tone (CS) was followed by an air puff (UCS). Results from this study revealed that regardless of age, individuals with DS and individuals with Fragile X showed significantly worse EBCC compared to individuals with typical development. Additionally, this study found a significant effect of age, such that older adults with DS and typical development performed significantly worse than younger adults. A subsequent analysis compared conditioning across younger and older adults with DS to both age-matched adults with typical development and older adults with probable Alzheimer's Disease. Young adults with DS demonstrated significantly worse conditioning compared to young adults with typical development but did not significantly differ from older adults with typical development.

However, the worst conditioning was found in older adults with DS whose performance was not only significantly impaired, but was not significantly different from the comparison sample of 20 patients with probable Alzheimer's Disease. The authors of this study interpreted these findings as a pattern of accelerated decline in individuals with DS. However, an alternative interpretation may be that individuals with DS may show an overall decrement in conditioning, but decline at a similar rate as typical adults. If there is poorer conditioning in 20 year-olds with DS, then typical age-related declines will likely result in significantly impaired conditioning by the time they reach mid-adulthood. This could also explain why the significant impairment in conditioning in 40 year-old adults with DS (mean age = 48 years) resembled that of patients with Alzheimer's Disease.

Another study of EBCC in individuals with Fragile X (ages 17 to 77) examined the retention and reacquisition of a conditioned response across two 12-month follow-up sessions (Tobia & Woodruff-Pak, 2009). Similar to Woodruff-Pak et al. (1994), these studies found significantly worse conditioning in the Fragile X group compared to age-matched individuals with typical development, and no effect of age. However, when participants were brought back for a 12-month follow-up session to re-assess EBCC, significant impairments were seen in older adults with FX (> 45 years old) compared to the younger adults with Fragile X (< 45 years old). Unfortunately, this study did not collect follow-up conditioning data for younger and older adults with typical development; therefore, it is unclear whether the effect of age on EBCC was specific to individuals with FX or was a function of normal aging. However, there is some previous evidence that supports that this age-related effect may be specific to older adults with FX. Numerous studies have identified the critical role of the cerebellum in EBCC (for review see Timmann et al., 2010). Post-mortem studies of individuals with FX have revealed significantly

greater volumetric reductions in the cerebellum with age in adults with FX compared to adults with typical development (Greco et al., 2011; Sabaratnam, 2000). Additionally, studies of individuals that carry the pre-mutation of FX, but are unaffected by FX, have identified a subgroup of older adults in this population that develop a neurological syndrome (onset between 50 and 70 years) known as fragile X – associated tremor/ataxia syndrome. Fragile X – associated tremor/ataxia is associated with progressive increases in tremor and ataxia, including greater disturbances in postural instability (i.e., balance) and gait control (e.g., tandem walking). These disturbances have been linked to greater cell loss in the cerebellum (Jacquemont et al., 2004). Therefore, the age-related changes in EBCC in FX may be associated with the cerebellar impairments (e.g., volumetric reductions) that are associated with this disorder.

These results suggest that there may be a pattern of accelerated decline in these DDs, though more research is needed to confirm these findings. The presence of cognitive impairment in young adults with DS, WS, and FX suggests that the level of impairment in effortful and some automatic processes (e.g., classical conditioning) associated with normal aging may appear 20 to 30 years before individuals with typical development reach this level of impairment. Additionally, it has been suggested that this pattern of early cognitive decline may signal an early behavioral marker for subsequent neuro-degenerative disease. These findings do not appear to be indicative of all types of DDs; for example, those with DS appear to be most negatively affected by early cognitive declines associated with increased risk for neuro-degenerative disease. Little research has examined whether a similar pattern is true for adults with ASD; thus it is important investigate the pattern of age-related cognitive change in ASD in order to understand whether this population may be at similar risk.

Cognitive Functioning in ASD

Cognitive performance on tests of effortful processing in ASD. There are few studies that have examined effortful processing in older adults with ASD. However, studies of effortful processes in younger adults with ASD, such as working memory, executive function, and explicit memory, reveal an inconsistent pattern of both impaired and intact performance. One of the more consistent findings in young adults with ASD is a pattern of impairment in free recall when lists of semantically-related items are used. Bowler and colleagues have argued this impairment is present because young adults with ASD are more likely to engage in item-specific processing, rather than relational processing or semantic organizational strategies (i.e., grouping words together into similar semantic categories; Gaigg, Gardiner, & Bowler, 2008; Minshew & Goldstein, 2001). It is interesting how similar this pattern of poorer relational processing in young adults with ASD is to the pattern found in older adults with typical development (Craik & Rose, 2012; Hogan, Kelly, & Craik, 2006). For instance, both young adults with ASD and older adults with typical development tend to show poorer relational processing when tested by free recall but better performance when assessed by recognition. The similar pattern of performance between young adults with ASD and older adults with typical development highlights the importance of investigating how explicit memory performance changes as individuals with ASD age. For instance, if declines in explicit memory processes in ASD follow a similar trajectory seen in normal aging, explicit memory performance in adults with ASD in middle-adulthood may mimic the performance of older adults with typical aging processes.

Cognitive performance on tests of automatic processing in ASD. In addition to investigating differences in effortful processes, it is equally important to examine differences in automatic processing in ASD. Studies of automatic or implicit processing in children,

adolescents, and young adults with ASD have shown inconsistent patterns of both impaired and intact performance (for review see Eigsti & Mayo, 2011).

*Studies of classical conditioning in ASD***.** Studies of classical fear conditioning in ASD have indicated patterns of both impaired learning (Gaigg & Bowler, 2007; South, Newton, & Chamberlain, 2012), intact learning (Bernier, Dawson, Panagiotides, & Webb, 2005; South, Larson, White, Dana, & Crowley, 2011), and, in one instance, more rapid learning compared to age and IQ-matched individuals with typical development (see Sears, Finn, & Steinmetz, 1994). However, several methodological differences seem to underlie the conflicting patterns of results. Studies finding intact or more rapid conditioning in ASD have measured eye-blink response to a single CS (Bernier et al., 2005; Sears et al., 1994), and is known to rely upon cerebellar and limbic system pathways (Steinmetz, Tracy, & Green, 2001); suggesting these pathways may be intact in ASD. Whereas studies of differential classical fear conditioning have measured associative learning by comparing changes in the skin conductance response (SCR) to a CS relative to one or more neutral stimuli. This type of conditioning has been linked to amygdala, hippocampal, and prefrontal brain regions (Jarrell et al., 1987; LaBar, LeDoux, Spencer, & Phelps, 1995; Morris, Friston, & Dolan, 1997).

Given that differential fear conditioning paradigms require greater communication between cortical and subcortical brain regions (Jarrell et al., 1987; Morris, Friston, & Dolan, 1997), prior evidence of poor connectivity between these brain regions in individuals with ASD (Belmonte et al. 2004; Just et al., 2004, Kana et al., 2006; Minshew & Williams, 2007) may be one explanation why differential conditioning paradigms have resulted in poorer learning in ASD. Furthermore, the medial temporal lobes (MTL), commonly associated with more explicit learning and memory processes, may also account for previously observed relationship between

explicit awareness and conditioning in samples of younger adults with ASD (Powell et al., 2016), as well as older adults (Labar et al., 2004). In light of these findings, differential fear condition paradigms provide an opportunity to explore the relationship between age, explicit awareness, and conditioning in adults with ASD.

*Studies of implicit category learning in ASD***.** Similar to studies of classical conditioning, studies of implicit category learning in ASD have indicated both impaired (Church et al., 2010; Gastgeb et al., 2009; 2011; 2012; Klinger and Dawson, 2001; Klinger, Klinger, & Pohlig, 2007; Schipul & Just, 2016; Valdusich, Olu-Lafe, Kim, Tager-Flusberg, & Grossberg, 2010) and intact (Molesworth, Bowler, & Hampton, 2005; 2008) learning in ASD. In contrast, studies of implicit category learning in older adults with typical development have largely indicated comparable performance between younger and older adults (Glass, Choibut, Pacheco, Schnyer, & Maddox, 2012; Hess & Slaughter, 1986a; 1986b). The prototype tasks used in these studies require participants to learn a category that has no explicit rules of membership. In this task, a typical example or prototype is thought to be created by averaging previously seen exemplars to form a "best" representation. Participants categorize new examples as a member of the category based on how closely the example matches the prototype. Studies of individuals with typical development suggest that formation of the prototype is considered a relatively automatic process; wherein a person is able to form a prototype with minimal explicit instruction, awareness, or effortful processing. In contrast, several studies of prototype learning in individuals with ASD have demonstrated a relationship between implicit category learning and effortful processing. That is, greater implicit category learning was associated with greater effortful processing (Gastgeb et al., 2012; Klinger et al., 2007; Vladusich et al., 2010). These findings indicate that, unlike individuals with typical development who demonstrate explicit and implicit learning

independent of one another, individuals with ASD who engage in more explicit processing demonstrate better implicit task performance.

Theoretical Frameworks for Cognitive Aging in ASD

The Processing Resource theory is one model that accounts for the differential pattern of age-related declines in effortful processing, yet relatively preserved automatic processing in normal aging. In contrast, studies of older adults with developmental disabilities (e.g., DS, FX) show age-related declines in both effortful and automatic processing which appear to emerge in mid-adulthood (i.e., 30-40) and continue to worsen with advancing age (Krinskey-McHale et al., 2005; Tobias & Woodruff-Pak, 2009; Woodruff et al., 1994). Similarly, studies of young adults with ASD have shown when automatic processing is impaired, individuals with ASD may compensate by using more effortful processing (Klinger et al., 2007; Gastgeb et al., 2012). Collectively, these findings can provide a framework for making predictions about patterns of age-related decline in ASD. For instance, if automatic processes are more dependent on effortful processing in ASD than in typical development, then as effortful processes declines with age, automatic processes might also decline. As a result, cognitive functioning may be more adversely affected as individuals with ASD enter late-adulthood because both controlled and automatic processes will be impacted by aging.

The processing speed account provides slightly different theoretical predictions for age-related declines in older adults with ASD. To date, only a few studies have examined processing speed in individuals with high-functioning ASD. These studies generally find slower processing speed compared to age and IQ matched individuals with typical development (Calhoun & Mayes, 2005; Mayes & Calhoun, 2003; 2007; Oliveras-Renta, Kenworthy, Roberson, Martin, & Wallace, 2012). More recently, Travers et al. (2013) conducted a longitudinal study (age range 6

to 42 years) finding pronounced processing speed impairments in adults with ASD, although the rate of age-related decline in processing speed was similar across groups (i.e., ASD vs. typical development). Given that slower processing speed is present in middle-aged adults with ASD, it is possible that any task dependent upon efficient information processing will likely be significantly disrupted. For example, classical condition paradigms designed to assess automatic aspects of learning involve forming an association between two pieces of sensory information. However, because declines in processing cause initial sensory processes to operate more slowly, the ability to quickly associate these two pieces of sensory information may be significantly impaired (e.g., learning the association between a visual CS with an auditory UCS). Likewise, effortful processes such as explicit memory, which requires efficient encoding and retrieval of information, may be disrupted because slower processing speed also slows initial encoding. Thus, declines in processing speed may mediate performance on both effortful and automatic tasks. Finally, it is important to consider whether the presence of slower processing speed in mid-adulthood signals an earlier onset of cognitive decline in ASD. With respect to this finding and previous evidence indicating that accelerated cognitive decline is associated with severe cognitive impairments in older adults with DD, it is possible that significant cognitive impairment is present by the time individuals with ASD reach late-adulthood.

Current Study Predictions

Based upon the empirical literature from studies of cognitive processing in ASD, normal aging, and older adults with DS, FX and WS, the current study has two specific aims. The first aim is to examine task performance on measures of effortful processing across age in a sample of adults with ASD and compare this performance to age and IQ-matched individuals with typical development. The second aim is to examine task performance on measures of automatic

processing across age in a sample of adults with ASD compared to age and IQ-matched adults with typical development.

Aim 1 – Effortful Processing in Adults with ASD

1. Previous evidence shows that by mid-adulthood individuals with DS and WS already demonstrate a pattern of decline in effortful processing tasks including tests of processing speed, block design, matrix reasoning, and concept formation. Research in younger adults with ASD suggests overall impairments in effortful processing tasks, particularly those that require coordination between several different cognitive processes. Thus, in the present study, is predicted that adults with ASD (ages 30 to 65) will demonstrate worse performance on measures of effortful processing compared to age and IQ-matched adults with typical development.
2. Based upon evidence of earlier decline in effortful processing in older adults with other DDs, it is predicted that adults with ASD will demonstrate a steeper age-related decline in effortful processing compared to age and IQ-matched adults with typical development (i.e., an age by diagnosis interaction is predicted), particularly when accounting for mediating variables such as IQ.

Aim 2 – Automatic Processing in Adults with ASD

1. Based upon previous evidence of impaired automatic processing in younger adults with ASD, it is predicted that adults with ASD will demonstrate poorer automatic processing compared to age and IQ-matched individuals with typical development.
2. Based upon the presence of decline in automatic processing by mid-adulthood in other DDs, it is predicted that adults with ASD will demonstrate a steeper age-related decline

in automatic processing compared to age and IQ-matched adults with typical development (i.e., an age by diagnosis interaction is predicted).

Method

Participants

Twenty-nine adults with ASD between 30 and 67 years old were recruited through the Autism & Neurodevelopmental Disorders Registry which is a recruitment tool available to UNC researchers through the NICHD-funded Intellectual and Developmental Disabilities Research Center at the Carolina Institute for Developmental Disabilities (CIDD) and through University of North Carolina TEACCH Autism Program. Individuals seen in UNC TEACCH clinics across the state are asked if they would be interested in participating in research on ASD. A diagnosis of ASD was confirmed by clinical interview using the Autism Diagnostic Observation Schedule– 2 (ADOS-2). The following exclusion criteria were applied: (a) evidence of pre-existing developmental disability etiologically-similar to autism (e.g., Fragile X, Down syndrome); (b) history of traumatic brain injury (TBI); (c) did not meet the ADOS-2 score below diagnostic cut-off (i.e., 6)[1] and Social Responsiveness Scale – 2 (SRS-2) diagnostic cut-off (i.e., 60)[2]; (d) intellectual functioning less than 85 (assessed by the Wechsler Abbreviated Scale of Intelligence).

Thirty adults with typical development were recruited through three sources: (1) UNC faculty and staff listserv which contains contact information of all UNC faculty and staff interested in research (4,000 plus contacts), (2) community fliers and newsletters, and (3)

[1]One participant with ASD did not meet ADOS-2 cut-off criteria (Total Score = 5), but did meet SRS-2 cut-off (Total Score = 68). Eliminating this participant from the analyses did not alter the pattern of findings, therefore, this participant was included in the final sample.

[2]Five adults with ASD did not meet SRS-2 cut-off criteria, however they did meet ADOS-2 cut-off criteria and were included in the final sample.

through the Cognitive Neuroscience of Memory Laboratory at the University of North Carolina at Chapel Hill database. This database included 212 healthy older adults (ages 60-80) with no history of neuro-degenerative disease and indicated interest in participating in cognitive psychology and neuroimaging research. The following exclusion criteria were applied: (a) a prior clinical diagnosis of ASD or Attention Deficit Hyperactivity disorder (ADHD); (b) prior clinical diagnosis of ASD in a close family member (e.g., parents, children, siblings) (c) history of neurological disorders, traumatic brain injury (TBI), or schizophrenia; (d) a Social Responsiveness Scale – 2 (SRS-2) total score of greater than 65[3]; (e) intellectual functioning less than 85.

Finally, both samples of adults with ASD and adults with typical development were stratified across age groups (i.e., ages 30-39, 40-49, 50-65), with an approximately even distribution of participants across age groups. Participants were group matched on age, gender, and IQ (Table 1). All participants, reported normal or corrected to normal hearing and vision (e.g., contact lenses or eye glasses). Fifteen adults reported antidepressant/anti-anxiety medication use (1 typical; 14 ASD)[4]. Although there were some differences in educational level, including 3 adults with ASD who only received a high school diploma, the large majority of participants received some type of post-secondary degree (e.g., associate's degree, bachelors, masters, or Ph.D.).

[3]The cut-off criterion was set at 65 for adults with typical development because this would have indicated moderate and clinically significant ASD symptomatology. All adults with typical development scored below 65.

[4]Preliminary analyses of cognitive performance on those with and without antidepressant/anti-anxiety medication revealed no significant differences (all p's > .22). Therefore, findings regarding the effects of medication use were not reported in the following Results section.

Measures

Demographic form. A demographic form gathered general information about participants. This form included information about the participant's age, gender, race, medication history, education level, employment history, and any current diagnoses (i.e., ASD, ADHD, learning disability, etc.).

Autism Diagnostic Observation Schedule – 2 (ADOS-2, Module 4; Lord et al., 2012). The ADOS-2, Module 4, (35-40 minutes) is a semi-structured assessment of social interaction, communication administered to adolescents and adults. The ADOS-2 was administered to individuals with ASD to both confirm a previous diagnosis of ASD and measure current symptomatology (i.e., social skills, and communication). The ADOS-2, Module 4 has good reliability ratings with mean weighted kappa (Mk_w = .66; see Lord et al., 2011). The ADOS has good sensitivity (.71) and specificity (.82) in adults with ASD (ages: 18 – 66), providing support for its use in adults with ASD.

The Social Responsiveness Scale- 2 (SRS-2; Constantino, 2012). The adult form of SRS-2 is a 65-item self-report measure of ASD symptoms. This test has been validated for clinical populations aged 2 to 89 years. The present study used the adult form self-report version (normed for ages 18 -89). The adult form SRS-2 has very good test-retest reliability after four months (r = .88). The SRS-2 was administered to both individuals with ASD and individuals with typical development. This measure served as an additional measure of ASD symptom severity in individuals with ASD and as a screening tool for autism characteristics in individuals with typical development. No individuals with typical development scored higher than 65; a score that would have indicated the presence of moderately severe ASD symptomatology.

Montreal Cognitive Assessment (MoCA; Nasreddine, Phillips, Bédirian, 2005). The MoCA is a 10-minute, 30-point cognitive screening test designed to assess and screen for mild cognitive impairment (MCI). The MoCA assesses short-term recall, delayed recall, visuospatial abilities, working memory, and language, but has more emphasis on tasks of frontal functioning and attention than other brief screeners for MCI or dementia such as the Mini Mental State Examination (Folstein, Folestein, & McHugh, 1975). The MoCA has good specificity (90%) and sensitivity (85%). The suggested cut-off point for MCI on the MoCA is 26, with scores below indicating the presences of MCI. However, it is possible that some individuals with ASD may demonstrate a level of impairment that is similar to MCI as a function of ASD rather than age, thus a more liberal cut-off point of 21 was used to indicate the presence of substantial cognitive impairment similar to that found in patients with dementia. No participant scored below 21, and therefore, no additional participants were excluded from analyses.

Wechsler Abbreviated Scale of Intelligence 2-subtests (WASI; Wechsler, 1999). The WASI is an abbreviated IQ measure appropriate for persons six to 89 years of age. This study administered the Vocabulary and Matrix Reasoning subtests to estimate IQ (FSIQ-2). The FSIQ-2 version takes approximately 20 minutes to administer. The FSIQ-2 has good validity compared to full-scale IQ and test-retest reliability (reliability coefficients for adults range from .93 to .98).

Matrix reasoning (Wechsler, 1999). This subtest of the WASI was used as a measure of fluid intelligence including visuospatial organization and abstract reasoning. Thirty-five incomplete geometric patterns are presented in a matrix or series to participants. Participants are asked to select the response option that completes the matrix or series. Matrix reasoning has good construct validity r = .84 (Canivez et al., 2009), and reliability, α =.94 (Abu-Hilal et al., 2011).

Woodcock-Johnson Concept Formation Test (WJ-III; Woodcock, Mather, & McGrew, 2001). The WJ-III Concept Formation (WJ-CF) test served as a measure of effortful category learning. This test is normed for individuals between 2 and 90 years of age. Participants were asked to look at colored shapes that vary on four dimensions (color, shape, size, and number) and derive the rule why as to why some are shapes are placed in a category (i.e., inside a box). For instance, participants viewed a set of shapes on the left and a box on the right that contains different shapes. Participants then stated the rule that defined how the shapes in the box were similar to one another and different from those on the left. The Concept Formation subtest of the WJ-III has a strong test-retest reliability coefficient of .94.

Trail Making Test (TMT) Parts A & B (Delis et al., 2001). The version of the TMT used in the present study was taken directly from the Delis-Kaplan Executive Function System (D-KEFS). This version of the TMT includes five subtests presented to participants in the following order: visual scanning, number sequencing, letter sequencing, number-letter switching, and motor speed. The visual scan sub-test presents participants with an array of numbered circles. Participants are asked to locate all the circles with the number '3' and make a small slash through only this number. The number sequencing sub-test presents participants with an array of numbered circles (1-16) among distractor letters. Participants are asked to draw a line to connect the numbers in ascending order (i.e., 1, 2, 3….). This same procedure is used for the letter sequencing sub-test wherein participants are asked to connect the letters in ascending alphabetical order (i.e., A, B, C…); distractor numbers appear on the same page. In the number-letter sequencing subtest, the circles include both numbers (1-16) and letters (A -P). Participants are asked to draw a line connecting the circles in an ascending pattern, while alternating between numbers and letters (i.e., 1-A-2-B, etc.). Unlike the previous three sub-tests, the number-letter

switching subtest measures both processing speed and executive functioning (i.e., switching from numbers to letters). Finally, the motor speed sub-test was designed to assess participant's baseline motor drawing speed. In this sub-test participants are simply asked to trace over a dotted line that passes through 32 blank circles. The path outlined for participants is the exact same path participants follow to complete the number-letter sequencing subtest. Participants are timed during each sub-test. During the first four sub-tests participants are told to work as quickly and accurately as possible. In the motor speed sub-test participants are told to work as quickly as possible, and it was emphasized that neatness (i.e., perfectly tracing over the dotted line) was not important. Total time to complete all five sub-tests is approximately three to five minutes. The internal consistency of the TMT ranges from .60 to .81 (Stephens, 2014).

Rey Auditory Verbal Learning Test (RAVLT; Rey, 1964; see Lezak, 1983 for English version). This brief memory test assessed episodic and recognition memory. During this task, participants listened to a 15-item list of semantically unrelated words. The list was pre-recorded and presented via computer speakers (1 word per second). Immediately following this, participants recalled as many of the words as they could remember; order did not matter. This procedure was repeated three times and included the same list of words for each trial. The percentage of freely recalled words on each trial was calculated by taking the average number of words freely recalled and dividing by 15. Similarly, total percent recalled was calculated by taking the average number of words recalled across all three trials and dividing by the total number of words that were possible (i.e., 45 total items). Following participants' third attempt to freely recall the list of words, they were given a recognition test. The recognition test consisted of 50 words: 15 previously heard (target words), 10 semantically-related lures, 7 phonetically-similar lures, and 18 unrelated words (i.e., neither semantically or phonetically similar to target

words). This task took approximately 5 minutes to complete. Recognition accuracy (%) was calculated by summing the number of hits (H) and correct rejections (CR) multiplied by 100, and divided by the total number of words.

Apparatus for conditioning task. Skin conductance response (SCR) was used as the dependent measure for the conditioning task and was monitored by a MP35 four channel data acquisition unit (BIOPAC Systems, Goleta, CA). SCRs from the participant's dominant hand using silver-silver chloride electrodes (Ag-AgCl) were attached to the top phalanges of the first and third digits by velcro straps. A saline-based gel (Signa Gel; BIOPAC Systems) was used as a conductive electrolyte and placed between the Ag-AgCl electrode and the participant's skin. Consistent with previous literature, skin conductance was monitored using a constant voltage of 0.5 V at a sampling rate of 200 Hz (Labar, Cook, Torpey, & Welsh-Bohmer, 2004; Lykken & Venables, 1971).

Stimuli for prototype task. The prototype formation task consisted of black-and-white drawings of imaginary cartoon animals each belonging to a different category (i.e., different animal family). The animal stimuli created for this task followed the methodology described by Younger (1985; 1990) and adapted by Klinger and Dawson (2001). Each category consisted of individual members. These individual members resembled one another but varied on specific features along five quantitative dimensions. For instance, one feature (e.g., wings) varied on length with a size of 1 designated the shortest and a size of 5 designated the longest (see Figure 1). Only four features were selected to vary (e.g., legs, nose, arms, wings) per category. There were a total of six different categories and each category was given a novel one-syllable name to denote category membership (e.g., MIP category, DAK category). All categories had a similar

number of overall features (i.e., a head, arms, legs, wings, nose, etc.), in order to equate visual complexity across categories.

Procedure

Participants with typical development were scheduled for a two-hour session. Participants with ASD were scheduled for a three-hour session due to the additional diagnostic evaluation. During this session, consent forms were signed, and participants were given an overview of the experiment (10 minutes). After the consent process, participants with ASD were administered the ADOS-2 followed by the automatic processing tasks. Because the ADOS-2 was not administered to participants with typical development, the two automatic processing tasks (i.e., classical conditioning and prototype formation) immediately followed the consent process. Competition time for across both of these two tasks was approximately 20-22 minutes. The order of presentation for the classical conditioning task and the prototype formation task was counterbalanced across participants in order to eliminate potential order effects. After completing both automatic processing tasks, participants were given a short break. Following the break, the participants were given the MoCA (10 minutes), the IQ assessment (WASI; approximately 20 minutes), and the WJ-III Concept Formation task (5-10 minutes). Following these tests participants were again offered a short break before completing the TMT and RAVLT to conclude the testing session. Each participant received $30 as compensation for their participation in this study.

Classical conditioning task. Prior to the classical conditioning task, electrodes were attached to the index and ring finger of the dominant hand. The experimenter then recorded the participant's baseline SCL (approximately 3-4 minutes) followed by presentations of the UCS to the participant at 95 dB through headphones. To ensure the volume level of the UCS so that it

was in no way painful to the participant, the experimenter adjusted the volume for each participant to loudest tolerable level while at the same time ensuring that the UCS continued to elicit a startle response (e.g., an increase in SCR). Following this, the experimenter allowed the participant's SCR to reach a stable baseline activity level before commencing with the conditioning task.

For the conditioning task, participants were told that they were about to participate in an experiment where they would view a series of colored squares presented on the computer screen, and they were warned that they would occasionally hear the loud noise (UCS). Participants were asked to remain as motionless as possible to reduce the possibility of excessive movement confounding the SCR recording. Following this instruction, participants were told to play close attention because a brief memory test would follow the experiment. The experimenter was present during the conditioning task to monitor SCRs and excessive anxiety in response to the loud noise and to ensure that participants were focused on the task. The experimenter was positioned adjacent to the participant. Only a computer screen and the electrodes attached to the fingers were in the line of sight of the participant.

The classical fear conditioning paradigm used to assess associative learning consisted of three phases: habituation, acquisition, and extinction. The presentation of the habituation and acquisition phases was continuous so participants were not aware of the transition from one phase to the next. Two colored squares (e.g., red and blue) were used as the stimuli. During the habituation phase participants were randomly presented with these two colored squares, with each colored square presented four times (8 trials total). During the acquisition phase one colored square was randomly paired with a co-terminating loud noise UCS and designated as the conditioned stimulus (CS). The other colored square was never paired by the UCS and

designated as the neutral stimulus (NS). Each colored square (CS and NS) was presented for five seconds. The UCS lasted for two seconds immediately following the presentation of the CS only (i.e., the CS-UCS trials lasted 7 seconds). The inter-stimulus interval between trials was set at 15 seconds and SCR was monitored to ensure that participant's SCR had returned to baseline. This phase consisted of eight presentations of CS reinforced by the UCS and eight presentations of the NS (16 trials total).

Following the acquisition phase participants received an explicit awareness questionnaire to assess declarative knowledge of the CS-UCS contingencies. Based on previous work (Bechara et al., 1995; Gaigg & Bowler, 2007; LaBar et al., 2004), participants were asked to recall the number and type of colors previously seen, how many colors preceded the presentation of the loud noise, and finally what specific color(s) preceded the loud noise. Participants were only classified as aware if they were able to accurately report that the CS and only the CS predicted the presentation of the UCS. Following the explicit awareness questionnaire, participants received the extinction phase. The extinction phase consisted of four presentations of the CS without the UCS (non-reinforced) and four presentations of the NS to ensure full extinction had taken place (8 trials total).

Prototype formation task. The prototype task consisted of five blocks (i.e., five animal categories) that each included a familiarization and a test phase. Each of the five blocks (including both a familiarization and test phase) included animals representing distinct animal categories. The order of each animal category (i.e., each block) was randomized for every participant. The familiarization phase consisted of individual animals created by combining feature values 1, 2, 4, and 5. Each individual animal was randomly presented once during familiarization with 8 different animals presented. Each presentation lasted for eight seconds

before continuing on to the next animal presentation. For the first three blocks a recognition test followed the familiarization phase and included three stimulus types: (1) animals previously viewed in the familiarization phase (old), (2) animals not presented in the familiarization phase (novel), and (3) the novel prototype. There were a total of nine test trials including three old animals, three novel animals, and three prototypical animals. During the test phase, participants were presented with these three stimulus types (old, new, prototype) one at a time and asked to say "yes" if they previously saw the animal or "no" if they did not see the animal. The last two blocks included an additional confidence rating for each recognition response. After each recognition judgment, participants were asked, "How confident are you that you did/did not see this animal?" The confidence rating included a scale of 0 (not confident at all) to 100 (entirely confident). Evidence of prototype learning was demonstrated when participants falsely recognized having seen the prototype more often than they falsely recognized novel animals.

Results

Statistical analyses. Data analyses were completed using SPSS IBM 23 statistical software. Means and standard deviations for each test of cognitive functioning are reported in Table 2. In order to compare diagnostic and age effects, separate regression analyses were conducted using MoCA, WJ-CF, RAVLT and TMT as the dependent variables. The predictor variables in these analyses included FSIQ-2, age, diagnosis, and the age by diagnosis interaction term. FSIQ-2 and age were used as continuous predictors and diagnosis was used as a categorical predictor. In order to prevent multi-collinearity between the predictor variables, age was mean-centered prior to analyses.

FSIQ-2 was treated as a covariate in these analyses for two reasons: (1) previous evidence has suggested individual differences in intellectual functioning account for substantial

variance in many measures of cognitive functioning, thus potentially masking age effects (Bolla, Lindgren, Bonaccorsy, & Bleecker, 1990; Schaie, 1983); (2) initial inspection of the current data revealed a significant correlation between age and FSIQ-2, $r(58) = .35$, $p < .01$. Therefore, including FSIQ-2 in the model provided an examination of the effect of age and diagnosis on measures of effortful processing above and beyond participants' level of intellectual functioning.

Using stepwise regression, FSIQ-2 was entered first. This was followed by age, diagnosis, and the age by diagnosis interaction term each being entered sequentially to determine whether age affected each cognitive measure and whether ASD was associated with a different rate of age-related decline than seen in typical development. Curvilinear relations were also examined in these analyses. However, no significant curvilinear effects were observed, thus only linear relationships were included in the final regression models reported here. Finally, when a significant interaction was observed, separate regression analyses were conducted on each diagnostic group to further probe the nature of the interaction. Standardized beta coefficients with *p*-values are reported for each dependent variable in Table 3.

Analysis of Effortful Processing Tasks

MoCA. Table 2 shows that performance on the MoCA was significantly lower in adults with ASD ($M = 25.9$; $SD = 2.4$) compared to adults with typical development ($M = 27.1$; $SD = 2.1$), $t(57) = 2.05$, $p = .05$. The regression analysis excluded the score from the delayed recall item from the MoCA. This item was excluded because it was determined that time between immediate recall and delayed recall depended upon the speed with which a participant completes the intervening items (i.e., items 5 – 10). Thus, the time between immediate recall and delayed recall was not consistent across participants.

As can be seen in Table 3, this analysis found FSIQ-2 to be positively related to MoCA performance, although this was not significant, explaining approximately 4% of the variance (FSIQ: $R^2_{change} = 0.04$, $F(1,57) = 2.51$, $p = .12$). In contrast, both age and diagnosis were negatively related to performance and accounted for 13% and 15% explained variance, respectively (Age: $R^2_{change} = 0.13$, $F(1,56) = 9.05$, $p = .004$; Diagnosis: $R^2_{change} = 0.15$, $F(1,55) = 11.85$, $p = .001$). Of particular interest, was the significant increase in variance explained by the interaction term ($R^2_{change} = 0.05$, $F(1,54) = 4.64$, $p = .04$), suggesting that both age and diagnosis had a negatively combined impact on MoCA performance. To further examine this interaction, separate regressions were conducted for each diagnostic group. Only age was included as a predictor in this analysis given that FSIQ-2 was not a significant predictor in the overall analysis. Examination of MoCA performance in adults with typical development revealed that age explained <1% of the variance in MoCA ($R^2_{change} < 0.01$, $F(1,28) = 0.01$, $p = .91$), whereas in adults with ASD, age explained 34% of the variance ($R^2_{change} = 0.34$, $F(1,27) = 13.67$, $p = .001$). These results suggest that age had a substantial and negative impact on MoCA performance in individuals with ASD but very little impact on performance in individuals with typical development. However, the smaller impact of age on adults with typical development was somewhat expected considering that the MoCA was originally designed to detect early signs of mild cognitive impairment (MCI), and, therefore, may be less sensitive to normal age-related declines (Julayanont, Phillips, Chertkow, & Nasreddine, 2013).

WJ-III concept formation. Using the same stepwise regression procedure, performance on the WJ-CF was examined[5]. Table 2 shows that adults with ASD scored lower on this measure ($M = 29.1$) compared to adults with typical development ($M = 33.2$). Regression analysis of these

[5]It should be noted that one individual with ASD did not complete this measure due to a time constraint

scores demonstrated FSIQ-2, age, and diagnosis were significant predictors of performance (see Table 3). FSIQ-2 explained 19% of the variance, (R^2_{change} = 0.19, F(1,56) = 13.37, p = .001, whereas age and diagnosis uniquely contributed an additional 10% and 9% of the variance, respectively (Age: R^2_{change} = 0.10, F(1,55) = 7.68, p = .008; Diagnosis: R^2_{change} = 0.09, F(1,54) = 7.43, p = .009). These findings indicated that FSIQ-2 was positively related to WJ-CF performance, with those with higher IQ performing better on the task. However, age and diagnosis were negatively related to performance, with both those with ASD and those who were older performing worse on the task. The interaction between age and diagnosis was also significant, explaining 5% of the variance, R^2_{change} = 0.05, F(1,53) = 4.95, p = .03 (see Table 3 for standardized regression coefficients).

To further examine this interaction separate regression analyses, using only FSIQ-2 and age as predictors, were conducted for each diagnostic group. Analyses of adults with typical development revealed FSIQ-2 was not a significant predictor of WJ-CF, explaining approximately 4% of the variance (R^2_{change} = 0.04, F(1,28) = 1.03, p = .32), whereas age resulted in a 29% increase in explained variance (R^2_{change} = 0.28, F(1,27) = 11.54, p = .002), suggesting that performance declined with age in this group. This same analysis performed on individuals with ASD revealed FSIQ-2 accounted for 48% of explained variance (R^2_{change} = 0.48, F(1,26) = 24.10, p <.01). However, age was not a significant predictor, accounting for only a 1% increase (R^2_{change} = 0.01, F(1,25) = 0.62, p = .44). Therefore, the lack of an age effect in individuals with ASD was largely related to intellectual functioning, such that higher IQ resulted in better WJ-CF performance.

Free recall and recognition memory. In order to examine the relation between age and performance on the RAVLT, separate analyses were conducted on free recall and recognition

memory. For the analysis of free recall, the total number of items recalled across all three trials was used as the dependent variable. As can be seen in Table 2, adults with ASD recalled fewer words (M = 49.1%) compared to adults with typical development (M = 57.6%). This was also reflected in the regression analysis, which found diagnosis to be negatively related to performance, even after accounting for the positive relationship between FSIQ-2 and performance. Both were significant predictors of performance [FSIQ-2: R^2_{change} = 0.10, $F(1,57)$ = 6.57, p = .01; Diagnosis: R^2_{change} = 0.10, $F(1,55)$ = 7.30, p < .01]. Age was also negatively related to performance, yet age was only a marginally significant predictor in the model [Age: R^2_{change} = 0.05, $F(1,56)$ = 3.08, p = .08]. There was no significant age by diagnosis interaction, R^2_{change} < 0.01, $F(1,54)$ = 0.20, p = .65.

Considering that age was found to be a marginally significant predictor of immediate free recall, a multivariate regression analysis was conducted using all three immediate free recall tests as dependent variables and regressed on the same predictors described above (i.e., FSIQ-2, age, diagnosis, & age by diagnosis interaction term). Results from the multivariate tests showed FSIQ-2 and diagnosis were significant predictors of immediate free recall performance, Wilks' Lambda = .850, $F(3, 52)$ = 3.06, p = .04, η_p^2 = .15; Wilks' Lambda = .787, $F(3, 52)$ = 4.69, p < .01, η_p^2 = .21, respectively. As above, neither age nor the interaction between age and diagnosis were significant, Wilks' Lambda = .897, $F(3, 52)$ = 1.98, p = .13, η_p^2 = .10; Wilks' Lambda = .945, $F(3, 52)$ = 1.01, p = .40, η_p^2 = .06. However, further inspection of the univariate tests revealed that age was a significant predictor of performance on trial 1, $F(1,54)$ = 5.93, p = .02, η_p^2 = .10, but not on trials 2 and 3, $F(1,54)$ = 2.14, p = .15, η_p^2 = .04; $F(1,54)$ = 1.69, p = .20, η_p^2 = .03 respectively. Together, these findings suggest that repeated tests of immediate free recall improved performance in older adults regardless of diagnosis.

Recognition memory. A similar regression analysis performed on recognition accuracy revealed that diagnosis was a significant predictor. Indicating that ASD was associated with relatively lower recognition accuracy (*M* = 92%) compared adults with typical development (*M* = 95%), R^2_{change} = 0.07, *F*(1,55) = 4.36, *p* = .04. According to Table 2, it is likely that the relatively lower recognition accuracy in adults with ASD was due to the higher false recognition of semantically-related lures (*M* = 1.7) compared to adults with typical development (*M* = 0.9). There were no significant effects for FSIQ-2 (R^2_{change} = 0.04, *F*(1,57) = 2.05, *p* = .06), age (R^2_{change} < .01, *F*(1,56) = 0.18, *p* = .67), or age by diagnosis (R^2_{change} = 0.01, *F*(1,54) = 0.45, *p* = .51)

TMT. Regression analyses were performed on raw scores (i.e., seconds) from the visual, number, letter, and number-letter switching subtests of the TMT. Table 2 shows that performance on these subtests (including motor) was significantly slower in adults with ASD compared to adults with typical development (all *p*'s $\leq$ .004). Due to the significantly slower baseline motor speed in adults with ASD, raw scores from this subtest were included in the model to control for baseline differences in motor ability. Similar to the above analyses motor speed and FSIQ-2 were entered in the first step followed by age, diagnosis, and the age by diagnosis interaction term. Results for these analyses can be found in Table 4. Across all four subtests, age was a significant predictor of performance such that increased age was associated with increasingly slower performance (i.e., time to complete). In contrast, diagnosis was negatively related to performance on the visual scanning and number-letter switching subtests (β = .34, *p* < .01; β = .29, *p* = .01, respectively), but not the number and letter sequencing subtests (β = .22, *p* = .07; β = .15, *p* = .15). This suggests that performance on the number and letter sequencing subtests was similar between diagnostic groups after accounting for FSIQ-2, motor

speed, and age. There were no significant interactions between age and diagnosis on any of these subtests (see Table 4, all p's $\geq$ .08).

Cognitive flexibility. Given that the number-letter switching subtest assesses both sequencing (i.e., connecting numbers and letters in ascending order) and cognitive flexibility (i.e., switching between numbers and letters), a follow-up regression analysis was conducted to examine the impact of age and diagnosis on cognitive flexibility apart from the underlying component of sequencing. In order to do so, the average completion time across both the number and letter sequencing subtests was used as a covariate in this analysis. Thus, the following predictors were entered sequentially into the model: FSIQ-2, sequencing, age, diagnosis, and the age by diagnosis interaction term. FSIQ-2, sequencing, and age were significant predictors of performance. FSIQ-2 explained 12% of the variance, (R^2_{change} = 0.12, F(1,56) = 7.74, p = .007, whereas sequencing and age uniquely contributed an additional 38% and 9% of the variance, respectively (Speed: R^2_{change} = 0.38, F(1,55) = 41.61, p < .001; Age: R^2_{change} = 0.09, F(1,54) = 11.40, p = .001), suggesting that slower sequencing speed and increased age were related to poorer cognitive flexibility. Diagnosis was not a significant predictor explaining roughly 2% of the variance, R^2_{change} = 0.02, F(1,53) = 2.73, p = .10. However, the interaction between age by diagnosis was significant, explaining 3% of the variance, R^2_{change} = 0.03, F(1,52) = 4.59, p = .03. To further examine this interaction, separate regression analyses were conducted for each diagnostic group using FSIQ-2, sequencing, and age as predictors.

Analyses of adults with typical development revealed FSIQ-2 and age were not significant predictors of cognitive flexibility, explaining approximately 8% and 4% of the variance (FSIQ-2: R^2_{change} = 0.08, F(1,27) = 2.49, p = .13; Age: R^2_{change} = 0.04, F(1,25) = 1.58, p = .22). In contrast, sequencing accounted for a substantial amount of variance, (R^2_{change} = 0.25,

$F(1,26) = 9.80$, $p < .01$). This same analysis performed on adults with ASD revealed FSIQ-2, sequencing, and age were all significant predictors of cognitive flexibility. FSIQ-2 accounted for 23% of explained variance ($R^2_{change} = 0.23$, $F(1,27) = 8.07$, $p < .01$), sequencing accounted for 25% of explained variance ($R^2_{change} = 0.25$, $F(1,26) = 12.62$, $p < .01$), and age accounted for 19% of explained variance ($R^2_{change} = 0.19$, $F(1,26) = 14.68$, $p < .01$). Despite the substantial amount of variance explained by sequencing, age remained a significant predictor of performance in adults with ASD, suggesting that aging in ASD may be characterized by greater declines in cognitive flexibility.

Analysis of Automatic Learning Tasks[6]

Prototype learning. For the prototype task, prototype learning was assessed by comparing the percentage of "yes" responses to the prototype versus the percentage of "yes" responses to the new stimulus. As can be seen in Table 5, on average, adults with ASD were 30% more likely to say "yes" to the prototype compared to the new stimuli, $t(28) = 6.41$, $p < .01$. Similarly, adults with typical development were 40% more likely to say "yes" to the prototype compared to the new stimuli, $t(29) = 9.77$, $p < .01$. Confidence ratings were also examined and revealed that both adults with ASD and adults with typical development were more confident that they had previously seen the prototype, $M = 75\%$, compared to the new stimuli, $M = 67\%$ (Table 5). Because confidence ratings did not differ across diagnostic groups or age (p's $\geq .25$), they are not discussed further in the following section.

Prior to the regression analysis d-prime (d') was calculated by subtracting the standard scores (Z-score) from recognition of the prototype by recognition of the new stimuli (i.e., Zprototype – Znew). This index of prototype learning was then used as the dependent variable in

[6]Preliminary analyses indicated that FSIQ-2 was not related to conditioning or prototype learning (p's > .41), therefore it was excluded from this analysis.

a hierarchical linear regression using a stepwise method. For this regression model, age was entered as the first predictor, followed by diagnosis, and the age by diagnosis interaction term. Results from this analysis revealed that age and diagnosis accounted for small, non-significant variance (Age: R^2_{change} = .01, p =.56; Diagnosis: R^2_{change} = .02, p =.27). Additionally, the age by diagnosis interaction term accounted for 4% of the variance, but it was also non-significant (R^2_{change} = .04, p = .14). These findings suggest that prototype learning was similar between adults with and without ASD, and unaffected by age.

Classical conditioning. To ensure that adults with ASD and adults with typical development had equivalent baseline skin conductance levels (SCL) prior to the acquisition phase (sampled at 200Hz), SCL values were examined during the habituation phase. This analysis demonstrated statistically equivalent baseline SCL values between adults with ASD (M = 6.81µS, SD = 3.84) and adults with typical development (M = 6.50µS, SD = 2.90), $t(29)$ = 0.35, p = .72. However, two participants with ASD and two participants with typical development were excluded from analyses due to extremely low baseline SCL that prevented reliable recording of electrodermal activity (ASD n = 27; Typical n =28).

Data processing. Following confirmation of equivalent baseline SCL values between diagnostic groups, a range-correction procedure was performed on the SCL values taken from the acquisition phase in order to correct for inter-individual variance (i.e., some participants showed large variability in SCL, while others showed small variability). This range correction was done by selecting each SCL value, subtracting this value from their minimum SCL, and then dividing by the maximum SCL minus the minimum SCL values for each participant. Following this correction SCR scores were calculated using a peak and valley method. Due to the slow potentiation of the SCRs, usually occurring 1000ms after stimulus onset, the peak skin

conductance (SC) value for each trial was taken within a 6 –second window prior to the UCR onset (typically occurring 7000ms after trial onset). This peak response was then subtracted by the SC value (valley) selected at trial onset (Pineles, Orr, & Orr, 2009; South et al., 2011).

Inspection of the SCR data revealed substantial positive skew. Thus, SCRs were square-root transformed to normalize distribution of the data (Gaigg & Bowler, 2007). Kolmogorov-Smirnov tests on the square-root transformed SCR scores indicated a normal distribution for both CS trials, $D = .09$, $p = .20$, and NS trials, $D = .11$, $p = .10$.

SCR amplitude to the UCS. Because participants were allowed to adjusted the volume of the UCS to be as loud as tolerable with a maximum allowed of 95 dB, we compared the adjusted decibel levels of the two diagnostic groups. The ASD group adjusted the intensity (dB level) of the UCS slightly, but significantly lower ($M = 83.25$dB, $SD = 3.38$) than individuals with typical development ($M = 85.25$dB, $SD = 2.10$), $t(56) = 2.33$, $p = .02$. Due to this difference we compared the unconditioned response (UCR) elicited by the aversive sound across the diagnostic groups. This comparison was necessary to ensure that any group differences in conditioning were not affected by these differences in volume of the stimuli and differences in sensitivity to those volumes. Both diagnostic groups exhibited a strong UCR (Typical: $M = 0.52\sqrt{\mu}S$; $SD = 0.26$; ASD: $M = 0.52\sqrt{\mu}S$; $SD = .21$), and did not significantly differ between diagnostic groups, $t(56) = 0.05$, $p = .96$.

Regression analysis. Table 5 shows the mean difference in SCR to the CS compared to NS stimuli across trials and diagnostic group (excluding the first CS trial). On average, there was a $.13\sqrt{\mu}S$ increase to the CS across trials compared to a $.11\sqrt{\mu}S$ increase to NS trials. This was a significant difference, $t(54) = 2.41$, $p = .02$, Cohen's $d = .35$. To examine age-related changes in conditioning as well as possible interactions between age and diagnosis, the difference score,

calculated by subtracting the average response to the NS from the average response to the CS, was used as the dependent variable in the regression analysis. Thus, positive values indicated relatively higher responses to the CS compared to the NS, and negative values indicated higher response to the NS compared to the CS.

A hierarchical linear regression using a stepwise method was then conducted using this difference score as a dependent variable. Due to the difference in adjusted volume level of the UCS between adults with ASD and adults with typical development, decibel level (dB level) was entered in the first step as a covariate followed by age, diagnosis, and the age by diagnosis interaction term.

This analysis found that dB level did not account for significant variance, explaining approximately 1% (dB level: $R^2_{change} = 0.01$, $F(1,53) = 0.30$, $p = .58$). Age was also a non-significant predictor accounting for less than 1% of explained variance ($R^2_{change} = 0.01$, $F(1,52) = 0.28$, $p = .60$, whereas diagnosis was a marginally significant predictor accounting 7% of explained variance, $R^2_{change} = 0.07$, $F(1,51) = 3.75$, $p = .06$, suggesting poorer conditioning in adults with ASD compared to adults with typical development (Table 5). However, this was qualified by a significant age by diagnosis interaction which explained 8% of the variance ($R^2_{change} = 0.08$, $F(1,50) = 5.04$, $p = .03$, see Figure 3). To further examine this interaction, separate regressions including the dB level and age as predictors were conducted for each diagnostic group. For adults with typical development neither the dB level nor age were significant predictors of learning. Decibel level explained less than 1% and age explained 4% [dB level: $R^2_{change} < 0.01$, $F(1,26) = 0.20$, $p = .89$; Age: $R^2_{change} = 0.04$, $F(1,25) = 0.95$, $p = .34$]. The same analysis performed on adults with ASD showed that dB level accounted for approximately 7% of the variance in learning [$R^2_{change} = 0.07$, $F(1,25) = 1.98$, $p = .17$] and age

contributed an additional 18% of explained variance, $R^2_{change} = 0.18$, $F(1,24) = 5.63$, $p = .03$]. As can be seen in Figure 3, the poorer conditioning observed in adults with ASD appears to be largely driven by older adults with ASD, suggesting age may adversely impact conditioning in adults with ASD compared to adults with typical development.

Multivariate Analysis of Effortful Processing Tasks

The pattern of performance across seven measures of effortful processing demonstrated relatively consistent effects of age (Tables 3 & 4) and diagnosis (Tables 2-4), yet evidence was mixed regarding the interaction between age and diagnosis. Therefore, a final multivariate analysis was conducted to examine the overall pattern of performance on measures of effortful process. To perform this analysis, scores were taken from the WJ-CF, RAVLT, MoCA, and the visual scanning, number sequencing, letter sequencing, and number-letter switching subtests of the TMT[7] and were converted into standard scores (Z-scores). Standard scores from these variables were then entered as dependent variables (7 total). Following this, FSIQ-2, age, diagnosis, and the age by diagnosis interaction term were entered into the model. Results from the multivariate analysis revealed FSIQ-2 to be a significant predictor of overall performance, Wilks' Lambda = .482, $F(7, 46) = 7.08$, $p < .01$, $\eta_p^2 = .52$. Age and diagnosis were also significant predictors of overall performance, Wilks' Lambda = .567, $F(7, 46) = 5.02$, $p < .01$, $\eta_p^2 = .43$; Wilks' Lambda = .532, $F(7, 46) = 5.78$, $p = .01$, $\eta_p^2 = .47$, respectively. The main effects of age and diagnosis were qualified by a significant age by diagnosis interaction, Wilks' Lambda = .710, $F(7, 46) = 2.70$, $p = .02$, $\eta_p^2 = .29$. As can be seen in Figure 4, age was associated with relatively poorer performance in both adults with and without ASD, except for

[7]Prior to converting to standard scores, unstandardized residuals were calculated by regressing each subtest on motor speed. Following this, the inverse of the unstandardized residuals was taken such that better performance was now associated with higher scores.

performance on the WJ-CF which showed poorer performance in adults with ASD regardless of age. Nevertheless, in six out of the seven measures of effortful processing (e.g., TMT, Free Recall, & MoCA), age appeared to have a disproportionately larger impact on performance in adults with ASD compared to adults with typical development, suggesting that aging in ASD may be associated with greater declines in cognitive functioning.

Processing Speed versus Processing Resources Account

In order to examine whether the pattern of age-related declines observed in the present study can be accounted for age-related declines in processing speed or age-related declines in processing resources, mediational analyses were performed on composite scores of processing speed (PS) and processing resources (PR). The composite measure of PS was calculated by first parceling out the effect of motor speed on the visual scanning, number sequencing, and letter sequencing sub-tests of the TMT and then converting them into standard scores (zScores). Following this, the overall average across the three subtests was computed. Importantly, the number-letter switching subtest was excluded from this composite score due to the additional cognitive resources (i.e., cognitive flexibility) associated with performance on this subtest, and instead included as part of the composite score for processing resources (PR). PR was calculated by averaging the standard scores (zScores) from the RAVLT, WJ-CF, Matrix Reasoning subtest (WASI), and Number-Letter Switching subtest. These variables were chosen because they were thought to represent measures which require a substantial amount of cognitive effort.

Following calculation of these composite scores, two mediation models were tested. Figure 5 shows the standardized regression coefficients from these models. As can be seen in Figure 5a, the relationship between age and PR, as mediated by PS, was examined first. In Step 1 of this mediation model, the regression of PR on age, ignoring the mediator of PS, was

significant, b = -.018, $t(57) = 2.16$, p =.04, suggesting age-related declines in processing resources. Step 2 tested the effect of potential mediator (PS) on PR. This was also significant, b = -.023, $t(57) = 5.53$, p <.001, indicating faster processing speed was associated with greater processing resources. Step 3 of the mediation analysis revealed that, controlling for the mediator (PS), age was no longer a significant predictor of PR, b = -0.01, $t(57) = 0.59$, p =.56. A Sobel test was conducted, indicating that PS fully mediated the relationship between age and PR ($z = 2.67$, $p = .01$).

A similar mediational analysis was performed using PR as the mediator in the model (Figure 5b). In Step 1 of this model, we regressed PS on age, ignoring the mediator of PR, and found this to be significant, b = .667, $t(57) = 3.16$, p =.003, suggesting a strong negative relationship between age and PS. Step 2 tested the effect of the mediator PR on PS. This was also significant, b = -15.15, $t(57) = 5.53$, p <.001. Step 3 of the mediation analysis revealed that, controlling for the mediator (PR), age was still a significant predictor of PS, b = .406, $t(57)$ = 2.18, p =.03. A Sobel's test was conducted and confirmed partial mediation ($z = 2.04$, $p = .04$).

Results from these analyses showed that controlling for PS eliminated all relationship between age and PR. In contrast, controlling for PR decreased but did not eliminate the relationship between age and PS, which remained significant. Taken together, these findings suggest that age-related declines in processing speed may represent the fundamental change in cognitive processing that influences performance on other measures of cognitive functioning in this study.

Discussion

This study examined age-related cognitive changes in adults with ASD and typical development using ten measures of cognitive processing. Adults with ASD demonstrated

relatively poorer performance in all of the measures designed to assess more effortful cognitive processing (e.g., MoCA, WJ-CF, RAVLT, & TMT), but relatively similar performance across two measures designed to assess more automatic processing (e.g., classical conditioning & prototype formation) compared to adults with typical development. However, analyses that sought to determine if age had a differential impact on cognitive performance in adults with ASD relative to adults with typical development revealed a mixed pattern of results. For instance, in three of these measures, adults with ASD showed greater age-related declines in ASD compared to age and IQ-matched adults with typical development. These included the MoCA, number-letter switching (e.g., TMT), and classical conditioning. Four measures showed a pattern of similar age-related decline in adults with ASD and typical development, free recall (RAVLT) and three of the TMT subtests (e.g., visual scanning, number sequencing, & letter sequencing). Finally, three measures showed no age-related decline in ASD: recognition memory (RAVLT), concept formation (WJ-CF), and the prototype learning. In order to further clarify the nature of age-related decline in ASD, the following sections will specifically address the pattern of performance for each task.

Evidence of atypical aging in ASD

Montreal cognitive assessment (MoCA). Performance on the MoCA revealed that age had a significant impact on performance in individuals with ASD, but performance did not change with age for individuals with typical development. Given that the MoCA was originally designed to detect early signs of mild cognitive impairment (MCI), the strong relationship between age and MoCA performance in adults with ASD suggests that aging in ASD may be associated with an increased risk for MCI. Although this interpretation may be tempered by the fact that the diagnostic validity of the MoCA has yet to be established in adults with ASD,

MoCA performance did not differ between adults with ASD and adults with typical development who were younger than 45 years old, suggesting that adults with ASD greater than 50 years-old may be experiencing significant cognitive difficulties associated with MCI.

Number-letter switching subtest. Further evidence that aging in ASD may be characterized by atypical declines in cognitive functioning was provided by analysis of the number-letter switching subtest of the TMT after controlling for sequencing speed. As previously mentioned, sequencing speed was included as a covariate in order to separate out the switching component or cognitive flexibility specifically associated with this subtest. Results from this analysis showed an overall effect of age on cognitive flexibility such that increased age was associated with poorer cognitive flexibility. Importantly, the effect of age was even more pronounced in adults with ASD, suggesting that age disproportionately impacted the cognitive flexibility needed to switch back and forth between numbers and letters. Because cognitive flexibility is often considered to be a component of executive functioning ability (Arbuthnott & Frank, 2000), it is possible that the greater impact of age on performance in adults with ASD suggests greater age-related declines in executive functioning. This is consistent with studies of executive dysfunction in individuals with ASD between 16 and 66 years old (Ambery et al., 2006; Bramham et al., 2009; Goldstein et al., 2001; Hill & Bird, 2006; Lopez et al., 2005; Minshew et al., 2002; Shafritz et al., 2008), although the effect of age was not examined in these studies. In fact, the only study to examine age-related changes in executive functioning using the TMT found no difference between older adults with ASD (> 50 years old) and older adults with typical development (Geurts & Vissers, 2012). They also did not find an effect of age on this task. However, unlike this study, the current study included both younger and older adults and

number and letter sequencing speed as a covariate which may have increased the likelihood of detecting age-related changes in cognitive flexibility.

The decision to include sequencing speed as a covariate highlights another important issue related to the study of cognitive aging and autism. That is, measures designed to assess specific cognitive abilities are rarely able to isolate a single cognitive process. This is particularly evident for measures of executive function wherein performance depends upon coordination of several cognitive processes including motor and perceptual speed, working memory, attention, cognitive flexibility, and inhibitory control (Diamond, 2013). As was the case the in the current study, the number-letter switching subtest involves two different cognitive components (i.e., sequencing and switching). Thus, properly controlling for sequencing speed permitted a specific examination of cognitive flexibility which aided in a more specific understanding of the pattern of cognitive decline in adults with ASD.

Classical conditioning. Initial examination of performance on the differential classical fear conditioning task showed similar conditioning between adults with ASD and adults with typical development. However, regression analyses revealed a significant age by diagnosis interaction, wherein age negatively impacted conditioning in adults with ASD, but did not impact conditioning in adults with typical development. Importantly, the slightly lower volume level preferred by adults with ASD compared to adults with typical development did not affect responses to the unconditioned stimuli (UCS), thus eliminating the possibility that decreased conditioning in older adults with ASD was the result of diminished physiological reactivity to the aversive sound. Rather, the significantly poorer conditioning observed in older adults with ASD appeared to be caused by similar conditioned responses to both the conditioned and neutral stimuli, as reflected by difference scores that were either at zero or negative. This pattern

suggests that older adults with ASD may have experienced greater difficulty in discriminating between potentially threatening stimuli (i.e., the CS that was always paired with the UCS) versus safe stimuli (i.e., the NS which was never paired with the UCS).

Age-related changes in adults with developmental disabilities. These findings are somewhat consistent with previous studies that have observed greater age-related changes in conditioning in adults with DS and FX compared to control groups. However, unlike these studies, which have documented age-related declines that begin in middle adulthood, the current study primarily observed poorer conditioning in adults with ASD in their early to mid-sixties. Furthermore, most of these studies have used eye-blink conditioning tasks which are known to rely upon cerebellar and limbic system pathways (Steinmetz, Tracy, & Green, 2001), compared to differential fear conditioning tasks (similar to the one used in the present study) which have been linked to amygdala, hippocampal, and prefrontal brain regions (Jarrell et al., 1987; LaBar, LeDoux, Spencer, & Phelps, 1995; Morris, Friston, & Dolan, 1997). Thus, the poorer performance observed in older adults with ASD may indicated dysfunctional communication within or between the medial temporal lobes and prefrontal cortex.

Evidence of Similar Age-related Decline in ASD

Free recall. Immediate free recall from the RAVLT revealed a significant effect of diagnosis and marginally significant effect of age, but no significant age by diagnosis interaction. That is, on tests of free recall older adults recalled fewer items compared to younger adults, and adults with ASD recalled fewer items compared to adults with typical development. In contrast, examination of recognition memory show relatively poorer recognition accuracy in adults with ASD compared to adults with typical development, whereas age had little impact on performance regardless of diagnosis. Evidence of age-related decline in immediate free recall but not

recognition is consistent with previous studies in healthy older adults (Spencer & Raz, 1995), as well as a more recent study examining age-related changes in explicit memory in both adults with ASD and adults with typical development between 20 and 79 years (see Lever & Geurts, 2015). In contrast, the overall poorer performance observed for adults with ASD in the present study diverges from previous work which has typically shown unimpaired free recall in ASD for lists of unrelated words (Bowler et al., 2008; 2009; Lever & Geurts, 2015; Minshew & Goldstein, 2001). These results may be better understood by consideration of task manipulations. In two separate studies Bowler et al., (2009) and Lever and Geurts (2015) assessed immediate free recall using the same RAVLT paradigm across five consecutive trials and found no differences in performance between individuals with ASD compared to age and IQ-matched individuals with typical development. It is possible that the current study's assessment of free recall across only three trials may have underestimated performance in adults with ASD. This seems to be a reasonable interpretation given that by Trial 3 in the current study, the two diagnostic groups' performance was more similar than on Trials 1 and 2. Thus, adding two additional, likely equivalent, trials in a five trial sequence may have caused the two diagnostic groups to look more similar in the studies using five trials. This interpretation may also explain the combined effects of age and diagnosis wherein older adults with ASD recalled fewer words compared to younger adults with ASD as well as older adults with typical development, and suggests combined effects of age and diagnosis. While it is important to emphasize that the non-significant interaction between age and diagnosis suggests similar age-related declines in both diagnostic groups, it does not preclude the possibility that if free recall performance is impaired in ASD it may in turn be further compromised by the aging process. Yet, it is also important to point out that age-related declines in free recall may be somewhat mitigated by repeated trials.

As was the case in present study, repeated trials boosted performance in both older adults with ASD and typical development.

Visual scanning and sequencing. Visual scanning, number sequencing, and letter sequencing are commonly used as measures of processing speed (Salthouse, 2011). In the current study, both age and diagnosis were related to poorer performance on these subtests. However, there was no significant age by diagnosis interaction, suggesting a similar pattern of age-related decline in both adults with ASD and adults with typical development. These findings are consistent with previous evidence of processing speed declines in normal aging (Salthouse, 1991; 1996; 2004; 2009; Verhaeghen & Salthouse, 1997) as well as evidence of processing speed deficits in individuals with ASD (Goldstein et al., 2001; Travers et al., 2016) Interestingly, both Goldstein et al. (2001) and Travers et al. (2014) demonstrated that deficits in processing speed in children with ASD were more pronounced in young adults with ASD compared to age and IQ-matched peers. Based on these findings and findings from the current study, it is argued that processing speed deficits are not only sustained in ASD across adulthood, but are also subject to a similar rate of decline as seen in normal aging.

Age-invariant Performance in ASD

Recognition memory. Consistent with previous studies of normal aging (Spencer & Raz, 1995; Verhaeghen & Salthouse, 1997), recognition memory performance was not significantly related to age. However, there was a significant effect of diagnosis, such that adults with ASD exhibited poorer recognition accuracy compared to adults with typical development. Studies of recognition memory in ASD have typically shown similar performance between individuals with ASD and individuals with typical development (Boucher, Mayes, & Bigham, 2012; Bowler et al., 2000; Lever & Geurts, 2015). For instance, in a study of recognition memory in older adults

with ASD, Lever and Geurts (2015) presented participants with 15 target words (i.e., previously heard) and 15 new words (i.e., distractors). In contrast, the current study presented participants with 15 target words intermixed with 35 distractors that included both semantically and phonetically-related lures. Therefore, it is possible that the greater number of distractors may have impacted recognition accuracy in adults with ASD. Support for this was also evident by the higher false recognition of semantically-related lures in adults with ASD compared to adults with typical development. Therefore, it is possible that recognition tests which include a higher proportion of semantically-related distractors may increase the rate of false recognition in adults with ASD. Nevertheless, it is important emphasize that in spite of higher false recognition of related items, recognition of previously studied items much higher compared to free recall in both adults with ASD (92% vs 49%) and adults with typical development (95% vs. 58%). Better memory performance on tests of recognition versus free recall is consistent with previous studies of explicit memory in ASD (Bowler et al., 1997; Gaigg et al., 2008), as well as studies of normal aging (Craik, 2002; Craik & McDowd, 1987). A pattern of performance that has led to the 'Task Support Hypothesis' wherein the memory difficulties experienced by younger individuals with ASD and older adults with typical development are attenuated when cues are provided to facilitate retrieval (Bowler et al., 1997; Gaigg et al., 2008). In light of the present evidence that free recall was impaired in ASD and disrupted by age, it is possible that the memory difficulties experienced by older adults with ASD will be more prominent in the absence of any environmental cues to facilitate retrieval of the previously studied material. Thus, future work should examine age-related changes in explicit memory using both supported (recognition, cued-recall) and un-supported memory tests (free recall) to better understand the nature of age-related changes in explicit memory in adults with ASD.

Concept formation. The Woodcock-Johnson concept formation test (WJ-CF) was designed to assess more explicit or effortful rule-based category learning. The pattern of performance on the WJ-CF showed significant effects of age and diagnosis, such that older adults demonstrated lower performance than younger adults, and adults with ASD demonstrated lower performance compared to adults with typical development. However, there was also a significant age by diagnosis interaction. Follow-up within-group analyses determined that the significant effect of age was primarily driven by adults with typical development. Adults with typical development showed greater age-related declines in performance relative to adults with ASD. In contrast, the non-significant effect of age in adults with ASD was due to significantly lower performance in adults with ASD irrespective or age. This finding is consistent with previous evidence of impaired category learning in children and adolescents with ASD (Klinger et al., 2007; Minshew, Meyer, & Goldstein, 2002), and further suggests that category learning may be a particular type of learning that is disrupted in ASD across the lifespan.

Prototype formation. In contrast to the WJ-CF, the prototype formation task was designed to assess more implicit or automatic category learning. As previously mentioned, this particular form of category learning has been shown to be relatively preserved in studies of normal aging (Glass et al., 2012; Hess & Slaughter, 1986a; 1986b), whereas studies of implicit category learning in ASD have been mixed (Gastgeb et al., 2012: Klinger & Dawson, 2001; Klinger et al., 2007; Molesworth et al., 2005; 2008). Nevertheless, results from the present study revealed no age effect and similar performance between adults with ASD and adults with typical development, suggesting that implicit category learning was preserved in both older adults with typical development and older adults with ASD.

Finally, when comparing performance across both measures of category learning, adults with typical development exhibited a pattern of performance consistent with the notion that age has a greater impact on more effortful rule-based learning tasks (e.g., WJ-CF) and a smaller impact on tasks involving more implicit or automatic learning (e.g., prototype formation). Yet in adults with ASD, age did not impact performance on either task. Failure to observed the expected age-related changes on the WJ-CF in adults with ASD was primarily due to poorer overall performance, and highlights the importance of choosing measures that are sensitive age-related changes in both adults with ASD and adults with typical development. That is, if the measures chosen to study age-related decline happen to be ones proven to be consistently disrupted in ASD, then the ability to detect age-related changes may be reduced. Thus, future studies of aging in ASD should carefully consider whether the cognitive measures chosen to assess age-related declines in ASD are suitable for this population.

Effortful versus Automatic Processing

In order to test the prediction that aging in ASD may impact both effortful and automatic processing, the current study included cognitive measures that are sensitive to age-related decline (i.e., explicit memory, processing speed, etc.) as well as measures that are relatively age-invariant or less impacted by age (i.e., conditioning and prototype learning). In line with previous studies of normal aging, the current study found age-related declines on several measures of effortful processing in adults with typical development and adults with ASD. However, these age-related declines were found to be greater in adults with ASD across two of the measures of effortful processing and one measure of automatic processing (i.e., classical conditioning). Taken together, these findings suggest that ASD may be characterized by greater age-related declines in some cognitive processes relative to the cognitive declines experienced by adults with typical

development. This conclusion is further supported by the neuro-cognitive scaffolding theory of cognitive aging. This theory suggests that when faced with age-related degeneration, the aging brain will adapt by recruiting a greater number of brain regions in order to achieve particular cognitive goals (Cabeza, 2002; Goh & Park, 2009; Park & Reuter-Lorenz, 2009). However, various neuroimaging studies have found that when engaging in complex cognitive tasks, this type of neural compensation strategy may already be in use and possibly disrupted in individuals with ASD (Just et al., 2007; Kana et al., 2007; Schipul & Just, 2016). Assuming adults with ASD undergo similar age-related neuro-degeneration, these findings suggest that adults with ASD may have difficulty undergoing the same neural reorganization needed to respond to the aging brain. In other words, if young adults with ASD already recruit a greater number of brain regions to complete a complex cognitive task, then less neural compensation may be achievable as they face greater volumetric and functional declines with age. This possibility would then explain why the current study observed the greatest age-related declines on measures that require coordination among several different brain regions. For instance, both the MoCA and TMT are frequently used as measures of executive function (Arbuthnott & Frank, 2000; Lam et al., 2013; Vogel et al., 2015). An umbrella term used to describe a set of cognitive processes such as controlled attention, working memory, cognitive flexibility, problem solving and reasoning (Diamond, 2013; Hill & Bird, 2006). Consequently, performance on these measures also requires coordination among different brain regions including the medial temporal lobes (MTL) and prefrontal cortex (PFC; Li et al., 2014; Paul et al., 2011). Likewise, several studies have identified these regions as important for the acquisition of conditioned responses. For instance, in studies of classical fear conditioning, efficient communication between the MTL and PFC brain regions seems to play an integral in distinguishing between safe (i.e., neutral stimuli) and

threatening stimuli (i.e., conditioned stimuli; for review see Rozeske, Valerio, Chaudun, & Herry, 2014). Therefore, it is possible that within ASD, age-related declines in performance across these three measures may be associated with greater age-related disruption in the PFC. Furthermore, because PFC dysfunction has been associated with an increased risk for MCI (Julayanont, Philips, Chertkow, & Nasreddine, 2013), it is possible that the greater age-related declines in ASD signals an increased risk for MCI in this population. Increased risk for MCI has also been documented in patients with Parkinson's disease (PD), who often suffer from subtle cognitive deficits in executive function, memory, and visual-spatial abilities prior to the manifestation of motor symptoms (Boyle et al., 2005; Costa et al., 2015; Hoops, 2009; Svenningsson, Westman, Ballard, & Aarsland, 2012). Recent evidence has also documented a higher risk for PD in older adults with ASD (Starkstein et al., 2015). Given this evidence, it is reasonable to assume that the Parkinsonian symptoms exhibited by these older adults with ASD were also accompanied by similar deficits in cognitive functioning. Thus, future studies that include more comprehensive assessment of cognitive, motoric, and neuro-anatomical changes associated with aging in ASD will be important for understanding the relationship between cognitive functioning in older adults with ASD and the associated risk for certain neuro-degenerative diseases such as PD.

Processing Resources versus Processing Speed

Another goal of the present study was to test the theoretical predictions derived from the Processing Resources and Processing Speed accounts of cognitive aging. According to the Processing Resources account, age is associated with a reduction in cognitive resources or 'mental energy' (Craik & Rose, 2012). As a result, performance on effortful processing tasks, which require greater cognitive effort, tends to be more disrupted with age compared to tasks

which require fewer resources (i.e., automatic processing tasks). In contrast, the Processing Speed theory suggests that declines in information processing speed may represent a more fundamental cognitive change, wherein age-related slowing of information processing leads to subsequent impairments in other cognitive domains.

In order to determine whether the pattern of cognitive performance can be better accounted for by the Processing Resources or the Processing Speed theory mediational analyses were conducted. The first analysis examined the relationship between age and processing resources after controlling for processing speed. Consistent with previous literature (Salthouse, 1996; Verhaeghen & Salthouse, 1997), processing speed significantly mediated the relationship between age and processing resources, such that controlling for processing speed virtually eliminated the relationship between age and processing resources. In contrast, processing resources did not appear to fully mediate the relationship between age and processing speed. A relationship that remained significant. Overall, these findings suggest that aging may be characterized by general slowing of information processing which in turn disrupts performance on other measures of cognitive functioning. This finding is particularly important considering that processing speed deficits are a common feature of ASD. A deficit that, according the present study, increases with age. As a result, it is possible that more complex tasks that require simultaneous coordination of several different cognitive processes may become even more disrupted as individuals with ASD age.

Limitations

As with any study, there are some limitations that complicate the conclusions drawn from the present findings. First, the present study used a cross-section design. This choice of research design was necessary in order to conduct the study in a timely fashion. Historically, cross

sectional designs have consistently revealed that increased age is associated with lower levels of cognitive performance (Salthouse, 2009). Nevertheless, there are limitations of cross-sectional designs including individual differences in demographic variables (e.g., educational level, income, etc.) and cohort effects that prevent any strong conclusions regarding the changes in cognition over time for adults with ASD. Therefore, the current findings should be interpreted with caution until future longitudinal studies are able to replicate these findings.

Second, virtually all of the adults with ASD were high functioning (IQ > 80), thus it is difficult to generalize the present findings to more impaired individuals with ASD who may have co-morbid intellectual ability. Still, it is important to highlight that despite similar levels of intellectual functioning between adults with ASD and adults with typical development, adults with ASD exhibited a pattern of performance that was not only effect by their diagnosis but also appeared to be further compromised by age. For that reason, it is possible that individuals with ASD, regardless of intellectual functioning, may experience significant difficulties in cognitive functioning as they age.

Second, even though the current study verified ASD diagnoses with a standard diagnostic interview, several adults with ASD disclosed that they had received their initial diagnosis in adulthood which was somewhat expected considering that mild forms of ASD were rarely diagnosed when these adults were children. Nevertheless, the current findings may not be representative of the whole autism population, particularly those with more severe ASD symptomatology who are typically diagnosed much earlier in life. However, it should be noted that approximately 30% of adults diagnosed with ASD later in life received some type of diagnosis in childhood (e.g., ADHD, bi-polar, schizophrenia). Again, this highlights the

limitations of the diagnostic instruments available at the time rather than attributing adulthood diagnoses only to those with milder ASD symptomatology.

Finally, a number of adults with ASD reported co-morbid diagnoses ranging from ADHD, depression, and anxiety. To date, it is still unknown whether having a co-morbid diagnosis has a differential impact on older adults with ASD, leading to greater age-related decline. Additionally, it is well known that anti-depressant medication can impact some forms of cognitive functioning. However, preliminary analyses revealed no differences in cognitive performance between those who use and do not use anti-depressant medication. Thus, it seems unlikely that co-morbid diagnoses affected the results seen. Nevertheless, it is possible that co-morbid diagnosis affects age-related cognitive decline in ASD and future studies should continue to investigate this link.

Conclusion

The current study represents one of only three systematic investigations comparing age-related decline in adults with ASD to age and IQ-matched to adults with typical development. While previous studies in normal aging have documented fairly consistent patterns of age-related cognitive decline, studies involving older adults with other DDs (e.g., Down syndrome, Fragile X, and William's syndrome) provide evidence that cognitive functioning may be disproportionately impacted by age such that age resulted in earlier declines in both effortful and some automatic processing tasks. Given the current findings and the similar etiology of ASD to those other DDs, it is possible that adults with ASD face a similar risk for atypical age-related decline.

However, as a final observation regarding the pattern of performance in adults with ASD, not every older adult with ASD in this study exhibited dramatic declines in cognitive

functioning. For example, performance on the number-letter switching subtest appeared to show that some older adults with ASD demonstrated performance that was comparable to older adults with typical development. Therefore, it is possible that the current findings signal different trajectories of age-related decline in adults with ASD, with some showing normal patterns of age-related decline while others showed more dramatic declines in cognitive functioning. Although the current study did not have a large enough sample to explore age-related changes within different sub-groups of adults with ASD, future work examining aging in ASD should consider the possibility that there may be different trajectories of age-related decline in ASD. For instance, one possible variable that may contribute to different patterns of age-related decline in ASD is employment and community involvement. It is well known that having a job and remaining activity involved in the community can buffer older adults with typical development against age-related declines in cognitive functioning (Rowe & Kahn, 1997). Unfortunately, the employment and community involvement in adults with ASD is incredibility low, with some studies estimating as little as 66% of young adults with ASD who have never been gainfully employed (Roux et al., 2013). Thus, it is possible that adults with ASD who are able to find and maintain employment may demonstrate less age-related decline in cognitive functioning compared to adults with ASD who have trouble finding or maintaining employment. Arguably, adults with ASD who struggle with employment may do so because of greater impairment in cognitive functioning, however this possibility is unlikely given that the current sample of high-functioning adults with ASD were more likely to be unemployed due the social and communication impairments associated with ASD rather than global deficits in cognitive functioning.

Alternatively, it has been suggested that maintaining community involvement, which is often considered to be an important component for 'successful aging' in adults with typical development (Rowe & Kahn, 1997), may be less important for adults with ASD. This is related to the idea that the feelings of social isolation and loneliness accompanied by reductions in community involvement may be less common in adults with ASD who prefer solitary hobbies and interests (Happe & Charlton, 2012). Unfortunately, the extent to which employment and community involvement impact age-related declines in adults with ASD remains unclear, thus future studies of environmental factors will be important for understanding whether factors such as community involvement and active lifestyles that contribute to the maintenance of cognitive functioning the aging in typical adults are also important for aging in adults with ASD. In order to do so, the first goal of this relatively new investigation of aging in ASD will be to increase our understanding age-related changes in ASD. Only then will clinicians and researchers be prepared to address ways in which to improve the lives and encourage more successful aging in individuals living with this lifelong disorder.

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Tables

Table 1. Demographics

	ASD (n = 29)		Typical (n = 30)		p
	Mean (SD)	Range	Mean (SD)	Range	
Chronological Age (years)	49.0 (11.7)	30-67	48.7 (12.1)	30-65	.94
Gender*	Male = 24 Female = 5		Male = 23 Female = 7		.42
WASI					
Vocabulary (t-score)	58.1 (7.8)	37-73	56.1 (7.1)	35-67	.57
Matrix Reasoning (t-score)	57.1 (7.0)	39-65	58.9 (5.9)	47-72	.29
2-subscale FSIQ	113.2 (9.5)	92-128	113.1 (10.2)	93-130	.95
SRS-2	67.2 (8.9)	52-92	48.1 (6.7)	39-65	<.001
ADOS-2 Module 4					
Communication	3.1 (0.9)	2 – 6	--	--	
Reciprocal Social Interaction	6.3 (1.9)	3 – 12	--	--	
Combined Total	9.9 (2.7)	5 – 18	--	--	

* X^2 = .34, p = .56

Table 2. *Measures of Effortful Processing*

Measure	Domain	Dependent Variable	ASD	Typical	*p*-value	Cohen's *d*
MoCA	MCI	Total Score	25.9 (2.4)	27.1 (2.1)	0.05	0.53
Concept Formation	Category Learning		29.1 (7.6)	33.2 (5.2)	0.02	0.63
RAVLT	Free Recall (%)	Trial 1	36.8 (9.5)	42.7 (11.2)	0.04	0.57
		Trial 2	48.5 (13.8)	60.9 (14.7)	0.002	0.87
		Trial 3	62.1 (19.2)	69.3 (17.6)	0.14	0.39
		Total	49.1 (13.0)	57.6 (13.1)	0.04	0.65
	Recognition Memory	Accuracy (%)	91.9 (7.0)	95.1 (4.4)	0.04	0.55
		Semantic Foils	1.6 (1.5)	0.9 (0.9)	0.02	0.57
		Phonetic Foils	0.3 (0.6)	0.1 (0.4)	0.26	0.39
TMT	Processing Speed (secs.)	Visual Scan	24.1 (6.1)	18.1 (4.1)	<0.001	1.15
		Number Seq.	39.4 (12.4)	28.1 (8.6)	<0.001	1.06
		Letter Seq.	35.9 (13.4)	25.6 (8.3)	0.001	0.92
		Number-Letter Seq.	102.1 (54.4)	69.3 (25.9)	0.004	0.77
		Motor Speed	38.5 (22.6)	16.9 (9.5)	<0.001	1.25

Table 3. *Standardized Beta Coefficients and p-values from Regression Models with FSIQ-2, Age, Diagnosis, and Age x Diagnosis as Predictors*

	MoCA		RAVLT		WJ-CF	
	B	*p*	β	*p*	β	*p*
FSIQ	0.21	0.12	0.32	<0.01	0.44	<0.01
Age	-0.38	<0.01	-0.23	0.08	-0.33	<0.01
Diagnosis	-0.38	<0.01	-0.32	<0.01	-0.29	<0.01
Age x Diagnosis	-1.03	0.04	-0.25	0.65	1.02	0.03

Table 4. *Standardized Beta Coefficients and p-values from TMT subtests with FSIQ-2, Motor Speed, Age, Diagnosis, and Age x Diagnosis as Predictors*

	Visual Scanning		Number Seq.		Letter Seq.		Number-Letter Switching	
	β	*P*	β	*p*	β	*p*	β	*p*
FSIQ	-.15	0.27	-.09	0.49	-.27	0.05	-.35	<0.01
Motor Speed	.49	<0.01	.60	<0.01	0.60	<0.01	.32	<0.01
Age	.32	<0.01	.34	<0.01	0.30	<0.01	.50	<0.01
Diagnosis	.34	<0.01	.22	0.07	0.15	0.18	.29	0.02
Age x Diagnosis	.43	0.37	.03	0.94	-0.11	0.80	.76	0.08

Table 5. *Measures of Automatic Processing*

Measure	Domain	Dependent Variable	ASD	Typical	*p*-value	Cohen's *d*
Classical Conditioning	Associative Learning	SCR Difference Score (CS-NS)	.01 (.07)	.04 (.08)	0.10	0.47
Prototype Task	Category Learning	Prototype Effect (Prototype – New)	30% (26%)	40% (22%)	0.10	0.41
	Confidence Rating	Recognition of Prototype	74% (18%)	76% (14%)	0.68	0.11
		Recognition of New Stimuli	71% (25%)	63% (22%)	0.25	0.30

Figures

Figure 1. *Sample stimuli for the prototype task: eight familiarization stimuli and the prototype for the "MIP" animal family*.

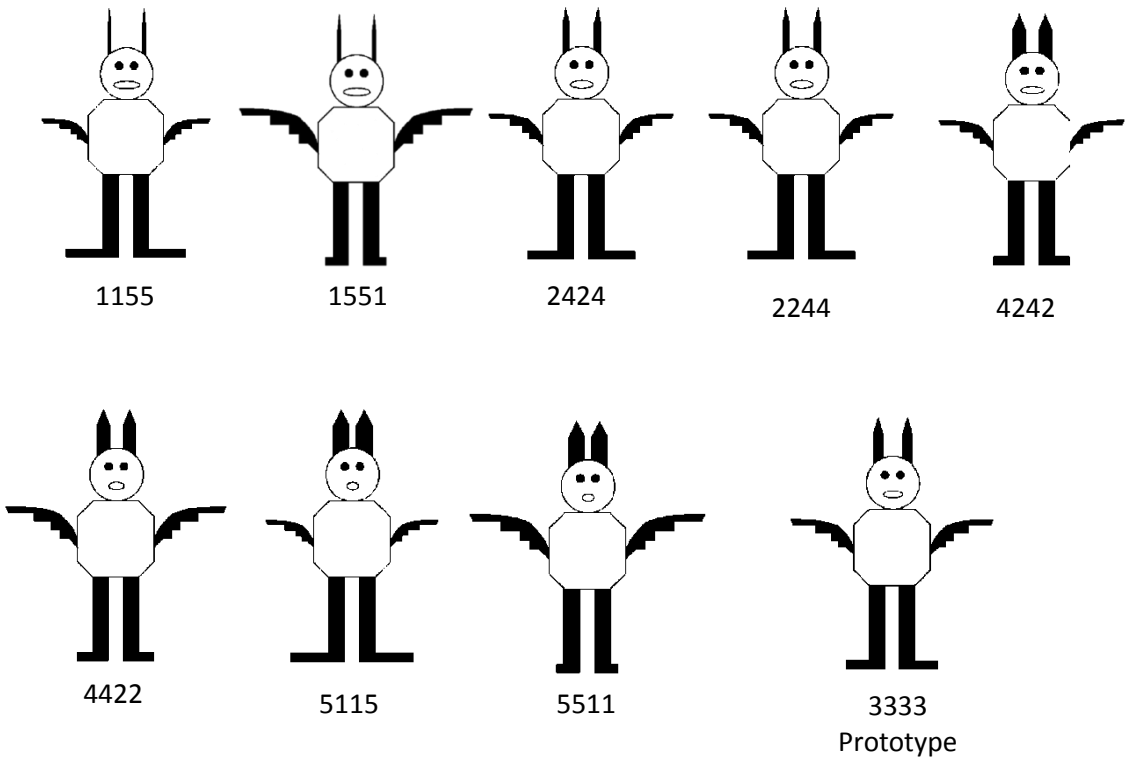
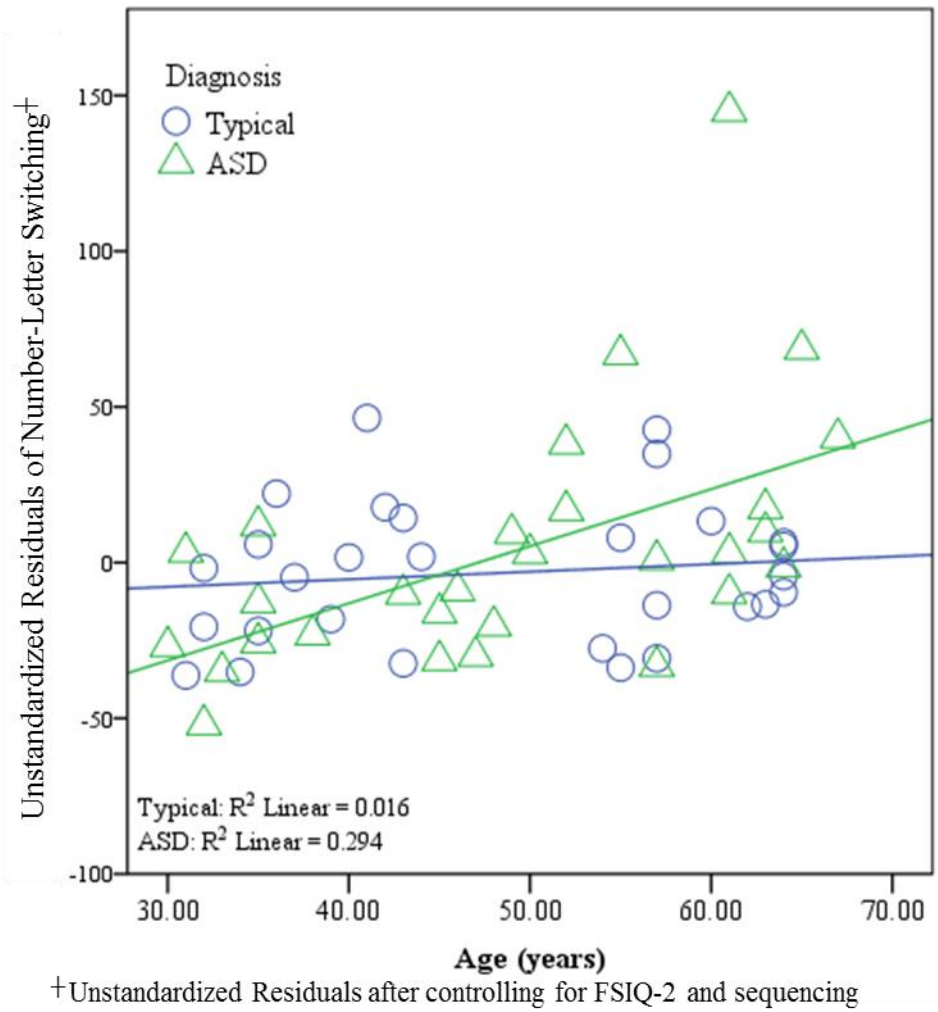


Figure 2. *Age-Related Differences in Cognitive Flexibility*



+Unstandardized Residuals after controlling for FSIQ-2 and sequencing

Figure 3. *Age-related comparisons of differential conditioning*

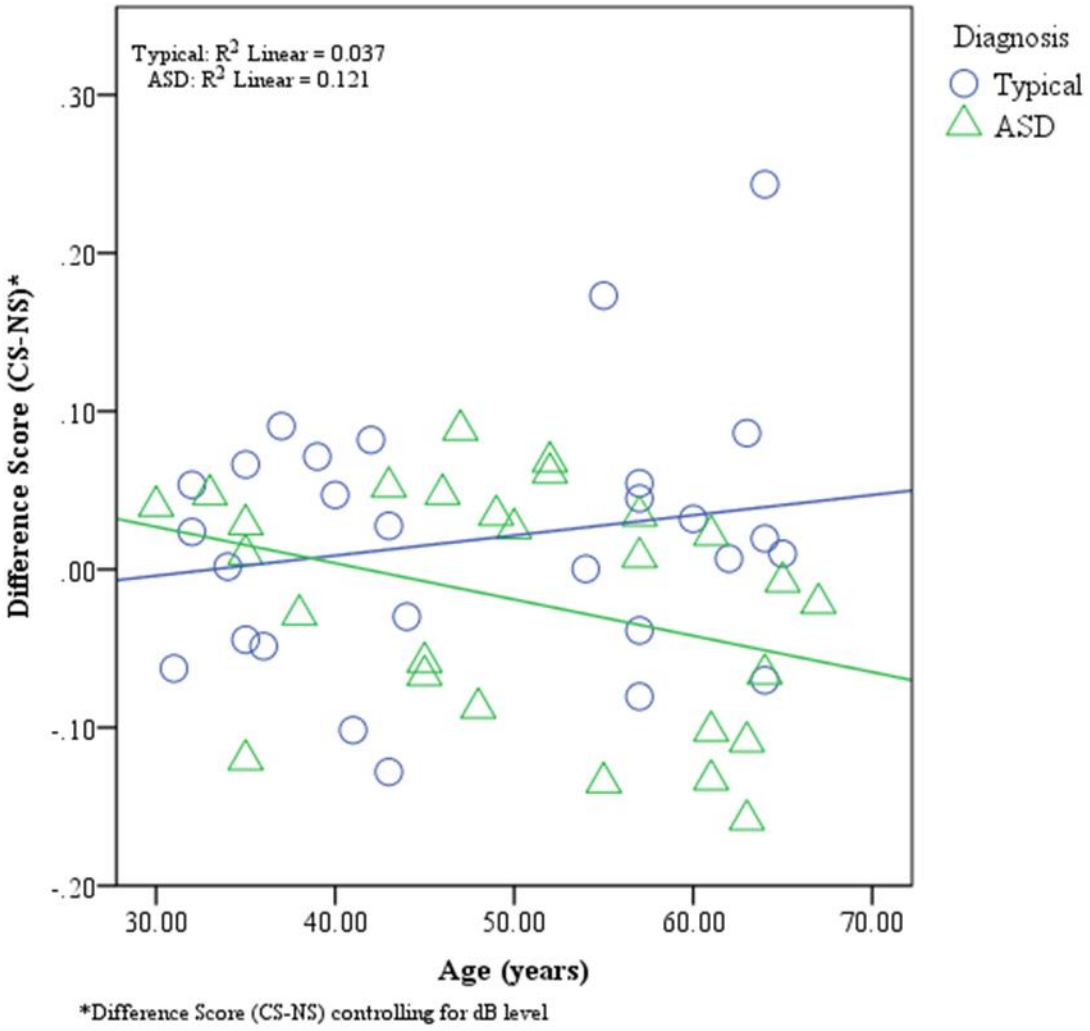


Figure 4. *Age-related Comparisons of Effortful Processing*

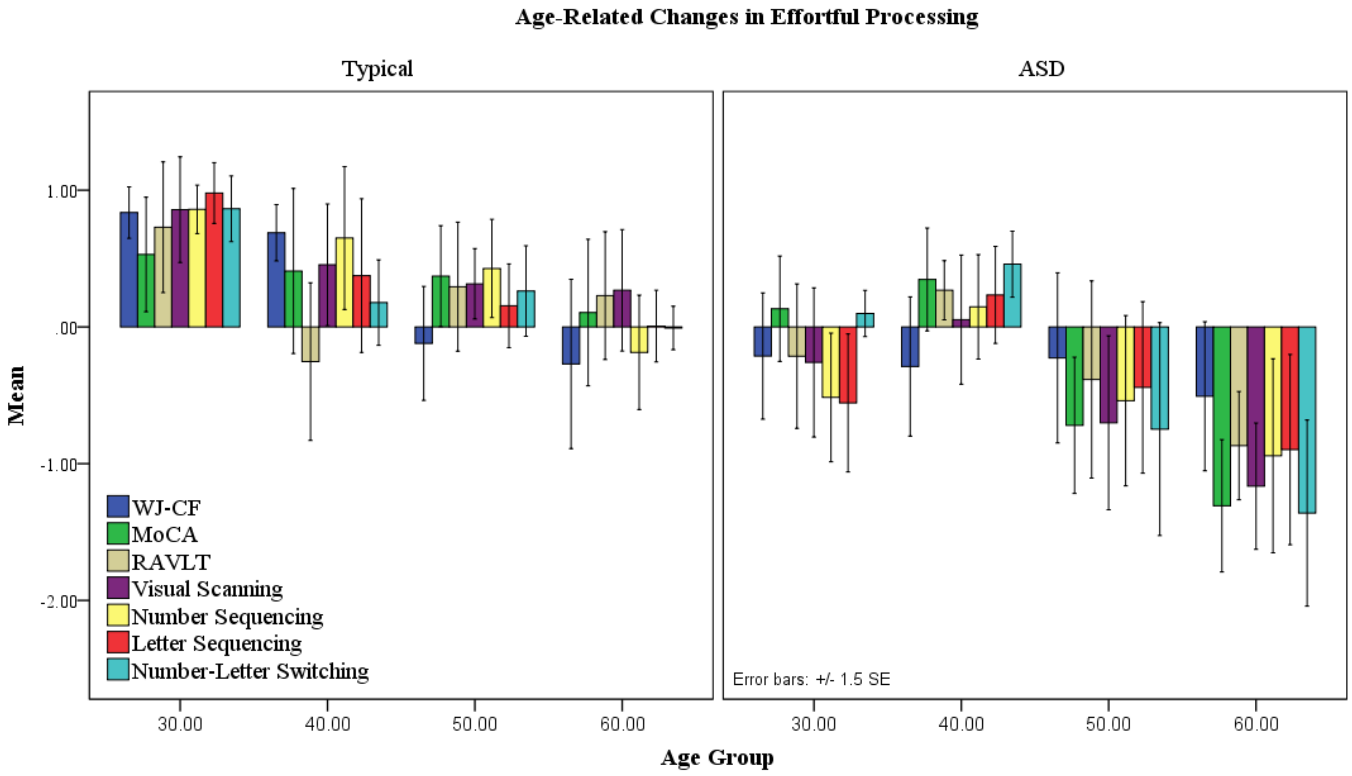


Figure 5. *Age as mediated by processing speed versus processing resources*

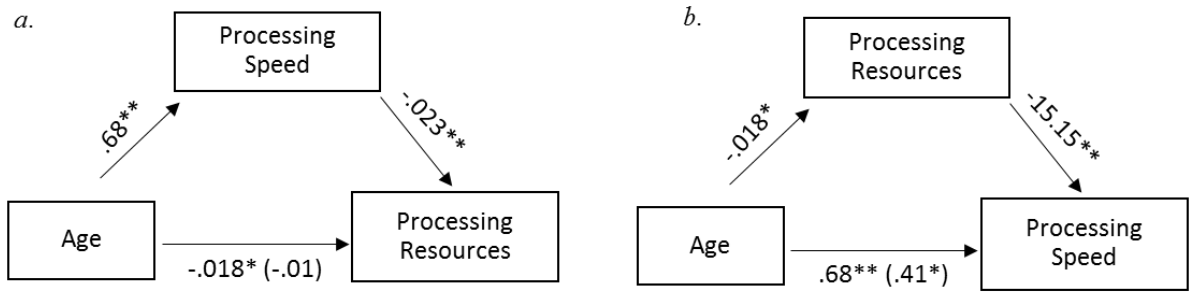


Figure 5a. Unstandardized regression coefficients for the relationship between age and processing resources (PR) as mediated by processing speed (PS). The unstandardized regression coefficients between age and PR, controlling for PS, is in parentheses. *Figure 5b*. Unstandardized regression coefficients for the relationship between age and processing speed (PS) as mediated by processing resources (PR). The unstandardized regression coefficients between age and PS, controlling for PR, is in parentheses.
*$p < .05$, ** $p < .01$