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**Ageing, Cognition and Quality of Life in Autism Spectrum
Disorder: Cross-Sectional and Longitudinal Studies**

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Thesis submitted in fulfilment of the requirements
for the degree of Doctor of Philosophy.

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Abstract

Few studies to date have included older autistic adults, meaning that lifespan outcomes for autistic adults are poorly understood. In this thesis, findings are presented from a four-year programme of research, which followed-up younger and older adults (aged 18-80 years) with a diagnosis of Autism Spectrum Disorder and comparison groups of younger and older typically ageing adults from the general population. The aim of this research was to understand how growing older affects the cognitive functioning, autistic traits, psychological well-being and quality of life of autistic adults. The work reported here extends the recent but sparse literature on autism and ageing and addresses some of the unanswered questions about the lifespan outcomes for autistic adults as they grow older. Is there a steeper risk of cognitive decline with ageing? Does ageing in autism mirror the trajectories of cognitive change seen in typical ageing? Does the cognitive profile of autistic adults stay the same as they grow older?

Previous literature has largely neglected the lifespan outcomes of autistic adults in older age, especially concerning questions on the persistence of mental health difficulties across the adult lifespan, and the degree to which cognitive differences and behavioural difficulties associated with autistic traits affects the quality of life of autistic adults as they grow older. The work presented here involved cross-sectional (Study 1) and longitudinal (Study 2) studies. Here, matched groups of older and younger adults with and without a diagnosis of autism spectrum disorder (ASD) were compared at two time points, T1 and T2, approximately 2.5 years apart. Measures were taken of autistic traits, cognitive functions (intellectual ability, language, memory and EF), mental health (anxiety, depression) and quality of life at both time points. The results showed that, at T1, younger autistic adults demonstrated the patterning of cognitive difficulties that resembled older typically ageing adults. Older autistic adults showed a different profile of age-related cognitive abilities, which may be explained by cognitive resilience developed across the lifespan. Nonetheless, co-existing physical and mental health conditions presented difficulties for more than half the autistic adults and were associated with poorer quality of life and well-being across the lifespan. An exploration of longitudinal change from T1 to T2 revealed no age-related changes in the cognitive profiles of

younger and older autistic adults. Nevertheless, mental health difficulties persisted, as did poor quality of life.

Then, to better understand the specific factors associated with cognitive functioning in older age, Study 3 extended the above work to Prospective Memory (remembering to remember) – a cognitive process that is among the most sensitive to age-related changes in typical ageing. For the first time, prospective memory was explored in ageing and autism, and its relation to quality of life. Ecologically relevant assessments of prospective memory were carried out in laboratory and naturalistic settings, in six experimental tasks. These tasks were designed to test pro-social and self-relevant motivations in event- and time-based aspects of prospective memory, which differentially draw on executive resources and are known to present difficulties for older typically ageing adults. Older autistic adults performed as well as or better than younger autistic adults across the six tasks, showing no age-related difficulties. By contrast, the older typically ageing adults showed age-related difficulties compared to younger typical adults. These findings replicate the previous literature of prospective memory in typical ageing and extend the limited research of PM in ASD to provide, for the first time, an explanation of the cognitive mechanisms associated with prospective memory and their relation to quality of life.

The programme of work just described directly addresses some of the key issues associated with ageing and ASD and identifies areas in which some autistic individuals may require supports across their lifespan.

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Declaration

A part of this programme of work, introduced in Chapter 2 of this thesis, was previously submitted for academic assessment in 2013. The dissertation: *Ageing and Psychological Functioning in Autism Spectrum Disorder* (Roestorf, A., September 2013) was awarded Master of Science in Research Methods and Psychology (Distinction), from City, University of London. This work is incorporated here for completeness, as a part of the 4-year programme of work resulting in this Doctoral Thesis. The work contained here is original research and writing produced for this thesis. I have exercised reasonable care to ensure that the work is original and does not, to the best of my knowledge, break any UK law or infringe any third party's copyright or other Intellectual Property Right. Where I have included third party copyright material, I have fully acknowledged its source.

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Publications and presentations of the work arising from this Thesis

Roestorf, A. & Bowler, D. M. (2016). Ageing and Psychological Functioning in Autism Spectrum Disorder. In Scott D. Wright (Ed.). *Autism Spectrum Disorder in Mid and Later Life*, (Part IV, Ch. 13, pp. 207-220). London: Jessica Kingsley Publishing.

Roestorf, A., Bowler, D. M., Deserno, M., Howlin, P., Klinger, L., McConachie, H., Parr, J., Powell, P., Van Heijst, B., & Geurts, H. M. (in press). Older Adults with Autism Spectrum Disorder: An International Perspective on Measurement. *Research in Autism Spectrum Disorders, Special Issue: Growing Older with Autism*.

Publications in preparation

Roestorf, A., Gaigg, S.B., Williams, D.M. & Bowler, D.M. (under review). *ASD traits and diagnostic convergence of self- and other-reports*.

Roestorf, A., Howlin, P., Povey, C. & Bowler, D.M. (in prep.). *Ageing on the Autism Spectrum: Mental health concerns across the adult lifespan*.

Roestorf, A., Howlin, P., Povey, C. & Bowler, D.M. (in prep.). *Ageing on the Autism Spectrum: Physical health concerns across the adult lifespan*.

Collaborative contributions

Autistica. (2018). *Global Summit on Autism throughout the Lifespan: Physical Health & Wellbeing*. A collaborative workshop to establish priority research areas to improve the physical health and wellbeing of autistic people, enabling them to live longer, happier, healthier lives. London: Autistica. <https://www.autistica.org.uk/downloads/files/Autistica-Research-Strategy-2017-2021.pdf>

Gaigg, S. B., Flaxman, P., Mclaven, G., Shah, R., Bowler, D. M., Meyer, B., **Roestorf, A.**, Haenschel, C., Rodgers, J. & South, M. (under review). *A pilot 8-month longitudinal randomized controlled trial of online mindfulness and cognitive behavioural self-help programs to alleviate anxiety in autistic adults*.

Oral presentations

Preliminary findings from studies included in this Thesis have been presented at the following conference proceedings:

Roestorf, A., Gaigg, S.B., Howlin, P., Povey, C. & Bowler, D.M. (May 2018). *Memory and Autism in the Context of Ageing*. Special Interest Group on Memory in ASD: Spared or Impaired? Insights from Behavioral and Neuroimaging Studies. Oral presentation at the International Society for Autism Research (INSAR), Rotterdam, Netherlands.

Roestorf, A., Gaigg, S.B., Williams, D.M. & Bowler, D.M. (May 2018). *Self-Reports Vs Observer Ratings of Autistic Traits: A Comparison of Their Validity over the Adult Lifespan*. Oral presentation at the International Society for Autism Research (INSAR), Rotterdam, Netherlands.

Roestorf, A., Howlin, P., Povey, C. & Bowler, D.M. (May 2018). *Ageing with Autism: A Later Lifespan Approach to Cognition, Mental Health and Quality of Life*. Oral presentation at the International Society for Autism Research (INSAR), Rotterdam, Netherlands.

Roestorf, A., Gaigg, S.B., Povey, C., Howlin, P. & Bowler, D.M. (January 2018). *Prospective memory in older adults with Autism Spectrum Disorder*. Oral presentation at the 5th International Conference on Prospective Memory (ICPM5). Australian Catholic University, Melbourne, Australia.

Roestorf, A. (November 2017). *The Next Big Step: what the future holds: Ageing on the Autism Spectrum*. Oral presentation at the Autistica Neurodiversity event series. Deutsche Bank, Birmingham, UK.
Video: <https://www.youtube.com/watch?v=fViW2HIT5mM>

Roestorf, A. (July 2017). *The Ageing with Autism Project: Longitudinal Research with Older Autistic Adults*. Oral presentation at the Symposium on Ageing and Autism Spectrum Disorder: Life course changes, well-being and service needs in later life, City, University of London.
Press: <https://www.city.ac.uk/news/2017/september/ageing-and-autism>

Roestorf, A., Gaigg, S.B., Howlin, P., Povey, C. & Bowler, D.M. (May 2017). *Prospective memory and quality of life in older adults with autism*. Measuring and predicting quality of life in older adults with autism. Oral presentation Panel session at the International Meeting for Autism Research (IMFAR), San Francisco, California, USA.
Press: <https://www.forbes.com/sites/emilywillingham/2017/05/18/half-of-autistic-adults-feel-a-low-sense-of-well-being/#16cd9e8f7bd1>

Gaigg, S.B., Shah, R., McLaven, G., Flaxman, P., Bowler, D. M., Meyer, B., **Roestorf, A.**, Haenschel, C., Rodgers, J., & South, M. (May 2017). *A Randomized Controlled Trial Comparing Online Mindfulness and CBT Programs to Alleviate Anxiety in Adults with ASD*. Poster Presentation at International Meeting for Autism Research (IMFAR), San Francisco, California, USA.

Roestorf, A., Howlin, P., Povey, C. & Bowler, D.M. (September 2016). *Ageing with Autism: Memory and quality of life in adults with autism spectrum disorder*. Oral presentation at the XI International Congress Autism Europe, Edinburgh, UK.

Video: https://drive.google.com/file/d/0B3_BfYlmRcKoc1BSR3E5UIFEdVU/view?usp=drivesdk

Press: www.city.ac.uk/news/2016/october/raising-awareness-of-mental-health-and-wellbeing-research-at-city

Roestorf, A., Howlin, P., Povey, C. & Bowler, D.M. (May 2016). *Memory and cognitive changes in later life in adults with autism spectrum disorder*. Growing older with autism: cognition, comorbidity and quality of life. Oral presentation Panel session at the International Meeting for Autism Research (IMFAR), Baltimore, Maryland, USA.

Press: <https://www.city.ac.uk/news/2016/april/autism-awareness-week>

Roestorf, A., Howlin, P. & Bowler, D.M. (May 2015). *Ageing and quality of life in autism spectrum disorder*. Oral presentation at the International Meeting for Autism Research (IMFAR), Salt Lake City, Utah, USA.

Press: <https://www.nrc.nl/nieuws/2017/01/27/nukopzorgenstrakskoppositie6425971a1543323>

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Abbreviations

ADOS-2	Autism Diagnostic Observation Schedule – Second Edition
AQ	Autism spectrum Quotient
ASD	Autism Spectrum Disorder
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory
CANTAB®	Cambridge Neuropsychological Test Battery
CFQ	Cognitive Failures Questionnaire
CREVT-3	Comprehensive Receptive and Expressive Vocabulary Test–Third Edition
CVLT-II	California Verbal Learning Task–Second Edition UK
DMS	Delayed Match to Sample task (CANTAB®)
DSM-5	Diagnostic and Statistical Manual – Fifth Edition
DSM-IV-TR	Diagnostic and Statistical Manual – Fourth Edition, Text Revised
DSM-III	Diagnostic and Statistical Manual – Third Edition
EBPM	Event-based Prospective Memory
EF	Executive Function(s)(ing)
FSIQ	Full Scale Intelligence Quotient (WAIS-R; WAIS-III; WAIS-IV)
FT	Future Thinking
ID	Intellectual disability
IQ	Intelligence Quotient
Lab-EBPM	Laboratory Event-based Prospective Memory task
Lab-TBPM	Laboratory Time-based Prospective Memory task
Nat-EBPM-OR	Naturalistic Event-based Prospective Memory ‘other-relevant’ task
Nat-EBPM-SR	Naturalistic Event-based Prospective Memory ‘self-relevant’ task
Nat-TBPM-OR	Naturalistic Time-based Prospective Memory ‘other-relevant’ task
Nat-TBPM-SR	Naturalistic Time-based Prospective Memory ‘self-relevant’ task
PAL	Paired Associates Learning task (CANTAB®)
PHQ	Patient Health Questionnaire
PIAS	Passport to Individual Autism Support (National Autistic Society)
PIQ	Performance Intelligence Quotient (WAIS-R; WAIS-III)
PM	Prospective Memory
POI	Perceptual Operating Index (WAIS-III)
PRI	Perceptual Reasoning Index (WAIS-IV)
PRMQ	Prospective Retrospective Memory Questionnaire
PSI	Processing Speed Index (WAIS-III; WAIS-IV)
PWI	Personal Wellbeing Index

QoL	Quality of life, as defined by World Health Organisation
RM	Retrospective Memory
RRB	Rigid and Repetitive Behaviours, including stereotyped interests (SRS-2; ADOS-2)
RTI	Reaction Time task (CANTAB®)
RVP	Rapid Visual Processing task (CANTAB®)
SCI	Social Communication Index (SRS-2)
SOC	Stockings of Cambridge task (CANTAB®)
SRS-2	Social Responsiveness Scale–Second Edition, Adult self-report version
SWB	Subjective Well-being. In the present research the term ‘well-being’ will be used as encompassing self-reported personal well-being.
SWM	Spatial Working Memory task (CANTAB®)
TA	Typical(ly) ageing; individuals in the general population with no associated neuropsychological disorders
TAS-20	Toronto Alexithymia Scale – 20 items
TBPM	Time-based Prospective Memory
TD	Typically developed(ing); children and adolescents in the general population who follow developmental trajectories with no associated neuropsychological disorders
ToM	Theory of Mind
VCI	Verbal Comprehension Index (WAIS-III; WAIS-IV)
VIQ	Verbal Intelligence Quotient (WAIS-R; WAIS-III)
WAIS-III	Wechsler Adult Intelligence Scales–Third Edition
WAIS-IV	Wechsler Adult Intelligence Scales – Fourth Edition
WCC	Weak Central Coherence
WMI	Working Memory Index (WAIS-III; WAIS-IV)
ZTPI	Zimbardo Time Perception Index

Chapter 1: Thesis Overview

The programme of work detailed in this thesis documents a collection of studies that set out to understand how the process of growing older affects individuals with a diagnosis of autism spectrum disorder (ASD) in terms of their autistic traits, cognitive functions, psychological well-being and quality of life (QoL). The primary aims of the research were to address the gap in knowledge of ageing and ASD which has been raised in recent literature identifying the key issues for this clinical population. Is there a risk of steeper cognitive decline with ageing? Does ageing with autism mirror ageing trajectories of cognitive change seen in typical development? Does the cognitive profile of autistic adults stay the same as they grow older? These issues are set out in Chapter 2, in a review of the literature on childhood developmental trajectories into adulthood and the core cognitive and functional differences of autistic individuals in relation to typically ageing individuals, culminating in the programme aims and overarching research questions for the 4-year programme of work that forms the framework for this thesis. Then, Chapter 3 identifies the conceptual issues related to assessing broad functioning in autistic individuals, followed by the selection of the measures that serve to achieve such assessment, and which are used in Studies 1 and 2 of this thesis. Accordingly, a series of inter-related studies were carried out to address the effects of ageing on cognitive change and QoL over time. To achieve the research aims, cross-sectional comparisons between younger and older autistic adults (ASD groups) and comparison groups of typically ageing adults (TA groups) were carried out at two time-points: assessment at Time 1 (T1; Study 1 in Chapter 4) established the baseline cognitive profile for the groups; these were then followed up after approximately 2.5 years and reassessed at Time 2 (T2; Study 2 in Chapter 5), the basis of the longitudinal analysis of change.

In Chapter 4, the T1 assessment involved exploratory information gathering about age-related *differences* in the cognitive profiles of younger and older ASD and TA adults. Cross-sectional data are reported on Age Group and Diagnostic Group comparisons for T1 assessments. Participants were assessed for a breadth of functions including general intellectual ability, language, memory and executive function, as well as the degree of everyday social and behavioural difficulties associated with autistic traits and mental health conditions, and the effects of these abilities and difficulties on

QoL. The results suggest a different profile of cognitive ageing in ASD. Here, older ASD adults displayed a profile of cognitive strengths that matched the performance of younger ASD adults and, in some assessments, outperformed older and younger TA adults. By contrast, the performance of older TA adults showed age-related cognitive limitations, such as in tasks of memory and EF, mirroring existing findings in the typical ageing literature. Nonetheless, ASD groups reported greater mental health difficulties and poorer QoL, across Age Groups, compared to TA adults.

This work is revisited in Chapter 5, which reports the T2 results from longitudinal follow-up where participants were re-assessed on the same cognitive, mental health and QoL domains measured at T1. A comprehensive summary of the cross-sectional Age Group and Diagnostic Group comparisons are reported for T2 assessments, followed by the longitudinal contrasts between T1 and T2 performance to paint a picture of how age-related *changes* affect younger and older ASD adults compared to age-matched TA adults. Whereas, no age-related differences were observed in the older ASD compared to younger ASD adults at T2, the older TA adults showed age-related difficulties that concurred with the findings from the T1 assessment. Further, for ASD adults no changes were observed in the degree of autistic traits, whereas, mental health difficulties and poor QoL persisted in comparison to TA adults. Once again, this profile of cognitive function points to a different patterning of ageing in ASD compared to typical ageing. Moreover, the persistence of specific mental health difficulties is an important finding that needs to be considered in the long-term well-being of autistic adults, given the high rate of co-existing physical and mental health concerns in ASD and which may further affect cognitive functioning, daily living and QoL outcomes across the lifespan (Wallace et al., 2016; Croen et al., 2017).

Because prospective memory (PM) is a cognitive function that is among the most sensitive to age-related changes, and one of the most important predictors of outcome in typical ageing, Chapters 6 and 7 investigate PM in ageing and ASD. PM is crucial to autonomous living and maintaining a sense of identity in older age, and it is one of the strongest predictors of QoL in older, typically ageing adults (Woods et al., 2012). Research findings from the typical ageing literature suggests that older adults have greater PM difficulties under increased EF demands, a domain that is known to present selective difficulties for autistic individuals. A small number of studies have explored PM in autistic

children and young adults, although the findings are not equivocal. The emerging picture is that PM presents difficulties in autistic individuals when task demands are increased, as in self-initiated time-based PM (remembering to execute an action at a specified future time), but not when PM is supported through event-based cues (remembering that is triggered by an external stimulus). Study 3, presented in Chapter 7 includes a series of six tasks that assess the event-based and time-based aspects of PM, and the role of pro-social motivation and self-interest in PM, across laboratory and naturalistic contexts. If autistic individuals have underlying difficulties in social reciprocity, then pro-socially motivated PM tasks should present greater challenges for autistic adults than PM tasks that represent self-interest. The findings converged in a similar patterning across the six tasks.

Observations of older TA adults in the laboratory tasks replicated previous literature in a profile of age-related difficulties, across time-based and event-based tasks, compared to younger TA adults. By contrast, there were no age-related losses in PM function in the older ASD adults, who performed as well as or better than younger ASD adults, across all tasks. What is more, in the naturalistic tasks younger ASD adults showed greater difficulties with PM function, making more forgetting errors (omissions) or inappropriate responses (commissions) than older ASD and younger TA adults.

Further analysis revealed Diagnostic Group differences in the underlying cognitive mechanisms involving EF processes used by ASD and TA adults to successfully complete the PM tasks. In relation to QoL, overall PM ability (as measured by accuracy in the respective tasks) was a strong predictor of overall QoL in the TA groups, but not in ASD. The one exception to this trend was that better performance by ASD adults in the naturalistic tasks predicted better health-related QoL. These findings show that PM ability in ASD follows a different age-related patterning, drawing on different cognitive mechanisms, and has different association with QoL compared to typical ageing.

Each Study Chapter (4, 5 and 7) begins with a review of the literature appropriate to the empirical work reported in that chapter. A brief discussion of the study findings is presented at the end of the respective Study Chapters. Then, in Chapter 8, the General Discussion draws together emerging themes from each of the studies and how they contribute to a better understanding of ageing in ASD. Possible explanations are presented for the patterning of findings observed throughout this work, together with future considerations and implications for research and practical applications. The

programme of work presented here extends the sparse literature thus far on ageing and autism, making substantial contributions to our understanding of the abilities and challenges for autistic adults across the adult lifespan.

1.1 Terminology

The diagnostic term autism spectrum disorder (ASD) follows the DSM-5 guidelines (American Psychiatric Association (APA), 2015) and includes autism, autism spectrum and Asperger's diagnoses that were previously classified under DSM-III and DSM-IV-TR guidelines. The term 'autism' can be used both as a general term for what we think of as the essential core of ASD, or it can be a clinical sign or feature of individuals with ASD (as in 'autistic aloneness' or showing signs of autism – i.e. withdrawal from or reluctance to engage in two-way social interaction). For clarity of expression, the terms ASD and 'autism' are used in this thesis to denote the above descriptions.

When referring to individuals who are ASD-diagnosed, 'identity-first' language (i.e. autistic individual; autistic adult etc.) is commonly the preferred terminology, as opposed to 'person-first' language (i.e. person with autism) which has often been used in previous literature (Kenny, Hattersley, Molins et al., 2016). Accordingly, identity-first terminology will be applied when describing the research presented in this thesis, out of respect for the autistic adults involved in this work. Any contrary references are made in relation to the preceding literature, in respect of the authors of those works.

Chapter 2: General Introduction and Literature Review

2.1 Opening remarks

Why is it important to consider ageing and Autism Spectrum Disorder?

Autism Spectrum Disorder (ASD) is a set of lifelong, pervasive neurodevelopmental conditions that affect the cognitive, behavioural and social functions of the diagnosed individual, across a spectrum of abilities and difficulties (Geschwind, 2009). ASD is characterised by specific difficulties in social interaction and communication, a preference for restricted interests and stereotyped behaviours (Elsabbagh, Divan, Koh, et al., 2012; DSM-5, APA, 2013). In turn, each of these features of autism affects the individual's everyday functioning, ability to maintain independence and social relationships, mental health and QoL (Howlin, 1997; Seltzer, Shattuck, Abbeduto & Greenberg, 2004; Shattuck, Orsmond, Wagner et al., 2011; Geurts & Visser, 2012; Howlin et al., 2014; Lever & Geurts, 2016a; Lever & Geurts, 2016b; Fortuna et al., 2016).

It is now recognised that the prevalence of ASD in the general population is more than 1% (National Autistic Society, 2012; Brugha, Cooper, McManus et al., 2012; Brugha, Spiers, Bankart et al., 2016), although global estimates vary by country (Elsabbagh et al., 2012). Autistic adults constitute the main proportion of diagnosed individuals (Charlton, 2017) and, whilst it is estimated that in the UK there are currently 500,000 autistic adults (Autistica, 2016), the prevalence rates for adulthood ASD are thought to be “underestimated, under-reported and underdiagnosed due to generational effects, evolving diagnostic criteria and developmental changes across the life course” (Wright, Brooks, D'Astous & Grandin, 2013, p. 34; and see van Niekirk et al., 2011; Wright, S.D, Wright, C.A., D'Astous & Maida Wadsworth, 2016; Brugha et al., 2012; Mukaetova-Ladinska, Perry, Baron & Povey, 2012).

The average life expectancy of an autistic adult is estimated to be beyond middle-age into later life, although a few years shorter than a typically ageing adult in the general population (Shavelle & Strauss, 1998) and may be further reduced for autistic women (Shavelle, Strauss & Pickett, 2001), and for individuals with intellectual disabilities (Shavelle et al., 2001; and see Heslop & Glover, 2015) and multiple co-existing complex health conditions (Fombonne, 2003; Mouridsen, Bronnum-Hansen,

Rich & Isager, 2008; Bilder et al., 2013; Hirvikoski, Mittendorfer-Rutz, Borman et al., 2016).

However, the longevity of autistic adults at age 65 is estimated to be on par with typically ageing adults. In the coming twenty years, there will be an estimated 700,000 autistic adults who are aged 65 years or older (Piven & Rabins, 2011; Kats et al., 2013) and it is, therefore, crucial to understand the functional changes and support needs for this clinical population as they grow older (Happé & Charlton, 2011; Perkins & Berkman, 2012; Howlin & Moss, 2012; Charlton, 2017).

The first cohort of diagnosed cases of ASD have already reached middle to older age (Howlin & Udwin, 2002; Donovan & Zucker, 2010; Donovan & Zucker, 2016), yet developmental studies have largely disregarded the trajectories and outcomes of autistic people beyond childhood (Howlin, 1997; Howlin & Moss, 2012; Charlton, 2017). To date, the few studies of autistic adults are limited to younger samples with narrow age ranges from 18-55 years of age and mean sample ages of approximately 30-35 years (e.g. Billstedt, Carina Gillberg & Gillberg, 2007; Esbenson, Bishop, Mailick Seltzer, Greenberg & Lounds Taylor, 2010; Howlin et al., 2015; Happé et al., 2016; Hong, Bishop-Fitzpatrick, Smith, Greenberg, & Mailick, 2016; Jones et al., 2018; and see Jang, Matson, Adams et al., 2014); very few studies have included adults over the age of 65 at all, even in cross-sectional comparisons of older autistic and healthy ageing adults (Geurts & Jansen, 2012; van Heijst & Geurts, 2014; Fortuna et al., 2016; Lever & Geurts, 2016a; Lever & Geurts, 2016b; Klinger, M.R. et al., 2017; Powell, Klinger, L.G. & Klinger, M.R., 2017). Of these studies, samples tend to include clinical groups who are identified to services, whilst very few investigations have extended research to community-based samples to understand the broader impact of ageing and ASD across the spectrum (Shattuck, Seltzer, Greenberg et al., 2007; and see Happé, Mansour, Barrett et al., 2016; and see Wise, Smith & Rabins, 2017). Consequently, still very little is understood about the needs, lifespan trajectories and outcomes of older autistic adults, in terms of cognitive functions, autistic traits, physical health, psychological well-being and QoL (Happé & Charlton, 2011; Howlin & Moss, 2012; Perkins & Berkman, 2012; Mukaetova-Ladinska et al., 2012; Volkmar, Reichow & McPartland, 2014; Michael, 2016; Wright et al., 2016). Moreover, long-term mental health difficulties, such as depression, are known to correspond to difficulties in memory, attention, EF and processing speed (McClintock, Husain, Greer, & Cullum, 2010), and is associated with increased risk of neurocognitive

disorders (i.e. dementia; Bauman, 2010) in the general population (Evans & Mottram, 2000).

Furthermore, depression is associated with an increase in autistic traits in older age (Geurts, Stek & Comijs, 2016), raising concerns about the extent to which depression and other co-existing conditions may increase the risk of later life cognitive difficulties for older autistic individuals (Hategan, Bourgeois & Goldberg, 2017). The absence of empirical research on ageing and autism gives rise to important questions about the lifespan outcomes for autistic individuals.

- Is there a steeper risk of cognitive decline with ageing?
- Does ageing with autism mirror the trajectories of cognitive change seen in typical ageing?
- Does the cognitive profile of autistic adults stay the same as they grow older?

To answer these questions, we first need to understand how ASD is conceptualised and how past classifications have informed theoretical attempts to explain the social and non-social features of autism. A summary of the diagnostic classifications of ASD, the patterning of cognitive differences associated with core autistic traits, and the theories arising from observations of these factors are discussed next.

Conceptualisation and classification of Autism Spectrum Disorder

The characteristic features of ASD emerged from Kanner's first classification of childhood autism (Kanner, 1943; 1971), in which he observed pathognomonic¹ behavioural characteristics in a group of children aged between 2-8 years:

- inborn “disturbances of affective contact”, resulting in inherent difficulties with reciprocal social interaction and difficulties in aspects of social communication;
- “an inability from the beginning of life to relate themselves to other people and situations in the ordinary way”, manifesting as an innate preference for aloneness;
- “an anxiously obsessive desire for the presentation of sameness”, displayed as stereotyped behaviours and a strong need for sameness and routine (Kanner, 1971, p. 140).

¹ pathognomonic is the term used to define the observations of clinically relevant features or characteristics of a condition or disorder.

Similar observations were made by Asperger (1944), whose findings became known some years later (see Frith, 1991). Kanner (1971) confirmed this patterning of autistic characteristics in one of the first longitudinal follow-up studies of children diagnosed with autism, after 28 years. The outcomes for most of the autistic individuals diagnosed in Kanner's first cases were of intellectual disability and a lifetime of institutional care, whilst few individuals received support to develop semi-independence and community integration (Kanner, 1971). Together, these clinical classifications formed the foundation of how autism was diagnosed over the decades that followed, evolving from Kanner's and Asperger's first observations, to a diagnostic model that largely focused on the early onset and clinical diagnosis of childhood autism (Wing & Gould, 1979; Volkmar et al., 1992; Brugha, McManus, Bankart, Scott, Purdon, Smith, Bebbington, Jenkins & Meltzer, 2011). Consequently, autism-related support services, treatments, interventions and research have largely focused on childhood development into adolescence (Mukaetova-Ladinska et al., 2012; Pellicano, Dinsmore & Charman, 2014; Fletcher-Watson et al., 2014), with little application and reliability in adulthood outcomes (Brugha et al, 2015).

The evolution of diagnostic criteria over the past 40 years has adopted a different conceptualisation of the core features of autism (Happé & Charlton, 2011; Hansen, Schendel & Parner, 2015; Charlton, 2017), which now embody a spectrum of developmental stages and ages, across the lifespan. The current clinical classification of ASD, outlined in the Diagnostic and Statistics Manual – Fifth Edition (DSM 5, American Psychiatric Association. 2013), now encompasses a broader range of cognitive and behavioural difficulties and strengths (Mazurek, Lu, Macklin & Handen, 2018), which has led to more adults self-identifying or being identified through auxiliary clinical settings for a diagnosis of autism spectrum disorder (Happé, Mansour et al., 2016).

The two core domains of difficulties central to a diagnosis of ASD are:

- reciprocal social and communication impairments;
- restricted interests and repetitive behaviours.

These domains have been broadly examined in previous literature which has highlighted a wide range of abilities and difficulties associated with ASD and the implications for developmental transitions beyond childhood and adolescence into young adulthood.

Wide range of functioning

Autism has historically been associated with “profound and devastating effects” on intellectual ability, cognitive functions, and QoL of diagnosed individuals (Wing & Gould, 1979). There is considerable variability between individuals on the autism spectrum – functional and behavioural abilities and cognitive functions are affected by intellectual disability (ID; Kats et al., 2013; Ratto & Mesibov, 2015), the degree of autistic traits (Mazurek et al., 2018) and co-existing physical and mental health conditions (Seltzer et al., 2004; and see Levy & Perry, 2011; Kats et al., 2013). Whereas, core symptoms of autism remain stable throughout childhood and adolescence (Billstedt, Gillberg & Gillberg, 2005; Seltzer, Krauss, Shattuck et al., 2003), some studies report an improvement in the severity of restricted and repetitive behaviours (RRBs) between adolescence and adulthood (Lounds et al. 2007; Howlin et al., 2014). Further, maladaptive behaviours associated with autism, such as aggression, withdrawal, self-injury and destruction of property, have also been shown to improve across developmental stages (Shattuck et al., 2007; Taylor & Seltzer, 2010; Howlin et al., 2013). The clinical characteristics of ASD can be classified as social and non-social features that are associated with an uneven cognitive profile (Minshew, Goldstein & Seigel, 1997; Minshew and Goldstein 2001; Williams, Goldstein & Minshew, 2006; de Schipper et al., 2016), which is reflected in the patterning of cognitive difficulties across a range of domains and ‘islets of ability’ (Shah & Frith, 1983, p. 1351; Happé, 1999, p. 216; but see Dawson, Soulières, Gernsbacher & Mottron, 2007).

The characteristics just mentioned also seem to be highly variable within the individual across their developmental course (Happé, Ronald, & Plomin, 2006; Jones, Simonoff, Baird et al., 2018). For instance, autistic children and adolescents show discrepancies among receptive and expressive language skills and narrative comprehension (Williams et al., 2006; Howlin et al., 2014) and selective difficulties in domains of general intellectual functioning (Mottron, Dawson, Soulières, Hubert & Burack, 2006; Dawson, Soulières, Gernsbacher & Mottron, 2007; Bölte, Dziobek & Poustka, 2009), motor function and sensory processing (Williams et al., 2006). What is more, metarepresentational difficulties (Bowler et al., 2005) that are thought to underlie social-cognitive functions such as theory of mind and mental state understanding (Baron-Cohen et al., 1985; Frith, 1989; Frith & Happé, 1994;

Frith & Frith, 2003; Leekam, 2016), in turn affect communication skills and understanding. Further, difficulties with higher-order cognitive functions extend to poor global information processing (Shah & Frith, 1989; 1993; Happé, 1999) and selective difficulties in sub-domains of EF such as cognitive flexibility, working memory and planning (Russell, 1997; Hill, 2004; Ozonoff et al., 2004; Bramham et al., 2009; White, Burgess & Hill, 2009; Rosenthal et al., 2013). These cognitive processes may further be mediated by a disordered organisation and processing of social and non-social information (Williams et al., 2006), giving rise to the core features associated with ASD (Just et al., 2012; Lawson et al., 2015; Van Eylen et al., 2017). Memory is affected by limited capacity for self-generated episodic recall (Gaigg, 2015), poor autobiographical memory and episodic memory for past events or information (Crane, Pring, Jukes & Goddard, 2012; Crane, Goddard & Pring, 2014) and difficulties with future thinking (Crane, Lind & Bowler, 2013) and prospective memory for future actions (Altgassen et al., 2012; Williams et al., 2014; and see Landsiedel, Williams & Abbot-Smith, 2017). Prospective memory – *remembering to remember* – is remembering to act on the intention to do something (a thought or action) at a specified point in the future, without a specific instruction to do so (Einstein & McDaniel, 1990; Brandimonte, Einstein, & McDaniel, 1996). Successful completion of an intended prospective memory action requires the self-directed retrieval of episodic information and self-initiated execution of the action, thus drawing on competing cognitive resources (Graf & Utl, 2001). Often, prospective memory is socially motivated since memory slips or errors could lead to social embarrassment (Brandimonte et al., 1996; Baddeley, 1997; Altgassen, Kliegel, Brandimonte & Filippello, 2009; Kretschmer, Altgassen, Rendell & Bölte, 2014), which would present specific challenges for autistic individuals. Prospective memory is a crucial index of age-related cognitive decline in typical ageing (Craik, 1986; Blanco-Campal et al., 2009) and core to maintaining good QoL (Woods, 2015).

Several cognitive strengths have also been widely reported in relation to ASD. These strengths and enhance cognitive abilities include proficiency in expressive language (Williams et al., 2006; but see Howlin et al., 2013), strong attention to detail (Shah & Frith, 1989; Mottron, Dawson, Soulières, Hubert & Burack, 2006; Bölte, Duketis, Poustka & Holtmann, 2011) and excellent memory for details (Williams et al., 2006), including semantic information (Gaigg, Bowler & Gardiner, 2013; Crane,

Lind & Bowler, 2013; Crane & Goddard, 2008; but see Kristen, Rossman & Sodian, 2014; and see Lever & Geurts, 2016). However, these talents come at a cost of other cognitive functions (Happé et al., 2006; Happé & Booth, 2008). For instance, the predisposition to enhanced perception of detail (Happé, 1999; Happé & Booth, 2008) has implications for poor context generalisation (Plaisted, O’Riordan & Baron-Cohen, 1998) and impaired complex memory for information in context (Williams et al., 2006; Ring, Gaigg & Bowler, 2015). Further, expressive language may be overly formal or nuanced, whilst difficulties with receptive language extend to narrative comprehension and interpreting metaphors or figures of speech (Williams et al., 2006).

The variable patterning of cognitive strengths and difficulties observed in autistic individuals, has given rise to several theories that have attempted to explain the social and non-social features of ASD. The respective contributions of these theories, together with a brief discussion of their strengths and limitations, is set out next.

2.2 Theories of ASD: underlying mechanisms of cognition and behaviour

Theoretical approaches to understanding autism have developed from Kanner’s notion of ASD as an affective disorder (Kanner, 1943) to the view that ASD is underpinned by impairments in social information processing (Pelphrey & Carter, 2008), mental state understanding (Baron-Cohen et al., 1985; Frith & Happé, 1994), local and global information processing (Frith, 1989; Happé, 1999; Mottron & Belleville, 1993) and EF of higher order cognitive processes (Russell, 1997; Minshew, Goldstein & Seigel, 1997; Hill, 2004; Ozonoff et al., 2004). The approaches just mentioned posit either domain-specific atypicalities as central to the aetiology of autism, which focus on particular brain regions, functions or processes, or domain-general atypicalities that consider the broader neural networks and cognitive processes.

Domain-specific accounts that have primarily focused on the social functioning difficulties associated with autism have gained increasing attention over the last 20 years; these include the *social brain* model (Pelphrey & Carter, 2008) which has extended to a *social motivation* account of autism (Chevallier et al., 2012). These approaches draw on the premise that specific brain functions associated with processing social stimuli, and the subsequent development of associated social skills,

are atypically functioning in autistic individuals. These atypicalities include attenuated emotion recognition (see meta-analysis by Uljarevic & Hamilton, 2013; and see review by Harms, Martin & Wallace, 2010) and social reciprocity (Hobson et al., 2009). Additionally, the social brain model posits that social-cognitive difficulties are central to broader functional atypicalities in ASD. Such difficulties include, for instance, speech perception (Gervais et al., 2004), facial recognition (Hobson, Ouston & Lee, 1988; Weeks & Hobson, 1987; and see critical review by Weigelt, Koldewyn & Kanwisher, 2012), and difficulties in mental state understanding (Baron-Cohen et al., 1985; Happé, 1995; Frith & Happé, 1994; White, Hill, Happé & Frith, 2009). These difficulties also extend to the recognition and interpretation of intentions from eye-gaze (Pelphrey & Carter, 2008) and biological motion (Pelphrey et al., 2005; Hubert et al., 2007; Kaiser et al., 2010; and see Castelli, Frith, Happé & Frith, 2002), which are important functions that aid the interpretation of non-verbal social communication. Variations of the social brain model have included attempts to provide integrated explanations that draw connections between the cognitive and behavioural features of autistic difficulties. Recently, two pragmatic arguments have suggested that the patterning of autistic traits may be the consequence of “disrupted interplay between socio-emotional and cognitive mechanisms” (Gaigg, 2012, p. 24), or the result of abnormalities in the top-down regulation of perception (Pellicano & Burr, 2012, p. 5). According to the positions set out by Gaigg (2012; see Table 2.1, p. 37), and by Pellicano and Burr (2012), ASD is characterised by abnormalities in predictive coding processes that render the environment more unpredictable and ambiguous. On the one hand, this can lead to sensory processing abnormalities due to atypicalities in how sensory input is modulated by prior expectations (see Pellicano & Burr, 2012; and see retort by Friston, Lawson & Frith, 2012), and on the other hand it renders the social environment particularly confusing due to the complex and probabilistically determined nature of social interaction (Gaigg, 2012). These emerging hypotheses offer interesting and potentially fruitful approaches to understanding some behavioural aspects of ASD and bridging the gap between domain-specific accounts of social brain models that do not always fully account for the non-social difficulties. More work is needed to understand the neural mechanisms involved and the broader, domain-general implications of these accounts. Therefore, these concepts will not be discussed further in this thesis but are summarised in Table 2.1 (p. 37) for completeness.

The more well-established domain-general explanations of ASD include the Weak Central Coherence theory and its variants (Frith, 1989; Happé, 1999; Mottron et al., 1999, 2006; Bölte et al., 2011), and the Executive dysfunction theory of autism (Pennington & Ozonoff, 1996; Russell, 1997; Hill, 2004). These theories have attempted to explain the cognitive mechanisms that underpin the core non-social and social features of autism and possible brain regions that might be implicated. For instance, functional connectivity of underlying brain networks are thought to explain the patterning of cognitive and behavioural features of autism, as a result of disordered processing of complex information (Minshew, Goldstein & Siegel, 1997; Williams et al., 2006; Minshew & Williams, 2007; Just et al., 2012) or its organisation at lower- and higher-order levels of cognitive functioning (Mottron, Bellville & Ménard, 1999; Mottron & Burack, 2001; Lai, Lombardo & Baron-Cohen, 2014).

Overall, the theories of ASD that have been most extensively explored in the literature are: Executive (dys)function (EF; Hill, 2004), Weak Central Coherence (WCC; Frith, 1989; Happé, 1999) and Theory of Mind (ToM; Baron-Cohen et al., 1985; 2001; Happé et al., 1996; Frith & Frith, 2003). The contributions of each of these theories is discussed next to highlight the potential implications for the programme of work presented in this thesis.

Table 2.1. Social Theories of Autism Spectrum Disorder

Theory	Author(s)	Tenets	Evidence
Social brain	Pelphrey & Carter (2008)	The social brain model argues that specific brain regions are associated with processing of social stimuli in an advanced and interconnected neural network geared toward social processing, e.g. orbito-frontal cortex (reward processing; Bechara, Damasio, Damasio, & Anderson, 1994); amygdala (emotion; facial expressions; Adolphs, Tranel, Damasio, & Damasio, 1995), superior temporal sulcus (agency; mental state understanding; interpretation of non-verbal body language; Pelphrey & Morris, 2006); fusiform face area (faces and emotional expressions; Kanwisher, McDermott & Chun, 1997); temporal poles (social knowledge and scripts). Further, it is posited that this neural network does not function in the same way in ASD as in typical development. This dysfunction results in impaired social perception, and difficulties representing and predicting the actions and intentions of other people as social stimuli e.g. detection of biological-motion cues (Pelphrey et al., 2005) and face processing (Kanwisher, McDermott & Chun, 1997). Findings are suggestive of implications for Theory of Mind and mental state understanding, and domain-general representational ability.	Neuroimaging studies using fMRI, show activation in social brain regions e.g. posterior superior temporal sulcus (STS), when TD children attributed intentions to the eye gaze of others (Pelphrey et al., 2003) and when TD adolescents watched the actions and attributed intentions to the biological motion of others (Pelphrey et al., 2005). Whereas, autistic children (Blake et al., 2003) and adults (Cook et al., 2009) showed behavioural difficulties in these domains, and atypical functional activation in social brain regions (e.g. STS) which did not differentially respond to social and non-social stimuli (Carter & Pelphrey, 2006). The evidence suggests that dysfunction in the STS region and reduced connectivity with other social brain structures (e.g. fusiform gyrus (face processing), amygdala (emotion), underpin impaired social perception associated with autism (Pelphrey & Carter, 2008). Further evidence indicated hypoactivation of the STS in autistic individuals during tasks involving the attribution of intentions, e.g. biological motion (Kaiser et al., 2010), moving geometric figures (Castelli et al., 2002), and human speech perception (Samson et al., 2004; see review by Harms et al., 2010; and see Gaigg, 2012).
Social motivation	Chevallier et al., (2012)	Social motivation theory draws on the social brain model, positing that a disruption in connectivity of the social brain network in autistic individuals provides fewer opportunities to experience the reward of social engagement and, therefore, the autistic child does not develop the social mechanisms that facilitate social skills (Hobson et al., 1988; Dawson et al., 2004). This effect has been shown in autistic adults who show reduced sensitivity to reward contingencies in decision-making behavior (Damiano et al., 2012).	Much evidence comes from studies that have shown atypicalities in the way in which autistic individuals orient to and process social information (e.g. faces; Hobson, Ouston & Lee, 1988a; see Weigelt et al., 2012 for a comprehensive review). Eye-tracking studies (in addition to the biological motion studies mentioned above) have shown that autistic individuals are more likely to attend to non-social (e.g. background) than social features (e.g. eye-gaze as social cues) when visually presented with complex social scenes. Autistic children demonstrate difficulties in processing facial expressions (Weeks & Hobson, 1987); this finding has been replicated in a multitude of studies with adolescents and adults (see review by Harms et al., 2010), which also indicate configural processing difficulties and tendency to focus on features e.g. mouth (Bird et al., 2011). However, it should be noted that the position set out by Bird and colleagues is that abnormalities in this domain – and any emotion processing difficulties – are due to Alexithymia and not autism, per se (Cook et al., 2003).

Emotional learning	Gaigg (2012)	Early emerging abnormalities in how emotional salience modulates cognitive processes leads to wide-spread abnormalities in how emotion shapes the organisation of knowledge and behaviour. These abnormalities in emotional learning affect the development of self (Hobson, 2002), self-regulation (Shah, Catmur & Bird, 2016) and self-awareness (Garfinkel et al., 2015) and the development of context-appropriate behavioural responses. This has disproportionate consequences for the development of social cognition and interpersonal behaviour because of the central role emotions play in shaping early social skills (see Hobson, 2002; Trevarthen, 1979; and see Bierman et al., 2008). Critically, however, the development of non-social functions are also affected (e.g., memory, attention, perception etc.), partly as a consequence of the disturbances in social cognition, but also because the emotional salience of events doesn't modulate these cognitive processes typically.	The evidence for Gaigg's account stems primarily from studies which show that basic emotion-related learning processes (e.g. fear conditioning; South et al., 2011) are characterised by abnormalities in ASD. Several studies suggest that declarative memory (Beverdort et al., 1998; Gaigg & Bowler, 2008, 2009a; but see Maras et al., 2012; and see South et al., 2008), attention (Gaigg & Bowler, 2009b) and decision making (Johnson et al., 2006; Shah, Catmur & Bird, 2016) are also atypically modulated by the emotional significance of stimuli – even when those stimuli are not really 'social' in nature (i.e. faces, bodies etc.; Hobson, Ouston & Lee, 1988b). Further evidence suggests that interoception difficulties – specifically the disconnection between awareness and accurate predictions of arousal – may provide clues to reduced emotional processing and self-regulation and decision making in ASD (Garfinkel et al., 2015).

Executive function: concepts, typical and atypical processing

Executive function (EF) is the complex array of higher-order cognitive processes that serve to regulate and guide lower-order goal-directed behaviours (Miyake et al., 2000; Friedman & Miyake, 2017; Diamond, 2006; 2013). An integrative network of several core components makes up the processes of EF that regulate the ability to process new or changing information (updating; working memory (WM); Baddeley, 2000), flexibly switch strategies for competing environmental or task demands (cognitive flexibility; switching/shifting; Friedman et al., 2006), and self-regulate context-inappropriate responses under specific conditions (response inhibition / impulse control; Perner & Lang, 1999; Zelazo et al., 2003; Friedman & Miyake, 2017). Theoretical approaches to EF as a construct has evolved from Baddeley's (2000) description of a "central executive" that oversees functions such as WM and inhibition (Baddeley & Hitch, 1974; Hoffman, Schmeichel & Baddeley, 2012), to cognitive models of EF as a unified construct with common underlying mechanisms (Miyake et al., 2000; Friedman & Miyake, 2017; Diamond, 2006, 2013; Anderson & Craik, 2017) and variations of these models include positions on the unity and diversity of EF conceptualisations (see Table 2.2, p. 45; and see Zelazo et al., 2003; Bagetta & Alexander, 2016 for a more detailed discussion of the unity and diversity of EF conceptualisations). The core components of higher-order EF processes are regarded as distinct processes (Friedman & Miyake, 2017) that guide thought and action (Zelazo, Craik & Booth, 2004), although there is some overlap between the "spheres of influence" involved. For instance, switching between task demands involves cognitive flexibility, as well as requires updating (WM) and regulation of prepotent responses (inhibition) or distracting information (Mäntylä et al., 2010; Bagetta & Alexander, 2016). These are just some of the subcomponents or broader *executive functions*² that draw on higher-order cognitive processes as an

² The majority of literature uses the terms *executive function* and *executive functions* interchangeably. Accordingly, there is some inconsistency in differentiation between core EF components and subcomponents that regulation goal-directed behaviours and other cognitive-behavioural regulation. A fine distinction is provided by Bagetta and Alexander (2016), who suggest that Executive function (EF; as referred to in this thesis) is the "overall construct" that comprises *salient attributes* (i.e. higher-order cognitive processes) and *spheres of influence* (e.g. flexibly switch strategies for competing environmental or task demands (cognitive flexibility)), and may include "the individual components or processes" (i.e. executive functions) that contribute to EF as a higher-order process. By contrast, *executive functions* are the putative "combination of individual abilities, components, subcomponents or [cognitive] processes" – as many as 39 identified subcomponents – that support higher-order information processing to guide thought and actions (Bagetta & Alexander, 2016; Friedman & Miyake, 2017; Zelazo, Craik & Booth, 2004).

integrative part of EF (Bagetta & Alexander, 2016; Friedman & Miyake, 2017). Additional subcomponents of EF include: attention (McCabe et al., 2010; Anderson & Craik, 2017), the use of effective strategies to make decisions and solve problems particularly in novel contexts (e.g. planning and strategy; Diamond, 2006; Zelazo, 2006), the ability to generate creative and specific responses to a given situation (fluency), and emotional control (Zelazo et al., 2003). There is some evidence for the interrelatedness of EF components and other higher-order cognitive processes. For instance, inhibition draws on planning, attention and conflict monitoring for effective modulation of responses (Braver et al., 2001; Kana et al., 20016; Just et al., 2012), whilst speed of information processing (whether automatic or strategic) may aid memory retrieval (McDaniel & Einstein, 2000), updating and conflict monitoring (Salthouse, 1996; Park et al., 1996; Hoffman, Schmeichel & Baddeley, 2012). It has also been suggested that attention underpins EF processes, playing a pivotal role in WM (Baddeley, 1993, 1997), cognitive flexibility (Zelazo et al., 2004) and inhibition, as well as other subcomponents of EF involved in processing speed and episodic memory (Salthouse, 1996; Park et al., 1996; McCabe et al., 2010). Consequently, tasks that measure EF cannot by their nature be “process pure” (McCabe et al., 2010, p. 223; and see Jacoby, 1999), but nonetheless converge to measure a composite of EF capabilities (e.g. Miyake et al., 2000; Salthouse et al., 2003; McCabe et al., 2010).

It is generally accepted that the frontal brain regions play a key role in EF processes (Dempster, 1992; Braver et al., 2001; McCabe et al., 2010). Research has identified the different developmental stages at which specific components of EF come online, based on discoveries of the late maturation of frontal brain regions relative to other brain structures (Huttenlocher et al., 1990; Eigsti, 2011; Purves, 2008). The typical developmental trajectory for EF emerges with the concurrent development of prefrontal cortices across childhood and adolescence (Huttenlocher et al., 1990) and deterioration of these brain regions into older age (Raz, 2000; West, 1996). Accordingly, in early typical development younger children display more reactive processing and bottom-up behavioural responses to their environment. The maturation of EF skills during adolescence and young adulthood facilitates a proactive top-down approach to directing attention and exerting cognitive control. This ability declines during typical older age, resulting in reactive responses once again (Dempster, 1992;

Diamond, 2013; Hasher & Zacks, 1979; Zelazo et al., 2004; McCabe et al., 2010; Anderson & Craik, 2017). For instance, EF (e.g. cognitive flexibility, updating, inhibition, and fluency), WM capacity and processing speed have all been shown to steeply decline from typical young adulthood to older age (Craik & Byrd, 1982; Dempster, 1992; Salthouse, 1996, 2010; Schaie, 2003; and see Reimers & Maylor, 2005; McCabe et al., 2010). What is more, processing speed appears to be a distinct process that modulates more general cognitive functioning in typical older age (Anderson & Craik, 2017), but does not mediate age-related declines in EF (McCabe et al., 2010; Cappelletti et al., 2015). Further evidence for the link between EF and frontal processes in typical development is supported from neuropsychological brain imaging studies. Several studies of typical ageing show reduced synchrony between frontal and other brain regions, such as the hippocampus of medial temporal lobes, and volumetric decreases with increasing age in both frontal and hippocampal regions (e.g. Raz et al., 1998; and see Hedden & Gabrielli, 2004).

The evidence just discussed shows an “inverted u-shape” for the typical developmental trajectory of EF across the lifespan (Dempster, 1992; Zelazo, Craik & Booth, 2004). Developmental gains in EF skills supports general intellectual ability (Freidman, Mitake, Young, DeVries & Hewitt, 2006) and has a broader impact on several domains of cognitive functioning and behaviours. For instance, improved cognitive flexibility facilitates perspective taking and the development of theory of mind (Perner & Lang, 1999; Kloo & Perner, 2008; Wimmer & Doherty, 2011), self-reflection (Zelazo, 2015) and self-generated representations for memory, including autobiographical and episodic memory (Mäntylä et al., 2010) and prospective memory for future actions (Marsh & Hicks, 1998; Finstad, Bink, McDaniel & Einstein, 2006). Further, EF gains across development mediate risk-taking behaviours, mental health problems and health-related QoL (Diamond, 2013). Consequently, EF may be considered a domain-general construct, which additionally governs domain-specific features of cognitive and behavioural functioning (Bagetta & Alexander, 2016).

It is clear from the evidence just described that EF is important in everyday functioning. Thus, the implications of EF difficulties on the broader functioning of autistic individuals is discussed next.

Executive function in autism

There is a large body of evidence that converges on executive dysfunction in autism, as possible explanation for the core social and non-social features of ASD (Hill, 2004; Lopez et al., 2005; Lawson et al., 2015; and see Lai, Lombardo & Baron-Cohen, 2014). Although the patterning of EF abilities and difficulties does not conclusively support this view (Geurts, Corbett & Solomon, 2009). A complicated patterning of EF is observed in autistic children (Minshew et al., 1992; 1996; Ozonoff, Pennington & Rogers, 1991; Pennington, Rogers, Bennett et al., 1997; Geurts et al., 2004; Williams et al., 2006; van Eylen et al., 2011), adolescents (Happé, Booth, Charlton & Hughes, 2006; Pellicano et al., 2006) and adults (Ozonoff et al., 2004; Geurts & Vissers, 2012; Lever & Geurts, 2016; Powell, Klinger & Klinger, 2017).

The diversity in methods and paradigms used in the ASD literature presents a complicated landscape to systematically quantify EF difficulties and abilities (but see Hill, 2004; Eigsti, 2011; Kercood et al., 2016 for comprehensive reviews). Studies of EF in ASD have largely explored cognitive flexibility, inhibition and planning. Investigations of WM are either specifically related to EF (Hoffman, Schmeichel & Baddeley, 2012; and see a comprehensive meta-analysis by Wang, Zhang, Liu et al., 2017), or separately assessed as an independent memory construct (Boucher, Mayes & Bigham, 2012; Geurts & Vissers, 2012; Lever et al., 2015; Geurts, 2016; and see review by Kercood et al., 2014). However, few studies have specifically explored attention in EF in relation to ASD (Minshew et al., 1992; Williams et al., 2006). Instead, the focus of previous literature has mainly been on attention to social information processing or perceptual processing (see Lai, Lombardo & Baron-Cohen, 2014), or attention switching in relation to cognitive flexibility (e.g. Landa & Goldberg, 2005). A summary of EF ability in ASD is presented next, in relation to the core components of EF involving cognitive flexibility, WM, and inhibition, followed by EF subcomponents related to planning and fluency.

Difficulties with cognitive flexibility in ASD have been shown to persist across adolescence (Ozonoff, Pennington & Rogers, 1991; Hughes et al., 1994; Ozonoff & Jensen, 1999; Geurts et al., 2004; South, Ozonoff & McMahon, 2007) and into older age (Ozonoff et al., 2004; Ambery et al. 2006; Bramham et al. 2009; Goldstein et al. 2001; Minshew et al. 2002; Hill and Bird 2006; Lopez et

al. 2005; Powell, Klinger & Klinger, 2017). However, at least two studies report no specific difficulties in older autistic adults (Lever et al., 2015; Davids et al., 2016) and no evidence of increased age-related difficulties (Geurts & Vissers, 2012; Lever et al., 2015). Therefore, it may be that cognitive inflexibility in ASD is not, necessarily, a pervasive deficit (Geurts, Corbett & Solomon, 2009), but that difficulties might instead be related to the type of task demands and whether other EF processes, such as WM and inhibition, are required for task success (van Eylen et al., 2011). The profile of WM in ASD is also not straightforward (Kercood et al., 2014; Geurts & Vissers, 2012; Lever & Geurts, 2015; Wang et al., 2017). Several studies show that WM as an EF component is generally intact across childhood (Ozonoff & Strayer, 2001; Geurts et al., 2004) and adulthood (aged 20-79 years) with no evidence of increased age-related difficulties (Lever et al., 2015; Wang et al., 2017). Studies with older autistic adults show intact verbal and visual memory across ages, whereas difficulties with verbal fluency and semantic memory are more pronounced in older individuals (Lever & Geurts, 2016). However, a different picture emerges when task type and cognitive load are varied, with more demanding tasks producing more pronounced difficulties across the lifespan (Wang et al., 2017). Further, tasks that require the integration of other EF processes, such as flexibility or inhibition, may also lead to increased WM difficulties in ASD (Kercood et al., 2014). The patterning of diversity is also observed in studies of inhibition as a core EF component. Early investigations suggest that inhibition is uncompromised in ASD (Ozonoff, Pennington & Rogers, 1991; Ozonoff & Strayer, 1997), even compared to individuals with ADHD and Tourette syndrome (Ozonoff & Jensen, 1999; Geurts et al., 2004). However, task demands again appear important, as do the measures of inhibition itself – whereas, inhibitory control is generally found to be intact (Ozonoff & Jensen, 1999; Geurts et al., 2004), difficulties inhibiting prepotent responses are more pronounced in autistic individuals across the lifespan (Geurts et al., 2004; Robinson, Goddard, Dritschel, Wisley & Howlin, 2009; Geurts, Van Den Bergh & Ruzzano, 2014). Planning difficulties are commonly observed in ASD at all ages, such as the need for additional planning time, increased planning errors and the inefficient use of strategies (Hughes et al., 1994; Geurts et al., 2004; Ozonoff et al., 2004; Robinson et al., 2009). Furthermore, planning difficulties appear to be a specific challenge for autistic individuals, as compared with other development disorders, such as Attention Deficit Hyperactivity Disorder

(Hill, 2004; Geurts et al., 2004). Finally, fluency completes the patterning of diversity presented here. Whereas, some studies with children and older autistic adults report marked fluency difficulties (e.g. verbal fluency and generativity; Geurts et al., 2004; Geurts & Vissers, 2012), other studies report no evidence of fluency difficulties (Ozonoff & Strayer, 1997; Robinson et al., 2009).

Summary of Executive function as a core impairment in ASD

There is converging evidence that difficulties with EF and its subcomponents – cognitive flexibility, WM, inhibition, planning and fluency – may be related to the core features of autism (Lai, Lombardo & Baron-Cohen, 2014; Lopez et al., 2005). Further, recent evidence suggests that some EF may present broader cognitive difficulties for autistic individuals across the lifespan. However, whereas fluency and planning are not strongly associated with the core behavioural features of autism, whereas cognitive flexibility, WM and inhibition are more strongly associated autistic features, such as restricted interests and repetitive behaviours (Pellicano, 2013; Lawson et al., 2015). The patterning of difficulties and abilities already mentioned does not present a unified view of EF as a core deficit in autism. However, several subdomains of EF impairment are observed across the autistic lifespan, and these difficulties may be mediated by cognitive load, the interdependency on other EF components and task complexity (Geurts et al., 2009; Kercood et al., 2014). EF necessarily recruits additional components and higher-order cognitive functions, leading to integrative network of cognitive processes that are not, as was noted earlier, “process pure” (McCabe et al., 2010, p. 223; Friedman & Miyake, 2017). Consequently, many cognitive tasks recruit multiple core and subcomponents of EF for successful completion. Since the majority of EF studies in ASD, to date, have not systematically measured the core and subcomponents of EF across the lifespan, it is also not possible to wholly discard this theory. Therefore, future work needs to address these issues in a systematic evaluation of the breadth of EF capabilities across the adult lifespan. More research is needed to understand the interdependence of EF components and other cognitive functions, such as language, general intellectual ability and memory, and of how these higher-order cognitive functions relate to core autistic traits, and their impact on the QoL of autistic individuals.

Table 2.2. Models of Executive Function

Author(s)	Model	Salient attributes	Spheres of influence	Evidence in support of model
Miyake, Freidman, Emerson, Witzki, Howerter & Wager (2000); Freidman & Miyake (2017)	Integrative framework	High-level cognitive processes that regulate goal-directed behaviour through the operation of common lower-level processes	Inhibition ¹ Working memory ² Cognitive flexibility ³	Early childhood (pre-school) EF skills predicted general learning ability, language skills, emotional development and social skills (Bierman et al., 2008); Difficulties with working memory and inhibition in young and middle-aged adults were associated with poorer metamemory (one's own awareness of one's memory capability) and greater source memory errors (Mäntylä et al., 2010); Inhibition and cognitive flexibility errors in older adults were associated with poorer driving performance (Adrian et al., 2011).
Diamond (2006; 2013)	Multidimensional construct	Top-down mental processes involved in effortful cognitive control	Inhibition ¹ Working memory ² Cognitive flexibility ³	The majority of evidence is centred around childhood outcomes: EF ability (inhibitory control, cognitive flexibility, working memory) in pre-schoolers (aged 3.5 – 6 years) was associated with adaptive social functioning / “social competence” (cooperation, interaction, independence; Razza & Blair, 2009, p. 7); cognitive flexibility was associated with school readiness (Vitiello et al., 2011) and inhibition with later academic outcomes (Roebbers et al., 2011). EF strategy selection and task efficiency in EF processes improved with age between childhood and adolescence (ages 8-13 years) and, in turn, improved inhibitory control and cognitive flexibility (Lemaire & Lecacheur, 2011).
Zelazo (2015)	Iterative reprocessing; reflection / reflective processing	“Goal-directed modulation of attention and behaviour”	Inhibition ¹ Working memory ² Cognitive flexibility ³	Dimensional Change Card Sorting task (DCCS) – the task involves matching a given card to a pre-defined rule (working memory) of matching to different dimension e.g. shapes or colours and switching between them. Young children aged 3-4 years are typically able to learn the rules (i.e. shapes vs. colours; working memory) but show greater difficulties applying the rules to a change in context (e.g. shapes to colours), which requires cognitive flexibility. Failure to inhibit use of the previous rule and update the strategy to apply a different rule results in perseveration and error responses (Zelazo, 2006).

Notes: Conceptual similarities are reported across all models described above, although the authors use variations in terminology, e.g.

¹Inhibition/ inhibiting (Miyake et al., 2000; Friedman & Miyake, 2017; Diamond, 2013); inhibitory control (Zelazo, 2015);

²Working memory (Diamond, 2013; Zelazo, 2015); updating, manipulation and maintenance (Miyake et al., 2000; Friedman & Miyake, 2017);

³Cognitive flexibility (Diamond, 2013; Zelazo, 2015), shifting / set shifting (Miyake et al., 2000; Friedman & Miyake, 2017).

Weak Central Coherence and its variants as a cognitive style in ASD

Central coherence is the drawing together of information to interpret meaning about a given context and to form a complete (coherent; *global*) view of the world (Frith, 1989; Happé, 1999a; Happé & Booth, 2008). Global processing of information is said to be achieved by obtaining the “gist” whilst eliminating the need to retain local detail, and is, therefore, at the cost of attention to detail and memory for competing detailed information (Happé, 1999b; Hill, 2004). Visual perception and conceptual interpretation, such as required in narrative comprehension, are both processes that rely on central coherence (Happé, 1997; Happé, 1999b; Brosnan et al., 2004), suggesting a broader implication for cognitive functions beyond the descriptions offered above. By contrast, weak central coherence (WCC; Frith, 1989; Happé, 1999a) is a tendency to focus on *local* detailed information of individual parts more readily than processing of the whole³, at the cost of reduced contextual interpretation. Theoretical explanations offered by the WCC account and its variants have attempted to describe the social and non-social behaviours in autism, as a domain-specific impairment in Central Coherence as the global integration of information (Frith, 1989; Happé, 1999a), Enhanced Perceptual Functioning (see Mottron, Dawson, Soulières, Hubert & Burack, 2006 for a detailed account of the principles underlying this model); and Attention to Detail (Bölte et al., 2011; but see Bölte et al., 2007 for alternate evidence of local-global processing differences). TD individuals routinely achieve central coherence, as demonstrated in tasks such as narrative completion or social scene construction. Whereas, a different patterning is observed in autistic people who show a tendency toward WCC. In ASD, WCC is also presented as strengths, in enhanced processing of local detail information (Shah & Frith, 1983, 1993; Plaisted et al., 1999; 2001; Mottron & Burack, 2001; Mottron et al., 2006; Dawson et al., 2007) and lower susceptibility to visual illusions (Mottron, Belleville & Ménard, 1999; Bölte et

³ The premise of Happé’s approach is that central coherence facilitates the integration of information for *gestalt* processing – i.e. perceiving the whole picture or context, by construal of constituent parts, whereby the ‘whole is greater than the sum of its parts’. To date, many researchers have interchangeably used the terms *gestalt* and *global* when describing configural processing (Happé, Briskman, & Frith, 2001; Plaisted et al., 1999), although it is argued that the hierarchical organisation of information involved in configural processing is supported by different cognitive mechanisms than gestalt perception. Bölte et al. (2007) emphasise that the whilst different mechanisms may be involved in gestalt and global processing, these may not be mutually exclusive, since gestalt perception may “be a [pre-attentive] prerequisite for more far reaching global perception.” (Bölte et al., 2007, p. 1494; and see discussion by Brosnan et al., 2004). For simplicity and consistency, the terms *global* and *local* will be used in this discussion of Weak Central Coherence theory, relating to particular experimental evidence in support of the performance differences in local-global processing by autistic individuals.

al., 2007), functions that depend on local information processing. However, resulting cognitive difficulties include reduced integration of information to configural wholes (Happé, 1999b) and impaired generalisation of perceptual learning to other contexts (Plaisted, O’Riordan & Baron-Cohen, 1998), difficulties in disambiguating narrative context from syntactic context (Snowling & Frith, 1986; Happé, 1997) and reduced recognition of individual items within scene constructions (Ring, Gaigg & Bowler, 2015).

Evidence for the patterning of WCC in ASD just mentioned, comes from tests of perceptual reasoning and visuo-spatial abilities, such as the Block Design (BD) task in Wechsler’s tests of general ability (e.g. Wechsler, 2008; Dawson, Soulières, Gernsbacher & Mottron, 2007), and the Embedded Figures Test (EFT; Witkin, Oltman, Raskin & Karp, 1971); autistic individuals generally show superior performance on these tasks compared to TD individuals (Mottron, Burack, Iarocci, Belleville & Enns, 2003; Frith & Happé, 2006). In the BD test, participants are shown a whole pattern (global configuration) and given all the individual pattern parts (local elements) as red/white blocks with which to construct the three-dimensional whole (the task is also time-limited, and each pattern should be constructed in as short a duration as possible). In the EFT, participants are shown a complex configuration of a two-dimensional geometric shape made up of smaller (embedded) geometric shapes – their task is to identify either the local elements (smaller parts) or global elements (larger whole). Both BD and EFT draw on visuo-spatial processing, with obvious differences in the dimensional features of the tasks mentioned above; specifically, the BD relies on “visual construction” of the whole, whereas the EFT is a “visual search” task (Bölte et al., 2007). In line with the WCC approach, if a local processing bias is present, then participants should be quicker and more accurate at identifying the embedded figures and replicating the block design patterns, whereas, if global processing is predominant then individuals should have greater difficulties reconstructing the whole from its fragmented constituent parts. Shah & Frith (1983; 1993) found that children with Kanner-type autism were faster and more accurate in identifying embedded figures than matched groups of TD and ID children (Shah & Frith, 1983), and autistic children with low and high IQ also showed superior performance on BD patterns (Shah & Frith, 1993).

Similar findings of superior performance have been reported in replication studies with other groups of autistic children (Dawson, Soulières, Gernsbacher & Mottron, 2007), adolescents (Ropar & Mitchell, 2001; Mottron et al., 2003) and adults (Joliffe & Baron-Cohen, 1997; Dawson et al., 2007; Bölte et al., 2007). However, previous research has yielded mixed results (Mottron et al., 2003; Dawson & Mottron, 2006; Bölte et al., 2007) and the findings of do not present a universal view in favour of WCC as global processing impairment in ASD (Plaisted et al., 1998; Mottron et al., 2006). Several studies have reported that autistic individuals are not only faster on local-level information processing, but that they are able to process global context as well as TD individuals, in certain circumstances (Mottron & Belleville 1993; Plaisted, Swettenham & Rees, 1999). Plaisted and colleagues argued that attentional mechanisms in ASD are biased toward discrimination between features, as opposed to integration of features for contextual meaning, meaning that configural processing is not deficient in autism, per se, but that processing of local information is preferentially processed where circumstances required divided as opposed to selective attention (Plaisted, Swettenham & Rees, 1999; Plaisted, Saskida, Alcántara & Weisblatt, 2003). The latter suggestion converges with earlier findings by Mottron & Belleville (1993) who posited that a lack of *global precedence* may underlie the patterning of global processing differences in ASD, and the resulting patterning of differences that may be explained by disordered organisation between lower and higher hierarchical levels of processing (hierarchization deficit model; Mottron, Bellville & Ménard, 1999; Mottron & Burack, 2001; Mottron et al., 2003; Mottron et al., 2006).

The patterning of findings just described suggests that autistic individuals make less use of contextual information in real-world information processing (Pellicano & Burr, 2012), which may be explained by reduced neural connectivity and synchrony between the underlying cognitive mechanisms (Boucher & Mayes, 2012) that modulate the accumulation of prior knowledge for future reference. Moreover, these findings hold potentially important implications for ageing autistic individuals who may demonstrate reduced tendency to global information processing. For instance, at least one study has shown weak central coherence in patients with Alzheimer's disease (Mårdh, 2013), who tended to identify fragmented details of an image (e.g. picture of a fire), rather than being able to process the whole context, highlighting potential risk factors for everyday living. However,

whilst more research is clearly needed to understand the potential implications of the varying positions just discussed, these are beyond the scope of work in this thesis (but see Yazar, 2017, for further exploration of these issues).

Theory of Mind theory of ASD

Theory of Mind (ToM) refers to a person's ability to construct representations of their environment, interpret their own mental states – which is also referred to as metacognition, or thinking about one's own thoughts – and to understand the intentions, beliefs, desires and inferred mental states of others (Premack & Woodruff, 1978; Berk, 2010). In this way, ToM is thought to be a core function in social communication and social interaction (Rozga, Anderson & Robins, 2011) and, therefore, provides a platform for understanding the social behavioural features of autism. The idea that representational difficulties may underpin ToM impairments is contextually relevant – in order to form an understanding of a given context, one first needs to be able to establish a representation of that context or experience. This ability extends to the development of self-awareness (Hobson, 1992) and episodic autobiographical memory, both of which draw on previously experienced representations, or “metarepresentational abilities” (Kristen, Rossman & Sodian, 2014; Perner, Kloo & Gornik, 2007). ToM skills are often assessed by false belief tests at two levels: first order and second order attributes. First order attributions of ToM extend metacognition to develop an understanding of the mental states of others and their goal-directed intentions (agency, e.g. Sam thinks there are smarties in the box; Perner, Frith, Leslie & Leekam, 1989), engage in symbolic play (pretend, e.g. John thinks that the banana is a telephone) and aid joint attention through social orienting (Charman, 2000; Rozga, Anderson & Robins, 2011). Whereas, second order attributions involve recursive thinking about mental states, to predict one person's (first order) thoughts about another person's thoughts about a particular scenario (e.g. Alex thinks that John thinks the football match is about to start; Perner & Wimmer, 1985; Baron-Cohen, 1989).

In TD children the acquisition of first order ToM develops in early childhood, usually by age 3-4 years, when children become aware of their sense of agency and how their own beliefs and desires, and those of other people, affect the ways in which people behave (Wimmer & Perner, 1983; Berk,

2010). This knowledge continues to develop throughout middle childhood, when children can conceptualise false belief (Leslie, 1987) – the understanding that people may hold beliefs that are incongruent with the reality of a situation (e.g. I wear a raincoat because I believe it is raining outside, but you know that it is not the case). This knowledge provides a platform for taking the perspectives of others as well as becoming aware of one’s own views of the world and, accordingly, developing a sense of self (Hobson, 2002; Berk, 2010). The continued development of ToM across early-middle childhood enables the child to develop self-reflection and the ability to exercise self-control (Perner & Lang, 1999; Berk, 2010). There is some evidence that ToM declines with cognitive ageing in later life in TD individuals (Charlton et al., 2009; Fischer, O’Rourke & Loken Thornton, 2016; but see Happé, Winner & Brownell, 1998), but this may depend on factors associated with metacognitive or affective reasoning abilities and the influence of other inter-related cognitive abilities such as episodic memory and EF (Fischer et al., 2016).

In ASD, a pivotal study by Baron-Cohen et al. (1985) led to the suggestion that ToM is often delayed or may not develop at all during the lifespan. In their study, 80% of autistic children, who also had lower cognitive abilities, failed first order ToM tasks; a subgroup of autistic children who did not show ToM impairments in first order ToM nonetheless still failed the second order false belief task, despite having equivalent mental age to a comparison group of TD children (Baron-Cohen et al., 1989). These findings led Baron-Cohen (1989) to suggest that ToM was a global impairment in autism, arguing that failure to carry out the second order false belief tasks inferred an underlying ToM deficit. Baron-Cohen (1989) went on to suggest that this impairment explained not only the social difficulties of ASD, but also non-social features such as repetitive behaviours (Jones et al., 2018; but see Boucher, 2014). Furthermore, “context-appropriate” mentalising presents specific difficulties for autistic difficulties, such as interpreting mental states of others in narratives on the Strange Stories tasks (Happé, 1994) – a test designed to assess how ToM skills relate to everyday social interactions, by evaluating an understanding of white lies, bluffing, pretence, joking, sarcasm and similar forms of nonliteral communication. These difficulties have been shown in autistic children (Happé, 1994), adolescents (White, Hill, Happé & Frith, 2009) and adults (Jolliffe & Baron-Cohen, 1999), indicating a broad difficulty in understanding agency and revisiting the idea that ToM impairments may be

underlying the core features of ASD (Jones et al., 2018) that may present difficulties across the lifespan.

However, compelling counter-evidence from several studies (Happé, 1995; Happé et al., 2006; and see review by Boucher, 2014) suggests that ToM deficits may not be uniformly problematic for all autistic individuals. Bowler (1992) and Happé (1995) proposed that reasons for the patterning of differences could be that more cognitively able autistic individuals are able develop compensatory solutions (“hack out”) to ToM tasks, rather than using innate ToM or mentalising abilities (Lind & Bowler, 2009, p. 930; and see Bowler, 1992; Frith & Happé, 1994). At least one other study suggested that difficulties did not extend to real world perspective taking, given that autistic individuals were as able as TD individuals in discriminating between their own beliefs and those of another person, but instead had specific difficulties with forming mental representations (Begeer, Malle, Nieuwland & Keysar, 2010). Bowler and colleagues (2005) extended the view that complex cognitive abilities are involved in false belief understanding. They argued that false belief tasks are more complex in their nature, requiring construal of episodic detail and an understanding of a person’s goal-directed actions in a given context, suggesting that false belief difficulties occur irrespective of mental state understanding, but instead reflect more general difficulties with metarepresentational comprehension and complex reasoning abilities about goal-directed action.

How are ToM, WCC and EF linked?

The links between ToM and EF have long been debated in typical development (Wimmer, 1989; Perner & Lang, 1999; Frye et al., 1995; Perner & Lang, 2000; Diamond, 2013) and autism (see Jones et al., 2018), suggesting that ToM is a precursor for EF even in early childhood development, and, reciprocally, that the development of EF skills may mediate the development of ToM (Pellicano, 2010, 2013), self-reflection (Zelazo, 2015), perspective taking and metamemory (Mäntylä et al., 2010). Specific components of EF, such as inhibition and WM appear to predict ToM abilities related to false belief and mental state understanding (Perner & Lang, 2000; Carlson & Moses, 2001; Carlson, Moses & Claxton, 2004), but this may be modulated by the complexity of inhibitory control demands (Carlson et al., 2004). The interplay between these cognitive mechanisms may explain some

of the behavioural features of autism associated with restricted interests and stereotyped behaviours (South et al., 2007; Jones et al., 2018). By contrast, the association between EF and WCC is less clear – EF planning ability (Frye, 2000; Atance & O’Neill, 2001) and inhibition may underlie cognitive flexibility in switching between global-local information processing (Wimmer & Doherty, 2011), but there is little evidence for this in ASD (South et al., 2007). Further, language is also thought to play an important role in the acquisition of ToM and EF (Perner & Lang, 1999; Carlson et al., 2004) and may mediate EF skills, such as planning, but is little understood about the extent to which language and general intellectual ability may shape the development of EF across the lifespan (Carlson et al., 2004; Eigsti, 2011).

Evaluation of theoretical contributions and their implications for ageing and ASD

The attempts by researchers to explain autism under a unitary model are not without challenges (Minshew, Sweeney & Luna, 2002; Waterhouse, London & Gillberg, 2016). The validity of a theory lies in its predictive value for the observations it is trying to explain (Hampton, 1998, p. 18; Elmes, Kantowitz & Roediger, 2006, p. 41), in a way that is parsimonious, precise and testable (Elmes et al., 2006, p. 46). Thus, the validity of a theory should first identify the “core deficit” that it is trying to explain, and that deficit should be: (a) universal; (b) specific; and (c) unique (Eigsti, 2011, p. 185). Accordingly, a patterning of (a) impairment should be observed “at some developmental period, regardless of symptom severity”; (b) both strengths and difficulties should be observed in the predicted domain or function; (c) functional differences should be clearly differentiated from other conditions that may be associated with those difficulties.

The theories of autism described above have focused on either social (e.g. theory of mind) or non-social (e.g. weak central coherence; executive dysfunction) features of ASD and the underlying cognitive mechanisms associated with functional difficulties. Social cognitive theories have posited domain-specific accounts of the difficulties in social functioning (Frith & Frith, 2012) explained by impaired ToM (Baron-Cohen et al., 1985), false belief understanding (Happé et al., 1995), and mentalising (Frith, 1996). It is evident from the equivocal findings presented earlier in this chapter that there are problematic issues with the Theory of Mind theory of autism. Firstly, ToM deficits in

ASD do not appear systematically in autistic individuals (Bowler, 1992; Bowler et al., 2005; Begeer et al., 2010). Accordingly, Bowler (2008) suggests that ToM deficits may be “more subtle and harder to detect” (p. 42) in autistic people at different developmental stages, level of cognitive functioning and with less problematic autistic traits (Bowler, 1992; 2008; and see Rozga et al., 2011). Second, difficulties with the first order ToM task were also observed in the children with Down syndrome who formed the comparison group in Baron-Cohen et al.’s (1985) study, suggesting that a ToM deficit is not unique to the difficulties associated with ASD (Boucher, 2014). Third, a lack of consensus in what is defined as ToM or mentalising, and false belief, remains problematic in uniformly endorsing the ToM theory of autism (Rozga et al., 2011). Furthermore, the differences between autistic individuals of greater or fewer cognitive abilities and language skills suggests that ToM difficulties in ASD may reflect difficulties associated with broader, domain general cognitive functions (see Bowler, 2008, and see Boucher, 2014 for review).

The above explanations are necessarily limited by accounting for only some aspects of the core features of ASD, whereas non-social theories have attempted to explain both behavioural and cognitive features of ASD. Non-social theories of ASD have centred on a Weak Central Coherence account (WCC; Frith, 1989; Happé, 1999), or an Executive Dysfunction account (EF; Hill, 2004; Ozonoff et al., 2004). The WCC account includes variants that posit Enhanced Perception (Plaisted et al., 1998; 2001; Mottron et al., 2006) and Attention to Detail (Bölte et al., 2007; Bölte et al., 2011) as explanatory mechanisms of the non-social difficulties associated with ASD. However, the WCC account has also produced mixed results (Mottron et al., 2003; Dawson & Mottron, 2006; Bölte et al., 2007) and the findings of do not present a universal view in favour of WCC as global processing impairment in ASD (Plaisted et al., 1998; Mottron et al., 2006). Whether the patterning of strengths in attention to detail (Bölte et al., 2011) and enhanced perceptual processing (Mottron et al., 2002) is the function of a cognitive style (Happé & Booth, 2008), or a disordered organisation between lower and higher hierarchical levels of processing (Mottron et al., 2003), requires more systematic exploration. Moreover, the many variants of WCC theory indicates that it is problematic in determining the universality and specificity of this model to explain the core features of ASD. In sum, whilst theories of WCC and ToM have offered valuable contributions to providing a richer description of some of the

cognitive and behavioural features of ASD but do not uniformly explain all the social and non-social features of autism, such as repetitive behaviours. Further, both these accounts attempt to describe single modular deficits, whilst other cognitive functions may be unaffected (e.g. IQ).

By contrast, the broader perspective of EF and Information Processing theories of ASD (Hill, 2004; Williams & Minshew, 2001; Williams et al., 2006) have attempted to explain the patterning of cognitive and behavioural features in ASD through an executive dysfunction account (Hill, 2004; Eigsti, 2011; Ozonoff et al., 2004; South et al., 2007). The EF theory of autism offers a compelling approach to understanding the broader picture of autism-related difficulties. For instance, the disordered organisation of information, associated with perceptual and conceptual processing – which relates to social and non-social processing mechanisms – elicits differences in the way in which autistic individuals experience the world. Consequently, these processing differences may lead to fewer opportunities to develop contextual representations and experiences (Williams & Minshew, 2010; Bowler, 2008; Gaigg, 2012). Similarly, the EF account offers an explanation of the cognitive and behavioural patterning in ASD, related to difficulties and strengths across various domains of functioning and their associated brain regions. Evidence from neuroimaging studies has highlighted the underconnectivity within these regions and atypical inter-connectivity across regions. Thus, the evidence would suggest a strong basis for the information processing differences that underlie EF in autism. The atypical patterning of functional connectivity, just mentioned, may account for difficulties with emotion recognition (Cook et al., 2013) and reciprocity (see Chevallier et al., 2012; and see review by Gaigg, 2012), global information processing (Frith, 1989; Happé, 1999a) and EF of general information processing (Hill, 2004; Williams et al., 2006; Just et al., 2012). Additionally, disordered processing may also account for difficulties in facial recognition (Weigelt, Koldewyn & Kanwisher, 2012), the interpretation of nonverbal cues in facial expressions (Pelphrey & Carter, 2008), biological motion (Castelli, Frith, Happé & Frith, 2002; Kaiser et al., 2010), speech perception (Gervais et al., 2004) and mental state understanding (Baron-Cohen et al., 1985; Happé, 1995; Frith & Happé, 1994; White, Hill, Happé & Frith, 2009). What is more, EF difficulties explain the patterning of repetitive behaviours and restricted interests in ASD (Lopez, Lincoln, Ozonoff & Lai, 2005; South

et al., 2007; Boyd et al., 2009; Bölte et al., 2011), which other theories of autism have not been able to explain.

However, the underlying challenge of the EF approach to explaining ASD is that most previous studies describe only a small subset of EF components in autistic groups compared to TD or other clinical groups. As we have seen in the review of EF presented earlier, the components that make up EF are many and complex, with inter-relations among those components. Few studies have evaluated the global contribution of EF, and individual differences in performance, in relation to the core difficulties of ASD – social communication and rigid and repetitive behaviours (Ozonoff et al., 2004; Lopez, Lincoln, Ozonoff & Lai, 2005; Boyd et al., 2009). Moreover, the findings to date have not explained (and few studies have explored) the associations between specific EF strengths or difficulties and other cognitive functions, or the inter-relatedness among EF components in ASD. Finally, the gap in the current literature requires a broader exploration of EF on multidimensional assessments that incorporate the range of executive functions (Janssen et al., 2014), and longitudinal evaluations of strengths and difficulties over time (Pugliese, Kenworthy et al., 2016) to understand the full impact of EF on developmental outcomes across the lifespan (Eigsti, 2011). Future investigations that incorporate these aspects serve to elucidate the core role of EF in ASD, in relation to difficulties and strengths, and importantly, establish a unified approach that maps EF as a construct on to the conceptualisations that have been identified in the literature on typical development and other clinical groups (Bagetta & Alexander, 2016; Janssen et al., 2014).

In sum, the prominent theories of autism described above, each has its own strengths and weaknesses in relation to the domains of functioning that they attempt to explain. No single theory has been able to explain the variable patterning of cognitive difficulties, absence of difficulties, or strengths in autistic individuals across the lifespan. However, each of these theories has contributed to the advance in knowledge about autism and the patterning of difference compared to typically developed individuals. The challenge for each of these theories is that they lack the unity and specificity to explain the specific patterning of strengths and difficulties associated with the core features of autism. However, these theories have advanced our understanding of autism and have illuminated the specific patterning of differences in ASD, such as perceptual processing, cognitive

flexibility, attention and WM, and mental state understanding. Moreover, the interplay between these functions and other domains of functioning provide foundations for future exploration of developmental trajectories across the lifespan (Gaigg, 2012).

The evidence described earlier in this chapter has highlighted multiple cognitive and behavioural difficulties that are associated with distinct neural mechanisms and brain regions as well as specific difficulties with cognitive functions that engage frontal lobe processes, such as those involved in EF, memory and global information processing. Furthermore, these cognitive functions are important for healthy cognitive ageing and QoL, as seen from research with typically ageing adults. Rather than attempting to explain a single defining cause of ASD, more valuable explanations offer insights to the cognitive profiles and trajectories associated with ASD, the specific patterning of clinical traits and behavioural features and which brain regions may be implicated in this patterning (Minshew, Sweeney & Luna, 2002) and captured by cognitive tasks that evaluate cognitive difficulties and strengths associated with specific cognitive processes in younger and older individuals across the lifespan (McCabe et al., 2010). Bowler (2001) echoes this view, proposing the need for longitudinal research to elucidate some of the unanswered questions that pertain to the developmental lifespan of autistic individuals.

2.3 Ageing and ASD: parallels to typical ageing, what is known, and what is yet unknown

As we have seen from the evidence presented above, the developmental trajectory for autistic individuals is quantitatively and qualitatively different from the patterning of cognitive functions in typical individuals. Estimates of intellectual functioning range from extreme disability to superior ability, and may be accompanied by specific difficulties in social cognitive processing (Chevallier, 2012; Gaigg, 2012), theory of mind (Happé, 1994; Frith & Frith, 2003; Jones et al., 2018), and executive functions such as cognitive flexibility (Hughes et al., 1994; Lawson et al., 2015) inhibition, planning and WM (Hill, 2004; South, Ozonoff & McMahon, 2007; Boucher & Bowler, 2008) and specific aspects of memory including self-generated episodic recollection (free recall; Boucher,

Mayes & Bigham, 2012). The patterning of cognitive difficulties just described is commonly observed in children and younger autistic individuals and resembles a similar profile of functioning observed in individuals with frontal lobe (EF) and hippocampal damage (memory; Boucher, Mayes & Bigham, 2008). Thus, these parallels highlight the importance of these brain regions and their interconnectivity in supporting broader cognitive functions. Furthermore, the patterning of strengths and difficulties observed in young autistic individuals and children parallels the cognitive and behavioural differences seen in older typically ageing individuals, for whom many cognitive functions are exposed to age-related declines in older age (Craik, 1986; Salthouse, 1996; Anderson & Craik, 2000; Kester, Benjamin, Castel & Craik, 2002; Anderson & Craik, 2017; Bowler et al., 2004; Bowler et al., 2007).

Parallels with typical ageing

It is well known that cognitive changes occur as typically ageing (TA) individuals approach old age (Salthouse, 2016). What is constituted as typical ‘old’ age remains an area of much discussion, whether aged 50+ years (Albert & Heaton, 1988), 55+ years (Ronnlund, et al., 2005; McCabe et al., 2010), 60+ (Salthouse, 2009) or 65 years and older (Salthouse & Saklofske, 2010). Nonetheless, it is generally acknowledged that neurocognitive changes include increased forgetting and memory decline (Craik & Byrd, 1982; Crook et al., 1986; Salthouse, 2009) which includes reduced capacity for episodic memory (McCabe et al., 2010) and prospective memory for future actions (Craik, 1986; McDaniel and Einstein, 2000), as well as selectively impaired EF in cognitive flexibility, WM, inhibition, attention, planning and information processing (Salthouse, 1996; Zelazo, Craik & Booth, 2004; Cappelletti et al., 2015; Anderson & Craik, 2017). Broader cognitive implications of age-related declines extend to diminished theory of mind (Charlton et al., 2009; Fischer et al., 2016; but see Happé et al., 1998), impaired perceptual reasoning (Salthouse & Saklofske, 2010), poor discrimination of item-context associations (Ward, Maylor, Poirier et al., 2017) and difficulties extracting local-to-global information (Cappelletti et al., 2015). The patterning of age-related difficulties accelerates between middle age and older age (McCabe et al., 2010; Salthouse, 2009) and although distinct domains of function are observed there is also considerable overlap between these

domains and their underlying processes. For instance, processing speed affects the capacity to process details and large amounts of information, and the retrieval of that information for later memory recall (Craik & Byrd, 1982; Dempster, 1992; Salthouse, 1996; McCabe et al., 2010; Salthouse & Saklofske, 2010). This is further compounded by age-related declines in episodic memory retrieval (Atance, 2010) and prospective memory ability (McDaniel and Einstein, 2001), which, in turn, are affected by age-related declines in EF and WM capacity (Zelazo, Craik & Booth, 2004; McCabe et al., 2010) and information processing (Salthouse, 1996). The association between processing speed and other higher-order cognitive functions, such as EF and WM capacity is a complex one. Whereas processing speed is associated with WM and EF, as well as independently associated with other cognitive functions such as episodic memory and numeracy (number acuity; Cappelletti et al., 2015) and prospective memory (Salthouse, Atkinson, and Berish, 2003; Mäntylä, 2003; Mäntylä, Rönnlund & Kliegel, 2010), it does not account for the variance in age-related declines explained by WM and EF alone. Furthermore, some recent evidence suggests that age-related cognitive difficulties can be attenuated by training (Cappelletti et al., 2015; Badham, Poirier, Gandhi et al., 2016) suggesting that cognitive decline in later life may not be a foregone conclusion, but there is some debate in the typical ageing literature (see Ramscar et al., 2014: and see Rabbit, 2016). For instance, age-related improvements are observed in verbal fluency (Park et al., 1996), crystallized intelligence and language into older age, with only slight declines observed in these domains after age 70-80 years (Salthouse & Saklofske, 2010; Anderson & Craik, 2017; and see Ramscar et al., 2014). This patterning of abilities and difficulties in older age highlights the distinctive contributions of specific cognitive functions and the potential interplay between them.

An assumption of neurocognitive theories of age-related cognitive changes is that changes in the functional connectivity between frontal, parietal and temporal brain regions may be associated with impairments in the domains just listed (Dempster, 1992; Salthouse, Atkinson & Berish, 2003; Cappelletti et al., 2015). Furthermore, the risk of onset of dementia, including Alzheimer's disease (AD), increases dramatically with older age (American Psychiatric Association, APA, 2011). The various forms of dementia are associated with fronto-temporal lobe dysfunction and are thought to exacerbate the effect of age-related cognitive decline, with an estimated half of all adults aged 85

years and older suffering from AD (Beckett & Taylor, 2010; Bishop, Lu & Yanker, 2010; APA, 2011). Age-related changes in the domains of memory, EF and fluid intelligence affect psychological functioning more broadly, including the ability to perform more demanding activities such as employment, planning skills, spatial orientation and navigation, independent living including life management, taking one's medication and even remembering a doctor's appointment or someone's name (McDaniel & Einstein, 2000). In this respect, age-related cognitive decline can also affect social interaction, independence and QoL (Crook et al., 1986; Salthouse et al., 2003; Salthouse, 2004; Hedden & Gabrieli, 2004).

In sum, the findings from many studies in the typical ageing literature, converge with this view that cognitive ageing can be observed after 50 years of age with decline in processing speed (Salthouse, 1996); attention (McCabe et al., 2010), metacognitive ability (Mäntylä et al., 2010), EF (Friedman & Miyake, 2017), memory (Craik & Byrd, 1982; Rabbitt, 2016) and general intellectual ability (Salthouse, 2004; Anderson & Craik, 2017; but see Hedden & Gabrieli, 2004 for review of lifespan changes and stability). These changes can have far-reaching and devastating effects on an individual's general functioning and independence, leading to social isolation and poorer QoL (Crook et al., 1986; Beckett & Taylor, 2010; Woods, 2015).

What is more, neurocognitive research involving the study of the interaction between the changes in the nervous system and changes in brain regions may provide clues to the cognitive mechanisms that underlie how individuals learn and process information, and the impairments in cognitive functioning within certain clinical groups including those with brain trauma or developmental disorders (Dempster, 1992; Cappelletti et al., 2015; but see Rabbitt, 2017). For instance, frontal brain regions (associated with EF) and the hippocampus and related structures of the medial temporal lobe (associated with memory) have been shown to decline in volume and functional connectivity with increasing age (e.g. Raz et al., 1998) – a picture that corresponds to the brain profiles of patients with amnesic and dementia pathologies (Hedden & Gabrieli, 2004).

As described earlier in this chapter, a similar profile of difficulties in EF, memory and atypical functional connectivity between associated brain regions has been shown in autistic individuals at various developmental stages (Mottron & Burack, 2001; Just et al., 2012; Boucher, Mayes & Bigham,

2012). These parallels suggest that younger autistic individuals may present as prematurely cognitively old (Bowler, 2007; Boucher & Bowler, 2008) and draw attention to important considerations of how ageing might affect older autistic adults (Happé & Charlton, 2011; Mukaetova-Ladinska et al., 2012). As we have seen, the evolution of the classification and resulting theories of autism has informed research developments of the past four decades. This has led to increasing awareness about transitions between childhood and adolescence into young adulthood, and the difficulties as well as strengths associated with the behavioural features (social and non-social) and cognitive differences (non-social) in ASD (Hill, 2004; Williams, Goldstein & Minshew, 2006). Much less is known about the way in which ageing affects cognitive functions such as memory, EF, prospective memory, language and intellectual ability and how these factors affect the QoL of autistic individuals (Happé & Charlton, 2011; Howlin et al., 2015). Together with the evidence just mentioned **about** age-related brain changes, these findings point to the potential risks and increased difficulties for autistic individuals in the context of ageing. For example, do autistic individuals experience the same path of age-related cognitive decline as healthy ageing individuals? Does ageing with autism present a more profound and severe effect on cognitive functioning in older autistic adults? (Geurts and Vissers, 2012). Insights from the few longitudinal and cross-sectional studies with older autistic adults highlight some of these issues and provide directions for the work that is still needed.

The strength of longitudinal studies is to assess within an individual over a period of time, accounting for developmental, biological (e.g. sensory, brain structure and volume) and social factors that influence the individual's lifespan. Thus, the value of longitudinal studies lies in their ability to provide a measure of *change* in performance or ability, which includes gains as well as losses, from one time point to another time point, through re-evaluation of a given function (Anderson & Craik, 2017). The downside of longitudinal evaluations being that societal changes in themselves may influence a person's ability from one time to another. Furthermore, repeated exposure to the same assessments may result in learning or practice effects which may "mask [actual] age-related declines" (Salthouse, 2010; Kliegl, Smith, & Baltes, 1989; Salthouse, 2004). By contrast, cross-sectional studies facilitate the measurement of age-related *differences* between age groups or cohorts, such as

comparing the ability of an individual who is 20 years old and one who is 70 years old. However, such differences may be mere artefacts (Schaie, 2005) of the between-person variability, such as maturational changes (Salthouse, 2010), or external factors that influence cognitive differences, such as educational attainment, life experience and motivation towards different goals (Schaie, 2005). For instance, a typically developing younger adult may be more motivated to learning and achieving, gaining meaningful employment or accumulating wealth, social status and environmental security. Whereas, older typical adults may be more motivated towards a slower pace and self-reflection. That said, these factors are important considerations for both cross-sectional and longitudinal methodologies. Accordingly, longitudinal observations of autistic adults should include the cumulative effect of co-existing physical and mental health conditions and the long-term effects of early life cognitive difficulties on later life outcomes (Kats et al., 2013; Waterhouse, 2013). Important contributions may be made by cross-sectional studies that provide insights into the current age-related cognitive and behavioural patterning of younger and older autistic adults, compared to typically ageing adults of the same age and general level of intellectual ability. Such studies serve to identify the potential risk factors associated with age-related differences and what this means for autistic adults in the context of ageing (Roestorf & Bowler, 2016; and see Lever & Geurts, 2016; Ring, Gaigg & Bowler, 2016).

Emerging insights about ageing and ASD

Kanner (1943; 1971) highlighted the need for continued investigation of long-term outcomes. Subsequently, few longitudinal studies have assessed the outcomes of autistic children into adulthood (Howlin et al., 2004; 2013; 2014; Lounds Taylor & Mailick Seltzer, 2010; Shattuck, Abbeduto & Greenberg, 2004; Shattuck et al., 2007; Woodman, Smith, Greenberg & Mailick, 2015; Taylor, Henninger & Mailick, 2015; Klinger, M.R. Bagatell, Meyer, Brooks & Klinger, L.G., 2017; and see Wise, Smith & Rabins, 2018). Adult outcomes are variable with respect to autonomy, self-care, social relationships, education and employment – some studies report stable diagnostic prognosis (Klinger et al., 2017) or modest improvements in autistic traits over time (Seltzer, Shattuck, Abbeduto & Greenberg, 2004; Shattuck et al., 2007; Taylor & Seltzer, 2010; Howlin et al., 2013; Woodman et al.,

2015; Taylor et al., 2015). Cognitive functions and general intellectual ability appear to stabilise over childhood and adolescence into adulthood, whilst modest but significant improvements in language skills are observed for the majority of individuals (Mawhood & Howlin, 2000; Howlin et al., 2014; Ratto & Mesibov, 2015) although functional language and adult outcomes remain poor (see Magiati, Wei Tay & Howlin, 2014; but see also Farley et al., 2009). However, these improvements may be mediated by general intellectual ability and co-occurring intellectual disability (ID; Shattuck et al., 2007; Taylor & Seltzer, 2010). Moreover, outcomes related to these factors may be mediated by higher order cognitive processes, such as EF (Lawson et al., 2015). Together, language ability, general intellectual ability outside the range of intellectual disability (ID), and the severity of autistic traits in early childhood, are strong predictors of adult outcomes in ASD (Shattuck et al., 2007; Taylor & Seltzer, 2010; Howlin et al., 2013; Ratto & Mesibov, 2015).

Nevertheless, few autistic individuals can live independently and require continued social care to maintain their QoL (Howlin et al., 2013; Parr, 2016; D'Astous et al., 2016; Mazurek et al., 2018). Only a small minority of individuals (12%) are reported to have 'very good' outcomes in relation to social relationships, educational attainment, employment and level of autonomy (Billstedt et al., 2007; Anderson et al., 2014; Fein et al., 2013; Orinstein et al., 2014); Shattuck et al., 2007; Magiati et al., 2014; Ratto & Mesibov, 2015). Whereas, others remain severely impaired in cognitive and social functions (e.g., Howlin et al., 2013; Howlin et al., 2017). Support from family members would unavoidably disappear as parent caregivers themselves approach old age and end of life (Howlin et al., 2015), presenting specific challenges for autistic individuals as they grow older, and which may be exacerbated for individuals who are not able to live independently. What is more, the majority of autistic individuals face persistent mental health difficulties and poor QoL compared to typically ageing adults (Howlin et al., 2004; Hofvander et al., 2009; Howlin & Moss, 2012; Ratto & Mesibov, 2015). As many as 84% of autistic individuals are reported to have long-term mental health difficulties associated with one or more complex psychiatric conditions (Hofvander et al. 2009; Howlin & Moss, 2012; Kats et al., 2013). However, because very little research has included older autistic adults, much less is known about how the process of ageing affects these outcomes and the need for long-term care. For instance, social integration and being able to work in meaningful

ongoing employment has been linked to greater QoL, and lower reported rates of anxiety and depression (Klinger et al., 2015; Van Heijst & Geurts, 2015; Lever & Geurts, 2016a; McConachie et al., 2017; but see Hong et al., 2016). The need for continued follow-up studies of autistic children into adulthood and older age is emphasized by recent reports that have documented the variable trajectories of autistic adults (e.g., Anderson et al., 2014; Henninger & Taylor, 2013; Howlin et al., 2013; Levy & Perry, 2011; Magiati, Wei Tay, & Howlin, 2012).

Recent cross-sectional studies with older autistic adults present a more mixed picture. Whereas, some studies highlight that memory difficulties in ASD persist into older age (Van Heijst and Geurts 2014; Ring, Gaigg & Bowler, 2016), others report a lack of differences compared to TA adults (Lever & Geurts, 2016). However, there does appear to be convergence in the patterning of age-related differences in ASD adults compared to TA adults (Lever et al., 2015; Ring et al., 2016). For instance, whilst autistic adults present difficulties in EF, episodic memory, WM (Altgassen et al. 2012; Geurts & Vissers, 2012; Lever et al., 2015), and forming associations between items, item-contexts, sequential order and spatial locations (Ring et al., 2016), they do not present the same ageing-related challenges in increased memory difficulties. As older TA adults tend to show incremental difficulties with increasing age (Roestorf & Bowler, 2016; Anderson & Craik, 2017). These differences have, to date, not been explained in ASD ageing trajectories by autistic traits, co-existing conditions or EF (Geurts & Vissers, 2012; Lever et al., 2015). Geurts and Vissers (2012) proposed three possible cognitive outcomes for the patterning of age-related differences observed in older ASD compared to older TA adults:

- “safeguarding” – fewer cognitive difficulties are observed in ASD with increasing age;
- “double jeopardy” – steeper age-related cognitive declines are observed in ASD;
- “parallel ageing” – following similar trajectories in ASD to typically ageing adults (see Figure 2.1, p. 65).

In the research presented in this thesis, a fourth possibility is proposed: that autistic individuals develop cognitive coping mechanisms across their lifespan, having to deal with adversity and the challenges of functioning in a neurotypical world. Accordingly, a lifetime of cognitive coping may serve to support the cognitive capabilities of autistic people in later life. Thus, older autistic

individuals may present more behaviours that appear to demonstrate cognitive resilience compared to younger ASD adults, and potentially also compared to older TA adults.

In relation to QoL, previous cross-sectional studies have highlighted significantly poorer QoL for autistic adults across the lifespan (Totsika et al., 2010; van Heijst & Geurts, 2015) and across QoL domains, namely: Physical QoL (e.g. health difficulties, medication dependence; daily living skills; work capacity); Psychological QoL (e.g. self-esteem; body image; positive/negative mood and thoughts; concentration, attention and memory); Social QoL (e.g. personal relationships; intimacy, social supports); and Environmental QoL (e.g. finances, living environment; access to and quality of health and social care) (Ayres, Parr, Rodgers et al., 2017; McConachie et al., 2017; but see Hong et al., 2016; QoL domains according to World Health Organisation (WHO), 2000). Moreover, factors such as degree of autistic traits (Khanna et al., 2014; Van Heijst and Geurts, 2015) and ability to self-reflect (Saldaña et al. 2009; Hong et al., 2016) may play a vital role in determining QoL outcomes for autistic adults as they grow older. Hong and colleagues (2016) compared self- and maternal reports of QoL ratings, showing consistency between those ratings and above-average QoL across all domains. However, their study also highlighted two crucial factors in lifespan QoL outcomes. More than half the autistic adults had experienced frequent bullying and increased perceived everyday stress. Both these factors negatively affected QoL across domains. Whilst good health predicted better Psychological QoL, recurring or frequent past experiences of bullying were predictors of poor quality of health across all domains (Hong et al., 2016; Bishop-Fitzpatrick et al., 2015; and see Kamp-Becker, Schroder, Remschmidt & Bachmann, 2010). More research is needed to understand the determinants of QoL for older autistic adults, and the changes that occur across the lifespan into older age. Further, the magnitude of effect and the direct of causality between cognitive difficulties and QoL in ASD are still unknown (Howlin et al., 2015).

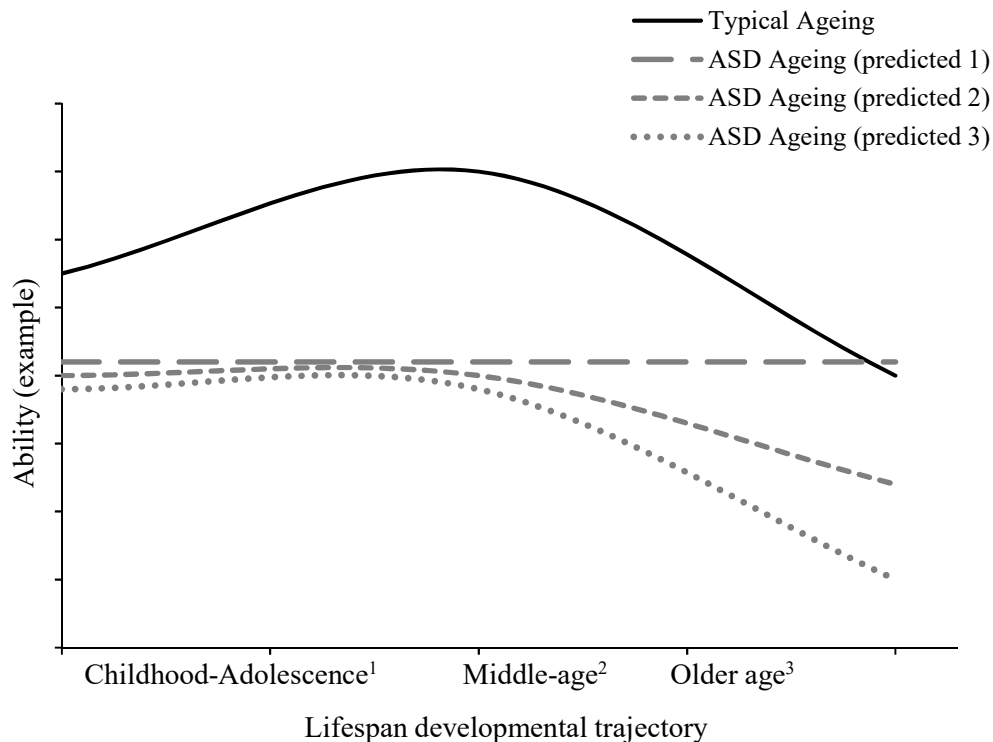


Figure 2.1. Predicted ageing trajectories in typical development and autism spectrum disorder.

Notes: Based on the literature of typical development and ASD in early life, the graph depicts that profile of estimated lifespan trajectories across ¹Childhood-Adolescence (< 18 years of age), ²Middle-age (<50 years of age) and ³Older age (≥65 years of age). Typical Ageing: as referred to earlier in this chapter, the lifespan trajectory for cognitive abilities begins to decline in middle-age and more steeply into older age (e.g. McCabe et al., 2010; Salthouse & Saklofske, 2010; Anderson & Craik, 2017). ASD Ageing: Based on the 'Ageing analogy' by Bowler and colleagues (Bowler et al. 2004; Bowler, 2007) and the subsequent hypothesis of Geurts & Vissers (2012), at least three possible outcomes are predicted for autistic adults as they grow older. 'Predicted 1' refers to stable trajectory (e.g. Howlin et al. 2004; 2013; Shattuck et al., 2007; Lever & Geurts, 2016a; Roestorf & Bowler, 2016; Ring et al., 2015; 2016); 'Predicted 2' refers to parallel age-related cognitive decline (e.g. Geurts & Vissers, 2012; Lever et al., 2015; Lever & Geurts, 2016a); 'Predicted 3' refers to steeper age-related cognitive decline (e.g. Howlin et al., 2013; Powell, Klinger & Klinger, 2017).

What is yet unknown about ageing and ASD

The discussion throughout this chapter has highlighted the similarities between the age-related cognitive changes in typical ageing and the patterning of cognitive difficulties and strengths in younger autistic individuals. These parallels have emphasised the need for a lifespan developmental approach to understand the long-term impacts on cognitive functions and QoL as autistic individuals grow older. The co-occurrence of one or more physical or mental health conditions are common in at least 50-84% of autistic individuals (Hofvander et al. 2009; Lounds & Mailick, 2010; Hirvikoski et al., 2016; but see Lever & Geurts, 2016b) but little is known about the health and social support services available for older autistic adults who may need continued support related to autistic traits,

co-occurring mental health difficulties or daily living skills (Seltzer et al., 2004; Nicolaidis et al., 2014; Hirvikoski et al., 2016; Fortuna et al., 2016; and see Wright et al., 2016). The cumulative effect of long-term psychiatric co-existing conditions on cognitive functions and QoL is largely unknown (Howlin & Moss, 2012; Kats et al., 2013; Howlin et al., 2015). Furthermore, the likelihood of pharmacological treatments for co-existing conditions may further affect cognitive difficulties, such as memory (Joss, Burton, & Keller, 2003).

Howlin et al.'s (2004; 2013; 2014) longitudinal work highlights important considerations of ageing in ASD: approximately two-thirds of participants in their longitudinal follow-up (approximately 20 years later) were still able to take part in the research and perform basic cognitive assessments. However, a significant sub-group of approximately 25% of individuals diagnosed in childhood, were not able to undergo the basic cognitive assessments. Overall, that research showed significantly improved language ability and social skills, but adult outcomes were nonetheless 'poor' (27%) to 'very poor' (33%) for most the autistic adults at follow-up, even for those with greater cognitive ability. For instance, the majority of adults (72%) did not obtain formal education qualifications (high school or above) and most were either unemployed (55%) or in sheltered/voluntary work (15%); few autistic adults had close friendship (24%) or long-term intimate relationships (17%), and only 13% lived autonomously without the need for support in daily living.

What is still unknown is the magnitude of age-related change that occurs in autistic adults over the age of 50 years (Wise, Smith & Rabins, 2017). Is there a steeper risk of cognitive decline with ageing? Does ageing with autism mirror the trajectories of cognitive change seen in typical ageing? Does the cognitive profile of autistic adults stay the same as they grow older? How do potential age-related cognitive changes and cognitive difficulties in prospective memory affect the QoL of autistic adults? The work reported in the forthcoming chapters will address the questions of the associations between cognitive functions and behavioural features of ASD and the developmental changes that occur across the lifespan of an autistic individual. Furthermore, assessments of multiple aspects of EF, memory (e.g. episodic, prospective), language and general intellectual ability are needed, to explore the magnitude of difficulties (or strengths) associated with ageing in ASD. The work presented here

directly addresses these factors. These issues are addressed in the studies presented in Chapters 4, 5 and 7, and revisited in the General Discussion presented in Chapter 8.

2.4 Programme of work: methodology, research questions and hypotheses

The programme of work presented in this thesis sets out to document the magnitude and patterning of age-related differences cognitive function and QoL of autistic adults compared to typically ageing adults, who were matched age and general intellectual ability.

The overall aims for this programme of research, are to:

- (a) Assess the extent and magnitude of cognitive differences in ASD, for older adults compared with younger adults;
- (b) Explore the specific relation of cognitive functions, including the range of EF, memory and language skills, to QoL in older age and ASD;
- (c) Establish an understanding of the relation between ASD symptoms and co-existing conditions across the lifespan, and the effect on QoL in older age;
- (d) Assess the trajectory and predictors of cognitive change in ASD, for older adults compared with younger adults, and compared to younger and older typically ageing adults;
- (e) Explore the nature and extent of prospective memory abilities in ASD and its role in supporting QoL of older autistic adults.

The chapters that follow present a multifaceted series of studies to directly address the aims set out above, and to gain a better understanding of how the process of ageing affects autistic adults across the lifespan. The predictions are based on what happens in typical ageing as well on the patterning of difficulties observed in younger autistic individuals that is similar to older typically ageing individuals (Bowler et al., 2004). Thus, it would be expected that some aspects of cognitive functioning (e.g. memory, EF) would be impaired in older age in the ASD adults. Whether this decline is steeper or shallower compared to the TA adults (Geurts & Vissers, 2012) is an open question, as is the patterning of the profile of any decline.

Study 1 sets out the T1 assessment (Chapter 4) which explores the patterning of age-related *differences* in autistic traits, cognitive functions, mental health and QoL. If the patterning of difficulties seen in younger autistic individuals persists into older age, then more pronounced age-related effects might be expected in ASD, in a patterning that is at least parallel to that seen in typical ageing. Whereas, if these difficulties stabilise or abate into older age, then a lesser effect on cognitive ageing in ASD would be expected compared to TA adults. The same sample was followed-up after 2.5 years, at the T2 assessment (Chapter 5), to evaluate longitudinal *changes* in autistic traits, cognitive functions, mental health and QoL. Here, the cross-sectional *differences* were explored at T2, and the longitudinal assessment of *change* was explored by comparing T1-T2 outcomes and predictors of cognitive and QoL outcomes. Based on the literature already discussed, it was predicted that ASD groups would present with greater physical and mental health conditions than TA groups, and that those difficulties would persist into older age for autistic adults. QoL was expected to be poorer overall for autistic adults, persisting into older age when followed up longitudinally. Chapter 6 presents a review of the prospective memory (PM) literature – a strong predictor of cognitive outcomes and QoL in typical ageing. Then, Chapter 7 sets out a collection of experimental tasks in Study 3, which explores PM for event-based and time-based actions, in laboratory and naturalistic contexts. Although we know nothing about how prospective memory affects older autistic adults, if the patterning of prospective memory difficulties is consistent with the profile reported by a small number of studies in younger autistic adults, then it would be expected that these difficulties would also present difficulties for older autistic individuals. Whether prospective memory predicts QoL in ASD is unknown since this issue has not been addressed in previous literature. These issues are explored in Chapter 7. Then, in conclusion, Chapter 8 set out the General Discussion of convergent findings from the series of studies presented in this thesis and addresses the possible implications of these findings for lifelong support needs of older autistic adults.

Ascertainment and continuity of participation across studies

Although there is an estimated increase in the number of autistic adults over age 65 years (Piven & Rabins, 2011; Kats et al., 2013), recent reports suggest there is also an increased risk of

early mortality, around 50 years of age, particularly where co-existing complex health conditions are a factor (Hirvikoski et al., 2016). The emerging research, presented earlier on ASD and ageing, highlights that previous work has not sufficiently included older adults over the age of 50 years (Wise, Smith & Rabins, 2018) and has been limited to narrow age ranges, resulting in a gap in understanding about how to define older age when referring to autism (Roestorf et al., in press). Moreover, evidence from the typical ageing literature converges with this view, demonstrating that even in typically developed individuals, the effects of ageing are observed after 50 years of age. Specifically, declines in processing speed (Salthouse, 1996), attention (McCabe et al., 2010), metacognitive ability (Mäntylä et al., 2010), EF (Friedman & Miyake, 2017), episodic memory (Craik & Byrd, 1982; Anderson & Craik, 2017), prospective memory (Mäntylä et al., 2013; Mäntylä et al., 2010) and general intellectual ability (Schaie, 2005; Salthouse, 2010; Anderson & Craik, 2017) are commonly observed in older typically ageing adults. For these reasons, in the programme of work presented here, comparisons are made between younger adults (aged 18- 49 years 11 months), and older adults (aged 50 years and older). Participants included autistic adults who had been diagnosed early in life, as well as those who received later life diagnosis of ASD. The comparison groups were typically developed adults with no personal or family history of ASD. Participants were invited to take part in several assessments during the course of the research (detailed in Chapters 4, 5, and 7). In consideration of the factors just mentioned, when conducting studies of older autistic adults, a degree of attrition was expected across all studies, since the programme of work extended across 4 years. Where possible, several attempts were made to retain contact with participants across the programme of work and the reasons for participants' discontinuation were recorded where available. These included: chronic or terminal illness, or death (ASD, n=5; TA, n=2); lost to follow-up, or moved to different town, city or country (ASD, n=4; TA, n=6); work commitments (ASD, n=1; TA, n=5); personal difficulties or family commitments (ASD, n=5; TA, n=4); self-exclusion or withdrawal from project for other reasons (ASD, n=3; TA, n=1); administrative reasons (ASD, n=3; TA, n=2). A summary of the participant overlap across studies is presented in Table 2.3, and a schematic of continuity is presented in Figure 2.2 (p. 71).

Table 2.3. Sample composition and continuity (percentage retention) across all studies

Participants in each study	Study 1: T1^a	Study 2: T2^b	Study 3: PM^c
Total	87	62 (71%)	57 (66%)
Autistic (ASD)	52	39 (75%)	35 (67%)
Younger	26	19 (73%)	18 (69%)
Older	26	20 (77%)	17 (65%)
Typically ageing (TA)	35	23 (66%)	22 (63%)
Younger	16	11 (69%)	10 (63%)
Older	19	12 (63%)	12 (63%)

Notes: Sample continuity in Study 2 (T2) and Study 3 (PM) are reported in relation to the sample recruited at Study 1 (T1).

^aT1: Time 1 cross-sectional study of age-related differences within Age Groups (younger; older) and Diagnostic Groups (ASD; TA).

^bT2: Time 2 longitudinal study of age-related changes within Age Groups and Diagnostic Groups. Sample continuity is reported in relation to the T1 sample.

^cPM: Prospective Memory study of event-based and time-based ability in laboratory and naturalistic contexts.

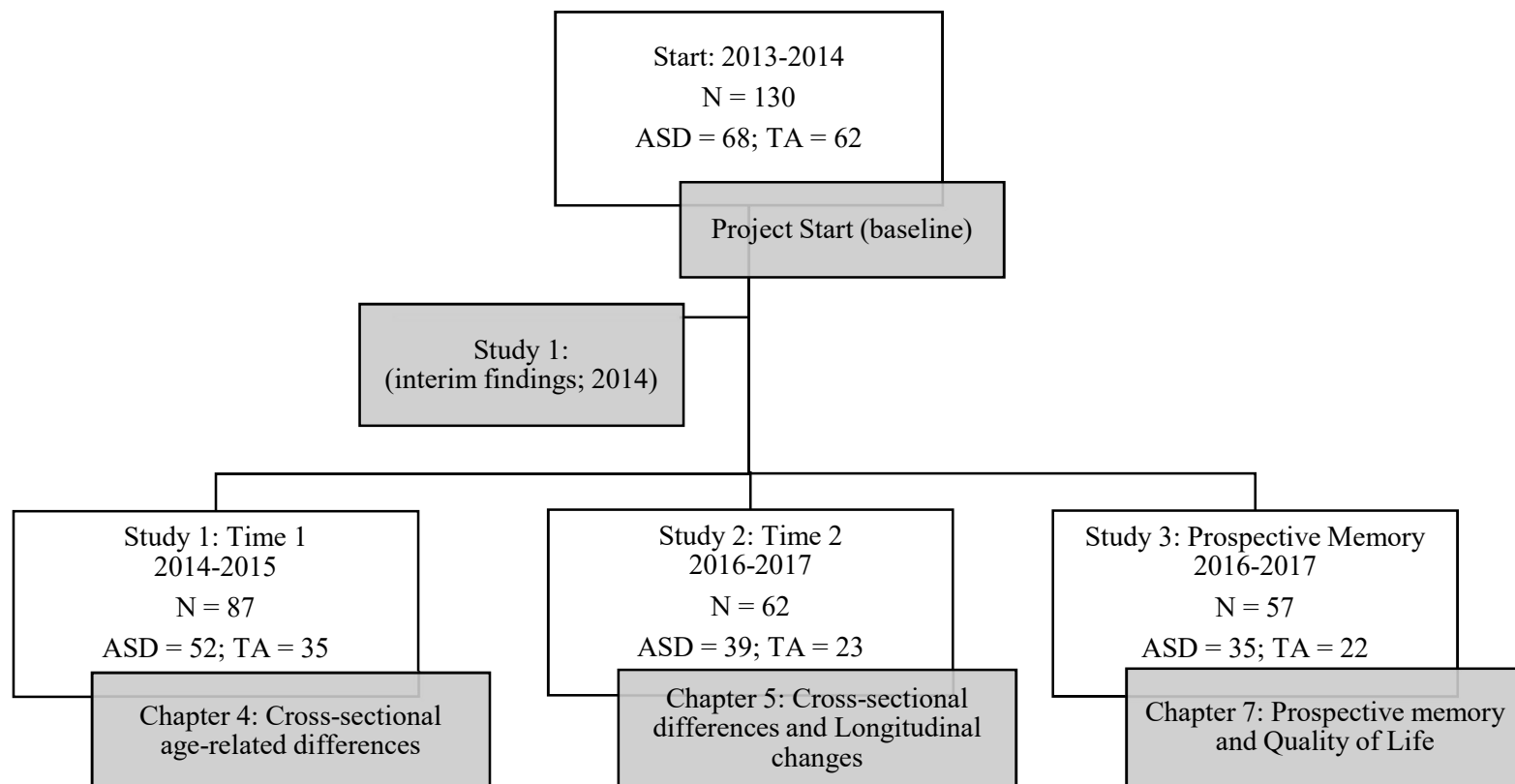


Figure 2.2. Sample continuity across all studies within the programme of research.

Next, Chapter 3 sets out the issues relevant to how assessments of cognitive functions, mental health and QoL are explored in the context of ageing in ASD. That is followed by Study 1 (Chapter 4), the T1 assessment, which addresses the questions of whether early life difficulties in ASD persist in younger and older adults, how the profile of ageing in ASD is different from that seen in typical ageing, and what this means for the QoL of autistic adults as they grow older.

Chapter 3: Conceptualisation of cognitive functions

The previous chapter set out a broad review of the profile of functioning in autistic individuals from longitudinal studies across childhood and into adulthood, and emerging discoveries from cross-sectional studies of older autistic adults (Gerhardt and Lanier; 2011; Smith, Maenner & Seltzer, 2012; Shattuck et al., 2012; Nicholas et al., 2017; Klinger, M.R. Bagatell, Meyer, Brooks & Klinger, L.G., 2017). A small number of studies have shown generally stable trajectories between childhood and adulthood, regarding language and general intellectual ability. Regarding autistic traits, persistent difficulties associated with the core features of ASD are reported to be associated with poor adult outcomes and poor QoL (see Magiati, Wei Tay & Howlin, 2014 for review). Those studies provide valuable insights about the developmental transitions between childhood and adulthood, although these are often limited to younger samples and seldom reflect outcomes beyond middle age. More recently, there has been a growing number of cross-sectional studies with older autistic adults (over 50 years of age) that report a similar profile of functioning and highlight specific challenges associated with EF, mental health and QoL (Totsika et al., 2010; Geurts & Vissers, 2012; van Heijst & Geurts, 2015; Lever & Geurts, 2016; Wang et al., 2017).

One of the limitations of the research to date is that it has, with a few exceptions, tended to focus on a narrow subset of cognitive measures within a given domain. This narrow focus has then been extrapolated to attempt to explain the broad cognitive profiles of autistic individuals. In order to remove any concerns about the impact of extrapolation, the work presented in Studies 1 and 2 (Chapters 4 and 5) of this thesis have included a more comprehensive set of measures on which the findings can be based. To provide the context for the measures that have been selected in these studies, it is helpful first to understand how each of the domains that they measure have been conceptualised to date.

3.1 Conceptualisation of cognitive functions: intellectual ability, language and memory

In the typical ageing literature, there is much variability in age of onset and degree of age-related cognitive decline (Salthouse, 2004; Schaie, 2005; Salthouse, 2010; Ramscar et al., 2014; Anderson & Craik, 2017). Nevertheless, there is convergence on three common domains that are associated with cognitive change in typical ageing (Anderson & Craik, 2017; Rabbit, 2016). These are memory, EF and language and their independent and collective effects on general intellectual ability and QoL (Salthouse et al., 2004; Rabbit, 2016). As briefly outlined in Chapter 2, the measurement of cognitive abilities such as EF is, in part, determined by the conceptualisation of the cognitive process(es) that are being measured. For instance, EF is a collection of separate but inter-related higher-order cognitive processes that regulate lower-order goal-directed behaviours, thoughts or actions, which includes distinct yet inter-related processes for core EF components (cognitive flexibility, WM, inhibition) and subcomponent processes (e.g. planning, fluency, attention). Similarly, other higher-order cognitive functions, such as general intellectual ability, language and memory are defined both in terms of broader conceptualisations of multidimensional constructs and distinct underlying cognitive processes that serve their specific functions. The challenge with assessing these functions in ASD is that, like research in typical ageing (TA), studies tend to conflate the measurement of these cognitive functions through the lens of a specific assessment e.g. visual WM. However, as with EF, the underlying mechanisms associated with general intellectual ability, language and memory may be linked with other cognitive processes and functions, and with each other.

Domains of cognitive functioning

(i) General intellectual ability and language

The concepts of intellectual ability and language will be discussed concurrently as these two features of cognition are often measured together in the context of intelligence (IQ) and as both aspects are particularly important in the context of cognitive outcomes in ASD (Magiati, Wei Tay &

Howlin, 2014; and see Howlin, 1997; Howlin et al., 2014). General intelligence is a multifaceted cognitive function and comprises both (i) the development of perceptual information processing, discrimination, reasoning and memory (fluid intelligence) through the maturation of the brain's development across childhood and adolescence, and (ii) acquired knowledge and wisdom through experience, episodic learning, cultural and social influences (crystallised intelligence; Cattell, 1963; Baltes, 1993; Schaie, 2003). Learned skills such as reading, writing, language comprehension and academic ability are forms of crystallised intelligence, as are self-knowledge and social skills (Baltes, 1993). Measures of IQ assess both fluid intelligence (aptitude) and crystallised intelligence (acquired knowledge), more broadly, through nonverbal (e.g. perceptual reasoning) and verbal (e.g. language and verbal comprehension) abilities encompassing a range of separate but inter-related tasks. These verbal and nonverbal domains include subtests of, for example, WM, processing speed, general knowledge, arithmetic, visual search, fluency and memory recall as subordinate, but related, cognitive functions. Together, verbal and nonverbal domains inform a composite score of general intellectual ability (GA) and full-scale IQ (FSIQ), depending on the subtest assessments that have been included in each domain. The Wechsler Intelligence Scales and its variants (Wechsler, 2008, 2010) is a widely used, standardised measure of IQ which assesses these multidimensional aspects of verbal and nonverbal IQ in children and adults, across a range of ages, cultures and, recently, neurodevelopmental and clinical conditions (Wechsler, 2010). Normative IQ scores are derived for population means (mean 100, standard deviation 15), providing a method of measuring an individual's IQ relative to a sample of age-range matched individuals in the population, with incremental age adjustments for older adults.

There is considerable overlap and inter-dependency between these features of intelligence, just outlined, across the lifespan, although they have somewhat different developmental trajectories in typical ageing and in ASD. In typical development, fluid intelligence and its underlying mechanisms continue to develop throughout adolescence and young adulthood, peak in middle-age and then decline across older age, from around 60 years of age (Schaie, 1996, 2003). Whereas, the pragmatics of crystallized intelligence tend to peak in early ageing, around 50 years of age, and are relatively stable until around 80 years of age when the first signs of age-related declines may appear (Schaie,

2003; and see Baltes, 1993; but see Hedden & Gabrielli, 2004; and see Anderson & Craik, 2017).

Declines in both fluid and crystallised intelligence with increasing older age (>80 years) may be amplified by associated age-related declines in sensory processing ability (e.g. vision, hearing, motor function) and atrophy of brain regions (Hedden & Gabrielli, 2004) and their associated neurocognitive processes such as verbal learning, information processing and memory (Park et al., 2002; Rabbitt, Lunn & Wong, 2008; Anderson & Craik, 2017; and see Schaie, 2003).

In ASD, cognitive profiles are more heterogenous and uneven in terms of verbal and nonverbal abilities (Bölte et al., 2009; Ankenman et al., 2014) which has substantial implications for outcomes across the lifespan (Howlin, 1997; Howlin & Moss, 2012; Howlin et al., 2014). The variability in autistic IQ profiles appears to be determined by a number of factors, including overall IQ with or without intellectual disability (ID), language development, age of testing, and the specific measures of IQ that are used to assess either verbal or non-verbal IQ (or both) and the respective cognitive functions that these tests tap into. Consequently, indicators of the direction of IQ strengths and difficulties in ASD are equally varied. Some research suggests enhanced nonverbal IQ associated with perceptual reasoning, compared with verbal IQ and language comprehension (e.g. Shah & Frith, 1989; Ozonoff et al., 2004; Minshew et al., 2006; Dawson et al., 2007; Bölte et al., 2009), whilst the opposite profile is presented in other research indicating better verbal IQ over nonverbal IQ (Minshew et al., 2005; Williams et al., 2008; but see Bölte et al., 2009; and see Ankenman et al., 2014 for review of verbal-nonverbal differences). Overall, language and childhood IQ in ASD are robust predictors of adult outcomes (Howlin, 1997, pp. 15-58; Howlin & Moss, 2012; and see Magiati et al., 2014). However, adult outcomes are variable even for individuals with average range IQ (70-130) – few individuals able to function autonomously in adulthood, and these difficulties are more pronounced for individuals with co-occurring ID (IQ <70, Howlin et al., 2014; IQ <85, Bölte et al., 2009). Further, ID appears to be associated with more uneven cognitive profiles in ASD and greater discrepancies between verbal and nonverbal abilities, difficulties in social functioning (Bölte et al., 2009; Ankenman et al., 2014) and more pronounced maladaptive behaviours such as aggression and self-injury (Howlin et al., 2014).

The development of language and verbal ability matures and changes across the lifespan, becoming, generally, more elaborate and fluent as the individual grows and matures. Language is arguably a crucial aspect of general intellectual ability and may underlie other cognitive functions such as memory and EF. Verbal ability and language comprehension are required to process information, interpret task instructions and respond to contextually specific content (Ramscar et al., 2014; Williams, Goldstein & Minshew 2006). Effective communication, therefore, involves not only the ability to comprehend and interpret information (receptive language), but also the ability to convey meaning and intention (expressive language). Across the autism spectrum, language profiles are qualitatively and quantitatively different in autistic individuals compared to age- and ability-matched non-autistic or typically developed individuals. Difficulties with complex language (Williams et al., 2006) and diverse profiles in receptive and expressive language abilities present difficulties for social communication (Howlin et al., 2014). Whereas, autistic individuals may have strongly developed vocabulary and complex speech, considerably difficulties with language comprehension, narrative interpretation, verbal fluency (EF), understanding metaphors and irony, and understanding nonverbal aspects of language (e.g. body language, facial expressions) are observed in autistic children and adults (Minshew, Goldstein & Siegel, 1997; Williams et al., 2006). At a behavioural level, verbal language in autistic individuals may be echolalic, overly formal or technical, with irregular rhythm, pitch or intonation (e.g. Howlin, 1997, pp. 15-58). The patterning of language difficulties does not correspond to overall intellectual abilities in ASD, since most comparative studies are between age- and ability-matched groups with equivalent verbal and nonverbal IQs (e.g. Minshew et al., 1997; Landa & Goldberg, 2005; Williams et al., 2006). Further, language may be an important mediator of cognitive processes involved in metacognition for episodic past and future thinking (e.g. Crane, Lind & Bowler, 2013) and theory of mind (Charlton et al., 2009; Happé, 1994; but see Boucher, 2012; and see Tager-Flusberg, 2000; Lind & Bowler, 2009), thus being of consequence for social communication, imagination and affective functioning (Hobson, 2002, pp. 83.94). Some research has suggested that the heterogenous patterning between language/verbal ability and cognitive functions in autistic individuals may be associated with using “compensatory verbal

strategies to succeed on cognitive experimental tasks” (Williams, Jarrold, Grainger & Lind, 2014, p. 40; and see Lind & Bowler, 2009; Wang et al., 2017).

(ii) Memory

Memory is a complex, multidimensional higher-order cognitive function that enables an individual to use information from knowledge and experience to distinguish between experiences and previously encountered information, guide behaviour and responses to present contexts, inform representations of the world, and establish meaning about personal experiences (Craig, 2000; Bowler, Gaigg & Lind, 2010). Memory is one of the core cognitive domains that is associated with age-related cognitive decline (Park et al., 1996; Park, 2000; Craig, 2000; Salthouse, 2014, 2016) which has implications for broader cognitive functioning and QoL. The construct of memory comprises several inter-related and subordinate features and processes that are, in turn, related to several other cognitive mechanisms, such as general intellectual ability, language, EF, information processing and metacognition and learning (Perner, Kloo & Stöttinger, 2007; Mäntylä et al., 2010; Rubin et al., 2014; Salthouse, 2016; and see Boucher, Mayes & Bigham, 2012). There are several accomplished texts on the theories and models underlying the study of memory. Among these, Montaldi & Mayes (2010), Boucher et al. (2012) and Gardiner (2008) each provide comprehensive reviews and relatively recent accounts of the underlying mechanisms of memory and theoretical constructs, respectively, as well as the implications for learning and everyday functioning. Craig (2000) provides a summary of the features of memory affected by cognitive ageing. The main interest in the present chapter is the challenges associated with memory difficulties in later life (Park et al., 1996; Park, 2000; Craig, 2000; Salthouse, 2014, 2016) and the implications for autistic adults as they grow older. In this section, a brief summary of typical age-related changes in memory processes will be provided, followed by the few studies of memory in adult ASD samples as a preface to the exploration that follows in Study 1 (Chapter 4).

(i) Memory processes

Theoretical approaches have endeavoured to explain memory as a network of systems (implicit, nondeclarative; explicit, declarative), processes (encoding, storage, updating, retrieval) and stores (working⁴, short-term, long-term). Declarative memory is defined as the conscious recollection of information from personal experiences, whilst nondeclarative memory involves more automatic or implicit processes that support the use of deeply rooted learned skills, information, or conditioning. An example of declarative/explicit memory is episodic or autobiographical memory about one's personal experiences; nondeclarative/implicit memory includes procedural memory involving, for instance, motor skills, spelling and reading (Craik, 2000, p.77). The features and processes of memory just described are associated with various brain regions, most prominently the medial temporal lobe structures including the hippocampus and related structures (Craik, 2000; and see Montaldi & Mayes, 2010 for a comprehensive discussion), and functions associated with the frontal lobes (see Rubin et al., 2014; and see Ben Shalom, 2003). Accordingly, memory involves the integration of information from present or past memory, discrimination of relevant or irrelevant information, and goal-oriented behaviours that are applied to changing contextual demands (Tulving, 1985; Salthouse, 2009; Rubin et al., 2014). Thus, it is clear that memory, broadly, involves other cognitive functions such as general intellectual ability, language (described earlier in this chapter), and EF (summarised below; and see Chapter 2 for theoretical discussion). The development and retention of memory across the lifespan has reciprocal effects on learning and metacognitive ability (Boucher et al., 2012), whilst processing speed, attention and EF may be particularly important in accessing information from memory (retrieval) in older age (Perner, Kloo & Stöttinger, 2007; and see Baddeley, 1997, pp. 336-338).

(ii) Memory in typical ageing

Whilst it is generally accepted that memory is broadly susceptible to age-related declines, self-reports of everyday memory difficulties do not appear to correspond to actual difficulties in

⁴ As mentioned in the previous chapter (Chapter 2), WM is primarily a subcomponent of EF, involving monitoring and updating of information (Friedman & Miyake, 2017; but see Baddeley & Hitch, 1974). WM is thought to be influenced by information processing capacity (Salthouse, 1996; Park et al., 1996) and attention (Baddeley, 1993, 1997).

remembering but may instead be mediated by a combination of brain changes, and process changes in cognitive mechanisms e.g. EF for shifting between goals, tasks and mental states (e.g. Salthouse et al. 2003; Mäntylä et al., 2010). The disparity between self-reports and actual cognitive performance suggests that self-awareness and metacognitive ability in later life may play a role in the interplay between EF and memory in older age (Perner et al., 2007; Rubin et al., 2014). The patterning and extent of age-related decline depends on the type of memory function, and whether declarative/explicit or nondeclarative/implicit processes are involved (Craik, 2000). The evidence from cross-sectional and longitudinal studies of TA adults provides a mixed profile of age-related cognitive function in older age. Some aspects of memory appear to steadily decline across adulthood into older age (e.g. WM; encoding of new episodic memory; Park, 1996; Schaie, 1996, 2003), whilst other aspects of memory are stable until older age with only slight declines (e.g. short-term; semantic; autobiographical; recognition and implicit memory; Schaie, 2003; Hedden & Gabrieli, 2004). By contrast, nondeclarative/implicit memory (including procedural memory) is generally protected from age-related declines (Craik, 2000). As mentioned earlier, what appears to create a distinction between preserved or eroded memory functions is the underlying mechanisms that are either automatic bottom-up, or effortful top-down processes – automatic processes are thought to withstand ageing effects to a greater extent than strategic effortful processing that places greater demands on competing cognitive resources for ongoing monitoring and maintenance of information (Craik, 1986, 2000; Tulving, 1989; McDaniel & Einstein, 2000; Salthouse, 2010). Consequently, functional and structural changes in brain regions associated with memory may be exacerbated by co-occurring pathologies in older age (Salthouse, 2010; and see Rabbitt et al., 2008). Moreover, difficulties with episodic memory in older age has potentially profound effects on everyday functioning, autonomy, social functioning and QoL (Schaie, 2005; Beckett & Taylor, 2010, pp.180-181; Toffalini et al., 2016).

(iii) Memory in ASD ageing

In the discussions presented so far it is evident that memory has a complex and important role in everyday functioning in typical ageing. Further, throughout this thesis we have seen that the specific cognitive profiles in ASD is affected by a range of separate yet inter-related mechanisms. The

profile of memory in ASD presents yet another complex and uneven profile of strengths and difficulties that affect the way in which autistic individuals engage with their environment. It is generally acknowledged that autistic individuals have some form of memory difficulties, although the degree and complexity of these vary between and within individuals across the autism spectrum (Boucher & Bowler, 2008; Bowler, Gaigg & Lind, 2010; Boucher et al., 2012). In contrast to the age-related difficulties that are observed in typical ageing (Park et al., 1996; Schaie, 1996, 2003; Anderson & Craik, 2017), explicit memory for items is not affected by older age differences in ASD, as reported in cross-sectional studies of younger and older adults (Bowler et al., 2007; Bowler et al., 2009; Geurts & Vissers, 2012; Lever & Geurts, 2015; but see Powell et al., 2017). Further, it would appear that language and intellectual ability may mediate memory in ASD, as do metacognitive ability and self-representation in supporting recollection of episodic detail (Bowler, Gardiner & Grice, 2000; Bowler et al., 2014; and see Kana et al., 2017). However, few studies have systematically explored the interplay between cognitive functions and the breadth of memory within the same autistic individuals across the lifespan (see Minshew & Goldstein, 2001; and see Williams et al., 2006). Further, in the context of ageing and ASD, the few studies with older autistic adults show equivocal patterns of verbal and visual memory across adulthood (Bowler et al., 2009; Geurts & Vissers, 2012; Powell et al., 2017; and see Lever & Geurts, 2015). A potential interpretation of the literature presented in this section is that autistic individuals employ compensatory strategies that draw on other cognitive processes to complete tasks with greater cognitive loads (Just et al., 2004; Williams et al., 2006). This view, in itself, presents a potentially problematic outlook for older adults, given the vulnerability of cognitive capacity for information processing in typical ageing (Schaie, 2003; Salthouse, 2004, 2016). This important consideration leads to the question of whether memory in ASD systematically declines or is preserved in older age, and the potential impacts of memory difficulties on everyday functioning and QoL.

Early studies suggested that ASD was an amnesic disorder underpinned by hippocampal dysfunction (e.g. Boucher & Warrington, 1976, amongst others), revised accounts have proposed that this specific profile of memory difficulties in ASD are associated with both hippocampal and frontal lobe structure and functional processes (Ben Shalom, 2003; Boucher et al., 2012; and see Kana et al.,

2012). Declarative/explicit memory in ASD is not uniformly affected. Semantic memory for specific factual information is, generally, preserved or enhanced in autistic individuals (Gaigg et al., 2014), whereas, episodic and explicit (declarative) memory is selectively impaired (Williams et al., 2006; Boucher & Bowler, 2008; and see Boucher et al., 2012; Gaigg et al., 2014). This patterning of memory function led Bowler and colleagues (Bowler et al. 2007) to propose the ‘ageing analogy’ – the similarity between the memory function of younger autistic individuals resembles the cognitively old profile of memory impairments seen in TA adults. For instance, autistic adults perform as well as TA adults on immediate recall of non-social and unrelated semantic information, show excellent memory for details (Williams et al., 2006), and self-generated (free) recall of semantic information in pre-defined categories (Crane, Lind & Bowler, 2013; Gaigg, Bowler & Gardiner, 2014). By contrast, the most substantial memory difficulties in ASD are associated with autobiographical and episodic memory. For instance, contextual memory is impaired for self-generated episodic recollection (Bowler et al., 2007; Gaigg et al., 2014; Gaigg, Bowler, Ecker et al., 2015); autobiographical memory and episodic thought is qualitatively impoverished of content (Crane & Goddard, 2008; Crane, Goddard, & Pring, 2009; Lind & Bowler, 2010; Lind, Williams, Bowler & Peel, 2014), compared to TA individuals. A possible explanation for the patterning of memory strength and difficulties is that semantic memory is generally free of contextual binding or associations, whereas episodic memory involves metacognition, self-representation, and effortful recollection of contextual information and personal experiences (Craik, 2000). These processes are, as described in Chapter 2, domains in which autistic individuals have substantial difficulties, to greater or lesser degrees, across the autism spectrum.

Where WM was considered in the context of memory function, a mixed patterning of intact and impaired WM has been observed, for example, visual and spatial WM is widely reported to present difficulties for autistic individuals (Minshew & Goldstein, 2001), whilst verbal WM does not (Kenworthy, 2008; Williams et al., 2014). However, several studies in adult ASD have reported difficulties in nonverbal short-term memory (Bowler et al., 2016) and verbal short-term memory and WM span for the number of items freely recalled (i.e. without cues or prompts; Poirier, Martin, Gaigg & Bowler, 2011; Geurts & Vissers, 2012; Powell et al., 2017; but see Lever et al., 2015). ASD adults

have higher error rates in falsely producing items and greater ordering difficulties (Minshew & Goldstein, 2001; Martin, Poirier, Bowler & Gaigg, 2006), particularly when tasks become more complex and cognitive load is increased (Minshew & Goldstein, 2001; Just et al., 2004; Powell et al., 2017; but see Lever et al., 2015). Overall, increasing cognitive load adversely affects response accuracy in both TA and ASD participants; whilst group differences in accuracy are not necessarily observed in older age (Minshew et al., 1992; Minshew & Goldstein, 2001), ASD adults tend to be significantly slower in their responses than TA adults, suggesting information processing difficulties, as described in Chapter 2. Further, since WM is a subcomponent of EF involving monitoring and updating of information (Baddeley & Hitch, 1974; Friedman & Miyake, 2017), mediated by information processing (Salthouse, 1996; Park et al., 1996) and attention processes (Baddeley, 1993, 1997), difficulties with these associated processes would potentially amplify any memory difficulties in ASD.

Minshew and colleagues assessed the broad cognitive profile of ASD and TD adults (see Minshew et al., 1992; Minshew & Goldstein, 2001; and Williams et al., 2006 for procedures). Using comprehensive test batteries of language, EF and tests of verbal and visual memory, Minshew et al. (1992; 2001) assessed ‘simple’ and ‘complex’ cognitive functions in each domain. The key differentiators between simple and complex memory were demands on lower-order or higher-order cognitive processes and self-directed retrieval of information and cue-referencing for successful completion, respectively. ASD participants performed as well as TD participants on tests of simple memory involving short-term and paired associate learning, visual and verbal WM (as well as formal language use, e.g. spelling, grammar; and simple task switching). However, on tasks of complex memory and, therefore, higher cognitive demands, ASD participants showed greater difficulties and more perseverative errors with increasing complexity (Minshew et al., 1992). Additionally, these difficulties included narrative comprehension, fluency and some difficulties in verbal or visual (perceptual) reasoning associated with cognitive flexibility (Minshew et al., 2001; Williams et al., 2006). The authors suggest that this patterning of strengths and difficulties for lower-order and higher-order functions, respectively, do not reflect a pervasive memory difficulty, per se, but rather point to a disordered information processing and impoverished encoding of information. Similarly,

Bowler, Gaigg and colleagues suggest that autistic individuals have difficulties ordering and organisation and binding of information at encoding, which presents difficulties for later retrieval (Gaigg, Gardiner & Bowler, 2008; Gaigg et al., 2015). Indeed, several studies have shown that autistic adults have difficulties with relational encoding and retrieval of visual (Bowler, Gaigg & Gardiner, 2014; Ring, Gaigg & Bowler, 2016), and verbal information (Minshew & Goldstein, 2001; Williams et al., 2006) is also impaired relative to other cognitive functions and compared to age- and ability-matched TD individuals.

Gaigg and colleagues (2015) suggest that organisational strategies during encoding and retrieval may be affected in ASD. In a study of relational memory processing, whereby individuals were required to implicitly form associations between items and their perceptual features and locations. Increased activation of the prefrontal cortex was observed during encoding of relational information for later recognition memory tests, whereas, hippocampal activity was observed much later in the retrieval process (Gaigg et al., 2015). Similar to the patterning described earlier, whereby older TA adults employ other cognitive processes, such as EF, to mediate memory difficulties in older age (Salthouse et al., 2003; Perner et al., 2007; Rubin et al., 2014), it would appear that autistic adults similarly draw on frontal lobe processes for more strategic and effortful encoding of information, possibly as compensatory mechanisms for the uneven patterns of memory abilities and difficulties. The profile of neural dysconnectivity just described (and see Chapter 2) has been further posited as a possible explanation for the patterning of episodic memory difficulties in ASD (Boucher, Mayes & Bigham, 2012). If, as the evidence suggests, the organisation at encoding and subsequent retrieval of information is disordered at a neural level, this would, in turn mediate the effective use of strategies for memory and other higher-order cognitive abilities, such as EF, problem solving and comprehension. Consequently, disordered organisation in memory and broader cognitive functions has potentially adverse effects on language comprehension and learning, since it would facilitate fewer opportunities for individuals to meaningfully interpret the environment and learn from prior experiences (Gaigg, 2012; and see Pellicano & Burr, 2012). This would pose specific challenges for autistic individuals across the spectrum in terms of social communication and everyday functioning. Further support of this view comes from neuroimaging studies that highlight wide-ranging cognitive

differences between autistic and TA adults, with increasing cognitive demands presenting specific challenges for the ways in which autistic individuals processed information. Whereas, the autistic and TA individuals achieved the same task success, the neuroimaging patterns in ASD showed decreased activation and underconnectivity of the memory and EF networks and reduced coordination between all brain regions, especially when cognitive load was increased (Just et al., 2004; Kana et al., 2006). However, some research has shown that autistic individuals benefit from structured ‘task supports’ (Bowler, Matthews & Gardiner, 1997; and see Bowler, Gardiner & Berthollier, 2004 for a description of the Task Support Hypothesis), that provide external cues to support the retrieval of previously encountered information. This finding suggests that compensatory strategies may facilitate not only memory function in ASD but may facilitate everyday memory and autonomous function in daily living skills. Further, given the patterning of selective memory difficulties in ASD presented above, tasks that place fewer cognitive demands, allow extended processing of information and present simpler information structures should facilitate improved memory in ASD adults (Minshew & Goldstein, 2001).

Subsequent studies with more cognitively able and older autistic individuals have also found that memory difficulties are not pervasive in ASD but, nonetheless, present atypical patterns of strengths and difficulties (Minshew et al., 1992; Williams et al., 2006; Bowler, Gaigg & Gardiner, 2008, 2010, 2014; Bowler, Limoges & Mottron, 2009; Geurts & Vissers, 2012; Lever & Geurts, 2015; Powell, Klinger & Klinger, 2017). In a cross-sectional study of older adults (aged 51-83 years), Geurts & Vissers (2012) found that, when matched on IQ, older ASD adults performed as well as older TA adults on tasks of visual and verbal memory, and that increasing age adversely affected performance in both groups (Geurts & Vissers, 2012). These findings hold important consequences in ASD and ageing. Given the converging evidence of selective memory difficulties in younger autistic individuals, further difficulties with increasing age would have potentially greater impacts across the adult lifespan (Powell et al., 2017).

(iii) Executive function

As we have seen in Chapter 2, a substantial body of research has provided evidence for EF impairments in ASD, across the lifespan (Ozonoff et al., 2004; Happé et al., 2006; Pellicano et al., 2006; Pellicano, 2010; Pellicano, 2013; Geurts & Vissers, 2012; Lever & Geurts, 2016; Powell, Klinger & Klinger, 2017). And the literature presented in this chapter reveals that higher-order cognitive processes, such as general intellectual ability, language, memory and EF, are inter-related and draw on underlying cognitive mechanisms that may be jointly mediated by disorder of information processing (Minshew, Sweeney & Luna, 2002; Kana, Keller, Minshew & Just, 2006).

Studies of TA individuals show EF, broadly, follows an inverted u-shaped trajectory between childhood and old age (Zelazo, Craik & Booth, 2004). EF difficulties in later life, in turn, affect other cognitive abilities. For instance, WM and inhibition impairments in young and middle-aged adults have been associated with poorer metamemory and greater source memory errors (Mäntylä et al., 2010). In autistic individuals, an uneven profile of difficulties and abilities is observed in several subdomains of EF, and these difficulties may be mediated by cognitive load, the interdependency on other EF components and task complexity across the lifespan (Geurts, Corbett & Solomon, 2009; Kercood et al., 2014). For instance, several studies report evidence for broad EF impairments in cognitive flexibility, working memory, planning and attention (Hughes et al., 1994; Geurts et al., 2004; Pellicano et al., 2006; but see Robinson et al., 2009; van Eylen et al., 2011; Lever & Geurts, 2015; Powell, Klinger & Klinger, 2017), but not inhibition, fluency or processing speed (Ozonoff & Strayer, 1997; Robinson et al., 2009; Geurts & Vissers, 2012). These difficulties have been shown to increase between childhood and adolescence (Rosenthal et al., (2013), and persist over time (Ozonoff & McEvoy, 1994). Whereas, WM as an EF component appears to remain intact across childhood (Ozonoff & Strayer, 2001; Geurts et al., 2004) and adulthood (aged 20-79 years) with little evidence of increased age-related difficulties (Lever et al., 2015; Wang et al., 2017). Further, age-related improvements are seen in inhibition of prepotent responses and cognitive flexibility (Geurts et al., 2014), although planning difficulties persist at least through adolescence (Van Den Bergh, Scheeren, Begeer, Koot & Geurts, 2014). However, a different picture emerges when task type and cognitive load are varied, with more demanding tasks producing more pronounced difficulties across the

lifespan (Wang et al., 2017). In tasks that involve simple and complex manipulations (i.e. fewer or more actions, respectively), autistic adults show corresponding ability to typically developed adults in simple tasks of planning, cognitive flexibility and inhibition (Hughes et al., 1994; South, Ozonoff & McMahon, 2007; Powell, Klinger & Klinger, 2017), but show greater difficulties with inhibition of prepotent responses (Robinson et al., 2009; Geurts et al., 2014), and complex planning and flexibility tasks (Kercood et al., 2014), even compared to individuals with other developmental disorders or intellectual disability (Ozonoff, Pennington & Rogers 1991; Hughes et al., 1994; Russel et al., 2003; Geurts et al., 2004). A recent study with older autistic adults shows intact verbal and visual memory across ages, whereas difficulties with verbal fluency and semantic memory are more pronounced with increasing age (Lever & Geurts, 2016).

As we have seen from the evidence presented so far, memory and EF are crucial for everyday cognitive function and may impact on cognitive functioning more broadly, with reciprocal involvement of language and general intellectual ability across the lifespan. With these cognitive considerations in mind, we turn to aspects of psychological well-being associated with mental health and QoL, given that long-term mental health difficulties, such as depression, are known to correspond to difficulties in memory, attention, EF and processing speed (McClintock, Husain, Greer, & Cullum, 2010).

3.2 Well-being: Physical and Mental health and Quality of Life

There is an increasing drive for awareness and support of older adults in the general population, related to cognitive change, well-being, social integration, physical healthcare and support needs, and dementia risk (Wright et al., 2016; Diaz-Moore et al., 2014). Studies in gerontology provide insights to the selective challenges of ageing, and the successful strategies that enable older adults to age well in maintaining cognitive function (Salthouse et al., 2004; Ramscar et al., 2014), and social integration to support better QoL (Wilson et al., 2010; World Health Organization [WHO], 2002). The World Health Organisation (2002), in their report on ‘Active Ageing’ suggest that 60 years of age should be a marker of “older” adulthood, but caution that “chronological age is not a precise marker for the

changes that accompany ageing. There are dramatic variations in health status, participation and levels of independence among older people of the same age” (WHO, 2002).

Physical and Mental health

The literature reviewed in the previous chapter highlighted that the co-occurrence of one or more physical or mental health conditions is common in at least 50% of autistic individuals (Hirvikoski et al., 2016; and see Lever & Geurts, 2016b). Thus, it is important to consider the impacts of physical and mental health difficulties in the context of cognitive functions and QoL, since poor mental health can have adverse effects on cognitive abilities such as memory and EF, and long-term adverse effects on QoL (van Heijst & Geurts, 2015; Lever & Geurts, 2015). Moreover, abilities associated with social integration and being able to work in meaningful ongoing employment have been linked to greater QoL, and lower reported rates of anxiety and depression (Klinger et al., 2015; Van Heijst & Geurts, 2015; Lever & Geurts, 2016a; McConachie et al., 2017; but see Hong et al., 2016).

(i) Depression

Depression is the catch-all term that is commonly used to describe disorders of mood or affect that encompass a pervasive dysphoria or anhedonia (lack of pleasure), feeling of low mood and or sadness that lasts for 2 weeks or more (Frank, 2011), and may present as a single episode, long-term event, or recurring episodes leading to major depressive disorder (MDD). The lifetime prevalence of depression is reported to be 15%, with as approximately two-thirds (70%) of individuals experiencing recurring episodes across their lifespan (Swaine, 2011). The effects of depression are far-reaching in terms of negative cognitive, social, psychological and QoL impacts, increased disability and earlier mortality than non-depressed individuals (Moussavi et al., 2007; McClintock, Husain, Greer & Cullum, 2010; Khanna et al., 2014), which may be exacerbated in individuals with co-occurring intellectual disability and/or neurodevelopmental disorders including ASD (Coppus, 2013; Ratto & Mesibov, 2015).

Although some ambiguity exists in the literature regarding the specific mechanisms associated with cognitive difficulties in depressed individuals, there is a growing evidence in the literature to suggest negative associations between depression and EF involved in planning, information processing, automatic and directed attention and working memory, as well as diminished ability in verbal fluency, verbal learning and declarative (episodic) memory (McClintock et al., 2010). The cognitive underpinnings of these deficits are thought to be linked with the front-temporal regions, given that MDD has been linked with the prefrontal cortex which is also known to be associated with EF (Tulving, 1985; West, 1996), and the hippocampus and surrounding structures in the medial-temporal lobe (Elliot, Rubinsztein, Sahakian & Dolan, 2002) which are thought to be involved in memory processes associated with encoding and retrieval of information over short and long durations (Eichenbaum, 1999; Montaldi & Mayes, 2010).

McClintock, Husain, Greer, & Cullum (2010) conducted a meta-analysis of 35 studies that have evaluated the effects of depression on cognitive functioning, between 1991 and 2007. In their review, McClintock and colleagues (2010) report greater cognitive difficulties in depressed individuals compared to non-depressed individuals from the general population, where performance was measured greater than 1 standard deviation difference on test outcome scores (across various measures). For instance, individuals with MDD performed significantly worse on IQ subtests involving working memory (Digit Span, Digit Symbol subtests on WAIS-R; Wechsler, 1981; e.g. Paradiso et al., 1997; Fossati et al., 1999; McBride & Abelese, 2000; Stordal et al., 2004) and higher scores on depression questionnaires were associated with increased perseverative errors in EF tests of planning, rule learning, inhibition and task-switching (Wisconsin Card Sorting Test; WCST; e.g. Martin, Oren & Boon, 1991; Grant, Thase & Sweeney, 2001; Naismith et al., 2003). Moreover, long-term mental health difficulties, such as depression, are known to increase the risk of neurocognitive disorders (i.e. dementia; Bauman, 2010); whether this risk is increased for individuals with ASD is unknown (Hategan, Bourgeois & Goldberg, 2017).

(ii) Anxiety

Stress and anxiety-related difficulties have substantive implications for autistic individuals, in terms of social functioning, cognitive ability and adaptive behaviours (Wallace et al., 2016; Maisel et al., 2016; South et al., 2017). The association between the clinical features of anxiety and the clinical features of ASD is a complex one, with considerable overlap (Rodgers et al., 2016) leading to potentially confounding profiles of autism-related difficulties (South et al., 2017) related to restricted interests and repetitive behaviours, and to social communication difficulties (Constantino & Gruber, 2012). Recent studies suggest that anxiety in ASD may be increased as a result of intolerance to uncertainty, coupled with emotional aversiveness and difficulties identifying and interpret their emotions (Maisel et al., 2016). Furthermore, anxiety and depression are thought to be potential mediators of maladaptive behaviours in ASD, as well as metacognition difficulties and cognitive inflexibility (Lawson et al., 2015; Wallace et al., 2016), resulting in poorer adult outcomes (Mazurek, 2014). Consequently, anxiety and depression may underlie internalising behaviours, which in turn affect higher order EF and behavioural self-regulation (Wallace et al., 2016).

The approach to supports for anxiety-related conditions requires careful consideration for autistic adults across the lifespan. Whilst auxiliary healthcare is important for the management and long-term support needs associated with co-existing conditions, such as anxiety and depression, it remains important for clinicians to dissociate the core symptoms associated with ASD from mental health conditions and physical health and to address the primary care needs in ASD.

Quality of life and subjective well-being

As already mentioned, there has been little systematic research into the impacts of ageing on outcomes for older autistic adults, whether diagnosed in childhood or later life (Perkins & Berkman, 2012; Damiano, Mazefsky et al., 2016; Charlton, 2017). What is known suggests a poorer QoL in older autistic adults (Totsika et al., 2010; van Heijst & Geurts, 2015; Ayres, Parr, Rodgers et al., 2017), and greater co-occurring health conditions and mental health needs than their age-matched peers (Hirvikoski et al., 2016; Fortuna et al., 2015; Happé, Mansour, Barrett et al., 2016; but see Lever & Geurts, 2016a), and do not have access to appropriate services in adulthood and across their

lifespan (Wright et al, 2016). Quality of life (QoL) and Subjective well-being (SWB) are affected by several features such as physical, psychological, environmental and social. The respective measures of QoL and SWB are discussed in the method section of Chapter 4.

Chapter 4: Study 1

Cross-sectional age-related differences in autistic traits, cognitive function and well-being

In the present chapter, Study 1 captures the profile of ageing in ASD through information gathering about older and younger autistic adults in relation to typically ageing (TA) adults. Given the limited knowledge around the effects of growing older with ASD, the objectives for this study were primarily explorative. A broad array of assessments at Time 1 (T1) established the starting point for the whole programme of work. The descriptive findings and possible interpretation of the cognitive profiles of younger and older autistic and TA adults are discussed later in this chapter.

4.1 Study Aims

The conceptual issues discussed in Chapters 2 and 3 outlined the need to understand the extent of age-related differences and the potential for increased magnitude of challenges for older autistic adults. In particular, such an evaluation would need to apply multidimensional exploration of cognitive features, mental and physical health and QoL to ascertain the effects of growing older in ASD.

Study 1, reported here, represents the first time-point (Time 1) assessment of the domains just mentioned. Given the limited knowledge around the effects of ageing in ASD, the objectives for this study were primarily explorative. The T1 assessments catalogued diagnostic history and autistic traits, physical and mental health, cognitive evaluations of general intellectual functioning, memory, language and EF, and measures of QoL and overall well-being.

The key research questions addressed in the present study were:

- (a) What is the extent and magnitude of cognitive differences in ASD, for older adults compared with younger adults, and how does this differ from typical ageing?
- (b) What is the specific profile of cognitive functions in older ASD adults, including EF, memory and language, and their relation to QoL?
- (c) What is the relation between the clinical features of ASD and co-existing conditions across the lifespan, and their effects on QoL?

4.2 Ethics

Ethical approval was obtained from the Psychology Department Research Ethics Committee PSYETH(UPTD) 13/14 28, for the research project titled: *Age-Related Effects on Cognition and Quality of Life in Adults with Autism Spectrum Disorder*.

Prior to enrolment in the study, a brief information leaflet was provided to individuals who were interested in participating, entitled: *What is involved?*. (see Appendix-X1). The information sheet containing details about the assessments involved in the study, consent form and screening questionnaire were sent to participants who registered their interest in taking part, prior to scheduling the first appointment. Participants were informed that the study would be conducted across multiple sessions (3-4 sessions, lasting approximately 3 hours each) across the 4-year programme of work. On arrival at the first appointment, the participant was verbally briefed about the assessments involved, and informed consent was verified prior to collecting background information and commencing the first assessments, described in the Measures section below.

4.3 Method

Procedure

Participants were screened for years of formal education (YFE) and English language proficiency, and for smoking, drug and alcohol use (all self-report). Diagnostic history for autistic participants required a confirmed diagnosis of ASD confirmed by clinical report. A pre-enrolment questionnaire was administered to TA participants to screen for no personal or family history of ASD

or other neuropsychological conditions. In the first research appointment, background medical and other diagnostic history was obtained, including co-occurring physical and mental health conditions, medication use and sensory sensitivities. TA participants were not screened out on this basis, given the increased incidence of co-occurring conditions in older age, it was intended that the sample were as representative of typical ageing as possible. Diagnostic Groups were matched on chronological age, Gender ratio, YFE (Figure 4.3, p. 123) and general intellectual ability (IQ).

On average, to complete the overall assessment for this study required three 3-hour sessions. If participants required more frequent comfort breaks, an additional fourth appointment was made. Where possible, the successive appointments were scheduled 2-4 weeks apart, allowing for participant availability. The standard protocol for the series of appointments was as follows:

- Appointment 1: informed consent; establish rapport; obtain background information (PIAS); IQ test (WAIS), and self-report questionnaires of autistic traits² (AQ), anxiety (BAI) and depression (BDI).
- Appointment 2: self-report questionnaires about physical and mental health (PHQ); Language test (CREVT-3); QoL (WHOQOL) or subjective well-being (PWI)¹; administered computerised EF tests (CANTAB)³.
- Appointment 3: Memory test (CVLT-II); measure of severity of autistic traits² (SRS-2); subjective well-being (PWI) or QoL (WHOQOL)¹; remaining EF tests (CANTAB)³.
- Appointment 4: ADOS (autistic participants only, where no recent ADOS (≤ 2 years) was available).

Notes:

¹ Since the PWI and WHOQOL both measures aspects of QoL and well-being across similar domains, these forms were administered in separate sessions, the order was counter-balanced to avoid order effects and matched by Diagnostic Group i.e. if the order for Participant 1 (ASD younger female) was PWI-WHOQOL, then the counterpart Participant 2 (TA younger female) also received PWI-WHOQOL, and so forth.

² Autistic traits measures were administered in separate sessions to avoid response bias and contamination.

³ The battery of EF tests was extended to include subtests that evaluated specific planning and strategy, and task switching abilities. These were administered in a separate session, since the first battery was already very long and demanding, thereby avoiding test fatigue.

Participants: T1 sample characteristics

One-hundred thirty participants were included in the Time 1 study (ASD n=68, mean age 44.07 (SD 15.48 years); TA n=62, mean age 47.29 (SD 16.64 years)); approximately evenly sampled from Younger (n=67, mean age 32.17 (SD 8.67 years) and Older (n=63, mean age 59.89 (SD 7.40 years) groups. Some participants were not able to complete all assessments, on account of ill health or personal difficulties (ASD) or owing to work or family commitments (TA; and see Figure 2.2 in Chapter 2, p. 71). The final sample reported here consists of 87 participants who were able to complete all or most of the assessments for the Time 1 study. The sample characteristics for these data are set out in Table 4.3a (p. 126) for Diagnostic Group comparisons, and in Table 4.3b (p. 127) for comparisons between Diagnostic Groups within Age Groups. This is followed by the data for cognitive (Tables 4.4a to 4.4c, pp. 136-140), and mental health, physical health and QoL (Tables 4.6a and 4.6b, pp. 148-149). Analysis of these data show that the Time 1 sample included 52 adults with a diagnosis of ASD and 35 typically ageing (TA) adults. Diagnostic Groups were matched on chronological age (18-80 years; mean 46.97 years, standard deviation (SD) 14.91 years). There were 25 females (21%) in the total sample (ASD n=11; TA n=14; see Table 4.3a, p. 126), who distributed approximately evenly between younger and older Age Groups (see Table 4.3b, p. 127).

4.4 Measures

Chapter 3 reviewed the complexities involved in assessing broad cognitive functions within a given domain, such as memory or executive function. Given those complexities, and the specific difficulties or strengths observed in previous ASD literature, careful consideration was given to the selection of appropriate measures in Study 1 and Study 2 (for Study 3 measures, see Chapter 7). Where possible, measures were selected that had been reliably used previous in ASD studies to demonstrate profiles of cognitive differences (e.g. CVLT). This was not possible, however, for many of the assessments of age-related difficulties in ASD, given the limited prior work in this area. Here, the typical ageing literature was consulted for widely used measures, with robust reliability and validity and, where possible, specificity. A summary of the multiple standardised measures that were administered during assessments is provided in Table 4.0 below.

Table 4.0. Measures administered at T1 and T2

Measure	Purpose and Outcome measures	Method	Age range	Reliability ¹	Validity ²	Specificity ³
ADOS-2, Module 4 ^a	Diagnostic profile for Autism ($A \geq 10$) or Autism Spectrum ($AS > 7$) as measured by Total score. Scale scores: Communication ($A \geq 3$; $AS \geq 2$), Social Interaction ($A \geq 6$; $AS \geq 4$), Restricted Interests and Repetitive Behaviours (RRB; $A \geq 2$; $AS \geq 1$), Imagination and Creativity ($A \geq 2$; $AS \geq 1$).	Administered semi-structured interview and participant enacted tasks. Observer-rated, double-coded where possible. Scoring guidelines and cut-off scores provided. Not timed but usually administered ≤ 60 minutes.	18+	.82	.74	.82
AQ ^b	Screening instrument for autistic traits. / Total score (0-50); SI (0-10); Attention Switching (0-10); Attention to Detail (0-10); Communication (0-10); and Imagination (1-10). Cut-off scores ≥ 26 are suggested (Woodbury-Smith et al., 2005).	50-item questionnaire, self-rated on a scale of 1 (definitely agree) to 4 (definitely disagree). Administrator scored and reverse coded items. Not timed but usually completed ≤ 10 mins.	17+	.70	.63-.77	.83
SRS-2 ^c	Diagnostic profile for Autism Spectrum (DSM-5). Subscales indicate degree of autistic traits with Total and T-Index scores (all 30-90): Social Communication Index (SCI); Restricted Interests and Repetitive Behaviours (RRB); Social Awareness (Awr); Social Cognition (Cog.); Social Motivation (Mot.).	65-item self-report questionnaire rated on a scale of 1 (definitely agree) to 4 (definitely disagree). Administrator scored, reverse coding on some items. Not timed.	19-99	.88-.95	.92	.94
WAIS-III / WAIS-IV ^d	General Intellectual Functioning and IQ index scores are calculated for: Full-scale IQ (FSIQ); Verbal Comprehension (VCI); Perceptual Reasoning (PRI); Working Memory (WMI); Processing Speed (PSI). Mean score 100 (SD 15). Standardised age-norms are provided for stratified age groups.	Administered structured questions and timed participant enacted tasks on 10 sub-tests. Observer-rated. Some sub-tests are timed.	16-90	.93-.97	.70	.65-.96
CREVT-3 ^e	General Vocabulary, Receptive Language and Expressive Language / Respective outcome index scores; descriptive level of language; significant differences between Receptive and Expressive language. Mean score 100 (SD 15). Standardised age-norms are provided for stratified age groups with age-level skill equivalents to 18 years.	Administered item-based identification of words through participant provided definitions (expressive) or picture-based word matching (receptive). Observer-rated. Not timed.	5-90	.97-.99	.80-.92	1 SD
CVLT ^f	Verbal learning and episodic memory / Learning Slope; Free- and Cued-Recall (short and long delays); Recognition memory; Source memory Discriminability (d'); Recall consistency (organization). Standardised age-norms are provided.	Administered 16-item list of words (4x4 semantic categories). Learning and memory assessed over short and long delays with either no prompts (free-recall) or category prompts (cued recall). Observer-rated. Not timed.	16-89	.82-.83	.66-.94	.69-.87

Measure	Purpose and Outcome measures	Method	Age range	Reliability ¹	Validity ²	Specificity ³
CANTAB ^g	Executive Function / Working Memory; Inhibition; Cognitive flexibility; Planning; Episodic Learning & Memory. Standardised age-norms are provided.	Administered computerized tasks from 10 sub-tests. Computer-coded to individual performance level across all task stages.	4-90	.87	.77	.73
PHQ ^h	Clinically-derived scores which align with DSM-IV-TR criteria for mental health conditions, such as depression and anxiety.	11-questions, Self-report measure rated on binary (yes/no) or degree of difficulty or frequency (e.g. not at all – very often). Administrator scored.	18+	.88	.85	.90
BAI-II ⁱ	Physical symptoms of anxiety, not explained by biological causes. Items represent related symptoms of subjective experiences (e.g. “nervous”), neurophysiology (e.g. “heart pounding or racing”), autonomic (e.g. “face flushed”), and panic (e.g. “fear of worst happening”).	21-item, Self-report questionnaire rated on a scale of 0 (not at all) to 3 (all the time), based on symptoms experienced during the past 1 month. Researcher-derived scores for Total and Levels of Anxiety.	13-80	.92	.94	.78-.90
BDI-II ^j	Physical and psychological symptoms associated with depression, including rumination, appetite, sleep disturbance and suicidal ideation. Researcher-derived scores for Total and Levels of Depression.	21-item, Self-report questionnaire rated on a scale of 0 (not at all) to 3 (all the time), based on symptoms experienced during the past 2 weeks.	13-80	.93	.92	.91
PWI-A ^k	Subjective well-being including religion-based item (optional) / Domain scores derived from each question item. Scale scores calculated to meet 0-100 scale (mean 50).	8-item, Self-report questionnaire rated on a scale of 0 (completely dissatisfied) to 10 (completely satisfied).	18+	.70-.85	.50-.78	not available
WHOQOL-BREF ^l	Quality of Life / Overall; Health; Physical; Psychological; Social; Environmental; Support in everyday life. Scale scores calculated to meet 0-100 scale (mean 50). Scores also equated to PWI.	Self-report 26-item questionnaire rated on a scale of 1 (completely dissatisfied) to 5 (completely satisfied).	12-97	.66-.87	not available	not available

Notes:

¹ Reliability scores are reported for test-retest, inter-rater and alternate forms, where relevant. Values reported here relate to Adult samples.

² Validity scores are reported for item / content validity and index / construct structure, and comparisons to similar scales / tests. For example, CREVT Validity is contrasted with Wechsler scales (WAIS), Clinical Evaluation of Language Fundamentals (CELF) and Peabody Picture Vocabulary Scales (PPVS), amongst others. Values reported here relate to Adult samples.

³ Specificity is reported as controlling for test bias toward specific groups (e.g. gender, race, clinical). Values reported here relate to Adult samples.

^a ADOS-2, Module 4: Autism Diagnostic Observation Schedule–Second Edition (Lord et al., 2012), Module 4 is suitable for administration to adolescents or adults with average to above-average verbal ability. Scores in parentheses indicate cut-offs for Autism (A) or Autism Spectrum (AS).

^b AQ: Autism Spectrum Quotient (Baron-Cohen et al., 2001).

^c SRS-2: Social Responsiveness Scale–Second Edition (Constantino & Gruber, 2012). Total score and T-Index (treatment subscale) scores are reported as described above.

^d WAIS-III: Wechsler Adult Intelligence Scales–Third Edition () was administered in full to all participants, wherever possible. For administrative reasons, the WAIS-IV (Wechsler Adult Intelligence Scales–Fourth Edition) was used in later assessments with participants after careful consideration of the concurrence between items and scale measures. All WAIS-III scale scores were calculated to parallel measures on WAIS-IV scales. Normative scores range from 70-130, Mean 100, SD 15.

^e CREVT-3: Comprehensive Receptive and Expressive Vocabulary Test–Third Edition. Normative scores range from 70-130, Mean 100, SD 15.

^f CVLT-2: California Verbal Learning Test–Second Edition. There are several possible outcome measures for this test. Those reported in the Results are with respect to previous literature and specific research objectives.

^g The CANTAB were administered in one session wherever possible. However, owing to time constraints or participant fatigue, some tests were administered at a later session (approximately 2-4 weeks later). Owing to logistical reasons, the CANTAB data is not available to report for participants tested during 2016. New data were collected where possible. All available data are reported in the Results section of Chapter 4.

^h PHQ: Patient Health Questionnaire.

ⁱ BAI-II: Beck Anxiety Inventory–Second Edition.

^j BDI-II: Beck Depression Inventory–Second Edition.

^k PWI-A: Personal Wellbeing Index–Adult version.

^l WHOQOL-BREF: World Health Organisation Quality of Life assessment–Short Form.

Set out in Tables 4.1a and 4.1b below are the measures used in Study 1 (and subsequently re-assessed in Study 2, Chapter 5). These measures are organised in the following way:

Table 4.1a. Study 1 Measures by domain

Domain	Sub-category domain	Measure
Autistic traits	-	ADOS-2 AQ SRS-2
Cognitive function	General intellectual ability Receptive and Expressive Language Memory Executive Function (EF) (see Table 4.1b below)	WAIS-III CREVT-3 CVLT-II CANTAB [®]
Well-being	Physical and Mental Health Subjective Well-being Quality of Life	PIAS PHQ BAI-II BDI-II PWI-A WHOQoL-BREF

EF is further broken down into a series of 10 sub-tests (Table 4.1b):

Table 4.1b. Study 1 EF Measures (CANTAB[®])

Domain	Sub-category domain	Measure
EF	Planning / Strategy / Working Memory (WM)	SWM (Planning, WM) SOC (Strategy, WM)
	Cognitive flexibility / Attention / WM	IED
	Attention / Inhibition	RVP RTI
	Visual / Episodic Memory and Learning	PAL DMS

The purpose of the questionnaire or assessment was explained to the participant together with instructions for completion. Participants had an opportunity to clarify their understanding of what was required for each assessment before continuing, and, in the case of questionnaires were then left alone to complete these free of interaction with the researcher. For administered tasks (i.e. ADOS, WAIS-III, CREVT-3, CVLT-II, and CANTAB) the researcher explained at which points the participant could ask further questions or request additional information, or where no further information could

be offered by the researcher (in line with task guidelines). Participant responses were checked for acquiescence bias and incomplete response sets upon completion. Next, a summary is provided of each measure and corresponding outcome variables.

Autistic traits

All participants in the ASD group were assessed using ADOS-2 (Lord et al., 2012), which is the gold standard measurement of the traits and difficulties associated with ASD in adults or persons with highly developed verbal ability. The researcher was trained and overseen by a certified ADOS trainer. Under supervision, more than one-third (37.2%) of ADOS assessments in this study were double-coded for inter-rater reliability which was maintained at a minimum level of 83.5% or above (recommended level of 80%; Hus & Lord, 2014). Self-report measures of autistic traits were administered to both autistic and TA participants to obtain a measure of difference on the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2000) and the Social Responsiveness Scale–2nd Edition (SRS-2; Constantino & Gruber, 2012). The profile of diagnostic differences in ASD adults were assessed by three measures, each tapping into specific aspects associated with ASD-related difficulties.

Autism Diagnostic Observation Schedule – Second Edition (ADOS-2)

The participants in the ASD group had prior diagnoses by clinical specialists, verified by clinical reports. Diagnoses were made at different life stages and using various diagnostic instruments (e.g. ADI-R, DISCO and other methods not disclosed in practitioner reports). Where possible, recent ADOS scores were obtained from clinician diagnostic reports. Otherwise, the ADOS-2 Module 4 was administered to ASD participants only, who had not recently been administered the ADOS in the past 2 years, as a uniform assessment of autistic characteristics in ASD participants. The assessment duration was approximately 60 minutes. Where prior consent was obtained from participants, the ADOS assessments were video recorded for reliability coding.

The ADOS (Lord et al., 2012) is considered the Gold Standard diagnostic instrument as part of a range of clinical tools used in multidisciplinary assessments of ASD. The ADOS-2 Module 4

algorithm for cut-offs at or above threshold, indicate the presence of Autism Spectrum (Communication >2; Social Interaction >4; Total >7) or Autism (Communication >3; Social Interaction >6; Total >10). The ADOS was administered to 47 participants (82.5%) in the ASD group. Scores varied in meeting the cut-offs for Autism and Autism Spectrum indices, both between participants and also within assessments for the same person. Total scores indicated approximately one-third (36.2%) of ASD participants met the cut-off for Autism and approximately one-third (36.2%) met the cut-off for Autism Spectrum, but just less than one-third (27.7%) did not meet the cut-off. On the Communication index, the majority of these participants the majority (53.2%) met the cut-off for Autism and more than one-third (38.3%) met the cut-off for Autism Spectrum Disorder. Only 4 participants (8.5%) did not meet the minimum cut-offs. On the Social Interaction index, approximately half of ASD participants met the cut-off for Autism and more than one-third (38.3%) met the cut-off for Autism Spectrum Disorder. Only 4 participants (8.5%) did not meet the minimum cut-offs for Communication and Social Interaction, respectively, but only 2.8% of individuals were below threshold for Total scores.

Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001)

The AQ is a 50-item self-report measure that assesses difficulties associated with autistic traits. The AQ takes approximately 5-10 minutes to complete. Items are scored on a 4-point Likert-type scale, from 1 (definitely agree) to 4 (definitely disagree), with specific items reverse-coded in the scoring algorithm (Baron-Cohen et al., 2001). A Total score with cut-off >26 are recommended, with scores at or above this threshold indicating the likelihood of autistic traits (Woodbury-Smith et al., 2005). Index sub-scores (range 0-10) are calculate for Social Interaction, Attention Switching, Attention to Detail, Communication and Imagination. Higher Total and Index scores indicate higher degree of autism-like traits.

Social Responsiveness Scale – Second Edition (SRS-2; Constantino & Gruber, 2012)

The SRS-2 is a standardised measure for screening the severity of characteristics associated with autism and clinical treatment pathways based on the threshold score obtained. The SRS-2 is

available for informant-report or as a self-report measure for adults aged 19 years and older, and who have at least average intellectual ability to enable comprehension of the questionnaire. The self-report form is based on the individual's perception of their behaviours during the preceding 6 months. Normative data for gender, ethnic group, level of education and regional location are provided (sample size = 1,906 individuals). The SRS-2 outcome measures are aligned with the diagnostic classification for Autism Spectrum Disorder in the Diagnostic and Statistical Manual–Fifth Edition (DSM-5; American Psychiatric Association, 2015), and differentiates characteristics associated with Autism Spectrum Disorder from traits seen in other psychological conditions such as anxiety and ADHD, to .78 sensitivity. In a study using the German-language version of the SRS-2 with groups of autistic adults (n=20) compared with a non-autistic clinical group (n=62) and a group of typically developed adults (n=163), sensitivity in ASD was reported at .85 and specificity at .83 (Bölte, 2012). The findings lend support to the use of the SRS-2 with autistic adults who have highly developed cognitive and verbal ability.

The SRS-2 takes approximately 10-20 minutes to complete. Participants rate 65 items on a four-point scale indicating how characteristic (true) the statement is of them, from 0 (not true) to 3 (almost always true). Items are coded according to an algorithm that is concealed from participants under a carbon-copy sealed-edge form. The items are coded from raw scores to T-scores (minimum >30 to ≥ 90 maximum; mean 50, standard deviation 10), which are calculated for the Total score and treatment sub-scale scores for Social Communication, Social Motivation, Restricted Interests and Repetitive Behaviours (RRBs), Social Awareness and Social Cognitive functioning.

The AQ and SRS-2 were administered to ASD and TA participants to establish Diagnostic Group differences in level of autistic traits above or below clinically significant thresholds.

Cognitive functioning

The cognitive measures and their respective key outcome measures are summarised below. In the Results section that follows, the respective outcomes are provided again for ease of reference. All tests were administered to evaluate general intellectual functioning on the Wechsler Adult Intelligence Scales (WAIS-IV; Wechsler, 2008); and language ability was measured by the

Comprehensive Receptive and Expressive Vocabulary Test–3rd Edition (CREVT-3; Wallace & Hammill, 2013). The California Verbal Learning Test–2nd Edition (CVLT-II; Ober et al., 2000) provides measures of verbal learning and memory related to simple and complex memory processes and mechanisms; whilst the range of EF components was assessed on the Cambridge Neuropsychological Test Battery (CANTAB®; Cambridge Cognition, 2013).

(i) General Intellectual Functioning

Wechsler Adult Intelligence Scales, Third Edition (WAIS-III, Wechsler, 1998)

The Wechsler Adult Intelligence Scales (WAIS; Wechsler, 2008) and its variants is widely used as a standardised measure of IQ, with normative scores (mean 100, standard deviation 15) in the general population, across a range of ages (16-90 years), cultures and, recently, neurodevelopmental and clinical conditions (Wechsler, 2010). These norms provide a method of measuring an individual's IQ relative to a sample of age-range matched individuals in the population, with incremental age adjustments for older adults. The WAIS relatively exempt from practice effects with high reliability (.93-.97), making it suitable for re-testing across the lifespan. Raw scores from the 10 sub-tests are computed as age-stratified standardised scores, resulting in Index scores for Verbal Comprehension (VIC), Perceptual Reasoning (PRI), Working Memory (WMI) and Processing Speed (PSI), and the composite FSIQ score.

(ii) Receptive and Expressive Language

Comprehensive Receptive and Expressive Vocabulary Scales, Third Edition (CREVT-3; Wallace & Hammill, 2013)

The CREVT-3 is a standardised test that is designed to assess receptive and expressive language skills and the differences between these which may of clinical of developmental significance. Normative data are provided for stratified age ranges (adults 18-89 years). The assessment is comprised of two tests: the receptive language test requires participants to indicate which picture (from a series of category templates) corresponds to a given word; and the expressive language requires participants to provide the definition or meaning of a given word. All words are

verbally administered by the researcher; words can be repeated for clarity but no further information is given to the participants (e.g. spelling). The CREVT takes approximately 30 minutes to complete, administered as 15 minutes each for the receptive and expressive language components. Index scores are calculated for Receptive and Expressive language, and a composite score is calculated for General Vocabulary; scores range from 70-130 (mean 100, SD 15). Descriptive data are calculated for age-level equivalent (to 18 years) and level of overall language skills ranging from “very poor” (<70) to “very superior” (>130).

(iii) Memory

California Verbal Learning Test, Second Edition (CVLT-II; Ober et al., 2002)

The CVLT-II is a standardised assessment of episodic verbal learning and memory, as measured by recall (self-initiated/free and cued) and recognition discriminability – defined as correctly identifying target words and correctly rejecting distractor words. Verbal learning and memory have been shown to be crucial component of everyday functioning (Zimbelman, 1990). Accordingly, the CVLT-II assesses the “process of memory performance” and “mechanisms of memory failure” (Delis et al., 2000) that occur in individuals with neuropsychological cognitive impairments (e.g. Parkinson’s disease (Massman et al., 1990; Buytenhuijs et al., 1994), Alzheimer’s disease (Deweert et al., 1994; Kramer et al., 1988), and affective disorders such as depression (Massman et al., 1992; Hill et al., 1993; but see Lyness et al., 1994), as well as memory decline seen in typically ageing adults (Delis et al., 1987; Van der Linden et al., 1997). The CVLT has been used as broad assessment of simple and complex memory abilities in autistic children and adults (Minshew et al., 1992; Minshew et al., 1997; Minshew & Goldstein, 2001; Williams et al., 2006).

Two parallel forms are available for administration of this test – a Standard form and an Alternate form, each containing a list of 16 words, comprising 4 categories of 4 words. The respective word lists for the parallel forms are matched on word frequency as “prototypical of the categories” (a short form is also available for individuals with intellectual disability or significant clinical memory impairment, but it was not required in this study). The test is administered in 4 parts (phase 1-4; see Figure 4.1, p. 106) and completed in a single test session, including two structured and timed breaks

of 20 minutes and 10 minutes to introduce long and short delays, respectively, as part of the recall test procedure. The total test duration is 60 minutes. Test norms are standardised for typically ageing adults aged 16 years up to age 89 years, with internal consistency reported as .82 across the lifespan (range: age 16-19 years = .80; age 80-89 years = .72) and .74 validity. The CVLT-II is reported to correlate highly with IQ as measured on the WASI Full Scale IQ (r .40, $p < .001$) and Vocabulary index score (r .46, $p < .001$), and similar observations are reported on WAIS-R (Delis & Kramer, 2000). However, some research has noted the declines in memory performance in older adults even when controlling for Verbal IQ (Van der Linden et al., 1997) and overall reduction in factor structure of the CVLT-II in older groups (Donders, 2008), suggesting that performance in older adults should be interpreted with caution.

In the present study, each participant was administered either the Standard form or the Alternate form⁵. The parallel forms were administered in counterbalanced order, controlling for age and gender across Diagnostic Groups (i.e. a ASD young female and a TA young female were administered the same form version). The list was verbally administered by the researcher reading out the list of target words, without repetition, followed by the participant recalling as many target words as possible, across 5 successive trials (phase 1 – List A Target Words, Immediate Free Recall). Thereafter, participants were administered a second list of 16 words (List B Interference Words, Immediate Free Recall), in the same fashion as phase 1 but with only one free recall trial, followed by a prompt for participants to recall as many target words from List A as they can (phase 2 – List A, Short Delay Free Recall). This was directly followed by cued recall for each of the 4 categories (phase 2 – List A, Short Delay Cued Recall). At this point, the long-delay interval was initiated by a 20-minute timed break, during which participants were engaged in non-verbal testing on an unrelated task⁶. On resuming, participants were straight away asked to recall as many target words from List A

⁵ The counterbalancing of parallel forms was extended from between-participants in this study to both between- and within-participants in the longitudinal study (Chapter 5). For example, if participant 1 was administered the Standard form in this study, the Alternate form was administered to that participant in the longitudinal study. Controlling for age, gender and Diagnostic Group were still applied.

⁶ A timed interval of not less than 15-minutes and not more than 25-minutes is recommended by the test guidelines, whilst in our study the average break was 20-minutes, ranging from 18- to 22-minutes. Some participants required a comfort break during this interval but were not permitted to use their mobile phones or read the newspaper or other material during this time.

as they could (phase 3 – List A, Short-Delay Free Recall), followed by category prompts as previously described (phase 3 – List A, Short-Delay Cued Recall). This was followed a recognition test (phase 3 - List A, Long-Delay Yes/No Recognition) whereby the researcher read aloud each word in this section (targets and lures) and participants were required to respond “Yes” if recognised that word being on List A, or respond “No” if they did not recognise that word being on List A. Next, a shorter 10-minute timed break followed the same format as previously indicated. The final phase involved a supported recognition test (phase 4 – List A Long-Delay Forced-Choice Recognition), during which the research read aloud 16 word-pairs comprised of a target word and either a concrete or abstract distractor word, and participants were required to repeat one word from each word-pair if they recognised that word as being on List A. Accuracy was calculated as the total number of correct items (hits), less the total number of intrusions and repetitions (false-positives) on free and cued recall trials and Yes/No Recognition trials, as well as serial order clustering for free recall trials, providing a measure of encoding strategies, learning rates across trials and error types. On the Long-Delay Forced-Choice Recognition trials, accuracy was calculated as total number of correct items (hits), transformed to a percentage correct score.

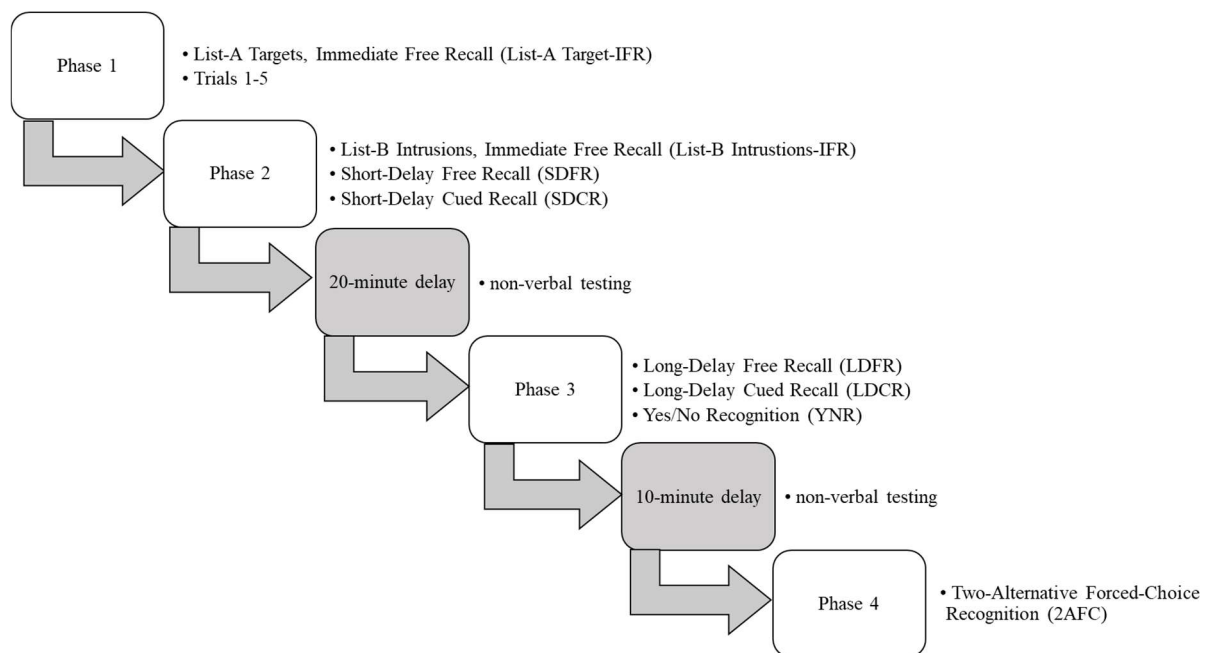


Figure 4.1. Schematic of CVLT-II Trials by Phase and Type of Assessment.

(iv) Executive function

Cambridge Neuropsychological Test Battery–CANTAB® (Cambridge Cognition, 2013)

The CANTAB® is a series of computerised tasks that assess broad range of EF and its subcomponents. The tasks are sensitive to detecting mild to moderate cognitive impairment and cognitive difficulties associated with depression, particularly in middle-aged and older adults. The CANTAB® tasks are easily administered across range of ages, with normative comparison data provided for assessing age-relevant EF performance. Moreover, digital administration allows for reduced social demands that may further disadvantage autistic individuals (White et al., 2009; but see Williams et al., 2014). A series of 10 sub-tests were individually administered with minimal interaction from the researcher during the tasks. Verbal instructions are provided prior to the start of each task, with short practice trials or demonstrations as specified by the CANTAB® administration guidelines. The ongoing tasks are non-verbal unless requiring specific prompts. Some tasks required touch-screen responses, whilst others required participants to press a button on a press-pad device. Outcome measures are unique to each task. Whilst many possible outcome measures can be derived, specific and relevant measures are reported for each task, in line with the research aims and questions of interest. Also, as previously highlighted, there is inevitable overlap between EF processes involved in various tasks. Accordingly, the overlap between CANTAB® tasks is outlined below with details of the respective tasks corresponding to each EF domain.

Working memory / Planning

Task: Spatial working memory (SWM)

The SWM is a test of working memory (maintenance/updating) and effective use of search strategies. Participants were visually presented an incremental number of on-screen boxes (practice 3; test: 4, 6, 8); each box contains a token which must be ‘found’ by using touch-screen responses to ‘search’ each box. The order in which tokens are revealed is not known by the participant and changes for each trial. Participants must, therefore, use a self-initiated search strategy to search for each token, and remember where they have previously attempted to find the tokens. Once a token has been found the participants must place it in the ‘vault’ (home area on the right of the screen).

Participants are instructed that once a token has been found they must never search that box again. Success is achieved by finding all tokens in a minimum number of moves. Errors are recorded as repeatedly searching in boxes that have already revealed tokens.

Outcomes:

- SWM Between errors (4, 6, 8 boxes) – perseverative errors measured as the total number of times a participant searches a box that already revealed a token. A lower score indicates better performance.
- SWM Strategy – good strategy is suggested as starting from the beginning of a set sequence each time a token is found. Each time the participant deviates from this strategy (boxes 6, 8 only), it is counted towards the Strategy outcome. A lower score indicates better performance and better use of the strategy.

Task: Stockings of Cambridge (SOC)

The SOC is a planning task equivalent to the Tower of Hanoi, which assess spatial planning and working memory ability. A split screen is shown with three pockets (stockings), like a billiards table, in the top half of the screen and three stockings in the bottom half. Coloured balls (red, green, blue) either stacked or individually suspended in the stockings. Participants use touch-screen responses to move each ball between the stockings, following a specific sequence. During practice trials, the administrator provides a demonstration and clearly defines the rules that must be followed: a number on the right of the screen indicates how many moves must be made; balls must be moved one at a time, so that the pattern in the bottom half of the screen matches the top half (reference pattern); a ball may be selected by tapping it once (the ball then flashes) and then touching the location it is to be moved to; a ball that is underneath another ball (stacked) cannot be selected, but the top ball needs to be moved out the way first; balls cannot be moved ‘into thin air’ (placed in an empty space above another empty space) but must be placed on top of another ball or in the bottom of a pocket. Participants are encouraged to plan their moves before moving the balls. A series of problems is then presented with incremental moves required (practice: 1; assessed: 2, 3, 4, 5). The

first assessed block includes two trials each of 2,3, and 4 moves, followed by a motor-control block in which participants need to follow the computer-led moves for the problems already completed. The second assessed block includes two trials each of 2 and 4 moves, and four trials of 5 moves, followed again by a motor-control block. The SOC responses are timed for duration of planning response time. Each trial terminates after the maximum number of moves has been exceeded.

Outcomes:

- SOC planning accuracy – measure as the total number of problems solved in minimum overall number of moves. A higher score indicates better performance.
- SOC Planning time, 2-moves, 5-moves – The latency (time taken) to plan a move strategy for each problem is calculated as the “difference in time taken to select the first ball for the same problem” in the motor-control block (p. 310 administration manual), measured as response time (RT) in milliseconds (ms). The 2-move outcome score corresponds to lower cognitive demand and the 5-move outcome with higher cognitive demand and associated performance. Lower RTs indicate better performance. However a score may be 0 if the participant was slower to respond in the motor-control block.

Cognitive flexibility / Attention / Working memory

Task: Intra/Extradimensional Set Shift (IED)

The IED measures the acquisition of rules and their application, and the ability to switch between rules (including rule reversal / set shifting). This task requires visual discrimination of information, maintained attention and flexibility in switching between sets as required by the task demands. In each trial two abstract shapes, each comprising a pink shape with a superimposed white line, are presented simultaneously. Participants are instructed to select the shape they think is the correct one, using touch screen responses. On each trial computerised response feedback is given about whether the selection is correct or incorrect, leading to implicit rule acquisition. No clues are given and the first trial has a 50:50 chance of accuracy. The task comprises 9 blocks (stages of dimensional shifting): stages 1-8 require intradimensional (ID/pre-ED) shifting on one dimension

(e.g. pink shape); rules alternate between shapes after success of 6 consecutive trials. Later trials, in the 9th stage, require an extradimensional shift (EDS) to a different dimension (e.g. white lines). Responses are not timed but the IED task terminates after the maximum of 50 trials has been reached, at any stage, without successfully switching between rules or dimensions.

Outcomes:

- IED Pre-ED errors – the total number of errors made during the ID trials, before the shift is made to ED. Errors are recorded as failure to adjust responses the correct (current) rule, whereby participants incorrectly select a stimulus that does not correspond to the rule for a given set of trials. Lower scores indicate better performance.
- IED EDS errors – failure to shift to the extradimensional set is recorded as errors here; perseveration with incorrect responses are accumulated for EDS errors. Lower scores indicate better performance.
- Total errors (adj). – a measure of overall response inefficiency, adjusted for the total number of errors made across the total number of trials completed. Participants who fail the task in the early stages have fewer opportunities to make cumulative errors in later trials. Accordingly, a score adjustment is made by weighting each uncompleted stage. Lower scores indicate better performance.
- Stages completed – the total number of stages successfully completed, to a maximum of 9 stages. Key dimensional shifts are made at stage 6 (intradimensional) and stage 8 (extradimensional). Higher scores indicate better performance.

Attention / Inhibition

Task: Rapid Visual Information Processing (RVP)

The RVP measures sustained visual attention over period of time and under increased task complexity. An array of numbers from 2 to 9 appear briefly on screen one at a time, in a random order. In simple trials, the objective is for participants to identify a target 3-number sequence (visible on screen as a prompt) that appear sequentially within the array of numbers. Participants are required

to respond as quickly as possible, each time the target sequence is identified. In complex trials, the same process takes place, but here, participants need to identify three 3-number target sequences, where the numbers of each sequence appear sequentially, in the array.

Outcomes:

- RVP A' (A prime) – the acquisition of information and resulting response accuracy is measured using Signal Detection Theory. Here, responses are adjusted for sensitivity to the target stimulus (hits) and response bias (false alarms). The speed-accuracy trade-off is accounted for as participants who are biased to speed of responses are likely to show reduced accuracy (correct rejections / non-responses to false alarms). Performance is scored from 0.00 (poor) to 1.00 (good). Higher scores indicate better performance.
- RVP B'' (B double prime) – measure of response rate, irrespective of whether the target sequence is displayed. Performance is scored from -1.00 to +1.00 (fewer false alarms). A perfect hit rate (100% hits) is scored as -1.00.
- RVP Mean latency (ms) – response time measured in milliseconds (ms), as the mean time taken for each correct response made within a window of 1800ms. This outcome is a measure of sustained attention. Lower scores indicate better performance.

Task: Reaction Time (RTI)

The RTI measures visual attention and corresponding speed of responses to a given stimulus at one or 5 on-screen locations. Accordingly, the RTI also provides a measure of motor function difficulties under increased task complexity. In this task a yellow spot (stimulus) appears briefly on the screen, and participants are required to touch the screen as quickly as possible when they see the stimulus (without hovering their hand over the screen i.e. reach and touch). There are several stages of this task, in the following order: simple touch is described above; simple release involves participants using the press pad to make the stimulus appear (press and hold until the spot appears), then immediately release; five-choice touch follows the protocol for simple touch but at 5 possible locations; simple release and touch involves the press pad (press and hold to make the stimulus

appear) followed by immediate release and touch the location of the stimulus; and five-choice release and touch follows the same protocol just described but with five possible locations.

Outcomes:

- RTI Simple / Five-choice accuracy score – the total trials in which a correct response was made, in one / five locations respectively. Higher scores indicate better performance.
- RTI Simple / Five-choice mean movement time – time taken in milliseconds (ms) to touch the stimulus after the press pad button has been released, in one / five locations respectively. A lower score indicates better performance.
- RTI Simple / Five-choice reaction time – time taken in milliseconds (ms) to release the press pad button once the stimulus has appeared, in one / five locations respectively. Reaction times will be longer for five-choice compared with simple trials. A lower score indicates better performance.

Visual / Episodic Memory and Learning

Task: Paired Associates Learning (PAL)

The PAL task assesses learning and memory of visual information. Across successive trials in 8 stages, a display of white boxes appears on screen with one to eight boxes each containing a pattern. The computer ‘opens’ each box in a random order by reveal if it is empty or has a pattern. Participants are required to remember which box (the location) contains that particular pattern. In later trials where all boxes contain pattern, memory load is greater. Once all boxes have been opened, the pattern(s) appear in the centre of the screen (in the case of more than one pattern at the response stage, these are presented one at a time in a random order). Participants are required to use touch screen responses to indicate the matching location. If an incorrect response is made the trial is repeated until all pattern-locations have been correctly entered. The task terminates after all stages have been successfully completed or after 10 consecutive trials have been failed.

Outcomes:

- PAL Total errors adjusted, 1 / 2 / 3 / 6 / 8 shapes – the total number of errors made at each of the incremental stages of task complexity. Scores are adjusted for participants who did not reach later stages as a result of earlier failures ($1 - 1 / \text{number of boxes}$). Lower scores indicate better performance.
- PAL Memory score – the number of patterns correctly located in the first trial for each stage, reported as the cumulative total for all stages. Higher scores indicate better performance.

Task: Delayed Matching to Sample (DMS)

The DMS is a task of recognition memory and detection of complex visual stimuli after a period of delay. A complex coloured visual pattern (target stimulus) made up of four abstract components is briefly presented on screen and then masked, followed by a delay of varied durations (0ms, 4000ms, 12000ms; delay trials). Then, four more patterns are presented, one of which matches the target stimulus. In some trials the four patterns are presented simultaneously to the target (simultaneous trials). There are three practice trials and two counterbalanced blocks of 20 assessed trials across all delays. Participants use the touch screen to select the pattern that matches the target. If the response choice is incorrect another response selection must be made.

Outcomes:

- DMS Percent correct, simultaneous – the percentage of correct first responses for all trials where the match patterns were simultaneously presented with the target stimulus (baseline, simple performance). A higher score indicates better performance.
- DMS Percent correct, 0ms / 4000ms / 12000ms delays – the percentage of correct first responses for trials where the match patterns were presented for respective intervals after the stimulus was masked. A higher score indicates better performance.

- DMS A' – a measure of the participant's sensitivity to errors, as the probability of making an error on subsequent trials, given previous errors made. Performance is scored from 0.00 to 1.00. A lower score indicates fewer subsequent errors and better performance.
- DMS B'' – a similar measure of probability of errors. Performance is scored from -1.00 to +1.00. A lower score indicates a greater overall tendency to make errors, whilst scores closer to +1.00 indicate a greater propensity for errors only if an error was just made. Accordingly, a score closer to -1.00 indicates better performance.

Physical and mental health

The Passport to Individual Autism Support (PIAS; The National Autistic Society, 2012) was administered as semi-structured interview during the first assessment together with background diagnostic and medical history. The PIAS was created to assist autistic individuals who have difficulties advocating for themselves when accessing health and social care services. The resulting information provides a summary of co-occurring diagnoses and other self-reported conditions or difficulties associated with autism, such as sensory sensitivities, motor function limitations, basic cognitive difficulties and issues related to social behaviours. The 2-page document is available from the National Autistic Society's website (www.autism.org.uk) for completion by the individual or their carer or advocate. The information set out in Figure 4.2 (p. 115) describes some of the information captured by the PIAS.

In addition, the following measures were administered, each with clinically relevant cut-offs for specific mental health difficulties, including anxiety and depression. All three measures were completed by participant self-reports:

- Patient Health Questionnaire (PHQ; Spitzer, Kroenke, Williams et al., 1999)
- Beck Anxiety Inventory – Second Edition (BAI-II, Beck & Steer, 1993)
- Beck Depression Inventory – Second Edition (BDI-II, Beck, Brown & Steer, 1996)

More information about these measures is detailed below and their respective psychometric properties are summarised in Table 4.0 (p. 96).

Co-existing conditions	Sensory sensitivity (under / over sensitive)	Sensori-Motor function	Cognitive function	Social function
• Sleep: irregular patterns / disturbances. • Other diagnosed e.g. • depression • anxiety • cancer • irritable bowel syndrome • other digestive problems (reflux, constipation) • menopause • enlarged prostate • hypertension • cholesterol • diabetes	• Light / Vision • Sound / Hearing • Smell • Taste • Touch / Texture	• Balance • Speed of responding • Co-ordination. • Multi-tasking. • Sense of direction	• Concentration • Short term memory • Time management • Following complicated instructions • Filling in forms • Planning ahead and changes to plans • Information processing	• Social conversation • Understanding body language • Understanding metaphors • Understanding social nuances • Ability to be brief • Being bullied • Eating in public • Stress and/or Anxiety related to: • busy or noisy places • social interaction • changes in routine or plans
• Medication • condition prescribed • dosage • type • duration			• Ability to travel independently • Preferred method of communication • Maladaptive stress response (e.g. physical or verbal outbursts and aggression [meltdown]; or passive and non-verbal response [shutdown]) • Support required during stress responses (if any, e.g. leave alone; sit in quiet room with dim lighting)	

Figure 4.2. Passport to Individual Autism Support (PIAS): Self-report record of autism-related difficulties.

Patient Health Questionnaire (PHQ; Spitzer, Kroenke, Williams et al., 1999)

The PHQ is a 3-page questionnaire comprised of core items designed to screen for depression and other disorders, including anxiety and panic syndrome, somatoform symptoms (e.g. pain, digestive problems), and risk of eating disorders and alcohol abuse. A single item is also included to reflect daily difficulties associated with these symptoms. The PHQ is a self-rated questionnaire that provides screening indicators to the aforementioned mental health conditions and is often used in primary care settings to inform treatment pathways (Kroenke, Spitzer, Williams, 2001). The measure is aligned with classification guidelines set out in the Diagnostic and Statistics Manual of Mental Disorders – Fourth Edition (DSV-IV, American Psychiatric Association, 1994) and the International Statistical Classification of Diseases and Related Health Problems–Tenth Edition (ICD-10; World Health Organisation, 2010), although the latter is thought to be “more parsimonious and suitable for clinical settings” (Fischer et al., 2011). Given that the research context is not intended to be a clinical diagnosis, but rather a screening of the co-existing conditions present in autistic individuals compared to typically ageing individuals, the PHQ provides a comprehensive overview that contributes to this exploratory study.

Participants completed the PHQ as self-reporting the degree to which they had experienced symptom difficulties during the previous 4 weeks (major depression, panic syndrome, other anxiety syndrome), 2 weeks (other depressive syndrome), 3 months (eating disorders), or 6 months (alcohol abuse). The questionnaire is set out so that each section pertains to a specific condition with either dichotomous (yes-no) response options, or options that are rated on a 4-point or 5-point Likert-type scale (e.g. “not at all” to very much”). An algorithm is provided for each section to calculate cut-offs according to the respective condition, where a specified number of symptoms must be present to align with diagnostic thresholds. Data from the original validation study (Kroenke, Spitzer, Williams, 2001) included a sample of 3000 adults aged 18-99 years (mean 46, SD 17.2 years; 66% female) who were recruited from primary care clinics. The authors compared outcomes on the PHQ with assessments by independent mental health practitioners, reporting “overall accuracy, 85% (CI: 82-88%); sensitivity,

75% (CI: 69-81%); and specificity, 90% (CI: 87-93%)⁷ for these conditions. Moreover, an increase in symptom severity was significantly associated with decreased functional ability relating to pain, physical mobility, social activities, work or household chores, mental health and perceptions of current health, and an increase in access to health care services.

Beck Anxiety Inventory—Second Edition (BAI-II, Beck & Steer, 1993)

The BAI-II is a standardised, self-rated measure that captures the physical symptoms associated with anxiety, not explained by biological reasons (e.g. symptoms may be related to hypoglycaemia; peripheral neuropathy, or other non-anxiety factors). The BAI provides an indicator of the severity of anxiety-related symptoms in adults aged 17 years and older, with no upper age limits. The questionnaire lists 21 items, as physical symptoms of anxiety (e.g. unable to relax; hand trembling) and associated cognitive symptoms (e.g. fear of the worst happening; fear of losing control). The individual is required to rate each item in terms of how much they have been bothered or distressed by each symptom during the past month (including the day of rating). The whole questionnaire takes approximately 5-10 minutes to complete.

Items are scored on a scale of 0-3 (0 = “not at all”; to 3 = “severely”), and the sum across all items results in a Total Anxiety score (maximum = 63). Missing items are pro-rated as the average of all other items, but where greater than 20% of items are missing the response sheet should be discarded as insufficient data are available for meaningful analysis. The total score provides a measure of anxiety severity according to several cut-offs: 0-7 = Minimal level of anxiety; 8-15 = Mild anxiety; 16-25 = Moderate anxiety; 26-63 = Severe anxiety. The BAI is valid in differentiating between individuals with and without anxiety, with reliability of .92. Test-retest reliability of .75 supports the use of the BAI in repeat assessment at different time points.

⁷ CI: Confidence Interval at 95% certainty (Spitzer, Kroenke, Williams et al., 1999).

Beck Depression Inventory–Second Edition (BDI-II, Beck, Brown & Steer, 1996)

The BDI-II is widely used in clinical and research settings to screen for severity of depression and related physical and psychological symptoms and behaviours e.g. suicidal ideation, rumination, sleep disturbances, weight loss and change in appetite. The 2-page questionnaire consists of 21 items which are self-rated by the individual based on the extent to which they have experienced changes and/or difficulties with each symptom on a four-point scale, from 0 (not at all) to 3 (severely). A total depression score is calculated by summing the items (maximum score = 63), and cut-off scores indicate the severity level of depression as follows: 1-10 = “normal ups and downs”; 11-16 = mild mood disturbance; 17-20 = borderline clinical depression; 21-30 = moderate depression; 31-40 = severe depression; over 40 = extreme depression. This questionnaire takes approximately 5-10 minutes to complete and is appropriate for use with adolescents aged 13 years and older, to an upper age limit in adults of age 80 years. The BDI-II is aligned with diagnostic criteria specified in the DSM-IV-TR (APA, 1994). Reliability is .86 across levels of depression, and .90 test-retest reliability makes the BDI-II a suitable measure for re-assessment of depressive symptoms in longitudinal cohorts.

Quality of life (QoL) and Subjective Well-being (SWB)

Assessments involved self-rating the WHOQOL-BREF (WHO, 2000) – a measure of overall QoL with index scores of Physical-QoL, Psychological-QoL, Social-QoL and Environmental-QoL, and the Personal Well-being Index for Adults (PWI-A; Cummins et al., International Well-being Group, 2003) – measuring overall SWB and global life satisfaction (GLS). These measures were selected since little research has evaluated QoL in ASD and even fewer studies have assessed this in older autistic adults (Geurts & Vissers, 2012). Consequently, it was unknown which measure would provide greater reliability within the ASD sample. Whereas, the World Health Organisation provides a standardised measure of QoL for adults in the general population (here the WHOQOL-BREF was used), alternatives are also provided for intellectual and physical disability, which were not suitable for ASD sample overall since physical disability was largely absent (however, some participants later disclosed difficulties with walking or prolonged sitting).

World Health Organisation Quality of Life, short form (WHOQOL-BREF; WHO, 2000)

The WHOQOL-BREF contains 26 items related to four outcome domains: Physical (e.g. activities of daily living, sleep, pain, illness), Psychological (e.g. negative and positive feelings, memory and concentration), Social (e.g. friendships and intimate relationships, social support), and Environmental (e.g. financial status, living arrangements, access to and quality of social care). Each item is self-rated on a Likert-type scale, from 1 (worst) to 5 (best), with slight variations in response naming conventions. The overall aim of the WHOQOL-BREF is to provide measure of the effects of physical and cognitive difficulties on everyday living and overall well-being. In addition to the four QoL domains, three additional standalone questions provide overall measures of QoL, health and level of support received from others. One of the benefits of the WHOQOL-BREF is that it asks about the individual's satisfaction with life-domains rather than assuming value of specific aspects of life. In this way, normative assumptions (e.g. about having a range of friends) are not hindering measuring the autistic perspective.

Whilst the WHOQOL-BREF and PWI measures are designed to capture similar domains, namely, Physical, Psychological, Social relationships, Environmental factors and overall QoL, the method of calculations differs between the measures. For this reason and in order to standardise the outcome scores for comparison and interpretation of the data, a conversion formula was applied to the WHOQOL scores for: overall QoL, overall health, and level of support received. The formula was derived from the PWI conversion guidelines for standardising scores to a 0-100 format, where raw scores may be derived from a different scale e.g. 1-5 rather than 0-10 (Cummins et al., 2003). Further, benefits of WHOQOL and PWI are that measures are self-rated not subjective associations e.g. number of friends; but potential challenges with self-report (e.g. Hong et al., 2016; see Chapter 2). A summary of each measure and its purpose is provided below.

Personal Well-being Index, Adult (PWI-A; Cummins, International Well-being Group, 2003)

The PWI-A is a standardised measure of subjective well-being (SWB), with good reliability (0.70-0.85) in a general adult population. The PWI-A is a self-rated questionnaire comprised of 7 core SWB items, which each encompass a single aspect (domain) of subjective well-being, related to broad outcomes including health, personal relationships, standard of living, and future prospects. Additionally, 2 optional questions are related to overall well-being and religion, respectively. Each item (including optional) is rated on a scale of 0 (no satisfaction at all) to 10 (completely satisfied). The core 7 items, together with the optional item about religion, provide an overall SWB outcome measure with a maximum score of 70 (minimum 0; range 70-80 in Western normative samples; Cummins et al. 2003). However, in our study many participants did not answer the optional item about religion: *“How satisfied are you with your spirituality or religion?”*, either declining to answer as “not applicable / no religion” or were unsure about how to rate it, whereas other participants provided a satisfaction rating despite no religion or spiritual practice. Therefore, the data for this item (spirituality / religion) are incongruous and were excluded from overall analysis. The data are reported for those individuals who answered all other items, where a maximum score of 70 is possible (see Table 4.6a, p. 150; Table 4.6b, p. 151; and see Figure 4.7, p. 149). The first questionnaire item (optional) is analysed separately, providing a measure of global life satisfaction (GLS; and see Diener et al., 1985): *“Thinking about your own life and personal circumstances, how satisfied are you with your life as a whole?”*). For the GLS item and remaining core 7 items, there were no missing data from participant responses.

4.5 Analysis strategy

Cross-sectional comparative analysis were carried out between Diagnostic Groups (TA; ASD) and Age Groups (younger; older) to determine the effects of ageing in the aforementioned domains, in line with the research questions and study aims accordingly: to understand the age-related differences in cognitive functioning, co-occurring mental and physical health conditions and their association with QoL in younger and older autistic adults, compared to typically ageing groups of younger and older adults. In the first instance, 2 (Diagnostic Groups: ASD; TA) x 2 (Age Groups: younger, older) Analyses of Variance (ANOVA) were carried out. Statistical significance ($\alpha, p < .05$) and effect sizes (η_p^2) are reported for relevant contrasts. Secondly, planned contrasts between Age Groups within each Diagnostic Group were carried out to assess the extent of differences associated with ageing for the ASD and TA groups, respectively. Third, planned contrasts between Diagnostic Group within each Age Group were carried out to assess the magnitude of differences between younger ASD (yaSD) and younger TA (yTA) adults, and between older ASD (oASD) and older TA (oTA) adults. Since the data for the Time 1 assessment were exploratory, no adjustments were made for multiple comparisons. A secondary analysis was carried out using Tukey and Bonferroni corrections for multiple comparisons. These methods both control for familywise errors and probability of Type I errors, whilst enabling detection of group differences and limiting of Type II error.

In sum, the key analyses related to:

- Diagnostic Group comparisons (ASD:TA)
- Diagnostic Group x Age Groups contrasts (yaSD; oASD; and yTA; oTA)
- Age Group x Diagnostic Group contrasts (yaSD; yTA; and oASD; oTA)

The data set out in Table 4.4a (p. 138) describe the sample characteristics for (i) Diagnostic Group comparisons, and in Table 4.4c (p. 142) for (ii) Diagnostic Group comparisons within each Age Group (Diagnostic x Age). Planned contrasts, using a multivariate ANOVA confirmed matching of Diagnostic Groups on chronological age, years of formal education (YFE) as self-reported in the

collection of background information, and IQ (FSIQ; VCI; PRI) as measured by the full Wechsler Adult Intelligence Scales–Third Edition (WAIS-III). Sample grouping was confirmed by levels of autistic traits for ASD and TA groups, as screened by Total scores (above threshold = ASD; below threshold = TA) on the Autism Spectrum Quotient (AQ). Social Responsiveness Scale (SRS) scores are reported for specific difficulties associated with autistic traits (see Table 4.3a, p. 126; and see Table 4.3b, p. 127). The ADOS was administered to ASD participants to obtain a profile of core difficulties associated with Communication, Social Interaction, Imagination and Repetitive Behaviours (see Table 4.2, p. 124).

4.6 Results

Sample characteristics

Chronological age did not differ between Diagnostic Groups (Table 4.3a, p. 126), or within Age Groups between Diagnostic Groups (Table 4.3b, p. 127). There were no Diagnostic Group differences in Gender ratio ($n=87$, $\chi^2(1) 3.628$, $p > .05$), nor was there a significant difference in Gender ratio within Diagnostic Groups between Age Groups (ASD $n=52$, $\chi^2(1) < 1.0$, $p > .05$; TA $n=35$, $\chi^2(1) < 1.0$, $p > .05$) or within Age Groups between Diagnostic Groups (Younger $n=42$, $\chi^2(1) 1.71$, $p > .05$; Older $n=45$, $\chi^2(1) 1.855$, $p > .05$).

There were no significant differences in verbal comprehension IQ (VCI), nonverbal perceptual reasoning IQ (PRI), or Full-scale IQ (FSIQ) between Diagnostic Groups (Table 4.3a, p. 126) or within Age x Diagnostic Groups with respect to verbal comprehension IQ and FSIQ (Table 4.3b, p. 127). However, within Age x Diagnostic Group contrasts, perceptual reasoning IQ was significantly lower for oTA adults compared to oASD adults, although scores for both groups were within the normal range of IQ (i.e. 70-130; see Table 4.3b, p. 127).

With respect to Years of formal Education (YFE), 7 older participants (6.9%) had fewer than 11 YFE (oTA $n=5$; oASD $n=2$). ANOVA revealed significant differences in YFE between Diagnostic Groups and within Diagnostic x Age Groups (Table 4.3a, p. 126; YFE sample mean 14.44 years, SD 2.87, median 15 years), which appeared to be explained by fewer YFE obtained by oTA adults (Table

4.3b, p. 127; and see Figure 4.3 below). For all matching analyses just described, pairwise comparisons were upheld after adjusting for multiple comparisons.

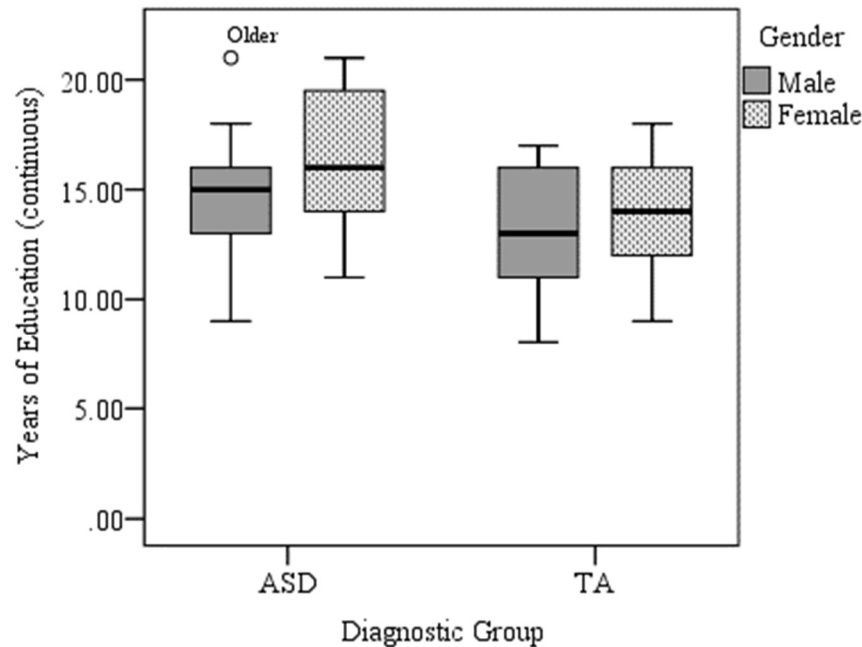


Figure 4.3. Years of Formal Education: Gender differences by Diagnostic Group and Age Group. Boxplot Error bars depict Standard Deviation (95% CI).

Autistic traits

Profiles of autistic traits, as measured by the AQ and SRS-2, differed significantly between Diagnostic Groups (Table 4.3a, p. 126; and see Figure 4.4, p. 125) and were upheld in planned contrasts between Age Groups within Diagnostic Groups (Table 4.3b, p. 127). There were no differences between Diagnostic Groups within Age Groups, nor were there any Gender differences (largest $F(1,65) 1.390$, smallest $p .243$, largest $\eta_p^2 .021$).

The profiles obtained for younger and older adults in the ASD group, did not differ on any of the ADOS index scores (Table 4.2, p. 124). Gender differences were only observed in the older ASD group in Communication – greater difficulties were observed for oASD males ($n=18$; mean 3.78, SD 1.31, 95% CI 3.13-4.43) compared to oASD females ($n=5$; mean 2.40, SD 0.55, 95% CI 1.72-3.08; $F(1,40) 5.403$, $p = .025$, $\eta_p^2 .119$). However, since the sample sizes are relatively small and few oASD

females were represented in this study, these data should be interpreted with caution. Self-report AQ and SRS scores did not correspond to ADOS outcome scores (largest $r(40) = .190$, smallest $p = .110$), whereas AQ and SRS scores correlated highly with each other (ASD: $r(40) \geq .627$, all $p \leq .001$; and TA: $r(33) \geq .560$, all $p \leq .001$) and were broadly correlated with measures of anxiety and depression (ASD: $r(44) \geq .505$, all $p \leq .001$; and TA: $r(33) \leq .389$, all $p \leq .023$). Possible interpretation of this discrepancy in self- vs observer-rated outcomes may be that self-report measures are not sensitive enough to detecting autistic difficulties, per se, but rather offer a transdiagnostic measure of general difficulties, such as those associated with mental health conditions, particularly in ASD (Roestorf, Gaigg, Williams & Bowler, 2018, INSAR). Another possible explanation may be related to a higher degree of self-reporting in cognitively able autistic individuals than would be expected. These are important factors to be considered in future work that aims to systematically differentiate between the core difficulties associated with ASD and co-occurring conditions.

Table 4.2. ADOS profiles: Cross-sectional comparisons within ASD Age Groups

Measure	ASD (n=44) Mean (SD)		Statistics		
	yASD (n=22)	oASD (n=22)	ANOVA $F(1,42)$	p	η_p^2
ADOS-2 Total	8.30 (2.86)	9.35 (3.60)	1.694	<i>n.s.</i>	.039
ADOS-2 Communication	2.90 (1.48)	3.45 (1.32)	3.392	<i>n.s.</i>	.075
ADOS-2 Social Interaction	5.40 (2.06)	5.90 (2.59)	.024	<i>n.s.</i>	.012
ADOS-2 Repetitive Behaviours	1.45 (1.32)	.85 (.88)	1.759	<i>n.s.</i>	.040

Notes: ADOS-2 = Autism Diagnostic Observation Schedule–Second Edition. Index score measures are calculated for: Communication; Social Interaction; Restricted interests and repetitive behaviours.

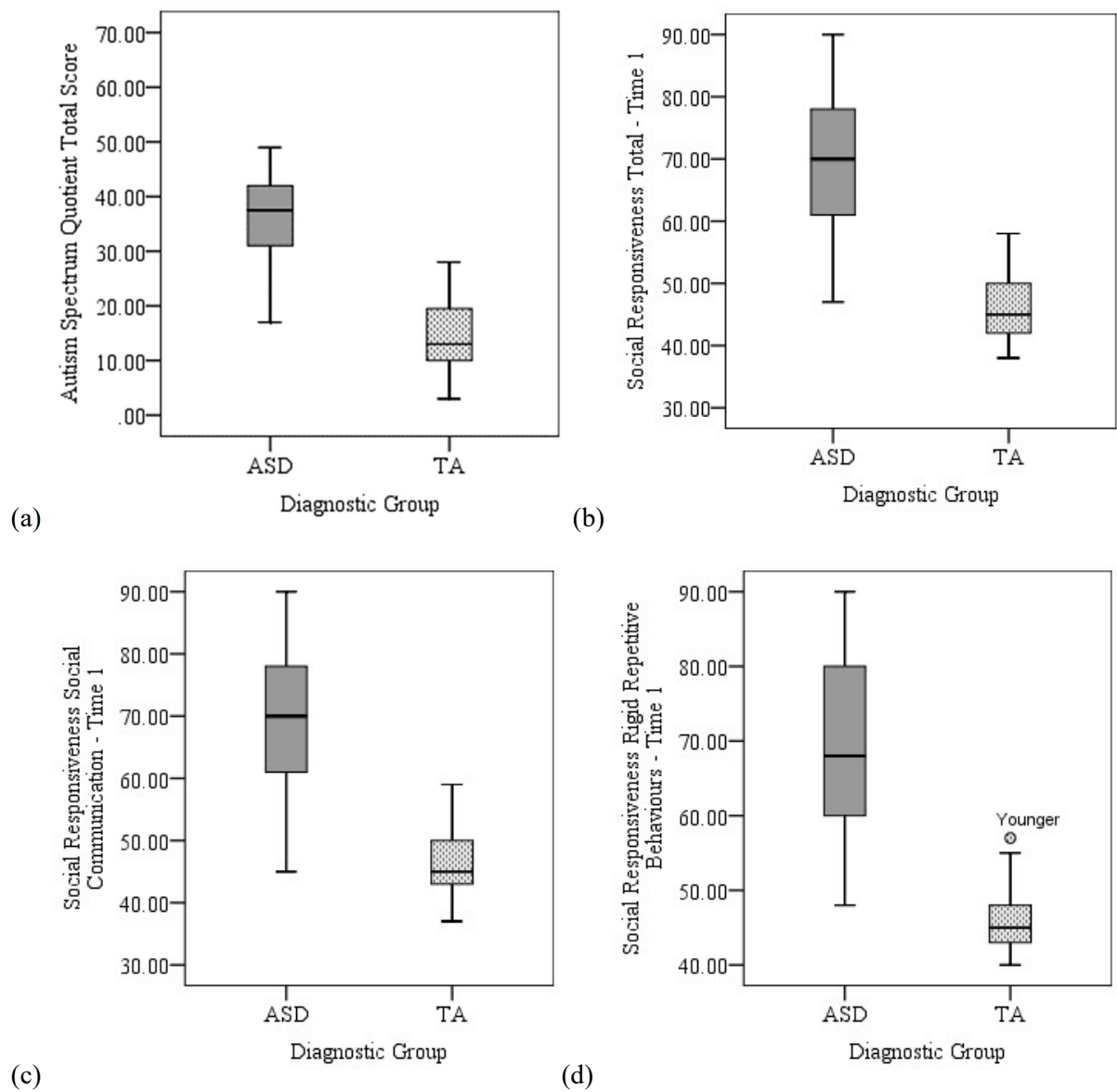


Figure 4.4. Boxplots of Diagnostic Group differences in autistic traits as measured by AQ and SRS-2. show as measured by the (a) AQ, where scores >26 indicate the degree of autistic traits and corresponds with ASD; and (b) SRS Total, (c) SRS SCI, Social Communication Index, (d) SRS RRB, Restricted Interests and Repetitive Behaviours, where SRS T-scores ≥ 59 (range 30-90) indicate a degree of autistic traits the corresponds with ASD-related difficulties.

Table 4.3a. Time 1 Sample characteristics: Cross-sectional comparisons between Diagnostic Groups

Measure	Diagnostic Groups (N=87)		Range	Statistics		
	Mean (SD)			ANOVA <i>F</i> (1,82)	<i>p</i>	η_p^2
	ASD (n=52)	TA (n=35)				
Age	45.53 (15.09)	48.82 (16.65)	18.08-79.74	1.40	<i>n.s.</i>	.017
Gender (m:f) ^a	41:11	21:14	-	-	-	-
YFE ^b	15.00 (2.74)	13.51 (2.80)	8-21	5.51	.021	.063
	Diagnostic Group x Age:			5.74	.019	.065
FSIQ ^c	113.31 (15.97)	110.14 (13.73)	72-138	1.065	<i>n.s.</i>	.013
VCI ^c	115.59 (15.62)	110.57 (13.05)	80-141	3.100	<i>n.s.</i>	.036
PRI ^c	111.51 (16.24)	107.23 (15.28)	58-138	1.650	<i>n.s.</i>	.019
AQ ^d	35.04 (7.85)	13.30 (5.68)	3-49	198.06	<.001	.742
SRS-2 Total ^e	69.87 (12.06)	46.33 (5.23)	38-90	111.96	<.001	.619
SRS-2 SCI ^e	69.16 (11.96)	46.55 (5.85)	37-90	101.24	<.001	.595
SRS-2 RRB ^e	69.91 (12.34)	46.21 (4.64)	38-90	123.35	<.001	.641

Notes: *n.s.* = not significant; ASD = Autism Spectrum Disorder; TA = Typically ageing

^a Gender (m:f) = ratio of male to female participants in each group

^b YFE = Years of formal education, including primary and secondary school, and further education (college, university or vocational).

^c Full-scale IQ (FSIQ) as measured by the Wechsler Adult Intelligence Scales – Third Edition. The FSIQ composite score is comprised of index scores for: Verbal Comprehension Index (VCI) as a measure of verbal IQ, semantic knowledge, and language comprehension; Perceptual Reasoning Index (PRI) as a measure of nonverbal IQ, problem solving and visual information processing. For matching purposes, verbal (VCI), nonverbal (PRI) and FSIQ are used to ensure Diagnostic Groups do not differ on core abilities that may influence their performance on other cognitive tasks. In the ASD group, one participant scored low on the PRI (58) but was not excluded since their overall IQ was within the average norms. Furthermore, exclusion of this participant's data would alter the representativeness of the ASD sample.

^d AQ = Autism Spectrum Quotient. Total scores are reported here (n=93; missing data =37: ASD n=18; TA n=19). Cut-off scores >26 indicate likelihood of autistic traits that correspond with ASD (Woodbury-Smith et al., 2005).

^e SRS-2 = Social Responsiveness Scale–Second Edition. Scores reported here are for SRS Total and index scores for Social Communication (SCI) and Restricted and Repetitive Behaviours (RRB; total data, n=78; missing data, n=52: of which ASD n=23; TA n=29).

Table 4.3b. Time 1 Sample characteristics: Cross-sectional comparisons between Diagnostic Group within Age Groups

Measure	Younger adults (n=42) (age 18:0-49:11 years)		Statistics			Older adults (n=45) (age 50:0-79:9 years)		Statistics		
	yASD (n=26)	yTA (n=16)	ANOVA <i>F</i> (1,40)	<i>p</i>	η_p^2	oASD (n=26)	oTA (n=19)	ANOVA <i>F</i> (1,42)	<i>p</i>	η_p^2
Age	32.54 (7.45)	33.48 (9.28)	.130	<i>n.s.</i>	.003	58.52 (7.60)	61.73 (8.18)	2.44	<i>n.s.</i>	.055
Gender (m:f)	21:5	10:6	-	-	-	20:6	11:8	-	-	-
YFE ^a	14.85 (2.60)	14.88 (2.03)	.001	<i>n.s.</i>	.041	15.16 (2.93)	12.37 (2.89)	9.93	.003	.191
FSIQ ^b	111.70 (17.24)	112.20 (10.60)	.367	<i>n.s.</i>	.009	113.00 (15.66)	108.00 (16.46)	3.69	<i>n.s.</i>	.081
VCI ^b	115.50 (15.59)	110.67 (11.11)	.546	<i>n.s.</i>	.013	118.14 (12.66)	110.47 (15.26)	3.52	<i>n.s.</i>	.077
PRI ^b	110.05 (20.51)	110.60 (12.03)	.158	<i>n.s.</i>	.004	114.76 (13.64)	104.06 (12.29)	5.55	.023	.117
AQ ^c	36.20 (8.33)	12.20 (5.13)	101.85	<.001	.694	35.87 (7.49)	14.22 (6.08)	99.02	<.001	.717
SRS Total ^d	70.95 (10.76)	46.56 (5.75)	67.26	<.001	.658	68.92 (13.84)	46.12 (4.86)	45.73	<.001	.500
SRS SCI ^d	70.10 (11.19)	46.94 (6.02)	55.95	<.001	.615	68.33 (12.78)	46.18 (5.31)	45.30	<.001	.537
SRS RRB ^d	71.90 (10.72)	45.88 (4.83)	81.28	<.001	.699	68.17 (13.59)	46.53 (4.58)	39.66	<.001	.504

Notes: ASD = Autism Spectrum Disorder; TA = Typically ageing

yASD = younger ASD; yTA = younger TA; oASD = older ASD; oTA = older TA

^a YFE = Years of Formal Education. Missing data (n=27): yASD (n=6); yTA (n=8); oASD (n=5); oTA (n=8).

^b FSIQ = Full-Scale IQ; VCI = Verbal Comprehension Index; PRI = Perceptual Reasoning Index

^c AQ = Autism Spectrum Quotient (Baron-Cohen et al., 2001)

^d SRS = Social Responsiveness Scale (Constantino & Gruber, 2012). SCI = Social Communication Index. RRB = Restricted Interests and Repetitive Behaviours.

Having confirmed that groups were well-matched, the exploratory analyses were carried out next. Here, the analyses compared the profiles for Diagnostic Groups and Age Groups with respect to cognitive functions (IQ, language, EF and memory), and Well-being associated with physical and mental health (co-existing conditions, e.g. anxiety, depression) and QoL. In the sections that follow, the analyses deal with the research questions set out at the end of Chapter 2 (p. 67) and summarised earlier in the present chapter. Accordingly, cross-sectional comparisons set out the profile of Age Group differences within Diagnostic groups (e.g. yASD; oASD; and yTA;oTA). First, cognitive functions involving broader IQ abilities (including WMI and PSI), receptive and expressive language, EF and memory are set out in Table 4.4a (p. 138, comparisons between Diagnostic Groups) and in Table 4.4b (p. 141, memory profiles between Diagnostic Groups). Planned contrasts within Age Group between Diagnostic Groups are set out in Table 4.4c (p. 142). Thereafter, Table 4.6a (p. 150) sets out Diagnostic Group comparisons for physical and mental health and QoL. These domains are also explored within Age Group between Diagnostic Groups, as set out in Table 4.6b (p. 151). Finally, the predictors of T1 QoL are presented in Table 4.7 (p. 156).

Cognitive functions

(i) General intellectual ability

There were no Diagnostic Group or Age Group effects on any IQ domains, but an interaction between Diagnostic Groups within Age Group was observed for Processing Speed with a small effect size ($\eta_p^2 .077$), which reflected age-related difficulties in the TA group that replicated findings from the typical literature (e.g. Salthouse, 2010). Whereas, an inverse patterning was observed in the ASD group where the Age Group differences were not significant; yASD showed a similar profile to oTA adults, extending Bowler's (2007) 'ageing analogy' to broader cognitive functioning. By contrast, the oASD adults showed faster processing speed, and therefore higher PSI IQ scores than yASD, which was further comparable to yTA adults (see Table 4.4a, p. 138).

(ii) Language

Three language outcome measures were derived from the CREVT: Receptive language, Expressive language and General Vocabulary. In addition, scores for significant differences between Receptive-Expressive ability (Difference) and Level of overall language (Level). Level of overall language (scale 70-130) is defined as: Very Poor < 70; Poor 70-79; Below Average 80-89; Average 90-110; Above Average 110-120; Superior 121-130; Very Superior > 130 (Table 4.4a, p. 138).

There were no main effects of Diagnostic Group or Gender (all $F(1,57) \leq 2.617, p > .05$, largest $\eta_p^2 .044$) on any of the above language outcome measures. An interaction effect between Diagnostic Groups within Age Groups was observed for Level of overall language ($F(1,60) 6.865, p = .011, \eta_p^2 .103$). Age Group differences were significant for Expressive language ($F(1,61) 4.989, p = .029, \eta_p^2 .064$) and differences between Receptive-Expressive ability ($F(1,60) 5.282, p = .025, \eta_p^2 .080$). Age Group differences in General Vocabulary were on the edge of significance, but with small effect size ($F(1,60) 3.831, p = .055, \eta_p^2 .059$). The Age Group effects appeared to be explained by higher scores for oASD adults compared to yASD, for Expressive language ($F(1,34) 6.434, p = .016, \eta_p^2 .159$), and General Vocabulary ($F(1,34) 5.680, p = .023, \eta_p^2 .143$) and Level of overall language ($F(1,34) 7.020, p = .012, \eta_p^2 .171$). In the TA group, there were no age-related differences between Receptive and Expressive language skills or General Vocabulary (Table 4.4c, p. 142). However, whereas both yTA and OTA adults showed better Expressive than Receptive language, the average significant Difference between Receptive-Expressive ability was greater for oTA adults ($F(1,27) 5.675, p = .025, \eta_p^2 .174$; see Table 4.4c, p. 142). These effects were upheld after Bonferroni adjustment for multiple comparisons.

Gender appeared to mediate Expressive language differences between Diagnostic Groups ($F(1,57) 4.946, p = .030, \eta_p^2 .080$), which appeared to be explained by lower ability for oASD males relative to yASD females, and relative to oASD performance (see Figure 4.5, p. 130). Diagnostic Group x Age Group interaction effect for Level of overall language was still significant but reduced in effect size ($F(1,56) 4.583, p = .037, \eta_p^2 .056$). There were no other Gender effects on language ability (all $F(1,56) \leq 1.161, p > .05$, largest $\eta_p^2 .020$).

To summarise, older TA showed larger differences between Expressive and Receptive language, compared to younger TA whose language scores did not vary as widely. Whereas, the older ASD adults showed better Expressive language and General Vocabulary compared to younger ASD, and higher overall Level of language compared to older TA. Neither ASD group showed clinically significant differences between Receptive and Expressive language.

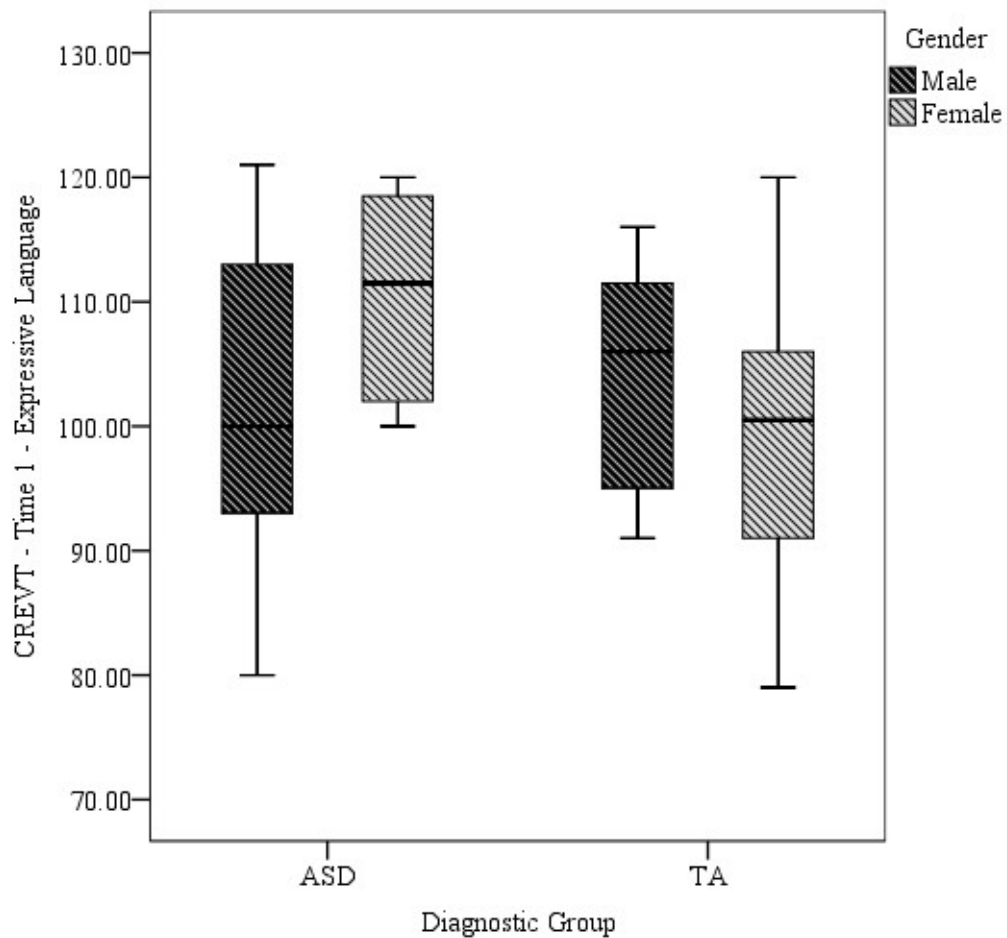


Figure 4.5. Expressive language scores: Gender differences by Diagnostic Group and Age group. Error bars show Standard Error.

(iii) Memory

Summary statistics and analysis of the outcome measures for the CVLT are set out below in Table 4.4a (p. 138; Diagnostic Group comparisonss) and Table 4.4c (p. 142; contrasts within Age Groups between Diagnostic Groups). Then, Table 4.4b (p. 141) presents a summary of the memory profiles of ASD and TA adults, including main effects and interaction effects, following the protocol by Minshew and colleagues (Minshew et al., 1992; Minshew & Goldstein, 2001). These data are organised according to *primary and process measures*, as identified by Woods et al. (2006), and then according to *learning strategies and memory profiles*, following work by Minshew et al., (1992; and see Minshew & Goldstein, 2001). Primary measures involve: Trials 1-5, Short-delay free recall (SDFR), Short-delay cued recall (SDCR), Long-delay free recall (LDFR), Long-delay cued recall (LDCR), and Recall discriminability across Trials 1-5 (d'). Process measures involve: Trial 1, Trial 5, Learning slope Trials 1-5, Recall consistency, Semantic clustering, Total repetitions, Total intrusions, Recognition source discriminability (d'), and Two-alternative forced-choice recognition percentage accuracy (2AFC). Corresponding performance is reported for: a. Incremental learning; b. Immediate recall (Simple memory); and c. Delayed recall (Complex memory). Respectively, these involve Trials 1-5 (and individual Trial outcomes), recall over short delays (free and cued), and recall over long delays (free and cued; and Recognition trials).

Primary and Process memory

The patterning of age-related differences replicated the typical ageing literature in that shows increasing age-related memory difficulties involving self-generated recall (e.g. free recall), but intact memory for supported tasks related to recognition (Craig & Anderson, 1990; Anderson & Craig, 2017). However, contrary to this literature, the older TA adults in our study also showed difficulties with cued recall, compared to younger TA. A similar patterning of difficulties was observed for younger ASD adults, replicating Bowler's (2007) ageing analogy. However, no incremental difficulties were observed in older ASD.

A main effect of Diagnostic Group was observed for differences on all primary measures, and on just one process measure (Trial 1; see Table 4.4b, p. 141). This appeared to be explained by lower

overall scores in primary memory ability, for ASD adults compared to TA adults. A main effect of Age Group was observed on two primary measures: SDFR and Recall discriminability Trials 1-5, which appeared to be explained by differences between oTA and yTA ($p < .001$; Table 4.4c, p. 142). Finally, an interaction effect of Diagnostic Group x Age Group was observed on all primary measures and three process measures: Trial 1, Trial 5, and Recall consistency. Once again, these patterns were explained by differences between oTA and yTA adults (Table 4.4c, p. 142).

Planned contrasts within Diagnostic Groups between Age Groups revealed significantly greater memory difficulties for oTA adults compared to yTA, on all the primary measures and more than half the process measures (see Table 4.3a, p. 126, and 4.3b, p. 127). Consequently, oTA recalled fewer items across Trials 1-5, and in both short- and long-delays irrespective of whether recall was free or cued. Recall discriminability, consistency, and source memory was poorer in oTA adults, who also made more intrusions compared to yTA adults. By contrast, there were no Age Group effects in the ASD group (all $F(1,35) \leq 1.606$, $p > .05$, largest $\eta^2_p .044$). The oASD adults not only performed as well as yASD adults, but mean scores were better across the majority of primary and process memory measures (see Table 4.4a, p. 138, and 4.4c for means and standard deviations).

Incremental learning, Simple and Complex Memory

The profile of memory in ASD replicated the findings by Minshew et al. (1992; 2001) and Bowler, Limoges and Mottron (2009), in relation to the organisation of information and retrieval strategies, and the findings by Ring et al., (2015) of the absence of age-related effects in ASD.

As set out in Table 4.4b (p. 141), the ASD group showed, overall, reduced trial-by-trial learning (number of words per Trial) compared to TA adults ($p < .05$). However, the learning slope across Trials 1-5 was equivalent for all groups, suggesting that ASD adults were able to improve across trials at approximately the same rate as TA adults (mean 1.42 words per Trial, SE .50, 95% CI 1.26-1.58). Further, whereas oTA adults showed significant age-related difficulties in trial-by-trial Incremental learning compared to yTA adults ($p \leq .01$), there were no age-related differences in the ASD group ($p > .05$). Moreover, oASD obtained higher mean scores than yASD and oTA on all

Incremental learning measures, and better learning slope compared to all groups, but these did not reach significance (see Table 4.4a, p. 138 and 4.4c).

In relation to Simple memory, the ASD group showed significantly greater difficulties compared to TA adults on Immediate recall as measured by short-delay free recall ($p < .01$) and cued recall trials, ($p < .05$). However, Age Group contrasts showed that, whereas age-related difficulties were significant for oTA adults compared to yTA adults ($p \leq .001$), there were no age-related effects in the ASD group ($p > .05$).

On Complex memory measures for Delayed recall, the ASD adults showed reduced long-delay free recall, compared to TA adults ($p < .05$), but Diagnostic Group differences on long-delay cued recall ($p > .05$). Further, the significant interaction effects between Diagnostic Groups within Age Group on both Delayed memory measures ($p < .05$; Table 4.4b, p. 141) appeared to be explained by significantly poorer performance by oTA compared to yTA adults ($p \leq .001$; see Table 4.4c, p. 142).

There were no significant differences between the ASD and TA groups on Semantic Clustering ($p > .05$), suggesting that whilst ASD adults were able to organise information semantically, they did not use these organisation strategies to facilitate free recall for Immediate or Delayed memory, replicating previous research on broad memory profiles in ASD (Minshew et al., 1992; Bowler, Matthews & Gardiner, 1997; Minshew & Goldstein, 2001). The patterning of age-related difference in TA adults and the absence of these differences in ASD adults are shown in Figure 4.6 (p. 134). The interpretation of the findings reported here are further supported by the magnitude of effect sizes observed (Tables 4.3a to 4.3c, pp. 136-140) as the proportion of variance explained in each group (e.g. ASD: incremental learning, $\eta_p^2 .14 - .44$; immediate/simple memory, $\eta_p^2 .01$; delayed/complex memory, $\eta_p^2 .04 - .15$; organisation, $\eta_p^2 .03 - .25$; where $\eta_p^2 > .01$ is a small effect, .06 medium, and .14 is a large effect, Cohen, 1988).

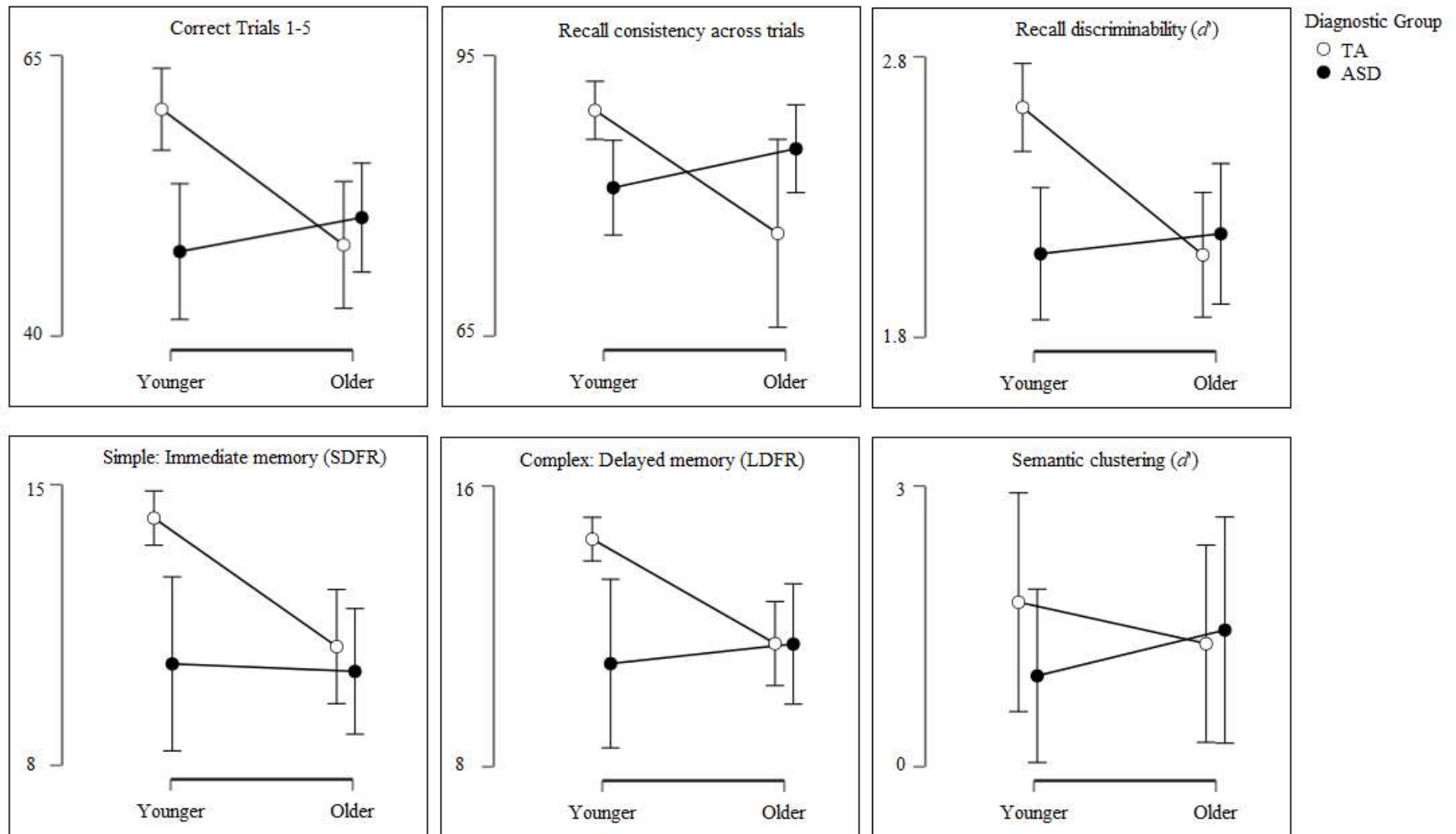


Figure 4.6. Time 1 Cross-Sectional Memory Profiles: Incremental Learning^a, Simple^b and Complex^c memory. Error bars show Standard Error.

(iv) Executive functions

The summary of key outcome measures for the EF tests are set out in Table 4.0 (p. 96) and Table 4.1b (p. 99). As with the previous cognitive measures, the data for the respective EF measures are set out in Table 4.4a (p. 138) for Diagnostic Group comparisons, and Table 4.4c (p. 142) for contrasts within Age Groups between Diagnostic Groups.

Overall, these findings show no Diagnostic Group differences on any EF tests for Working Memory / Planning; Cognitive Flexibility; Attention / Inhibition; and Visual Learning / Episodic Memory (all $F(1,43) \leq 1.35, p > .05$, largest $\eta_p^2 .031$). However, interaction effects between Diagnostic Groups within Age Groups were significant on specific outcome measures, which bore significance for EF in the context of ageing. These findings are summarised in relation to specific EF components.

Planning / Strategy / Working memory

The ASD adults, overall, performed as well as the TA adults on the broad range of EF tasks. However, Age Group differences were significant for spatial Working Memory (SWM task) for the total number of perseverative errors (SWM Between Errors; $F(1,43) 5.53, p .023, \eta_p^2 .114$) and effective use of strategic search to complete each task stage (SWM Strategy; $F(1,43) 5.40, p .025, \eta_p^2 .111$; see Table 4.4a, p. 138 and 4.4c for descriptive scores). Furthermore, interaction effects between Diagnostic Groups within Age Groups were significant on the SWM task for perseverative errors ($F(1,43) 5.45, p .024, \eta_p^2 .112$) and effective use of strategic search ($F(1,43) 4.47, p .040, \eta_p^2 .094$), and on the SOC task for planning accuracy in solving each problem in the minimum number of moves (SOC Planning Accuracy; $F(1,43) 4.11, p .049, \eta_p^2 .087$). These Age Group main effects and interaction effects were explained by poorer performance in oTA and yASD, but not oASD adults, compared to yTA adults (see Table 4.3a, p. 126, and 4.3b, p. 127). By contrast, there were no differences between the older ASD and TA groups (see Table 4.4a, p. 138). Furthermore, within the ASD group, there were no age-related differences in EF components related to Working Memory or Planning (Table 4.4c, p. 142).

Cognitive flexibility / Attention / Working Memory

There were no Diagnostic Group differences in overall cognitive flexibility (IED task), for outcome measures associated with number of stages completed or intradimensional perseverative errors (pre-ED errors; largest $F(1,70) \leq 1.09, p > .05, \eta_p^2 .015$; see Table 4.4a, p. 138 for descriptive data). The number of extradimensional shift errors (EDS errors) were also not significantly different between Diagnostic Groups ($F(1,70) < 1.41, p > .05, \eta_p^2 .020$). However, there was a significant Diagnostic Group x Age Group interaction effect ($F(1,70) 4.89, p .03, \eta_p^2 .065$) in the number of EDS errors, which was explained by better performance in oASD compared to yASD adults. Planned contrasts within Age Groups showed no significant differences between yASD and yTA adults on any cognitively flexibility outcomes (Table 4.4). Whereas, within the older Age Group, the difference in EDS errors was on the edge of significance, with oASD adults making fewer errors in extradimensional shift stage of the task than oTA adults, and all other groups (Table 4.4c, p. 142).

Attention / Inhibition

There were no Diagnostic Group differences in visual sustained Attention (RVP task) in responses accuracy or number of errors (all $F(1,42) < 1.0, p > .05$, largest $\eta_p^2 .017$), but there were significant Age Group differences in mean response time ($F(1,42) 6.88, p .012, \eta_p^2 .141$) which appear to be explained by slower response times by oTA adults than all other groups (see Table 4.4c, p. 142).

However, on Attention / Response Inhibition (RTI task) tasks, performance was significantly different between Diagnostic Groups for mean movement time in the simple task (RTI simple movement (ms); $F(1,42) 6.67, p .013, \eta_p^2 .137$) but not on the 5-choice task, and there were no differences in reaction time or accuracy scores at all levels of the RTI task (all $F(1,42) \leq 3.30, p > .05$, largest $\eta_p^2 .073$).

Visual / Episodic Memory and Learning

On the visual learning and memory task (PAL) there were no Diagnostic Group differences in the number of errors made (PAL Total Errors adjusted), or in the memory score for correctly identified on the first trial of each stage (PAL Memory; all $F(1,49) \leq 2.55, p > .05$, largest $\eta_p^2 .049$). However, there was a significant effect of Age Group for memory score ($F(1,49) 7.13, p .01, \eta_p^2 .127$), which was explained by significantly greater number of errors and lower memory score for older TA and younger ASD adults, but not older ASD adults, compared to younger TA adults (see Table 4.4c, p. 142).

On the visual episodic memory task (DMS) there were no differences between Diagnostic Group or Age Groups and no interaction effects on the outcome measures related to sensitivity to errors (DMS A'; DMS B'') or response accuracy across all stimulus presentations (DMS accuracy simultaneous / delays; all $F(1,50) \leq 2.72, p > .05$, largest $\eta_p^2 .061$). There were no differences within Diagnostic Groups (Table 4.4a, p. 138) or within Age Group contrasts (Table 4.4c, p. 142).

Summary EF performance and ageing effects

The findings from the broad battery of EF assessments replicated the age-related difficulties in TA adults that have been identified in previous literature. The present findings show that older TA adults had specific EF difficulties in planning execution and made poorer use of strategy and more errors in spatial working memory, and in dimensional set shifting. Older TA adults showed slower motor function and attention execution in visual attention and inhibition tasks, and greater difficulties with visual Episodic memory and learning. By contrast, the older ASD adults did not show the same difficulties. There were no increased age-related differences on the EF task performance for older ASD compared to younger ASD adults. The older ASD adults performed better than all groups on dimensional set-shifting and performed at least as well as older TA adults on Working Memory, Planning, Cognitive flexibility, visual Attention and response Inhibition, and visual Learning and Episodic Memory tasks.

Table 4.4a. Time 1 Cognitive function: Cross-Sectional comparisons between Age Groups within Diagnostic Group (ASD)

	Performance / Outcome ¹	ASD (n=37)		Statistics			TA (n=33)		Statistics		
		yASD (n=16)	oASD (n=21)	ANOVA	p	η ² _p	yTA (n=16)	oTA (n=17)	ANOVA	p	η ² _p
General Intellectual ability^a			F(1,40)						F(1,31)		
WMI	Higher	87.24 (13.70)	95.52 (15.40)	3.40	<i>n.s.</i>	.078	98.25 (12.41)	96.11 (17.91)	<1.0	<i>n.s.</i>	.001
PSI	Higher	94.43 (20.89)	101.70 (17.40)	<1.0	<i>n.s.</i>	<.001	109.44 (11.55)	97.47 (13.65)	9.08	.005	.227
		Gender		4.34	.04	.098	Gender		4.13	.051	.118
		Age Group x Gender		4.09	.05	.092					
Language^b				F (1,34)					F (1,27)		
Receptive (RL)	Higher	90.78 (14.04)	96.72 (7.51)	2.51	<i>n.s.</i>	.069	96.29 (6.97)	94.20 (8.76)	<1.0	<i>n.s.</i>	.018
Expressive (EL)	Higher	98.78 (10.94)	108.11 (11.13)	6.43	.016	.159	101.00 (9.33)	103.6 (11.06)	<1.0	<i>n.s.</i>	.017
Difference (RL-EL)	Lower	11.89 (7.81)	12.83 (8.88)	<1.0	<i>n.s.</i>	.020	8.14 (7.79)	12.87 (9.22)	5.68	.025	.174
General Vocabulary	Higher	94.06 (11.89)	102.39 (8.87)	5.68	.023	.143	97.86 (7.23)	98.73 (8.44)	.09	<i>n.s.</i>	.003
Level of Language	Higher	2.50 (.86)	3.11 (.47)	7.02	.012	.171	2.93 (.27)	2.87 (.52)	.16	<i>n.s.</i>	.006
Memory^c				F (1,35)					F (1,29)		
<u>Primary measures</u>											
Trial 1-5	Higher	47.53 (12.54)	50.56 (9.75)	<1.0	<i>n.s.</i>	.019	60.20 (6.59)	48.13 (10.60)	14.27	.001	.330
SDFR	Higher	10.63 (4.50)	10.44 (3.15)	<1.0	<i>n.s.</i>	.001	14.27 (1.22)	11.06 (2.67)	18.03	<.001	.383
SDCR	Higher	11.53 (4.02)	11.78 (3.19)	<1.0	<i>n.s.</i>	.001	14.67 (1.35)	11.75 (2.59)	15.12	.001	.343
LDFR	Higher	11.05 (4.98)	11.61 (3.45)	<1.0	<i>n.s.</i>	.004	14.60 (1.12)	11.63 (2.25)	21.29	<.001	.423
LDCR	Higher	11.37 (4.60)	12.33 (3.45)	<1.0	<i>n.s.</i>	.015	14.93 (1.10)	11.75 (2.38)	22.30	<.001	.435
Recall (<i>d'</i>) Trial 1-5	Higher +	2.08 (.49)	2.15 (.50)	<1.0	<i>n.s.</i>	.005	2.60 (.28)	2.08 (.42)	16.59	<.001	.364
<u>Process measures</u>											
Trial 1	Higher	5.84 (1.61)	6.28 (1.78)	<1.0	<i>n.s.</i>	.017	8.07 (2.22)	6.00 (1.55)	9.14	.005	.240
Trial 5	Higher	11.32 (3.82)	12.61 (2.38)	1.515	<i>n.s.</i>	.041	14.20 (1.26)	12.00 (2.83)	7.63	.010	.208
Learning slope Trials 1-5	Higher	1.32 (.79)	1.49 (.69)	<1.0	<i>n.s.</i>	.014	1.43 (.52)	1.45 (.56)	<1.0	<i>n.s.</i>	<.001
Recall consistency %	Higher	81.00 (10.50)	85.17 (9.43)	1.606	<i>n.s.</i>	.044	89.27 (5.60)	76.13 (18.85)	6.72	.015	.188
Semantic clustering	Higher +	1.00 (1.92)	1.49 (2.43)	<1.0	<i>n.s.</i>	.013	1.79 (2.11)	1.34 (1.99)	<1.0	<i>n.s.</i>	.012
Total repetitions	Lower	6.845 (4.79)	6.28 (5.31)	<1.0	<i>n.s.</i>	.003	4.47 (4.66)	5.38 (3.03)	<1.0	<i>n.s.</i>	.014
Total intrusions	Lower	3.00 (4.28)	5.00 (8.17)	<1.0	<i>n.s.</i>	.025	1.20 (1.74)	4.00 (3.90)	6.51	.016	.183
Source (<i>d'</i>)	Higher +	2.98 (1.20)	2.75 (1.05)	<1.0	<i>n.s.</i>	.011	3.50 (.39)	2.95 (.77)	6.17	.019	.176
2AFC recog. %	Higher	98.05 (5.89)	97.00 (4.24)	<1.0	<i>n.s.</i>	.011	99.60 (1.55)	91.88 (24.66)	1.46	<i>n.s.</i>	.048
Executive function^d											
<u>Working memory / Planning</u>				F(1,28)					F(1,15)		
SWM											
Between Errors, 4-8 boxes	Lower	29.05 (21.44)	31.33 (19.75)	<1.0	<i>n.s.</i>	.000	11.33 (12.27)	40.73 (21.79)	10.12	.006	.403
Strategy	Lower	32.45 (6.16)	34.38 (4.55)	<1.0	<i>n.s.</i>	.001	28.78 (6.65)	35.27 (3.82)	8.88	.009	.372

		Performance / Outcome ¹	ASD (n=37)		Statistics			TA (n=33)		Statistics		
			yASD (n=16)	oASD (n=21)	ANOVA	p	η_p^2	yTA (n=16)	oTA (n=17)	ANOVA	p	η_p^2
SOC												
Planning time (ms), 2 moves	Lower	2507.26 (2820.20)	2068.21 (815.98)	<1.0	<i>n.s.</i>	.018	1790.58 (732.77)	1393.36 (1012.78)	<1.0	<i>n.s.</i>	.045	
Planning time (ms), 5 moves	Lower	19203.88 (19956.60)	21638 (13143.61)	<1.0	<i>n.s.</i>	.012	15466.25 (9459.34)	15626.23 (12133.30)	<1.0	<i>n.s.</i>	<.001	
Planning accuracy	Higher	8.50 (3.22)	9.57 (2.06)	<1.0	<i>n.s.</i>	.002	10.50 (1.05)	8.27 (2.33)	4.84	.044	.244	
<u>Cognitive flexibility / Attention / WM</u>			<i>F</i> (1,41)				<i>F</i> (1,29)					
IED												
Stages complete	Higher	7.59 (2.70)	8.55 (.80)	2.79	<i>n.s.</i>	.064	7.92 (1.98)	7.89 (1.97)	<1.0	<i>n.s.</i>	.000	
Pre-ED errors	Lower	7.19 (9.33)	8.64 (10.43)	<1.0	<i>n.s.</i>	.006	9.86 (9.64)	11.17 (11.29)	<1.0	<i>n.s.</i>	.002	
EDS errors	Lower	13.24 (12.71)	6.27 (2.76)	6.30	.016	.133	7.86 (5.63)	10.33 (9.20)	<1.0	<i>n.s.</i>	.018	
Total errors (adj).	Lower	41.33 (54.71)	22.91 (20.56)	2.18	<i>n.s.</i>	.050	35.29 (44.09)	40.33 (49.39)	<1.0	<i>n.s.</i>	.001	
<u>Attention / Inhibition</u>			<i>F</i> (1,28)				<i>F</i> (1,14)					
RVP												
A' (0.00–1.00)	Higher	.93 (.07)	.93 (.05)	<1.0	<i>n.s.</i>	.000	.95 (.05)	.91 (.05)	<1.0	<i>n.s.</i>	.034	
B'' (-1.00–+1.00)	Higher +	.83 (.50)	.95 (.08)	<1.0	<i>n.s.</i>	.027	.94 (.08)	.93 (.09)	<1.0	<i>n.s.</i>	.014	
Mean latency (ms)	Lower	401.50 (94.50)	448.74 (67.23)	1.46	<i>n.s.</i>	.050	380.64 (74.76)	493.97 (134.54)	4.21	.059	.231	
RTI												
Simple accuracy	Higher	8.89 (.32)	8.71 (.47)	1.17	<i>n.s.</i>	.040	8.67 (.71)	8.90 (.32)	1.21	<i>n.s.</i>	.079	
Simple movement	Lower	504.33 (118.88)	539.51 (60.87)	<1.0	<i>n.s.</i>	.032	426.86 (110.81)	563.69 (125.25)	4.80	.046	.255	
Simple reaction	Lower	301.97 (88.66)	312.56 (41.84)	<1.0	<i>n.s.</i>	.001	280.17 (54.76)	326.27 (56.61)	1.82	<i>n.s.</i>	.115	
5-choice accuracy	Higher	7.83 (.38)	7.93 (.27)	<1.0	<i>n.s.</i>	.029	7.89 (.33)	7.90 (.32)	1.31	<i>n.s.</i>	.086	
5-choice movement	Lower	358.32 (145.44)	360.53 (73.51)	<1.0	<i>n.s.</i>	.029	307.44 (72.40)	339.58 (52.49)	2.02	<i>n.s.</i>	.126	
5-choice reaction	Lower	303.01 (60.72)	331.26 (52.04)	<1.0	<i>n.s.</i>	.025	298.00 (71.04)	335.55 (61.69)	2.65	<i>n.s.</i>	.159	
<u>Visual / Episodic memory and learning</u>												
PAL												
Total Error adj.	Lower	13.70 (28.70)	20.61 (23.40)	<1.0	<i>n.s.</i>	.007	3.20 (3.01)	12.91 (7.35)	15.07	.001	.442	
Memory score	Higher	20.24 (4.96)	19.07 (4.22)	<1.0	<i>n.s.</i>	.017	23.80 (1.99)	19.00 (3.10)	17.42	.001	.478	
DMS												
Correct % sim.	Higher	89.52 (12.29)	90.44 (7.08)	<1.0	<i>n.s.</i>	.005	95.25 (4.63)	91.50 (5.92)	2.491	<i>n.s.</i>	.122	
Correct % 0ms	Higher	89.44 (13.05)	90.00 (11.09)	<1.0	<i>n.s.</i>	.001	97.00 (6.75)	91.00 (7.38)	3.600	<i>n.s.</i>	.167	
Correct % 4000ms	Higher	91.67 (14.25)	89.29 (11.41)	<1.0	<i>n.s.</i>	.009	97.00 (4.83)	93.00 (10.59)	1.180	<i>n.s.</i>	.062	
Correct % 12000ms	Higher	77.22 (22.40)	83.57 (17.37)	<1.0	<i>n.s.</i>	.025	88.00 (10.33)	85.00 (10.80)	<1.0	<i>n.s.</i>	.022	
A' (0.00 to 1.00)	Lower	.16 (.68)	.65 (.11)	1.47	<i>n.s.</i>	.197	.67 (.06)	.5 (.07)	1.98	<i>n.s.</i>	.498	
B'' (-1.00 to +1.00)	Higher -	-.72 (.43)	-.75 (.51)	<1.0	<i>n.s.</i>	.001	-.64 (.62)	-.76 (.47)	.213	<i>n.s.</i>	.015	

Notes: ms = milliseconds.¹ Performance / Outcome: this column is a reminder of the measure of good performance on each measure; where higher (or lower) scores indicate better performance, respectively. 'Higher +' indicates better performance for positive scores, whilst 'Higher -' indicates better performance for higher negative scores (usually in reference to d' , A' or B'' which ranges from '–' to '+' values).

^a General intellectual ability index scores derived from the WAIS index scores reported here. Normative range 70-130 (mean 100, SD 15). WMI = Working Memory Index, as a measure of updating, arithmetic, verbal span; and PSI = Processing Speed Index, as a measure of attention and organisation of information.

^b Language scores derived from the CREVT for Expressive and Receptive language ability and General Vocabulary. Normative range 70-130 (mean 100, SD 15). Level of language is scored as: 0=Very Poor, <70; 1=Poor, 70-79; 2=Below Average, 80-89; 3=Average, 90-110; 4=Above Average, 111-120; 5=Superior, 121-130; 6=Very Superior, >130. Significant differences between Receptive and Expressive language: not significant, <12; statistically significant, 12-26; clinically significant, >26).

Recall (d') T1-5 = Recall discriminability (d') Trial 1-5, as the ability to detect all correct target words from distractor items. Scores range from +3.73 to -3.73; positive values indicate 100% correct (all target responses) and negative values indicate 100% incorrect (all distractor responses).

Source (d') = Source recognition discriminability (d') ability to accurately identify (recall or recognition) target items and avoid inclusion of distractor items (interference).

This measure accounts for hits and false alarms. Scores range from +4.02 to -4.02; positive values indicate 100% correct (all target responses) and negative values indicate 100% incorrect (all distractor responses).

EF SOC: planning time = initial thinking time to move; planning accuracy = number of problems solved in minimum number of moves.

^c Memory scores are reported for: Incremental learning; Immediate recall (Simple memory); and Delayed recall (Complex memory). Respectively, these involve Trials 1-5 (and individual Trial outcomes), recall over short delays (free and cued), and recall over long delays (free and cued; and Recognition trials). SDFR = short-delay free recall; SDCR = short-delay cued recall; LDFR = long-delay free recall; LDCR = long-delay cued recall; 2AFRC = 2-Alternatives Forced Choice recognition.

^d Executive function scores. SWM = Spatial Working Memory; SOC = Stockings of Cambridge; IED = Intra/Extradimensional Set Shift; RVP = Rapid Visual Information Processing; RTI = Reaction Time; PAL = Paired Associated Learning; DMS = Delayed Matching to Sample. Signal Detection Theory ratios = A' (A-Prime; range 0.00 to 1.00) and B'' (B-double Prime; range -1.00 to +1.00).

Table 4.4b. Time 1 Memory Profiles: Cross-sectional comparisons between Diagnostic Groups.
Primary, Process, Incremental Learning^a, Simple^b and Complex^c memory and organisational^d mechanisms of memory.

	Diagnostic Groups (N=68)	Statistics		
	ASD (n=37), TA (n=31)	ANOVA	<i>p</i>	η_p^2
Memory	Main effect	<i>F</i>(1,64)		
<u>Primary measures</u>				
Trial 1-5 ^a	Diagnostic Group	4.20	.045	.062
	Diagnostic Group x Age Group	9.13	.004	.125
SDFR ^b	Diagnostic Group	7.38	.008	.103
	Age Group	4.69	.034	.068
	Diagnostic Group x Age Group	3.72	.058	.055
SDCR ^b	Diagnostic Group	4.43	.039	.065
	Diagnostic Group x Age Group	4.59	.036	.067
LDFR ^c	Diagnostic Group	4.61	.036	.067
	Diagnostic Group x Age Group	4.53	.037	.066
LDCR ^c	Diagnostic Group	3.50	.066	.052
	Diagnostic Group x Age Group	6.78	.011	.096
Recall discrim. Trials 1-5	Diagnostic Group	4.35	.041	.064
	Age Group	4.51	.038	.066
	Diagnostic Group x Age Group	7.77	.007	.108
<u>Process measures</u>				
Trial 1 ^a	Diagnostic Group	4.98	.029	.072
	Diagnostic Group x Age Group	8.23	.006	.114
Trial 5 ^a	Diagnostic Group x Age Group	6.59	.013	.093
Learning slope Trials 1-5 ^a	-	<1.0	<i>n.s.</i>	.001
Recall consistency % ^a	Diagnostic Group x Age Group	8.72	.004	.120
Semantic clustering ^d	-	<1.0	<i>n.s.</i>	.006
Total repetitions ^d	-	2.17	<i>n.s.</i>	.033
Total intrusions ^d	-	1.22	<i>n.s.</i>	.019
Source recognition discrim. (<i>d'</i>) ^d	-	2.52	<i>n.s.</i>	.038
2AFC recognition % ^e	-	<1.0	<i>n.s.</i>	.005

Notes: discrim. = discriminability; ability to accurately identify (recall or recognition) target items and avoid inclusion of distractor items (interference). This measure accounts for hits and false alarms. Recall discriminability (*d'*) scores range from +3.73 to -3.73, whilst Recognition discriminability scores range from +4.02 to -4.02; positive values indicate 100% correct (all target responses) and negative values indicate 100% incorrect (all distractor responses).

^a Incremental learning.

^b Immediate recall, simple memory.

^c Delayed recall, complex memory.

^d organisation strategies.

^e Task support recognition memory.

Table 4.4c. Time 1 Cognitive function: Cross-sectional comparisons between Age Groups within Diagnostic Groups

	Performance / Outcome ¹	Younger (n=34)	Statistics			Older (n=34)			
		yASD (n=19); yTA (n=15)	ANOVA	Sig.	η_p^2	oASD (n=18); oTA (n=16)	ANOVA	Sig.	η_p^2
WMI	Higher	Diagnostic	6.36	.016	.154		<1.0	n.s.	<.001
PSI	Higher	Diagnostic	6.68	.014	.160		<1.0	n.s.	.018
		Gender	11.03	.002	.270	Diagnostic x Age	6.42	.013	.080
<i>Language^b</i>									
Receptive (RL)	Higher		1.80	n.s.	.057		<1.0	n.s.	.025
Expressive (EL)	Higher		<1.0	n.s.	.012		1.35	n.s.	.042
Difference (RL-EL)	Lower		2.81	n.s.	.086		<1.0	n.s.	.002
General Vocabulary	Higher		1.11	n.s.	.036		1.45	n.s.	.045
Level overall Language	Higher		3.23	n.s.	.097		2.02	n.s.	.061
<i>Memory^c</i>									
<u>Primary measures</u>									
Trial 1-5	Higher	Diagnostic	12.53	.001	.281		<1.0	n.s.	.015
SDFR	Higher	Diagnostic	9.20	.005	.223		<1.0	n.s.	.012
SDCR	Higher	Diagnostic	8.37	.007	.207		<1.0	n.s.	<.001
LDFR	Higher	Diagnostic	7.27	.011	.185		<1.0	n.s.	<.001
LDCR	Higher +	Diagnostic	8.58	.006	.211		<1.0	n.s.	.010
<u>Process measures</u>									
Trial 1	Higher	Diagnostic	11.50	.002	.264		<1.0	n.s.	.007
Trial 5	Higher	Diagnostic	7.84	.009	.197		<1.0	n.s.	.014
Learning slope Trials 1-5	Higher		.25	n.s.	.008		<1.0	n.s.	.001
Recall consistency %	Higher	Diagnostic	7.57	.010	.191		3.24	n.s.	.092
Semantic clustering	Higher +		1.29	n.s.	.039		<1.0	n.s.	.001
Total repetitions	Lower		2.11	n.s.	.062		<1.0	n.s.	.011
Total intrusions	Lower		2.33	n.s.	.068		<1.0	n.s.	.006
Source recognition discrim. (d')	Higher +		2.59	n.s.	.075		<1.0	n.s.	.012
2AFC recognition %	Higher		<1.0	n.s.	.030		<1.0	n.s.	.023
<i>Executive function^d</i>									
<u>Working memory / Planning</u>			<i>F</i> (1,21)			<i>F</i> (1,22)			
SWM	Lower								
Between errors (4 to 8 boxes)	Lower	Diagnostic	5.32	.029	.160		2.24	n.s.	.093
Strategy			2.20	n.s.	.095		2.45	n.s.	.100
SOC	Lower								

	Performance / Outcome ¹	Younger (n=34)		Statistics			Older (n=34)		
		yASD (n=19); yTA (n=15)		ANOVA	Sig.	η_p^2	oASD (n=18); oTA (n=16)		
Planning time – 2moves (ms)	Lower			1.99	<i>n.s.</i>	.086		2.16	<i>n.s.</i>
Planning time – 5 moves (ms)	Higher			<1.0	<i>n.s.</i>	.017		2.87	<i>n.s.</i>
Planning accuracy				<1.0	<i>n.s.</i>	.009		<1.0	<i>n.s.</i>
<u>Cognitive flexibility/Attention/WM</u>				<i>F</i>(1,32)			<i>F</i>(1,38)		
IED	Higher								
Stages complete	Lower			<1.0	<i>n.s.</i>	.006		2.05	<i>n.s.</i>
Pre-ED errors	Lower			<1.0	<i>n.s.</i>	.025		<1.0	<i>n.s.</i>
EDS errors	Lower			1.78	<i>n.s.</i>	.053		3.876	.056
Total errors (adj).				<1.0	<i>n.s.</i>	.002		2.269	<i>n.s.</i>
<u>Attention / Inhibition</u>				<i>F</i>(1,21)			<i>F</i>(1,22)		
RVP	Higher								
<i>A'</i> (0.00 to 1.00)	Higher +			<1.0	<i>n.s.</i>	.002		<1.0	<i>n.s.</i>
<i>B''</i> (-1.00 to +1.00)	Lower			<1.0	<i>n.s.</i>	.017		<1.0	<i>n.s.</i>
Mean latency (ms)				<1.0	<i>n.s.</i>	.024		1.48	<i>n.s.</i>
RTI	Higher								
Simple accuracy score	Lower			1.73	<i>n.s.</i>	.076		<1.0	<i>n.s.</i>
Simple mean movement time (ms)	Lower			2.13	<i>n.s.</i>	.092		<1.0	<i>n.s.</i>
Simple mean reaction time (ms)	Higher			<1.0	<i>n.s.</i>	.022		<1.0	<i>n.s.</i>
Five-choice accuracy score	Lower			<1.0	<i>n.s.</i>	.003		<1.0	<i>n.s.</i>
Five-choice movement time (ms)	Lower			<1.0	<i>n.s.</i>	.030		<1.0	<i>n.s.</i>
Five-choice reaction time (ms)	Lower			<1.0	<i>n.s.</i>	.037		<1.0	<i>n.s.</i>
<u>Visual/Episodic Memory/Learning</u>				<i>F</i>(1,30)			<i>F</i>(1,19)		
PAL									
Total errors (adj.)	Lower			1.69	<i>n.s.</i>	.063		<1.0	<i>n.s.</i>
Memory score	Higher		Diagnostic	4.67	.041	.157		<1.0	<i>n.s.</i>
DMS									
Percent correct – simultaneous	Higher			2.69	<i>n.s.</i>	.094		<1.0	<i>n.s.</i>
Percent correct – 0ms delay	Higher			2.89	<i>n.s.</i>	.100		<1.0	<i>n.s.</i>
Percent correct – 4000ms delay	Higher			1.30	<i>n.s.</i>	.048		<1.0	<i>n.s.</i>
Percent correct – 12000ms delay	Higher			2.04	<i>n.s.</i>	.073		<1.0	<i>n.s.</i>
<i>A'</i> (0.00 to 1.00)	Lower			1.00	<i>n.s.</i>	.166		<1.0	<i>n.s.</i>
<i>B''</i> (-1.00 to +1.00)	Higher -			<1.0	<i>n.s.</i>	.006		<1.0	<i>n.s.</i>

Notes: Diagnostic = main effect of Diagnostic Group; Diagnostic x Age = interaction effect between Diagnostic Group and Age Group.

¹ Performance / Outcome: this column is a reminder of the measure of good performance on each measure; where higher (or lower) scores indicate better performance, respectively; 'higher +' indicates better performance for positive scores (usually in reference to d' which ranges from '-' to '+' values).

^a General intellectual ability index scores derived from the WAIS index scores reported here. Normative range 70-130 (mean 100, SD 15).

^b Language scores derived from the CREVT for Expressive and Receptive language ability and General Vocabulary. Normative range 70-130 (mean 100, SD 15).
discrim. = discriminability; ability to accurately identify (recall or recognition) target items and avoid inclusion of distractor items (interference). This measure accounts for hits and false alarms. Recall discriminability (d') scores range from +3.73 to -3.73, whilst Recognition discriminability scores range from +4.02 to -4.02; positive values indicate 100% correct (all target responses) and negative values indicate 100% incorrect (all distractor responses).

Physical and Mental health

Participant self-reports of difficulties related to social functioning, and mental and physical health are outlined in Table 4.5 (p. 148). Corresponding data from the T1 assessments are set out in Table 4.6a (p. 150) for comparisons between Diagnostic Groups, and in Table 4.6b (p. 151) for contrasts within Age Groups between Diagnostic Groups (and see Figure 4.7, p. 149).

Mental health concerns were greater for ASD adults than TA adults (Table 4.6a, p. 150), with respect to Anxiety and Levels of Anxiety as measured by BAI-II outcome scores, and greater on Depression and Levels of Depression as measured by BDI-II outcome scores. Planned contrasts between Age Groups within Diagnostic Groups were not significant for TA or ASD adults (all $F \leq 2.51$, $p \geq .12$, $\eta_p^2 \leq .073$). Data from the PHQ concurred with Diagnostic Group differences in Anxiety and Depression. In particular, these differences were clinically significant in identifying the following specific conditions in ASD adults: Major Depressive syndrome ($F(1,70) 9.11$, $p = .004$, $\eta_p^2 .115$), Panic syndrome ($F(1,70) 4.19$, $p = .045$, $\eta_p^2 .056$) and Other Anxiety syndrome ($F(1,70) 6.06$, $p = .016$, $\eta_p^2 .080$). Furthermore, ASD adults reported higher tendency to Binge Eating Disorder ($F(1,70) 7.84$, $p = .007$, $\eta_p^2 .101$) than TA adults, and although clinical cut-offs were not met for Somatic complaints significantly more associated symptoms were reported by ASD adults ($\bar{x}$ 5.49, SD 3.28) than TA adults ($\bar{x}$ 3.29, SD 2.69 ; $F(1,70) 9.45$, $p = .003$, $\eta_p^2 .119$). Somatoform conditions that presented greatest difficulties for ASD adults were associated with pain (stomach 27.6%; back 13.8%; joints or limbs 41.4%), digestive problems (bowel 20.7%; indigestion 41.4%) and neurophysiology symptoms (heart racing 13.8%; shortness of breath 20.7%). For TA adults the greatest somatoform difficulties were pain (stomach 13.3%; back 26.7%; joints or limbs 13.3%) and digestive problems (bowel 26.7%; indigestion 13.3%). The conditions just mentioned were associated with greater Difficulty in Daily Living for ASD adults ($F(1,70) 25.70$, $p < .001$, $\eta_p^2 .269$) compared to TA adults.

Planned contrasts within Diagnostic Groups revealed no significant Age Group differences between younger and older TA adults (all $F \leq 1.27$, $p \geq .269$, $\eta_p^2 \leq .073$) or between younger and older ASD adults (all $F \leq 3.43$, $p \geq .071$, $\eta_p^2 \leq .077$).

Planned contrasts between Diagnostic Groups within Age Groups confirmed that younger ASD adults reported a significantly higher prevalence of Major Depressive syndrome ($F(1,35) 6.05, p = .019, \eta_p^2 .147$) and greater symptoms associated with Other Anxiety syndrome, Binge Eating Disorder and Somatic complaints (all $F(1,35) 4.73, p = .036, \eta_p^2 .119$), compared to younger TA adults. In comparisons of older adults, there were no significant Diagnostic Group differences on any of the clinically relevant outcomes (all $F \leq 3.15, p \geq .084, \eta_p^2 \leq .083$). Although scores did not meet clinical cut-offs, the older ASD adults reported more symptoms associated with Depression ($F(1,35) 5.97, p = .020, \eta_p^2 .146$) and Anxiety ($F(1,35) 8.03, p = .008, \eta_p^2 .187$) compared to older TA adults.

Difficulties with Daily Living were significantly more challenging for younger ASD ($F(1,35) 28.27, p < .001, \eta_p^2 .447$) and older ASD adults ($F(1,35) 4.73, p = .037, \eta_p^2 .119$) compared to the respective TA Age Groups.

Quality of Life (QoL) and Subjective Well-being (SWB)

Our findings replicate observation from previous studies that have identified poorer QoL in younger autistic adults (Kamp-Becker et al., 2010) and older autistic adults (Totsika et al., 2010; van Heijst & Geurts, 2015; Roestorf & Bowler, 2016; Ayres, Parr, Rodgers et al., 2017). Compared to the TA adults in our study, subjective well-being (SWB) and all domains of QoL were significantly poorer for the ASD adults (all $F(1,70) \geq 8.69, p \leq .004, \eta_p^2 \geq .110$; see Table 4.6a, p. 150), who also reported lower levels of Support from others in everyday life ($F(1,42) 9.59, p = .003, \eta_p^2 .186$; see Figure 4.8, p. 153). There were no Age Group differences with TA or ASD Diagnostic Groups (all $F < 1, p \geq .69, \eta_p^2 \leq .01$). However, older Age was associated with poorer Social QoL outcomes in TA adults ($r(32) -.44, p = .02$) but no other domains (all $r(32) \leq .27, \text{all } p \geq .14$). Age was not a factor in the reduced QoL of ASD adults (all $r(44) \leq .17, \text{all } p \geq .25$). When exploring the specific domains associated with poorer SWB in ASD, whilst all factors were significantly lower than TA adults, the most concerning factors for ASD adults were related to lack of Personal Relationships and feeling isolated from the Community; lack of Achievement; and concerns about Health and Future, respectively – ASD scores for these factors were also below the general population mean scores. Standard of Living and feeling safe (Safety) were scores amongst the highest SWB in the ASD group,

but these factors were still significantly lower than SWB in TA adults. In TA adults, the greatest SWB concerns were sense of Achievement, Health concerns, social isolation from the Community, whilst Future, Standard of Living, Personal Relationships and Safety featured the highest levels of satisfaction for TA adults (Table 4.6a, p. 150). Age did not contribute to SWB in TA adults (all $F(1,28) < 1, p \geq .35, \eta_p^2 \leq .03$). However, in the ASD group, Age was related to SWB for Standard of Living, Personal Relationships, and concerns about the Future (Table 4.6a, p. 150). This reflected higher SWB scores for older ASD adults compared to younger ASD adults, for each of these factors.

Overall, the profile of substantially affected QoL and subjective well-being in ASD holds important implications for the lifespan outcome for autistic adults. The potential influence of co-existing physical and mental health conditions and cognitive functions on QoL are explored next.

Self-reported conditions and well-being

ASD participants reported multiple physical health conditions and social difficulties, as displayed in Figure 4.7 (p. 149). The type of co-existing conditions reported included alexithymia, anxiety and depression (mental health), sleep disturbances (e.g. difficulty falling asleep; frequent waking), and sensory sensitivities (hyper and hypo) and related sensory conditions (e.g. rhinitis; psoriasis). Social difficulties were associated with communication (e.g. from social nuances and figurative language, to understanding complex instructions), and extended to social anxiety and loneliness or social isolation. The majority of younger ASD adults (69%) and almost half the older ASD adults (49%) reported difficulties in each of these areas (see Table 4.5, p. 148).

Table 4.5. Percentage of self-reported co-occurring conditions in ASD adults

Condition	ASD younger	ASD older
<i>Mental health</i>		
Anxiety	75%	62%
Depression	75%	54%
Alexithymia	38%	38%
<i>Sleep</i>		
Sleep Disturbance	88%	46%
<i>Sensory</i>		
Hypersensitivity	88%	69%
Hyposensitivity	13%	31%
Sensory condition	63%	15%
<i>Social</i>		
Social communication	88%	54%
Social anxiety	88%	62%
Lonely / socially isolated	75%	62%

Notes: self-reported conditions previously diagnosed and that are currently or previously treated.

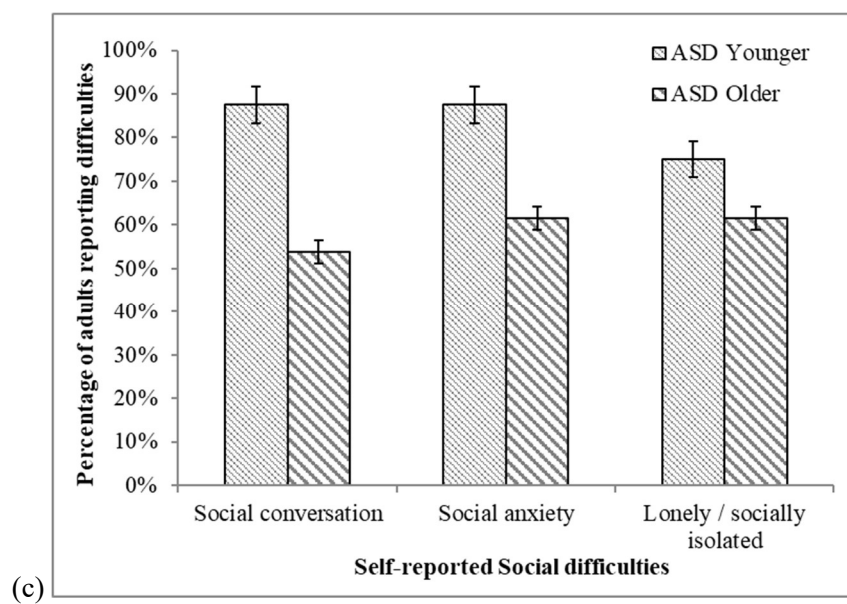
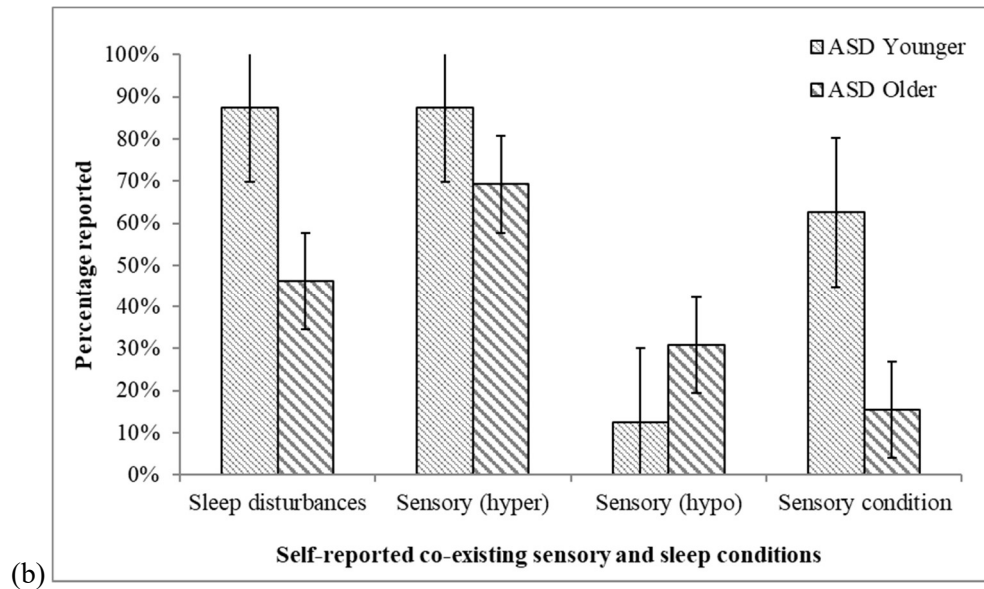
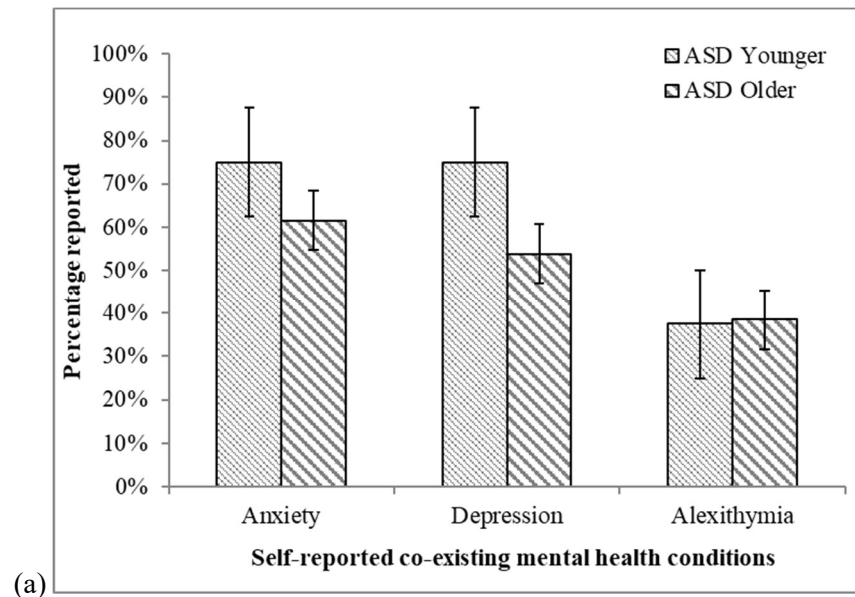


Figure 4.7. Physical and Mental health conditions reported by ASD adults.

Table 4.6a. Time 1 Mental Health and Quality of Life: Cross-sectional comparisons between Diagnostic Groups

Measure	Diagnostic Groups (N=87)		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η_p^2
	ASD (n=52)	TA (n=35)				
<i>Mental Health</i>				<i>F(1,75)</i>		
Anxiety ^a	13.53 (8.80)	5.18 (5.84)	0-35	23.23	<.001	.236
Levels of Anxiety ^a	2.16 (.95)	1.09 (.79)	0-4	28.70	<.001	.277
Depression ^b	17.13 (13.54)	4.76 (4.27)	0-37	26.58	<.001	.262
Levels of Depression ^b	1.71 (1.82)	.09 (.29)	0-5	26.47	<.001	.261
<i>Subjective Well-being & Quality of Life</i>				<i>F(1,70)</i>		
Subjective wellbeing ^c	39.40 (12.17)	52.81 (9.12)	5-67	27.42	<.001	.281
Global life satisfaction ^c	53.95 (21.40)	74.40 (12.62)	10-100	23.37	<.001	.250
Personal Relationships	41.46 (16.67)	74.81 (11.85)	0-100	30.35	<.001	.312
Community	42.01 (19.97)	69.63 (11.29)	0-100	16.47	<.001	.197
Achieving	42.01 (17.70)	65.56 (9.80)	0-100	13.58	<.001	.168
Health	45.80 (15.32)	68.52 (10.93)	0-100	13.75	<.001	.170
Future	47.15 (18.47)	72.96 (12.17)	0-100	15.72	<.001	.190
Standard of Living	57.45 (12.32)	75.19 (6.32)	0-100	11.47	.001	.146
Safety	58.54 (14.59)	85.19 (3.86)	0-100	24.59	<.001	.268
Overall QoL ^d	59.88 (20.51)	80.65 (15.42)	25-100	24.50	<.001	.259
Health ^d	52.33 (28.25)	70.1 (22.75)	0-100	8.69	.004	.110
Physical ^d	60.00 (18.47)	82.23 (15.00)	25-100	30.84	<.001	.306
Psychological ^d	55.00 (19.05)	70.90 (14.33)	25-94	16.63	<.001	.192
Social ^d	48.51 (18.95)	68.55 (20.37)	0-94	21.01	<.001	.231
Environmental ^d	65.07 (16.73)	76.52 (13.33)	13-100	10.41	.002	.129
Support ^d	45.00 (27.39)	70.31 (20.85)	0-100	9.58	.003	.186

Notes:

^a Anxiety Total score as measured by Beck Anxiety Inventory (BAI); Levels of Anxiety reported according to Total score thresholds, where: 0-7 = Minimal; 8-15 = Mild; 16-25 = Moderate; and 26-63 = Severe anxiety.

^b Depression Total score as measured by Beck Depression Inventory (BDI); Levels of Depression reported according to Total score thresholds, where: 1-10 = normal ups and downs; 11-16 = Mild mood disturbance; 17-20 = Borderline clinical depression; 21-30 = Moderate depression; 31-40 = Severe depression; and over 40 = Extreme depression.

^c Subjective well-being and Global life satisfactions as measured by the Personal Well-being Index (PWI). Scores range from 0-100 (mean = 100, SD = 15).

^d Quality of Life and Support from others as measured by World Health Organisation Quality of Life assessment – short form (WHOQO-BREF). Scores range from 0-100 (mean = 100, SD = 15).

Table 4.6b. Time 1 Mental Health and Quality of Life: Cross-sectional comparisons between Diagnostic Groups within Age Groups

Measure	Younger (N=36)			Older (n=38)			Older (n=38)			Older (n=38)		
	Mean (SD)		Range	Statistics			Mean (SD)		Range	Statistics		
	yASD (n=21)	yTA (n=15)		ANOVA	Sig.	η_p^2	oASD (n=22)	oTA (n=16)		ANOVA	Sig.	η_p^2
Mental Health				F(1,36)						F(1,39)		
Anxiety ^a	14.77 (8.25)	4.25 (3.77)	0-35	22.48	<.001	.384	12.35 (9.32)	6.00 (7.22)	0-34	5.67	.022	.127
Levels Anxiety ^a	2.32 (.89)	.94 (.57)	0-4	29.28	<.001	.449	2.00 (1.00)	1.22 (.94)	0-4	6.42	.015	.141
Depression ^b	18.91 (13.07)	3.56 (4.16)	0-47	20.42	<.001	.362	15.43 (14.05)	5.83 (4.19)	0-43	7.82	.008	.167
Levels Depression ^b	1.95 (1.70)	.06 (.25)	0-5	19.29	<.001	.349	.88 (1.60)	.11 (1.93)	0-5	8.81	.005	.184
Somatoform dis. ^c	6.10 (3.74)	3.38 (2.39)	0-14	6.43	.016	.155	4.91 (2.74)	3.20 (3.05)		3.164	<i>n.s.</i>	.083
Depressive syn. ^c	11.10 (6.57)	1.75 (2.54)	0-23	28.89	<.001	.452	7.36 (6.64)	2.73 (3.75)		5.97	.020	.146
Anxiety syn. ^c	9.71 (7.46)	1.81 (2.01)	0-25	16.92	<.001	.326	7.09 (5.75)	2.40 (3.40)		8.03	.008	.187
Binge eating dis. ^c	1.33 (1.49)	.06 (.25)	0-5	11.26	.002	.243	.64 (1.18)	.20 (.41)		1.89	<i>n.s.</i>	.051
Alcohol abuse ^c	1.05 (.50)	1.25 (.58)	0-9	1.31	<i>n.s.</i>	.036	1.05 (1.89)	1.00 (.93)		.007	<i>n.s.</i>	.000
Diff. Daily Living ^c	1.33 (.86)	.13 (.34)	0-3	28.27	<.001	.447	.86 (.83)	.27 (.80)		4.73	.037	.119
QoL & SWB				F(1,34)						F(1,36)		
SWB overall ^d	36.38 (11.97)	52.67 (9.01)	5-66	19.73	<.001	.367	42.27 (11.92)	52.94 (9.52)	12-67	8.73	.005	.195
GLS ^d	50.95 (16.40)	74.00 (11.83)	10-100	21.53	<.001	.388	56.82 (13.65)	75.63 (12.08)	10-90	7.25	.011	.168
Pers. Relation.	28.99 (11.55)	57.41 (14.81)	0-100	14.01	.001	.264	74.07 (13.62)	75.56 (10.79)	22-100	.03	<i>n.s.</i>	.001
Community	40.58 (19.34)	43.83 (21.56)	0-100	.108	.744	.003	65.93 (15.6)	73.33 (6.1)	22-100	.82	<i>n.s.</i>	.028
Achieving	36.71 (13.38)	48.77 (21.92)	0-100	1.80	.187	.044	62.96 (12.52)	68.15 (7.13)	11-100	.45	<i>n.s.</i>	.016
Health	40.58 (11.76)	52.47 (18.61)	0-100	2.10	.155	.051	69.63 (9.23)	67.41 (13.18)	22-100	.07	<i>n.s.</i>	.003
Future	35.27 (13.2)	52.35 (18.2)	0-89	10.46	.002	.212	68.89 (16.17)	77.04 (7.42)	11-100	.92	<i>n.s.</i>	.032
Std. Living	50.72 (11.57)	66.05 (10.94)	0-100	4.72	.036	.108	74.81 (3.11)	75.56 (9.55)	22-100	.01	<i>n.s.</i>	<.001
Safety	56.04 (11.73)	61.73 (18.22)	0-89	.488	.489	.012	87.41 (4.53)	82.96 (3.36)	44-100	.65	<i>n.s.</i>	.023
Overall QoL ^e	52.38 (17.51)	80.00 (16.90)	25-100	22.40	<.001	.397	67.05 (20.97)	81.25 (14.43)	25-100	5.45	.025	.131
Health ^e	46.43 (25.34)	73.33 (22.09)	0-100	10.94	.002	.243	57.95 (30.26)	67.19 (23.66)	0-100	1.03	<i>n.s.</i>	.028
Physical ^e	57.90 (19.24)	86.00 (12.98)	25-100	24.05	<.001	.414	62.00 (17.92)	78.69 (16.29)	25-94	8.66	.006	.194
Psychological ^e	49.43 (17.69)	74.53 (11.86)	25-94	22.80	<.001	.401	60.32 (19.16)	67.50 (15.94)	31-94	1.49	<i>n.s.</i>	.040
Social ^e	41.95 (19.38)	74.13 (22.28)	0-100	21.30	<.001	.385	54.77 (16.62)	63.31 (17.50)	25-94	2.34	<i>n.s.</i>	.061
Environmental ^e	61.43 (18.11)	79.27 (13.82)	13-100	10.25	.003	.232	68.55 (14.88)	73.94 (12.74)	25-94	1.37	<i>n.s.</i>	.037
Support ^e	46.43 (23.73)	67.86 (12.20)	0-75	4.96	.038	.207	43.75 (30.96)	72.22 (26.35)	0-100	5.39	.029	.190

Notes: dis. = disorder; syn. = syndrome; Diff. = Difficulty; QoL = Quality of Life; SWB = Subjective well-being; GLS = Global life satisfaction; Pers. Relation. = Personal Relationships; Std. Living = Standard of Living; Support = Support from others.

^a Anxiety Total score as measured by Beck Anxiety Inventory (BAI); Levels of Anxiety reported according to Total score thresholds, where: 0-7 = Minimal; 8-15 = Mild; 16-25 = Moderate; and 26-63 = Severe anxiety.

^b Depression Total score as measured by Beck Depression Inventory (BDI); Levels of Depression reported according to Total score thresholds, where: 1-10 = normal ups and downs; 11-16 = Mild mood disturbance; 17-20 = Borderline clinical depression; 21-30 = Moderate depression; 31-40 = Severe depression; and over 40 = Extreme depression.

^c Mental health disorders as measured by the Patient Health Questionnaire (PHQ). Binary scores were derived for the following (range 0 'not present' to 1 'present at clinical cut-off'): Somatic complaints, Depressive syndrome, Anxiety syndrome, Binge eating disorder, and Alcohol abuse; the actual range of scores indicated above. The item Difficulty in Daily Living was scored in terms of degree of difficulty, on a scale of 0 'not at all difficult' to 3 'extremely difficult'.

^d Subjective well-being and Global life satisfactions as measured by the Personal Well-being Index (PWI). Scores range from 0-100 (mean = 100, SD = 15).

^e Quality of Life and Support from others as measured by World Health Organisation Quality of Life assessment – short form (WHOQO-BREF). Scores range from 0-100 (mean = 100, SD = 15).

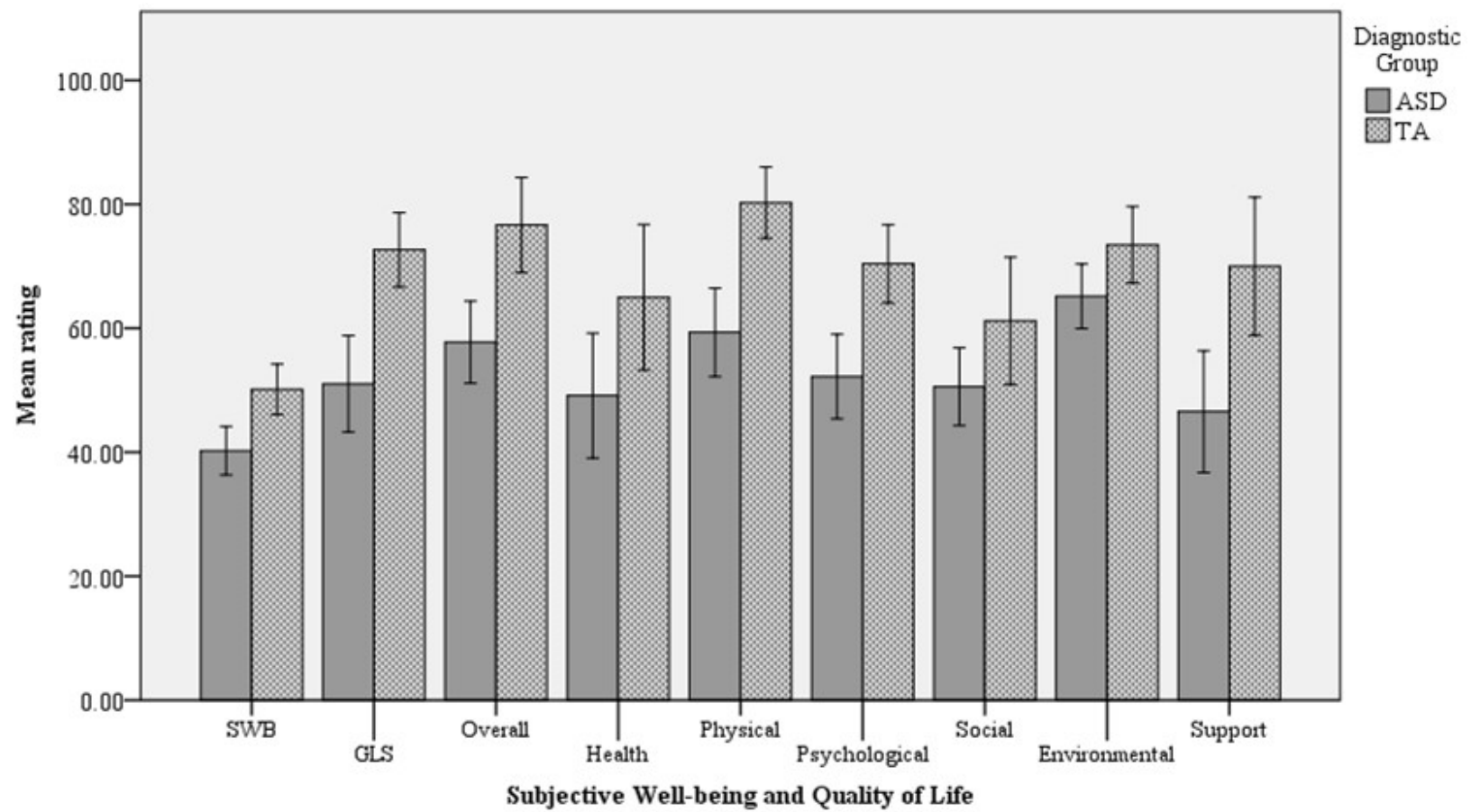


Figure 4.8. Quality of Life ratings for ASD and TA adults. All $p \leq .003$. Error Bars represent 2 SE.

Correlations and predictors of performance

Cognitive difficulties were associated with QoL outcomes in TA and ASD adults, but the patterning of association was different for the respective groups. In TA adults, difficulties in receptive language (RL) and spatial working memory (SWM) were associated with Physical (RL, $r(28) = -.38$, all $p < .05$; SWM, $r(28) = -.45$, all $p < .05$) and Psychological QoL (SWM, $r(20) = -.44$, all $p = .05$), and RL difficulties were also associated with poorer overall subjective-well-being ($r(28) = -.42$, all $p = .03$). Whereas, planning ability ($r(17) = .77$, all $p < .001$) and memory recall consistency were associated with better Social ($r(31) = .41$, all $p < .05$) and Psychological ($r(31) = .36$, all $p < .05$) QoL. Although processing speed in itself was not directly associated with QoL outcomes in TA adults, the relation between other factors just mentioned suggests that processing speed may play a mediating role in later life functioning, as already discussed.

In ASD adults, Episodic Memory and Learning had an unexpectedly inverse association with SWB, whereby individuals who had memory difficulties nonetheless showed better SWB than those individuals with better Episodic memory and Learning ability ($r(33) = -.41$, all $p = .02$). The same profile of associations were observed between memory ability and Psychological ($r(28) = -.40$, all $p = .04$), Environmental ($r(32) = -.40$, all $p = .02$) and Health-related QoL ($r(32) = -.37$, all $p = .04$). Regarding other cognitive abilities, Verbal Comprehension IQ was also associated with poorer SWB ($r(46) = -.30$, all $p = .04$), whilst poorer Health-related QoL was associated with Processing Speed ($r(36) = -.38$, all $p = .02$), and with memory organisation ($r(37) = -.39$, all $p = .02$) and recall ($r(37) = -.38$, all $p = .02$). Even when ASD participants had short-term memory difficulties, this did not adversely affect Psychological QoL ($r(37) = -.34$, all $p = .04$). However, these findings do not indicate causal links and should, therefore, be interpreted with caution. Next, regression analyses were carried out to assess which factors were predictors of QoL outcomes.

As set out in Table 4.7 (p. 156), the outcome variables were entered into a stepwise regression model to detect the strongest predictors of Health and Overall QoL outcomes. First variables related to age, gender, autistic traits, IQ, language, memory, mental health and physical health factors were entered as a stepwise backward regression. This method was used since the data were exploratory and no prior theoretical basis for selecting particular variables as predictors over other variables.

Furthermore, the backward method controls for suppression effects in analysing the relative contribution of each variable to the regression model. The following variables were significant predictors and were subsequently included in a second regression using the Enter method: age, processing speed, self-report autistic traits, self-report RRBs, anxiety, depression, Somatic complaints, and difficulties in daily living. The second regression confirmed these factors as predictors of Health QoL in both ASD and TA groups but did not uniformly predict Overall QoL in both groups (see Table 4.7, p. 156).

In the overall sample, the strongest predictor of overall QoL was depression ($F(1,56) 55.63, p < .001, r^2 .50$), which alone explained almost half of the variance in the group outcomes; Diagnostic Group explained an addition 4% ($F(2,55) 34.46, p < .001, r^2 .54$).

When exploring predictive factors within each Diagnostic Group, there was no model predictor of overall QoL in the TA group, although difficulties in daily living were significantly associated with poorer QoL ($r^2 -.34, p < .05$). However, when exploring these factors within Age and Diagnostic groups, in the older TA adults difficulties with daily living was the strongest predictor of QoL Diagnostic Group ($F(1,12) 6.94, p < .03, r^2 .37$).

In the ASD group, Depression remained the strongest predictor of overall QoL ($F(1,29) 22.36, p < .001, r^2 .44$), which explained more than 43% of the variance in the ASD group. For younger ASD, Depression was maintained as the strongest predictor of overall QoL ($r -.758; F(1,14) 18.88, p = .001, r^2 .574$) followed by age ($r^2 -.67; F(2,13) 15.20, p < .001, r^2 .70$), and Depression was the only model predictor for older ASD ($r^2 -.55; F(1,13) 6.20, p < .03, r^2 .32$). These findings highlight the critical impact of mental health difficulties on the well-being of autistic adults across the lifespan.

Table 4.7. Time 1 Predictors of Quality of Life in ASD and TA adults

	ASD							TA						
	Health QoL	Overall QoL	β	B	SE	t	sig.	Health QoL	Overall QoL	β	B	SE	t	sig.
Age	-.04	.01	.06	.12	.35	.33	.74	-.32*	-.01	.04	.06	.25	.22	.83
Processing Speed	-.42**	-.24	-.18	-.24	.29	-.80	.43	.19	-.17	.05	.09	.27	.33	.75
AQ	-.35*	-.39**	-.13	-.42	.85	-.49	.63	-.37*	.10	-.38	-1.63	.74	-2.20	.04
SRS RRB	-.13	-.40**	.41	.91	.58	1.57	.13	-.45**	-.01	-.11	-.61	1.10	-.55	.59
Anxiety	-.46**	-.43**	-.29	-.86	.61	-1.42	.17	-.30	.04	-.06	-.22	.64	-.35	.73
Depression	-.53***	-.68***	-.50	-.99	.46	-2.15	.04	-.53***	-.25	-.27	-1.52	1.18	-1.29	.21
Somatoform	-.44**	-.50***	-.18	-12.88	13.52	-.95	.35	-.38*	-.07	-.40	-49.47	18.07	-2.74	.01
Daily Living	-.40**	-.55***	.14	4.30	6.61	.65	.52	-.33*	-.30	-.39	-14.95	6.42	-2.33	.03

Notes: * $p < .05$; ** $p < .01$; *** $p \leq .001$

Health QoL R^2 : ASD .483*; TA .622**

Overall QoL R^2 : ASD .540**; TA .216 (*n.s.*)

4.7 Discussion

The literature described in Chapters 2 and 3 highlighted selective memory difficulties and uneven cognitive profiles in autistic individuals (Bowler, Gaigg & Lind, 2010; Boucher et al., 2012; and see Bölte et al., 2009; Ankenman et al., 2014), and the declines in memory that are observed in typical ageing, and which are reliable markers of cognitive decline (Frerichs & Tuokko, 2005; Salthouse, 2004). The aim of the present study was to establish a profile of the factors associated with ageing and ASD as a first time-point assessment. Accordingly, this study involved a multidimensional approach to investigating the complexity of general intellectual ability, language, memory processes and mechanisms, and multiple components of EF across the lifespan (Minshew & William, 2001; Hill, 2004; Eigsti, 2011).

The research questions were:

- What is the extent and magnitude of cognitive differences in ASD, for older adults compared with younger adults, and how does this differ from typical ageing?
- What is the specific profile of cognitive functions in older ASD adults, including EF, memory and language, and their relation to QoL?
- What is the relation between the clinical features of ASD and co-existing conditions across the lifespan, and their effects on QoL?

To directly address these questions, a broad range of assessments were carried out to explore the patterning of age-related *differences* in cognitive functions, mental health and QoL outcomes. The key findings from cross-sectional comparisons between younger and older autistic (ASD) and typically ageing (TA) adults are discussed, followed by implications of the research findings and suggested future directions.

What is the extent and magnitude of cognitive differences in ASD, for older adults compared with younger adults, and how does this differ from typical ageing?

The ASD and TA Diagnostic Groups were matched on age, gender, verbal and nonverbal IQ and years of formal education (YFE) as closely as possible, however, the older ASD adults had more YFE than the older TA adults. In both Diagnostic Groups, higher educational attainment was associated with better cognitive outcomes in terms of IQ and memory. A possible explanation for this might be that, on average, participants had achieved high levels of educational attainment, despite some older TA participants having only achieved primary education levels. In the ASD group YFE was also associated with greater depression and anxiety, but educational attainment was not related to QoL in either Diagnostic Group.

Overall, cognitive function in ASD adults was not affected by older age. The older autistic adults performed as well as or better than the younger autistic adults in memory, language, and in IQ associated with working memory and processing speed. Expressive language and general vocabulary skills were significantly better in the older ASD adults, suggesting that language may mediate other cognitive functions as highlighted in previous ASD literature (Bowler, Gardiner & Grice, 2000; Bowler et al., 2014; and see Kana et al., 2017). Indeed, previous research has shown expressive language proficiency as well as cognitive strengths in ASD (Williams et al., 2006). The findings from Study 1 align with these suggestions, since better IQ and language skills were associated with better memory performance in ASD adults, overall. In line with previous research findings, the cognitive profiles of younger ASD adults appeared to mirror the difficulties observed in the older TA adults (Bowler et al., 2005). For instance, previous literature has highlighted discrepancies among receptive and expressive language skills, and in narrative comprehension in autistic children and adolescents (Williams et al., 2006; Howlin et al., 2014), and with selective difficulties in domains of general intellectual functioning in younger autistic adults (Mottron et al., 2006; Dawson et al., 2007; Bölte et al., 2009).

In the present study, the profile of difference was particularly evident for younger ASD adults in memory function. Broadly, primary memory processes related to simple memory (short delay free and cued recall) and complex memory (long delay free and cued recall), as well as the underlying

mechanisms of memory failure associated with incremental learning and recall consistency was comparably impaired in younger ASD and older TA adults. Although there were no significant differences between the ASD and TA groups on semantic clustering, this patterning of memory ability suggests that whilst ASD adults were able to organise information semantically, they did not use these organisation strategies to facilitate free recall for immediate or delayed memory. These findings replicate the memory profiles observed in previous studies, whereby (young) autistic adults show difficulties in complex memory (Minshew et al., 1992; Minshew & Goldstein, 2001) and organisation of information for later retrieval (Bowler, Matthews & Gardiner, 1997). The work presented here extends previous findings to highlight the potential mechanisms associated with memory difficulties and strengths in ageing and ASD.

EF performance of ASD adults in the present study, overall, did not concur with the profile of impairments reported in previous literature. For instance, previous literature has identified selective ASD-related difficulties in sub-domains of EF, such as cognitive flexibility, working memory and planning (Russell, 1997; Hill, 2004; Ozonoff et al., 2004; Bramham et al., 2009; White, Burgess & Hill, 2009; Rosenthal et al., 2013). In the present study there were no differences between ASD and TA adults on tasks of Working memory / Planning, Cognitive flexibility / Attention / Working Memory, Attention / Inhibition, or on Visual / Episodic Memory and Learning. However, when exploring these functions within younger and older Age Groups, the younger ASD adults showed a similar profile of EF difficulties to older TA adults. Both these groups showed poor planning accuracy related to Working Memory / Planning, and greater difficulties in Episodic Memory and Learning as demonstrated by increased perseverative errors and lower retention of contextual information for later retrieval. Here, older TA adults showed the same patterning age-related EF difficulties that have been identified in previous literature (Zelazo, Craik & Booth, 2004). Specifically, compared to the performance of younger TA adults, the older TA adults in our study showed greater difficulties with visual episodic memory and learning, poorer planning ability, increased difficulties with cognitive flexibility and dimensional set shifting, and increased perseverative errors and poor efficiency of search strategies related to spatial working memory. Further, motor function was slower, and sustained attention and response inhibition were reduced in

older TA adults. What is more, the medium effect sizes observed for Attention/Inhibition suggest that the findings from these data are relatively robust with practical relevance, despite non-significance of group differences.

What is the specific profile of cognitive functions in older ASD adults, including EF, memory and language, and their relation to QoL?

The older ASD adults showed a different patterning of age-related performance. There were no increased age-related differences on the EF task performance for older ASD compared to younger ASD adults. The older ASD adults performed better than all groups in cognitive flexibility for dimensional set-shifting and performed at least as well as older TA adults on tasks of visual Attention and response Inhibition.

Previous literature has reported that executive dysfunction may underlie the core difficulties associated with ASD (Hill, 2004; Lopez et al., 2005; Lawson et al., 2015; and see Lai, Lombardo & Baron-Cohen, 2014), but the findings across lifespan developmental studies are equivocal and the patterning of EF abilities and difficulties does not conclusively support this view (Geurts, Corbett & Solomon, 2009; and see Ozonoff et al., 2004; Geurts & Vissers, 2012; Lever & Geurts, 2016; Powell, Klinger & Klinger, 2017). There is some evidence that planning difficulties persisting into adolescence and young adulthood (Van Den Bergh, Scheeren, Begeer, Koot & Geurts, 2014), whereas better performance has been shown older ASD adults in cognitive flexibility and the inhibition of prepotent responses (Geurts et al., 2014). Furthermore, working memory has been shown to remain intact across adulthood (aged 20-79 years) with little evidence of increased age-related difficulties (Lever et al., 2015; Wang et al., 2017). The findings from the present study concur with the suggestion of preserved EF in older age, as evidenced by the performance of older ASD adults in the present study.

How does this explain the patterning of positive age-related differences between older compared to younger ASD adults, and the relative absence of age-related difficulties that were observed in typically ageing adults? A possible explanation for the patterning of age-related performance in our study may be that few previous studies of EF in ASD have systematically

measured EF in a multidimensional approach (but see Ozonoff et al., 2004; see also Pellicano, 2013). The present study included a systematic evaluation of the breadth of EF capabilities across the adult lifespan to explore the interdependence of EF components and other cognitive functions, such as language, general intellectual ability and memory. Further, as already mentioned (Chapters 2 and 3), the nature of EF processes is multidimensional and, therefore, not a “process pure” mechanism of operation (McCabe et al., 2010, p. 223; Friedman & Miyake, 2017). EF operations are optimised by the integration of multiple higher-order cognitive processes, whereby most the successful completion of cognitive tasks draws on an integrative network of cognitive functions. Consequently, EF difficulties may be mediated by a disordered organisation and processing of information (Williams et al., 2006), giving rise to broader cognitive difficulties. If true, then the difficulties observed in younger ASD and older TA adults may be associated with underlying EF difficulties involving the planning, organising and retrieval of contextual information for later use. This suggestion is evident in previous literature that has shown that autistic adults can achieve the same task performance as typically developed individuals, but via different cognitive mechanisms (Minshew, Sweeney & Luna, 2002; Kana, Keller, Minshew & Just, 2006). Moreover, the profile of cognitive abilities and difficulties in ASD may be mediated by the interdependency on other EF components and task complexity, whereby cognitive difficulties in one domain may have a cumulative effect across other domains (Geurts, Corbett & Solomon, 2009; Kercood et al., 2014). Consequently, disordered organisation in memory and broader cognitive functions has potentially adverse effects on language comprehension and learning since it would facilitate fewer opportunities for individuals to meaningfully interpret the environment and learn from prior experiences. Such difficulties would pose particular challenges for autistic individuals across the spectrum in terms of everyday functioning and social communication.

A second potential interpretation of these findings is that older ASD adults employ compensatory strategies that draw on other cognitive processes to complete tasks with greater cognitive loads (Just et al., 2004; Williams et al., 2006). It could be argued that this cognitive competition would be more problematic in the context of ageing, given that cognitive capacity for information processing is reduced in typical ageing (Schaie, 2003; Salthouse, 2004, 2016). As

previously indicated, processing speed appears to be a distinct process that modulates more general cognitive functioning in older age (Anderson & Craik, 2017), but does not mediate age-related declines in EF (McCabe et al., 2010; Cappelletti et al., 2015). If, as the evidence suggests, the organisation at encoding and subsequent retrieval of information is disordered at a neural level, this would, in turn mediate the effective use of strategies for memory and other higher-order cognitive abilities, such as EF, problem solving and comprehension. For instance, processing speed affects the capacity to process details and large amounts of information, and the retrieval of that information for later memory recall (Craik & Byrd, 1982; Dempster, 1992; Salthouse, 1996; McCabe et al., 2010; Salthouse & Saklofske, 2010). This is further compounded by age-related declines in episodic memory retrieval (Atance, 2010) which, in turn, are affected by age-related declines in EF and WM capacity (Zelazo, Craik & Booth, 2004; McCabe et al., 2010) and information processing (Salthouse, 1996). In the present study, the findings on memory, language and general intellectual ability and EF were strongly positively correlated with each other. However, whilst this finding was robust in the ASD group, it was only partially supported in the TA group, suggesting that other factors may be mediating performance in typical ageing, as already discussed. In the present study, there was a strong association between age and declines in processing speed in the TA group. This was explored following the earlier suggestion that processing speed may be a potential precursor to age-related cognitive difficulties in typical ageing. When controlling for processing speed, any associations between cognitive measures in the TA group disappeared, whilst in the ASD group these were altered but still strongly evident. The TA adults showed greater processing speed difficulties with increasing age, and increased difficulties associated with memory and EF. This was not the case for older ASD adults. Consequently, it may be that older autistic adults are more adept at dealing with cognitive challenges across the lifespan, as suggested by previous literature and the patterning of performance in younger ASD adults in our study. It is possible that autistic adults, through repeated experiences of cognitive adversity in earlier life, develop cognitive coping mechanisms that serve to support their functioning in later life. By contrast, as typically developed individuals grow older and first experience cognitive challenges associated with ageing, the contrast of performance differences is more pronounced compared to the younger typical adults.

Quality of life

The relation between cognitive function and QoL was different in ASD than observed in typically ageing adults. Whereas, older TA adults showed difficulties in processing speed, receptive language, memory and EF compared to younger TA adults, the older ASD adults did not show the same patterning of age-related difficulties. In TA adults, these difficulties were associated with poorer Physical, Psychological and Social QoL and well as poorer subjective well-being (*“overall, how satisfied are you with your life?”*).

In the ASD group, the findings from the present study concur with observations in the previous literature of poorer QoL in younger and older autistic adults (Kamp-Becker et al., 2010) and older autistic adults (Totsika et al., 2010; van Heijst & Geurts, 2015; Roestorf & Bowler, 2016; Ayres, Parr, Rodgers et al., 2017). Overall subjective well-being and QoL across domains was significantly lower for younger and older ASD adults compared with TA adults. However, increasing age was not a factor in reduced QoL in either group. Thus, the findings presented here add to the evidence of QoL concerns that affect autistic individuals across the lifespan. Subjective well-being was scored lowest by ASD adults, followed by Support (*“how much support do you receive from others?”*), and then Social, Health, Psychological and Physical QoL domains, respectively. ASD adults rated Environmental QoL most highly, although this was still significantly lower than for TA adults. By contrast, the highest rated domain for TA adults was Physical QoL, followed by Environmental QoL, and then similar ratings for Social, Psychological and Support domains. The difficulties observed across the range of QoL domains in the present study hold important implications for general well-being and health-related outcomes for autistic adults across the lifespan. For instance, Social QoL relates to the friendships and intimate relationships a person has as well as the degree of social support they receive. Given that ASD adults, overall, reported receiving very little support from others, and considering the social difficulties that were reported earlier, it is unsurprising that Social QoL was the most substantially affected QoL domain. What is more, Health-related QoL in the ASD group is also of concern, given the high degree of co-existing physical and mental health conditions in autistic adults, which have also been highlighted in previous studies (Hirvikoski et al., 2016; Fortuna et al., 2015; Croen et al., 2017).

The domain of Psychological QoL includes self-esteem, negative and positive feelings about oneself, as well as psychological functioning difficulties associated with learning, memory and concentration. In the present study, although the autistic adults performed comparably well, overall, on assessments of general intellectual ability, language, memory and EF, there were some notable difficulties in these domains, particularly for younger autistic adults. Further, self-perceived difficulties in everyday functioning are important factors that impact the psychological well-being and mental health of autistic adults. The difficulties associated with daily living, sleep, pain and illness, as well as dependency on long-term medication, reduced mobility and capacity for employment were reflected in the reduced Physical QoL for autistic adults in the present study. Whereas, Environmental factors associated with financial status, living arrangements, access to and quality of social care, which were also affected in ASD compared to TA adults, these factors were perceived as less problematic by some autistic adults.

Subjective well-being also substantially affected well-being for ASD adults, across domains. Older ASD adults reported greater satisfaction with Standard of Living, Personal Relationships, and their outlook to the Future compared to younger ASD adults. However, the degree to which these (and other) domains were affected compared to TA adults raises specific consideration of the long-term outcomes of autistic adults. Moreover, social isolation and the absence of meaningful personal relationships highlight the lack of supports, already mentioned. Autistic adults identified concerns for the future and their health and care needs. These findings have significant implications for the lifespan outcome for autistic adults in the context of autism-related difficulties and co-existing physical and mental health conditions, which are discussed next.

What is the relation between the clinical features of ASD and co-existing conditions across the lifespan, and their effects on QoL?

Self-reported autistic traits were expectedly higher in the ASD group, and these were associated with better language skills, but increased difficulties in everyday life, anxiety and depression and poorer QoL across domains. However, observer-rated ADOS profiles did not concur with these findings, although higher ADOS scores were associated with poorer Health QoL. A possible

explanation for these findings is that higher scores on observer-rated measures, such as the ADOS are designed to reflect the behavioural difficulties associated with ASD, which appeared to be mediated by General Vocabulary and Gender in this sample. By contrast, individuals who have better language skills appeared to be more able to self-report their ASD-related difficulties in daily living. Moreover, more highly verbal and cognitive able autistic adults also appeared to demonstrate greater self-awareness of how these factors affected their QoL. Recent findings from related work in this area suggest that caution should be applied in comparing self-report and observer rated measures of autistic traits, since these do not correspond to predicted value of ASD diagnostic profiling (Roestorf, Gaigg, Williams & Bowler, in prep.). In that study self-report AQ and SRS scores did not correlate with ADOS outcome scores, whereas AQ and SRS scores correlated highly with each other and with measures of anxiety and depression. A possible interpretation of this discrepancy in self- vs observer-rated outcomes may be that self-report measures are not sensitive enough to detecting autistic difficulties, per se, but rather offer a transdiagnostic measure of general difficulties, such as those associated with mental health conditions. Moreover, since self-awareness is known to present difficulties for many autistic individuals, self-reporting of metacognitive awareness of associated difficulties would consequently prove problematic for some autistic individuals (Ashwood et al., 2010). What is more, findings from recent independent studies concur with the above suggestion in three key ways. First, the predictive value of self-report measures does not appear to sensitive enough in ASD for those individuals who have difficulties in self-reporting, given that 20-80% of individuals below threshold nonetheless go on to obtain a diagnosis of ASD (e.g. Bishop & Seltzer, 2012; Sizoo et al., 2015). Second, individuals with higher cognitive ability and the ability to self-reflect on their autism-related difficulties, in turn increases the extent of high self-reports across measures (van Niekerk et al., 2010). Finally, ASD traits are known to overlap other conditions e.g. anxiety, ADHD, OCD, dementia (van Niekerk et al., 2010). In contrast to Constantino & Gruber's (2012) reporting that the SRS discriminates ASD from conditions as ADHD and anxiety, a recent study by South and colleagues (2018) highlighted that the SRS did not significantly differentiate between 'ASD traits' in an autistic adult sample compared with an anxiety sample (South et al., 2018). The convergence of the findings presented in the present chapter, and previous studies already mentioned, suggests that

careful consideration needs to be given to using self-report measures as screening instruments in the diagnosis of older autistic adults. Given that ASD is underdiagnosed in older people (Ashwood et al., 2016; van Niekkerk et al., 2010) there are additional challenges in obtaining a diagnostic history and using measures that are age- and gender-appropriate (Roestorf, Bowler, Deserno, Howlin ... Geurts, in press). Consequently, metacognitive difficulties, which may account for some these differences, in turn affect communication skills and understanding (Bowler et al., 2005). More work is needed to understand the nature and extent of these factors.

Co-existing conditions

Recent research has highlighted greater co-occurring health conditions and mental health needs in ASD compared to typically developed individuals (Hirvikoski et al., 2016; Fortuna et al., 2015; Happé, Mansour, Barrett et al., 2016; but see Lever & Geurts, 2016a), and do not have access to appropriate services in adulthood and across their lifespan (Wright et al, 2016). The presence of co-existing physical and mental health conditions in the present study were highly prominent in younger and older autistic adults. These findings showed that the majority 49-69% of autistic adults reported multiple co-existing physical and mental health conditions, difficulties with social abilities and everyday life. Anxiety and depression presented significant difficulties across the lifespan, and gender differences highlighted more extreme levels of depression in autistic women. In addition, autistic adults reported multiple co-existing conditions associated with physical health concerns which, in turn, increased difficulties in everyday life. Social difficulties (e.g. anxiety, loneliness, isolation) were associated with communication difficulties (e.g. from social nuances and figurative language, to understanding complex instructions), while physical health concerns (e.g. pain, sensory sensitivities) increased difficulties with daily living and predicted poorer QoL in younger and older autistic adults. The findings presented here converge with previous literature that shows the majority of autistic adults face persistent mental health difficulties and poorer QoL compared to typically ageing adults, regardless of intellectual ability (Howlin et al., 2004; Hofvander et al., 2009; Howlin & Moss, 2012; Kats et al., 2013; Ratto & Mesibov, 2015). What is more, ASD adults reported lower levels of support from others in everyday life compared to typically ageing adults, and this did not differ with age. The

implications of these findings are that many autistic adults require social care support across the lifespan (Howlin et al, 2013; D'Astous, Manthorpe, Lowton & Glaser, 2016). These factors pose particular challenges for autistic individuals as they grow older and may be exacerbated for individuals who are not able to live independently – support from family members would unavoidably disappear as parent caregivers themselves approach old age and end of life (Howlin et al., 2015). The implications of the factors just discussed are outlined in the section that follows, raising important considerations for future research.

Implications of the present research

The predictions for the work presented here were based on what happens in typical ageing, and the patterning of difficulties in younger autistic individuals that is similar to older typically ageing individuals (Bowler et al., 2004). Some aspects of cognitive functioning were expected to be impaired in older age. For instance, if the patterning of difficulties seen in younger autistic individuals were to persist into older age, then it would be expected to see evidence of age-related effects in older ASD adults that at least parallel the patterning seen in typical ageing. Whereas, if those potential difficulties were to stabilise or abate into older age, then fewer age-related differences in cognitive ageing would be expected in autistic compared to typically ageing adults.

Based on the patterning of difficulties and abilities observed in younger and older autistic adults, compared to typically ageing adults, the findings from the present study suggest this latter profile to be the case, showing *age-neutral* outcomes for older autistic adults. The older ASD adults in this cohort were cognitively able individuals, with average to above-average intellectual ability, who performed well across a range of standardised cognitive assessments. Specifically, the performance of older ASD adults was better on some cognitive functions compared to younger ASD adults and compared to older TA adults. Conversely, increasing age adversely affected the cognitive, physical and mental health and QoL of older typical adults. Moreover, as we have seen from previous literature (Chapters 2 and 3), long-term mental health difficulties, such as depression, are known to correspond to difficulties in memory, attention, EF and processing speed in the context of ageing (McClintock, Husain, Greer, & Cullum, 2010).

Summary of key findings and their implications

The crucial outcomes from this study relate to the poor QoL and well-being, degree of co-existing physical and mental health difficulties, and the difficulties in everyday life reported by autistic adults, regardless of cognitive abilities across adulthood. The high cognitive abilities in the ASD adults, despite their greater difficulties with daily living, suggests that they may be employing compensatory mechanisms to facilitate a level of autonomous functioning in everyday life (Schaie, 2003; Baltes, Dittman-Kohli, & Dixon, 1984; Baltes, Staudinger, & Lindenberger, 1999). In line with this suggestion, Damiano, Mazefsky et al. (2016) describe autonomous living as the ability to achieve everyday functioning without the need for care supports (e.g. caregiver). That is, the ability to undertake small and moderate daily tasks such as self-care, bathing and dressing, to home management tasks including cleaning, laundry, shopping, organising and preparing meals, and more demanding tasks such as paying bills and managing finances. The functions just mentioned are examples of daily living skills that are essential to maintaining autonomy in everyday life, especially into older age (Schaie, 2003). What is more, in some communities, health and social welfare provide care packages to facilitate autonomy or the provision of care to support these daily living needs. However, few services provide individualised care packages tailored to specific needs, such as ASD (Wright et al., 2016). Provision of such treatment and care plans would serve to build on the strengths of individuals and develop and maintain coping strategies for cognitive maintenance and social integration (Wright et al., 2016). In order to facilitate such social care strategies, a need for “societal adaptation and acceptance” is required, together with a broader understanding of the needs of older autistic adults across the lifespan (Wright et al., 2016). More research is needed on the effects of co-existing conditions on daily living skills in ASD across the lifespan. The factors just mentioned need to be addressed in future work, not only to identify factors that may mediate QoL and well-being outcomes for autistic adults, but to establish care pathways to enable autistic adult to lead healthy and fulfilling lives.

Study contributions

The work presented in this chapter applied a broad range of cognitive assessments to get a sense of the profile of functioning in ageing autistic adults. The array of tasks used presents several potential advantages that have not characterised previous studies using single test paradigms (e.g. Geurts & Vissers, 2012; Lever & Geurts, 2016; Powell, Klinger & Klinger, 2017). The cognitive battery used in this study extends previous research on memory in ASD (e.g. Minshew & Williams, 2001; Williams et al., 2006; Geurts & Vissers, 2012; Lever & Geurts, 2015; Powell et al., 2017) and the implications for ageing. In previous studies of typical development, similar test batteries have been used with younger and older adults, with ecologically valid outcomes (e.g. McCabe et al., 2010; Chan & McDermott, 2007; Glisky & Kong, 2008). Yet, few studies in ASD have explored the breadth of EF components and their relation to other cognitive functioning in ASD, but these are limited to younger groups (e.g. Ozonoff et al., 2004, Pellicano, 2013). The findings from the present work extend the observations of previous ASD research. Moreover, these new findings highlight the potential challenges associated with EF difficulties in early life, and how potential coping mechanisms may serve to support cognitive performance in older age. Thus, the present work offers new knowledge and understanding of the breadth of memory and EF, in conjunction with cognitive domains involved in language and general intellectual functioning in ageing and ASD. The majority of studies reviewed in Chapters 2 and 3 have explored specific or only singular aspects of memory or EF, or other cognitive abilities. Few studies have explored the association between cognitive functions, and the patterning of abilities across the lifespan. The present study fills these gaps by addressing several components of simple and complex memory that facilitated an evaluation of primary and process-related mechanisms of memory function.

Limitations and Future Directions

The possible explanations for the patterning of findings from the Time 1 study have already been discussed. However, several important considerations arise from these findings, some of which have been mentioned in the study implications. Further, few autistic women were represented in the present study, despite efforts to include as broad a sample as possible. Although some gender

differences were observed in profiles of autistic traits and selective cognitive functions, it is not possible to generalize the current findings to the wider autism community. A potential reason for the lower ratio of females would be concordant with recent work that has highlighted camouflaging of autistic features in women on the autism spectrum (Mandy & Lai, 2017). Thus, future work that focuses on the inclusion of autistic women across the lifespan would serve to explain any true gender differences in the context of ageing and ASD. Another important consideration is that the older autistic adults in this study were of average to above average intellectual and verbal ability. Therefore, age-related comparisons were between highly cognitively able younger and older adults. Future work would need to consider how these findings translate to the profile of ageing across abilities on the autism spectrum, for individuals with more variable cognitive profiles and abilities.

The cross-sectional comparisons from the present study and previous research offer valuable insights to the potential outcomes for adults who are able to maintain optimal cognitive functioning into older age. However, a consequence of cross-sectional studies is that they cannot account for cohort differences, or other factors associated with the presence or absence of age-related cognitive *changes*. Findings from the few longitudinal studies of autistic adults at least into middle age (e.g. Howlin et al., 2013) suggest that the features of autism remain stable, as does general intellectual ability. Nevertheless, emerging evidence from cross-sectional studies with older autistic adults suggests that mental health and individual differences in cognitive abilities may mediate age-related gains or losses in later life (e.g. Geurts & Vissers, 2012; Lever & Geurts, 2015; Lever et al., 2015; Powell, Klinger & Klinger, 2017). More longitudinal work is needed to understand the extent to which these cognitive profiles *change* over time.

4.8 Conclusion

The findings of the exploratory cross-sectional study presented here make a substantial contribution to our advancing understanding of ageing and ASD. The patterning of age-related difference in older TA adults, and the absence of those differences in older ASD adults, suggests that autistic individuals may adopt cognitive scoping strategies across the adult lifespan, which serve to benefit their cognitive function in older age. Nevertheless, the substantial physical and mental health concerns observed in younger and older ASD adults highlights the need for ongoing supports across the lifespan. In the next chapter, this work is taken forward into a longitudinal evaluation of the *changes* that occur with ageing. The same cohort from the present study was followed-up through a second time-point (T2; approximately 2.5 years after T1), to assess the predictors of changes in the cognitive domains described in Study 1, as well as the possible influences of these domains on mental health difficulties and QoL.

Chapter 5: Study 2

Stability and change in autistic traits, cognitive function and quality of life: a two year follow-up

The findings from Study 1 extend previous research, providing new knowledge about the profile of ageing in older cognitively able autistic adults, and the associations between mechanisms of cognitive function, mental health and QoL. However, to date, previous research has not addressed the issue of age-related *changes* that occur in older age and autism or how the process of growing older differs from typical ageing (Wise et al., 2017). To address this gap in the literature, Study 2 sets out the second time-point (T2) cross-sectional assessment and longitudinal follow-up of the same cohort involved in Study 1. Here, a broad range of measures was once again used to assess the degree of autistic traits, general intellectual ability, language and multidimensional evaluation of cognitive function, mental health and QoL (see Chapter 4 for Methods and Measures). The T2 cross-sectional findings present further data on the age-related *differences between* younger and older ASD and TA adults in the domains just mentioned, and the comparison of T1 with T2 outcomes provides a longitudinal measure of *changes within* individuals. Accordingly, the analysis explores predictors of change for ageing in ASD compared to typical ageing, and the associations of potential changes with QoL. The patterning of findings from the T2 cross-sectional and longitudinal assessments are discussed later in the present chapter.

5.1 Study Aims

Several research questions were set out in Chapter 2 (p. 67) in order to understand the extent of age-related differences and the potential for increased magnitude of challenges for older autistic adults. In Chapter 4, Study 1 set out to address the first three questions (p. 91), in the T1 cross-sectional assessments. In the present chapter, Study 2 revisits those research questions in the T2 cross-sectional assessments of age-related *differences*. Additionally, the fourth question (Chapter 2, p. 67) explores trajectories of *change* through an evaluation of longitudinal changes from T1 to T2 outcomes. The issues related to longitudinal research and their bearing on Study 2 are discussed.

5.2 Ethics

Study 2 followed the ethical guidelines and procedure outlined in Study 1 (Chapter 4, p. 93), and the recruitment and ascertainment procedure already discussed (p. 92).

5.3 Method

Longitudinal research presents many operational challenges, including cost, time and resources, and the retention of participants over time (e.g. Salthouse, 2014). Study 2 was no less at risk of these challenges. For instance, the longitudinal study required sustained participation across multiple assessments for the duration of the 4-year programme of work. Related challenges included the need for flexible arrangements for ASD and TA adults over the age of 60 years, and individuals with complex physical and mental health concerns as already discussed (p. 68). Moreover, a possible self-selection sampling bias may limit the generalisability of the findings. For instance, less cognitively able ASD and TA adults and those with greater difficulties arising from co-existing conditions may not have been able to take part in the extended study.

The methods and procedure for the individual assessments in the present study follows those of Study 1 (Chapter 4), and so will not be repeated here. Cross-references will be provided to the relevant details where appropriate.

Procedure

Participants who confirmed their availability to take part in the T2 follow-up assessments were once again provided with the information sheet as a reminder of what was involved, informed consent was confirmed, and an appointment was scheduled. On arrival at the appointment, the participant was verbally reminded of what each assessment involved.

The choice of the set of measures was determined by the need to document meaningful change in the domains that affect everyday functioning and communication abilities and QoL in older age, within the time available for testing. Accordingly, the amount of assessments used at T2 enabled complete assessment in a single session. Each appointment lasted approximately 3 hours, allowing

time for comfort breaks as needed by the participant. The measures that were administered at T2 are briefly outlined below (for more detail see Chapter 4, pp. 97-118).

Participants: T2 sample characteristics and continuity

Sixty-two participants from the T1 study (see Figure 2.2 in Chapter 2, p. 71) took part in the T2 study, representing a 71% retention rate across both groups. Analysis of these data showed that the T2 sample included 39 adults with a diagnosis of ASD and 23 TA adults (Figure 5.1, p. 175). There were 18 females (29%) in the T2 sample, the greater proportion of those being TA adults (see Table 5.1, p. 185). Diagnostic Groups ended up being matched on general ability (verbal and non-verbal IQ), and broadly matched on chronological age. Sample attrition from T1 to T2 resulted in a marginally significant ($p = .045$) age difference between Diagnostic Groups, which is looser than one might like. Regarding sample continuity, an analysis of the time between T1 and T2 assessments confirmed a mean interval of 2.26 years (see Figure 5.1, p. 175), which was not significantly different between Diagnostic or Age Groups ($p > .05$).

The YFE T1 data, which were carried over from T1 to the T2 sample, yielded no significant differences between Diagnostic Groups in YFE at T2. Regarding group matching on non-verbal and verbal IQ, analyses of the T2 data for Full-scale IQ (FSIQ), Verbal Comprehension (VCI; verbal IQ) and Perceptual Reasoning (PRI; nonverbal IQ) showed no differences between Diagnostic Groups (see Table 5.1, p. 185). There was a small, but significant mean age difference of 4 years between ASD and TA Diagnostic Groups (mean sample age 49.87 years, standard deviation (SD) 7.84 years), with the TA group being slightly older (Table 5.1, p. 185). However, this main effect disappeared in planned contrasts within Age Group between Diagnostic Group (i.e. yASD vs. yTA; oASD vs. oTA; mean ages: younger Age Group, mean 37.00 years, SD 7.81; older Age Group, mean 62.15 years, SD 7.76; see Table 5.2b, p. 189).

Table 5.1 (p. 185) shows a higher proportion of females in the older TA group compared to than other groups. Overall, and the TA group overall had an approximately even gender distribution (males, $n=12$; females, $n=11$), whereas the in the ASD group included fewer females (males, $n=32$; females, $n=7$), for reasons previously discussed in Chapter 2 (p. 71). However, this effect disappeared

in planned contrasts within Diagnostic Groups between Age Groups (Table 5.2a, p. 188) and within Age Groups between Diagnostic Groups (Table 5.2b, p. 190).

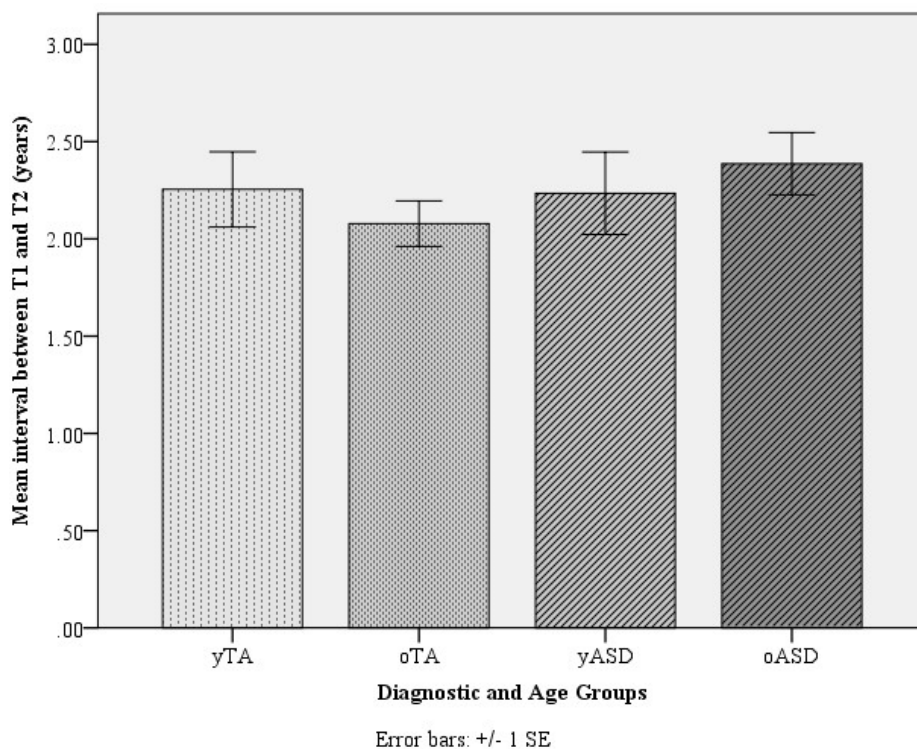


Figure 5.1. Mean time interval in years between T1 and T2 assessments.

5.4 Measures

Assessments of cognitive function at T2 included general intellectual ability (Wechsler Adult Intelligence Scales–Fourth Edition; WAIS-IV; Wechsler, 2008), language (Comprehensive Receptive and Expressive Vocabulary Test–Third Edition; CREVT-3; Wallace & Hammill, 2013) and memory (California Verbal Learning Test–Second Edition; CVLT-II; Ober et al., 2002). As already described, the measures reported in Chapter 4 were once again administered here; where alternate forms were available for language, memory, wellbeing and QoL, these were administered in counter-balanced order at T1 and T2⁸. The EF measures are not reported for T2 because of administrative reasons that resulted in insufficient data being available. However, previous literature has indicated the importance

⁸ At T2 the counterbalanced measures were administered in reverse order to T1 (Chapter 4).

of EF ability in QoL outcomes in ASD children (e.g. de Vries & Geurts, 2015) and adults (e.g. Dijkhuis, Ziermans, van Rijn, Staal & Swaab, 2017). Thus, the T1 EF data (Study 1, Chapter 4) was used to predict outcomes at T2.

Mental health measures were also administered at T2 to assess co-occurring psychiatric conditions (Patient Health Questionnaire; PHQ; Spitzer, Kroenke, Williams et al., 1999), anxiety (Beck Anxiety Inventory–Second Edition; BAI-II; Beck & Steer, 1993) and depression (Beck Depression Inventory–Second Edition; BDI-II; Beck, Brown & Steer, 1996). Finally, wellbeing (Personal Wellbeing Index–Adult version; PWI-A; Cummins et al., International Wellbeing Group, 2003) and QoL (World Health Organisation Quality of Life questionnaire–short form; WHOQOL-BREF; WHO, 2000), and the clinical features of autism and degree of related difficulties (Social Responsiveness Scale–Second Edition; SRS-2; Constantino & Gruber, 2012) were administered again.

5.5 Analysis strategy

Given that, at T2, there were no within Diagnostic Group differences in age, or within Age Group differences in Gender or YFE, these variables were not individually included in any analyses. Therefore, the Results section sets out cross-sectional *differences* for: (i) Diagnostic Groups comparisons, (ii) planned contrasts within Diagnostic Groups between Age Groups, and (iii) planned contrasts within Age Groups between Diagnostic Groups. This is followed by an analysis of the *change* in outcomes between T2 and T1 assessments. The explanation below sets out how the data were handled longitudinally.

Missing data analysis

Prior to analysing the data, a Missing Value Analysis (MVA) was carried out using an Expectation Maximisation (EM) method in SPSS (v.24). The MVA confirmed that any missing data at T2 were Missing Completely at Random (MCAR), according to Little's (1995) MCAR test. Overall, the MVA EM analysis confirmed no significance of missing data patterns between Diagnostic Groups, (Little's MCAR test: Chi-Square = 555.06, DF = 632, Sig. = .99). The only

exception to that pattern of missing data was associated with Memory assessment (45.2% missing). The MVA confirmed that the Memory data were not MCAR, but rather more systematically missing (Little's MCAR test: Chi-Square = 24.69, DF = 13, Sig. = .025). This seemed to be driven by missing data in the younger TA group. However, it is noted that just more than half the participants in each group were able to complete the T2 memory assessment. Consequently, the test-retest reliability of the CVLT-II memory measure (Woods et al., 2006) is addressed in the longitudinal change analyses below. The missing data associated with all T2 variables are summarised in Figure 5.2 below.

Missing data imputation

Following the MCA procedure just explained, the missing values were imputed using the EM method. Accordingly, a new data file was generated with imputed values to previously identified missing data. Then, the main statistical analyses (MANOVA) between Diagnostic Groups and Age Groups were re-run with the new data set that contained no missing values. This procedure was repeated for planned contrasts between Age Groups within Diagnostic Groups; and between Diagnostic Groups within Age Groups. Overall, the patterning of data showed no differences between Diagnostic Groups in general ability ($\eta_p^2 \leq .011$), language ($\eta_p^2 \leq .035$) and memory ($\eta_p^2 \leq .035$), but significant differences in mental health ($\eta_p^2 = .08 - .28$) and QoL ($\eta_p^2 = .07 - .29$), which follows the patterning of findings observed at T1. Moreover, in the MVA analyses there were significant gender differences and interactions with diagnosis and age, with medium to large effect sizes ($\eta_p^2 = .07 - .23$) across a range of measures that bear consideration in future work. Nevertheless, the breadth of measures and the Diagnostic Groups involved in the overall programme of work represent a complex sample and study configuration. The complexities of conducting longitudinal research in this cohort have already been discussed. It is, therefore, important to note that the MVA is provided for completeness but is not intended to replace the primary analysis which is summarised in the Results below. Accordingly, the MVA imputed data and secondary analyses are provided as a supplementary file in Table 5.0 (Appendix 2, p. 354).

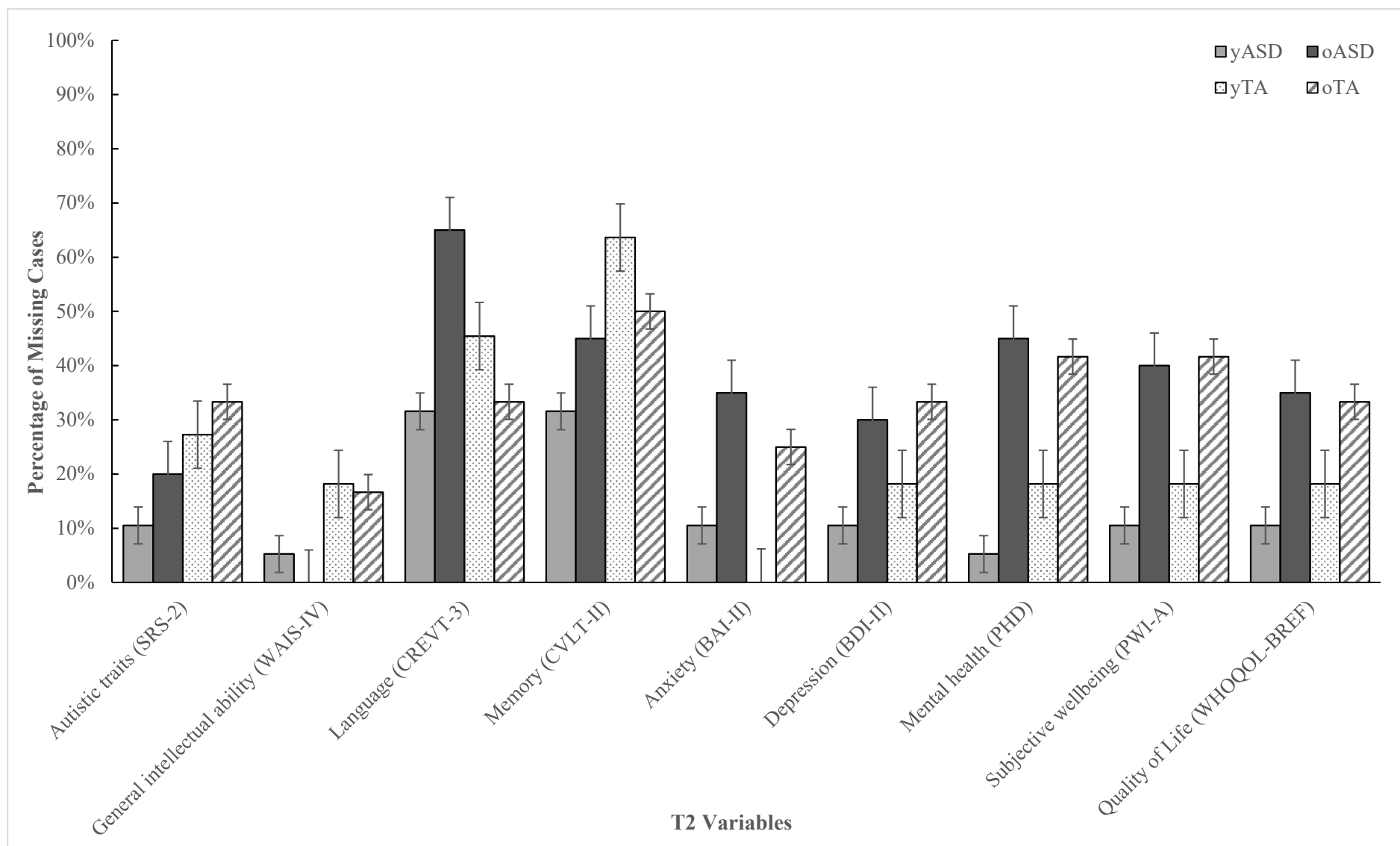


Figure 5.2. Missing Value Analysis on T2 variables within Diagnostic Groups and Age Groups.

Cross-sectional analysis of age-related differences at Time 2

The primary analysis at T2 focused on cross-sectional comparisons between Diagnostic Groups (TA; ASD) and Age Groups (younger; older) to confirm group *differences* at T2 assessment. The cross-sectional analysis follows the procedure set out in Chapter 4 (p. 95). The T2 data set out in Table 5.1 (p. 185) summarises the sample characteristics related to chronological age, gender ratio and years in formal education (YFE), as well as the Diagnostic Group differences in autistic traits, cognitive functioning, physical and mental health, and QoL. The domains just mentioned are set out in Table 5.2a (p. 188) for planned contrasts within Diagnostic Group between Age Groups, and in Table 5.2.b (p. 190) for planned contrasts within Age Group between Diagnostic Group. The cross-sectional analysis concludes with correlations between autistic traits, cognitive, physical and mental health factors and QoL, as set out in Table 5.3 (p. 198).

The Diagnostic Groups were assessed for homogeneity of variance (Levene's test), which confirmed homogeneity (smallest $p = .06$) and met the assumptions for ANOVA calculations. However, Kolmogorov-Smirnov Z was carried out to test normal distributions between Diagnostic Groups and highlighted unequal distribution in Autistic traits as would be expected, as well as in anxiety, depression and QoL, but normal distribution on age and IQ scores. Accordingly, a 2 x 2 Multivariate Analysis of Variance (MANOVA) analysed the differences between Diagnostic Groups (TA; ASD) and Age Groups (younger; older), on the primary outcome variables of interest, namely: autistic traits, general intellectual ability, memory, anxiety, depression, subjective wellbeing and QoL. The overall results from the MANOVA are reported with Bonferroni correction for multiple comparisons.

Longitudinal analysis of change

Then, longitudinal *changes* were assessed by comparing test-retest scores between T1 and T2 across measures of autistic traits, cognitive functioning (general intellectual ability, language, memory), mental health (anxiety, depression) and QoL. Given the limited prior literature on ageing and ASD, to our knowledge there are no precedents for the statistical approaches to evaluating longitudinal change in ASD. Therefore, the typical ageing literature was consulted for best practice

methods to evaluate longitudinal change scores, and to account for test-retest reliability and practice effects in younger and older cohorts. Accordingly, the longitudinal analysis in Study 2 applied two methods of analysis to detect statistical change from T1 to T2 scores (see Frerichs & Tuokko, 2005, for a review of the methods summarised below).

The first assessment of change followed the Standard Deviation (SD) method, using the formula:

$$(X_2 - X_1) / SD$$

where X_2 represents the individual score at T2 (averaged for each Diagnostic Group) and X_1 represents the individual score at T1 (averaged for each Diagnostic Group), and SD is the T1 standard deviation of the mean for each Diagnostic Group. The calculation results in SD-change scores, where ± 1 SD indicates change. Respectively, scores $\geq +1SD$ indicate improved change, whereas scores ≤ -1 SD indicate deterioration (Appendix 3, Table 5.4b, p. 359).

A secondary analysis used the Reliable Change Index (RCI) method to “correct for measurement error and practice effects” (Frerichs & Tuokko, 2005, p. 324). RCIs are the statistical methods used for “determining the significance of test score changes in serial neuropsychological assessment of older adults” (Frerichs & Tuokko, 2005, p. 321). The RCI method has been widely used to assess change scores between two or more time-points (i.e. T1 to T2) as a measure of cognitive change in older typically ageing adults (Woods et al., 2006; Frerichs & Tuokko, 2005; Gavett, Ashendorf & Gurnani, 2015). This approach has been also applied to behavioural assessment of autistic children (see Barber, 2012), in assessing longitudinal outcomes on a given measure. The RCI is calculated as follows for ASD and TA Diagnostic Groups, respectively:

$$(X_2 - X_1) - (M_2 - M_1) / SED$$

where X_2 is the individual participants’ scores at T2 and X_1 is their score at T1; M_2 is the mean score for each Diagnostic Group at T2, and M_1 is the group mean score at T1. The Standard Error of the difference score (SED) is calculated as the Standard Deviation of the mean observed difference (M_{diff}) score. In order to calculate these scores, M_1 and M_2 are calculated first, obtaining M_{diff} and SED accordingly. The RCI was then applied to individual difference scores ($X_2 - X_1$), using the above formula. The confidence interval (CI) for these scores were calculated for 95% ($SE \times 1.96$) and 90%

(SE x 1.645), as recommended by Frerichs & Tuokko (2005). Accordingly, difference scores that are outside the CI range (above or below) are indicators of reliable change. For cognitive assessments and QoL measures, scores that fall below the CI indicate decline or ageing-negative outcomes, and those scores above CI indicated improvement or ageing-positive outcomes, whilst scores that fall within the CI are deemed stable or ageing-neutral outcomes. Accordingly, better cognitive and wellbeing outcomes refer to ageing-neutral or ageing-positive RCIs. Whereas, for the clinically relevant assessments, such as the degree of autistic traits as measured by the SRS-2 and measures of anxiety and depression, a decline in symptoms or ageing-negative RCI would be suggestive of better outcomes (Table 5.4a, Appendix 3, p. 358).

In order to confirm the results arising from the above methods, paired sample t-tests were used to confirm test-retest (T1 to T2) change scores for each Diagnostic Group. The appropriateness of each statistical method relative to Study 2 is addressed in the Discussion of the present chapter, in relation to a reliable and meaningful evaluation of change in ageing and ASD.

5.6 Results

Cross-sectional comparisons of Diagnostic Group differences

The main comparisons between Diagnostic Groups and Age Groups are set out below. Additionally, the summary findings from planned contrasts between Age Groups within Diagnostic Groups are set out in Table 5.2a (p. 188), and between Diagnostic Groups within Age Groups in Table 5.2b (p. 190). Accordingly, those data are reported below for autistic traits, cognitive functions (general intellectual ability; language; memory), mental health, and wellbeing (SWB and QoL).

Autistic traits

Profiles of autistic traits, as measured by the SRS-2, differed significantly between Diagnostic Groups across domains, as would be expected (see Table 5.1, p. 185) and these differences were upheld in planned contrasts between Diagnostic Groups within Age Groups (Table 5.2b, p. 190).

Cognitive functions

The main effects of Diagnostic Group differences are reported below, where observed. There were no significant effects of Age Group, and no interaction effects between Diagnostic Groups within Age Groups on any T2 cognitive outcomes.

(i) General intellectual ability

There were no Diagnostic Group differences on any of the indices of IQ, namely: full-scale IQ, verbal comprehension (VCI), perceptual reasoning (PRI), working memory (WMI) and processing speed (PSI; Table 5.1, p. 185). Furthermore, there were no differences between Age Groups within Diagnostic Groups (Table 5.2a, p. 188) or between Diagnostic Groups within Age Groups (Table 5.2b, p. 190).

(ii) Language

There were no Diagnostic Group differences in Receptive and Expressive Language, or General Vocabulary (Table 5.1, p. 185). However, planned contrasts between Age Groups within Diagnostic Groups (Table 5.2a, p. 188) revealed significant differences between oASD and yASD adults in Expressive Language, which was explained by higher scores for older ASD adults. In the TA group, there were significant differences between yTA and oTA adults in Receptive and Expressive Language, and General Vocabulary resulting from lower scores in the oTA adults. The planned contrasts between Diagnostic Groups within Age Groups (Table 5.2b, p. 190) revealed significant differences between yTA and yASD adults in Expressive Language, which was explained by lower scores for yASD adults. There were no Language differences between oASD and oTA adults.

(iii) Memory

Regarding *Primary and Process Memory* and *Incremental learning, Simple and Complex Memory*, there were no Diagnostic Group differences in any of the outcome measures (Table 5.1, p. 185). Nor were there any differences in planned contrasts between Age Groups within Diagnostic Groups (Table 5.2a, p. 188; and see Figure 5.3, p. 192). The planned contrasts between Diagnostic

Groups within Age Groups revealed a significant difference only in: *ii. simple learning* between oASD and oTA adults, reflected by fewer correct responses at Trial 1 for oASD adults (Table 5.2b, p. 190).

Mental Health

The main effects of Diagnostic Group differences are reported below, where observed. There were no significant effects of Age Group, and no interaction effects between Diagnostic Group within Age Group on any T2 mental health outcomes. Where relevant, Age Group effects and interactions between Diagnostic Groups and Age Groups are reported for subjective wellbeing and QoL.

On the self-report measure of anxiety (BAI), there were significant Diagnostic Group differences in anxiety, which was driven by higher self-reported symptoms in the ASD group compared to the TA group. Whereas, on the self-report measure of depression (BDI-II), scores were on the edge of significance ($p = .056$), with higher depression scores in the ASD group compared to the TA group. There were no significant differences between Diagnostic Groups in levels of anxiety or depression. In addition, a significantly greater degree of psychiatric symptoms (PHQ) associated with depressive disorders, anxiety disorders, and increased difficulties with daily living, were observed in the ASD group compared to TA group (Table 5.1, p. 185). However, there were no Diagnostic Group differences in symptoms of Somatic complaints, binge eating disorder, or alcohol abuse.

Planned contrasts between Age Groups confirmed no age-related differences in mental health within either the ASD group or the TA group (Table 5.2a, p. 188). However, planned contrasts between Diagnostic Groups within Age Groups revealed significant differences between yTA and yASD adults, but not between oASD and oTA adults (Table 5.2b, p. 190). This was because yASD adults score higher than yTA and oASD adults, on measures of anxiety and depression. Furthermore, a greater degree of psychiatric symptoms (PHQ) was associated with depressive disorders, anxiety disorders, and increased difficulties with daily living in younger ASD adults.

Quality of Life and Subjective Well-being

(i) Subjective Wellbeing (SWB)

There were significant Diagnostic Group differences associated with overall SWB, global life satisfaction (GLS), satisfaction with personal relationships (pers. relation.), feeling part of the community (community), and sense of safety (safety; Table 5.1, p. 185). This reflected lower scores in the ASD group compared to the TA group. There were no Diagnostic Group differences in SWB outcomes associated with sense of achievement (achieving), and satisfaction with health, future, or standard of living. Nevertheless, significant Age Group effects were observed for overall SWB, community, future, and standard of living. There were also significant interaction effects between Diagnostic Groups within Age Group for overall SWB, personal relationships, and future outcomes.

Planned contrasts between Age Groups within Diagnostic Groups revealed significant differences between yASD and oASD adults in the majority of SWB outcomes (Table 5.2a, p. 188). These effects reflected lower SWB reports by yASD compared to oASD adults. However, there were no age-related differences in health or safety outcomes in the ASD group. In the TA group, there were no age-related differences in any of the SWB outcomes.

The planned contrasts between Diagnostic Groups within Age Groups confirmed significant differences between yASD and yTA adults on the majority of SWB outcomes, with the exception of community, health, and standard of living (Table 5.2b, p. 190). The significant differences were explained by lower scores by younger ASD compared to younger TA adults. However, in the older groups there were no significant differences on any SWB measures.

(ii) Quality of Life

Significant Diagnostic Group differences were observed in overall QoL, and in physical, psychological, social and environmental domains (Table 5.1, p. 185). A significant interaction effect between Diagnostic Groups within Age Group was observed for social QoL but did not reach significance for Health QoL ($p = .088$, $\eta_p^2 = .071$). Further, support from others (support) was on the edge of significance with a medium effect size ($p = .059$, $\eta_p^2 = .086$), suggesting that Diagnostic Group differences may be masked in the smaller sample.

Planned contrasts between Age Groups within Diagnostic Groups revealed significant age-related differences in Social QoL between yASD and oASD adults, but not on any other QoL outcome (Table 5.2a, p. 188). This was driven by lower Social QoL reports by yASD compared to oASD adults. In the TA group, significant age-related differences in Social QoL and Support were explained by lower scores for oTA compared to yTA adults.

In planned contrasts between Diagnostic Groups within Age Groups significant differences were observed between yASD and yTA adults on across QoL domains (Table 5.2b, p. 190). However, there were no differences in QoL between older ASD and TA groups, although Psychological QoL was on the edge of significance ($p = .058$). This reflected lower scores for Psychological QoL by oASD compared to oTA adults.

Table 5.1. Time 2 Cross-sectional comparisons between Diagnostic Groups.

Measure	Diagnostic Groups (N=62) ^a		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η ² _p
<i>Characteristics</i>	ASD (n=39)	TA (n=23)		F(1,51)		
Age (years) ^b	48.65 (14.27)	52.71 (16.22)	23.02-80.33	4.20	.045	.061
Gender (m:f) ^c	32:7	12:11	-	χ²(1) 6.27	.012	.097
YFE ^d	15.29 (2.65)	14.09 (2.45)	9-21	2.97	<i>n.s.</i>	.049
<i>Autistic traits^e</i>	ASD (n=33)	TA (n=16)		F(1,45)		
SRS-2 Total	71.97 (11.50)	46.25 (4.91)	39-90	70.33	<.001	.610
SRS-2 SCI	70.82 (11.48)	46.56 (4.87)	39-90	63.19	<.001	.584
SRS-2 RRB	73.42 (11.49)	46.25 (5.30)	40-90	79.48	<.001	.638
<i>General intellectual ability^f</i>	ASD (n=38)	TA (n=19)		F(1,51)		
FSIQ	114.72 (17.32)	113.68 (11.19)	69-145	<1.0	<i>n.s.</i>	.001
VCI	114.75 (13.95)	113.53 (9.59)	87-141	<1.0	<i>n.s.</i>	.002
PRI	112.75 (17.36)	112.05 (11.87)	65-144	<1.0	<i>n.s.</i>	<.001
WMI	113.56 (17.46)	108.47 (14.25)	80-150	2.44	<i>n.s.</i>	.021
PSI	104.06 (17.11)	106.53 (14.32)	59-137	<1.0	<i>n.s.</i>	.006
<i>Language^g</i>	ASD (n=20)	TA (n=14)		F(1,30)		
Receptive (RL)	94.40 (14.27)	97.00 (7.41)	72-115	<1.0	<i>n.s.</i>	.015
Expressive (EL)	102.90 (11.71)	105.21 (7.20)	86-120	<1.0	<i>n.s.</i>	.005
Difference (RL-EL)	11.70 (7.33)	8.78 (6.67)	1-27	1.78	<i>n.s.</i>	.056
General Voc.	98.75 (12.94)	100.79 (7.11)	78-119	<1.0	<i>n.s.</i>	.006
Level of Language	2.85 (.75)	3.14 (.36)	1-4	1.33	<i>n.s.</i>	.042

Measure	Diagnostic Groups (N=62) ^a		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η _p ²
Memory^h	ASD (n=24)	TA (n=10)		F(1,29)		
<u>Primary</u>						
Trial 1-5	52.91 (11.68)	56.50 (9.98)	Higher	<1.0	<i>n.s.</i>	.003
SDFR	11.17 (3.97)	11.60 (4.74)	Higher	1.21	<i>n.s.</i>	.040
SDCR	12.39 (3.01)	11.60 (4.65)	Higher	2.44	<i>n.s.</i>	.078
LDFR	11.91 (3.63)	13.50 (2.42)	Higher	<1.0	<i>n.s.</i>	.001
LDCR	12.43 (3.09)	13.10 (3.18)	Higher	<1.0	<i>n.s.</i>	.000
Recall (<i>d'</i>) Trial 1-5	2.25 (.52)	2.43 (.45)	Higher +	<1.0	<i>n.s.</i>	.000
<u>Process</u>						
Trial 1	6.39 (1.62)	7.6 (1.65)	Higher	<1.0	<i>n.s.</i>	.016
Trial 5	12.74 (2.82)	13.40 (2.32)	Higher	<1.0	<i>n.s.</i>	.001
Learning slope Trials 1-5	1.44 (.42)	1.41 (.42)	Higher	<1.0	<i>n.s.</i>	.018
Recall consistency %	83.00 (12.57)	86.20 (7.18)	Higher	<1.0	<i>n.s.</i>	.028
Semantic clustering	2.04 (2.37)	2.66 (2.47)	Higher +	<1.0	<i>n.s.</i>	.008
Total repetitions	6.30 (5.54)	6.40 (4.67)	Lower	<1.0	<i>n.s.</i>	.009
Total intrusions	3.53 (5.03)	2.40 (3.03)	Lower	<1.0	<i>n.s.</i>	.000
Source (<i>d'</i>)	3.04 (.96)	3.49 (.20)	Higher +	<1.0	<i>n.s.</i>	.004
2AFC recog. %	91.04 (27.85)	99.40 (1.90)	Higher	<1.0	<i>n.s.</i>	.000
Mental healthⁱ	ASD (n=30)	TA (n=20)		F(1,38)		
Anxiety	14.63 (10.67)	5.53 (5.01)	0-37	3.31	.030	.207
Levels Anxiety	2.20 (1.15)	1.40 (.74)	0-4	2.35	<i>n.s.</i>	.156
Depression	16.15 (12.16)	6.60 (6.07)	0-44	2.75	.056	.178
Levels Depression	1.56 (1.60)	.33 (.82)	0-5	2.48	<i>n.s.</i>	.164
Somatoform dis.	5.56 (3.87)	4.13 (3.54)	0-14	1.66	<i>n.s.</i>	.116
Depressive syn.	9.04 (7.27)	2.93 (3.65)	0-25	4.65	.007	.269
Anxiety syn.	9.52 (7.62)	2.80 (5.19)	0-25	3.37	.028	.210
Binge eating dis.	.93 (1.41)	.13 (.35)	0-5	1.46	<i>n.s.</i>	.103
Alcohol abuse	1.22 (1.67)	1.00 (.76)	0-9	.39	<i>n.s.</i>	.029
Diff. Daily Living	.89 (.80)	.22 (.41)	0-3	3.61	.022	.222
QoL^j & SWB^k	ASD (n=30)	TA (n=14)		F(1,35)		
SWB overall	42.48 (10.04)	50.29 (8.88)	7-64	6.36	.016	.154
	Age Group			5.66	.023	.139
	Diagnostic Group x Age Group			5.04	.031	.126
GLS	58.80 (17.16)	75.71 (12.23)	20-100	10.57	.003	.232
Pers. Relation.	47.48 (23.22)	70.86 (20.70)	0-100	10.84	.002	.236
	Diagnostic Group x Age Group			10.56	.003	.232
Community	53.80 (23.02)	69.07 (22.49)	0-100	4.68	.037	.118
	Age Group			7.29	.011	.172
Achieving	50.28 (20.81)	59.71 (20.89)	0-89	1.37	<i>n.s.</i>	.038
Health	53.84 (22.58)	63.57 (24.64)	0-100	1.38	<i>n.s.</i>	.038
Future	53.72 (24.53)	62.79 (28.57)	11-100	<1.0	<i>n.s.</i>	.023
	Age Group			5.00	.032	.125
	Diagnostic Group x Age Group			6.85	.013	.164
Std. Living	66.32 (22.68)	73.29 (13.45)	11-100	<1.0	<i>n.s.</i>	.025
	Age Group			4.83	.035	.121
Safety	69.00 (16.83)	82.64 (15.57)	33-100	5.66	.023	.139

Measure	Diagnostic Groups (N=62) ^a		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η _p ²
Overall QoL	60.34 (20.61)	80.00 (14.02)	0-100	10.21	.003	.203
Health	53.45 (24.75)	68.33 (29.07)	0-100	3.06	.088	.071
Physical	58.83 (16.70)	81.80 (17.23)	19-100	17.58	<.001	.305
Psychological	52.45 (16.85)	74.73 (14.08)	25-100	18.43	<.001	.315
Social	45.07 (19.44)	71.73 (18.48)	19-100	21.45	<.001	.349
	Diagnostic Group x Age Group			13.00	.001	.245
Environmental	63.86 (16.32)	78.87 (15.13)	19-100	8.06	.007	.168
Support	48.28 (26.67)	65.00 (26.39)	0-100	3.77	.059	.086
		Age Group		3.77	.059	.086

Notes: Findings are reported for effects between Diagnostic Groups, and between Age Groups, and interaction effects between Diagnostic Groups within Age Groups (Diagnostic x Age).

^a Diagnostic Groups: yASD = younger ASD; oASD = older ASD; yTA = younger TA; oTA = older TA.

^b Age (years): Chronological age at the time of testing.

^c Gender (m:f): ratio of male to female participants in each Diagnostic Group, as reported at T1 assessment.

^d YFE: Years of Formal (full time) education according the UK education system (primary, secondary, tertiary).

^e SRS-2: Social Responsiveness Scale–Second Edition (Constantino & Gruber, 2012). SCI = Social Communication Index; RRB = Restricted Interests and Repetitive Behaviours (see Table 4.0, p. 96). Missing data, N=13 (ASD, n=6; TA, n=7).

^f General intellectual function as measured by WAIS-IV (Wechsler, 2008). Data compared for sample matching: FSIQ = Full-scale IQ; VCI = Verbal Comprehension Index; PRI = Perceptual Reasoning Index. Missing data, N=5 (ASD, n=1; TA, n=4). Additional IQ measures for total intellectual functioning profile: WMI = Working Memory Index; PSI = Processing Speed Index.

^g Language: Receptive and Expressive Language measured by the CREVT-3 (Delis et al., Wallace & Hammill, 2013). Level of language reported on scale of 0-6 (0=Very poor; 1=Poor; 2=Below average; 3=Average; 4=Above average; 5=Superior; 6=Very superior; See Chapter 4). Missing data, N=28 (ASD, n=19; TA, n=9).

^h Memory as measured by CVLT-II (Ober et al., 2002). Primary and Process measures are detailed in Chapter 4. Missing data, N=28 (ASD, n=15; TA, n=13).

ⁱ Mental health: self-report; measured by the BAI (Beck & Steer, 1993); BDI (Beck et al., 1996); and PHQ (Spitzer et al., 1999). BAI Missing data N=12 (ASD, n=9; TA, n=3). BDI Missing data N=14 (ASD, n=8; TA, n=6). PHQ Missing data N=17 (ASD, n=10; TA, n=7).

^j QoL: Quality of Life; self-report; domains measured by the WHOQOL-BREF (WHO, 2000). Missing data N=15 (ASD, n=9; TA, n=6).

^k SWB: Subjective wellbeing domains measured by the PWI-A (Cummins et al., 2003). Missing data N=15 (ASD, n=9; TA, n=6).

Table 5.2a. Time 2 Cross-sectional comparisons between Age Groups within Diagnostic Groups.

Measure	ASD (n=39) ^a		Range	Statistics			TA (n=23) ^a		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η ²	Mean (SD)			ANOVA	Sig.	η ²
	yASD (n=19)	oASD (n=20)					yTA (n=11)	oTA (n=12)				
Age (years) ^b	36.05 (7.58)	59.95 (6.02)	24.53-73.22	120.70	<.001	.801	38.63 (8.30)	65.62 (9.13)	23.02-80.33	34.38	<.001	.726
Gender (m:f) ^c	16:3	15:4	-	<1.0	<i>n.s.</i>	.007	7:4	5:7	-	2.17	<i>n.s.</i>	.143
YFE ^d	15.47 (2.59)	15.11 (2.77)	9-21	<1.0	<i>n.s.</i>	.001	15.00 (1.84)	13.25 (2.70)	9-17	2.67	<i>n.s.</i>	.170
<i>Autistic traits^f</i>				F(1,33)						F(1,16)		
SRS-2 Total	71.47 (8.70)	72.50 (14.16)	40-90	<1.0	<i>n.s.</i>	.003	47.25 (5.97)	45.25 (3.69)	39-52	1.57	<i>n.s.</i>	.108
SRS-2 SCI	69.76 (8.58)	71.94 (14.15)	42-90	<1.0	<i>n.s.</i>	.010	47.38 (5.78)	45.75 (3.99)	39-54	1.23	<i>n.s.</i>	.086
SRS-2 RRB	75.24 (10.67)	71.50 (12.36)	45-90	<1.0	<i>n.s.</i>	.021	48.00 (5.53)	44.50 (5.75)	40-57	2.79	<i>n.s.</i>	.176
<i>General intellectual ability^e</i>				F(1,36)						F(1,17)		
FSIQ	111.06 (18.31)	118.39 (15.95)	69-145	1.64	<i>n.s.</i>	.046	117.11 (9.89)	110.60 (11.88)	92-136	1.66	<i>n.s.</i>	.089
VCI	111.67 (14.24)	117.83 (13.33)	87-141	1.80	<i>n.s.</i>	.050	115.11 (9.12)	112.10 (10.26)	100-130	<1.0	<i>n.s.</i>	.026
PRI	109.61 (18.88)	115.89 (15.58)	65-144	1.18	<i>n.s.</i>	.034	115.44 (8.55)	109.00 (13.97)	90-135	1.43	<i>n.s.</i>	.078
WMI	110.67 (18.70)	116.44 (16.13)	80-150	<1.0	<i>n.s.</i>	.028	113.22 (13.21)	104.20 (14.25)	88-136	2.01	<i>n.s.</i>	.106
PSI	101.72 (20.82)	106.39 (12.57)	59-132	<1.0	<i>n.s.</i>	.019	109.22 (14.98)	104.10 (14.04)	84-137	<1.0	<i>n.s.</i>	.034
<i>Language^g</i>				F(1,18)						F(1,12)		
Receptive (RL)	93.46 (15.43)	96.14 (12.76)	72-115	<1.0	<i>n.s.</i>	.008	102.17 (8.30)	93.12 (3.56)	87-111	7.76	.017	.393
Expressive (EL)	99.15 (11.28)	109.86 (9.65)	86-120	4.50	.048	.200	109.83 (4.92)	101.75 (6.86)	91-115	5.97	.031	.332
Difference (RL-EL)	10.62 (6.68)	13.71 (8.60)	2-27	<1.0	<i>n.s.</i>	.043	8.33 (7.00)	9.13 (6.88)	1-21	<1.0	<i>n.s.</i>	.004
General Voc.	95.92 (13.32)	104.00 (11.21)	78-119	1.85	<i>n.s.</i>	.093	106.33 (6.53)	96.63 (4.14)	90-113	11.64	.005	.492
Level of Language	2.69 (.75)	3.14 (.69)	1-4	1.73	<i>n.s.</i>	.088	3.33 (.52)	3.00 (0)	3-4	3.43	.089	.222
<i>Memory^h</i>				F(1,21)						F(1,13)		
<u>Primary</u>												
Trial 1-5	52.08 (14.80)	53.82 (7.59)	Higher	<1.0	<i>n.s.</i>	.006	56.00 (11.49)	56.83 (9.99)	Higher	<1.0	<i>n.s.</i>	.002
SDFR	10.33 (4.50)	12.09 (3.27)	Higher	1.13	<i>n.s.</i>	.051	10.50 (7.19)	12.33 (2.80)	Higher	<1.0	<i>n.s.</i>	.040
SDCR	11.83 (3.54)	13.00 (2.32)	Higher	<1.0	<i>n.s.</i>	.039	9.75 (6.85)	12.83 (2.48)	Higher	1.06	<i>n.s.</i>	.117
LDFR	11.17 (3.97)	12.732 (3.20)	Higher	1.06	<i>n.s.</i>	.048	14.25 (2.06)	13.00 (2.68)	Higher	<1.0	<i>n.s.</i>	.071
LDCR	11.92 (3.68)	13.00 (2.32)	Higher	<1.0	<i>n.s.</i>	.032	13.75 (2.87)	12.67 (3.56)	Higher	<1.0	<i>n.s.</i>	.031
Recall (<i>d'</i>) Trial 1-5	2.28 (.61)	2.23 (.43)	Higher +	<1.0	<i>n.s.</i>	.002	2.40 (.62)	2.45 (.36)	Higher +	<1.0	<i>n.s.</i>	.003
<u>Process</u>												
Trial 1	6.25 (2.18)	6.55 (.69)	Higher	<1.0	<i>n.s.</i>	.009	7.25 (1.71)	7.83 (1.72)	Higher	<1.0	<i>n.s.</i>	.033
Trial 5	12.67 (3.42)	12.82 (2.14)	Higher	<1.0	<i>n.s.</i>	.001	13.25 (2.50)	13.50 (2.43)	Higher	<1.0	<i>n.s.</i>	.003
Learning slope Trials 1-5	1.48 (.44)	1.39 (.41)	Higher	<1.0	<i>n.s.</i>	.013	1.50 (.28)	1.35 (.51)	Higher	<1.0	<i>n.s.</i>	.034
Recall consistency %	84.42 (13.54)	81.45 (11.87)	Higher	<1.0	<i>n.s.</i>	.014	89.25 (5.97)	84.17 (7.68)	Higher	1.24	<i>n.s.</i>	.134
Semantic clustering	1.99 (2.79)	2.10 (1.93)	Higher +	<1.0	<i>n.s.</i>	.001	3.28 (3.09)	2.25 (2.19)	Higher +	<1.0	<i>n.s.</i>	.046
Total repetitions	6.58 (4.83)	6.00 (6.45)	Lower	<1.0	<i>n.s.</i>	.003	4.75 (4.27)	7.50 (4.97)	Lower	<1.0	<i>n.s.</i>	.092
Total intrusions	3.58 (5.37)	3.55 (4.89)	Lower	<1.0	<i>n.s.</i>	<.001	2.25 (2.06)	2.50 (3.73)	Lower	<1.0	<i>n.s.</i>	.002
Source (<i>d'</i>)	3.04 (.86)	3.05 (1.10)	Higher +	<1.0	<i>n.s.</i>	<.001	3.63 (.15)	3.40 (.19)	Higher +	3.93	.083	.329
2AFC recog. %	91.12 (26.92)	90.91 (30.15)	Higher	<1.0	<i>n.s.</i>	<.001	98.50 (3.00)	100 (0)	Higher	1.6	<i>n.s.</i>	.167
<i>Mental healthⁱ</i>				F(1,25)						F(1,13)		
Anxiety	15.65 (11.01)	12.90 (10.40)	0-37	<1.0	<i>n.s.</i>	.016	6.13 (5.87)	4.86 (4.18)	0-17	<1.0	<i>n.s.</i>	.017
Levels Anxiety	2.29 (1.21)	2.10 (1.10)	0-4	<1.0	<i>n.s.</i>	.007	1.63 (.74)	1.14 (.69)	0-3	1.68	<i>n.s.</i>	.114
Depression	17.24 (11.50)	14.30 (13.65)	0-44	<1.0	<i>n.s.</i>	.014	6.75 (6.86)	6.43 (5.56)	0-22	<1.0	<i>n.s.</i>	.001
Levels Depression	1.65 (1.62)	1.40 (1.65)	0-5	<1.0	<i>n.s.</i>	.006	.38 (1.06)	.29 (.49)	0-3	<1.0	<i>n.s.</i>	.003
Somatoform dis.	6.47 (3.99)	4.00 (3.27)	0-14	2.74	<i>n.s.</i>	.099	3.38 (2.97)	5.00 (4.16)	0-12	<1.0	<i>n.s.</i>	.056
Depressive syn.	10.88 (7.44)	5.90 (6.06)	0-25	3.21	<i>n.s.</i>	.114	3.00 (3.34)	2.86 (4.26)	0-12	<1.0	<i>n.s.</i>	<.001
Anxiety syn.	10.59 (6.93)	7.70 (8.74)	0-25	<1.0	<i>n.s.</i>	.035	2.88 (5.54)	2.71 (5.19)	0-16	<1.0	<i>n.s.</i>	<.001
Binge eating dis.	.88 (1.50)	1.00 (1.33)	0-5	<1.0	<i>n.s.</i>	.002	.13 (.35)	.14 (.38)	0-1	<1.0	<i>n.s.</i>	.001
Alcohol abuse	1.41 (2.03)	.90 (.74)	0-9	<1.0	<i>n.s.</i>	.023	1.13 (.83)	.86 (.69)	0-3	<1.0	<i>n.s.</i>	.033
Diff. Daily Living	1.00 (.87)	.70 (.67)	0-3	<1.0	<i>n.s.</i>	.034	.13 (.35)	.29 (.49)	0-1	<1.0	<i>n.s.</i>	.040

Measure	ASD (n=39) ^a		Range	Statistics			TA (n=23) ^a		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η ² _p	Mean (SD)			ANOVA	Sig.	η ² _p
	yASD (n=19)	oASD (n=20)					yTA (n=11)	oTA (n=12)				
<i>QoLⁱ & SWB^k</i>												
<u>SWB</u>				<i>F(1,23)</i>						<i>F(1,12)</i>		
SWB overall ^k	36.79 (6.83)	49.73 (8.86)	7-64	17.07	<.001	.426	50.13 (6.83)	50.50 (11.81)	33-62	<1.0	<i>n.s.</i>	<.001
GLS	52.14 (16.72)	67.27 (14.21)	20-90	5.74	.025	.200	75.00 (9.26)	76.67 (16.33)	50-100	<1.0	<i>n.s.</i>	.005
Pers. Relation.	33.93 (16.66)	64.73 (18.68)	0-89	18.94	<.001	.452	75.25 (18.36)	65.00 (23.87)	22-100	<1.0	<i>n.s.</i>	.065
Community	45.14 (19.90)	64.82 (22.77)	0-100	5.31	.031	.188	61.00 (25.45)	79.83 (12.86)	22-100	2.72	.125	.185
Achieving	42.07 (17.28)	60.73 (20.90)	0-89	5.98	.023	.206	62.75 (14.62)	55.67 (28.30)	22-89	<1.0	<i>n.s.</i>	.030
Health	50.07 (18.09)	58.64 (27.44)	0-100	.882	<i>n.s.</i>	.037	62.63 (22.40)	64.83 (29.54)	22-89	<1.0	<i>n.s.</i>	.002
Future	37.86 (15.61)	73.91 (18.09)	11-100	28.59	<.001	.554	64.00 (22.20)	61.17 (37.78)	11-100	<1.0	<i>n.s.</i>	.003
Std. Living	55.57 (22.01)	80.00 (15.41)	0-100	9.75	.005	.298	72.50 (14.40)	74.33 (13.32)	56-89	<1.0	<i>n.s.</i>	.005
Safety	66.00 (14.31)	72.82 (19.63)	33-100	1.01	<i>n.s.</i>	.042	82.13 (11.67)	83.30 (20.95)	44-100	<1.0	<i>n.s.</i>	.002
<u>QoL</u>				<i>F(1,27)</i>						<i>F(1,13)</i>		
Overall QoL ⁱ	56.25 (21.41)	65.38 (19.20)	25-100	<1.0	<i>n.s.</i>	.050	81.25 (17.68)	78.57 (9.45)	50-100	<1.0	<i>n.s.</i>	.010
Health	56.25 (25.00)	50.00 (25.00)	0-75	<1.0	<i>n.s.</i>	.016	78.13 (8.84)	57.14 (40.09)	0-100	2.097	<i>n.s.</i>	.139
Physical	59.50 (14.80)	58.00 (19.36)	19-88	<1.0	<i>n.s.</i>	.002	86.88 (10.83)	76.00 (21.95)	31-100	1.546	<i>n.s.</i>	.106
Psychological	50.00 (19.47)	55.46 (12.58)	31-94	<1.0	<i>n.s.</i>	.027	80.50 (11.26)	68.14 (14.83)	44-100	3.358	<i>n.s.</i>	.205
Social	37.56 (17.81)	54.31 (17.82)	19-75	6.34	.018	.190	82.13 (17.84)	59.86 (10.82)	50-100	8.21	.013	.387
Environmental	61.00 (16.99)	67.38 (15.39)	19-88	1.10	<i>n.s.</i>	.039	80.50 (14.41)	77.00 (16.85)	44-100	<1.0	<i>n.s.</i>	.014
Support	50.00 (28.87)	46.15 (24.68)	0-100	<1.0	<i>n.s.</i>	.005	78.13 (20.86)	50.00 (25.00)	25-100	5.65	.034	.303

Notes:

^a Diagnostic Groups: yASD = younger ASD; oASD = older ASD; yTA = younger TA; oTA = older TA.

^b Age (years): Chronological age at the time of testing.

^c Gender (m:f): ratio of male to female participants in each Diagnostic Group, as reported at T1 assessment.

^d YFE: Years of Formal (full time) education according the UK education system (primary, secondary, tertiary).

^e Measures of general intellectual function for matching: FSIQ = Full-scale IQ; VCI = Verbal Comprehension Index; PRI = Perceptual Reasoning Index. Missing data, N=5 (yASD, n=1; yTA, n=2; oTA, n=2).

^f SRS-2: Social Responsiveness Scale–Second Edition (Constantino & Gruber, 2012;). SCI = Social Communication Index; RRB = Restricted Interests and Repetitive Behaviours (see Table 4.0, p. 96). Missing data, N=13 (yASD, n=2; oASD n=4; yTA, n=3; oTA, n=4).

^g Language: Receptive and Expressive Language measured by the CREVT-3 (Delis et al., Wallace & Hammill, 2013). Level of language reported on scale of 0-6 (0=Very poor; 1=Poor; 2=Below average; 3=Average; 4=Above average; 5=Superior; 6=Very superior; See Chapter 4). Missing data, N=28 (yASD, n=6; oASD, n=13; yTA, n=5; oTA, n=4).

^h Memory as measured by CVLT-II (Ober et al., 2002). Primary and Process measures are detailed in Chapter 4. Missing data, N=28 (yASD, n=6; oASD, n=9; yTA, n=7; oTA, n=6).

ⁱ Mental health: self-report; measured by the BAI (Beck & Steer, 1993); BDI (Beck et al., 1996); and PHQ (Spitzer et al., 1999). BAI Missing data N=12 (yASD, n=2; oASD, n=7; oTA, n=3). BDI Missing data N=14 (yASD, n=2; oASD, n=6; yTA, n=2; oTA, n=4). PHQ Missing data N=17 (yASD, n=1; oASD, n=9; yTA, n=2; oTA, n=5).

^j QoL: Quality of Life; self-report; domains measured by the WHOQOL-BREF (WHO, 2000). Missing data N=15 (yASD, n=2; oASD, n=7; yTA, n=2; oTA, n=4).

^k SWB: Subjective wellbeing domains measured by the PWI-A (Cummins et al., 2003). Missing data N=15 (yASD, n=2; oASD, n=7; yTA, n=2; oTA, n=4).

Table 5.2b. Time 2 Cross-sectional comparisons between Diagnostic Groups within Age Groups.

Measure	Younger (n=30)		Range	Statistics			Older (n=32)		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η^2	Mean (SD)			ANOVA	Sig.	η^2
	yASD (n=19)	yTA (n=11)					oASD (n=20)	oTA (n=12)				
Age (years) ^b	36.05 (7.58)	38.63 (8.30)	23.02-48.91	<1.0	<i>n.s.</i>	.045	59.96 (6.02)	65.62 (9.13)	51.65-80.33	4.10	.06	.157
Gender (m:f) ^c	16:3	7:4	-	<1.0	<i>n.s.</i>	.001	16:4	5:7	-	2.86	<i>n.s.</i>	.115
YFE ^d	15.47 (2.59)	15.00 (1.84)	11-21	<1.0	<i>n.s.</i>	.001	15.11 (2.77)	13.25 (2.70)	9-21	2.58	<i>n.s.</i>	.105
<i>Autistic traits^f</i>												
SRS-2 Total ^f	71.47 (8.70)	47.25 (5.97)	40-86	38.84	<.001	.649	72.50 (14.16)	45.25 (3.69)	39-90	28.07	<.001	.561
SRS-2 SCI ^f	69.76 (8.58)	47.38 (5.78)	40-85	34.20	<.001	.620	71.94 (14.15)	45.75 (3.99)	39-90	25.84	<.001	.540
SRS-2 RRB ^f	75.24 (10.67)	48.00 (5.53)	40-90	35.41	<.001	.628	71.50 (12.36)	44.50 (5.75)	40-90	34.91	<.001	.613
<i>General intellectual ability^e</i>												
FSIQ	111.06 (18.31)	117.11 (9.89)	69-143	<1.0	<i>n.s.</i>	.033	118.39 (15.95)	110.60 (11.88)	83-145	1.81	<i>n.s.</i>	.065
VCI	111.67 (14.24)	115.11 (9.12)	87-136	<1.0	<i>n.s.</i>	.017	117.83 (13.33)	112.10 (10.26)	92-141	1.39	<i>n.s.</i>	.051
PRI	109.61 (18.88)	115.44 (8.55)	65-135	<1.0	<i>n.s.</i>	.030	115.89 (15.58)	109.00 (13.97)	84-144	1.35	<i>n.s.</i>	.049
WMI	110.67 (18.70)	113.22 (13.21)	80-150	<1.0	<i>n.s.</i>	.005	116.44 (16.13)	104.20 (14.25)	88-150	4.01	.06	.134
PSI	101.72 (20.82)	109.22 (14.98)	59-136	<1.0	<i>n.s.</i>	.036	106.39 (12.57)	104.10 (14.04)	84-137	<1.0	<i>n.s.</i>	.007
<i>Language^g</i>				<i>F</i> (1,17)						<i>F</i> (1,13)		
Receptive (RL)	93.46 (15.43)	102.17 (8.30)	72-115	1.65	<i>n.s.</i>	.089	96.14 (12.76)	93.12 (3.56)	72-108	<1.0	<i>n.s.</i>	.031
Expressive (EL)	99.15 (11.28)	109.83 (4.92)	86-117	4.83	.042	.221	109.86 (9.65)	101.75 (6.86)	91-120	3.59	.081	.216
Difference (RL-EL)	10.62 (6.68)	8.33 (7.00)	1-23	<1.0	<i>n.s.</i>	.027	13.71 (8.60)	9.13 (6.88)	2-27	1.32	<i>n.s.</i>	.092
General Voc.	95.92 (13.32)	106.33 (6.53)	78-119	3.23	.090	.160	104.00 (11.21)	96.63 (4.14)	82-116	3.02	.106	.189
Level of Language	2.69 (.75)	3.33 (.52)	1-4	3.54	.077	.172	3.14 (.69)	3.00 (0)	2-4	<1.0	<i>n.s.</i>	.026
<i>Memory^h</i>				<i>F</i> (1,14)						<i>F</i> (1,15)		
<i>Primary</i>												
Trial 1-5	52.08 (14.80)	56.00 (11.49)	Higher	<1.0	<i>n.s.</i>	.016	53.82 (7.59)	56.83 (9.99)	Higher	<1.0	<i>n.s.</i>	.032
SDFR	10.33 (4.50)	10.50 (7.19)	Higher	<1.0	<i>n.s.</i>	.000	12.09 (3.27)	12.33 (2.80)	Higher	<1.0	<i>n.s.</i>	.002
SDCR	11.83 (3.54)	9.75 (6.85)	Higher	<1.0	<i>n.s.</i>	.045	13.00 (2.32)	12.83 (2.48)	Higher	<1.0	<i>n.s.</i>	.001
LDFR	11.17 (3.97)	14.25 (2.06)	Higher	2.14	<i>n.s.</i>	.133	12.732 (3.20)	13.00 (2.68)	Higher	<1.0	<i>n.s.</i>	.002
LDCR	11.92 (3.68)	13.75 (2.87)	Higher	<1.0	<i>n.s.</i>	.055	13.00 (2.32)	12.67 (3.56)	Higher	<1.0	<i>n.s.</i>	.004
Recall (<i>d'</i>) Trial 1-5	2.28 (.61)	2.40 (.62)	Higher +	<1.0	<i>n.s.</i>	.009	2.23 (.43)	2.45 (.36)	Higher +	1.16	<i>n.s.</i>	.072
<i>Process</i>												
Trial 1	6.25 (2.18)	7.25 (1.71)	Higher	<1.0	<i>n.s.</i>	.047	6.55 (.69)	7.83 (1.72)	Higher	4.938	.042	.248
Trial 5	12.67 (3.42)	13.25 (2.50)	Higher	<1.0	<i>n.s.</i>	.007	12.82 (2.14)	13.50 (2.43)	Higher	<1.0	<i>n.s.</i>	.023
Learning slope Trials 1-5	1.48 (.44)	1.50 (.28)	Higher	<1.0	<i>n.s.</i>	.000	1.39 (.41)	1.35 (.51)	Higher	<1.0	<i>n.s.</i>	.002
Recall consistency %	84.42 (13.54)	89.25 (5.97)	Higher	<1.0	<i>n.s.</i>	.032	81.45 (11.87)	84.17 (7.68)	Higher	<1.0	<i>n.s.</i>	.016
Semantic clustering	1.99 (2.79)	3.28 (3.09)	Higher +	<1.0	<i>n.s.</i>	.041	2.10 (1.93)	2.25 (2.19)	Higher +	<1.0	<i>n.s.</i>	.001
Total repetitions	6.58 (4.83)	4.75 (4.27)	Lower	<1.0	<i>n.s.</i>	.031	6.00 (6.45)	7.50 (4.97)	Lower	<1.0	<i>n.s.</i>	.016
Total intrusions	3.58 (5.37)	2.25 (2.06)	Lower	<1.0	<i>n.s.</i>	.016	3.55 (4.89)	2.50 (3.73)	Lower	<1.0	<i>n.s.</i>	.011
Source (<i>d'</i>)	3.04 (.86)	3.63 (.15)	Higher +	1.74	<i>n.s.</i>	.111	3.05 (1.10)	3.40 (.19)	Higher +	<1.0	<i>n.s.</i>	.038
2AFC recog. %	91.12 (26.92)	98.50 (3.00)	Higher	<1.0	<i>n.s.</i>	.020	90.91 (30.15)	100 (0)	Higher	<1.0	<i>n.s.</i>	.034
<i>Mental healthⁱ</i>				<i>F</i> (1,23)						<i>F</i> (1,15)		
Anxiety	15.65 (11.01)	6.13 (5.87)	0-37	5.20	.032	.184	12.90 (10.40)	4.86 (4.18)	0-37	3.70	<i>n.s.</i>	.198
Levels Anxiety	2.29 (1.21)	1.63 (.74)	0-4	2.04	<i>n.s.</i>	.082	2.10 (1.10)	1.14 (.69)	0-4	4.11	.061	.215
Depression	17.24 (11.50)	6.75 (6.86)	0-44	5.63	.026	.197	14.30 (13.65)	6.43 (5.56)	0-38	2.05	<i>n.s.</i>	.120
Levels Depression	1.65 (1.62)	.38 (1.06)	0-5	4.07	.055	.150	1.40 (1.65)	.29 (.49)	0-5	2.97	<i>n.s.</i>	.165
Somatoform dis.	6.47 (3.99)	3.38 (2.97)	0-14	3.79	.064	.142	4.00 (3.27)	5.00 (4.16)	0-12	<1.0	<i>n.s.</i>	.020
Depressive syn.	10.88 (7.44)	3.00 (3.34)	0-25	8.07	.009	.260	5.90 (6.06)	2.86 (4.26)	0-20	1.30	<i>n.s.</i>	.080
Anxiety syn.	10.59 (6.93)	2.88 (5.54)	0-25	7.57	.011	.248	7.70 (8.74)	2.71 (5.19)	0-25	1.81	<i>n.s.</i>	.108
Binge eating dis.	.88 (1.50)	.13 (.35)	0-5	1.96	<i>n.s.</i>	.078	1.00 (1.33)	.14 (.38)	0-3	2.69	<i>n.s.</i>	.152
Alcohol abuse	1.41 (2.03)	1.13 (.83)	0-9	<1.0	<i>n.s.</i>	.006	.90 (.74)	.86 (.69)	0-2	<1.0	<i>n.s.</i>	.001
Diff. Daily Living	1.00 (.87)	.13 (.35)	0-3	7.44	.012	.244	.70 (.67)	.29 (.49)	0-2	1.92	<i>n.s.</i>	.113

Measure	Younger (n=30)		Range	Statistics			Older (n=32)		Range	Statistics		
	Mean (SD)			ANOVA	Sig.	η_p^2	Mean (SD)			ANOVA	Sig.	η_p^2
	yASD (n=19)	yTA (n=11)					oASD (n=20)	oTA (n=12)				
<i>QoL^j & SWB^k</i>												
SWB				<i>F</i> (1,20)						<i>F</i> (1,15)		
SWB overall ^k	36.79 (6.83)	50.13 (6.83)	7-62	19.41	<.001	.493	49.73 (8.86)	50.50 (11.81)	26-64	<1.0	<i>n.s.</i>	.002
GLS	52.14 (16.72)	75.00 (9.26)	20-90	12.56	.002	.386	67.27 (14.21)	76.67 (16.33)	30-100	1.53	<i>n.s.</i>	.093
Pers. Relation.	33.93 (16.66)	75.25 (18.36)	0-100	29.13	<.001	.593	64.73 (18.68)	65.00 (23.87)	22-89	<1.0	<i>n.s.</i>	<.001
Community	45.14 (19.90)	61.00 (25.45)	11-100	2.65	<i>n.s.</i>	.117	64.82 (22.77)	79.83 (12.86)	0-100	2.18	<i>n.s.</i>	.127
Achieving	42.07 (17.28)	62.75 (14.62)	0-78	8.10	.010	.288	60.73 (20.90)	55.67 (28.30)	11-89	<1.0	<i>n.s.</i>	.012
Health	50.07 (18.09)	62.63 (22.40)	0-78	2.07	<i>n.s.</i>	.094	58.64 (27.44)	64.83 (29.54)	22-100	<1.0	<i>n.s.</i>	.012
Future	37.86 (15.61)	64.00 (22.20)	11-89	10.51	.004	.345	73.91 (18.09)	61.17 (37.78)	11-100	<1.0	<i>n.s.</i>	.057
Std. Living	55.57 (22.01)	72.50 (14.40)	0-89	3.77	.067	.158	80.00 (15.41)	74.33 (13.32)	56-100	<1.0	<i>n.s.</i>	.037
Safety	66.00 (14.31)	82.13 (11.67)	44-100	7.32	.014	.268	72.82 (19.63)	83.30 (20.95)	33-100	1.06	<i>n.s.</i>	.066
QoL				<i>F</i> (1,22)						<i>F</i> (1,18)		
Overall QoL ^j	56.25 (21.41)	81.25 (17.68)	25-100	8.09	.009	.269	65.38 (19.20)	78.57 (9.45)	25-100	2.87	<i>n.s.</i>	.138
Health	56.25 (25.00)	78.13 (8.84)	0-100	5.66	.026	.205	50.00 (25.00)	57.14 (40.09)	0-100	<1.0	<i>n.s.</i>	.013
Physical	59.50 (14.80)	86.88 (10.83)	38-100	21.38	<.001	.493	58.00 (19.36)	76.00 (21.95)	19-94	3.59	.074	.166
Psychological	50.00 (19.47)	80.50 (11.26)	25-100	16.24	.001	.425	55.46 (12.58)	68.14 (14.83)	31-88	4.09	.058	.185
Social	37.56 (17.81)	82.13 (17.84)	19-100	33.35	<.001	.603	54.31 (17.82)	59.86 (10.82)	25-75	<1.0	<i>n.s.</i>	.030
Environmental	61.00 (16.99)	80.50 (14.41)	19-100	7.72	.011	.260	67.38 (15.39)	77.00 (16.85)	44-94	1.67	<i>n.s.</i>	.085
Support	50.00 (28.87)	78.13 (20.86)	0-100	5.97	.023	.213	46.15 (24.68)	50.00 (25.00)	0-100	<1.0	<i>n.s.</i>	.006

Notes:

^a Diagnostic Groups: yASD = younger ASD; oASD = older ASD; yTA = younger TA; oTA = older TA.

^b Age (years): Chronological age at the time of testing.

^c Gender (m:f): ratio of male to female participants in each Diagnostic Group, as reported at T1 assessment.

^d YFE: Years of Formal (full time) education according the UK education system (primary, secondary, tertiary).

^e Measures of general intellectual functioning for matching: FSIQ = Full-scale IQ; VCI = Verbal Comprehension Index; PRI = Perceptual Reasoning Index. Missing data, N=5 (yASD, n=1; yTA, n=2; oTA, n=2).

^f SRS-2: Social Responsiveness Scale–Second Edition (Constantino & Gruber, 2012;). SCI = Social Communication Index; RRB = Restricted Interests and Repetitive Behaviours (see Table 4.0, p. 96). Missing data, N=13 (yASD, n=2; oASD n=4; yTA, n=3; oTA, n=4).

^g Language: Receptive and Expressive Language measured by the CREVT-3 (Delis et al., Wallace & Hammill, 2013). Level of language reported on scale of 0-6 (0=Very poor; 1=Poor; 2=Below average; 3=Average; 4=Above average; 5=Superior; 6=Very superior; See Chapter 4). Missing data, N=28 (yASD, n=6; oASD, n=13; yTA, n=5; oTA, n=4).

^h Memory as measured by CVLT-II (Ober et al., 2002). Primary and Process measures are detailed in Chapter 4. Missing data, N=28 (yASD, n=6; oASD, n=9; yTA, n=7; oTA, n=6).

ⁱ Mental health: self-report; measured by the BAI (Beck & Steer, 1993); BDI (Beck et al., 1996); and PHQ (Spitzer et al., 1999). BAI Missing data N=12 (yASD, n=2; oASD, n=7; oTA, n=3). BDI Missing data N=14 (yASD, n=2; oASD, n=6; yTA, n=2; oTA, n=4). PHQ Missing data N=17 (yASD, n=1; oASD, n=9; yTA, n=2; oTA, n=5).

^j QoL: Quality of Life; self-report; domains measured by the WHOQOL-BREF (WHO, 2000). Missing data N=15 (yASD, n=2; oASD, n=7; yTA, n=2; oTA, n=4).

^k SWB: Subjective wellbeing domains measured by the PWI-A (Cummins et al., 2003). Missing data N=15 (yASD, n=2; oASD, n=7; yTA, n=2; oTA, n=4).

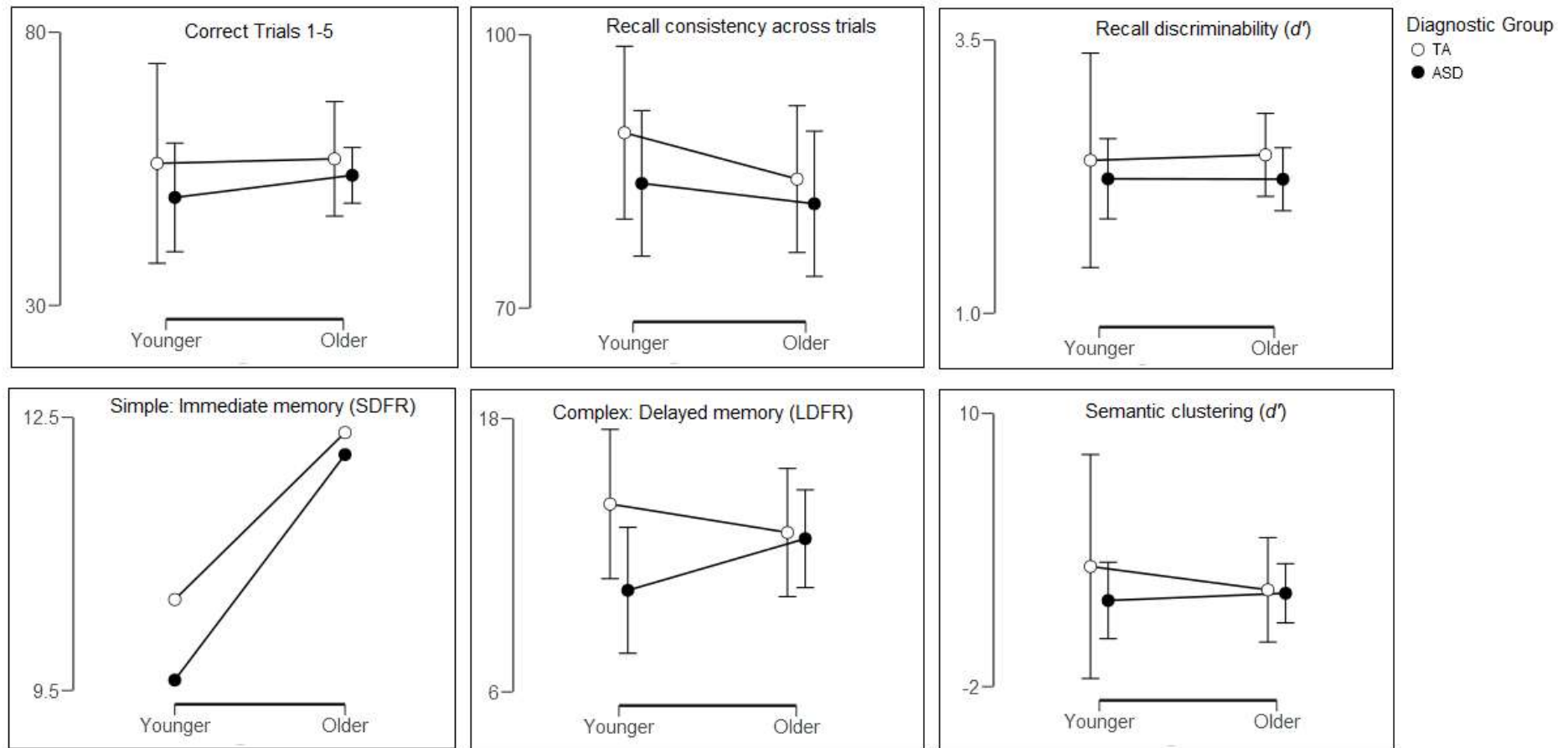


Figure 5.3. Time 2 Cross-sectional Memory Profiles: Incremental Learning^a, Simple^b and Complex^c memory. Error bars show ± 1 standard error.

Correlations and predictors of T2 outcomes

The T2 correlation analysis were exploratory, but the patterns justified further follow-up of associations between chronological age and cognitive outcomes with wellbeing, mental health and QoL. These factors were observed in both ASD and TA groups, but with different patterns of association, respectively.

(i) Typical ageing outcomes and QoL

In the TA group, older age was associated with greater language difficulties (receptive language, $r(14) = -.63, p < .02$); expressive language, $r(14) = -.58, p < .04$) and general vocabulary, $r(14) = -.70, p < .01$), but no other cognitive domains (all $r \leq -.37$, all $p \geq .17$). Older age was also associated with poorer Social QoL ($r(17) = -.599, p < .01$) and Support from others ($r(17) = -.56, p < .03$). Higher receptive language ability was associated with better overall SWB ($r(12) = .58, p < .05$) and better general vocabulary was significantly associated better Environmental QoL ($r(12) = .61, p < .04$). However, there was also a moderately negative association between poor general vocabulary and psychiatric symptoms associated with Somatic complaints, although the implications of this association are unclear as it did not reach significance ($r(12) = -.57, p = .052$). In terms of general intellectual ability and memory, there were significant correlations between verbal comprehension (VCI) and semantic clustering ($r(9) = .86, p < .004$). Perceptual reasoning (PRI) ability was associated with and primary simple memory (Correct Trials 1-5; ($r(9) = .67, p < .05$)) and primary complex memory (LDFR; ($r(9) = .78, p < .02$), and sustained process memory (recall consistency; ($r(9) = .79, p < .02$)). Processing speed (PSI) was positively correlated with primary complex memory (LDFR; ($r(9) = .69, p < .05$). None of the general intellectual ability outcomes were associated with any wellbeing, mental health or QoL domains (all $r(10) \leq .45$, all $p > .11$). Therefore, although processing speed was not directly associated with QoL outcomes in TA adults, its association with memory suggests that processing speed may play a mediating role in later life functioning (see p. 24 & p. 162). Furthermore, these findings mirror the patterning of data presented at T1 (p. 175). Primary complex memory (LDFR) was the only memory measure that positively correlated with overall SWB ($r(10) = .71, p < .02$); no other memory measures shared associations with QoL or wellbeing. As

previously indicated, these data should be interpreted with caution given the large amount of missing data for the T2 memory assessment.

Mental health difficulties adversely affected QoL and wellbeing across multiple domains. Increased anxiety was associated with poorer overall SWB ($r(16) = -.70, p < .003$), GLS ($r(16) = -.70, p < .004$), Physical QoL ($r(17) = -.65, p < .01$), Psychological QoL ($r(17) = -.72, p < .002$), and Environmental QoL ($r(17) = -.50, p < .05$), and less support from others ($r(15) = -.74, p < .003$), but not Social QoL ($r(17) = -.10, p < .07$). Similar patterns were observed between higher self-reported depression scores and lower overall SWB ($r(16) = -.60, p < .02$), GLS ($r(16) = -.50, p < .05$), Health QoL ($r(17) = -.52, p < .04$), Physical QoL ($r(17) = -.64, p < .006$) and Psychological QoL ($r(17) = -.87, p < .001$), and less support from others ($r(15) = -.67, p < .01$). Moreover, Anxiety and depression were highly positively correlated ($r(17) = .71, p < .002$), and both were associated with other psychiatric conditions. Depression was further positively associated with Eating Disorders ($r(15) = .73, p < .003$), Alcohol abuse ($r(15) = .72, p < .004$), and Difficulties in Daily Living ($r(15) = .52, p < .05$). Whereas, anxiety was positively correlated with Alcohol abuse ($r(17) = .66, p < .01$), but did not reach significance for associations with Eating Disorders ($r(15) = .49, p = .054$), or with Difficulties in Daily Living, $r(15) = .37, p > .10$). These findings suggest that anxiety and depression may have broader impacts on health-related behaviours that affect everyday life in typical ageing.

Finally, the analysis revealed that lower levels of autistic traits (social communication (SCI), restricted interests and repetitive behaviours (RRB)) were associated with better Overall SWB (all $r(15) \geq .66$, all $p \leq .01$), GLS (all $r(15) \geq .55$, all $p \leq .04$), and Psychological QoL (all $r(15) \geq .56$, all $p \leq .03$). Whereas, higher SCI and RRB scores at T2 were positively correlated with anxiety (all $r(16) \geq .54$, all $p \leq .04$) and depression (all $r(16) \geq .51$, all $p \leq .05$). Together, these findings highlight implications for communication difficulties and more inflexible behaviours on wellbeing in older age.

(ii) ASD ageing outcomes and QoL

In ASD group, older age was positively associated with expressive language ($r(20) = .55, p < .05$) and overall SWB ($r(20) = .40, p < .04$). Autistic traits related to SCI difficulties were unexpectedly positively correlated with expressive language ($r(20) = .61, p < .01$) and were on the

edge of significance for general vocabulary ($r(20) = .46, p = .056$). Nevertheless, both SCI and RRB difficulties were negatively associated with QoL across all domains (all $r(27) \geq -.38$, all $p \leq .05$), with the exception of Support from others and Social QoL, for which correlations did not reach significance (all $r(27) \leq -.19$, all $p \geq .36$). Moreover, greater difficulties related to both SCI and RRB were positively associated with higher scores for anxiety, depression and Difficulties in Daily Living (all $r(27) \geq .38$, all $p < .05$).

Analysis of cognitive functions revealed positive correlations between all memory outcomes (simple, complex, primary and process) and general intellectual ability for verbal (VCI) and non-verbal (PRI) IQ and processing speed (PSI), as well as all language measures (all $r(23) \geq .52, p < .01$). Working memory (WMI) was significantly associated with primary simple memory (SDFR; (all $r(23) .43, p < .05$), primary complex memory (LDFR; (all $r(23) .47, p < .03$) and process memory for recall consistency (all $r(23) .43, p < .04$), and was on the edge of significance for incremental learning ($r(23) = .41, p = .052$). Regarding QoL and mental health, higher VCI ability was negatively associated with Social QoL ($r(29) = -.37, p < .05$), and was moderately associated with Environmental QoL which was on the edge of significance ($r(29) = -.36, p = .055$). Social QoL was also negatively associated with higher WMI scores ($r(29) = -.42, p = .025$). Further, PRI ability was moderately associated with higher levels of anxiety ($r(29) = .367, p = .05$), and PSI with self-reported difficulties in daily living ($r(29) = .36, p = .057$). Finally, ability for expressive language (but not other language measures) was associated with higher depression ($r(29) = .45, p = .051$), and was negatively correlated with Environmental QoL ($r(19) = -.53, p < .03$). There no other significant patterns of association between cognitive functions and mental health, wellbeing and QoL. As with the patterning of findings in the TA group, the findings may be an indication of a person's ability to express their difficulties. Thus, the generalisability of these findings should be interpreted with caution.

Nevertheless, as observed at T1, higher anxiety and depression scores at T2 were associated with poorer QoL across multiple domains, namely: overall SWB (all $r(27) \geq -.59$, all $p < .001$), GLS (all $r(27) \geq -.46$, all $p \leq .016$), overall QoL (all $r(27) \geq -.54$, all $p \leq .003$), Health QoL (all $r(27) \geq -.54$, all $p \leq .003$), Physical QoL ($r(27) \geq -.54, p \leq .003$), Psychological QoL (all $r(27) \geq -.46$, all $p \leq .013$), and Environmental QoL (all $r(28) \geq -.39$, all $p \leq .035$). Anxiety and Depression were both

significantly associated with other psychiatric symptoms related to Somatic complaints (all $r(27) \geq -.49$, all $p \leq .007$) and increased Difficulties in Daily Living (all $r(27) \geq -.49$, all $p \leq .01$), whereas only depression was associated with Eating Disorder ($r(28) = -.39$, $p < .05$). In addition, a higher degree of Somatic complaints symptoms were associated with lower overall SWB ($r(26) = -.41$, $p < .04$), GLS ($r(26) = -.47$, $p < .02$), Health QoL ($r(28) = -.49$, $p < .01$), and Physical QoL ($r(28) = -.61$, $p < .002$). Moreover, Difficulties in Daily Living that arose from psychiatric symptoms, were significantly correlated with lower overall SWB ($r(26) = -.55$, $p < .004$), GLS ($r(26) = -.53$, $p < .01$), Psychological QoL ($r(28) = -.59$, $p < .002$) and Environmental QoL ($r(28) = -.39$, $p < .05$). As previously indicated, none of these findings indicates causal links and should, therefore, be interpreted with caution.

(iii) Predictors of QoL in ASD and typical ageing

Next, regression analyses were carried out to assess which factors were predictors of QoL outcomes (see Table 5.3, p. 198 for summary). Following the T1 results, the same predictor variables were included in the T2 regression model, to confirm whether these still predicted Health and Overall QoL outcomes at T2. These variables were: age, RRBs, processing speed, anxiety, depression, Somatic complaints, and difficulties in daily living. In addition, the T1 EF variables: strategy, planning, cognitive flexibility (perseverative errors and task switching), episodic memory and learning, and visual attention (rapid visual information processing) were also entered into the model to assess their impact on T2 QoL outcomes. These data are all illustrated in Table 5.3 (p. 198).

In the TA group, both Somatic complaints and Difficulties in Daily Living were significantly associated with Health QoL (Table 5.3, p. 198), whilst only the latter was a significant predictor in the regression model ($F(7,7) = 4.82$, $p = .027$). Whereas, for Overall QoL, Somatic complaints were the only significantly associated variable but it was not a significant predictor in the regression model ($F(7,7) < 1.0$, $p > .05$). When removing Age as a variable, the pattern of associations remained the same, but here no models emerged as significant predictors (all $F(6,8) < 1.59$, all $p > .05$). Since the TA sample size was very small at T2, the models did not hold when exploring predictors within Age Groups. Regarding T1 EF predictors, there were three variables that were significant correlated with Overall QoL at T2; these were planning ($p = .021$), episodic memory and learning ($p = .014$), and

sustained attention ($p = .048$), and. A fourth EF variable, attention reaction – which provides a measure of motor function difficulties – was on the edge of significance ($p = .052$). These four EF variables, as well as cognitive flexibility and strategy were included in the regression model to determine their predictive association with QoL. The overall model was significant when including episodic memory and learning, planning and strategy ($F(4,6) = 30.78, p < .001, R^2 = .95$). The strongest of these predictors was planning ($F(1,9) = 6.75, p = .029, R^2 = .43$). For Health QoL, only visual attention was significantly correlated ($p = .017$), but the model was significant for visual attention, planning and cognitive flexibility related to perseveration ($F(3,7) = 4.89, p = .039, R^2 = .95$), and visual attention was the strongest predictor of Health QoL ($F(1,9) = 6.33, p = .033, R^2 = .41$).

By contrast, in the ASD group all variables except for age and processing speed were significantly correlated with Health QoL and Overall QoL (Table 5.3, p. 198). However, the regression model showed that Depression was the only significant predictor of Health QoL ($F(7,15) = 4.76, p < .005$) and Overall QoL ($F(7,15) = 4.87, p < .005$). When age was removed as a variable, the association was still significant between depression and Health QoL ($F(6,16) = 5.68, p < .003$), and was on the edge of significance in the Overall QoL model ($p = .053$; model $F(6,16) = 5.92, p < .002$). When the predictors were explored within each Age Group, the regression models were not significant for either younger or older ASD groups (all $F \leq 3.23$, all $p > .05$). Nevertheless, Depression was still a significant predictor of Overall QoL in the younger ($R^2 = .71$; $t = -2.44, p = .041$), but not older adults ($R^2 = .68$; $t < -1.0, p > .05$). Regarding EF, whilst the model was significant when including cognitive flexibility (task switching, perseveration), attention reaction, planning and strategy ($F(5,18) = 3.04, p = .037, R^2 = .46$), the only significant predictor of Overall QoL in the model was cognitive flexibility (perseveration; $F(1,22) = 9.21, p = .006, R^2 = .30$). However, for Health QoL only attention reaction was significantly correlated ($p = .02$), and was the only factor contributing to the model ($F(1,22) = 4.77, p = .04, R^2 = .18$).

Table 5.3. Time 2 Predictors of Quality of Life in ASD and TA adults

	ASD							TA						
	Health QoL	Overall QoL	β	B	SE	t	sig.	Health QoL	Overall QoL	β	B	SE	t	sig.
Age	-.03	.13	-.11	-.19	.29	-.67	.52	-.31	.18	-.75	-1.19	.35	-3.41	.01
Processing Speed	-.16	-.16	-.04	-.05	.24	-.21	.83	-.19	-.22	.37	.69	.35	2.00	.09
SRS RRB	-.62***	-.62***	-.24	-.57	.50	-1.14	.27	-.09	-.34	-.54	-2.78	1.47	-1.90	.10
Anxiety	-.61***	-.60***	-.08	-.20	.61	-.33	.75	-.38	-.39	-.48	-2.66	1.49	-1.78	.12
Depression	-.72***	-.71***	-.43	-.90	.40	-2.24	.04	-.26	.08	.49	2.74	1.54	1.78	.12
Somatoform	-.51**	-.50**	-.19	-1.39	1.58	-.88	.39	-.51*	-.45*	.31	2.78	2.89	.96	.37
Daily Living	-.49**	-.55**	-.18	-6.98	6.97	-1.00	.33	-.62**	-.05	-.95	-32.99	9.40	-3.51	.01
Executive function														
Strategy	-.01	.08	.41	1.52	.87	1.75	.10	-.01	-.11	.58	1.41	.38	3.70	.03
Planning	-.03	.08	.16	1.47	2.01	.73	.47	-.03	.62*	.53	2.49	.94	2.66	.08
Cog. Flex. – perseveration	.23	.54**	.55	1.19	.37	3.17	.005	.23	-.27	-.04	-.04	.22	-.19	.86
Cog. Flex. – switching	.14	-.16	-.32	-.57	.46	-1.25	.23	.14	-.16	.02	.06	.49	.12	.91
Episodic memory and learning	-.30	-.16	-	-	-	-	-	-.30	.66**	.70	2.45	.58	4.20	.03
Sustained attention	-.18	.09	-	-	-	-	-	-.18	.53*	.35	79.85	34.98	2.28	.11
Attention reaction	.42*	.21	.29	15.72	10.19	1.59	.13	.42*	-.52 [†]	-.07	-2.72	7.53	-.36	.74

Notes: [†] $p = .052$; * $p < .05$; ** $p < .01$; *** $p \leq .001$

Health QoL R^2 : ASD .690**; TA .828*

Overall QoL R^2 : ASD .694**; TA .287 (*n.s.*)

Longitudinal changes within Diagnostic Groups

Using the RCI formula previously indicated in the Analysis Strategy (p. 179), the RCI change scores were calculated as the mean difference between test-retest scores at T1 and T2, respectively (see Table 5.4a, Appendix 3, p. 358). and re-checked for consistency. Lower and upper Confidence Intervals (CI) reported for 90% and 95% CI, with RCI calculated for each variable and CI. No RCI differences were observed across T1 to T2 in either Diagnostic Group (see Table 5.4a, p. 356). Overall, there were no declines in cognitive abilities, mental health and autistic traits, and QoL. Thus, the findings across all cognitive, mental health and QoL domains suggest *age-neutral* outcomes in both the ASD and TA groups, which did not differ with age. However, although the overall patterning of findings does not indicate age-related cognitive declines in ASD, the *age-neutral* longitudinal outcomes also do not indicate improvements in mental health, autistic traits and QoL.

To further confirm the longitudinal statistics, the secondary SD change analysis (p. 179) was carried out (see Table 5.4b, Appendix 3, p. 359). The change scores were manually calculated and re-checked using SPSS for consistency. Lower and upper Confidence Intervals (CI) are reported for 90% and 95% CI, with SD change scores calculated for each CI and each variable, respectively. The results revealed mean differences on some variables. However, the SD method also showed *age-neutral* effects on all variables (Table 5.4b, Appendix 3, p. 359). This patterning was the same in both ASD and TA groups and there were no age-related differences in either younger or older groups.

The final stage analysis involved paired *t*-Tests comparing T1 and T2 scores within each Diagnostic Group. The *t*-Tests revealed significant mean difference scores in the ASD group, suggesting a decline in RRB, and improvements in working memory and incremental learning and memory (see Table 5.4b, Appendix 3, p. 359). These results could be interpreted as *age-negative* outcomes for autistic traits associated with restricted interests and repetitive behaviours (RRB), and *age-positive* outcomes for working memory and learning in ageing and ASD. Whereas, in TA adults, the mean difference scores suggested significant improvements in working memory and expressive language and declines in subjective well-being. Thus, the *t*-test results can be interpreted as *age-negative* wellbeing outcomes, and *age-positive* working memory and language outcomes in typical ageing. Possible explanations for this patterning findings at T2 compared to T1 is discussed next.

5.7 Discussion

The aim of Study 2 was to establish a second time-point of cross-sectional data in the same cohort of younger and older autistic and typically ageing adults, in order understand the extent of age-related differences and the potential for increased magnitude of challenges for older autistic adults. The work presented in the present chapter, set out to revisit the research questions raised in Chapter 2, which were first addressed in the T1 cross-sectional assessments (Chapter 4). An additional question (p. 68) explored the trajectories of change, through longitudinal analysis of the change in test-retest scores from T1 to T2. The key findings from Study 2 are discussed in relation to the primary research questions. The implications of these findings are discussed in the context of challenges in conducting longitudinal research, with suggestions for future work.

Age-related cognitive differences in ageing and ASD compared to typical ageing

The cross-sectional findings at T2 showed no Diagnostic Group differences in cognitive function. Age-related differences in the ASD group were observed in expressive language, subjective wellbeing and QoL. These differences were driven by better language skills and higher self-reported levels of wellbeing and QoL, respectively, in older compared to younger ASD adults. These findings contrasted with the T1 cross-sectional findings of QoL, which showed no age-related differences. Whereas, the patterning of language abilities in older compared with younger ASD adults were aligned with the T1 findings.

In the TA group, language skills, social QoL and support from others at T2 were significantly lower for older TA adults compared to younger TA adults. Once again, these findings contrasted with the T1 cross-sectional findings that showed no age-related differences in QoL. Whereas, for language, the T1 data highlighted a significant difference in receptive and expressive language ability of older TA adults compared to younger TA adults. By T2, these differences were more pronounced and extended to general vocabulary difficulties. These findings appear to concur with research in the typical ageing literature that age-related declines in verbal ability (Ramscar et al., 2014) may have more far-reaching consequences for social functioning (Hobson, 2002). It may be, therefore, that the

language difficulties in older TA adults at T1 and T2 may mediate the social QoL and support difficulties observed at T2.

Cognitive profiles of older ASD adults and associations with QoL

A correlation analysis of the T2 variables revealed no significant associations between cognitive functions and QoL in the TA group. The two exceptions were language ability, already mentioned, and primary complex memory which was associated with overall subjective wellbeing. However, none of the cognitive variables was a significant predictor of Health QoL or Overall QoL – the two primary QoL outcomes.

In the ASD group, a negative association was observed between Social QoL and verbal comprehension and working memory aspects of general intellectual ability, where higher ability was associated with poorer Social QoL. Overall, mental health difficulties were the prevailing variables associated with poor QoL. Additionally, EF abilities in cognitive flexibility were also a predictor of QoL in ASD. This patterning of findings suggests that future support programmes of possible interventions may be beneficial if targeted to maintaining cognitive flexibility and improving everyday difficulties associated with mental health conditions.

Associations between the clinical features of ASD, co-occurring conditions and QoL

The T1 findings in the ASD group showed that autistic traits related to RRBs were negatively correlated with overall QoL, but not Health QoL. However, RRBs were not a predictor of QoL outcomes at T1. However, at T2, RRBs were significantly associated with both Overall and Health QoL, but were once again not significant predictors. Nevertheless, since RRBs were the only variables to show age-negative longitudinal outcomes in the ASD group, it would suggest that related difficulties continue to present challenges for autistic individuals into older age. Whereas, in the TA group, RRBs were a significant predictor of Health QoL at T1 (see Chapter 4, p. 157), but at T2 this was no longer the case. A possible explanation may be that RRBs abate in TA adults as they grow older. Alternately, it may be that factors related to sample attrition at T2 may be associated with the participants who had greater overall autistic difficulties that made ongoing participation challenging.

Regarding mental health, the overall profile of findings across the longitudinal study reflect enduring mental health difficulties in the ASD group, resulting in poor QoL. What is more, co-existing psychiatric conditions were significant predictors of QoL, suggesting a potential causal link. However, the direction of any association is unknown. Somatic complaints were associated with poorer overall and Health QoL in both TA and ASD participants but was only a significant predictor of QoL in the TA group, whereas depression was the significant predictor for ASD adults. Once again, these findings mirror the patterning of associations observed at T1. What is more, anxiety, depression, and difficulties in everyday life were all significantly associated with Health and Overall QoL in the ASD group, at T1 and T2. Given that these difficulties were still significant and enduring at T2, these findings raise important issues about the mental health and wellbeing needs of autistic adults in the context of ageing.

Possible explanations for longitudinal findings

There are several possible explanations for the patterning of results in the longitudinal analyses described above. First, it may be that the T2 sample included participants who were able to take part in the longitudinal study but did not include participants from the T1 cross-sectional study who had greater cognitive, functional or mental health difficulties resulting in their absence at T2. Furthermore, T1 difficulties related to processing speed were a significant predictor of Health QoL in the ASD group, as were anxiety and depression. Thus, it is possible that an interplay between cognitive factors and health status of autistic adults may have affected their ability to take part in the longitudinal study. As such, the T2 sample was broadly representative of the survivor effect in a vulnerable clinical population. This was particularly the case in the ASD group and older TA adults, raising important concerns for the wellbeing of individuals who were lost to follow-up and whose current status is unknown.

A second possible explanation is that the longitudinal methods used to analyse the data from this study were drawn from methods used in the typical ageing literature. Thus, it is possible that these methods may not be sufficiently sensitive to detecting changes in adult ASD samples, or between only two time-points. However, the data provide meaningful insights to possible ageing

trajectories in an adult ASD cohort. Third, the time between T1 and T2 assessments was relatively short, leaving open the possibility of practice effects for some of the assessments. However, all possible methodological precautions were taken to reduce practice effects, by using alternate forms of the language and memory assessments, and counterbalancing order of the QoL measures. Moreover, the statistical analyses used to assess longitudinal change (RCI method) is designed to account for test-retest effects, thus offering potentially reliable indicators of longitudinal outcomes.

Summary contribution of the T1-T2 longitudinal comparison

Time 1: When considering possible explanations about the observed profile of difficulties and abilities in ASD adults across the lifespan, it was proposed that cognitive coping resilience may be advantageous to autistic adults in later life. Whereas, TA adults incrementally adapt to cognitive declines with ageing and, therefore, show more marked changes in cognitive abilities, it was suggested that ASD adults have already developed the coping mechanism across their lifespan in response to dealing with life-long neurocognitive diversity. The overall findings appeared to concur with this suggestion, as evidenced by the high degree of mental health concerns in ASD adults and poor QoL and wellbeing across adulthood.

Time 2: The challenges of conducting longitudinal research have already been outlined and the mixed patterning of cross-sectional and test-retest longitudinal data from this study highlight those challenges. Nevertheless, the present study offers important contributions in that it is one of the few follow-up studies that tested the same cohort of younger and older autistic adults, and typically ageing comparison groups, with both cross-sectional and longitudinal data. The profile of strengths and difficulties was relatively consistent at both T1 and T2 cross-sectional data, suggesting that younger ASD adults may appear “prematurely cognitively old” (Bowler, 2008) – a patterning which mirrored the cognitive difficulties observed in the older TA adults in both Study 1 and Study 2. By contrast, older ASD adults who were matched on general intellectual ability at T1, appear to have fewer cognitive difficulties associated with their older age. What is more, the role of EF in ageing QoL outcomes appears to draw on different EF components in ASD adults than in TA adults. Planning, episodic memory and sustained attention are predictors of overall QoL for TA adults, whereas for

ASD adults, cognitive flexibility was the only predictor of Overall QoL. In addition, attention involving physical reactivity was a predictor of Health QoL in both TA and ASD adults.

These differences and similarities may provide insights into reasons associated with participant attrition over the course of the study, particularly given the high rate of co-occurring conditions already discussed. Moreover, these factors highlight important considerations in understanding the long-term health behaviours and QoL outcomes for autistic adults as they grow older. For instance, autistic traits related to social communication difficulties were unexpectedly positively correlated with expressive language and were on the edge of significance for general vocabulary. An important consideration is whether these findings may be an artefact of differences in ability to self-report more general difficulties, rather than intrinsically associated with autistic traits per se (Roestorf, Gaigg, Williams & Bowler, 2018, INSAR). Nevertheless, the findings across the T1 and T2 studies suggest that anxiety and depression may have broader impacts on health-related behaviours that affect everyday life. Consequently, these findings highlight implications for communication difficulties and more inflexible behaviours on wellbeing in older age.

Limitations and Future directions

The limitations of this study have already been discussed. An apparent explanation for the gap in the ageing and autism literature to date is the complex nature of conducting longitudinal research in a community cohort. Previous research has indicated that the presence of co-occurring complex health conditions can dramatically reduce life expectancy and exacerbate early mortality, particularly in ASD where the average age of death has been reported to be 55 years of age, and lower for individuals with lower cognitive ability including learning disabilities and epilepsy (Mouridsen, Bronnum-Hansen, Rich & Isager, 2008; Hirvikoski et al., 2016). However, other studies suggest that the average life expectancy in ASD for autistic individuals with higher cognitive ability is only minimally less than typically ageing individuals in the general population, for autistic individuals with higher cognitive ability. A report by Rosenblatt and colleagues (2008) reported the average life expectancy of individuals who were living independently was more than 65 years. Bancroft, Batten, Lambert and Madders (2012) reported the average life expectancy in ASD to be greater than 55 years

of age. Yet few studies have systematically explored ageing outcomes in older autistic samples over 50 years of age (e.g. Seltzer et al. 2011; Howlin et al., 2013; Howlin et al., 2014; Bishop-Fitzpatrick et al., 2016) and those have been mainly cross-sectional in nature (e.g. Geurts & Vissers, 2012; Lever & Geurts, 2015; Ring et al., 2016; Powell et al., 2017; Braden et al., 2017), or a retrospective examination of clinical records (Totsika et al., 2010; Croen et al., 2015; Wise et al., 2018).

In the present longitudinal study, a smaller study sample was involved at T2, with approximately 67% retention of the T1 cohort. There were approximately 2.5 years between the T1 and T2 data collection. At T2, groups were still broadly matched, although there was a small age-difference in the older ASD and TA groups owing to the loss of older ASD participants from the study. Over time, some participants who were recruited into the cross-sectional study reported here were lost to follow up (and see Chapter 3). The precise reasons for this are unknown since participants attended a day facility, which ceased operating during the second year of this programme of work. In addition, participants had several co-occurring physical and mental health conditions which prevented them from traveling to the research. However, although individual contact had been established with each person, there were no subsequent responses to follow-up over the course of the project, despite several attempts to contact these individuals.

Then, to establish reliability of change over time, two statistical approaches were used to analyse the mean differences between T1 to T2 outcomes. To our knowledge, there were no existing longitudinal studies in the ASD ageing literature to draw on, necessitating a recourse to the typical ageing literature for robust analyses to detect changes in scores and to account for test-retest reliability and practice effects. Although these analyses did not reveal any changes in T1 to T2 scores in the present study, they offer potential insights to the factors associated with wellbeing and QoL in the context of ageing.

The findings from the T1 and T2 studies highlighted that mental health difficulties persist over time in both younger and older autistic adults and have profound consequences for difficulties in everyday living. They are also linked to poor QoL. By contrast, the profile of cognitive abilities appears to suggest that older ASD adults may have developed a cognitive resilience across their lifespan, to facilitate cognitive coping in later life. Despite the profile of cognitive strengths, both

older and younger ASD adults had profound and enduring mental health difficulties and poor QoL and wellbeing, compared to typically ageing adults. These mental health difficulties, too, were ‘age-neutral’ in that they did not decline over the period of time covered here. Thus, although, on average the symptoms associated with anxiety, depression and other psychiatric conditions did not increase in severity during the course of the current study, they did not decrease either. Moreover, these conditions were significantly associated with increased difficulties in everyday life. Taken together, the profile of abilities and difficulties in the ASD group suggests that cognitive coping across the lifespan may be at the cost of mental health and wellbeing. Future work would need to specifically investigate the causal links between these factors, with a view to support autistic adults as they grow older.

5.8 Conclusions

The longitudinal research presented here is one step towards understanding what challenges are associated with ageing and ASD. Much more work is needed in broader cohorts, with sustained longitudinal follow-ups at multiple time points. Only through continued efforts can we understand the potential factors that may help or hinder the transition from premature cognitive ageing in young adulthood to cognitive resilience in older age.

Picking up on that theme, Part 2 of this thesis explores Prospective Memory (PM) – remembering to remember – and its relation to QoL. PM is a crucial factor in age-related cognitive decline in the typical population and is one of the primary mechanisms associated with QoL in typical ageing. Yet, although a small amount is known about PM in ASD, nothing is known of its role in ageing and QoL in this population. A review of the PM literature and methodological issues is set out in Chapter 6, highlighting the gap in knowledge about PM in ASD. This is followed by the presentation of findings from a collection of studies, set out in Chapter 7, which address that gap in knowledge.

Part 2

Chapter 6: Prospective memory, ageing and quality of life

The research presented in the previous Study Chapters (4 & 5) highlighted a profile of cognitive function in younger autistic adults that resembled that of older typically ageing adults. These difficulties appeared to be stabilised or improved in older autistic adults, suggesting that lifetime cognitive coping mechanisms may support autistic people in dealing with later-life cognitive challenges. Nonetheless, QoL and mental health were significantly poorer for the ASD compared to the TA adult, although these were not associated with either broader cognitive difficulties in ASD. Poorer QoL outcomes were, however, strongly associated with mental health concerns and depression which in turn correlated with self-reported difficulties in everyday life. Moreover, factors such as inter-participant variability in Studies 1 and 2, and the absence of a standardised measure of daily living skills, means that it was not possible to draw conclusions about how cognitive and wellbeing factors interact with QoL. Therefore, the present chapter takes a further step to explore the specific cognitive factors known to be associated with QoL outcomes in typical ageing.

Here, the mechanisms of everyday memory are explored through Prospective Memory (PM) – ‘remembering-to-remember’, or remembering to act on a planned intention (thought or action) at a specific point in the future (Brandimonte, Einstein, & McDaniel, 1996). In typical ageing, PM is a crucial index of age-related cognitive decline (Craik, 1986; Blanco-Campal et al., 2009) and core to maintaining good health-related QoL (Zogg, Woods, Saucedo, Wiebe, & Simoni, 2012; Woods, 2015). It is also important to everyday functioning and maintaining independence (Maylor, 1996; Brandimonte et al., 1996) and, consequently, plays a crucial role in QoL in older age (Pirogovsky et al., 2012; Woods et al., 2012). Furthermore, PM is often socially motivated, since memory slips or errors could lead to social embarrassment, withdrawal and isolation (e.g. forgetting a friend’s birthday; Brandimonte et al., 1996; Baddeley, 1997; Altgassen, Kliegel, Brandimonte & Filippello, 2010; Kretschmer, Altgassen, Rendell & Bölte, 2014).

However, the existing literature presents a complex and sometimes paradoxical picture of PM abilities and difficulties in typical older age. Moreover, very little is known about PM functions in ASD and nothing is known of its role in ageing and QoL in autism. The present chapter sets out a

broad review of the PM literature in typical ageing, followed by a summary of the few PM studies that have been carried out in younger ASD samples. The review will frame important methodological considerations for the work presented in Chapter 7. There, Study 3 aims to address the gap in knowledge just mentioned through a series of six experimental tasks designed to explore PM and ageing in ASD. A discussion of the study findings and their implications for ageing and ASD will conclude this section.

6.1 Literature Review

A distracted teenager is stranded after forgetting to leave the party on time for the last train home. An engineer is electrocuted after forgetting to switch off the power supply before working with live wiring. An elderly person who lives alone forgets to switch off the gas after they have finished cooking. A person taking life-critical medication forgets that they have already taken it earlier in the day, thus accidentally overdosing. All of these examples point to the critical role of PM in everyday life (Cohen, 1996; Kliegel & Martin, 2003; Will et al., 2009; Brewer et al., 2011). PM research has broadly focused on event-based (EBPM) or time-based (TBPM) remembering and its role in everyday functioning (McDaniel & Einstein, 2000; Einstein et al., 2005; Dismukes, 2008, 2012; Craik & Rose, 2012; Hering et al., 2015). EBPM is the self-directed execution of a planned intention, where remembering is cued by specific event. For instance, remembering to post a letter on the way to work may be prompted by the event cue of seeing the envelope before leaving the house, or passing the mail box en route. TBPM is also self-directed but requires execution of the planned intention at a specific future time, such as remembering to telephone the doctor at 2pm, or to attend an appointment at 9am next Thursday. A third, less well-established phenomenon is activity-based PM, whereby the end of one activity triggers remembering to carry out the next activity (e.g. remembering to switch off the gas after cooking, or to lock the door when leaving the house; Kvavilashvili and Ellis; 1996; Kumar, Nizamie & Jahan, 2008; Gonen-Yaacovi & Burgess, 2012). It has been argued that activity-based PM differs from EBPM in the type of cue (event) that triggers the PM action, and which “coincides with another activity and thus does not require the interruption of the [ongoing] activity” (Shum, Ungvari, Tang & Leung, 2004, p. 694; Brandimonte & Ferrante, 2015). However, study findings are equivocal

and the theoretical underpinnings of activity-based PM are not well established (Brewer, Marsh, Clark-Foos, Meeks et al., 2011; Rummel, Wesslein & Meiser, 2017). Consequently, it is not well understood which cognitive processes are involved or whether activity-based PM is merely a more or less cognitively demanding variant of EBPM (Shum et al., 2004; Brewer et al., 2011; Rummel et al., 2017; Walter & Meier, 2017). Therefore, the work presented in this chapter will not include activity-based PM but will instead focus on EBPM and TBPM. In order to understand the complex network of associated cognitive processes involved in PM in everyday functioning, a detailed description of the mechanisms underlying PM are described next.

Mechanisms of Prospective Memory

PM is a complex memory process involving the coordination and direction of multiple cognitive resources to complete everyday activities (McDaniel & Einstein, 2000; Ellis & Kvavilashvili, 2000; Craik & Rose, 2012; Penningroth & Scott, 2013). The core features of PM are the ability to generate internal intentions (*intention formation*) to execute a particular activity or function at some point in the future (*prospective*; remembering ‘*when*’ to remember; *intention initiation*) and *retrieve* the corresponding information from memory (*retrospective*; remembering ‘*what*’ needs to be remembered or actioned; *intention retrieval*) after a delayed period of time (retention interval) in order to perform the activity (*intention execution*) (McDaniel & Einstein, 2000; and see Einstein & McDaniel, 1996; Ellis, Kvavilashvili, & Milne, 1999; Uttl, Graf, Miller & Tuokko, 2001). Because everyday PM requires the ability to carry out a future intention during the course of unrelated activities, laboratory based PM tasks are usually embedded in ongoing (typically unrelated) tasks which prevent rehearsal of the *to-be-remembered intention*. Thus, conscious and active retention is interrupted during the delay between *intention formation* and *execution*.

Although it is evident that PM involves similar processes to everyday memory, such as encoding and retrieval of information over time, it contrasts with standard working memory or episodic memory tasks. This is because PM involves not only the retrieval of retrospective information, but also the prospective initiation of an intention in context at a future point, such as remembering to take medication with supper (Rose et al., 2015; and see Craik, 1986; Gonen-Yaacovi

& Burgess, 2012; Mattle et al., 2013). The specific combination of these cognitive processes involved in PM indicate that it is a separate cognitive construct with dissociable functional mechanisms involved in successful remembering (Kliegel, Martin, McDaniel & Einstein, 2002; Ball & Aschenbrenner, 2018). Furthermore, PM involves other memory mechanisms and EF (planning, task switching, inhibition; initiation) required to form the intention and plan future actions (binding), and to hold information in mind during distractions of unrelated tasks or event (working memory). These functions are deployed whilst simultaneously attending to information or ignoring irrelevant cues, whether internally or externally directed (e.g. attention; inhibition). Then, at the appropriate point, recalling from memory the content and context (retrospective memory; retrieval) of the to-be-remembered intention and initiating an action (initiation; execution; Einstein & McDaniel, 1996; Kliegel et al., 2002). All the factors just mentioned are particularly important in later life, since older adults may have fewer available resources to process the multiple competing cognitive demands associated with ongoing attention. What is more, self-initiation of internal responses may also prove more problematic for older adults, since these functions draw more heavily on EF which is known to decline in older age.

Several of the core differences between EBPM and TBPM are relevant in this regard. Both EBPM and TBPM rely on some aspect of internal referencing, in the *planning* of future goal-based actions and intentions (intention formation; remembering to do something in the future), *retention* of that intention over a period of time (retention interval) and their *execution* (remembering to do something at a particular time in the future), which may either be carried out (initiated) in response to an external cue (EBPM) or an internally triggered response (TBPM; Graf & Utl, 2001; Rendell, McDaniel, Forbes & Einstein, 2007; Einstein & McDaniel, 2010; Ball & Aschenbrenner, 2018). Whilst both require intention formation and successful execution of that intention (see Figure 6.1, p. 213), EBPM relies on external cues which serve as memory supports, such as a memo note or use of diary reminders to do a particular task in the future, or EBPM may be in conjunction with another event (e.g. remembering to fulfil a prescription when passing the pharmacy).

Therefore, although EBPM appears to make use of contextual binding of information which appears to draw more on frontal processes but not hippocampal (e.g. Lecouvey et al., 2015), it is

thought to place fewer demands on frontal lobe activation of executive resources, overall (Brewer et al., 2011; and see McDaniel & Einstein, 2000; Hicks, Marsh & Cook, 2005). By contrast, TBPM depends more extensively on the *self-directed* initiation of all PM features for successful execution, as well as internal time-keeping and monitoring, and self-directed use of planning, strategy and inhibition (stopping what one is currently engaged in to do the PM activity). Thus, TBPM draws not only on the construction of information, but also on the self-initiated execution of the task at the appropriate time (frontal processes), such as remembering to attend a doctor's appointment in 6 months' time or to turn off the cooker after 30 minutes (Block & Zakay, 2006; Gonen-Yaacovi & Burgess, 2012). The parallel demands of self-regulation, self-monitoring and self-generated action make TBPM more demanding on executive resources and associated frontal lobe functions (Craig, 1986; Gonen-Yaacovi & Burgess, 2012). What is more, recent fMRI data suggests that the underlying brain regions involved in EBPM and TBPM are sufficiently varied to suggest the reliance on different cognitive processes involved in external target-checking (EBPM) and internal monitoring and time-estimation (TBPM; Gonneaud et al., 2014). However, mnemonics, memos, and technological supports such as an alarm clock, timer or smart technology, are sometimes used as external cues for additional supports to aid retrieval in TBPM (Graf & Grondin, 2006; McDaniel & Einstein, 2007; Rabin, Chi, Wang, Fogel, Kann & Aronov, 2014). Thus, the distinction between TBPM and EBPM has become slightly blurred by the presence of an event-based component in both task types. Overall, successful PM depends, broadly, on self-directed action since there is an absence of instructions to attend to a specific time (TBPM) or cue (EBPM; also TBPM in the case of alarms etc.). PM success also depends on one's ability to execute the intended action at the appropriate point (Uttl, Graf, Miller & Tuokko, 2001; Gonen-Yaacovi & Burgess, 2012). There is, typically, no external feedback about whether the PM intention has been successfully executed or not, or whether it has already been completed. Consequently, the self-directed self-monitoring nature of PM means that there are no external indicators by which a person can gauge whether they have failed to remember the PM intention (omission error), or forgotten that they have already completed it and, thus repeat it in error (commission error; Boywitt, Rummell & Meiser, 2015).

Temporal perception is another important part of normal memory function in that it underpins a person's ability to organise their behaviours in relation to their experiences across time – past, present and future (*metarepresentational ability*; e.g. Perner, 1991; 2001; Bowler et al., 2011). For instance, time perception helps an individual to organise and process information about events, including setting intentions, “imagine scenarios” and expected outcomes, “make decisions” and form “contingencies”, and “take action” (Zimbardo & Boyd, 1999). Moreover, time perception combined with an individual's perspectives on time, is thought to be of social and cognitive importance for their behavioural self-regulation. For example, motivation, emotion and goal-directed behaviour draw on information from past experiences in relation to a given current situation and future objectives (Zimbardo & Boyd, 1999; Carstensen, Jaascowitz & Charles, 1999; Bandura, 1997). Thus, time perception arguably plays an important role in both retrospective and prospective memory and may offer important clues to successful PM functioning.

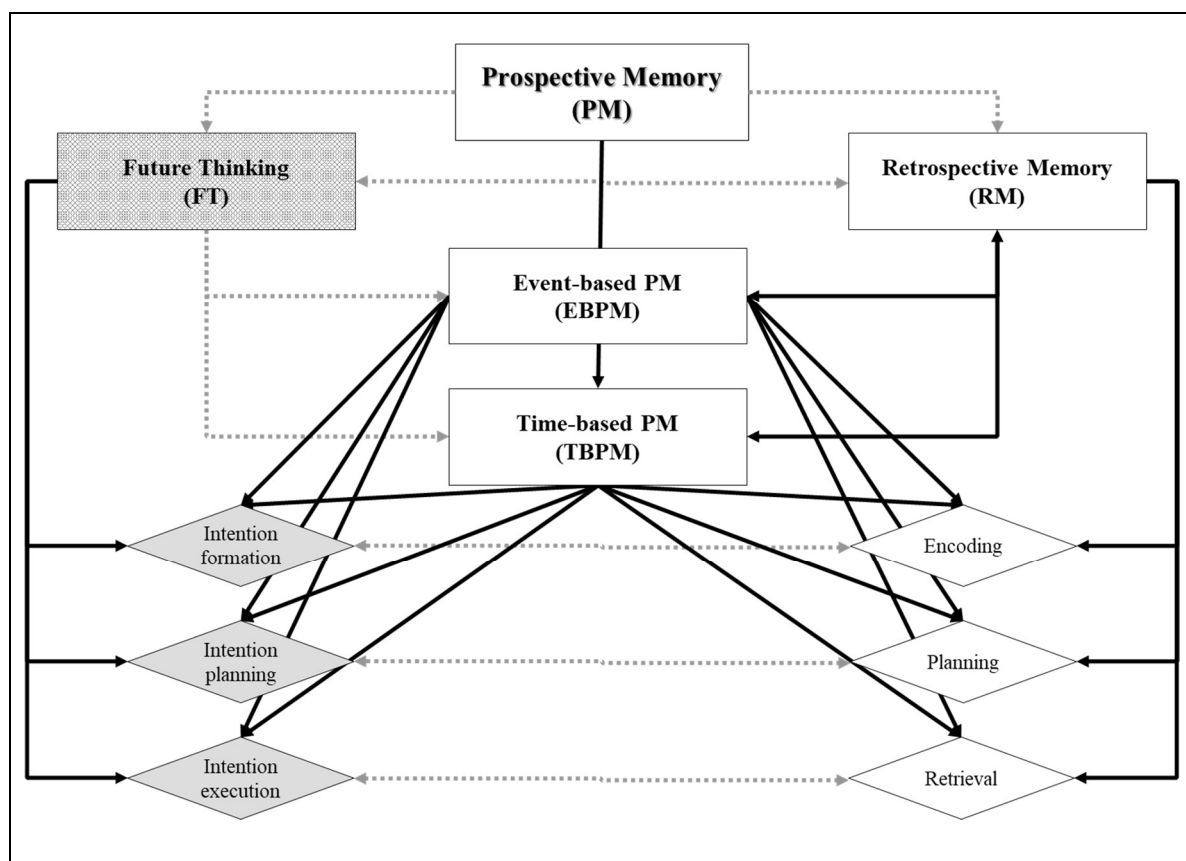


Figure 6.1. Schematic of Prospective Memory processes. Solid lines indicate core processes and dotted lines indicate testable relations between associated processes.

Prospective memory and everyday functions in typical ageing

As we have seen, everyday forgetting as a result of PM failures can have profound and far reaching impacts with practical and potentially life-threatening consequences (Brewer et al., 2011; Dismukes, 2012; Islam, 2014). For instance, PM failures are reported to contribute substantially to everyday forgetting that leads to medical or surgical errors (Dembitzer & Lai, 2003; Rogers et al., 2006; Dismukes, 2012), airline disasters (Dismukes, 2008), and health-related incidents resulting from medication mismanagement (Rendell & Thomson, 1993; Rendell & Craik, 2000; Pirogovsky et al., 2012). Consequently, the cognitive processes associated with EBPM and TBPM and their respective demands on executive processes, as outlined above, can offer insights into some of the consequences for PM difficulties in older age (Craik, 1986; Brewer et al., 2010; and see Hicks, Marsh & Cook, 2005). Moreover, some of cognitive difficulties highlighted in previous ASD literature suggests that the self-directed components of PM may be more problematic for autistic than typically ageing individuals (see Lind & Bowler, 2010; Bowler, Gaigg & Lind, 2011; and see Lind, Williams, Bowler & Peel, 2014). The sections that follow present what is known about PM functioning in each of these groups, and what is as yet unknown in relation to PM in ageing and ASD.

In typical ageing, PM performance is reportedly worse for older TA adults (i.e. above 60 years of age) on both EBPM and TBPM tasks, compared to younger TA adults (i.e. under 30 years of age), particularly in laboratory based tasks (see Henry et al., 2004, for a meta-analysis; and see Kvavilashvili et al., 2000; d'Ydewalle, Bouckaert, & Brunfaut, 2001; Altgassen, Kliegel, Brandimonte, & Filippello, 2010; Smith, Horn & Bayen, 2012; Ihle et al., 2013; Walter & Meier, 2015). However, a small number of other studies have found the opposite patterning under varying conditions, such as the use of additional memory aids, cue focality, and varied task load (e.g. Patton & Meit, 1993; Perez et al., 2013; and see McDaniel & Einstein, 2000). Moreover, the patterning of age-related PM difficulties corresponds to brain functions that are susceptible to cognitive decline in typical ageing (Salthouse, 1996; Neath & Surprenant, 2003). For instance, frontal brain regions associated with EF such as strategy, attention and self-initiation (West, 1996; Anderson & Craik, 2000; Bastin & Meulemans, 2002; Altgassen et al., 2010), and the hippocampal processes involved in memory encoding and retrieval of information are brain regions that are known to be vulnerable to

typical age-related declines (Cabeza et al., 1997; Hedden & Gabrieli, 2004; Anderson & Craik, 2017). Possible explanations for these differences point to reduced capacity of executive resources in older adults, as fundamental to successfully completing TBPM tasks (Henry et al., 2004; Schnitzspahn et al., 2013). According to this view, in typical ageing TBPM is particularly vulnerable to failure under the high demands on EF of the frontal lobes and the competing cognitive load of *internal* processes that are already subject to age-related declines (Craik & Rose, 2012; Perez et al., 2013; and see Ball & Aschenbrenner, 2018 for a statistically-derived theoretical discussion). However, the contrasts between TBPM and EBPM performance differences and their associated cognitive functions have not been widely researched (Einstein & McDaniel, 1990; Brewer et al., 2010) and are not definitively reported (Schnitzspahn et al., 2013; and see Henry et al., 2004; Rose et al., 2010; Altgassen et al., 2010). Moreover, few studies have contrasted TBPM and EBPM performance, and many studies have assessed both PM functions in a single paradigm. This presents potential methodological confounds, since performance difficulties on one task type may influence performance outcomes on another task type (Williams et al., 2014).

In naturalistic settings, however, the opposite pattern of age-related PM performance is observed (Gonen-Yaacovi & Burgess, 2012). Several studies have found that older adults often perform as well as or better than younger adults on EBPM and TBPM tasks in naturalistic settings – the so-called *age-paradox* of PM (Uttl, Graf, Miller & Tuokko, 2001; Bastin & Meulemans, 2002; Aberle et al., 2010; Bailey et al., 2010; Schnitzspahn et al., 2009; Schnitzspahn et al., 2011). What is more, the enhanced performance of older adults in naturalistic tasks (Walter & Meier, 2015) appears to be associated with better adaptation to the social demands of such tasks (Altgassen et al., 2010). A possible explanation for this paradoxical patterning is that older adults may not encode contextual information in the same way as younger adults (Simon, 1979; Neath & Surprenant, 2003), yet they appear to display more adaptive cognitive ability when PM intentions are aligned with their everyday life activities, for example regular use of medication (Will et al., 2009; Penningroth & Scott, 2013; Islam, 2014). Another explanation is that older adults are more motivated to complete tasks that mimic everyday life (Rendell & Thomson, 1999; Gonen-Yaacovi & Burgess, 2012), but this may be heavily dependent on the type and quantity of cues available during the retrieval context (Anderson & Craik,

2000; Penningroth & Scott, 2013). Furthermore, older adults are said to rely more on internal cues, whereas younger adults are more attentive to external PM cues. PM errors or failures occur when these cognitive processes fail to support the retrieval of the PM action and its related retrospective content. Accordingly, the *multiprocess framework* of PM suggests that “people use multiple approaches for solving the problem of retrieving an intention”, which may be inherently more automatic or strategic, depending on the nature of the task, cues and the individual doing the task (McDaniel & Einstein, 2000, p. 127; Smith & Bayen, 2004; Rummell, Boywitt & Meier, 2011). Thus, PM performance, across the lifespan, is likely to be the result of the interplay between a network of mechanisms underlying memory and EF processes, rather than a single process in itself (Hicks et al., 2005; Gonneaud et al., 2014). Moreover, PM performance is likely to include the combined processing of bottom-up perceptual information, and top-down strategic monitoring (Gilbert, Hadjipavlou & Raoelison, 2013).

The picture is further complicated by several other factors that have been shown to influence PM success, across the lifespan (Kretschmer et al., 2014). For instance, PM task importance (Walter & Meier, 2016), task regularity, cue focality and vigilance to the PM action (Rose et al., 2010; Ihle et al., 2013) are all factors that influence successful PM completion. Furthermore, the introduction of breaks or task-switching (Finstad, Bink, McDaniel & Einstein, 2006), and the complexity of the ongoing task (d’Ydewalle et al., 2001) and its cognitive load, in turn, are associated with increased demands on executive resources (Kretschmer et al., 2014). Therefore, if the PM task has low importance or the target cue has low salience, then more effortful and strategic monitoring is like to be more beneficial than automatic processes (Smith & Bayen, 2004). These factors appear to be of particular importance in older age (Jacoby, 1999; Rajah & D’Esposito, 2005; Smith et al., 2015). Given that attentional resources and strategic planning skills are sensitive to age-related declines (Salthouse, 1991; Anderson & Craik, 2017), increased demands on these cognitive resources are likely to result in greater costs (e.g. slowed response times or missed responses) to either the PM or ongoing tasks, or both. These costs are thought to be a consequence of impaired strategic planning and “dividing attention” between multiple tasks that draw on the cognitive processes (McDaniel & Einstein, 2000, p. 142; and see Einstein et al., 1995; Hicks et al., 2005; Smith et al., 2015).

What is more, PM has social importance since memory slips or errors could lead to social embarrassment (Brandimonte et al., 1996; Baddeley, 1997; Altgassen et al., 2010), but ageing-effects and factors such as the use of reminders, task importance, and everyday stress may mediate PM performance in older age (Ihle, Schnitzspahn, Rendell, Luong & Kliegel, 2011). Thus, personally-relevant factors such as goals, pro-social motivations or reward incentives all contribute to age-related performance differences (Altgassen et al., 2010; Brandimonte et al., 2012; Penningroth & Scott, 2013; Brandimonte & Ferrante, 2015). Although the number of studies exploring motivational aspects of PM is somewhat limited, the consensus thus far is that *pro-social* motivation appears to be of particular importance in PM performance in older age, autonomy and social functioning (Brandimonte & Ferrante, 2008; 2015; Bastin & Meulemans, 2002; Altgassen et al., 2010; Kretschmer et al., 2014). The theoretical models set out below have attempted to describe the role of motivations – whether self-directed or instructed (Islam, 2014). These positions are discussed next in the context of PM and ageing.

Motivations in PM: personal vs. pro-social ‘altruism’ (self-relevant vs. other-relevant)

Whilst PM errors in themselves can have social consequences, many studies of PM are by their nature socially motivated. Experimental tasks often involve remembering to do something that is not personally relevant to the participant but is instead for the benefit of the researcher, whether inferred or explicitly stated. Consequently, still little is understood about the cognitive processes that underlie the interaction between social motivation and prospective memory processes (Albrecht et al., 2014). Recent theoretical attempts to establish cognitive associations have given rise to variants of motivational-cognitive models: the *value-added intentions* framework (Cook, Rummel & Dummel, 2015); the *goal-based motivational-cognitive* model (Penningroth and Scott (2007; 2013); and an adapted *motivational-cognitive* model (Islam, 2014), have all endeavoured to explain the cognitive processes associated with motivation in PM.

The *value-added intentions* framework (Cook, Rummel & Dummel, 2015) posits that a person’s overarching motivation to complete the PM action is determined by *self-relevant* gains. Thus, if a person’s primary motivation is self-gain, then larger (*value-added*) monetary incentives – which are

contingent on gains or losses – serve to improve PM performance, without costs (performance impairments) to the ongoing task performance. Similarly, Penningroth & Scott (2007; 2013) argued that PM success is improved by goal-oriented intentions. Specifically, according to the *goal-based motivational-cognitive* view, PM intentions are purportedly better fulfilled when they are in line with an individual's personal goals, than PM intentions which are unrelated to goals. The authors extended this notion to incorporate goal *importance* (Penningroth & Scott, 2013), whether self-directed or externally driven (e.g. assigned by another person; Islam, 2014), as relevant factors.

There is some evidence that motivational factors enhance PM performance without affecting the ongoing task (Walter & Meier, 2017). However, very few studies have explicitly investigated the role of social motivation (pro-social behaviours) and their effect on PM (Altgassen et al., 2010; Brandimonte, Ferrante, Bianco & Villani, 2010; Cook, Rummel & Dummel, 2015; Brandimonte & Ferrante, 2015) and ageing (e.g. Altgassen et al., 2010; Niedźwieńska & Barzykowski, 2012). Altgassen and colleagues reported that PM task success was improved for older (but not younger) adults, when specific goal-based intentions were formed (Altgassen et al., 2010; Altgassen, Ktreshmer & Schnitzspahn, 2017; and see Zimmermann & Meier, 2010; McFarland & Glisky, 2011). A possible explanation for the positive age effects of pro-social motivation on PM performance, is that older adults may have a stronger sense of social norms and courtesies than younger adults (Maylor, 1993). Thus, if older adults attend more to “social [and] emotionally meaningful interactions” than younger adults (Altgassen et al., 2010, p. 314), then motivations serve to enhance PM in ageing (Penningroth & Scott, 2013). However, Brandimonte and Ferrante (2015) caution that these same motivational factors may attenuate PM success, particularly when the importance of goals and task instructions are in conflict for attentional priority. What is more, pro-social PM (other-relevant) that is incentivised either by monetary reward or self-image gains appear to conflict with ‘altruistic’ motives. In a series of studies, Brandimonte and colleagues demonstrated that pro-social motivations can improve PM task performance, compared to tasks that are not inherently socially motivated (Brandimonte et al., 2010; Brandimonte and Ferrante, 2008; 2015; D’Angelo, Bosco, Bianco & Brandimonte, 2012). However, these effects appear to be attenuated by the presence of low-value monetary rewards (e.g. €1) or self-image-based incentives (e.g. future public recognition of altruistic

behaviour; D'Angelo et al., 2012). In both instances, self-relevant motivations served to decrease the number of pro-social PM responses made, compared to conditions that involved no reward or high monetary rewards (e.g. €20). Further, low-value incentives, or incentives that are negatively perceived (e.g. being controlled by the outcome), in turn negatively affected the ongoing task performance. Consequently, monitoring costs resulted in slower response times and reduced response accuracy for both the PM and ongoing tasks (Brandimonte et al., 2010; Brandimonte & Ferrante, 2015). The authors suggest that the paradoxical effects of specific reward incentives on task performance may be the result of unconscious reconciling between internal motives and altruistic intentions when reward gains are involved.

In an adapted *motivational-cognitive* model, Islam (2014) reasoned that motivations are not limited to purely personal goals or altruistic motivations, but that a PM task instruction can independently form a goal (e.g. to do the task well; see Islam, 2014, p. 134). Thus, the importance of the PM intention is enhanced and, in turn, serves to facilitate memory strategies for encoding and retrieval. Thus, the inherent PM goal directs the cognitive resources associated with the ongoing task to the successful completion of the PM task. In this way, the combined “effortful and automatic processing” (Islam, 2014, p. 134; and see McDaniel & Einstein, 2000; Brandimonte & Ferrante, 2015) draw together strategic cognitive processes, to direct one’s attention and awareness to the respective internal time monitoring or external event cues, to successfully execute the PM response at the appropriate point. The associated cognitive mechanisms that are linked with PM failures, holds importance for clinical groups who may already have difficulties with selective cognitive functions, such those individuals with Parkinson’s disease, Schizophrenia, or Autism Spectrum Disorder. Next we turn to the profile of PM function that has been documents in clinical groups, to date.

Prospective memory in clinical groups

As we have seen, PM difficulties in typical ageing affect the ability to autonomously carry out everyday tasks, such as medication management. Consequently, PM is linked with poorer health-related outcomes, reduced QoL, and risk of early mortality (Evans & Mottram, 2000; Zogg et al., 2012; Woods et al., 2012). In addition to affecting typically ageing individuals, PM performance is

also reported to contribute to cognitive difficulties in a range of clinical groups, such as individuals diagnosed with anxiety and depression disorders, ADHD, Schizophrenia and ASD (Shum et al., 2004; Kvavilashvili et al., 2009; Brandimonte et al., 2011; Pirogovsky et al., 2012; Woods et al., 2012; Altgassen et al., 2013; Gonneaud et al., 2014; Kretschmer et al., 2014; Williams et al., 2014). The emerging findings from these recent studies suggest that the additional executive demands of time-based tasks may present greater difficulties for these individuals than for typically developed persons. What is more, PM-related difficulties, more generally, appear to be influential in basic functional skills for everyday living.

Woods and colleagues explored PM in relation to daily living skills and QoL in older adults with Parkinson's disease (PD) with and without dementia (mean age 71.2 years, SD 1.4) and a typically ageing (TA) comparison group (mean age 69.8 years, SD 1.3; Pirogovsky et al., 2012). The clinical groups in those studies were free of diagnosed substance use disorders and co-occurring mental health conditions (e.g. major depression, schizophrenia), although self-reported depression scores were elevated in the PD participants. What is more, PM difficulties in those studies were a significant predictor of everyday difficulties in the clinical groups. Compared to the TA group, the adults with PD displayed significantly greater difficulties in memory for intentions, self-cued PM and everyday challenges associated with self-management of medication and personal finances. The PD group also reported more everyday PM failures, increased difficulties with daily living skills, and poorer health-related QoL (see Pirogovsky et al., 2012 for methods). Crucially, memory for intentions was significantly correlated with medication management in the PD group, particularly where the PM action involved a time-based component. Further, the results from that study suggest that difficulties in self-care, such as self-management of chronic medication use, may be underpinned by executive difficulties that draw on the self-directed strategic organisation of cognitive resources (e.g. planning, attention, time monitoring). In typical ageing, older adults tend to apply more strategic time monitoring in the lead up to a time-bound PM action, although this is often at the cost of the ongoing task (Mäntylä & Carelli, 2006). In the studies just described, strategic time monitoring seemed to present greater difficulties for both PD adults (e.g. medication management) and TA adults alike (e.g. finance management), when they were engaged in ongoing tasks.

Similar patterns of PM difficulties have been consistently reported in other clinical groups, including obsessive compulsive disorder, Schizophrenia, traumatic brain injury and, recently, ASD (Gonen-Yaacovi & Burgess, 2012; and see Shum et al., 2004; Henry et al., 2004 and Landsiedel et al., 2017 for reviews). The overall picture appears to be associated with the frontal and temporal brain regions that facilitate PM intention formation and retrieval in everyday life (Gonen-Yaacovi & Burgess, 2012; and see Will et al., 2009; Woods et al., 2012). Consequently, increased cognitive difficulties in those domains and greater resulting PM failures, are linked with reduced autonomy with potentially profound consequences for health and wellbeing (Rendell & Craik, 2000; d'Ydewalle et al., 2001; Henry et al., 2004). Moreover, PM difficulties such as those just mentioned have also been linked with an individual's sense of identity in older age (Maylor, 1996; Brandimonte et al., 1996; d'Ydewalle, Bouckaert & Brunfaut, 2001; Henry, MacLeod, Phillips, & Crawford, 2004), which holds important clues to risk of age-related neurocognitive disorders (NCD's e.g. dementia; Desgranges et al., 2008; 2009; Blanco-Campal et al., 2009; Rabin et al., 2014). For instance, problems with *autonoetic* or self-referential processing (e.g. self-awareness, self-generated memory recall, and self-judgement; Kana, Klinger et al., 2017) affects one's ability to self-organise everyday events in time and space, or to self-report any forgetting associated with PM difficulties (Brandt, 2007; see Lind & Bowler, 2010, and see Bowler et al., 2011 regarding autonoetic awareness in episodic memory and future thinking in ASD). Thus, it is evident that impaired PM has far-reaching consequences on broader cognitive functioning and everyday functional ability in older age (Cockburn & Smith, 1988; Einstein & McDaniel, 1996; Maylor, 1996; d'Ydewalle, Bouckaert, & Brunfaut, 2001; Kester et al., 2002; Blanco-Campal et al., 2009).

The cognitive processes just mentioned are known to be selectively affected by ASD, across the lifespan. This patterning of difficulties in ASD is also known to persist in older autistic adults but does not appear to manifest in the same way as cognitive difficulties seen in typical ageing (Ring et al., 2015; 2016; Lever & Geurts, 2015; Happé, Mansour et al., 2016; Roestorf & Bowler, 2016; and see Chapters 4 and 5 of this thesis). However, it is unknown that specific challenges PM and its association with a broader array of cognitive functions already mentioned, may present for autistic individuals, particularly during ageing. Nor is it known what effect any PM difficulties may have on

QoL outcomes in ASD, which is known to be diminished compared to typical persons (Howlin et al., 2014; van Heijst & Geurts, 2014; Roestorf & Bowler, 2016; and see Chapters 4 and 5, this thesis).

The social motivation accounts of PM set out earlier, describe the ways in which goals, motivations and rewards (e.g. personal, altruistic, task inherent) may underpin successful PM encoding, retrieval and actions, more than PM tasks that do not include a motivational component (Cook et al., 2015; Brandimonte & Ferrante, 2015; Walter & Meier, 2017). Accordingly, pro-social and self-relevant motivations may be of importance in mediating specific age-related PM difficulties (Einstein et al., 1992; Rendell & Thomson, 1999; Altgassen et al., 2010). Arguably, the nature of many PM tasks involving pro-social interactions (e.g. with/for the experimenter) and is motivated by fulfilling goals which are not self-relevant but rather pro-social (although not necessarily altruistic i.e. socially conforming). The factors just mentioned are important considerations in the context of ageing and autism, since social communication is one of the core impairments associated with ASD. Thus, socially motivated PM may present greater challenges for autistic individuals. To date, very few studies have researched EBPM and TBPM in younger autistic adults and children (see Landsiedel et al., 2015; and see Sheppard, Bruineberg, Kretschmer-Trendowicz & Altgassen, 2018 for reviews). A summary of findings from those studies is briefly presented next. However, to my knowledge, no studies have explored PM in ageing and ASD, or the association between PM and QoL.

Prospective memory in Autism Spectrum Disorder

A literature search was carried out in 2015 and again in July 2017, for completeness. The Web of Science was searched, using the terms “autism*” and “prospective memory”, for all years from the earliest database record (1970). The search produced a list of 30 results for publications from the last 17 years (1999-2017). However, only 15 of the listed records were relevant studies of prospective memory in ASD. Those studies were published during the last eight years (2009-2017), including one review paper of PM in ASD (Landsiedel et al., 2017), one study of everyday memory in adolescents which involved a single event-based memory task that was likened to PM as well as other memory measures (Jones et al., 2011), and one theoretical paper that focused on episodic future thinking and its potential role for PM in ASD (Lind & Williams, 2012). Subsequently, one additional review paper has

been published (Sheppard et al., 2018). Of the remaining 12 papers, seven were studies of PM in children (Altgassen, Williams, Bölte & Kliegel, 2009; Rajendran et al., 2011; Brandimonte, Filippello, Coluccia, Altgassen, & Kliegel, 2011; Williams, Boucher, Lind, & Jarrold, 2013; Yi et al., 2014; Sheppard, Kvavilashvili, & Ryder, 2016), and one was a study of adolescents (Altgassen, Schmitz-Hubsch & Kliegel, 2010). Two studies explored TBPM only in ASD (Altgassen et al., 2009; Altgassen et al., 2017), and these both reported greater PM difficulties in the ASD but not the control group.

However, only four papers were experimental PM studies with autistic adults (Kretschmer, Altgassen, Rendell, & Bölte, 2014; Williams, Jarrold, Grainger, & Lind, 2014; Altgassen, & Koch, 2014; Altgassen, Koban, & Kliegel, 2012). The overall findings from those four studies point to specific cognitive difficulties in ASD that may be of relevance to PM functioning. Broadly, the findings indicate that autistic adults have more difficulties on TBPM tasks than EBPM tasks and varying difficulties with intention formation, rule adherence and higher self-reported difficulties with everyday memory than their typically developed peers (Altgassen et al., 2012; 2013; Williams et al., 2014). In addition to the small number of studies, the findings are also variable between studies (see Landsiedel et al., 2017; and see Sheppard et al., 2018 for reviews). Whilst the specific reasons for the different outcomes between those studies are unknown, factors such as self-motivation, internal self-referencing, task relevance, and demands on executive resources were factors that would likely have influenced PM performance in the autistic individuals (Walter & Meier, 2014; Kana et al., 2017).

In TBPM, for instance, PM success inherently involves *self-referencing* (i.e. without supportive external cues). As already mentioned, TBPM places greater demands on executive function, which is known to present specific challenges for autistic individuals (e.g. Bowler et al., 2004; Hill, 2004; Kana et al., 2017). Consequently, combined dual-task paradigms – testing EBPM and TBPM in the same paradigm – may lead to adverse confounding effects on task performance (Williams et al., 2014; Walter & Meier, 2015; Landsiedel et al., 2017). As previously noted, many standard PM tasks are socially demanding, since they require the participant to remember to do something for the researcher. Thus, previous studies of PM in ASD that have inherently included pro-social demands, together the methodological limitations of single-paradigm tasks may have presented further disadvantages for autistic individuals (Bowler, Gardiner & Berthollier, 2004; Williams et al., 2014). Consequently,

“atypical, compensatory strategies” (Williams et al., 2014, p. 31), by which autistic individuals employ more effortful strategic processes (e.g. continuous rehearsal), would place greater demands on working memory and executive resources; whereas, typical individuals tend to rely more on automatic processes (e.g. spontaneous retrieval in response to a particular cue; Einstein & McDaniel, 1996; Ward et al., 2005). The cognitive patterning for autistic individuals would, therefore, demonstrate the effects of increased cognitive load on one task which would, necessarily, impair performance on subsequent tasks in the same paradigm.

Bowler, Lind and colleagues have explored the metacognitive abilities of autistic individuals in episodic memory and future thinking tasks (Lind & Bowler, 2010; Crane, Lind & Bowler, 2013; Lind et al., 2014; and see Terrett et al., 2013). They found that autistic adults recalled fewer specific events (episodic memory), and that these memories were also impoverished of content, compared to a typically developed group of adults. Further, they found that difficulties in episodic generativity extended to self-directed planning of events related to future intentions and actions (Lind & Bowler, 2010; and see Terrett et al., 2013). In a separate study, Klinger and colleagues reported significant difficulties in self-referential processing in younger autistic adults. Self-referential processing entails the ability to incorporate self-awareness, self-judgment and self-memory in information processing (Bowler et al., 2011). It is also key to orienting of attentional resources (Zhao, Uono, Li, Yoshimura & Toichi, 2018). Consequently, underlying difficulties have broader impacts on “perspective-taking, language processing and self-other representation” (Kana et al., 2017, p. 116). Similar findings have also been reported in the few studies of PM in ASD (e.g. Altgassen et al., 2012; Williams et al., 2014). Reduced functional connectivity between brain networks involved in the processing of social and self-referencing information may provide a possible explanation for the cognitive and social differences associated with ASD (Kana et al., 2017; and see Lind & Bowler, 2008; 2009 for evidenced discussion of self/other processing difficulties).

Preliminary research by Desgranges and colleagues (Bensaber et al., 2012; Lecouvey et al., 2015) indicated that the retrospective component of PM may present greater difficulties for some autistic individuals. It has been proposed that difficulties in binding information in working memory arise from atypical hippocampal function that impairs the ability to form “a unified representation of

the individual features of an event” and organise these for later retrieval (Lecouvey et al., 2015, p. 1). Rendell and colleagues proposed that PM difficulties may be supported by explicit implementation intentions, which in turn support deeper encoding of information (Kretschmer, Altgassen, Rendell, & Bölte, 2014). Given the previously mentioned cognitive difficulties in ASD and the known implications of PM difficulties in typical ageing, it is important to understand how the underlying mechanisms of PM might affect autistic adults as they grow older.

At the start of the present study, no other studies had explored social motivations in EBPM and TBPM in relation to ASD and ageing. Subsequently, however, one study of TBPM in autistic adolescents (Altgassen, Sheppard, & Hendriks, 2017) published findings on the exploration of personal motivations and social motivations. The findings from that study showed enhanced PM performance for ASD and typically developed control groups in the personal motivation condition compared to the social condition, although this appeared to be related more to performance in the control group. Nevertheless, the authors also reported a trend to time monitoring costs (impaired performance) in the ongoing task, which is an indicator of difficulties in allocating limited cognitive resources between the PM and ongoing tasks (McDaniel & Einstein, 2000; Brewer et al., 2011). Evidence from the broader PM literature already mentioned showed that older TA adults struggle with PM under greater cognitive loads and (competing) EF demands. These cognitive functions are also known to present selective difficulties for autistic individuals across the lifespan (Boucher, Mayes, & Bigham, 2012; Geurts & Vissers, 2012). For instance, some of the selective cognitive difficulties that are commonly associated with ASD include planning, strategy use and cognitive flexibility, and memory related to episodic recall, temporal order and complex information (Williams et al., 2006; Boucher & Bowler, 2008; Martin, Poirier & Bowler, 2010; Crane, Pring, Jukes & Goddard, 2012; Crane, Goddard & Pring, 2014; Gaigg, Bowler, Ecker, Calvo-Merino & Murphy, 2015; Gaigg, 2015; Ring, Gaigg & Bowler, 2015; Bowler, Gaigg & Gardiner, 2015). Furthermore, difficulties associated with autobiographical memory present specific challenges with respect to self-image representation in time (e.g. Crane & Goddard, 2008; Bowler et al., 2011), which has also been demonstrated in studies involving episodic future thinking (e.g. Lind & Bowler, 2009). Therefore, it could be that autistic individuals may have greater difficulties in the time-related aspects of prospective memory, because of

difficulties with *autonoetic* awareness – one’s self-awareness, self-generated memory recall, and self-judgement (Kana, Klinger et al., 2017). Autonoetic awareness is an important feature of episodic memory (e.g. Bowler, Gardiner & Gaigg, 2007; Bowler et al., 2011). Accordingly, difficulties reflecting on one’s past or imagining one’s future (or future actions), may be reflective of more pervasive difficulties in personal reflections about one’s internal mental states (Lind & Bowler, 2009; Hurlburt, Happé, & Frith, 1994; Williams, 2010; Williams & Happé, 2009, 2010). The patterning of cognitive difficulties just described are known to persist into older age (Ring, Gaigg & Bowler, 2016; and see Lever, Werkle-Bergner, Brandmaier, Ridderinkhof & Geurts, 2015; Lever & Geurts, 2016). However, some aspects of cognitive functioning appear to present fewer difficulties for autistic adults in older age (Roestorf & Bowler, 2016; and see Bowler et al., 2009; Geurts & Vissers, 2012; Lever et al., 2015; Lever & Geurts, 2016; Powell et al., 2017; Wang et al., 2017; and refer to Chapter 5 this thesis). What is yet unknown, however, is whether PM functioning is affected in autistic adults, in the same way as it is in typical ageing. It is also unknown how PM affects QoL outcomes in autistic adults. These issues are addressed in Chapter 7, through a series of tasks that assess PM ability in ASD and TA adults, in laboratory and naturalistic contexts.

As we have seen, PM performance draws on a broad range of cognitive resources, including EF (e.g. planning, inhibition, attention monitoring and initiation), and social cognition factors (e.g. motivation and reward). These cognitive functions are known to present difficulties in ASD and are further influenced by difficulties associated with the clinical traits of ASD. Moreover, difficulties in self-awareness, temporal perception, and the ability to form and recall contextual representations, may be exacerbated by co-occurring conditions, such as depression and anxiety, presenting potentially acute difficulties for some autistic individuals (Crane & Goddard, 2008; Lind & Bowler, 2009; Martin, Poirier & Bowler, 2010; Bowler et al., 2011; Bowler, Gaigg & Gardiner, 2015). Further, difficulties with episodic memory and planning for future intentions (e.g. Lind & Bowler, 2008; 2009), which are fundamental features of prospective remembering (see Figure 6.1. Schematic of Prospective Memory Processes, p. 213), may present even greater PM challenges for autistic individuals. Moreover, problems with self-referencing and processing of social information (e.g. social motivation) may further exacerbate any PM difficulties (Bensaber et al., 2012; Kana et al., 2017; Zhao et al., 2018).

Thus, PM impairments associated with the core features of ASD and related cognitive difficulties would be expected to lead to poor everyday functioning, social isolation and even greater adverse impacts on cognitive function and QoL in older age. An understanding of prospective memory performance in older ASD may, therefore, provide important clues to potential cognitive changes in ageing and autism. What is more, studies that explore the functions just mentioned may provide insights to the the potential cognitive adaptations that occur with ageing and the mechanisms underlying cognitive ageing in ASD. To our knowledge, no studies have explored these features. The patterning of differences in PM ability in autistic individuals may provide clues to how different cognitive processes underlie prospective memory functions, compared to other forms of memory (Rendell & Craik, 2000; Einstein & McDaniel. 2000' Craik & Rose, 2012; Ball et al., 2018). Furthermore, where comparisons are made between laboratory and naturalistic PM performance, especially during the course of ageing, it is important that laboratory tasks mimic real world everyday tasks as much as possible, to establish ecological validity of those tasks (Gonen-Yaacovi & Burgess, 2012).

Challenges associated with the evaluation of naturalistic PM

The types of 'naturalistic' tasks used in the ASD PM studies already discussed, tend to be based in laboratory settings or have pro-social demands (other-relevant) that are not accounted for in analyses of performance differences. That is, the effects of pro-social demands have only been considered in one prior study of PM in ASD, already mentioned (Altgassen et al., 2017). A commonly use PM task is the Virtual Week (e.g. Henry et al., 2014; Kretschmer et al., 2014; and see Sheppard et al., 2018 for review), which has also been widely used in the TA literature (Rendell & Craik, 2000; Rose et al., 2010; Penningroth & Scott, 2013; and see Hering et al., 2014). In that task (Rendell & Craik, 2000), participants perform activities on a board game (or computer version) which is laid out from 9am to 4pm, with either time stamps (TBPM e.g. call the plumber at 2pm) or events (e.g. take medication with breakfast) as cues for PM actions. Thus, the EBPM and TBPM cues are embedded in the game as the ongoing task. The main challenge with the Virtual Week is that it is carried out in a laboratory setting and accelerates the representation of a daily routine. Consequently, for individuals

with representation difficulties, as in ASD (e.g. Bowler et al., 2011), this type of task would necessarily present methodological challenges. Furthermore, the TBPM and EBPM tasks are contained in a single paradigm, which may conflate any performance difficulties already mentioned. Another study of PM in ASD has used the Dresden Breakfast Task (Altgassen et al., 2012), which requires participants to prepare breakfast for four other (imaginary) people.

In the Dresden Breakfast Task (Altgassen et al., 2012), the task instructions set out the rules about what needs to be done first, and in what order (e.g. boil the water before making the tea). The primary challenge with that task is that, arguably, autistic adults are likely to have less experience in preparing breakfast for four other people. Moreover, the Dresden Breakfast Task arguably involves inherent social demands, as is the case in many PM tasks which require the participant to remember to do something for the researcher. For instance, in the typical ageing literature, naturalistic tasks may require participants to return a post-card (to the researcher) or to call the researcher every day for a week (e.g. Maylor, 1995). In the examples just described, the primary concerns were about the relevance of the type of task to individuals with autism in an everyday context. For instance, the degree to which autistic individuals – the majority of whom may not live independently (Howlin & Moss, 2012) or may be socially isolated – have the opportunity to engage in such tasks, and thus develop relevant experience necessary for carrying out the task itself, regardless of PM function.

The challenges just described were important considerations in designing naturalistic tasks that are appropriate for ASD participants in their content and contextual relevance, with distinct self-relevant and other-relevant parameters. The naturalistic tasks in the PM study presented in Chapter 7, set out to explore EBPM and TBPM performance differences in self-relevant vs other-relevant tasks as factors that may affect PM performance differences in ASD.

6.2 Study Aims

In Chapter 7, Study 3 sets out a series of experimental tasks that explore EBPM and TBPM in ASD, in laboratory and naturalistic settings. The work set out next provides, for the first time, information about the age-related differences in PM function in ASD compared to typically ageing adults, and the role of PM ability in QoL of autistic adults.

The present study addressed the following questions:

- (1) How does prospective memory ability differ in younger and older autistic adults? Does this pattern follow the same age-related trajectory seen in typical ageing?
- (2) Are autistic adults more or less likely to be influenced by, or have difficulties with, socially-motivated prospective memory compared to self-relevant prospective remembering?
- (3) To what extent does prospective memory (ability or difficulty) predict QoL in ASD?

Given the additional cognitive demands of TBPM and their respective challenges in ASD already described, three additional research questions were addressed:

- (4) How frequently do older and younger adults (ASD and TA) monitor the time during the TBPM task?
- (5) How accurately do older and younger participants perform the TBPM action, at pre-defined (2-minute) intervals?
- (6) What Diagnostic Group and Age Group differences are there in average difference between time monitoring and PM accuracy of responses (internal clock speed)?

The predictions about PM performance are summarised by task type below.

Laboratory tasks

- no Diagnostic Group differences in EBPM performance; but
- greater TBPM difficulties than EBPM, in both younger and older ASD adults; and
- impaired TBPM in younger ASD and older TA compared to younger TA adults; but
- no TBPM differences between older ASD and TA adults; and
- no age-related differences between younger and older ASD adults for EBPM or TBPM.

Naturalistic tasks

- no Diagnostic Group differences in ‘self-relevant’ EBPM and TBPM; but
- impaired naturalistic ‘other-relevant’ EBPM and TBPM in ASD compared to TA adults.

Prospective Memory and Quality of Life

- poorer PM performance would be associated with poorer QoL in TA adults

However, since there is no precedence for PM and QoL in ASD, it was not practical to predict any associations, but instead raised the question:

- How are EBPM and TBPM performance related to QoL and ageing in ASD compared to TA adults?

6.3 Ethics

Study 3 followed the ethical guidelines and procedure outlined in Study 1 (Chapter 4). Participant recruitment to this study is summarised in Table 2.3 (Chapter 2, p. 70). The nature of the PM study was to assess participants' performance as a reflection of how they would engage with tasks and use memory cues in everyday life. Consequently, for all experimental PM tasks, it was necessary to withhold some information about the precise reasons for the tasks. The withheld information was necessary to avoid confounding of true memory performance in both laboratory and naturalistic settings. Consequently, the reasons for the naturalistic tasks were not provided to participants at the start of the study. Further, participants were not informed about the retrospective Remember-Know memory test at the end of the laboratory tasks. There was no perceived risk to participants and a full debrief on the study aims and the nature of the tasks was provided at the end of the study.

Chapter 7: Study 3

Ageing and Prospective Memory in ASD. Evidence from event- and time-based tasks in laboratory and naturalistic settings

7.1 Method

Procedure

A challenge in the design of Study 3 was to use paradigms that provided sufficient demands on cognitive processing within the ongoing tasks, but that did not present greater challenges for ASD participants than for TA participants. Since lexical knowledge is reported to not present specific difficulties in ASD (Ullman & Pullman, 2015), a lexical decision task was used as the ongoing task for the PM laboratory studies. Following the procedural guidelines by Williams et al. (2014), EBPM and TBPM tasks were designed and administered as separate tasks to individually assess time- or event-based PM abilities in ASD. All participants completed the six separate tasks for EBPM and TBPM, which comprised two laboratory tasks and four naturalistic tasks. The two laboratory tasks (Lab-EBPM-lab; Lab-TBPM) were embedded in a computerised lexical decision paradigm (Kliegel, Martin, McDaniel & Einstein, 2001; Boywitt, Rummel & Meiser, 2015). The four naturalistic tasks (Nat-EBPM self-relevant, Nat-EBPM other-relevant; and Nat-TBPM self-relevant, Nat-TBPM other-relevant) were integrated into the participants everyday activities, with the time intervals varied for the TBPM tasks. Figure 7.1 (p. 236) presents a schematic of the six tasks and related self-report measures.

At the end of each task, participants were asked to feedback on: (i) what they were required to do in each task; (ii) whether they had followed the task instructions and remembered to perform related PM actions; and (iii) how they remembered to complete the task and what strategies were used (following the procedure set out in Maylor, 1996). In order to test the retrospective memory aspect of PM, a surprise recognition memory test was incorporated at the end of the Lab-EBPM and Lab-TBPM tasks (following the procedure by Gardiner et al., 1990). Participants also completed self-report measures of prospective and retrospective memory (PRMQ; Smith, Della Salla, Logie & Maylor, 2000; Crawford, Smith, Maylor, Della Salla & Logie, 2003), cognitive failures (CFQ; Broadbent et al., 1982) and time perception (ZTPI; Zimbardo & Boyd, 1999). Correlation analyses explored the

associations between self-reported everyday memory difficulties (on the CFQ and PRMQ) and observed difficulties on the PM tasks. Finally, the self-report and behavioural measures of PM difficulties were correlated with QoL measures captured in Study 2 (Chapter 5), to explore the association between PM and QoL in ASD.

7.2 Measures

Self-report measures of PM difficulties

Three standardised self-report questionnaires (Table 7.4a, p. 258) were administered after completion of the laboratory and naturalistic tasks, to explore the association between self-reported cognitive failures or cognitive strategies and EBPM and TBPM difficulties in both laboratory and naturalistic settings (see Maylor, 1995; Williams et al., 2014; Woods et al., 2015). The selected measures have been widely used in PM studies with older adults, and more recently in the emerging studies of PM in ASD. Their respective relevance and psychometric properties are detailed next.

(i) Everyday Prospective Memory failures

Prospective and Retrospective Memory Questionnaire (PRMQ; Smith et al., 2000; Crawford et al., 2003; Crawford et al., 2006)

The PRMQ is a 16-item self-rated questionnaire that reflects everyday memory slips and errors related to future planned intentions (8 items) and past memory content (8 items; see Sheppard et al., 2018 for review). The questionnaire is appropriate for use across adulthood, from ages 18-93 (Crawford et al., 2006). Items are self-rated for the frequency of which difficulties are experienced on an average day (scale 1 (never) to 5 (very often)). Total and index scores are calculated for prospective (PRMQ-PM) and retrospective (PRMQ-RM) memory components (range 0-100; normative range 17-94; see Smith et al., 2000 for the original paper). High reliabilities are reported for Total (.89 – .92), PM index (.84 – .87) and RM index scores (.80 – .83; see CI ratings in Crawford et al., 2003; 2006). Regarding, construct validity, the index scores are also reported as standardised *T* scores, for which *T*-scores and *True*-scores are derived for PRMQ Total, PRMQ-PM and PRMQ-RM indices (see Table 7.4b, p. 260), and where lower scores indicate poorer everyday memory (using the method set

out by Crawford et al., 2003 in Table 2, p. 266 and Table 3, p. 267 of their paper). Additionally, outcome scores are measured against a three-factor model of prospective and retrospective memory difficulties. These factors are: (i) general memory; (ii) short-term and long-term memory; (iii) self-cued and environmentally-cued memory (Smith et al., 2000). Finally, ecological validity of the PRMQ has been demonstrated in differentiating performance difficulties in the PM and RM components of everyday prospective remembering (Kliegel & Jäger, 2006).

(ii) Everyday Cognitive failures

Cognitive failure Questionnaire (CFQ; Broadbent et al., 1982; Wallace et al., 2002)

The CFQ is a 25-item self-report questionnaire, designed to assess specific cognitive difficulties in everyday memory “slips” and lapses. The primary factors of everyday cognitive difficulties are related to ‘forgetfulness (memory)’, ‘distractedness (distractibility)’ and ‘false triggering (blunders)’ – the three primary factors identified (Broadbent et al., 1982; Wallace et al., 2002). Subsequently a fourth factor has been identified – ‘memory for names’ (Wallace et al., 2002). The CFQ has high reliability overall (.91), as well as for each factor (‘forgetfulness / memory’, .86; ‘distractedness / distractibility’, .84; ‘false triggering / blunders’, .82; ‘memory for names’, .76”.

Although the CFQ is an older version self-report measure, it is still widely used in gerontological studies to assess age-related cognitive difficulties. Everyday memory slips and lapses, including forgetfulness, distractedness and false triggering indicate the failure to execute an intended action because of “interrupted processing of sequences of cognitive and motor actions” (Norman, 1981; Rast et al., 2008). Individual question items related to everyday memory failures are scored on a Likert-type scale from 0 (never) to 5 (very often) resulting in total scores (range 0-75), with higher scores indicating greater self-reported everyday cognitive failures associated with Forgetfulness (range 0-24), Distractibility (range 0-24) and False Triggering (range 0-24; Rast et al., 2009). McDonald et al., (1995) suggested that lay awareness of ageing facilitates an implicit expectation of decline with older age, which may overshadow (bias) self-reports towards higher ratings of everyday difficulties. However, self-report ratings may not, necessarily concur with independent observer-rated assessments (Rast et al., 2009; Cook, Ball & Brewer, 2014). Nevertheless, the CFQ has been reliably used to assess

memory difficulties that affect everyday functioning and social participation across various domains (Wallace et al., 2002; Chan et al., 2008; Rast et al., 2008; Bridger, Johnsen & Brasher, 2013).

(iii) Time perception difficulties

Zimbardo Time Perception Index (ZTPI; Zimbardo & Boyd, 1999)

The ZTPI is a measure of a person's ability to hold temporal representations of present, past and future. The measure has been standardised in several samples, ranging from age 16-62 years. The outcome of interest in the present study was whether participants' scores for 'future' time perception, on this measure, were related to the experimental PM task success, errors, or response inhibition of irrelevant information (avoidance of commission errors; Kestrel et al., 2002; Boywitt et al., 2015). The ZTPI is a 56-item questionnaire, self-rated on a 5-point Likert-type scale; individuals are asked to rate 'how characteristic' each item is of oneself, with responses ranging from 1 (very uncharacteristic) to 5 (very characteristic). The resulting scores for each item are averaged to calculate Total time perception score (range 1-5) and five index scores (range 1-5) with high reliability: Past Negative (.82); Past Positive (.80); Present Hedonistic (.79); Present-Fatalistic (.74), and Future (.77). Outcome scores represent stability or disintegrated time perception (temporal representation) across past, present and future. A theoretical discussion of time perspective as a construct, details of the ZTPI design and scale construction is described in the original paper (see Zimbardo & Boyd, 1999).

As explained in Chapter 6, time perception plays an important role in everyday functioning and processing of information, since it helps an individual to make sense of past and present experiences (and their own place in time in those experiences; Zimbardo & Boyd, 1999; Perner, 1991; 2001; Bowler et al., 2011). Accordingly, through the encoding, storage, organisation and retrieval of information, one is able to relate this information (in time) to current behaviours or future goals and expectations. The psychological perspective of time has been linked with positive or negative outcome behaviours associated with factors such as risk taking, academic achievement and mental health (Zimbardo & Boyd, 1999). The ZTPI provides a measure of the individual's difficulties with the perception of past, present and future time and their relative impulsiveness (risk-taking) in a given situation. Thus, the ZTPI was designed to capture an individual's sense of control over these temporal

dimensions (i.e. fatalistic, future oriented, present or past), and the degree to which these temporal perspectives influence the individual's view of past situations and their decision-making, self-regulation of behaviours and actions in response to a given current situation or future objectives (Zimbardo & Boyd, 1999). In this study, of particular interest was whether participants' scores on the ZTPI would reflect their individual impulsive behaviours and how these might be related to PM success, errors, or response inhibition of irrelevant information (i.e. avoidance of commission errors; Kestrel et al., 2002; Boywitt et al., 2015).

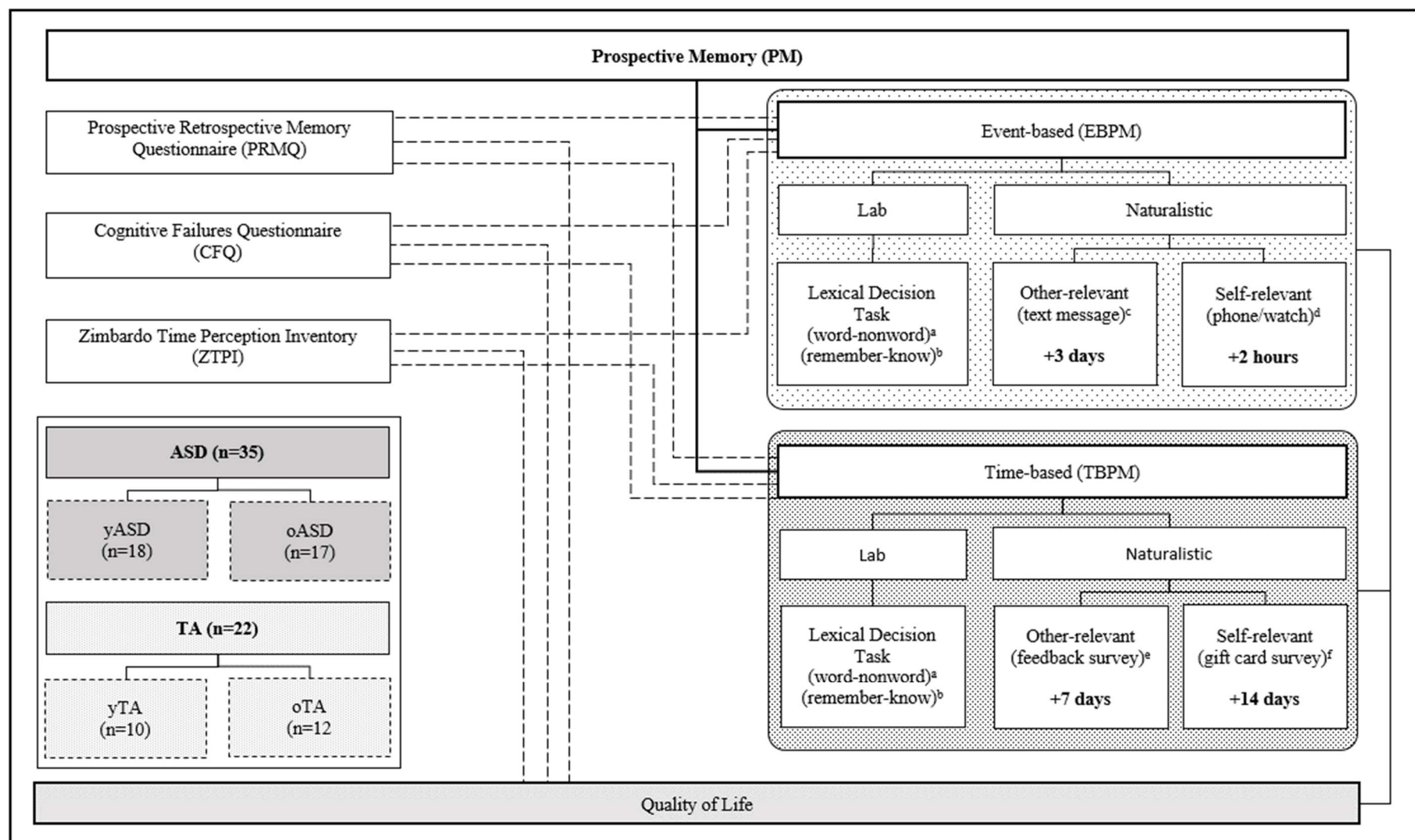


Figure 7.1. Prospective Memory study design in laboratory and naturalistic contexts.

Naturalistic tasks

The main challenge for measuring naturalistic PM performance was to design tasks that can adequately fit into the natural behaviours of autistic individuals. The aim of the naturalistic tasks was to capture PM ability when motivated by relevance to the participant (self-relevant), or to the researcher (other-relevant). Accordingly, the naturalistic tasks were designed to be carried out during the participants' everyday lives or during the course of other activities and were, as far as possible, equated for consistency (between participants) of the ongoing task.

The naturalistic EBPM and TBPM tasks were administered by text message, email and survey data, since most routine correspondence with research participants was via digital means rather than telephone. Accordingly, email correspondence was used to confirm appointments, provide information about the study, and provide instructions for the TBPM tasks. Given that these methods of communication had been used throughout the programme of work, they were deemed appropriate for all participants. An online survey method was used to collect data for the TBPM naturalistic tasks, which facilitated validation of individual participant responses against date and timestamps. Thus, whether the survey was accessed and completed (or abandoned), and the individual's time spent on each task. The outcome measures for all naturalistic tasks were responses made on time (accuracy; hits), commission errors (premature or late responses; false alarms), or omission errors (forgetting to do the task; misses).

In the naturalistic EBPM *other-relevant* task, participants were contacted by text message three days before their next appointment. They were instructed to contact the researcher (by text message), upon leaving the house to start their journey on the day of the scheduled appointment, to say they were on their way. The reason provided to participants for this request, was "to ensure that the researcher would be there to meet them when they arrived". Thus, the PM task was framed as important and relevant to the researcher in that instance. The outcome measures for this task were PM hits (correct action), and PM false alarms (action at incorrect point or stage; commission errors), and PM misses (non-response; omission errors).

In the naturalistic EBPM *self-relevant* task, participants were asked at the start of their appointment to securely store all personal items (i.e. mobile phone, watch) in a locked safety box out

of view, for which only the researcher had the key. Participants were instructed to remember to ask for their belongings before they leave. Thus, the PM action for that task was frame as important and personally relevant to the participant. To reduce anxiety about handing over their personal belongings, and thus reduce potential task interference, participants were shown that the researcher held the key around her neck (which was hidden from view during the experimental tasks). The safety box practice also fulfilled a secondary objective, which was to remove any personal time-keeping devices so that the participants only made use of the clock in the Lab-TBPM task, described later in this chapter. For ethical reasons, in the case that participants had forgotten to retrieve their belongings, these were returned before they left the research office. The outcome measures for the EBPM *self-relevant* task were PM hits (accuracy; correct action), and PM false alarms (commission errors; action at incorrect point or stage – e.g. asking for belongings during the experimental tasks), and PM misses (omission errors; non-response). At the point of retrieval, or departure in the case of forgetting, participants were asked how they had remembered (or why they had forgotten e.g. distracted, or just forgot). This procedure is described after the self-reported measures.

In the naturalistic TBPM tasks, instructions were sent to the participants by email (see Appendix 4, Figures 7.10a to 7.10e, pp. 372-377). At the end of the final research appointment, participants were told that they would soon receive an email requesting some additional information, which was related to the overall programme of work. No hints or clues were given about any of the tasks. Email instructions for the TBPM *other-relevant* task requesting participants to complete a short online survey to provide feedback about their involvement in the research. The task was marked as important to the researcher's ongoing assessment. Participants were advised that the survey would only be available on a future specified date, which was 7 days in the future. Further, participants were instructed to remember to include a unique code provided in their email, at the time of completing the survey.

Finally, in the naturalistic TBPM *self-relevant* task, participants were sent an automated email upon completion of the previous task (TBPM other-relevant). In that email, participants were thanked for their participation in the study and invited to collect a £10 voucher in 14 days' time. Once again, they needed to include a unique code which was provided in the email instructions. Finally, all

participants were asked once again how they remembered to complete the surveys. The extended time frame here, in contrast to the TBPM *other-relevant task*, was to account for the enhanced effects of reward motivation on PM performance in the TBPM *self-relevant task* (Penningroth & Scott, 2013; Brandimonte et al., 2015). If participants failed to complete the TBPM *other-relevant task*, a separate email was sent to those individuals with the same instructions as just described. Accordingly, whilst the TBPM *other-relevant task* would be marked as a PM failure (omission errors; misses), participants still had the opportunity to complete the *self-relevant task*, thus allowing for comparisons of motivations underlying performance differences. The outcome measures for both the TBPM-naturalistic tasks were PM hits (accuracy; correct action), and PM false alarms (commission errors; action at incorrect time – e.g. too early or too late), and PM misses (omission errors; non-response). An observation of whether participants correctly remembered to include their unique code was also coded but was not a key outcome measure (see Table 7.5, p. 265, for a summary of outcome measures).

Laboratory based experimental tasks

The EBPM and TBPM were both embedded in computerised lexical decision paradigms as the ongoing tasks (Walter & Meier, 2012; Williams et al., 2014). In general, lexical decision tasks offer variation in task difficulty in that words are more easily recognisable (e.g. money), and therefore response times are generally quicker, than for nonwords (e.g. muntty) which require more processing to distinguish the item from a real word (Meyer & Schvaneveldt, 1971; Gardiner et al., 1990). Despite inherent problems with lexical processing in older age (e.g. speed of processing; Rabbit, 1993; Salthouse, 1996; 2003; Neath & Surprenant, 2003; and see Kester, Benjamin, Castel & Craik, 2002), lexical decision tasks are widely used in PM studies and were used in the present study.

Here, the words comprised letter strings (items) that formed standard recognisable words that varied in length (4-7 characters), number of syllables (1-4), and familiarity (low-high). The nonwords mimicked those rules just described, to give the appearance of words, in that they were pronounceable and had a similar form in their construction of consonants and vowels. Each lexical decision task involved making judgements for 120 words and 120 nonwords. The lists contained equal numbers of

1, 2, 3 syllable words and nonwords, and equal numbers of items containing 4, 5, 6 or 7 letters (word length), which made up words/nonwords of one-syllable (for 4 letter items), or two- to four-syllables (for 5, 6, and 7 letter items). Words were sourced from the SUBTLEX-UK database (Duyck, Desmet, Verbeke & Brysbaert, 2004) and nonwords were sourced from the WordGen database (Van Heuven, Mandera, Keuleers & Brysbaert, 2014). An initial search produced a cleansed data file which included word frequencies for 160,022 word types with corresponding Zipf-values: (values 1-3 = low frequency words; 4-7 = high frequency words). Item length was restricted to 4-7 characters since words less than 3 characters were mainly acronyms or very low frequency or multiple consonant letter strings (e.g. zzy), which were subsequently excluded. Further, words exclusions were those containing repetitive letters, apostrophes and hyphenation (e.g. zzzz, o'clock, non-British), as well as curse and emotional salient words (e.g. anger, murder) and names (e.g. Adam, Mike). The resulting list comprised 3,694 words of 4-7 characters and 1-3 syllables in length, with frequencies ranging from 4.0-7.19 (low to high). Examples of words are: ARMY, BALL, MUSEUM, STATE, FLIGHT.

However, the SUBTLEX-UK database did not produce a comparable list of nonwords. Thus, the WordGen⁹ tool was used to generate nonword items (Boywitt et al., 2015). Nonwords are generated from actual words by combining up to 7 lexical rules, such as number of letters, lexical relatedness (neighbourhood size), word frequency and others (see Rastle, Harrington & Coltheart, 2002 for full description of lexicon methods). Accordingly, nonword items from the WordGen tool comprised letter strings of vowels and consonants which were easily recognisable as nonwords, but pronounceable in one, two or three syllables of 4-7 characters, respectively. Examples of nonwords are: WABBY, MOOF.

A final list of lexical items was created, and their order randomised to produce the task lists of 240 words (120 EBPM; 120 TBPM) and 240 nonwords (120 EBPM; 120 TBPM) for the lexical decision tasks. Additionally, 120 words (60 EBPM; 60 TBPM) and 120 nonwords (60 EBPM; 60

⁹ The WordGen database incorporates properties of the CELEX and Lexique lexical databases, producing nonwords in English – Dutch, German, and French versions are also available; <http://expsy.ugent.be/wordgen.htm>; and see Boywitt et al., 2015. CELEX database: Baayen, Piepenbrock, & Gulikers (1995); and see Max Planck Institute for Psycholinguistics. <http://celex.mpi.nl/>. Lexique database: New B., Pallier C., Ferrand L., Matos R. (2001) A lexical database of contemporary French on the Internet: LEXICON, *The Psychological Year*, 101, 447-462. <http://www.lexique.org>

TBPM) formed novel items in the remember-know procedure at the end of each laboratory task. The rate of presentation (on-screen time for each item) was pre-randomised and fixed to words and nonwords, and the presentation order of items was randomised across trials and between participants.

Procedure for laboratory EBPM and TBPM tasks

The laboratory EBPM and TBPM paradigms were equated for complexity of the ongoing tasks, procedure, frequency of stimuli (presentation rates), demands of the PM task (aside from the obvious PM cue and action differences), PM target times, and the task duration. The order of EBPM and TBPM tasks was systematically counterbalanced to avoid the effects of biased performance by task type. A starting central fixation cross was set to a duration of 1000ms; the fixation cross also appeared briefly between trials (1000ms; adjusted). The words and nonwords were presented in the centre of the screen, one at a time in random order, and in a single block of 240 trials (120 words; 120 nonwords). Using a standard keyboard, participants were instructed to press 'w' for words and 'n' for nonwords (Kliegel et al., 2001; Boywitt et al., 2015). All lexical items appeared in lowercase black font (Courier New, 18 pt). The presentation rate was varied (2000 milliseconds (ms), 3000ms or 4000ms; mean 3000ms; see Figure 7.2, p. 244) to avoid participants using precise timing of stimulus intervals as an additional cue to the PM task, which would potentially bias task performance. The fixation duration between trials (inter-trial stimulus interval) was automatically adjusted between trials to ensure consistency of presentation and trial duration between participants. Thus, if a participant took slightly longer to make a response on one trial, the presentation of the inter-trial stimulus interval was reduced so that the next trial could be presented. The PM-ongoing task was 12 minutes in duration.

In the Lab-EBPM task, with the presentation of each word and nonword a small coloured box was presented simultaneously in the top left corner of the screen. The colour of the box was pseudo-randomised: blue, green, magenta, yellow, cyan, teal, lime, purple, or red (EBPM cue). For the PM action, participants were required to press '1' (PM action) when a red box was presented, before they made the 'w' or 'n' response in the ongoing task. There were seven Lab-EBPM opportunities and the PM outcome measures were hits (correct action), false alarms (action at incorrect colour), misses (non-

response), and response times in milliseconds (RT, ms; see Table 7.5, p. 265, for a summary of outcome measures).

In the Lab-TBPM task, participants were shown an on-screen clock (actual time of day) at the start of the task and were instructed to make a mental note of the time. During the task, participants could access the on-screen clock at any point, as frequently as they wished, by pressing ‘C’ to check the current clock time. The time was displayed for 1000ms before returning to the word/nonword item. As in the Lab-EBPM task, participants made word (press ‘w’) or nonword (press ‘n’) judgements and corresponding keyboard responses. The PM action was to press ‘SPACE’ at 2-minute intervals (every 120000ms; time-based PM cue) to log their time (PM action) during the ongoing task. There were six lab-TBPM opportunities across the task. The key performance measures followed previous studies of TBPM in ASD, assessing *time monitoring*, *strategy* and *accuracy*. This was expanded further to understand how the response rate related to *internal clock speed* in ASD, as measured by the rate of elapsed time for a given individual. To assess *time monitoring* and strategy, the analysis included the total frequency of responses for *clock checks* and *PM actions*, and the average time intervals between their subsequent responses (Altgassen et al., 2010; Williams et al., 2014). *Accuracy* was measured as correct TBPM responses, within prescribed window before or after target times (Williams et al., 2014). Thus, *accuracy* was determined by applying “strict and lenient” rules (Dobb & Rule, 1987; Rendell & Thomson, 1999), to evaluate responses within a range of 20 second (s) intervals before and after the target times. The mean duration of response intervals across all Lab-TBPM trials provided a measure of *internal clock speed*. In summary, Lab-TBPM response times (RT, ms) are reported for mean difference from target time, and mean interval between responses as a measure of the individual’s internal clock speed (i.e. their average estimation of a two-minute interval). The extraction of PM accuracy presented complex issues related to the Lab-TBPM targets. For clarity, Table 7.5 (p. 265) sets out the key reference terms and related performance measures for the Lab-TBPM task.

Then, directly following the last trial in each task, a new instruction was given for the recognition memory task (Gardiner et al., 1990) to assess the demands on information processing for PM performance (Kestrel et al., 2002), and the link between the successful execution of PM intentions

and the retrospective memory processes involved in incidental memory. Here, participants were shown a list of 120 words and 120 nonwords. Half of those were previously seen words (60) and nonwords (60) as *familiar* items, and half were new words (60) and nonwords (60) as *novel* items. The novel items were generated from the word and nonword lexicons described earlier, and with the same characteristics as familiar items. Participants were instructed to make judgements about (1) whether the item was presented in the task just completed (e.g. Lab-EBPM), by the question: *“Have you seen this item before? Press Y for Yes or N for No”*. If participants pressed N, then the next item was shown with the same question just stated. However, if participants pressed Y, then a second question was asked: (2) *“How sure are you that you have seen this item before? Press ‘R’ for Remember, ‘K’ for Know or ‘G’ for Guess”*. In this way, participants were required to place confidence values on their previous answers. According to Gardiner et al., (1990), the highest confidence would be in remember (R) responses where clear memory recall of the item (or event) is supported by contextual detail. Similarly, medium confidence would elicit know (K) responses as a measure of recognition memory, where the item appears familiar but without clear contextual reference. By contrast, the lowest confidence judgements would produce guess (G) responses, where participants were unsure of their recognition memory. In this task, participants were given the freedom to self-regulate their speed of responses but were instructed to apply their instinctive first response and not think too long about each item. Thus, participants were encouraged to complete the task as efficiently as possible, relying on incidental memory for previously presented items. This portion of the tasks lasted approximately 12-18 minutes (mean 15 minutes).

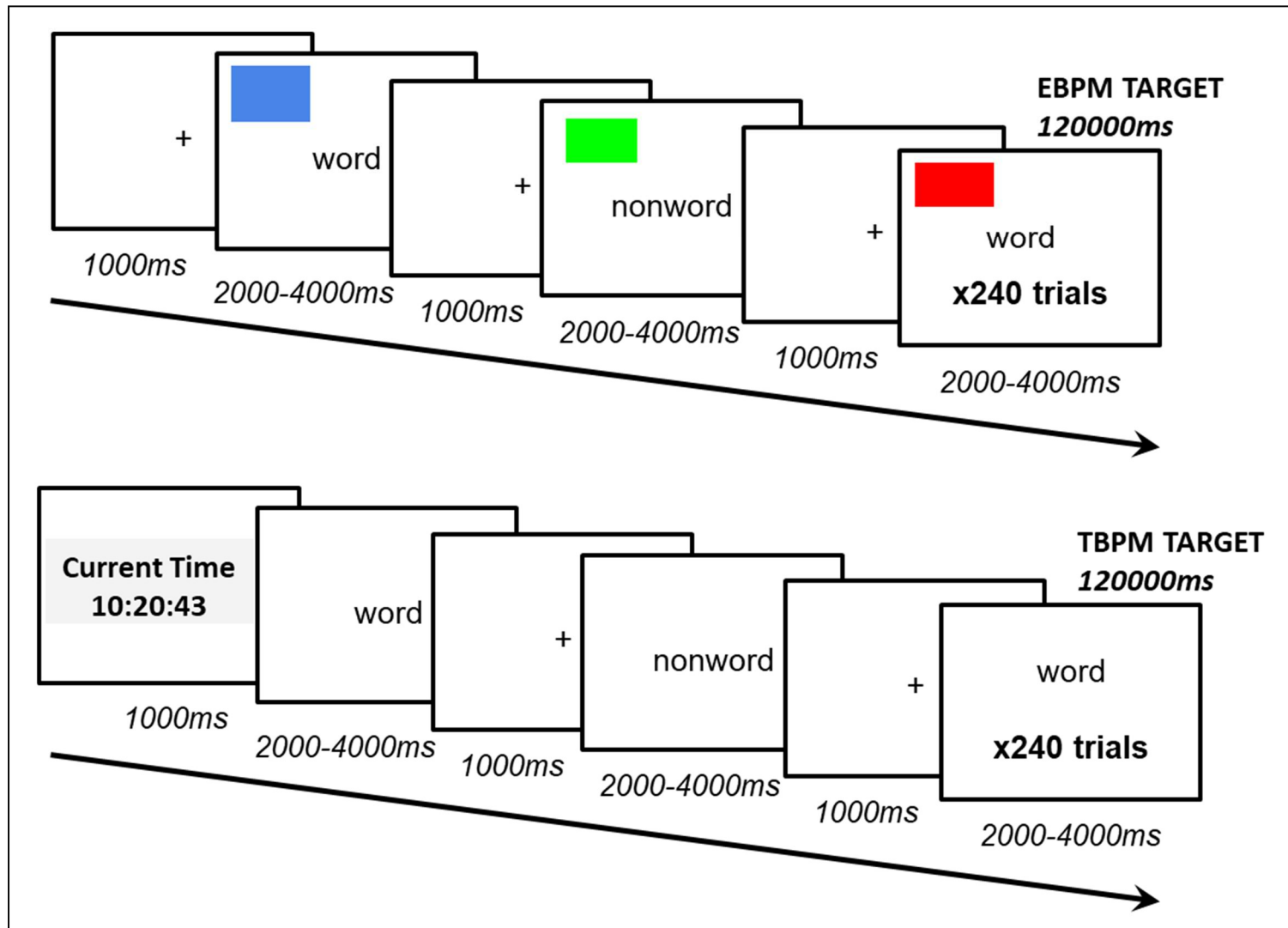


Figure 7.2. Schematic of Lab-EBPM and Lab-TBPM tasks

7.3 Analysis strategy

Multiple statistical methods were used as appropriate to the specific PM tasks, using IBM® SPSS Statistic v. 25 for the main analyses, unless otherwise specified. Partial η_p^2 and Pearson's correlation r -values are reported for effect sizes (Field, 2007, p. 308 and p. 785), where relevant, in addition to the reported p -values of statistical outcomes. Given that no a priori data are available for expected effects of PM differences in ageing and ASD compared to older TA groups, effects sizes indicate the magnitude of any group differences or their absence with practical and theoretical significance (see Crane, Lind & Bowler, 2013; Lind et al., 2014). Accordingly, η_p^2 effect sizes $\geq .01$ indicate small effects, $\geq .06$ medium effects, and $\geq .14$ large effects, whilst R^2 equivalent values are $\geq .10$, $\geq .30$, $\geq .50$ respectively (Cohen, 1973, 1988; and see Hox, 2002, p. 173-196).

A series of Analyses of Variance (ANOVA) were used to compare performance differences between Diagnostic Groups (ASD; TA) and Age Groups (younger; older). Further, chronological age was included as a continuous independent variable, in order to understand the effects of ageing in ASD on any behavioural differences that may be observed in PM ability, compared to TA adults. Respectively, the following outcomes were analysed:

Self-report measures

- A series of ANOVA were used to compare differences between Diagnostic Groups and Age Groups on the self-report measures (PRMQ, CFQ and ZTPI)
- Pearson's correlation analysis explored the associations between performance on PM experimental tasks and self-report difficulties in PM (PRMQ), everyday cognitive functioning (CFQ), and time perception (ZTPI).

Naturalistic tasks

- For both the EBPM- and TBPM-naturalistic tasks, a chi-square analysis was used to confirm *pass* (PM hits; accuracy) and *fail* (PM misses; forgetting; omission and commission) differences between Diagnostic Groups and Age Groups.
- Secondly, t-tests were used to analyse the commission errors (false alarms; i.e. too soon, or too late) between Age Groups and Diagnostic Groups.

Laboratory tasks

- To recap, EBPM performance measures were accuracy (hits), commission errors (false alarms), failures (misses), and response time (RT ms).
- TBPM performance was analysed for *time monitoring* (frequency of clock checks, and PM actions), *accuracy* (hits; absolute difference from target times), and *internal clock speed* (interval time between responses; and average length of time either side of target times). The data were first analysed using MATLAB® R2017a to code the PM actions and time monitoring (clock checking) relative to the PM target times (every 120000ms). Accordingly, time bins were created to demarcate responses into 20 second (s) bins either side of the PM target (to a maximum of +/- 60s). This method provided a clear picture of whether participants responded on target (i.e. 120000ms, 240000ms, 360000ms, 480000ms, 600000ms and 720000ms), or responded too soon (before target, -20s to -60s) or too late (after target, +20s to +60s).
- MATLAB® v. R2701a was used for data preparation to extract Clock check (time monitoring, clock speed) and PM action (accuracy, clock speed) responses within ± 20 s of the target time (i.e. 0s). Responses were allocated to incremental time bins of 20s in duration, as determined by their computerised time stamp: 0s to +/- 20s; +/-20s to +/- 40s; and +/-40s to +/- 60s of the target times. Responses were classified as ‘early’ (-ve) when made before the target times, or ‘late’ (+ve) when made after the target times.

- The interactions between TBPM performance measures were explored for: (i) *Time monitoring* and *Internal clock speed* to confirm if participants increase their *Clock check* frequency (and therefore run a faster internal clock) as the PM target approaches (Mäntylä & Carelli, 2006); and (ii) *Time monitoring* and *Accuracy*, to establish whether that (potential) increase in *Clock check* frequency, is related to improved PM *accuracy* (i.e. smaller absolute difference from target times).
- Finally, JASP v.0.9.0.1 was used post-hoc to calculate Bayes factors, to establish the magnitude of the effects being true and to avoid Type II error.

PM success were assessed according to ‘strict’ and ‘lenient’ criteria (Dobbs & Rule, 1987), following the practice of previous literature. As inferred, the premise here is that for PM actions to be considered, the PM response needs to be either strictly aligned to the target (strict, e.g. within a specific timeframe, or exactly as specified in the instructions), or alternately more lenient criteria are accepted (e.g. whether the participant attempts a PM response at all). In the case of the latter, the present study explores all PM actions for the EBPM and TBPM tasks. However, the strict criteria vary somewhat more between EBPM and TBPM tasks. In both cases, strict (adjusted) criteria consider the PM responses made within the target window, adjusted for false alarms or PM responses not related to the target.

Further, for TBPM additional factors are considered. The ‘strict’ coding approach considers accuracy (Hits) to be PM actions made within 20 seconds (s) of the target times (T) i.e. every 120000ms. Thus, the data reported for the outcome *PM hits (strict unadjusted)* are the proportion of correct PM actions for $T \pm 20s$. Additionally, *PM hits (strict adj.)* refers to the proportion of correct (adjusted) PM actions for $T \pm 20s$, excluding the proportion of inaccurate PM actions made $\pm 21s$ to $\pm 60s$ of the target times. Responses made within the $\pm 21-60s$ time bands are considered commission errors under the ‘strict’ coding approach. There is 0.33 chance of success if responding randomly. Negative scores here indicate more random responses, and positive scores indicate more strategic responses, whilst a zero-value indicated performance at chance. Whereas, the ‘lenient’ approach credits the individual for all PM actions made within $T \pm 60s$, recognising that the individual

remembered the content of the task, but with flexibility on the specific timing of responses, reported as *PM hits (lenient)*. These findings are set out in Table 7.6 (p. 273).

Overall, the analyses aimed to address these questions and explore the possible (mechanisms underlying) performance differences, through related sub-questions:

- (a) Are ASD adults more or less accurate in their PM actions and estimations of 2-minute intervals (on-target time) than TA adults?
- (b) Regarding age-related effects on PM performance, does the ASD group show a similar or different PM ability (e.g. steeper difficulties or fewer difficulties) compared to the TA group?
- (c) What is the relation between PM and QoL in ASD and TA groups?

Recognition memory test (remember-know)

A multivariate ANOVA compared the remember (R), know (K) and guess (G) responses as a measure of recognition memory differences between Diagnostic Groups and Age Groups.

Quality of life

Finally, the impact of PM difficulties on QoL in ASD and ageing were explored through Pearson's correlation analysis. Here, PM accuracy (EBPM and TBPM all tasks) was correlated with the self-report QoL measure of psychological, physical, social and environmental domains (WHOQOL-BREF, WHO, 2002). The QoL data were those obtained in the Time 2 longitudinal study (Chapter 5).

7.4 Results

Background data and sample characteristics

The participants involved in Study 3 were a sub-group of 57 individuals who took part in the T1 cross-sectional (Chapter 4) and T2 longitudinal assessments (Chapter 5). Background measures of chronological age, autistic traits, general Intellectual ability, and QoL were analysed from the T2 data (Chapter 4). Educational attainment and EF data were included from T1 data (Chapter 4). The EF data reported here relate to strategy and planning skills, cognitive flexibility, episodic memory & learning, attention for visual information processing, and reaction time (refer to Chapter 4, pp. 105-112 for EF outcome measures).

Participants were aged 23-80 years of age (ASD $n=35$, men age 47.34 years, SD 14.13; TA $n=22$, mean age 52.64 years, SD 16.93; see Table 7.1, p. 250). Despite the small number of females in ASD and TA groups, an exploratory analysis between Age Groups within Diagnostic Groups, confirmed that there were no gender effects on any of the measures just mentioned (all $F(1,55) \leq 3.68$, all $p \geq .07$, largest $\eta_p^2 = .120$).

Regarding EF, there were no Diagnostic Group differences in Strategy ($F(1,47) = <1.0$, $p > .05$, $\eta_p^2 .002$) or Planning skills ($F(1,45) <1.0$, $p > .05$, $\eta_p^2 <.001$). Nor were there any differences in Cognitive flexibility related to set-shifting ($F(1,52) = <1.0$, $p > .05$, $\eta_p^2 .008$), but fewer perseverative errors in the ASD group (mean 7.27, SD 9.28) than in the TA group (mean 13.38, SD 11.41; $F(1,52) = 4.64$, $p = .036$, $\eta_p^2 .082$). Furthermore, there was comparable performance between Diagnostic Groups in speed-accuracy trade-offs measured by Sustained visual attention ($F(1,52) = <1.0$, $p > .05$, $\eta_p^2 .015$), and Reaction Time ($F(1,46) = <1.0$, $p > .05$, $\eta_p^2 <.001$).

Regarding QoL, there were no age-related differences on QoL domains within each Diagnostic Group, except for Social-QoL which was significantly better for oASD compared to yASD, but significantly poorer for oTA compared to yTA adults (Table 7.1, p. 250). Nevertheless, following the pattern reported in Chapters 4 and 5, there were significant differences between Diagnostic Groups across all QoL domains (Overall, Health, Physical, Psychological, Social, Environmental), which was driven by poorer QoL for ASD compared to TA adults (all $F(1,42) \geq 4.17$, all $p \leq .05$, all $\eta_p^2 \leq .090$).

Table 7.1. Sample characteristics and Diagnostic Group matching for PM tasks

Measures	ASD		Statistics			TA		Statistics		
	yASD (n=18)	oASD (n=17)	ANOVA	sig.	η_p^2	yTA (n=10)	oTA (n=12)	ANOVA	sig.	η_p^2
Age (years)	35.47 (7.35)	59.96 (6.23)	110.14	<.001	.803	37.66 (8.06)	66.25 (9.30)	25.63	<.001	.681
Gender (m:f)	15:3	13:4	<1.0	<i>n.s.</i>	.007	6:4	5:6	<1.0	<i>n.s.</i>	.021
Education (years)	15.44 (2.66)	15.00 (2.92)	<1.0	<i>n.s.</i>	<.001	15.20 (1.81)	13.27 (2.83)	2.27	<i>n.s.</i>	.159
<i>Autistic traits^a</i>										
SRS-Total	71.47 (8.70)	74.79 (12.41)	<1.0	<i>n.s.</i>	.021	47.25 (5.97)	45.25 (3.69)	1.11	<i>n.s.</i>	.085
SRS-SCI	69.76 (8.58)	74.21 (12.50)	1.09	<i>n.s.</i>	.039	47.38 (5.78)	45.75 (3.99)	<1.0	<i>n.s.</i>	.072
SRS-RRB	75.24 (10.67)	73.29 (10.89)	<1.0	<i>n.s.</i>	.010	48.00 (5.53)	44.50 (4.75)	2.35	<i>n.s.</i>	.164
<i>General intellectual ability^b</i>										
FSIQ	111.29 (18.84)	116.94 (15.17)	2.36	<i>n.s.</i>	.080	115.75 (9.63)	110.11 (12.49)	1.67	<i>n.s.</i>	.122
VCI	111.29 (14.58)	116.47 (12.38)	1.64	<i>n.s.</i>	.057	114.50 (9.55)	113.44 (9.90)	<1.0	<i>n.s.</i>	.009
PRI	110.06 (19.37)	114.71 (15.21)	2.42	<i>n.s.</i>	.082	115.50 (9.13)	107.44 (13.87)	3.39	<i>n.s.</i>	.220
WMI	111.29 (19.08)	115.47 (16.07)	1.43	<i>n.s.</i>	.050	111.75 (13.54)	103.11 (14.67)	2.75	<i>n.s.</i>	.186
PSI	101.82 (21.46)	105.35 (12.14)	1.07	<i>n.s.</i>	.038	106.75 (13.92)	103.00 (14.42)	<1.0	<i>n.s.</i>	.018
<i>Executive function^c</i>										
Strategy	32.45 (6.16)	34.39 (4.52)	1.22	<i>n.s.</i>	.031	29.78 (6.65)	35.27 (3.82)	7.52	.013	.295
Planning	8.50 (3.22)	9.57 (2.67)	1.17	<i>n.s.</i>	.038	10.50 (1.05)	8.27 (2.33)	4.84	.044	.244
Cognitive flexibility	13.24 (12.71)	6.27 (2.76)	6.30	.036	.133	7.86 (5.63)	10.33 (9.20)	<1.0	<i>n.s.</i>	.025
Episodic memory	20.24 (4.96)	19.07 (4.22)	<1.0	<i>n.s.</i>	.017	23.80 (1.99)	19.00 (3.10)	17.42	<.001	.478
Visual Attention	.92 (.07)	.93 (.05)	<1.0	<i>n.s.</i>	<.001	.95 (.05)	.91 (.05))	2.14	<i>n.s.</i>	.112
Reaction time	8.89 (.32)	8.71 (.47)	1.55	<i>n.s.</i>	.049	8.67 (.71)	8.90 (.32)	<1.0	<i>n.s.</i>	.050
<i>Quality of Life^d</i>										
Overall	56.25 (21.41)	65.38 (19.20)	1.43	<i>n.s.</i>	.050	80.56 (16.67)	78.13 (8.87)	<1.0	<i>n.s.</i>	.009
Health	54.69 (26.17)	50.00 (25.00)	<1.0	<i>n.s.</i>	.009	77.78 (8.33)	59.38 (37.65)	2.05	<i>n.s.</i>	.120
Physical	58.75 (15.30)	58.00 (19.36)	<1.0	<i>n.s.</i>	.001	84.22 (12.88)	77.50 (20.76)	<1.0	<i>n.s.</i>	.042
Psychological	49.25 (19.70)	55.46 (12.58)	<1.0	<i>n.s.</i>	.035	76.44 (16.09)	69.75 (14.46)	<1.0	<i>n.s.</i>	.051
Social	34.81 (15.93)	54.31 (17.82)	9.66	.004	.264	82.00 (16.69)	61.00 (10.53)	9.33	.008	.383
Environmental	61.81 (16.73)	67.38 (15.39)	<1.0	<i>n.s.</i>	.031	78.56 (14.69)	77.50 (15.67)	<1.0	<i>n.s.</i>	.001

Notes: ^a Autistic traits as measured by SRS: Social Responsiveness Scale (Constantino & Gruber, 2012): data are reported for autistic traits in each Age Group and Diagnostic Group, as captured in Study 2 of longitudinal assessment (Chapter 5).

^b General intellectual functioning: as measured by Wechsler Adult Intelligence Scales (WAIS; Wechsler, 2008; see P-Appendix 2 for a detailed summary of subtests). Scores were extracted from data collected in the longitudinal follow-up study (Chapter 5) to ensure associations with IQ related to the most recent assessment prior to this study.

^cExecutive function as measured by the CANTAB®. Scores are reported for Strategy (lower is better); Planning (higher is better); Cognitive flexibility for set-shifting (lower is better) and perseverative errors (lower is better); Sustained Visual Attention for information processing (higher is better);

^d Quality of Life as measured by the short form World Health Organisation Quality of Life questionnaire (WHOQOL-BREF); Domain index scores are reported (0-100).

Bonferroni corrections were made to α .05 for multiple comparisons using, to avoid Type I error. The statistical analyses were compared without the α adjustment for multiple comparisons, to avoid Type II error, using the Least Significant Difference (LSD) method no changes to the significance of the above outcomes were observed.

Levene's test for Homogeneity of Variance confirmed that the distribution of variance was equal between Diagnostic Groups and did not violate the assumptions for ANOVA (Table 7.2). However, Kolmogorov-Smirnov and Shapiro-Wilk tests confirmed that the data were non-normally distributed, in both Diagnostic Groups (Table 7.3, p. 253).

Table 7.2. Levene's Test of Homogeneity of Variance

Outcome measure	Levene ^a Statistic, df (1,52)	Sig.
<i>Laboratory tasks</i>		
<i>EBPM</i>		
<i>Accuracy^b</i>		
PM actions (strict, adj.)	.904	.346
PM actions (lenient)	1.462	.232
<i>TBPM</i>		
<i>Accuracy</i>		
PM actions (strict)	.984	.326
PM actions (lenient)	6.565	.013
<i>Time monitoring</i>		
Clock checks	.371	.545
PM actions	.212	.647
<i>Clock speed</i>		
Clock checks	1.517	.232
PM actions	4.203	.053
<i>Naturalistic tasks</i>		
EBPM other-relevant	.061	.806
EBPM self-relevant	4.365	.042
TBPM other-relevant	1.364	.248
TBPM self-relevant	.410	.525

Notes: ^a based on mean for each outcome measure

Table 7.3. Tests of Normality

Outcome measure	Diagnostic Group	Kolmogorov-Smirnov ^a	df	Sig.	Shapiro-Wilk	df	Sig.
Laboratory tasks							
EBPM							
<i>Accuracy^b</i>							
PM actions (strict, adj.)	TA	.199	20	.036	.859	20	.008
	ASD	.224	34	.000	.699	34	.000
PM actions (lenient)	TA	.218	20	.014	.828	20	.002
	ASD	.193	34	.002	.856	34	.000
TBPM							
<i>Accuracy^b</i>							
PM actions (strict)	TA	.263	20	.001	.748	20	.000
	ASD	.201	34	.001	.811	34	.000
PM actions (lenient)	TA	.	20	.	.	20	.
	ASD	.442	34	.000	.332	34	.000
<i>Time monitoring^c</i>							
Clock checks	TA	.148	20	.200	.964	20	.632
	ASD	.092	34	.200	.952	34	.141
PM actions	TA	.234	20	.005	.862	20	.009
	ASD	.156	34	.034	.957	34	.202
<i>Clock speed^d</i>							
Clock checks	TA	.255	19	.002	.813	19	.002
	ASD	.178	31	.014	.831	31	.000
PM actions	TA	.198	19	.048	.801	19	.001
	ASD	.265	31	.000	.578	31	.000
Naturalistic tasks							
EBPM other-relevant	TA	.387	20	.000	.626	20	.000
	ASD	.399	34	.000	.617	34	.000
EBPM self-relevant	TA	.487	20	.000	.495	20	.000
	ASD	.429	34	.000	.591	34	.000
TBPM other-relevant	TA	.387	20	.000	.626	20	.000
	ASD	.338	34	.000	.638	34	.000
TBPM self-relevant	TA	.387	20	.000	.626	20	.000
	ASD	.414	34	.000	.606	34	.000

Notes: ^aLilliefors Significance Correction was automatically applied to correct the significance value for use of the sample mean and SD.

^bTBPM accuracy: strict = proportion of PM action responses made that fell within +/-0s to +/-20s of target, adjusting for PM actions made within +/-21s to +/-60s of target (lenient). Strict accuracy is adjusted for hits (PM target) minus false alarms (random, non-targeted PM actions; lenient accuracy)

^cTBPM time monitoring: frequency of *clock checks* and *PM actions*.

^dTBPM clock speed: average time between responses i.e. *clock check to clock check*, or *PM action to PM action*.

Self-reported difficulties in everyday memory

A series of ANOVA were used to compare differences between Diagnostic Groups and Age Groups on the self-report measures of difficulties in PM (PRMQ), everyday memory and cognitive functioning (CFQ), and time perception (ZTPI). Then, following the analysis of the laboratory and naturalistic tasks, Pearson's correlation analysis explored the associations between the self-reported difficulties just mentioned and performance on the laboratory and naturalistic tasks (p. 103).

(i) Everyday Prospective and Retrospective Memory failures

The main self-report data from the PRMQ (Smith et al., 2000; Crawford et al., 2003) are set out in Table 7.4a (p. 258) and Figure 7.3 (p. 257). Additional analyses for the *T*-scores and *True*-scores associated with General, Prospective and Retrospective memory components are presented in Table 7.4b (p. 260). The descriptive data suggest greater everyday PM-related difficulties (higher raw scores), in the younger and older ASD groups compared with the TA groups (Table 7.4a, p. 258). The ANOVA revealed statistically significant differences between Diagnostic Groups for General memory ($F(1,51) = 16.17, p < .001; \eta_p^2 = .241$), Prospective memory ($F(1,51) = 10.53, p < .003; \eta_p^2 = .171$) and Retrospective memory components ($F(1,51) = 17.73, p < .001; \eta_p^2 = .258$), as indicated by lower scores overall in the ASD group (see Figure 7.3, p. 257). Furthermore, the ASD group also reported significantly greater difficulties with the self-cued aspects of short-term PM ($F(1,51) = 9.07, p < .005; \eta_p^2 = .151$), long-term PM ($F(1,51) = 9.22, p < .005; \eta_p^2 = .153$), and for short-term RM ($F(1,51) = 6.34, p < .02; \eta_p^2 = .111$), and long-term RM ($F(1,51) = 7.67, p < .009; \eta_p^2 = .131$).

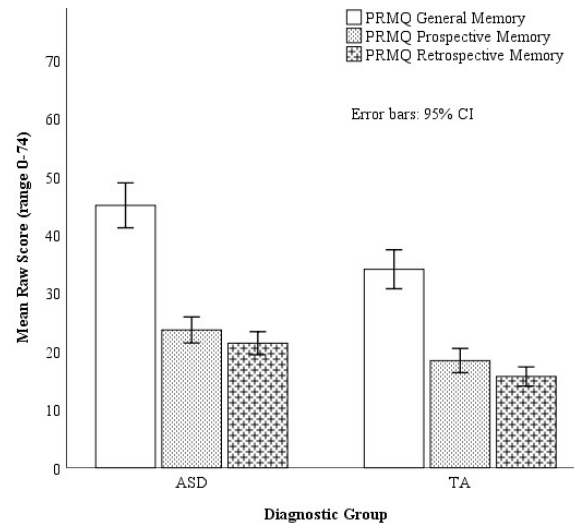
Environmentally-cued aspects were more problematic for the ASD group in short-term RM ($F(1,51) = 31.92, p < .001; \eta_p^2 = .385$), long-term RM ($F(1,51) = 4.06, p < .05; \eta_p^2 = .074$), and long-term PM ($F(1,51) = 12.55, p < .002; \eta_p^2 = .197$), but not in short-term PM ($F(1,51) = 3.67, p = .061; \eta_p^2 = .067$). The moderate to large effect sizes indicate that, overall, the ASD group experienced more pervasive everyday difficulties associated with both prospective and retrospective memory components over the short-term and long-term, largely irrespective of whether forgetting was self-generated or environmentally supported. Consequently, these difficulties may reflect underlying issues with contextual binding of information (Bowler et al., 2015; Lecouvey et al., 2015). Moreover, when

examining the T-scores and True-scores (Table 7.4b, p. 260), there were significant Diagnostic Group differences across all indices ($F_{\min}(1,51) = 10.46, p_{\max} = .002; \eta_p^2_{\min} = .170$). However, at Diagnostic Group level, there were no age effects and no interactions between Diagnostic Group and Age Group ($F_{\max}(1,51) = 2.58, p_{\min} = .115; \eta_p^2_{\max} = .048$).

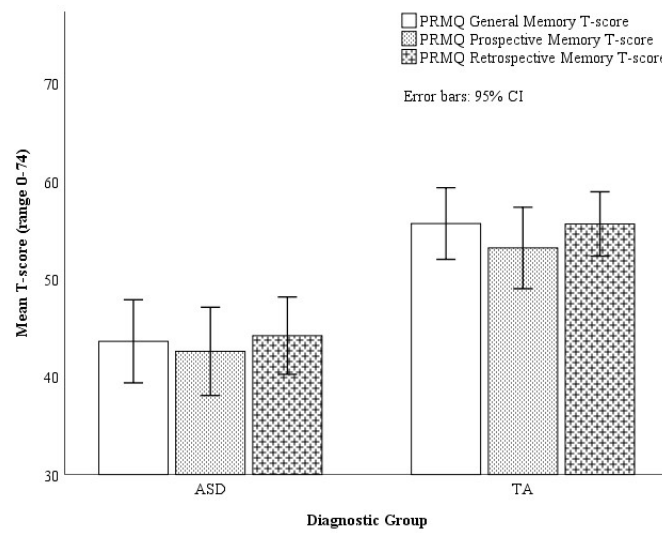
Then, planned contrasts between Age Groups within Diagnostic Groups showed no significant differences between yTA and oTA adults. Overall, the small effect sizes indicated similar level of self-report scores within the TA group. However, the large effect size for retrospective memory for short-term environmentally-cued events suggests that oTA adults reported marginally greater difficulties with this domain. Whereas, the absence of significance may be related to the smaller group sizes. In the ASD group, there were no significant differences across any of the PRMQ outcome measures (Table 7.4a, p. 258).

Finally, planned contrasts between Diagnostic Groups within Age Groups highlighted the broad PM-related difficulties for yASD compared to yTA adults, for General ($F(1,26) = 6.72, p < .02; \eta_p^2 = .205$), Prospective ($F(1,26) = 5.48, p < .03; \eta_p^2 = .174$) and Retrospective memory ($F(1,26) = 6.36, p < .02; \eta_p^2 = .196$). significant Diagnostic Group differences in the younger adults were also observed in the T-scores for True-scores for General, Prospective and Retrospective memory ($F_{\min}(1,26) = 5.41, p_{\max} = .028; \eta_p^2_{\min} = .172$). The profile of short- and long-term prospective and retrospective memory showed a slightly mixed picture. Regarding the PM short-term component, self-cued memory difficulties were significantly greater for yASD than yTA adults ($F(1,26) = 6.50, p < .02; \eta_p^2 = .200$), but environmentally-cued were not ($F(1,26) = 2.69, p = .113; \eta_p^2 = .094$). Whereas, for the PM long-term component, self-cued memory difficulties in yASD compared to yTA adults were on the edge of significance ($F(1,26) = 3.99, p = .056; \eta_p^2 = .133$), and environmentally-cued difficulties were now significantly more problematic for yASD than yTA adults ($F(1,26) = 4.91, p < .04; \eta_p^2 = .159$). There were no significant Diagnostic Group differences in RM short-term self-cued memory ($F(1,26) = 2.49, p = .127; \eta_p^2 = .087$), or in RM long-term self-cued ($F(1,26) = 1.31, p = .264; \eta_p^2 = .048$), or RM long-term environmentally-cued memory ($F(1,26) = 2.14, p = .156; \eta_p^2 = .076$). By contrast, substantially more difficulties in RM short-term environmentally-cued memory were reported by yASD compared to yTA adults ($F(1,26) = 17.94, p < .001; \eta_p^2 = .408$).

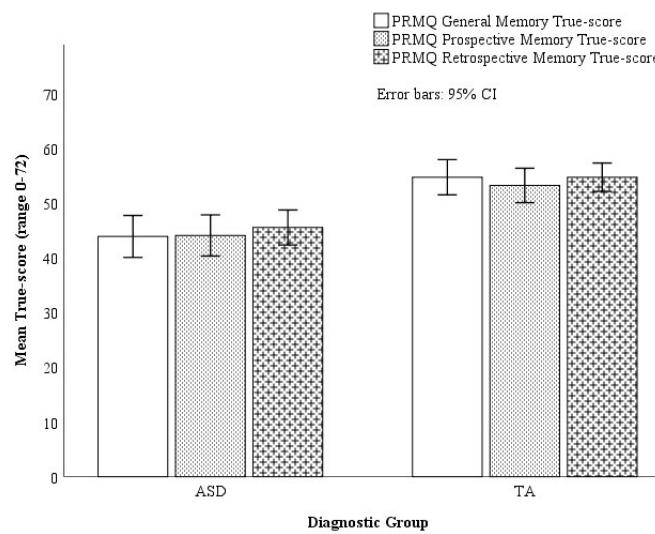
A similar patterning was observed in the older adults, as indicated by consistently higher self-reported difficulties by oASD compared to oTA adults. As with the younger group, oASD adults reported significantly more difficulties in General ($F(1,25) = 10.73, p < .004; \eta_p^2 = .300$), Prospective ($F(1,25) = 5.24, p < .04; \eta_p^2 = .173$), and Retrospective memory ($F(1,25) = 13.07, p < .002; \eta_p^2 = .343$). This picture was consistent for T-scores and True-scores ($F_{\min}(1,25) = 5.24, p_{\max} = .031; \eta_p^2_{\min} = .173$). Further, the yASD adults reported significantly greater difficulties in PM long-term self-cued ($F(1,25) = 6.02, p < .03; \eta_p^2 = .194$) and PM long-term environmentally-cued memory ($F(1,25) = 8.37, p < .009; \eta_p^2 = .251$). These difficulties extended to RM short-term environmentally-cued memory ($F(1,25) = 14.17, p < .002; \eta_p^2 = .362$), and RM long-term self-cued memory ($F(1,25) = 7.86, p < .02; \eta_p^2 = .239$). Difficulties related to RM short-term self-cued memory was on the edge of significance ($F(1,25) = 4.09, p = .054; \eta_p^2 = .140$). However, there were no significant differences between oASD and oTA adults for RM long-term environmentally-cued memory, nor were there any differences for PM short-term self-cued or environmentally-cued memory ($F_{\max}(1,25) = 2.61, p_{\min} = .119; \eta_p^2_{\max} = .095$). Overall, the very large effect sizes highlighted the magnitude of difficulties for younger and older autistic individuals compared to typically ageing persons.



(a) PRMQ Index Raw scores



(b) PRMQ Index *T*-scores



(c) PRMQ Index *True*-scores

Figure 7.3. PRMQ scores between Diagnostic Groups.

Note: Normative mean = 50 (SD = 10). Lower scores reflect greater difficulties.

Table 7.4a. Self-reported everyday difficulties with prospective and retrospective memory, cognitive failures, and time perception.

Measures	Diagnostic Groups									
	ASD (n=35)					TA (n=22)				
	yASD (n=18)	oASD (n=17)	ANOVA, <i>F</i> (1,31)	Sig.	η_p^2	yTA (n=10)	oTA (n=12)	ANOVA, <i>F</i> (1,20)	Sig.	η_p^2
Prospective & Retrospective Memory^a										
<i>PRMQ General memory</i>	45.17 (12.27)	44.93 (9.38)	<1.0	<i>n.s.</i>	.000	33.70 (8.88)	34.42 (6.65)	<1.0	<i>n.s.</i>	.002
<i>PRMQ PM^a</i>	24.39 (7.13)	22.80 (5.20)	<1.0	<i>n.s.</i>	.016	18.40 (5.06)	18.42 (4.60)	<1.0	<i>n.s.</i>	.000
<u>Short-term Memory</u>										
Self-cued	6.83 (1.92)	6.33 (1.50)	<1.0	<i>n.s.</i>	.021	5.00 (1.63)	5.50 (1.09)	<1.0	<i>n.s.</i>	.036
Externally-cued	6.00 (2.03)	5.80 (1.82)	<1.0	<i>n.s.</i>	.003	4.80 (1.48)	5.08 (1.68)	<1.0	<i>n.s.</i>	.009
<u>Long-term Memory</u>										
Self-cued	5.78 (1.83)	5.00 (1.07)	2.10	.16	.063	4.40 (1.58)	3.83 (1.40)	<1.0	<i>n.s.</i>	.038
Externally-cued	5.78 (2.01)	5.67 (1.68)	<1.0	<i>n.s.</i>	.001	4.20 (1.32)	4.00 (1.21)	<1.0	<i>n.s.</i>	.007
<i>PRMQ RM^a</i>	20.78 (6.14)	22.13 (4.94)	<1.0	<i>n.s.</i>	.015	15.30 (4.06)	16.00 (3.54)	<1.0	<i>n.s.</i>	.009
<u>Short-term Memory</u>										
Self-cued	6.11 (1.81)	6.47 (1.68)	<1.0	<i>n.s.</i>	.011	5.10 (1.20)	5.33 (1.07)	<1.0	<i>n.s.</i>	.011
Externally-cued	4.78 (1.80)	5.20 (1.86)	<1.0	<i>n.s.</i>	.014	2.30 (.48)	2.92 (1.08)	2.76	.11	.121
<u>Long-term Memory</u>										
Self-cued	4.72 (2.08)	5.87 (1.92)	2.66	.11	.079	3.90 (1.20)	3.92 (1.62)	<1.0	<i>n.s.</i>	.000
Externally-cued	5.17 (2.04)	4.60 (1.64)	<1.0	<i>n.s.</i>	.024	4.00 (2.00)	3.83 (.94)	<1.0	<i>n.s.</i>	.003
Cognitive failures^b										
CFQ Total	53.33 (17.46)	45.27 (15.38)	1.94	<i>n.s.</i>	.059	32.30 (16.08)	29.17 (12.17)	<1.0	<i>n.s.</i>	.013
Forgetfulness	18.22 (5.84)	17.27 (5.23)	<1.0	<i>n.s.</i>	.008	12.00 (5.12)	12.25 (4.75)	<1.0	<i>n.s.</i>	.001
Distractibility	19.50 (6.83)	15.67 (5.60)	3.03	.09	.089	11.40 (6.98)	8.83 (3.35)	1.28	.27	.060
False Triggering	13.50 (6.83)	10.60 (5.73)	1.70	<i>n.s.</i>	.052	7.90 (4.23)	7.58 (3.90)	<1.0	<i>n.s.</i>	.002
Time perception^c										
ZTPI Total	3.26 (.22)	3.10 (.38)	2.39	.13	.069	3.10 (.27)	3.13 (.36)	<1.0	<i>n.s.</i>	.002
Past negative	3.84 (.61)	3.34 (.79)	4.33	.045	.119	2.65 (.83)	2.61 (.73)	<1.0	<i>n.s.</i>	.001
Past positive	2.94 (.62)	2.88 (.93)	<1.0	<i>n.s.</i>	.002	3.58 (.71)	3.53 (.65)	<1.0	<i>n.s.</i>	.001
Present hedonistic	3.03 (.50)	2.78 (.71)	1.49	.23	.045	3.42 (.52)	3.39 (.57)	<1.0	<i>n.s.</i>	.001
Present fatalistic	3.04 (.54)	2.91 (.61)	<1.0	<i>n.s.</i>	.013	2.42 (.47)	2.66 (.51)	1.25	<i>n.s.</i>	.059
Future	3.45 (.54)	3.60 (.81)	<1.0	<i>n.s.</i>	.012	3.43 (.61)	3.47 (.45)	<1.0	<i>n.s.</i>	.001

Notes:

^a PRMQ: Prospective and Retrospective Memory Questionnaire (Smith et al., 2000). Raw data are reported here for PM and RM indices, and General memory (as combined Total for PM and RM). Short-term and Long-term self-cued and environmentally-cued memory difficulties are reported as raw data from specific questionnaire items (Crawford et al., 2006). Higher scores indicate more PM difficulties. See Table 7.4b for T-scores.

^b Cognitive failures as measured by the CFQ (Broadbent et al., 1982). Scores are reported as CFQ Total (range 0-100) and index scores (range 0-24): Forgetfulness, Distractibility, and False Triggering (Wallace et al., 2002; Rast et al., 2009).

^c Time perception as measured by the Zimbardo Time Perception Index (ZTPI; Zimbardo & Boyd, 1999)

Table 7.4b. PRMQ T-Scores and True-Scores for General, Prospective and Retrospective Memory indices

Measure	Diagnostic Groups								
	ASD (n=33)		Statistics			TA (n=22)		Statistics	
	yASD (n=18)	oASD (n=15)	ANOVA, <i>F</i> (1,31)	Sig.	η_p^2	yTA (n=10)	oTA (n=12)	ANOVA, <i>F</i> (1,20)	Sig. η_p^2
PRMQ indices									
General memory									
<i>T</i> -score	43.56 (13.62)	43.73 (10.15)	<1.0	<i>n.s.</i>	.000	55.10 (9.62)	55.33 (7.36)	<1.0	<i>n.s.</i> .002
<i>True</i> -score	43.89 (12.27)	43.93 (9.18)	<1.0	<i>n.s.</i>	.000	55.00 (8.39)	54.50 (6.56)	<1.0	<i>n.s.</i> .001
Prospective memory									
<i>T</i> -score	41.11 (14.50)	44.40 (14.50)	<1.0	<i>n.s.</i>	.017	53.20 (10.12)	53.17 (9.20)	<1.0	<i>n.s.</i> .000
<i>True</i> -score	42.89 (12.03)	45.53 (8.79)	<1.0	<i>n.s.</i>	.016	52.90 (8.39)	53.50 (6.14)	<1.0	<i>n.s.</i> .002
Retrospective memory									
<i>T</i> -score	45.44 (12.28)	42.73 (9.88)	<1.0	<i>n.s.</i>	.015	56.40 (8.11)	55.00 (7.08)	<1.0	<i>n.s.</i> .009
<i>True</i> -score	46.44 (9.93)	44.47 (8.00)	<1.0	<i>n.s.</i>	.012	55.40 (6.31)	54.17 (5.65)	<1.0	<i>n.s.</i> .012

Notes: The T-scores and True-scores reported here (Crawford et al., 2003, *Table 2* p. 266 and *Table 3* p. 267 of their paper) are for everyday difficulties in General memory (PRMQ Total score; *T*-range = 5-74; *True* range = 10-72), Prospective memory (PRMQ-PM score; *T*-range = 9-74; *True* range = 16-70) and Retrospective memory (PRMQ-RM score; *T*-range = 7-71; *True*-range = 16-67). Lower scores indicate more extreme difficulties. See Table 7.4a for Index scores calculated from raw scores.

(ii) Everyday Cognitive failures

Everyday cognitive failures, as measured by the CFQ (Broadbent et al., 1982), were self-reported to a greater extent by ASD adults (CFQ Total mean 49.67, SD 16.80) compared to TA adults (CFQ Total mean 30.59, SD 13.82; $F(1,51) = 18.49, p < .001; \eta_p^2 = .266$; and see Figure 7.4, p. 262). These findings replicate recent cross-sectional study of cognitive difficulties in older autistic compared to typically ageing adults (Lever & Geurts, 2015). Similar findings have also been observed in relation to PM difficulties in younger autistic adults (Cherkaoui & Gilbert, 2017). Furthermore, the Diagnostic Group differences observed here extended to self-reported difficulties in Forgetfulness (ASD mean 17.79, SD 5.50; TA mean 12.14, SD 4.80; $F(1,51) = 14.58, p < .001; \eta_p^2 = .222$), Distractibility (ASD mean 17.76, SD 6.50; TA mean 10.00, SD 5.34; $F(1,51) = 20.75, p < .001; \eta_p^2 = .289$) and False Triggering (ASD mean 12.18, SD 6.42; TA mean 7.73, SD 3.95; $F(1,51) = 7.84, p < .008; \eta_p^2 = .133$; see Figure 7.4, p. 262). Age Group effects related to Distractibility difficulties in everyday life, were on the edge of significance ($F(1,51) = 3.81, p = .056; \eta_p^2 = .070$).

Planned contrasts within Diagnostic Groups and between Age Groups showed similar mean scores in the yTA and oTA groups, across domains (Table 7.4a, p. 258). Everyday Distractibility difficulties were reported less by oTA than yTA adults, which is in line with findings from the typical aging literature (Rast et al., 2009). Further, although these differences did not reach significance in the present study (Table 7.4a, p. 258), the moderate effect size suggests that age-related differences may be masked by smaller group sizes reported here. A possible explanation the lower scores in oTA adults is that they have more available time in a typical day, commonly as a result of retirement or similar change in circumstance (Rast et al., 2009). In the ASD group a similar profile was observed in relation to not only Distractibility, but lower mean scores across domains for oASD compared to yASD adults (Table 7.4a, p. 258). However, none of those differences reached significance in the ASD group. Once again, the moderate effect sizes suggest that age-related differences may be less pronounced for oASD adults.

Planned contrasts within Age Groups and between Diagnostic Groups revealed that yASD adults reported greater difficulties than yTA adults for everyday cognitive failures (CFQ Total, $F(1,26) = 9.85, p < .005; \eta_p^2 = .275$), and across domains of Forgetfulness ($F(1,26) = 7.94, p < .01; \eta_p^2 = .234$), Distractibility ($F(1,26) = 8.91, p < .007; \eta_p^2 = .255$), and False Triggering ($F(1,26) = 5.50, p < .03; \eta_p^2 = .175$). In the older groups, oASD also reported greater difficulties than oTA adults in everyday cognitive failures (CFQ Total, $F(1,25) = 8.74, p < .008; \eta_p^2 = .259$), Forgetfulness ($F(1,25) = 6.65, p < .02; \eta_p^2 = .210$) and Distractibility ($F(1,25) = 13.82, p < .002; \eta_p^2 = .356$). However, Diagnostic Group differences in False Triggering did not reach significance ($F(1,25) = 2.42, p = .13; \eta_p^2 = .088$), which may have been masked by smaller group sizes.

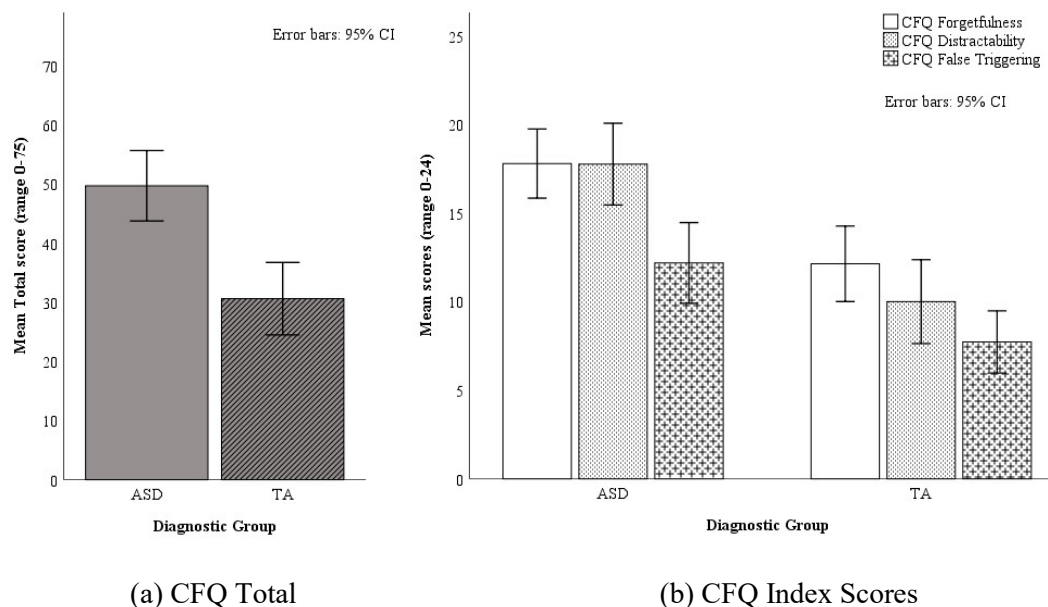


Figure 7.4. CFQ scores between Diagnostic Groups.

Notes: Scores are reported for (a) Total (range 0-75), and Index scores (range 0-24) related to Forgetfulness, Distractibility and False Triggering. Higher scores reflect greater difficulties.

(iii) Time perception difficulties

Overall, time perception did not significantly differ between Diagnostic Groups (ZTPI Total: ASD mean 3.19, SD .31; TA mean 3.12, SD .31; $F(1,52) < 1.0$, $p = .44$; $\eta_p^2 = .011$; and see Figure 7.5, below). However the Diagnostic Groups varied in their temporal representations for Past Negative (ASD mean 3.61, SD .73; TA mean 2.62, SD .76; $F(1,52) = 23.22$, $p < .001$; $\eta_p^2 = .309$), Past Positive (ASD mean 2.91, SD .77; TA mean 3.55, SD .66; $F(1,52) = 9.90$, $p < .004$; $\eta_p^2 = .160$), Present Hedonistic (ASD mean 2.91, SD .61; TA mean 3.41, SD .53; $F(1,52) = 9.86$, $p < .004$; $\eta_p^2 = .159$), and Present Fatalistic representations (ASD mean 2.98, SD .57; TA mean 2.55, SD .49; $F(1,52) = 8.44$, $p < .006$; $\eta_p^2 = .140$). However, there were no significant Diagnostic Group differences for Future representations (ASD mean 3.52, SD .68; TA mean 3.45, SD .51; $F(1,52) < 1.0$, $p = .66$; $\eta_p^2 = .004$), which was the primary outcome measure for correlation analyses against the experimental PM tasks.

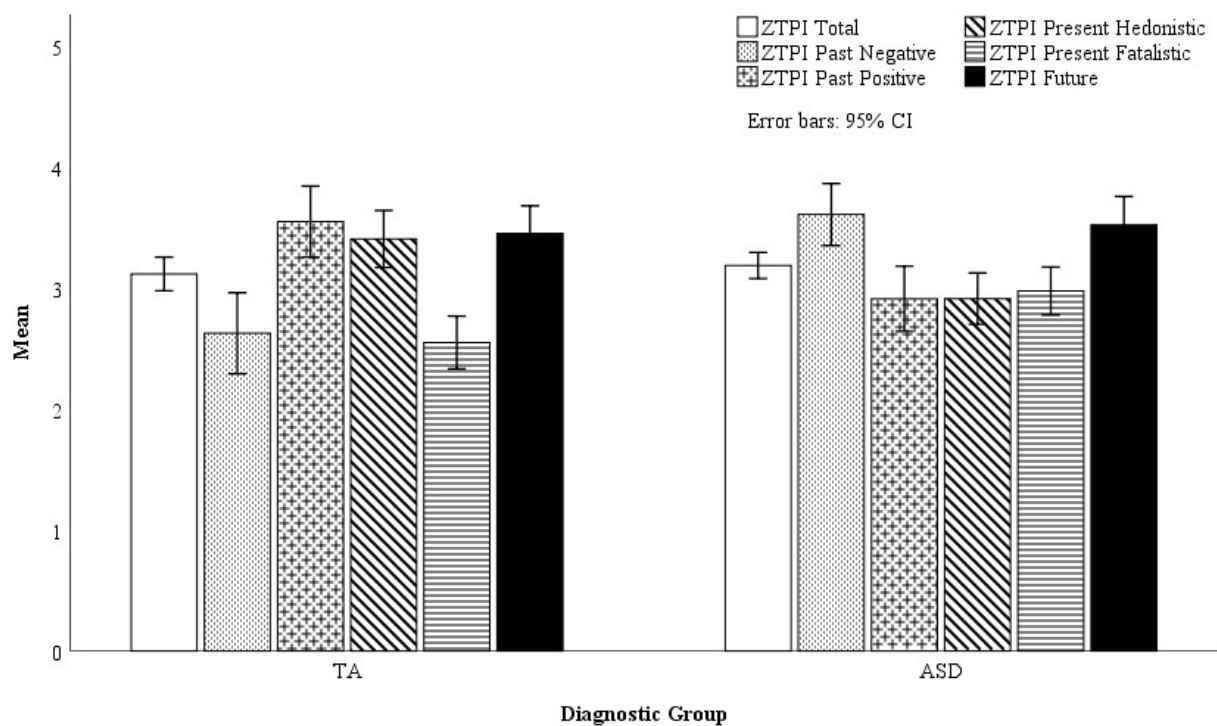


Figure 7.5. ZTPI scores between Diagnostic Groups.

Notes: Scores are reported (range 1-5) for Total, and Index scores related to Total, Past Negative, Past Positive, Present Hedonistic, Present Fatalistic, and Future time perception. Outcome scores represent stability or disintegrated time perception (temporal representation) across past, present and future.

Planned contrasts within Diagnostic Groups and between Age Groups showed no significant age-related differences within the TA group, across temporal indices (Table 7.4a, p. 258). A similar profile of no age-related temporal differences was observed within the ASD group, across all but one index of time – Past Negative (Table 7.4a, p. 258). Whilst the negligible effect sizes in the TA group indicate that the younger and older adults were closely matched in self-reported temporal representations, the small to moderate effect sizes in the ASD group suggest that there may be greater individual differences associated with time perception in ASD. However, when analyses included age as a continuous variable, rather than a grouping variable, the age-related effects disappeared ($F_{\max}(1,31) = 3.14, p_{\min} = .086; \eta_p^2_{\max} = .089$). Nor did any age effects emerge in the TA group ($F_{\max}(1,20) = 1.69, p_{\min} = .208; \eta_p^2_{\max} = .078$).

Planned contrasts within Age Groups and between Diagnostic Groups highlighted selective temporal representation differences between yASD and yTA adults (Table 7.4, p. 258), as indicated by large effect sizes. There were significant Diagnostic Group differences only in Past Negative ($F(1,26) = 18.979, p < .001; \eta_p^2 = .422$), Past Positive ($F(1,26) = 6.07, p < .03; \eta_p^2 = .189$), and Present Fatalistic representations ($F(1,26) = 9.03, p < .007; \eta_p^2 = .258$). In the older groups, a similar profile was observed with significant differences between oASD and oTA adults only in Past Negative ($F(1,26) = 6.38, p < .02; \eta_p^2 = .197$), Past Positive ($F(1,26) = 4.27, p < .05; \eta_p^2 = .141$), and Present Hedonistic representations ($F(1,26) = 6.13, p < .03; \eta_p^2 = .191$). In sum, the similarities were associated with Past and Present temporal representations. Regarding *past*, both yASD and oASD adults reported more Past Negative and fewer Past Positive biases than yTA and oTA adults. Whereas for *present*, the oASD adults reported fewer Present Hedonistic biases than oTA (and yASD) adults, whilst yASD reported more Present Fatalistic biases than yTA (and oASD) adults. Although these outcome measures were not directly of interest to the present study, they may provide clues to the way in which temporal processing might affect the everyday cognitive performance of autistic individuals. This would need to be considered in future work.

Next, Table 7.5 presents a summary of each outcome measure for the EBPM and TBPM naturalistic and laboratory tasks. This is followed by the presentation of results for each task.

Table 7.5. PM key reference terms and outcome measures for laboratory and naturalistic tasks

Outcome variable	Description
Nat-EBPM & Nat-TBPM	
Accuracy (Hits)	Hits indicate the correct PM action for each event in the Naturalistic EBPM, TBPM, self- and other-relevant tasks (e.g. remembering to send a message at the start of the journey (Nat-EBPM other-relevant)).
Commission Errors (False alarms)	False alarms indicate commission errors or PM actions that are not related to the target (e.g. responding immediately to Nat-TBPM survey for which the instruction is to respond in 14 days' time)
Omission Errors (Misses)	Misses indicate omission errors or no PM action, where one should have been made (e.g. remembering to ask for personal belongings (Nat-EBPM self-relevant), or remembering to complete the voucher survey (Nat-TBPM self-relevant)).
Lab-EBPM	
Accuracy (Hits) – strict (adj.)	Hits, using 'strict (adjusted)' criteria consider the PM actions made for a PM target (i.e. red square). These are adjusted for false alarms.
Accuracy (Hits) – lenient	Hits, using 'strict (adjusted)' criteria consider all PM actions made.
Commission Errors (False alarms)	False alarms indicate commission errors or PM actions that are not related to the target (e.g. PM action on blue square).
Omission Errors (Misses)	Misses indicate omission errors or no PM action, where one should have been made (i.e. on red square).
Response time (ms)	Response time in milliseconds indicates that average time taken to make a PM action.
Lab-TBPM	
Target times (t)	The intended time stamp for each of 6 TBPM actions at 2-minute intervals i.e. t1 = 120s; t2 = 240s; t3 = 360s; t4 = 480s; t5 = 600s; and t6 = 720s.
Clock check	The action of checking the time, by pressing the “c” keyboard button, to display the on-screen clock.
PM action	The action of executing the intended PM action to log the time every 2 minutes, by pressing the “space” keyboard button, to record a PM response.
Time monitoring;	The frequency of responses for <i>Clock checks</i> and <i>PM actions</i> across the task duration.
Strategy	Average rate of response across trials for <i>clock checking</i> and <i>PM actions</i> .
Accuracy	Measured in seconds (s) as the absolute difference ^a between <i>Clock check</i> and <i>PM action</i> responses from the target times. Respectively, accuracy of Clock checks as absolute difference ^a from target times (averaged across the task); accuracy as absolute difference ^a of PM actions from target time (0s): +/-10s; +/-20s; +/-30s; +/-40s; +/-50s; +/-60s ^b . Responses with negative absolute differences are consistently before target times, and positive absolute differences are consistently after target times.
Internal clock speed	A measure of the average duration of responses between <i>Clock checks</i> or <i>PM actions</i> , respectively, measured in seconds (s) across trials. The average interval of Clock check responses across the target times as a measure of the rate at which participants estimate time. The average interval of PM action responses across target times, as a measure of the rate at which participants estimate time.

Notes: ^a Absolute differences are the difference in seconds (s) between responses and target times; negative values denote *early* responses (before target times) and positive values denote *late* responses (after target times); zero (0s) represents the actual target times across the six, two-minute intervals (120s, 240s, 360s, 480s, 600s, 720s) during the task.

^b Responses outside of +/-60s were excluded because these would refer to both the next and previous target window. Thus, 60s is mid-point between two target times.

Naturalistic EBPM and TBPM performance in socially motivated tasks

The data set out in Figure 7.6 (p. 267) summarises the responses in ASD and TA groups in the naturalistic PM tasks. A series of *t*-tests explored the Diagnostic Group differences for pass (PM hits) or fail (PM misses) responses in Nat-EBPM and Nat-TBPM tasks, and the role of self- other-motivations in each of those tasks. Chi-square analysis explored the frequencies of naturalistic PM actions within Diagnostic Groups. Overall, fewer TA adults made attempts to complete the Nat-TBPM other-relevant task (PM hits = 36.36%) and Nat-TBPM self-relevant (PM hits = 45.45%) tasks, respectively, compared to Nat-EBPM self- or other-relevant tasks. This was followed by greater success in Nat-EBPM other-relevant (PM hits = 63.64%) and significantly more PM actions for Nat-EBPM self-relevant tasks (PM hits = 81.82%; $\chi^2(1) = 8.91, p = .003$). A similar patterning was observed in the ASD group, overall, although PM actions did not significantly differ across naturalistic tasks ($\chi^2(1) = 3.46, p = .063$). The highest PM successes for ASD adults were in Nat-EBPM self-relevant (PM hits = 65.71%) and Nat-EBPM other-relevant tasks (PM hits = 60.00%), with fewer successes in the Nat-TBPM other-relevant (PM hits = 48.57%) and Nat-TBPM self-relevant tasks (PM hits = 34.29%). In sum, an interesting variant in the patterning observed here, is that whilst TA adults had more PM successes in the Nat-TBPM self-relevant than Nat-TBPM other-relevant tasks, the opposite patterning was observed in the ASD group. Nevertheless, there were no Diagnostic Group differences across any of the naturalistic tasks (all $t(55) \leq 1.23, p > .05, \eta_p^2 \leq .030$).

Planned contrasts within Diagnostic Groups between Age Groups showed no significant age-related differences in PM success between yTA and oTA adults, across the naturalistic tasks (all $t(20) \leq 1.38, p > .05, \eta_p^2 \leq .071$). Whereas, in the ASD group performance on the Nat-EBPM self-relevant task was driven by a significantly higher proportion of responses from oASD compared to yASD adults – those responses were also higher than all other conditions (Table 7.6, p. 273). However, these data should be interpreted with caution since approximately half or more of the participants did not successfully complete all of the naturalistic tasks. Thus, any group differences are based on substantially reduced samples. Consequently, the effect sizes may offer more meaningful interpretation of the data than the *p*-values, in this study.

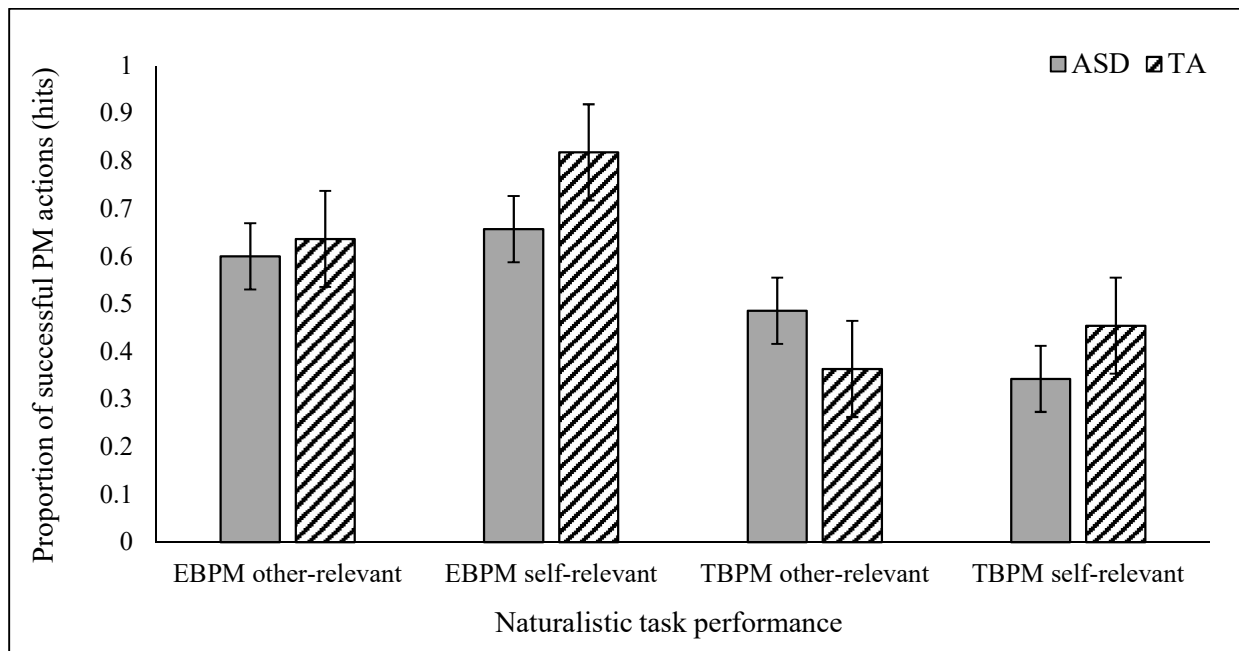


Figure 7.6. Naturalistic EBPM and TBPM task performance between Diagnostic Groups

Laboratory EBPM performance

(i) Lab-EBPM accuracy (strict)

The first analysis looked at Lab-EBPM *accuracy* as the proportion of hits, contrasted with the proportion of false alarms and misses, and the overall response time (ms) for PM hits (Table 7.6, p. 273). In the TA adults, there were no significant age-related differences in accuracy, which replicated the previous TA literature (e.g. Hering et al., 2014). Furthermore, there were no significant differences in accuracy between Diagnostic Groups ($F(1,52) = 1.05, p > .05, \eta_p^2 = .020$), which corresponds to the overall pattern of findings in the recent ASD PM literature (Landsiedel et al., 2017; Sheppard et al., 2018). In addition, there were no Diagnostic Group differences in hits when applying lenient accuracy (Dobbs & Rule, 1987), or in the proportion of false alarms (ASD: mean .45, SD 2.01; TA: mean .06, SD .11; $F(1,54) = <1.0, p > .05, \eta_p^2 = .015$), misses (ASD: mean .30, SD .29; TA: mean .20, SD .23; $F(1,54) = 1.77, p > .05, \eta_p^2 = .031$) or response times (ASD: mean 1080.84 ms, SD 271.20 ms; TA: mean 1100.50 ms, SD 311.56 ms; $F(1,54) = <1.0, p > .05, \eta_p^2 = .001$). These findings indicate that, overall, ASD adults are as able in Lab-EBPM as TA adults, regardless of the stringency of success

criteria. However, one participant in each of the yASD, oASD and oTA groups made substantially more errors than other participants. Only in the ASD group did these errors result in negative scores for adjusted hits. Post-hoc questions indicated that those participants appeared to have misunderstood the task instructions and, although they had remembered to make PM actions, their responses were not aligned with the task rules for accuracy (i.e. red box indicates PM action to press “1”). Subsequently, on excluding those participants from the analysis, the results were unchanged ($F(1,51) = <1.0, p > .05, \eta_p^2 = .009$). Thus, all participants were retained in the subsequent analyses.

Planned contrasts between Age Groups within Diagnostic Groups showed a slightly different patterning. In the TA adults, although there were no significant age-related differences in the proportion of hits (strict and lenient), false alarms or misses, the oTA adults were significantly slower in responding than were the yTA adults (Table 7.6, p. 273). These findings, once again replicate the TA literature, suggesting that a speed-accuracy trade-off (task interference) is of greater consequence for oTA in successfully completing EBPM actions (as measured by the Lab-EBPM accuracy), than it is for yTA adults (Cook, Ball & Brewer, 2014). Indeed, previous literature has shown that response time *costs* are a measure of general PM difficulties, as a consequence of conflicting cognitive demands (Brewer et al., 2011). By contrast, in the ASD group, there were no significant age-related differences in Lab-EBPM accuracy, false alarms, misses, or response times (Table 7.6, p. 273).

Planned contrasts between Age Groups within Diagnostic Groups showed no significant differences between younger ASD and TA groups in hits ($F(1,26) = 1.30, p > .05, \eta_p^2 = .048$), false alarms ($F(1,26) = <1.0, p > .05, \eta_p^2 = .005$), or misses ($F(1,26) = 2.41, p > .05, \eta_p^2 = .085$). By contrast, response times were significantly slower in the yASD group compared to the yTA group ($F(1,26) = 5.24, p = .03, \eta_p^2 = .168$). In contrasts between the older groups, there were no significant Diagnostic Group differences in hits ($F(1,26) = <1.0, p > .05, \eta_p^2 < .001$), false alarms ($F(1,26) = <1.0, p > .05, \eta_p^2 = .029$), or misses ($F(1,26) = <1.0, p > .05, \eta_p^2 = .004$). However, slower response times were observed in the oASD compared to oTA adults, although this did not reach significance ($F(1,26) = 3.96, p = .057, \eta_p^2 = .132$). A possible explanation of these findings is that oASD adults may take a more considered approach to processing the information in order to make correct judgments, thus incurring a speed-accuracy trade-off. Another explanation is that cognitive mechanisms associated with

processing speed, or EF involved in cognitive flexibility for task switching may be involved. These factors are considered later in this chapter when exploring the role of EF in PM and ASD.

(ii) Ongoing task performance in the EBPM laboratory task

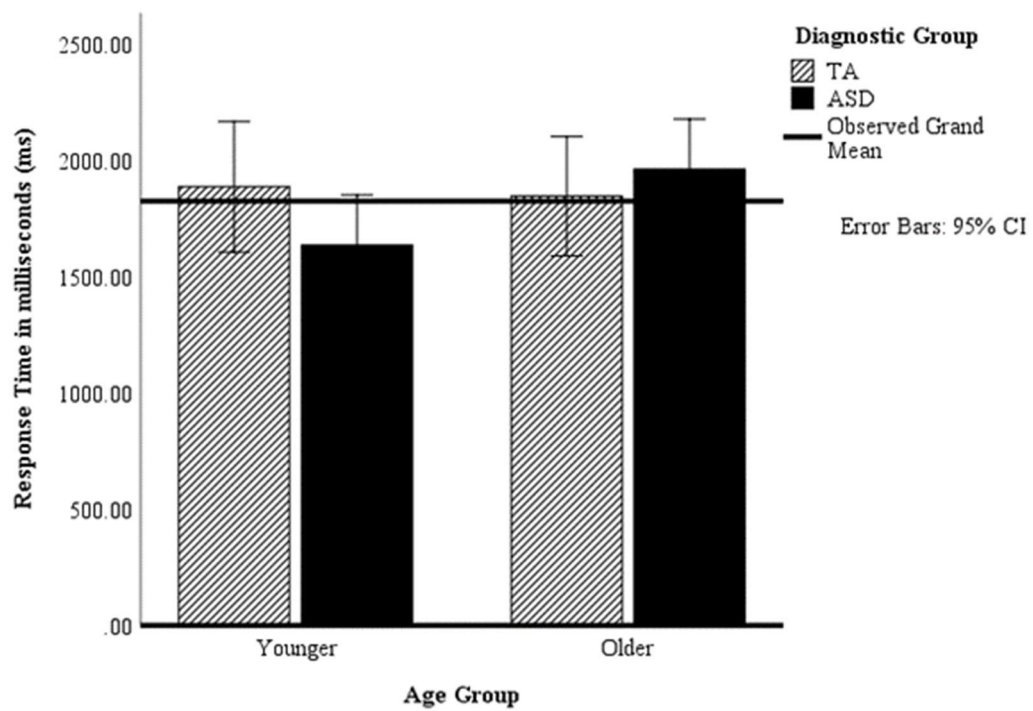
Despite the varied performance differences on the Lab-EBPM task just mentioned, there were no Diagnostic Group differences in overall costs to the ongoing task performance, related to hits ($F(1,55) = 2.09, p > .05, \eta_p^2 = .037$) or response times ($F(1,55) = 3.96, p = .05, \eta_p^2 < .001$). Moreover, all groups performed close to ceiling in the ongoing Lab-EBPM task (ASD: mean .93, SD .10; TA: .96, SD .03; Table 7.6, p. 273), indicating that there were no costs to ongoing task performance whilst performing the EBPM task. However, planned contrasts within Diagnostic Groups and between Age Groups showed that oTA adults, once again, had significantly slower response times than yTA adults ($F(1,20) = 8.47, p = .009, \eta_p^2 = .297$), despite no age-related performance differences in the ongoing task ($F(1,20) < 1.0, p > .05, \eta_p^2 < .001$). A similar pattern was observed in the ASD group. Although there were also no observed age-related performance differences ($F(1,33) < 1.0, p > .05, \eta_p^2 < .005$), the oASD adults were slower in the ongoing Lab-EBPM task than were yASD adults ($F(1,33) = 10.66, p = .003, \eta_p^2 = .244$). Finally, planned contrasts between Diagnostic Groups within Age Groups confirmed the absence of performance differences in the ongoing Lab-EBPM task (younger, $F(1,26) = 1.10, p > .05, \eta_p^2 = .041$; older, $F(1,27) < 1.0, p > .05, \eta_p^2 = .032$). Nor were there any differences in response times in the ongoing Lab-EBPM task (younger, $F(1,26) < 1.0, p > .05, \eta_p^2 = .009$; older, $F(1,26) < 1.0, p > .05, \eta_p^2 < .001$).

(iii) Retrospective memory in the EBPM laboratory task

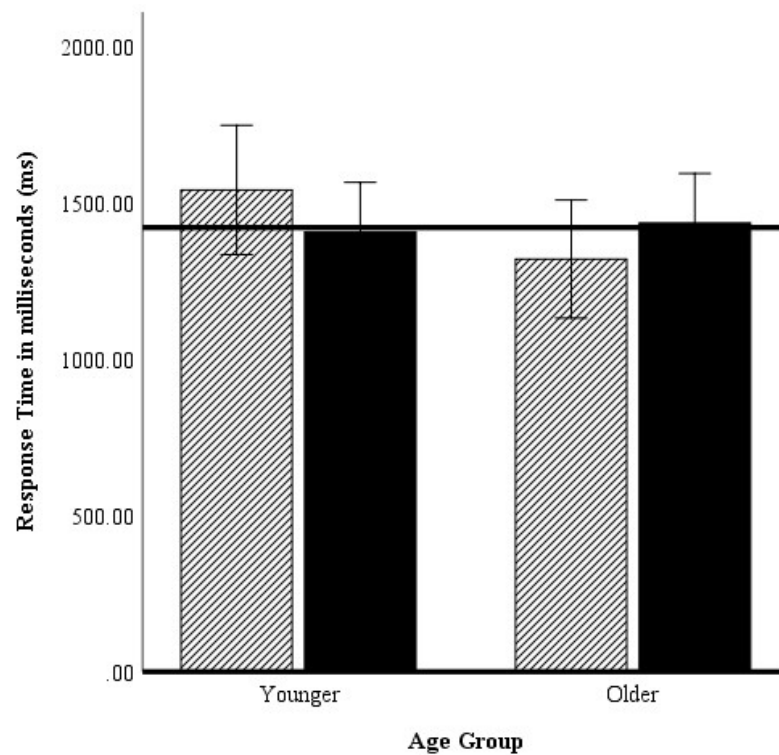
Next, the analysis looked at whether participants were able to correctly recognise familiar or novel words in the surprise recognition memory test from the Lab-EBPM task. Using the RKG procedure described earlier, responses were analysed for adjusted *accuracy* (hits minus false alarms) in identifying previously presented (familiar) from new (novel) items (Figure 7.7, p. 271), and confidence of response (R/K/G; Figure 7.8, p. 272). There were no Diagnostic Group differences in the recognition of familiar words and nonwords (ASD: mean .45, SD .22; TA: mean .46, SD .17;

$F(1,55) = <1.0, p > .05, \eta_p^2 < .001$), or identifying novel items (ASD: mean .72, SD .20; TA: mean .74, SD .14; $F(1,55) = <1.0, p > .05, \eta_p^2 = .002$). Overall, both groups were better able to detect a higher proportion of novel than familiar items.

Planned contrasts within Diagnostic Groups between Age Groups confirmed that there were no age-related differences in the recognition of familiar items by the TA ($F(1,20) = <1.0, p > .05, \eta_p^2 = .036$) or ASD group ($F(1,33) = <1.0, p > .05, \eta_p^2 = .020$). Nor were there any age-related differences in identifying novel items (ASD: $F(1,33) = <1.0, p > .05, \eta_p^2 = .016$; TA: $F(1,20) = <1.0, p > .05, \eta_p^2 = .001$). Furthermore, response times did not differ between Diagnostic Groups for either familiar items (ASD: mean 1797.23 ms, SD 487.43 ms; TA: 1863.00, SD 393.95; $F(1,54) <1.0, \text{all } p > .05, \eta_p^2 = .005$), or for novel items (ASD: mean 1409.21 ms, SD 347.52 ms; TA: 1417.22, SD 288.77; $F(1,55) <1.0, \text{all } p > .05, \eta_p^2 < .001$). Finally, R/K/G responses were analysed only for correctly identified familiar items. There were no significant differences between Diagnostic Groups on R/K/G responses (all $F(1,52) \leq 2.77, \text{all } p > .05$, largest $\eta_p^2 = .051$; and see Figure 7.8, p. 272). However, the overall patterning of responses showed a higher proportion of R (remember) judgements in both ASD (mean .42, SD .26) and TA groups (mean .39, SD .22) for items that had been correctly identified as familiar. This was followed by G (guess) responses (ASD: mean .32, SD .21; TA: .37, SD .20), and then by K (know) responses (ASD: mean .26, SD .20; TA: mean .24, SD .14). Thus, the overall picture would seem to indicate that there were not any greater retrospective memory difficulties underlying the Lab-EBPM performance in ASD compared to TA adults.

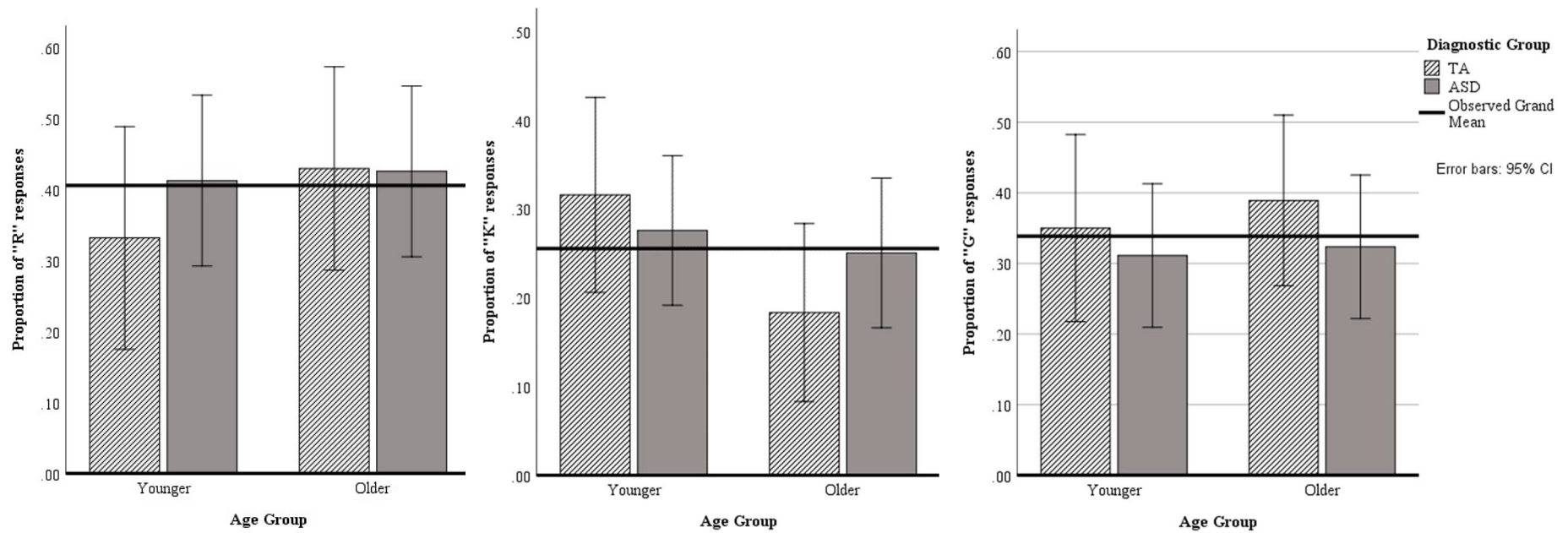


(a) correctly identified familiar items



(b) correctly identified novel items

Figure 7.7. Lab-EBPM Recognition memory test response times.



(a) Proportion of Remember "R" responses

(b) Proportion of Know "K" responses

(c) Proportion of Guess "G" responses

Figure 7.8. Lab-EBPM Recognition memory as measured by response certainty. Error bars indicate 95% CI.

Table 7.6. Diagnostic Groups performance on EBPM, TBPM and ongoing laboratory tasks and naturalistic (self- and other-relevant) tasks

Measure	ASD		Statistics			TA		Statistics		
	yASD (n=18)	oASD (n=17)	ANOVA	sig.	η^2	yTA (n=10)	oTA (n=12)	ANOVA	sig.	η^2
<u>Nat-EBPM</u>										
<i>Self-relevant</i>	.61 (.50)	.59 (.51)	<1.0	<i>n.s.</i>	.001	.50 (.53)	.75 (.45)	1.44	<i>n.s.</i>	.067
<i>Other-relevant</i>	.50 (.51)	.82 (.39)	4.33	.045	.116	.80 (.42)	.83 (.39)	<1.0	<i>n.s.</i>	.002
<u>Nat-TBPM</u>										
<i>Self-relevant</i>	.33 (.49)	.65 (.49)	3.60	.066	.098	.40 (.52)	.33 (.49)	<1.0	<i>n.s.</i>	.005
<i>Other-relevant</i>	.33 (.49)	.35 (.49)	<1.0	<i>n.s.</i>	.000	.60 (.52)	.33 (.49)	1.53	<i>n.s.</i>	.071
<u>Lab-EBPM</u>										
<i>PM Accuracy</i>										
Proportion Hits (strict) ^a	.52 (.55)	.73 (.26)	2.61	<i>n.s.</i>	.060	.73 (.26)	.74 (.31)	<1.0	<i>n.s.</i>	.027
Proportion Hits (lenient) ^b	.67 (.29)	.79 (.23)	1.74	<i>n.s.</i>	.052	.73 (.26)	.77 (.26)	<1.0	<i>n.s.</i>	.014
False alarms	.15 (.41)	.79 (2.91)	<1.0	<i>n.s.</i>	.026	.10 (.15)	.04 (.06)	1.83	<i>n.s.</i>	.084
Misses	.33 (.29)	.26 (.29)	<1.0	<i>n.s.</i>	.016	.17 (.20)	.23 (.26)	<1.0	<i>n.s.</i>	.013
Response time (ms)	1081.99 (274.06)	1079.54 (289.69)	<1.0	<i>n.s.</i>	<.001	866.75 (149.66)	1295.28 (275.57)	19.32	<.001	.491
<i>Ongoing tasks</i>										
Hits (adj.)	.92 (.12)	.94 (.09)	<1.0	<i>n.s.</i>	.005	.96 (.03)	.96 (.03)	<1.0	<i>n.s.</i>	.010
Response time (ms)	834.02 (144.49)	1017.89 (187.09)	10.66	.003	.244	807.09 (133.74)	1024.52 (201.79)	8.47	.009	.297
<u>Lab-TBPM</u>										
<i>PM Accuracy</i>										
Proportion Hits (strict adj.) ^b	.11 (.62)	.25 (.62)	<1.0	<i>n.s.</i>	.010	.27 (.73)	-.08 (.56)	1.57	<i>n.s.</i>	.080
Proportion Hits (lenient) ^c	.93 (.24)	.97 (.07)	<1.0	<i>n.s.</i>	.020	1.00 (0.0)	1.00 (0.0)	-	-	-
<i>Time monitoring^d</i>										
Clock checks	9.39 (6.28)	11.56 (7.90)	<1.0	<i>n.s.</i>	.024	17.00 (7.65)	10.55 (7.89)	3.61	<i>n.s.</i>	.160
PM actions	4.33 (2.06)	5.00 (5.07)	<1.0	<i>n.s.</i>	.027	4.70 (1.70)	3.45 (2.07)	2.24	<i>n.s.</i>	.106
<i>Clock speed^e</i>										
Clock checks	82.78 (44.28)	95.3 (70.46)	<1.0	<i>n.s.</i>	.012	48.21 (14.73)	90.97 (52.24)	6.19	.02	.267
PM actions	134.55 (36.27)	168.18 (126.06)	<1.0	<i>n.s.</i>	.033	145.14 (44.38)	199.16 (99.69)	2.42	<i>n.s.</i>	.124
<i>Ongoing tasks</i>										
Hits (adj.)	.97 (.05)	.97 (.03)	<1.0	<i>n.s.</i>	.008	.98 (.02)	.98 (.01)	.53	<i>n.s.</i>	.026
Response time (ms)	1134.83 (379.70)	1227.19 (382.92)	<1.0	<i>n.s.</i>	.015	1001.04 (333.26)	1205.90 (269.98)	2.32	<i>n.s.</i>	.104

Notes: ^aHits (strict unadj.) is reported here as the unadjusted count of all PM actions made within the target window (± 20 s).

^bHits (strict adj.) is reported as the adjusted proportion of correct PM actions (± 20 s), after excluding false alarm responses (commission errors; ± 21 s to ± 60 s). There is 0.33 chance of success if responding randomly. Negative scores here indicate more random responses, and positive scores indicate more strategic responses, whilst a zero-value indicated performance at chance.

^cHits (lenient) is reported as the total PM attempts made. The 'lenient' coding approach credits the individual for all PM action responses made within $T \pm 60s$, recognising that the individual remembered the content of the task, but not the specific timing.

^dTime monitoring is reported as the frequency of clock checks or PM actions.

^eClock speed is reported as the mean time in milliseconds (ms) between clock checks, or PM actions, respectively.

Laboratory TBPM performance

As a reminder, a summary of the main outcome measures for the Lab-TBPM task are set out in Table 7.5 (p. 265). A descriptive view of TBPM performance indicated that oTA made just more than half the required six PM actions (unadjusted; mean 3.45, SD 2.07), which was fewer than yTA adults (mean 4.70, SD 1.70; $F(1,26) = 2.19, p > .05, \eta_p^2 = .103$). Although both groups made a similar proportion of clock checks for each PM action ($F(1,26) < 1.0, p > .05, \eta_p^2 < .001$), the oTA adults checked the clock less frequently, overall, but none of these differences reached significance (Table 7.6, p. 273). However post-hoc t-tests revealed a trend to significance for oTA making fewer responses compared to yTA ($t(19) = 2.04, p = .056$), and a significantly higher proportion of responses for oASD compared to yASD ($t(32) = -2.26, p = .031$) and compared to oTA adults ($t(19) = -3.12, p = .006$).

(i) Lab-TBPM accuracy (strict)

There were 6 expected responses per participant, amounting to an expected total 330 PM actions across TA and ASD groups. More than one-quarter (26.3%) of all participants made exactly 6 PM actions, whilst 35.1% of all participants made 3 or fewer PM actions (not including non-responses), and 12.4% made 7 or more PM actions ($N=7$; oTA = 1; yASD = 2; oASD = 4). Failure to make any PM actions occurred in just 2 participants (3.5%; oTA = 1; oASD = 1), whilst one participant (oASD) made a maximum of 9 PM actions. In the ASD group, oASD adults made slightly more of the required PM actions (mean 5.00, SD 2.07) than yASD adults (mean 4.33, SD 2.06; $F(1,26) < 1.0, p > .05, \eta_p^2 < .001$). Further, the oASD adults also made more clock checks than yASD, although a similar number of clock checks per PM action were made by both oASD (mean 2.92, SD 2.73) and yASD adults (mean 2.81, SD 2.88). None of these differences were significant (Table 7.6, p. 273). Planned contrasts between older and younger participants within Diagnostic Groups, on the average number of PM actions, confirmed there were no significant differences between yTA and oTA ($F(1,20) = 3.22, p > .08, \eta_p^2 = .139$), or between yASD and oASD ($F(1,33) < 1.0, p > .05, \eta_p^2 = .011$; Table 7.6, p. 273). Planned contrasts comparing Diagnostic Groups within older and younger Age groups revealed no Diagnostic Group differences in the average number of PM actions between yTA and yASD ($F(1,26)$

$< 1.0, p > .05, \eta_p^2 = .009$), and no differences between oTA and oASD ($F(1,26) = 3.19, p > .05, \eta_p^2 = .106$).

In comparisons between Diagnostic Groups, there were no significant differences on TBPM accuracy ($F(1,51) < 1.0, p > .05, \eta_p^2 = .006$) or time monitoring ($F(1,51) = 2.61, p > .05, \eta_p^2 < .049$). However, a significant Diagnostic Group x Age Group interaction effect emerged in time monitoring ($F(1,51) = 4.46, p < .05, \eta_p^2 < .08$). Planned contrasts revealed that this was driven by a greater frequency of clock check by yTA adults compared to oTA and all other groups (Table 7.6, p. 273). Furthermore, planned contrasts between Diagnostic Groups within Age Groups showed no significant differences across TBPM measures ($F_{\max}(1,26) = 3.65, p_{\min} > .05, \eta_p^2_{\max} = .127$), with one exception. In the younger group, there was a significant difference in time monitoring of yTA adults compared to yASD adults ($F(1,26) = 8.09, p < .01, \eta_p^2 = .237$). However, no significant differences emerged in planned contrasts between Age Groups within Diagnostic Groups (Table 7.6, p. 273).

In sum, there were no Diagnostic Groups differences on EBPM or TBPM tasks, and no age-related effects on PM performance in either the TA or ASD group.

Next, the *accuracy* of PM responses was explored in relation to *time monitoring*, by measuring the absolute difference from target times, with smaller difference from target times representing greater accuracy. Earlier in the present chapter, the question was asked:

“Does the frequency of Lab-TBPM Clock check responses increase and does the average time interval between Clock check responses decrease, as the target time approaches?”

To answer this question, the detailed patterning of switching behaviours – from clock checks to PM action responses – was explored within defined time bins -60s to +60s of the six PM target times (T; i.e. T1=120s, T2=240s, T3=360s, T4=480s, T5=600s, T6=720s), averaged across the task.

Responses that were made outside of +/-60s (i.e. +/-61s to +/-119s), across trials, were not included in the analyses for *accuracy* or *clock speed*, since these responses fell equally between two given target times and were, therefore, difficult to quantify as ‘early’ or ‘late’ responses associated with a given target time. The data were checked to ensure that the proportion of such responses did not result in exclusion of large amounts of data. A frequency count revealed that only 7.34% of Total clock check responses (47 of 640; N=25) were made on the first trial, within the first 0s to -60s from the start of the

trial and before the first target time (i.e. 120s). The results indicated one outlier in the oASD participants who made 4 clock checks in the first 0s to -60s in the first trial, but there were no differences in the number of responses made between Diagnostic Group ($F(1,23) < 1.0, p > .05, \eta_p^2 = .041$) or Age groups ($F(1,23) < 1.0, p > .05, \eta_p^2 = .013$). Five participants made PM action responses in the first -60s before the target time (yTA = 1; yASD = 3; oASD = 2). These numbers were too small for meaningful analysis and the data were not considered further.

The time bins of primary interest for PM accuracy were -20s to 0s before the target times, and 0s to +20s after the target times, since descriptive data suggested that the greatest distribution of scores were within these time bins and would, therefore, indicate the greatest accuracy of responses as being closest to the target times (Figure 7.9 (a) and (b), p. 281). Overall, the ASD group made a greater proportion of responses within the target time, than did TA adults. Further exploration revealed that 64% of the ASD PM actions were within 30s of the target, compared with 51% of the TA group. However, this did not reach significance. Furthermore, there were no Diagnostic Group differences within 20s or 10s of the target time. Post-hoc analysis of age-related effects within ASD and TA groups showed that oTA adults had a significantly lower response accuracy than did yTA adults ($t(16) = 2.13, p < .05$). Moreover, the oASD adults also showed significantly higher accuracy than oTA adults ($t(17) = -2.47, p < .03$). In sum, the ASD group were closer to the target times across trials ($F(1,51) = 11.36, p < .001$), but this group also had a higher response rate overall, compared to TA adults. Consequently, as previously indicated in the overview, when the responses were adjusted for response rate (including false alarms), these differences were not significant. There were no significant Diagnostic Group differences in PM actions -20s before the target times ($F(1,53) = 1.97, p > .05, \eta_p^2 = .036$), but Diagnostic Group differences in PM accuracy emerged in the +20s after target times ($F(1,53) = 4.11, p < .047, \eta_p^2 = .070$), which were explored in planned contrasts. No other time bins were significant between Diagnostic Groups, and there were no observed Age effects and no Diagnostic Group x Age interactions (largest $F(1,55) = 2.74, p > .05, \eta_p^2 = .049$).

Planned contrasts comparing older and younger participants within Diagnostic Group showed no significant Diagnostic Group differences in PM actions between younger and older Age groups within TA or ASD groups ($F_{\max}(1,20) = 2.33, p_{\min} = .14, \eta_p^2_{\max} = .104$). Further, planned contrasts

comparing Diagnostic Groups within younger and older Age groups showed no differences between younger participants (TA/ASD; $F_{\max}(1,26) = 3.82, p_{\min} = .06, \eta_p^2 = .128$). However, between older participants (TA/ASD), oTA made significantly fewer PM actions within 0 to +20s after the target time, compared to oASD ($F(1,27) = 4.90, p < .04, \eta_p^2 = .153$). The older ASD participants, therefore, showed greater PM accuracy than older TA participants.

(ii) Lab-TBPM time monitoring

The frequency of clock check responses was explored in relation to possible time monitoring differences between Diagnostic Groups and Age Groups. An average of 6 to 12 clock checks were expected across the task, if participants checked the clock time every 2-minutes to 1-minute, respectively. There were no imposed limits imposed on the number of clock checks that participants were able to make during the course of the task. A total of 640 clock check responses were made across TA and ASD groups. Overall, participants made an average 11.23 (SD 7.80; range 0-32) clock check during the TBPM task, but time monitoring behaviour was variable between participants. Overall, 54.4% of participants ($N=31$) made 12 or fewer clock check (not including non-responses), with 12.3% of participants making exactly 12 clock check, and 36.8% of participants making 13 or more responses. Five participants (8.8%) did not check the clock at all (oTA = 2; yASD = 2; oASD = 1), and one participant (yTA) checked 32 times (maximum response).

In comparisons between Diagnostic Groups, the TA adults made an average of 13.00 (SD 8.57) clock checks and ASD adults made an average of 10.11 (SD 7.18) clock checks, but there were no significant differences between Diagnostic Groups ($F(1,55) = 2.44, p > .05, \eta_p^2 = .044$) or Age groups ($F(1,53) = 2.04, p > .05, \eta_p^2 = .037$). However, there was significant Diagnostic Group x Age Group interaction effect ($F(1,53) = 4.65, p < .04, \eta_p^2 = .081$). Post-hoc tests confirmed that there were no age-related effects in the ASD group, but in the TA group there were fewer clock checks by oTA than yTA adults ($t(19) = 2.57, p < .02$).

There were no significant Diagnostic Group differences in clock check responses at target +/- 20s ($F_{\max}(1,55) < 1.0, p_{\min} > .05$). However, overall, participants increased the frequency of clock checks between -40s to 0s of the target times (43% of responses), and then the response behaviours

were directed toward PM actions between -20s and +20s of the target time (42% of responses; as shown in Figure 7.9 (a) and (b), p. 281). Significant differences were observed between the Diagnostic Groups in clock check frequency at -40s to -20s before the target time ($F(1,53) = 6.47, p < .02, \eta_p^2 = .109$), and between Age groups at -60s to -40s before the target time ($F(1,53) = 8.03, p < .007, \eta_p^2 = .132$). Furthermore, a significant Diagnostic Group x Age interaction effect was observed for both -60s to -40s ($F(1,53) = 5.331, p = .025, \eta_p^2 = .091$), and -40s to -20s time bins ($F(1,53) = 7.28, p < .01, \eta_p^2 = .121$). No other time bins showed significant Diagnostic Group or Age differences.

Planned contrasts within Diagnostic Groups and between Age Groups showed age-related performance differences only in the TA group, with oTA adults making fewer clock check responses than yTA at -60s to -40s (yTA $\bar{x}$ 3.60, SD 2.37; oTA $\bar{x}$ 1.17, SD 1.19; $F(1,20) = 9.78, p < .006, \eta_p^2 = .328$), and at -40s to -20s (yTA $\bar{x}$ 4.40, SD 2.55; oTA $\bar{x}$ 1.92, SD 2.02; $F(1,20) = 6.51, p < .02, \eta_p^2 = .246$). In the ASD group there were no age-related differences in clock checks across time bins ($F_{\max}(1,33) = 2.14, p_{\min} > .05, \eta_p^2_{\max} = .066$).

Planned contrasts within Age Groups and between Diagnostic Groups revealed that yASD adults made significantly fewer successive clock checks than yTA adults in the -60s to -40s time bin ($F(1,26) = 5.362, p = .029, \eta_p^2 = .171$), and in the -40s to -20s time bin ($F(1,26) = 12.424, p = .002, \eta_p^2 = .323$). There were no Diagnostic Group differences between oASD and oTA adults in *time monitoring* at any time point ($F_{\max}(1,27) < 1.0, p_{\min} > .05, \eta_p^2_{\max} = .029$).

(iii) Lab-TBPM response clock speed

Internal *clock speed* is measured as the average duration in seconds (s) between responses (interval), taken as an estimate of participants' judgement of elapsed time before making the required responses (either clock checks or PM actions). Accordingly, the average interval between successive clock check or PM action responses was calculated as measures of *clock speed*. For PM actions, the *clock speed* interval was taken as the average of PM action responses across target times. Here, no significant Diagnostic Group differences were observed in PM action *clock speed* ($F_{\max}(1,46) = 2.934, p_{\min} = .093, \eta_p^2_{\max} = .060$).

Planned contrasts between Age Groups within Diagnostic Group showed no significant age-related differences for *clock speed* in PM actions, in the TA group ($F(1,29) = 2.42, p > .05, \eta_p^2 = .124$; yTA: mean 145.14, SD 44.38; oTA: mean 199.16, SD 99.69), or in the ASD group ($F(1,29) < 1.0, p > .05, \eta_p^2 = .033$; yASD: mean 134.54, SD 36.27; oASD: mean 168.18, SD 126.06).

Further, planned contrasts between Diagnostic Groups within Age Groups, showed no significant Diagnostic Groups differences in *clock speed* for clock check intervals between yASD and yTA adults ($F(1,23) < 1.0, p > .05, \eta_p^2 = .018$), and no significant differences between oASD and oTA adults ($F(1,23) < 1.0, p > .05, \eta_p^2 = .017$). What is more, in the TA group chronological age was a significant predictor of *clock speed* for clock checks ($F(1,17) = 14.07, p < .003, \eta_p^2 = .421$) and PM actions ($F(1,17) = 6.51, p < .03, \eta_p^2 = .234$), but this was not the case in the ASD groups ($F_{\max}(1,29) < 1.0, p_{\min} > .05, \eta_p^2_{\max} = .030$).

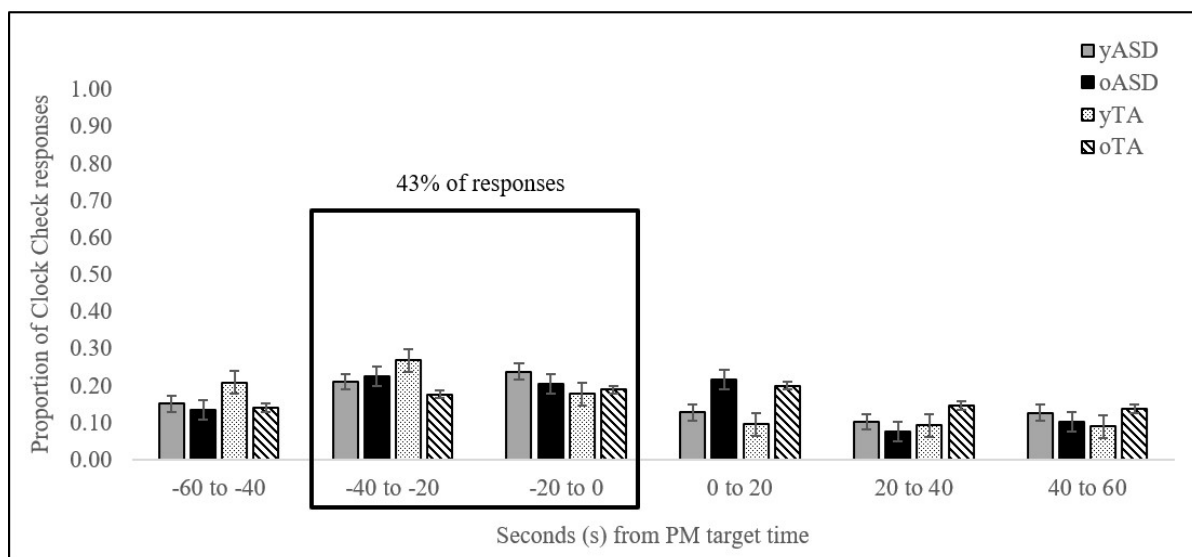
(iv) Associations between time monitoring and PM accuracy

Finally, the relation between *clock checks* and PM response *accuracy* was analysed, as well as the role of internal *clock speed* in PM *accuracy*.

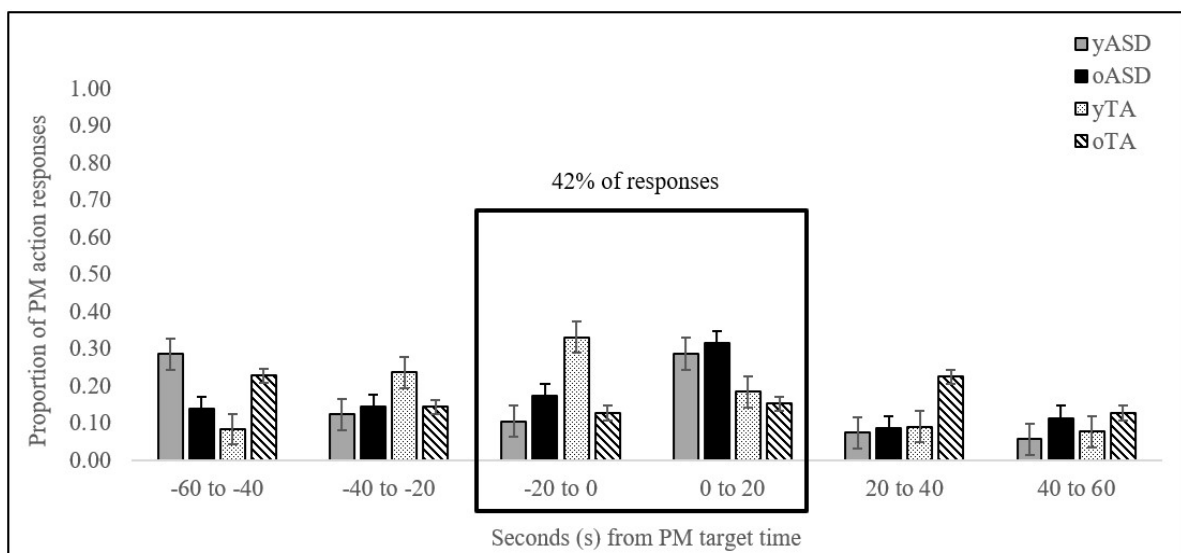
How does clock checking behaviour relate to PM action accuracy?

Overall, there was no significant correlation with the average interval (*clock speed*) of PM actions ($r(51) = -.138, p > .05$). Nevertheless, participants tended to increase their frequency of clock checks and, therefore, increase their *clock speed* immediately prior to the target time (see Figure 7.9, p. 281). In the time bins of interest, at -20s to 0s before the target time, there was a strong correlation between clock checks and PM actions at +/-40s of the target times, as follows: clock checks at -40s to -20s were significantly associated with PM actions at -20s to 0s ($r(57) = .38, p = .004$); and clock checks at -20s to 0s were significantly associated with PM actions at -20s to 0s ($r(57) = .29, p < .03$) and at 0s to +20s ($r(57) = .44, p = .001$). Thus, the results indicate support for the prediction that increased frequency of clock check responses closer to the target times would be associated with greater accuracy of PM actions. However, given the within Diagnostic Group differences for TA adults, these data should be interpreted with caution.

Correlations within Age Groups and Diagnostic Group reconfirmed the associations between clock checks and PM actions for yTA adults at -20s to 0s ($r(10) = .77, p < .01$) accounting for 58.68% of the variance in accuracy for younger TA adults. By contrast, no associations were observed for oTA adults, or for yASD or oASD adults, in any time bins. This patterning of results suggests that older TA adults, and similarly ASD adults, used different time monitoring strategies to complete the Lab-TBPM task compared to younger TA adults.



(a) Time Monitoring: Clock Check responses in relation to Lab-TBPM Target Times



(b) Time Monitoring: PM action responses in relation to Lab-TBPM Target Times

Figure 7.9. Lab-TBPM time monitoring behaviours in relation to PM actions, within Diagnostic Groups and Age groups.

(v) Ongoing task performance in the TBPM laboratory task

There were no Diagnostic Group differences in the ongoing Lab-TBPM task performance related to hits ($F(1,53) = 1.41, p > .05, \eta_p^2 = .026$) or response times ($F(1,53) < 1.0, p > .05, \eta_p^2 = .012$), indicating that all participants were able to complete the ongoing task as instructed (Table 7.6, p. 273).

Planned contrasts within Diagnostic Groups and between Age Groups analysed performance costs to hits and response times of the ongoing tasks. In the TA group, performance costs in response times, but not hits, were observed for oTA compared to yTA adults. Although the response time differences did not reach significance, the large effect sizes indicate that younger and older TA groups did differ in performance (Table 7.6, p. 273). Within the ASD group, there were no differences in performance on the ongoing task in terms of hits or response times (Table 7.6, p. 273).

Planned contrasts within Age Groups and between Diagnostic Groups showed no differences in ongoing task performance between yASD and yTA adults, for hits ($F(1,26) < 1.0, p > .05, \eta_p^2 = .018$) or response times ($F(1,26) < 1.0, p > .05, \eta_p^2 = .032$). Nor were there any differences between oASD and oTA adults for ongoing task hits ($F(1,27) = 1.58, p > .05, \eta_p^2 = .055$) or response times ($F(1,27) < 1.0, p > .05, \eta_p^2 = .001$).

(vi) Retrospective memory in the TBPM laboratory task

Following the RKG procedure described earlier, responses were analysed for adjusted *accuracy* (hits minus false alarms) in identifying previously presented (familiar) from new (novel) items, and confidence of response (R/K/G). In the Lab-TBPM recognition memory test, there were no Diagnostic Group differences in recognition accuracy or response times for familiar items or for novel items (all $F(1,53) < 1.0$, all $p > .05$, all $\eta_p^2 \leq .007$). Furthermore, there were no Diagnostic Group differences in recognition response type (i.e. R/K/G; all $F(1,53) < 1.0$, all $p > .05$, all $\eta_p^2 \leq .013$). What is more, planned contrasts within Diagnostic Groups and between Age Groups showed no age-related differences between younger and older TA adults in recognition accuracy, response time, or response type (all $F(1,20) \leq 1.91$, all $p \geq .18$, all $\eta_p^2 \leq .087$). In the ASD group, there were no age-related

differences in recognition accuracy ($F(1,33) = 1.57, p > .05, \eta_p^2 = .045$) or response times ($F(1,33) < 1.0, p > .05, \eta_p^2 = .001$) for familiar items. However, for novel items the difference in recognition accuracy was on the edge of significance ($F(1,33) = 3.81, p = .059, \eta_p^2 = .104$), which appeared to be driven by more accurate recognition of novel items by oASD (mean .80, SD .09) compared to yASD adults (mean .68, SD .23). There were no age-related differences between yASD and oASD adults in response times for recognition of novel items ($F(1,33) < 1.0, p > .05, \eta_p^2 = .001$). Nor were there any differences in the type of recognition response (R/K/G; all $F(1,33) < 1.0$, all $p > .05$, all $\eta_p^2 \leq .008$).

Executive function as a factor in laboratory and naturalistic EBPM and TBPM performance

Finally, the role of EF was explored in relation to EBPM and TBPM in the laboratory and naturalistic tasks. The data set out below summarise the main correlations between EF data collected at T1 (Chapter 4) and performance on the laboratory PM tasks within ASD and TA groups.

In the TA group, only Episodic Memory and Learning was significantly correlated with Lab-TBPM performance ($r(19) = .54, p = .02$), but there were no other associations between any of the EF measures and Lab-TBPM or Lab-EBPM performance (all $r(19) \leq .41$, all $p \geq .13$). However, in the naturalistic tasks, EF cognitive flexibility was significantly positively associated with Nat-EBPM *other-relevant* performance ($r(22) = .55, p = .007$), whereas Nat-TBPM *other-relevant* performance was significantly negatively associated with EF Planning skills ($r(16) = -.56, p = .025$) and EF Attention/Inhibition ($r(18) = -.47, p = .048$). In the ASD group a somewhat different profile of EF associations emerged. In the laboratory tasks, EF Planning skills were significantly positively correlated with performance in both Lab-EBPM ($r(31) = .45, p = .01$) and Lab-TBPM tasks ($r(31) = .50, p = .005$). Furthermore, ASD performance in the Lab-EBPM task was also significantly positively correlated with EF Attention/Inhibition ($r(31) = .64, p < .001$) and EF Episodic Memory and Learning ($r(31) = .54, p = .002$). In addition, the ASD group's Lab-TBPM performance was also significantly positively correlated with EF Episodic Memory and Learning ($r(31) = .54, p = .002$). In the naturalistic tasks, the ASD group performance in the Nat-EBPM *other-relevant* task was significantly positively correlated with EF Attention/Inhibition ($r(31) = .56, p = .001$) and EF Episodic Memory and Learning ($r(31) = .54, p = .002$). Although EF Strategy difficulties (higher score) were associated

with poorer Nat-EBPM *other-relevant* performance, this did not reach significance ($r(33) = -.32, p = .066$). Finally, although Nat-TBPM *self-relevant* performance in the ASD group was positively correlated with EF Attention/Inhibition ($r(31) = .34, p = .064$) and EF Episodic Memory & Learning ($r(31) = .32, p = .076$), these associations did not reach significance.

In sum, for TA adults EBPM in the laboratory appeared to be supported by EF for Episodic Memory and Learning, whereas EBPM naturalistic tasks appeared to draw on EF cognitive flexibility. In TBPM, however, whilst no EF associations emerged in the TA group for the laboratory task, both EF Planning and Attention/Inhibition appeared to facilitate naturalistic TBPM performance when remembering involved pro-social motivations. By contrast, the ASD group appeared to draw on a broader range of EF skills across the PM tasks and settings. First, laboratory EBPM performance in the ASD group drew on several EF skills – Planning, Attention/Inhibition, and Episodic Memory and Learning – whilst laboratory TBPM performance also drew on EF Planning skills, as well as Episodic Memory and Learning. In the naturalistic tasks, several EF skills were again associated with successful task completion. Pro-social motivations in the naturalistic EBPM task appeared to draw on EF Attention/Inhibition, as well Episodic Memory and Learning, and to some extent on Strategy skills. Further, these EF skills also appeared to play some role in supporting *self-relevant* TBPM in naturalistic settings. Overall, then, these findings suggest that ASD adults tend to draw on more strategic than automatic cognitive processes in completing PM tasks, compared to TA adults.

Self-reported cognitive failure as predictors of PM ability in laboratory and naturalistic tasks

Regression analyses were carried out to assess which variables were predictors of PM accuracy (EBPM and TBPM; laboratory and naturalistic tasks). The outcome variables were PM accuracy for Lab-EBPM strict accuracy (hits minus false alarms), Lab-TBPM strict accuracy (+/- 20s hits minus false alarms), Nat-EBPM-OR (other-relevant) accuracy, Nat-EBPM-SR (self-relevant) accuracy, Nat-TBPM-OR accuracy, and Nat-TBPM-SR accuracy, respectively. The variables entered into the Regression model were Diagnostic Group, and self-report scores for PRMQ-PM True-score, CFQ Total and ZTPI Future scores. The Regression analyses within Diagnostic Groups also included Age Group and, separately included chronological Age as a continuous variable (Table 7.7, p. 289).

(i) Self-reported cognitive failures as predictors of PM accuracy in laboratory tasks

Overall, Lab-EBPM *accuracy* was not significantly associated with Diagnostic Group ($r = -.12$, $p = .19$), or any of the self-report measures across all indices (PRMQ, $r(57) = -.14$, $p = .17$; CFQ, $r(57) = -.01$, $p = .46$; ZTPI, $r = -.03$, $p = .41$). Further, none of the variables just mentioned were significant predictors of Lab-EBPM *accuracy* ($R^2 = .38$; $F(22,31) < 1.0$, $p = .67$; $B_{\max} = -.263$, $\beta_{\max} = 1.06$, $t_{\max} = -1.40$, $p_{\min} = .17$). Whereas, Lab-TBPM *accuracy* was moderately but significantly associated with ZTPI-Future ($r = -.23$, $p = .047$; $B = -.349$, $SE = .134$, $\beta = -.496$, $t = -2.61$, $p = .014$), but not with any other variables (all $r < .07$, all $p > .32$). None of the variables were significant predictors of Lab-TBPM *accuracy* ($R^2 = .38$; $F(22,32) < 1.0$, $p = .59$; $B_{\max} = -.349$, $\beta_{\max} = -.496$, $t_{\max} = -2.61$, $p_{\min} = .014$).

Within Diagnostic Groups, Lab-EBPM *accuracy* in the TA group was moderately negatively correlated with ZTPI-Future, but this did not reach significance ($p = .065$). There was no significant regression model ($F(1,20) = 2.48$, $p > .10$) in the TA group and neither Age Group nor chronological age were associated with Lab-EBPM *accuracy*; there were also no associations with any of the self-report measures (Table 7.7, p. 289). By contrast, in the ASD group Lab-EBPM *accuracy* was moderately positively correlated with Age Group and chronological age, but again there were no associations between Lab-EBPM *accuracy* and any of the self-report measures, and there was no significant model of predictors ($F(1,30) = 3.25$, $p > .07$). Whereas, Lab-TBPM *accuracy* in the TA group was moderately negatively associated with chronological age but this did not reach significance ($p = .057$). Nevertheless both age and self-reported cognitive failures were predictors of Lab-TBPM *accuracy* in the TA group (Table 7.7, p. 289), although the overall model was not significant ($F(2,19) = 3.06$, $p > .06$). These findings replicate previous literature of PM difficulties in the general population, where it one's subjective awareness of cognitive difficulties is often a greater predictor of PM performance, than objective measures of cognitive impairment (Reese & Cherry, 2006). In the ASD group, Lab-TBPM *accuracy* was significantly negatively associated with ZTPI-Future ($p = .02$), but not with any other variables (Table 7.7, p. 289). Furthermore, ZTPI-Future and Age Group were significant predictors of Lab-TBPM *accuracy* in the overall regression model ($R^2 = .12$; $F(1,31) =$

4.58, $p < .04$). These findings align with the overall patterning of objective PM performance already presented, whereby older autistic adults were more accurate than younger autistic adults in the Lab-TBPM task. Moreover, the significant model of predictors suggests autistic adults who have a greater awareness of their difficulties with future-based time perception, may develop compensatory strategies to support memory for future events in older age.

(ii) Self-reported cognitive failures as predictors of Naturalistic PM performance

Overall, none of the variables (PRMQ, CFQ, ZTPI, Diagnostic Group) were significant predictors of *accuracy* for any of the Naturalistic EBPM or TBPM tasks (Nat-EBPM-OR, $R^2 = .44$; Nat-EBPM-SR, $R^2 = .40$; Nat-TBPM-OR, $R^2 = .46$; Nat-TBPM-SR, $R^2 = .19$; $F_{\max}(22,32) \leq 1.23$, $p_{\min} = .29$). In the Nat-EBPM-OR task, *accuracy* in the TA group was significantly positively correlated with chronological age ($p = .038$), which replicates findings from the typical ageing literature that has found enhanced age-related PM performance in naturalistic settings (Schnitzspahn et al., 2011; Walter & Meier, 2015), and which appears to be associated with better adaptation to the social demands of such tasks (Altgassen et al., 2010). However, none of the self-report measures in the TA group were significant predictors of Nat-EBPM-OR performance, nor was there a significant regression model ($F(1,20) = 3.52$, $p > .07$). In the ASD group, Nat-EBPM-OR performance was not associated with any variables (Table 7.7, p. 289), nor was there a significant regression model ($F(2,30) = 1.81$, $p > .18$).

For the Nat-EBPM-SR task, PM *accuracy* in the TA Group was moderately negatively correlated with ZTPI-Future but this did not reach significance ($p = .089$). No variables predicted Nat-EBPM-SR performance in the regression model (Table 7.7, p. 289; $F(1,20) = 1.95$, $p > .17$). In the ASD group only Age Group and age were positively significantly associated with Nat-EBPM-SR performance (Table 7.7, p. 289). Nevertheless, there was a significant model of predictors associated with PRMQ-PM True-score, ZTPI-Future, CFQ-Total, and age ($R^2 = .29$; $F(4,28) = 2.86$, $p < .05$). Thus, the findings indicate that combined awareness of cognitive difficulties in everyday life were associated with everyday self-relevant EBPM task performance in autistic adults. However, this awareness appeared to facilitate compensatory strategies for PM functioning, particularly in older age.

Nat-TBPM-OR *accuracy* in the TA group was significantly positively correlated with ZTPI-Future ($p = .005$) – the opposite pattern of associations to that seen in the Lab-TBPM task. Here, a significant model of predictors encompassed PRMQ-PM True-score, Age Group, ZTPI-Future and CFQ-Total scores ($R^2 = .39$; $F(3,18) = 3.80$, $p < .03$). However, ZTPI-Future was the strongest predictor of Nat-TBPM-OR accuracy in the TA group, overall, which alone accounted for 29% of the variance in performance ($R^2 = .29$; $F(1,20) = 8.16$, $p < .01$). In the ASD group, Nat-TBPM-OR accuracy was significantly positively correlated with Age Group ($p = .029$) and age ($p = .009$). Here, the significant predictors in the regression model were ZTPI-Future and age ($R^2 = .19$; $F(2,30) = 3.57$, $p < .05$), although age alone accounted for 16.6% of the variance in performance ($R^2 = .17$; $F(1,31) = 6.18$, $p < .02$).

Finally, Nat-TBPM-SR *accuracy* in the TA group was not significantly associated with any of the variables (Table 7.7, p. 289). The regression model indicated that Age Group and age variables were both significant predictors of Nat-TBPM-SR performance in the TA group (Table 7.7, p. 289), in that older TA adults performed better than younger TA adults in this task. Although the overall model did not reach significance in the TA group ($R^2 = .26$; $F(2,19) = 3.42$, $p = .054$), the findings are in line with the typical ageing literature already discussed (e.g. Schnitzspahn et al., 2011; Walter & Meier, 2015). By contrast, in the ASD group there were no variables associated with Nat-TBPM-SR performance (Table 7.7, p. 289) and no significant model of predictors ($R^2 = .047$; $F_{\max}(1,31) = 1.53$, $p_{\min} = .23$).

(iii) Summary of self-reported cognitive failures as predictors of PM performance

In sum, the patterning of PM task performance across laboratory and naturalistic tasks was different between the ASD and TA groups. Whereas, age was negatively associated with Lab-TBPM performance in the TA group, it held a positive association with performance in the ASD group, suggesting age-related difficulties in TA adults and age-related enhancements in ASD adults. Furthermore, age was also positively associated with Lab-EBPM performance in the ASD group, but not in the TA group, suggesting that PM ability may be more broadly enhanced in ageing and ASD compared to typical ageing. Temporal perceptions of future time also showed different patterning in

the ASD and TA groups. Here, a lack of Future time perception was associated with poorer Lab-EBPM but not Lab-TBPM performance in the TA group. Whereas, a greater awareness of Future time perception was associated with better Lab-TBPM but not Lab-EBPM performance in the ASD group. These findings point to the temporal processing differences in ASD and TA adults that were reported earlier in the present chapter, suggest that a greater temporal awareness for Future time in ASD may be related to the profile of age-related Lab-TBPM enhancements observed in this study.

The patterning of associations in the naturalistic tasks was similar, in that age was once again a positively associated with PM performance in the ASD group. However, this was only in the naturalistic TBPM tasks (Nat-TBPM-OR; Nat-TBPM-SR) but not in the naturalistic EBPM tasks (Nat-TBPM-OR; Nat-TBPM-SR). Further, awareness of Future time perception in the ASD group was once again associated with better TBPM performance, this time in the Nat-TBPM-OR task, suggesting that time perception may be social motivated in ageing and ASD. An alternate explanation is that older autistic adults may be more aware of social pressures that inform their time perception for future events.

In the final section of results, the associations between PM ability and QoL are presented in relation to ageing and ASD compared to typical ageing.

Table 7.7. Correlates and predictors of PM accuracy in EBPM and TBPM laboratory and naturalistic tasks

PM task	Outcome variable	Diagnostic Groups											
		ASD (n=35)						TA (n=22)					
		Pearson's <i>r</i>	B	SE	β	<i>t</i>	sig.	Pearson's <i>r</i>	B	SE	β	<i>t</i>	sig.
Lab-EBPM^a	Age Group	.31*	.26	.35	.29	.76	.46	.02	-.15	.29	-.26	-.51	.62
	Age	.32*	<.001	.01	.06	.16	.88	<.001	.01	.01	.35	.64	.53
	PRMQ ^g	.12	.01	.01	.28	1.15	.26	.04	.01	.01	.18	.54	.60
	CFQ ^h	.05	.01	.01	.30	1.22	.23	.11	<.001	.01	.13	.37	.72
	ZTPI ⁱ	.15	.09	.13	.13	.72	.47	-.33 [†]	-.23	.15	-.41	-1.46	.17
Lab-TBPM^b	Age Group	.11	.15	.15	.18	1.00	.32	-.30	.09	.42	.11	.22	.83
	Age	.05	-.01	.01	-.19	-.51	.62	-.35 [†]	-.01	.01	-.42	-2.08	.05 [†]
	PRMQ ^g	-.07	<.001	.01	.09	.37	.72	.21	<.001	.02	.03	.08	.94
	CFQ ^h	.13	-.01	.01	-.19	-.51	.62	-.27	-.01	.01	-.36	-1.77	.09
	ZTPI ⁱ	-.35*	-.23	.11	-.36	-2.14	.04*	<.001	.01	.22	.01	.04	.97
Nat-EBPM-OR^c	Age Group	-.01	-.59	.35	-.60	-1.69	.10	.26	-.53	.46	-.55	-1.16	.26
	Age	.15	.02	.01	.67	1.90	.07 [†]	.39*	.01	.01	.41	1.95	.07 [†]
	PRMQ ^g	-.15	-.01	.01	-.16	-.91	.37	-.11	-.01	.02	-.17	-.79	.44
	CFQ ^h	.18	<.001	.01	.07	.26	.79	-.09	-.01	.01	-.33	-1.02	.32
	ZTPI ⁱ	.16	.11	.13	.15	.83	.41	.01	-.25	.25	-.27	-1.03	.32
Nat-EBPM-SR^d	Age Group	.31*	.02	.01	.42	2.6	.02*	.04	-.09	.40	-.12	-.22	.83
	Age	.40**	-.09	.12	-.12	-.74	.47	-.01	<.001	.01	.18	.33	.75
	PRMQ ^g	.21	.02	.01	.42	2.59	.02*	-.17	-.02	.02	-.31	-.92	.37
	CFQ ^h	.06	.02	.01	.45	2.10	.05*	.06	-.01	.01	-.27	-.77	.45
	ZTPI ⁱ	-.06	.01	.01	.41	1.90	.07 [†]	-.30	-.29	.22	-.38	-1.35	.20
Nat-TBPM-OR^c	Age Group	.33*	.03	.37	.03	.07	.94	-.07	-.51	.40	-.53	-1.27	.22
	Age	.41**	.02	.01	.41	2.49	.02*	.18	.02	.01	.54	1.25	.23
	PRMQ ^g	-.00	.01	.01	.10	.41	.67	.28	.03	.02	.44	1.66	.12
	CFQ ^h	.21	.12	.12	.16	.98	.33	.54**	.52	.18	.54	2.86	.01**
	ZTPI ⁱ	.10	.01	.01	.24	1.00	.33	-.14	.01	.01	.40	1.42	.17
Nat-EBPM-SR^f	Age Group	.07	.45	.38	.46	1.18	.25	-.27	-1.04	.40	-1.04	-2.61	.017*
	Age	-.01	-.01	.01	-.38	-.99	.33	-.01	.03	.01	.89	2.24	.038*
	PRMQ ^g	-.17	<.001	.01	<-.001	-.01	.99	.12	.01	.02	.12	.39	.70
	CFQ ^h	.22	.01	.01	.28	1.08	.29	-.03	<.001	.01	.06	.20	.85
	ZTPI ⁱ	.07	.08	.13	.10	.56	.58	.01	-.27	.25	-.27	-1.08	.30

Notes: [†] $p \leq .07$; * $p < .05$; ** $p < .01$; *** $p < .001$

^a Lab-EBPM = Laboratory task for Event-based Prospective Memory.

^b Lab-TBPM = Laboratory task for Time-based Prospective Memory.

^c Nat-EBPM-OR = Naturalistic task for *other-relevant* Event-based Prospective Memory.

^d Nat-EBPM-SR = Naturalistic task for *self-relevant* Event-based Prospective Memory.

^e Nat-TBPM-OR = Naturalistic task for *other-relevant* Time-based Prospective Memory.

^f Nat-TBPM-SR = Naturalistic task for *self-relevant* Time-based Prospective Memory.

^g PRMQ = Prospective and Retrospective Memory Questionnaire (Smith et al., 2000). Correlates are reported on PM index scores.

^h CFQ = Cognitive Failures Questionnaire (Broadbent et al., 1982). Correlates are reported on Total scores.

ⁱ ZTPI = Zimbardo Time Perception Index (Zimbardo & Boyd, 1999). Correlates are reported on Future index scores.

Prospective memory, Executive function and Quality of Life

The results set out in Table 7.8 (p. 293) present a summary of the correlational and regression analyses for ASD and TA groups, across all PM tasks. The overall patterning of correlations between PM and EF in relation to Overall QoL and Health QoL, showed some similarities and several differences between the TA and ASD groups. Further, the regression analyses highlighted that greater self-reported everyday PM difficulties (PRMQ-PM *True*-scores; lower scores represent greater difficulties) were significantly correlated with Health QoL in both the TA and ASD groups (Table 7.8, p. 293). Furthermore, greater self-reported everyday cognitive failures (CFQ-Total; higher scores represent greater difficulties) were significantly correlated with Health QoL and Overall QoL in both TA and ASD groups. These findings replicate observations from the broader typical ageing literature in that subjective experiences of cognitive difficulties are often strong indicators of actual PM performance (Reese & Cherry, 2006) and everyday functioning for wellbeing (Woods et al., 2012). However, the patterning of PM ability and EF skills as predictors of QoL were not consistent among the Diagnostic Groups.

In the TA group, good EF Planning skills and strong performance in the Lab-TBPM task and Nat-EBPM-SR task were associated with better Overall QoL (Table 7.8, p. 293). Given the small TA group size, the regression model was not significant ($R^2 = .53$; $F(3,8) = 3.05$, $p = .09$), but highlighted significant associations with the variables just mentioned. Health QoL in the TA group was also negatively associated with self-reported everyday cognitive failures but was positively associated with EF-Attention/Inhibition ability (Table 7.8, p. 293). However, the regression model was not significant ($R^2 = .94$; $F(10,3) = 4.90$, $p = .11$). Nevertheless, significant correlations were observed between Health QoL and Lab-TBPM performance, in that greater PM accuracy was associated with better Health QoL. Whereas, everyday PM failures (PRMQ-PM *True*-score) and everyday cognitive failures (CFQ-Total), as well as EF difficulties associated with Attention/Inhibition and Episodic Memory and Learning were significantly correlated with poorer Health QoL. However, only cognitive failures emerged as a predictor of Health QoL, although this did not reach significance ($p = .057$).

In the ASD group, a somewhat different patterning of associations was observed. Better performance in Lab-EBPM was significantly correlated with both Overall QoL and Health QoL (Table

7.8, p. 293). A somewhat surprising finding indicated that ASD adults who had poor Lab-TBPM ($p = .052$) and poor Nat-EBPM-OR ($p = .052$) performance, nonetheless had good Health QoL. However, neither of those PM abilities were associated with Overall QoL, nor were any other naturalistic PM tasks associated with Health or Overall QoL, which suggests that other factors may be involved. Nevertheless, the regression model was significant for Health QoL in ASD ($R^2 = .94$; $F(15,5) = 5.32$, $p < .04$). The significant predictors of good Health QoL in ASD were Lab-EBPM ability, awareness of future time perception and strong EF skills related to Planning, Cognitive Flexibility, Attention/Inhibition, and Episodic Memory and Learning (Table 7.8, p. 293). Regarding Health QoL in ASD, there were several significant regression models and the most significant of these explained 69.5% of the variance in performance ($R^2 = .70$; $F(7,13) = 4.23$, $p < .02$). The significant predictors of poor Overall QoL in ASD were Lab-TBPM difficulties, lower awareness of future time perception, and difficulties in EF Strategy, Planning, and Episodic Memory and Learning skills (Table 7.8, p. 293). The overall patterning of findings indicates that EF function skills may play a greater role in everyday functioning and wellbeing in ASD, than PM difficulties alone.

Table 7.8. PM as correlates and predictors of Quality of Life

Outcome variable	Diagnostic Groups											
	ASD (n=35)						TA (n=22)					
	Pearson's <i>r</i>	B	SE	β	<i>t</i>	sig.	Pearson's <i>r</i>	B	SE	β	<i>t</i>	sig.
Overall QoL												
Age	.20	.32	.32	.22	1.01	.34	-.32	-.47	<.001	-.69	-	-
Lab-EBPM ^a	.54**	34.71	13.68	.47	2.54	.03*	-.32	2.78	<.001	.06	-	-
Lab-TBPM ^b	-.02	-12.71	7.04	-.40	-1.81	.09	.61*	6.76	4.73	.41	1.43	.19
Nat-EBPM-OR ^c	-.13	-4.24	17.47	-.09	-.24	.82	.25	-	-	-	-	-
Nat-EBPM-SR ^d	-.23	-.86	15.00	-.02	-.06	.96	-.52*	-13.42	10.22	-.34	-1.31	.23
Nat-TBPM-OR ^e	.05	-9.92	16.31	-.24	-.61	.57	-.15	2.84	<.001	.13	-	-
Nat-TBPM-SR ^f	-.13	6.68	14.22	.14	.47	.66	-.32	6.93	<.001	.31	-	-
PRMQ ^g	.14	-.91	.47	-.47	-1.96	.08	.31	-1.36	<.001	-.88	-	-
CFQ ^h	-.44*	.40	.63	.27	.64	.54	-.53*	-1.03	<.001	-1.25	-	-
ZTPI ⁱ	.12	-23.75	8.03	-.63	-2.96	.01**	-.22	-	-	-	-	-
EF-Strategy/Working Memory	.08	-1.30	.98	-.36	-1.34	.21	-.06	4.05	<.001	1.63	-	-
EF-Planning	.19	6.17	2.73	.64	2.26	.04*	.57*	.89	1.42	.19	.63	.55
EF-Cognitive flexibility	-.16	.99	.46	.57	2.15	.05*	.21	2.07	<.001	.57	-	-
EF-Attention/Inhibition	.34†	520.05	141.05	1.03	3.69	.003**	.48†	-	-	-	-	-
EF-Episodic Learning/Memory	-.29-.30	-3.71	1.31	-.72	-2.82	.01**	.46	-1.54	<.001	-.45	-	-
Health QoL												
Age	.06	.44	.36	.26	1.23	.28	-.42†	.61	.56	.36	1.08	.36
Lab-EBPM ^a	.41*	41.18	32.33	.47	1.27	.26	-.31	8.68	25.20	.06	.34	.75
Lab-TBPM ^b	-.37†	-22.89	6.84	-.61	-3.35	.02*	.48*	.86	10.90	.02	.08	.94
Nat-EBPM-OR ^c	-.41*	-18.85	12.15	-.33	-1.55	.18	-.40	-	-	-	-	-
Nat-EBPM-SR ^d	-.14	5.50	10.43	.11	.53	.62	-.25	-	-	-	-	-
Nat-TBPM-OR ^e	.16	-9.85	11.34	-.20	-.87	.43	-.28	-12.98	13.35	-.22	-.97	.40
Nat-TBPM-SR ^f	-.14	15.65	9.90	.27	1.58	.18	-.04	-16.44	13.16	-2.8	-1.25	.30
PRMQ ^g	.66***	.63	.56	.27	1.12	.31	.43†	-.33	1.09	-.09	-.30	.78
CFQ ^h	-.56**	-.05	.54	-.03	-.09	.93	-.58*	-1.77	.59	-.94	-3.06	.06†
ZTPI ⁱ	.21	-19.63	8.58	-.44	-2.29	.07†	-.40	-	-	-	-	-
EF-Strategy/Working Memory	-.06	-3.22	1.06	-.76	-3.04	.03*	-.38	-3.43	1.50	-.63	-2.29	.11
EF-Planning	-.12	7.31	2.62	.65	2.79	.04*	.44	-	-	-	-	-
EF-Cognitive flexibility	-.16	1.65	.88	.80	1.87	.12	-.08	-4.79	2.00	-.49	-2.39	.10
EF-Attention/Inhibition	.11	259.26	160.38	.44	1.62	.17	.58*	-	-	-	-	-
EF-Episodic Learning/Memory	-.31	-4.46	1.46	-.74	-3.05	.03*	.44*	4.78	2.47	.52	1.94	.15

Notes: † $p \leq .07$; * $p < .05$; ** $p < .01$; *** $p < .001$

^a Lab-EBPM = Laboratory task for Event-based Prospective Memory.

^b Lab-TBPM = Laboratory task for Time-based Prospective Memory.

^c Nat-EBPM-OR = Naturalistic task for *other-relevant* Event-based Prospective Memory.

^d Nat-EBPM-SR = Naturalistic task for *self-relevant* Event-based Prospective Memory.

^e Nat-TBPM-OR = Naturalistic task for *other-relevant* Time-based Prospective Memory.

^f Nat-TBPM-SR = Naturalistic task for *self-relevant* Time-based Prospective Memory.

^g PRMQ = Prospective and Retrospective Memory Questionnaire (Smith et al., 2000). Correlates are reported on PM index scores.

^h CFQ = Cognitive Failures Questionnaire (Broadbent et al., 1982). Correlates are reported on Total scores.

ⁱ ZTPI = Zimbardo Time Perception Index (Zimbardo & Boyd, 1999). Correlates are reported on Future index scores.

(i) Subjective PM difficulties and cognitive failures as predictors of Quality of Life

Regarding self-reported difficulties in yTA adults, there were no significant associations between everyday PM failures or awareness of future time perception and QoL outcomes. Only everyday cognitive failures was a significant variable, which explained 36.9% of the variance for yTA adults and was a predictor of Overall QoL ($t(8) = -2.78, p < .03; R^2 = .37; F(1,7) = 4.10, p = .083$) but not Health QoL ($t(8) < 1.0, p > .05$). For oTA adults, once again cognitive failures was a significant predictor of Overall QoL ($t(7) = -2.63, p < .05$), and together with everyday PM failures explained around 60% of the variance in Overall QoL for oTA adults ($R^2 = .60; F(1,7) = 4.10, p = .083$). Moreover, cognitive failures on its own accounted for 87% of the variance in Health QoL for oTA adults ($R^2 = .87; F(1,6) = 40.50, p < .001$) and together with PM failures and low awareness of time perception explained around 92% of the variance in Health QoL ($R^2 = .92; F(1,7) = 14.94, p = .012$), although only cognitive failures was a significant predictor ($t(7) = -6.40, p < .001$).

In the yASD group, together the self-report measures explained 53.7% of the variance in Overall QoL ($R^2 = .54; F(3,12) = 4.63, p = .02$), whilst cognitive failures alone explained around 40% of the variance and was the only predictor of Overall QoL ($t(14) = -3.33, p < .007; R^2 = .39; F(1,14) = 9.01, p = .01$). Whereas, for Health QoL, once again all self-report measures contributed to the significant regression model ($R^2 = .69; F(3,12) = 8.81, p = .002$), but everyday PM ability alone explained more than 62% of the variance and was the only predictor of Health QoL in yTA adults ($t(14) = 4.82, p < .001; R^2 = .62; F(1,14) = 23.22, p < .001$). By contrast, for oASD adults, none of the self-reported difficulties were predictors of either Overall QoL ($R^2 = .18; F(3,7) < 1.0, p > .05$) or Health QoL ($R^2 = .36; F(3,7) = 1.29, p > .05$).

(ii) Objective PM difficulties as predictors of Quality of Life

In the yTA group, TBPM ability in both laboratory and naturalistic settings (self- and other-relevant) predicted Overall QoL (all $t(8) > 5.40$, all $p < .03; R^2 = .98; F(6,2) = 14.05, p = .068$) but not Health QoL (all $t(8) < -1.68$, all $p > .05$). Whereas, EBPM ability in both laboratory and naturalistic settings (self- and other-relevant) did not reach significance for Overall QoL (all $t(8) < 3.98$, all $p \geq .06$), or Health QoL (all $t(8) < -1.46$, all $p > .05$) for yTA adults. By contrast, in oTA adults the

regression models were highly significant for Overall QoL associated with TBPM in laboratory and self-relevant naturalistic contexts as well as EBPM in laboratory and other-relevant naturalistic contexts ($R^2 = .996$; $F(6,4) = 116.07$, $p = .009$). Here, the strongest predictor of was Lab-TBPM ability which alone explained 94% of the variance in Overall QoL in oTA adults ($t(6) = 9.10$, $p < .001$; $R^2 = .943$; $F(1,5) = 82.71$, $p < .001$), but not Health QoL (all $t(7) < 2.20$, all $p > .05$). Overall, these findings broadly replicate the previous literature on typical ageing (e.g. Woods et al., 2012).

In the yASD group, EBPM in a self-relevant naturalistic context was the strongest predictor of Overall QoL ($t(15) = -2.83$, $p = .013$; $R^2 = .36$; $F(1,14) = 8.00$, $p = .01$) and together with EBPM in laboratory settings explained around 40% of the variance in Overall QoL in yASD adults ($R^2 = .39$; $F(1,14) = 4.13$, $p = .04$). However, predictors of Health QoL in yASD adults was driven by other-relevant EBPM in naturalistic contexts ($t(15) = -2.68$, $p = .018$), which together with other-relevant TBPM in naturalistic contexts also explained around 40% of the variance in Health QoL in yASD adults ($R^2 = .41$; $F(2,13) = 4.57$, $p = .03$). By contrast, for oASD adults none of the objective measures of PM predicted Overall QoL (all $t(11) < 1.08$, all $p > .05$) or Health QoL (all $t(11) < -2.09$, all $p > .06$). Thus, overall, PM did not predict QoL in the same way in ageing and ASD as it did in typical ageing.

The overall findings, whilst offering interesting associations between potential factors that affect everyday PM and QoL, should be interpreted with caution given the potential limitations of smaller group sizes. Nevertheless, the potential implications of these findings for current understanding of PM in ASD and future directions for this work are considered next, in the Discussion.

7.5 Discussion

The series of tasks presented in the present chapter investigated the profile of PM and ageing in autistic compared to typically ageing individuals. Further, the role of self-relevant and pro-social motivations were explored in relation to limitations on PM performance in ASD, given the potential implications of inherent social difficulties associated with the condition. The nature of social motivations in PM has particular relevance for ageing and ASD, in that health behaviours may be more or less affected by whether an individual is able to carry out PM tasks that involve others but may nonetheless be of personal benefit (e.g. calling the doctor at 2pm). The broader impact of everyday PM slips and errors that are diminished for socially motivated PM, and that may be exacerbated by factors such as the absence of reminders, low task importance, and everyday stress (Ihle, Schnitzspahn, Rendell, Luong & Kliegel, 2011), have been known to lead to social isolation in older age (Brandimonte et al., 1996; Baddeley, 1997; Altgassen et al., 2010). To date, there has been very little research on social motivations in PM and ageing (Brandimonte & Ferrante, 2008; 2015). In ASD, only one prior study explored these factors on PM performance, albeit in a younger sample (Altgassen et al., 2017). Moreover, no prior studies have explored the age-related differences of PM performance between younger and older autistic adults. The work set out in the present chapter addressed this gap. Further, since autistic individuals are known to experience selective difficulties with memory and EF, the present study examined the role of EF in PM functioning to better understand the real-world impacts for autistic adults as they grow older. The key findings from the laboratory and naturalistic tasks carried out in Study 3, are summarised below.

Event-based prospective memory in a laboratory setting

The finding of significantly slower response times for older TA adults compared to younger ones replicated previous literature (Cook, Ball & Brewer, 2014), suggesting that speed-accuracy trade-off was a compensatory factor that enabled older TA adults to successfully complete the PM task and deal with conflicting cognitive demands (Brewer et al., 2011). In the ASD group, the findings showed greater PM task difficulties in younger ASD adults, who had a lower proportion of EBPM hits compared to older ASD adults and all other groups. By contrast, the overall performance older ASD

did not appear to show the age-related PM difficulties that were observed in the older TA adults. Although age-related differences within the ASD group did not reach significance, and there were no significant differences in response times for EBPM actions, the medium effect sizes observed in this task point to the extent of PM difficulties in younger, but not older autistic adults. Possible reasons for this may be that older autistic adults have developed cognitive coping strategies that enable them to deal with competing cognitive demands in everyday life. However, the older ASD adults also made substantially more false alarm responses than all other groups. Nevertheless, the absence of significance in age-related differences in the ASD groups accompanied by the relatively small effect sizes, indicates that the age did not affect PM in the same way for older ASD adults as it did for younger ones.

Regarding the number of commission and omission errors in PM actions, the profile of responses observed in younger ASD and older TA adults, indicate specific difficulties in retrieving content from retrospective memory, which is in line with previous literature in both these groups (Bowler et al., 2010; Smith & Bayen, 2004). Further, the small to medium effect sizes indicate that Diagnostic Group performance was similar for inhibitory control of responses. However, the effect sizes for EBPM hits and misses indicate that vigilance for the PM target was slightly hindered in the younger ASD adults, compared to younger TA and older ASD adults. Moreover, these processes appear to draw on slower, more effortful cognitive processing by younger ASD adults, as observed by the large effect sizes for PM response times (Craik, 1986; Ball & Aschenbrenner, 2018). Furthermore, this method of slow strategic processing, rather than automatically driven responses, were also observed in the older ASD and TA adults. The large effect sizes between Diagnostic Groups and between Age Groups in the TA sample, indicate that the organisation of demands on underlying cognitive functions may present more difficulties for ASD adults overall. This was also the case for the older TA, but not younger TA adults.

Regarding the ongoing task, although there were no age-related performance differences, the older ASD adults were somewhat slower in the ongoing task than were younger ASD adults. Here, the magnitude of effect sizes show that these age-related differences were substantial in both older ASD and older TA adults, compared to younger groups. As previously suggested, these processing

differences indicate that more effortful cognitive processing is fundamental to the way in which older ASD and TA adults can complete everyday EBPM tasks.

Time-based prospective memory in a laboratory setting

The TBPM results showed a more differential pattern. In comparisons of clock check response frequency between Diagnostic Groups, the TA adults checked the clock on average 3 more times (instances) than ASD adults, but this was not a significant. Nevertheless, *time monitoring* behaviours observed in older TA adults were fewer than those of younger TA adults, a patterning that replicated the age-related effects found in the typical PM literature (Einstein & McDaniel, 1990; Rendell & Thomson, 1999; Henry, McLeod, Phillips & Crawford, 2004; Einstein, Richardson, Guynn, Cunfer & McDaniel, 1995; Salthouse, 1996). Furthermore, in the present study, older TA adults showed significantly reduced clock check behaviour compared to younger TA adults. A similar profile was observed in the younger and older ASD participants (refer to Table 7.6, p. 273). Nevertheless, although ASD participants tended to monitor the time less frequently than younger TA adults, there were no age-related differences in *time monitoring* within the ASD group. Finally, age-related differences in PM responses were observed in the TA and ASD groups, but for different reasons. Whereas, older TA adults made fewer of the required PM responses than younger TA adults, the opposite patterning was observed in the ASD group. Here, older ASD adults made more PM actions than all other groups. However, as was explained in the results, increased PM response do not necessarily equate to greater accuracy of responses. Nevertheless, the older ASD adults were also more accurate in their clock speed for elapsed time, and therefore in their accuracy for responding to the TBPM target time, than all other groups, even after adjusting for PM false alarms. The overall patterning of findings suggests that older ASD adults had more robust PM ability than younger ASD, and older TA adults.

Retrospective memory and recognition processes in EBPM and TBPM laboratory tasks

All groups performed similarly across the 240 familiar and 120 novel trials, with performance at chance (~50%) for correct recognition of familiar items and above chance (~72%) for correct dismissal of novel items. All groups had faster response times for novel items. Then, the type of confidence judgements that participants made were explored – either Remember (R = absolute certainty, recognition in context), or Know (K = moderate certainty, familiarity and recollection without context), or Guess (G = minimal certainty, could be recency effect). Overall, ASD adults made more ‘R’ judgements for familiar items than TA adults. Only one younger ASD participant did not identify any familiar items, although they correctly identified all novel items, which may have been related to a response bias or a more pervasive memory difficulty. Additionally, the overall patterning of responses showed a higher proportion of ‘R’ judgements in both ASD and TA groups for familiar items compared to novel items. This was followed by ‘G’, and then ‘K’ responses in both groups. Thus, the overall picture would seem to indicate that, in these tasks, there were not any retrospective memory difficulties underlying the PM performance in either ASD or TA adults.

Summary of laboratory PM performance in ASD

In the present study, younger and older ASD adults performed well, overall, in self-directed TBPM tasks, which reflects a somewhat different picture to the previous ASD literature (e.g. Altgassen et al., 2010; Williams et al., 2014). What is more, whilst older TA adults showed age-related difficulties that were in line with previous literature (e.g. McDaniel & Einstein, 2000; Hering et al., 2015), there were no age-related effects in the ASD group. Moreover, on both EBPM and TBPM tasks, all groups did well in the ongoing task, without costs to the ongoing task as a function of the PM response. However, since the older ASD adults made more PM responses overall (EBPM and TBPM), some of which were to irrelevant cues, suggests that self-regulated inhibitory control may present some difficulties for autistic individuals in older age. Despite this, the older ASD adults were still closest to target times in the TBPM task, than all other groups, even when adjusting for false alarms.

In sum, the overall picture extends the findings from the studies presented in Chapters 4 and 5 of this thesis. The implications of these findings should, however, be interpreted with caution, given the heterogeneity of responses within the ASD group. Previous literature has shown that autistic individuals, at least in younger adulthood, may have greater difficulties in time-related aspects of self-awareness involved in episodic memory (Bowler et al., 2007; Bowler et al., 2011). Autistic difficulties have also been observed for self-reflections, imagining future actions and personal reflection on internal or mental states (Lind & Bowler, 2009; Hurlburt et al., 1994; Williams, 2010; Williams & Happé, 2009, 2010). Future work would need to take these factors into account in exploring the functions that may influence PM ability or difficulty in ASD. Future research that aims to disentangle the mechanisms of self-awareness in relation to prospective memory, may provide more evidence to establish a truer reflection of the cognitive strategies that autistic individuals might use to facilitate prospective memory function in everyday life.

As indicated at the outset of this chapter, PM involves other memory and executive resources. For instance, forming intentions and planning of future actions (binding), attention maintenance and regulation of responses are important to acting at the correct moment in time or event (attention; inhibition). Furthermore, holding information in mind during distractions of unrelated tasks or event (working memory), as well as calling from memory the content and context (retrospective memory) of the to-be-remembered activity, are crucial to acting at the required point (initiation). This collection of cognitive functions involved in PM performance suggests that PM is likely to be the result of the interplay between a network of mechanisms underlying memory and EF processes than a single process. In the present study, the overall findings seem to support this view. This was especially evident in the ASD group. Not only were autistic adults able to perform the PM task as well as, and in some instances better than the TA groups, but they also did not appear to have difficulties in retrieving information in the retrospective recognition memory task. Furthermore, the ASD adults showed none of the age-related difficulties that were observed in the older TA adults, which indicates a dissociation in the cognitive mechanisms used in each group. Furthermore, the associations between EF and PM suggests, not only that different cognitive processes are involved in event-based compared to time-

based PM, but that in ASD these cognitive processes are differentially drawn on, compared to typically ageing individuals.

Social motivation and PM in a naturalistic context

The present study explored the role of social-motivation on PM performance in autistic adults. Given the difficulties associated with social reciprocity in ASD, it was predicted that autistic adults would be less influenced by pro-social motives and, therefore, make fewer PM responses for *other-relevant* tasks than for *self-relevant* tasks. Since social reciprocity and social motivation have broader reaching ecological impacts in ASD (e.g. Gaigg, 2012; Chevallier, 2012), the present study involved separate naturalistic tasks with *other-relevant* and *self-relevant* conditions to assess the role of social motivation in PM performance in ASD. Furthermore, given the sparse literature of PM in adult ASD and the ambiguity of these findings with respect to EBPM and TBPM, separate tasks explored *other-relevant* and *self-relevant* conditions explored in both EBPM and TBPM. The overall picture in TA adults replicated previous literature (Schnitzspahn et al., 2011; Walter & Meier, 2015), with the finding that older TA adults were better at both self-relevant and socially-motivated EBPM and TBPM tasks in naturalistic settings, compared to younger TA adults (and see Altgassen et al., 2010). Moreover, self-reported cognitive failures and everyday PM difficulties were not associated with naturalistic EBPM performance in the TA group, for either self-relevant or other-relevant PM. In the ASD group, older adults made more PM responses than the younger ASD adults, which followed the picture observed in the laboratory tasks. However, in contrast to the TA group, self-reported everyday difficulties in time perception, as well as PM difficulties and cognitive failures were all predictors of self-relevant EBPM performance in naturalistic settings. What is more, in the naturalistic TBPM tasks, future time perception was a significant predictor of socially-motivated TPBM performance, in both TA and ASD groups. These findings have potential implications for the health behaviours of autistic individuals in everyday life, with practical and theoretical relevance.

Prospective Memory and Quality of Life

Once again, the different profiles of PM functioning were observed in relation to QoL. In the present study, self-reported PM difficulties resulting from everyday cognitive failures were a significant predictor of Overall QoL in both younger and older TA adults. What is more, self-reported cognitive failures were a robust predictor of Health QoL in the older TA adults, a finding that aligns with the typical ageing literature (Woods et al., 2012; Rendell et al., 2012). In the ASD group, self-reported cognitive failures predicted Overall QoL in younger ASD adults, whilst self-reported everyday PM difficulties predicted Health QoL. By contrast, there were no PM predictors of QoL in older ASD adults. Thus, although the overall QoL of older ASD adults was significantly poorer than older TA adults, it did not appear to be driven by PM difficulties. Moreover, the strength of overall PM performance observed in older ASD adults suggests that other factors may be involved in QoL. These findings suggest that age-related cognitive functioning may not occur in the same way in older ASD individuals as it does in typically ageing adults (van Heijst & Geurts, 2012; Lever & Geurts, 2015). Together with the findings from the previous Study Chapters (4 and 5), these insights provide important clues to how age-related differences in cognitive strategies and memory processes might operate in ASD.

Limitations, implications and future directions

The limitations of the series of tasks in the present study have already been discussed. Primarily, a key consideration for future work is to attempt to replicate these findings in larger samples. Additionally, future work involving an exploration of individual differences may illuminate some of the specific cognitive mechanisms involved in the PM abilities and difficulties in ASD. Although significant effects were not found in relation to some PM outcomes in the present study, the magnitude of effects sizes may provide a truer reflection of the broader picture of PM ability in ageing and ASD. What is more, smaller effect sizes have been reported in previous studies of PM and ageing in the general population, particularly where naturalistic PM tasks are involved, compared to the effect sizes in more standard laboratory tasks and where the range of possible outcome scores is limited (e.g. van den Berg et al., 2012; Rabin et al., 2014). This was also the pattern of findings in the *self-relevant* and

other-relevant naturalistic PM tasks in the present study. Nevertheless, the overall picture of PM in ASD indicates potentially substantial implications for everyday functioning and wellbeing in older age. For instance, the older ASD adults appeared to self-regulate their time monitoring in TBPM. This ability may provide insights into self-managed healthcare behaviours. Further, the relation between subjective PM complaints and objective PM difficulties and QoL outcomes warrants further exploration. In this regard, future work that includes an exploration of cognitive interventions (e.g. Hering et al., 2015) for memory strategies in everyday life, may attenuate everyday PM failures and improve QoL outcomes in ASD, particularly in older age. Finally, as mentioned at the outset of this study, PM difficulties are known to affect daily living skills (Pirogovsky et al., 2012). The more generalised memory difficulties associated with ASD and daily living skills, already mentioned elsewhere in this thesis, combined with the importance of PM in healthy ageing, raise important questions about how remembering to perform activities in the future may have real-world impacts in ASD. For instance, self-care and health related behaviours, such as remembering to take medication, schedule and attend medical appointments and check-ups, as well as effectively performing various tasks associated with daily living skills, financial independence and regular employment are all affected by PM functioning. Moreover, as already mentioned, many of these domains are known to present particular challenges for autistic individuals across adolescence and early adulthood, to at least middle-age (e.g. Howlin et al., 2003). Thus, a better understanding of the associations between PM and daily living skills in ASD would serve to identify particular supports that may be needed for autistic individuals across adulthood. What is more, an understanding of the influence of social motivational factors in PM may provide insights to challenges associated with social functioning in ASD (Sheppard et al., 2018). Finally, a continued exploration of PM in ASD may be especially relevant to understanding the mechanisms of independent functioning that enable autistic adults to lead fulfilling and rewarding lives.

7.6 Conclusion

To my knowledge, this work is the first to assess the age-related differences in PM functioning and social motivations in ASD, in both laboratory and real-life naturalistic tasks. The findings reported in the present chapter provide, for the first time, new understanding about the specific difficulties and strengths associated with PM in ASD. Moreover, these findings outline the different cognitive mechanisms and strategies that may be involved in supporting PM functioning in ASD, compared to the mechanisms used by typically ageing individuals.

In the final chapter, the General Discussion (Chapter 8) draws together the emerging themes from this prospective memory study in relation to the patterning of findings from the cross-sectional and longitudinal work presented in Chapters 4 and 5, respectively.

Chapter 8: General Discussion

At the outset of this thesis, several questions were raised about the trajectories for ageing autistic individuals. These questions were:

- Is there a steeper risk of cognitive decline at autistic individuals grow older?
- Does the trajectory of cognitive change in ASD ageing mirror that seen in typical ageing?
- Does the cognitive profile of autistic individuals remain stable as they grow older?

The subsequent work, presented in study chapters 4 and 5, addressed those questions in a series of cross-sectional studies, and longitudinally for data collected at two time points (T1, T2), approximately 2.5 years apart. The findings contribute new knowledge and a broader understanding of how the process of growing older affects the cognitive function, mental health and QoL of adults on the autism spectrum.

Study 1 (Chapter 4) involved exploratory information gathering and the cross-sectional analysis of autistic traits, mental health and cognitive functions (memory, EF, language and general intellectual ability), and their relation to QoL. Profiles of abilities and differences were compared between younger and older autistic adults, and typically ageing groups that were matched on age, general intellectual ability and years of formal education.

Study 2 (Chapter 5) presented the second time point of cross-sectional assessments and the patterning of longitudinal changes in ASD and TA groups.

Study 3 (Chapter 7) explored, for the first time, prospective memory (PM) and ageing in ASD and its relation to QoL outcomes. Study 3 involved six PM tasks to assess the mechanisms associated with everyday forgetting (PM failures) as a measure of age-related cognitive decline, in laboratory and naturalistic settings. The PM studies explored the role of social motivations (self-relevant; other-relevant) and their effect on everyday memory in ASD and ageing.

In this final chapter, the emerging themes and key findings of all these studies are drawn together with reference to their contributions to the existing literature on ageing and ASD. To conclude, the strengths and limitations of this research are considered in the context of future directions to build on this work.

8.1 Emerging themes and key findings

Key finding 1: Ageing and cognitive strengths in older autistic adults

The cross-sectional research at T1 (Chapter 4) and T2 (Chapter 5) explored a broad range of cognitive functions including memory, EF, language and general intellectual ability. The findings were consistent across both time points, showing no age-related differences in the cognitive functions of younger and older autistic adults. Furthermore, although the profile of younger autistic adults presented as prematurely cognitively old (Bowler et al., 2004; Bowler, 2007), in that their overall patterning of cognitive function mirrored that observed in older typically ageing adults, this was not seen in the older autistic adults. By contrast, in the typically ageing group, age-related difficulties were observed in the older adults, which replicated findings in the general literature (Zelazo et al., 2004; Cappelletti et al., 2015; Anderson & Craik, 2017). These findings are important for several reasons.

Firstly, the sample was matched on general intellectual ability, years of formal education, and age at the start of the study (T1). Thus, any cognitive differences that were observed could be attributed to that specific cognitive domain, rather than being driven by any underlying intellectual or comprehension difficulties.

Second, a broad range of cognitive assessments was used to assess general intellectual ability (resulting in IQ outcomes for verbal comprehension, perceptual reasoning, working memory, and processing speed), language (for receptive, expressive and general vocabulary), primary and process memory (with outcomes for incremental learning, simple and complex memory), and EF (planning, strategy, cognitive flexibility, attention, episodic memory and learning). To date, few studies have extensively explored such a breadth of functions within the same cohort (Ozonoff et al., 2004; Williams et al., 2006), but have instead focused on specific aspects of a subset of those domains (e.g. cognitive flexibility, or working memory; Geurts et al., 2004; South et al., 2007; Kercood et al., 2014; Powell et al., 2017).

Third, the patterning of differences between younger and older autistic adults reflects a cognitive resilience that appears to be refined with ageing in ASD. Thus, the older autistic adults, who had average to above-average intellectual ability, did not display the same patterning of age-related difficulties seen in typical ageing. Not only did the older autistic adults perform as well as or better

than younger autistic adults, but their performance also compared positively to older typical adults. For instance, difficulties related to processing speed, language, cognitive flexibility, primary memory (simple and complex learning and recall) and process memory (clustering, repetitions, intrusions, source memory) are known to decline steeply in typical older age (Zelazo et al., 2004; Craik & Bialystok, 2006; Mäntylä et al., 2010; McCabe et al., 2010; Anderson & Craik, 2017). This profile of age-related difficulties was not observed in the older ASD adults presented in this thesis. What is more, the older autistic adults showed better memory recall consistency than both younger autistic and older typically ageing adults, as evidenced by the moderate to large effect sizes observed in group comparisons. These findings coincide with previous typical ageing literature, which has shown that older adults have difficulties in accessing information (Craik & Bialystok, 2006). Recent cross-sectional studies with older autistic adults have shown similar findings to those presented in this thesis (Geurts & Vissers, 2012). For instance, Geurts and colleagues observed better cognitive flexibility in older compared to younger autistic adults (Geurts et al., 2014). They also found that older autistic adults performed as well as typically ageing adults in verbal and visual memory (Lever & Geurts, 2016) and working memory tasks (Lever & Geurts, 2015). Similar findings have also been reported in other studies (e.g. South et al., 2007; Powell et al., 2017; Wang et al., 2017). By contrast, some functions such as planning, ordering of information and memory errors related to recall difficulties have been shown to be problematic in autistic adults (Minshew & Goldstein, 2001; Martin et al., 2006; Van den Bergh et al., 2014; Bowler et al., 2016). However, whilst the younger autistic adults studied here showed a similar profile of functioning to that reported in the studies just mentioned, these difficulties did not appear to present greater challenges for the older autistic adults. Overall, the profile of cognitive ability, observed in the T1-T2 longitudinal data, indicates age-neutral trajectories for ageing and ASD.

Key finding 2: Longitudinal cognitive stability and ‘age-neutral’ ageing trajectories in ASD

Older autistic adults did not show the same profile of age-related cognitive difficulties seen in typical ageing. By contrast, younger autistic adults showed comparably more difficulties than younger typical adults in cognitive functions across a range of domains, including general memory (simple and

complex; primary and process mechanisms) and EF (planning, strategy, attention/inhibition, cognitive flexibility, episodic memory/learning). Arguably, although the cognitive profile of autistic individuals starts at a lower baseline of abilities compared to typically developing individuals, any observed cognitive stability with ageing may, nevertheless, indicate the absence of age-related declines. This raises the question of whether the observed profile of age-neutral outcomes in ASD reflects ‘protection’ from age-related declines (Geurts & Vissers, 2012). The picture is likely to be a more complex one, for several reasons. The profile of strengths in older autistic adults, outlined throughout this thesis, suggests that the underlying cognitive mechanisms involved in information processing may follow a different pathway in ASD than in typical development. This view has been suggested in previous studies that have highlighted the functional differences in brain activity in ASD, despite behavioural performance on certain cognitive tasks being equivalent to that of typical individuals (Just et al., 2004; Kana et al., 2006; Braden et al., 2015, 2018). The studies presented in this thesis reflect a potential profile of premature cognitive ageing in younger ASD adults (Bowler et al., 2004), a profile that mirrored the difficulties observed in older typical adults. Nevertheless, the profile of age-neutral cognitive outcomes for older autistic adults suggests that cognitive declines with ageing in ASD may not be a foregone conclusion following early life cognitive difficulties. The longitudinal assessments of cognitive functions in ASD showed age-neutral (stable) outcomes across a range of domains, and age-positive (improvements) outcomes for language ability (Chapter 5). However, age-negative (declines) outcomes were observed for RRBs, which reflected more pronounced difficulties for autistic individuals at T2.

The typical ageing literature has consistently shown age-related declines across various domains (e.g. cognitive flexibility, inhibitory control, language, memory; Park et al., 2001; Craik & Bialystok, 2006; Anderson & Craik, 2017). However, others argue that some cognitive declines can be attenuated by training (e.g. episodic memory; prospective memory; Park et al., 2001; Hering et al., 2015; Chan, Haber, Drew & Park, 2016; and see Cappelletti et al., 2015; Ward et al., 2016). The findings from Study 3 (Chapter 7) showed age-positive outcomes for working memory in typical ageing, whilst all other cognitive domains reflected age-neutral (stable) outcomes. These findings align with the third

key finding and explanation of the profile of ageing and autism demonstrated across the programme of work reported here.

Key finding 3: Prospective memory and everyday functioning in ageing and ASD

The six experimental PM studies presented in Chapter 7 explored time-based and event-based PM in laboratory and naturalistic settings. The naturalistic tasks involved social motivations that were either of importance to the participant (self-relevant) or the researcher (other-relevant). Overall, the findings highlighted age-related difficulties for older typically ageing adults across the PM tasks, whereas older ASD adults did not show the same profile of age-related PM difficulties. In all naturalistic tasks the older ASD adults outperformed both younger ASD and older typical adults. In the laboratory tasks, older ASD adults were more accurate than older typical adults on time-based PM and closer in response times to the PM target (i.e. within 20 seconds). What is more, age was significantly associated with time monitoring difficulties in the typically ageing group, but not in the ASD group. In relation to QoL the findings indicated that, for autistic adults, better PM ability in naturalistic contexts was associated with better health-related QoL. By contrast, for typically ageing adults better PM ability in all contexts was associated with better QoL across all domains (physical, psychological, social, environmental). Thus, for older typical adults the PM difficulties observed here were reflective of broad-spectrum consequences for QoL, but for autistic adults it was health-related outcomes that were affected.

Prior to the present research, the small number of studies that have explored PM in ASD showed that autistic children and young adults have difficulties in time-based PM (Altgassen et al., 2010; Kretschmer et al., 2014; Landsiedel et al., 2017; Sheppard et al., 2018), but not in event-based PM tasks (Williams et al., 2014). Similar difficulties were observed here in the PM performance of younger, but not older autistic adults. Several possible reasons may explain this finding. Firstly, it may be that there are no ‘real’ differences in PM ability in older autistic adults, compared to older typical adults. This suggestion is supported by the profile of age-related difficulties observed in the typically ageing group but not in the ASD group. Second, older autistic adults may be more adept at applying compensatory mechanisms to everyday remembering and tasks that draw on the mechanisms

associated with prospective memory, EF and social strategies. The findings observed in Study 1 (Chapter 4) and Study 2 (Chapter 5) converge to support this argument. Consequently, if PM ability is not problematic in ASD then a similar patterning would be expected across younger autistic individuals, and across abilities on the autism spectrum. Alternately, if PM is a core difficulty associated with ASD then it might be expected that more specific difficulties would emerge, irrespective of age or cognitive ability. Accordingly, future work should aim to consider these factors in autistic adults who represent the range of abilities on the autism spectrum. Third, unlike the majority of previous PM studies in ASD that have explored only event-based or only time-based PM, or that have combined both tasks in a single paradigm (Williams et al., 2014; Sheppard et al., 2018), the work presented here individually assessed time- and event-based PM ability in separate tasks. Whilst one other study has explored social motivations in PM and ASD (Altgassen et al., 2012), the work presented here is the first to assess these factors in both time- and event-based PM in laboratory and real-life naturalistic settings. Moreover, this is the first study to systematically explore PM and ageing in ASD. The findings that emerged from these studies contribute to our understanding of the age-related differences and the cognitive strategies and memory processes used by autistic adults as they grow older. Furthermore, these studies provide new information about the strengths and potential challenges of everyday memory difficulties as autistic individuals in the context of ageing, and their effects on QoL.

Key finding 4: Cognitive resilience with ageing in ASD

The findings from the research presented in this thesis suggest that ageing in ASD is a different kind of ageing than for typical individuals. This notion is aligned with the view of some researchers that autism represents a different intelligence (Mottron et al., 2006; Dawson et al., 2007), which is reflected in a patterning of cognitive strengths and differences across a range of domains (Brosnan et al., 2004; Hill, 2004; Bölte et al., 2007; Boucher, 2012). As already suggest, a possible explanation for the patterning of age-related strengths observed in the studies presented here is that older autistic individuals are more adept at using cognitive coping strategies to deal with everyday problems, based on a lifetime of experience adapting to a complex neurotypical world. By contrast, typically ageing

individuals are learning to adapt to the changes in their cognitive abilities as they grow older (Craik, 1986; Crawford et al., 2003; Anderson & Craik, 2017). Thus, everyday cognitive compensation in ASD would, characteristically, present differently in different contexts (Williams & Bowler, 2014). A feature of compensation would be the ability to ‘hack out’ solutions for tasks that are low in cognitive demands (Lind & Bowler, 2009, p. 930; and see Bowler, 1992). However, performance costs are more pronounced under increasing demands for competing cognitive resources, such as attentional vigilance and self-initiated action monitoring or inhibition. Such costs are frequently observed in PM tasks that involve time monitoring (Altgassen et al., 2012; Williams et al., 2014). These competing cognitive demands also present greater challenges for older typically ageing adults (Einstein & McDaniel, 2000; Craik & Rose, 2012; Hering et al., 2015). However, in the PM findings presented in Chapter 7, the autistic adults used different EF processes (task switching) to complete the ongoing and PM tasks, compared to TA adults (planning, strategy).

This contrast offers a possible explanation for the profile of cognitive strengths observed in the older autistic adults who took part in the cross-sectional (Chapter 4) and longitudinal studies (Chapter 5). Thus, the cognitive strengths across a range of functions demonstrate the degree to which autistic adults may be able to flexibly and continuously adapt to cognitive challenges across their lifespan (Happé et al., 2016). However, the consequence of a lifetime of cognitive coping would appear to be at the cost of mental health difficulties, and which persist across adulthood and into older age. In turn, the prolonged effects of co-existing mental health conditions appear to amplify difficulties in daily living, social isolation and poorer QoL (Chapter 4). This suggestion needs to be explored in future work that attempts to identify factors associated with greater mental health difficulties and their effects on cognitive functions and daily living (Gardiner & Iarocci, 2012; Hong et al., 2016; Happé et al., 2016). Such an exploration might include interventions that target aspects of mental health concerns (e.g. anxiety; Gaigg) that are age, gender and ability-appropriate (Roestorf et al., in press). Further, a central feature of potential interventions should be an evaluation of their efficacy for long-term outcomes (Brugha et al., 2015). A second, alternate approach would be to identify individual differences in cognitive resilience and other factors that may be associated with positive outcomes in older age (Seltzer et al., 2004; LeBlanc, Riley & Goldsmith, 2008; Khanna et al., 2014; Hirvikoski et

al., 2016; Ball et al., 2018). Yet a third approach, which encompasses facets of both these suggestions, is to explore the individual differences in autistic traits and the ability to self-report specific difficulties associated with mental health and daily living (Roestorf, Gaigg, Williams & Bowler, 2018).

Key finding 5: Mental health concerns in ASD

Previous literature has shown that mental health problems in typical ageing, such as those related to chronic health conditions (e.g. diabetes, cardiovascular disease) are significantly associated with poorer health QoL and increased mortality, with greater risks for men than women (Landman et al., 2012). In ASD, several retrospective studies of clinical records have reported increased risk of early mortality and suicidality associated with mental health problems such as anxiety and depression, in particular, and chronic or complex health concerns (e.g. epilepsy; heart disease; Parkinson's disease; Totsika et al., 2010; Croen et al., 2015; Fortuna et al., 2015; Hirvikoski et al., 2016).

In the present research, several mental health concerns were highlighted across the T1 and T2 studies. In particular, anxiety and depression were present in more than half of all autistic adults. Both conditions were significantly associated with poor QoL outcomes. Moreover, mental health concerns were strongly associated with everyday difficulties and depression was the strongest predictor of health and overall QoL in ASD across T1 (Chapter 4) and T2 (Chapter 5). What is more, depression presented particular challenges for autistic women. The underlying causes and exacerbating factors related to these difficulties needs to be explored in future research, along the lines of the suggestions already made (see Key finding 4). Recent studies on gender differences suggest that masking of ASD symptoms by autistic females may underpin more pronounced mental health concerns (Lai et al., 2016; Mandy & Lai, 2017). Moreover, this masking may also explain some of the cognitive and behavioural differences observed in younger autistic individuals, such as difficulties in working memory, processing speed and cognitive flexibility, which present as premature cognitive ageing profiles. However, it is unknown whether these factors continue to present challenges during the course of ageing (Livingston et al., 2018). Thus, these factors need to be explored in future work with broader representation of autistic women in the context of ageing and ASD.

Key finding 6: Decreased quality of life and its correlates

Across both T1 and T2 assessments, QoL was consistently poorer for autistic compared to typically ageing individuals. QoL domains included overall wellbeing, global life satisfaction, health QoL and physical, psychological, social and environmental domains, as well as the level of support received in everyday life. The one exception was Environmental QoL, which was similar between Diagnostic Groups. Overall, older autistic adults reported better Social QoL than younger individuals, and this patterning was upheld longitudinally. However, an interesting observation in the QoL assessments emerged at T2, whereby group differences were no longer evident for Health QoL or level of support received, although the latter was still below average in the ASD group. Despite lower QoL scores for autistic adults across domains, these were not significantly different from older typically ageing adults at T2. In contrast, among younger individuals, the QoL was significantly reduced for autistic compared to typical adults at both time points. There were several predictors of QoL in addition to depression and anxiety. In the typically ageing group, Health QoL was predicted by difficulties related to EF (cognitive flexibility, episodic memory, strategy, planning), as well as mental health related to psychiatric disorders and difficulties in daily living. In the ASD group, EF (cognitive flexibility, attention), predicted overall QoL and Health QoL. What is more, both overall and Health QoL in ASD were predicted by RRBs and mental health associated with anxiety, depression, psychiatric disorders and difficulty in daily living.

What is more, QoL outcomes were also different between groups when exploring PM ability – one of the strongest predictors of QoL in typical ageing (Woods et al., 2012), and in clinical samples (Pirogovsky et al., 2012). The findings from Study 3 (Chapter 7) replicated previous literature in relation to time-based PM. Overall, the findings suggest that it was time monitoring, rather than PM ability *per se*, that was significantly associated with poorer QoL in both groups. In line with previous research, in the present study overall PM ability (as measured by *accuracy*) was also significantly associated with better Social QoL in both older ASD and TA adults (Penningroth & Scott, 2013; Brandimonte & Ferrante, 2015). Further, within the typical ageing group, better time monitoring was significantly associated with better overall wellbeing, and physical, psychological and Social QoL for younger but not older individuals. However, overall PM ability did not appear to be a predictive factor

for QoL in typical ageing. By contrast, in the ASD group time monitoring difficulties were associated with poorer Physical QoL in both younger and older adults, and with overall wellbeing in younger adults. In addition, for older autistic adults PM ability was associated with better Psychological and Social QoL.

These findings offer important insights in relation to everyday remembering, since PM ability is important to maintaining autonomy in older age (Altgassen et al., 2010). Furthermore, it is widely accepted that time monitoring places greater demands on cognitive and executive resources, which are particularly susceptible to age-related decline in typical ageing (Hering et al., 2015). Thus, a potential interpretation of these findings is that PM ability in ASD is associated with better QoL. Alternately, it may be that ability to self-regulate time may be associated with better QoL in ASD. In addition, better self-awareness and the ability to organise experiences and behaviours across time, as the result of better metarepresentational skills (Bowler, Gaigg & Lind, 2011), may underlie the PM ability in autistic adults. Future work that specifically addresses these aspects would also illuminate the broader impact of metarepresentational difficulties on time perception and prospective memory in everyday tasks.

8.2 Longitudinal research, ageing and autism

The majority of research in ASD relies on cross-sectional studies between autistic and typically developed (or other non-autistic) comparison groups. When exploring age-differences, those studies infer age-related ‘changes’ from those cross-sectional *differences* (Raz et al., 2005). However, cross-sectional studies do not account for cohort differences other than age, which may reflect potential differences in cognitive ability (Schaie, 2013; Salthouse, 2014). Thus, it is only in longitudinal evaluations that true *changes* can be observed and reported as such (Salthouse, 2014). Accordingly, the generalisability of the key findings presented throughout this thesis are considered in the context of the broader range of intellectual abilities and differences across the autism spectrum. Given that the sample included here represented a group of adults with higher cognitive ability, it is important to consider how these findings align with studies of older autistic adults with intellectual difficulties. Howlin and colleagues conducted a 40-year longitudinal study of autistic individuals who were

diagnosed with ASD in childhood (Howlin et al., 2004; 2013). Their findings showed a similar profile of age-neutral (autistic traits) or age-positive outcomes (IQ; language; social skills) for majority of autistic adults. Nevertheless, a significant sub-group of approximately twenty-five percent of individuals diagnosed in childhood, were not able to undergo the basic cognitive assessments. Consequently, a challenge in conducting research with older autistic adults is that those who take part, necessarily, are those who are more able to do so. The individuals involved in the studies presented in this thesis, represent a group of highly motivated, highly cognitive able individuals. What is more, many of those individuals were able to live at least semi-independent to independent lives. Thus, the sample included here are similar to the 75% of individuals in Howlin et al.'s (2003; 2013) work who achieved 'good' outcomes in their 40-year longitudinal study. However, the individuals involved in the present research, unfortunately, do not represent those who are less cognitively able, or those individuals who face greater challenges in everyday living.

Retention, attrition and generalisability of longitudinal findings

Helles and colleagues conducted a three-stage longitudinal follow up of children diagnosed with ASD (Helles, Gillberg, Gillberg and Billstedt, 2015), reporting changes in diagnostic profiles at T1 and T2 follow-ups. Overall, less than half their original sample (47%) were able to complete both longitudinal assessments (i.e. T0-T1 and T1-T2). At T1 the attrition rate was 24% (76% retention T0-T1) and by T2 this had reduced by a further 38% (61.8% retention T1-T2; Helles et al., 2015). Nevertheless, their findings showed stable trajectories into adulthood for ASD symptoms, which was underpinned by a higher degree of core autistic traits in younger autistic individuals (childhood and adolescence). In the context of growing older, given the patterning of findings observed in the studies present in this thesis, one might expect that as the younger ASD adults transition into older age, that the core features of ASD would remain stable. This, broadly, reflects the pattern of findings in the T2 longitudinal study. Overall, core autistic traits related to social communication difficulties were age-neutral in both younger and older autistic adults, reflecting stable trajectories across the adult lifespan. However, an age-negative trend was observed for restricted interests and repetitive behaviours (RRBs), which increased between T1 and T2. The findings reported here also confirm those of

previous longitudinal studies (Helles et al., 2015; Howlin et al., 2013), in that greater severity of core autism symptoms continues to present difficulties across the lifespan. For instance, Howlin et al. (2013) found that the majority of individuals in their longitudinal study had stable ASD profiles, but also had improved social skills, and therefore age-positive outcomes for some individuals. However, despite those gains, the overall outcomes in that cohort were poor. Moreover, significant declines were observed in one-quarter or their original sample who were untestable at follow-up in adulthood. The present programme of work included a sample of the highly motivated and cognitively able individuals who were able to take part in this research. In this cohort, approximately 70% retention was maintained at T2 follow-up. Whilst this group may not be representative of the wider autistic community, the key findings and themes presented in the present chapter outline the profile of strengths and challenges associated with ageing in this group of autistic individuals. Nevertheless, it is important to recognise that the findings may not be replicated in the significant sub-groups of individuals who are not able to take part in research, for reasons already discussed (Chapter 2, p. 71). Accordingly, it is important for future work to involve representative samples that address the issues related to generalisability of findings across the range of intellectual ability and ages, and across the autism spectrum (Roestorf, Bowler, Deserno, Howlin, Klinger ... & Geurts, in press).

Limitations, future directions and implications for ageing and ASD research and practice

Picking up on the last point, a challenge in conducting research with older autistic adults is that those who take part, necessarily, are individuals who are more able to do so. Indeed, the World Health Organisation (WHO, 2002) states that:

“...broad social policies based on chronological age alone can be discriminatory and counterproductive to wellbeing in older age.” (Active Ageing: A Policy Framework, p. 4).

Many of the participants in the present set of studies were highly motivated individuals with average to above average cognitive ability, who were able to live at least semi-independent to independent lives (Chapter 4). Thus, this sample, on the face of it, mirrors the samples of previous studies with older autistic adults (Geurts & Vissers, 2012; Howlin et al 2004; 2013; Lever & Geurts

2015; 2016) – thus, akin to the 75% of individuals who achieved ‘good’ outcomes in the 40-year longitudinal study by Howlin and colleagues (2004; 2013). However, there are some important differences between these cohorts. Firstly, Howlin’s sample included individuals who were diagnosed in childhood, and who were followed up at multiple time points into young adulthood and middle-age. The present sample involved a community cohort of individuals, many of whom had been diagnosed with ASD later in life. Moreover, age of diagnoses ranged from childhood to older age, and diagnoses were obtained from a broad range of assessments (Chapter 3). Second, the average intellectual ability of Howlin’s sample, although within the normal range of IQ (70-130) had a mean IQ of 80, whereas the sample included in the longitudinal work presented here, had above average intellectual ability with a mean IQ of 117 (Chapter 4). Accordingly, the present sample is similar to participants in the studies by Geurts and colleagues (Geurts & Vissers, 2012; Lever & Geurts, 2015). However, another important difference is that the present sample is a community based group of individuals, who do not necessarily have access to clinical supports, despite the extent of mental health difficulties (Chapters 4 and 5). Whereas, both Geurts’ and Howlin’s samples were clinical cohorts and as such, they may present with a different profile of underlying difficulties compared with participants involved in the present research. Thus, it is acknowledged that these individuals may not be representative of the wider autistic community or of others who have greater cognitive or intellectual difficulties or more profound challenges in everyday functioning. Furthermore, it was not possible to statistically explore gender differences in these studies owing to small sample sizes. However, indications at T1 suggested that gender differences might account for some of the Diagnostic Group and Age Group differences observed in language, processing speed and memory. This suggestion converges with recent studies by Happé and colleagues, which highlighted that men and women may compensate for cognitive difficulties in different ways, including masking of autism related difficulties (e.g. social communication), leading to adverse mental health impacts, such as increased stress, anxiety and depression (Lai et al., 2016; Livingston et al., 2018; and see Baldwin & Costa, 2015). In the context of the studies presented here, the moderate to large effect sizes for gender differences in specific cognitive domains, such as processing speed and language, bears consideration for future work on ageing and ASD to include a systematic exploration of gender differences.

The work presented in this thesis outlines the strengths and challenges facing cognitive able individuals on the autism spectrum. The observations of cognitive functions, mental health conditions and QoL provide clues to what can be expected for autistic adults of average intellectual ability as they grow older. Moreover, the studies have highlighted the significant mental health difficulties for younger and older autistic adults, compared to typically ageing individuals, despite the observed cognitive strengths and differences (Chapters 4 and 5). These findings have also highlighted the consequences of co-existing medical conditions in ASD for everyday functioning and QoL (Chapter 4). Further, the findings raise important considerations regarding therapeutic and treatment care plans, that are appropriate to the age, gender and ability differences in autistic adults across the adult lifespan and across the autism spectrum (Roestorf et al., in press). Consequently, future research needs to consider the individual differences in positive ageing in ASD. For instance, what factors are associated with increased depression in autistic women? What are the long-term effects of multiple pharmacological treatments on health and wellbeing, such as side-effects and cognitive declines associated with specific medications? How can social anxiety and isolation be alleviated for autistic adults who live alone? What is the association between autistic traits and adaptive skills required for everyday functioning in older age? Can prospective memory training alleviate the difficulties that autistic adults experience in everyday living? These are just a few of the questions that need to be addressed in future research. In order to achieve this, a broader understanding is needed about the daily needs and difficulties experienced by older autistic adults across the lifespan (Duncan & Bishop, 2013; Wright et al., 2016).

8.3 Concluding remarks

Programme review

Prior to this thesis, few papers had addressed the gap in evidence-based knowledge about cognitive functioning and wellbeing in older cohorts and the potential consequences for ageing with ASD (Geurts & Vissers, 2012; Howlin et al., 2013; 2014; Lever & Geurts, 2015; Bishop-Fitzpatrick et al., 2016; Roestorf & Bowler, 2016; Powell et al., 2017; Braden et al., 2017; Wise et al., 2018). Moreover, little previous research had addressed the factors associated with the provision of services and supports needed across the adult lifespan (Howlin et al., 2013; Parr, 2016). For instance, health care practitioners and services continue to be unaware of how to support autistic adults as they grow older, with respect to changing ASD symptoms, cognitive functioning, mental health and wellbeing, and age-related physical health (Happé et al., 2016). Clinicians who specialise in ASD tend to focus on children and adolescents and few are familiar with geriatric care and the needs of older autistic adults. Gerontology clinicians who work with older individuals are, largely, unfamiliar with the clinical presentation and needs of autistic individuals (Hategan et al., 2017).

In a policy report, the UK Government set out several areas to improve awareness of autism and identified key areas that need to be addressed in supporting autistic individual across the lifespan. That report stated:

“The HM Government vision is that all adults with autism are able to live fulfilling and rewarding lives within a society that accepts and understands them; they can get a diagnosis and access support if they need it, and they can depend on mainstream public services to treat them fairly as individuals, helping them make the most of their talents (p. 6) [and] improving access for adults with autism to the services and support they need to live independently within the community (p. 14)” (HM Government, 2010, p. ; Fulfilling and rewarding lives: The strategy for adults with autism in England).

The research presented in this thesis has attempted to address this gap in knowledge, by contributing new knowledge and understanding about the issues related ageing and ASD. The findings from all three studies indicated that autism is a different kind of ageing. Older autistic adults demonstrated cognitive strengths compared to younger autistic and older typically ageing adults, across a range of cognitive domains. However, although older autistic individuals appear to have developed a cognitive resilience from a lifetime of coping with the challenges of functioning in a neurotypical world, this appears to be at the consequence of mental health and QoL. For instance, although younger autistic individuals presented a significant degree of mental health difficulties associated with anxiety and depression, these did not improve with age but were, however, a significant predictor of poorer QoL in older age. Thus, findings from the current research indicate the need for future work to address the specific mental health needs of autistic adults as they grow older. Overall, the findings from the work reported here contribute new understanding to the challenges and benefits of growing older with ASD and provide evidenced based pointers to the specific supports needed to enable autistic individuals to lead fulfilling and rewarding lives.

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Appendix 1

Table 5.0. Time 2 Supplementary Data for Cross-sectional Missing Variable Analysis.

Measure	Diagnostic Groups (N=62) ^a ASD (n=39); TA (n=23)	Statistics		
		ANOVA <i>F</i> (1,54)	<i>p</i> .	η^2_p
Age (years) ^b	Diagnostic Group	<1.0	<i>n.s.</i>	.006
	Age Group	114.66	<.001	.680
	Diagnostic Group x Age Group x Gender	4.94	.03	.084
<i>Autistic traits^f</i>				
SRS-2 Total	Diagnostic Group	26.89	<.001	.332
	Diagnostic Group x Gender	4.31	.043	.074
SRS-2 SCI	Diagnostic Group	26.05	<.001	.325
	Diagnostic Group x Gender	4.28	.043	.073
SRS-2 RRB	Diagnostic Group	25.76	<.001	.323
<i>General intellectual ability^e</i>				
FSIQ	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
VCI	Diagnostic Group	<1.0	<i>n.s.</i>	.011
PRI	Diagnostic Group	<1.0	<i>n.s.</i>	.001
WMI	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
PSI	Diagnostic Group	<1.0	<i>n.s.</i>	.002
	Diagnostic Group x Age Group x Gender	5.23	.026	.088
<i>Language^g</i>				
Receptive (RL)	Diagnostic Group	1.96	<i>n.s.</i>	.035
	Age Group	5.28	.026	.089
	Diagnostic Group x Age Group	5.00	.029	.085
Expressive (EL)	Diagnostic Group	<1.0	<i>n.s.</i>	.008
	Gender	8.91	.004	.142
	Diagnostic Group x Age Group	9.21	.004	.146
Difference (RL-EL)	Diagnostic Group	<1.0	<i>n.s.</i>	.008
	Gender	8.53	.005	.136
	Diagnostic Group x Age Group	9.02	.004	.143
<i>Memory^h</i>				
<u>Primary</u>				
Trial 1-5	Diagnostic Group	<1.0	<i>n.s.</i>	.002
	Gender	11.10	.002	.171
SDFR	Diagnostic Group	1.40	<i>n.s.</i>	.025
	Gender	10.75	.002	.166
	Diagnostic Group x Age Group	4.64	.036	.079
SDCR	Diagnostic Group	7.26	.009	.118
	Gender	4.08	.048	.070
	Diagnostic Group x Age Group	8.82	.004	.140
LDFR	Diagnostic Group	1.08	<i>n.s.</i>	.020
	Gender	7.57	.008	.123
LDCR	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
	Gender	9.53	.003	.150
	Diagnostic Group x Age Group x Gender	5.28	.025	.089
Recall (<i>d'</i>) Trial 1-5	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
	Gender	14.23	<.001	.209
<u>Process</u>				
Trial 1	Diagnostic Group	1.35	<i>n.s.</i>	.024
	Gender	15.69	<.001	.225
Trial 5	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
Learning slope Trials 1-5	Diagnostic Group	1.86	<i>n.s.</i>	.033
Recall consistency %	Diagnostic Group	<1.0	<i>n.s.</i>	.015
	Age Group	9.73	.003	.153
	Gender	7.02	.011	.115

continued

Measure	Diagnostic Groups (N=62) ^a ASD (n=39); TA (n=23)	Statistics		
		ANOVA <i>F</i> (1,54)	<i>p</i> .	η^2_p
Memory^h				
Process				
Semantic clustering	Diagnostic Group	<1.0	<i>n.s.</i>	.003
	Gender	11.97	.001	.181
Total repetitions	Diagnostic Group x Age Group	3.86	.055	.067
	Diagnostic Group x Age Group x Gender	6.15	.016	.102
Total intrusions	Diagnostic Group	<1.0	<i>n.s.</i>	<.001
	Gender	10.42	.002	.162
Source (<i>d'</i>)	Diagnostic Group	1.38	<i>n.s.</i>	.025
	Gender	13.63	.001	.202
2AFC recog. %	Diagnostic Group	1.31	<i>n.s.</i>	.024
	Gender	5.13	.028	.087
Mental healthⁱ				
Anxiety	Diagnostic Group	15.87	<.001	.227
Levels Anxiety	Diagnostic Group	21.07	<.001	.281
Depression	Diagnostic Group	4.64	.036	.079
	Age Group	5.21	.029	.085
	Gender	10.80	.002	.167
	Diagnostic Group x Gender	5.32	.025	.090
Levels Depression	Diagnostic Group	28.43	<.001	.345
Somatoform dis.	Diagnostic Group	16.03	<.001	.229
	Diagnostic Group x Age Group	3.95	.052	.068
	Age Group x Gender	5.23	.026	.089
	Diagnostic Group x Age Group x Gender	4.75	.034	.081
Depressive syn.	Diagnostic Group	1.93	<i>n.s.</i>	.034
	Gender	10.93	.002	.168
	Diagnostic Group x Age Group	8.81	.004	.140
	Diagnostic Group x Gender	5.82	.019	.097
Anxiety syn.	Diagnostic Group x Age Group x Gender	5.80	.019	.097
	Diagnostic Group	<1.0	<i>n.s.</i>	.016
	Diagnostic Group	13.30	.001	.198
	Diagnostic Group	15.87	<.001	.227
Binge eating dis.	Diagnostic Group	21.07	<.001	.281
Alcohol abuse	Diagnostic Group	21.07	<.001	.281
Diff. Daily Living	Diagnostic Group	21.07	<.001	.281
	Diagnostic Group x Age Group	5.00	.029	.085
QoL^j & SWB^k				
SWB overall ^k	Diagnostic Group	15.36	<.001	.221
GLS	Diagnostic Group	18.24	<.001	.252
Overall QoL ^j	Diagnostic Group	9.52	.003	.150
Health	Diagnostic Group	7.29	.009	.119
	Diagnostic Group x Age Group x Gender	5.79	.020	.097
	Diagnostic Group	21.14	<.001	.281
	Age Group x Gender	4.59	.037	.078
Physical	Diagnostic Group x Age Group x Gender	4.01	.050	.069
	Diagnostic Group	22.08	<.001	.290
	Diagnostic Group x Age Group	4.49	.039	.077
	Diagnostic Group x Age Group x Gender	4.06	.049	.070
Psychological	Diagnostic Group	16.91	<.001	.238
	Diagnostic Group x Age Group	12.65	.001	.190
	Diagnostic Group	11.11	.002	.171
	Diagnostic Group	10.02	.003	.157
Social	Age Group	5.17	.027	.087
	Diagnostic Group x Age Group	4.64	.036	.079
	Age Group x Gender	5.98	.018	.100
	Age Group x Gender	5.98	.018	.100

Notes: Main effects and interaction effects shown above for MVA analysis on imputed data with no missing values.

Appendix 2

Table 5.4a. Reliable Change Index (RCI) analysis for test-retest scores across T1 to T2 assessments.

	ASD										TA													
	Time 1 (n=52)		Time 2 (n=36)		M _{diff}	SE _{diff}	CI 90%			CI 95%			Time 1 (n=35)		Time 2 (n=21)		M _{diff}	SE _{diff}	CI 90%			CI 95%		
	Mean	SD	Mean	SD			lower	upper		lower	upper		Mean	SD	Mean	SD			lower	upper		lower	upper	
<i>Autistic traits</i>																								
SRS Total	69.87	12.06	71.91	11.50	0.133	0.091	-0.016	0.282	stable	-0.044	0.310	stable	46.33	5.23	46.25	4.91	0.042	0.081	-0.09	0.175	stable	-0.117	0.201	stable
SRS-SCI	69.16	11.96	71.94	14.15	0.203	0.091	0.052	0.353	stable	0.024	0.382	stable	46.55	5.85	46.56	4.87	0.047	0.080	-0.08	0.178	stable	-0.109	0.203	stable
SRS-RRB	69.91	12.34	73.42	11.49	0.142	0.081	0.009	0.276	stable	-0.016	0.301	stable	46.21	4.64	46.25	5.30	0.031	0.076	-0.09	0.157	stable	-0.119	0.181	stable
<i>General intellectual ability</i>																								
FSIQ	113.31	15.97	114.72	17.32	-0.023	0.085	-0.162	0.117	stable	-0.189	0.144	stable	110.14	13.73	112.05	15.86	-0.041	0.084	-0.18	0.096	stable	-0.205	0.122	stable
VCI	115.59	15.62	114.75	13.95	-0.113	0.100	-0.276	0.051	stable	-0.308	0.083	stable	110.57	13.05	112.05	11.70	-0.147	0.111	-0.33	0.036	stable	-0.364	0.071	stable
PRI	111.51	16.24	112.75	17.36	-0.049	0.100	-0.214	0.116	stable	-0.246	0.147	stable	107.23	15.28	111.29	15.93	0.159	0.147	-0.08	0.401	stable	-0.130	0.448	stable
WMI	91.57	15.04	113.56	17.46	2.126	0.408	1.454	2.798	stable	1.325	2.926	stable	97.09	15.46	106.43	18.08	0.553	0.204	0.22	0.889	stable	0.153	0.954	stable
PSI	98.23	19.27	104.06	17.12	0.953	0.359	0.362	1.543	stable	0.249	1.657	stable	102.94	13.93	104.81	17.18	0.216	0.140	-0.01	0.446	stable	-0.059	0.490	stable
<i>Language</i>																								
Receptive	93.75	11.50	94.40	14.27	-0.005	0.019	-0.036	0.027	stable	-0.042	0.033	stable	95.21	7.87	97.00	7.40	0.064	0.011	0.05	0.083	stable	0.042	0.086	stable
Expressive	103.44	11.86	102.90	11.71	0.076	0.162	-0.190	0.341	stable	-0.241	0.392	stable	102.34	10.16	105.21	7.20	0.071	0.176	-0.22	0.362	stable	-0.274	0.417	stable
General Vocabulary	98.22	11.17	98.75	2.94	0.005	0.018	-0.025	0.034	stable	-0.030	0.039	stable	98.31	7.76	100.79	7.11	-0.006	0.015	-0.03	0.018	stable	-0.035	0.023	stable
<i>Memory</i>																								
Incremental learning	49.00	11.22	52.91	11.68	0.211	0.097	0.052	0.370	stable	0.022	0.401	stable	53.97	10.68	56.50	9.98	0.188	0.142	-0.05	0.422	stable	-0.091	0.467	stable
Simple	10.54	3.85	11.17	3.97	0.000	0.113	-0.186	0.186	stable	-0.221	0.221	stable	12.61	2.63	11.60	4.74	-0.338	0.360	-0.93	0.253	stable	-1.043	0.367	stable
Complex	11.32	4.26	12.73	3.12	-0.078	0.089	-0.223	0.068	stable	-0.251	0.096	stable	13.06	2.32	13.50	2.42	0.071	0.144	-0.17	0.308	stable	-0.212	0.354	stable
<i>Mental health</i>																								
Anxiety	13.72	8.78	14.42	10.75	0.029	0.200	-0.300	0.357	stable	-0.363	0.420	stable	5.18	5.84	5.58	6.21	0.038	0.170	-0.24	0.317	stable	-0.295	0.370	stable
Anxiety level	2.17	0.94	2.19	1.17	-0.069	0.203	-0.402	0.265	stable	-0.466	0.329	stable	1.09	0.79	1.42	0.88	0.310	0.207	-0.03	0.650	stable	-0.096	0.715	stable
Depression	16.76	13.62	16.00	11.66	-0.016	0.090	-0.164	0.132	stable	-0.192	0.160	stable	4.76	4.27	6.59	6.12	0.115	0.070	0.00	0.231	stable	-0.022	0.253	stable
Depression level	1.67	1.81	1.55	1.59	0.145	0.162	-0.122	0.411	stable	-0.173	0.462	stable	0.09	0.29	0.35	0.79	0.550	0.225	0.18	0.919	stable	0.110	0.990	stable
<i>Wellbeing / Quality of life</i>																								
Subjective wellbeing	38.37	12.58	38.90	13.41	0.016	0.138	-0.211	0.244	stable	-0.255	0.287	stable	52.39	9.00	50.38	10.13	-0.160	0.059	-0.26	-0.063	stable	-0.275	-0.045	stable
Global life satisfaction	52.39	21.83	54.14	19.91	0.157	0.163	-0.111	0.425	stable	-0.162	0.476	stable	74.55	12.27	75.63	12.63	0.058	0.083	-0.08	0.193	stable	-0.105	0.220	stable
Overall QoL	59.09	20.94	60.00	20.34	0.159	0.148	-0.085	0.403	stable	-0.132	0.450	stable	80.47	15.21	77.78	14.57	0.000	0.136	-0.22	0.224	stable	-0.267	0.267	stable
Health QoL	52.27	27.92	52.50	24.87	0.089	0.157	-0.170	0.348	stable	-0.219	0.397	stable	70.31	22.39	66.67	28.44	0.050	0.114	-0.14	0.238	stable	-0.174	0.274	stable
Physical QoL	60.20	18.30	58.33	16.63	-0.156	0.105	-0.329	0.018	stable	-0.362	0.051	stable	82.00	14.81	79.00	18.53	-0.152	0.133	-0.37	0.066	stable	-0.413	0.108	stable
Psychological QoL	54.75	18.90	52.17	16.62	-0.019	0.114	-0.207	0.169	stable	-0.243	0.205	stable	71.03	14.12	72.33	15.35	0.082	0.093	-0.07	0.236	stable	-0.101	0.265	stable
Social QoL	47.98	19.06	44.40	19.45	0.027	0.130	-0.186	0.240	stable	-0.227	0.281	stable	68.56	20.04	70.89	17.71	-0.036	0.184	-0.34	0.267	stable	-0.396	0.325	stable
Environmental QoL	65.16	16.55	63.83	16.04	-0.040	0.131	-0.255	0.176	stable	-0.296	0.217	stable	76.09	13.33	75.83	17.09	0.007	0.185	-0.30	0.311	stable	-0.356	0.369	stable

Notes: Using the RCI formula indicated in Analysis Strategy (p. 181), the RCI were manually calculated and re-checked for consistency. Lower and upper Confidence Intervals (CI) reported for 90% and 95% CI, with RCI calculated for each CI and each variable, respectively.

Table 5.4b. Change scores using Standard Deviation (SD) analysis for test-retest scores across T1 to T2 assessments.

ASD (n=33)												TA (n=18)														
Time 1		Time 2		M _{diff}	SE _{diff}	Statistics		CI 90%		CI 95%			Time 1		Time 2		M _{diff}	SE _{diff}	Statistics		CI 90%		CI 95%			
Mean	SD	Mean	SD			t	p	lower	upper	lower	upper		Mean	SD	Mean	SD			t	p	lower	upper	lower	upper		
Autistic traits						≤1.76	≥.088							≤1.91						≥.079						
SRS Total	70.21	11.70	71.97	11.50	1.76	1.00		n.s.	0.12	3.40	-.20	3.71		46.22	5.07	46.61	4.73	.39	.95		n.s.	-1.17	1.95	-1.47	2.25	
SRS-SCI	69.21	11.62	70.82	11.48	1.61	1.09		n.s.	-0.19	3.40	-.54	3.75		46.39	5.17	46.89	4.70	.50	.97		n.s.	-1.10	2.10	-1.41	2.41	
SRS-RRB	71.00	11.36	73.42	11.49	2.42	1.09	2.22	.03	0.63	4.22	.28	4.57	worse	46.06	5.41	46.61	5.16	.56	.95		n.s.	-1.01	2.13	-1.31	2.43	
General intellectual ability																										
FSIQ	113.81	15.78	112.97	17.26	-.37	1.36		n.s.	-2.60	1.86	-3.03	2.29		112.71	13.76	112.05	15.86	-.67	1.33		n.s.	-2.86	1.53	-3.28	1.95	
VCI	116.55	13.60	114.00	14.35	-1.76	1.56		n.s.	-4.32	.79	-4.81	1.28		114.33	11.08	112.05	11.70	-2.29	1.74		n.s.	-5.14	.57	-5.69	1.12	
PRI	112.29	17.61	111.03	17.80	-.79	1.63		n.s.	-3.47	1.89	-3.98	2.40		108.71	15.06	111.29	15.33	2.57	2.40		n.s.	-1.37	6.51	-2.13	7.27	
WMI	91.68	14.05	111.39	16.58	20.28	2.64	7.70	<.001	15.95	24.62	15.12	25.45	better	98.10	17.20	106.43	18.08	8.33	3.08	2.71	.01	3.27	13.39	2.31	14.36	better
PSI	99.52	20.10	102.39	16.55	2.87	2.41		n.s.	-1.09	6.83	-1.84	7.59		100.67	15.31	104.81	17.18	4.14	2.70		n.s.	-.29	8.58	-1.14	9.43	
Language																										
Receptive	93.50	11.96	94.40	14.27	.90	1.92		n.s.	-2.25	4.05	-2.86	4.66		96.14	4.77	97.00	7.41	.86	2.09		n.s.	-2.58	4.29	-3.24	4.95	
Expressive	103.80	13.36	102.90	11.71	-.90	1.91		n.s.	-4.04	2.24	-4.64	2.84		98.57	9.17	105.21	7.20	6.64	2.54	2.61	0.02	2.46	1.83	1.66	11.63	better
General Vocabulary	98.30	12.18	98.75	12.94	.45	1.65		n.s.	-2.27	3.17	-2.79	3.69		96.71	5.74	100.79	7.11	4.07	2.13		n.s.	.56	7.58	-.11	8.25	
Memory																										
Incremental learn.	49.25	11.65	51.63	13.05	2.38	1.08	2.19	0.04	.59	4.16	.25	4.50	better	54.40	7.56	56.50	9.98	2.10	1.60		n.s.	-.52	4.72	-1.03	5.23	
Simple	10.75	3.84	10.75	4.41	<.001	.43		n.s.	-.71	.71	-.85	.85		12.90	2.08	11.60	4.74	-1.30	1.38		n.s.	-3.58	.98	-4.01	1.41	
Complex	11.83	3.97	11.50	4.09	-.33	.38		n.s.	-.96	.29	-1.08	.41		13.20	2.10	13.50	2.42	.30	.62		n.s.	-.71	1.31	-.91	1.51	
Wellbeing / Quality of life																										
Subjective wellbeing	41.12	10.30	39.65	11.33	-.71	1.53		n.s.	-3.23	1.80	-3.71	2.28		52.38	8.18	50.38	10.13	-2.00	.74	-2.72	0.02	-3.21	-.79	-3.44	-.56	worse
Global life satisfaction	54.23	20.82	55.00	17.94	2.50	3.55		n.s.	-3.34	8.340	-4.46	9.46		74.38	9.64	75.63	12.63	1.25	1.80		n.s.	-1.71	4.21	-2.27	4.77	
Overall QoL	57.69	18.40	61.54	20.29	3.33	3.11		n.s.	-1.78	8.45	-2.76	9.43		79.69	16.38	79.69	13.60	<.001	2.86		n.s.	-4.70	4.70	-5.60	5.60	
Health QoL	49.04	26.91	52.88	25.81	2.50	4.38		n.s.	-4.70	9.70	-6.08	11.08		67.19	21.83	68.75	28.14	1.39	3.18		n.s.	-3.84	6.62	-4.84	7.62	
Physical QoL	61.19	20.15	58.15	17.85	-2.83	1.93		n.s.	-6.00	.33	-6.61	.94		82.63	15.81	80.63	17.29	-2.78	2.42		n.s.	-6.76	1.21	-7.53	1.97	
Psychological QoL	54.12	16.15	52.50	17.24	-.37	2.16		n.s.	-3.92	3.18	-4.60	3.86		71.81	15.47	74.38	15.07	1.56	1.76		n.s.	-1.34	4.45	-1.89	5.00	
Social QoL	43.69	13.88	42.58	18.80	.50	2.47		n.s.	-3.56	4.56	-4.34	5.34		74.25	17.46	73.50	17.03	-.67	3.51		n.s.	-6.44	5.11	-7.54	6.21	
Environmental QoL	63.81	14.38	65.46	16.43	-.67	2.17		n.s.	-4.23	2.90	-4.91	3.58		76.19	12.15	77.44	14.93	.11	3.05		n.s.	-4.91	5.13	-5.87	6.10	
Mental health																										
Anxiety	14.16	8.50	14.42	10.75	.26	1.75		n.s.	-2.63	3.14	-3.18	3.70		5.25	6.35	5.58	6.21	.33	1.49		n.s.	-2.12	2.79	-2.59	3.26	
Anxiety level	2.26	0.89	2.19	1.17	-.07	.19		n.s.	-.38	.25	-.44	.31		1.13	0.85	1.42	.88	.29	.20		n.s.	-.03	.61	-.09	.67	
Depression	16.23	12.40	16.00	11.66	-.23	1.23		n.s.	-2.24	1.79	-2.63	2.18		5.00	4.90	6.59	6.12	1.59	.96		n.s.	.01	3.17	-.29	3.47	
Depression level	1.55	1.67	1.55	1.59	<.001	.21		n.s.	-.35	.35	-.42	.42		0.12	.33	.35	.79	.24	.14		n.s.	.01	.46	-.03	.50	

Notes: Using the SD formula indicated in Analysis Strategy (p. 180), the change scores were manually calculated and re-checked using SPSS for consistency. T-tests were carried out within each Diagnostic Group to compare mean differences between T1 and T2 scores. Lower and upper Confidence Intervals (CI) reported for 90% and 95% CI, with RCI calculated for each CI and each variable, respectively.

Appendix 3

Figure 7.10a Email to Participants for Naturalistic TBPM-OR Task (other-relevant)

 **TBPM_OR Email** Edit Select Language ▾

Dear \${m://FirstName},

In your last research appointment, I mentioned that I would be in contact by email to ask for some final information for the Ageing with Autism Research Project.

I would be very grateful if you could complete this short survey to provide feedback about being involved in the project. Your feedback is important to our research.

The survey will be available on **Friday 30th June**, please go to the link on that day only.

You will need to enter your unique code: **\${m://ExternalDataReference}**

Follow this link to the Survey:

\${l://SurveyLink?d=Take the Survey}

Or copy and paste the URL below into your internet browser:
\${l://SurveyURL}

Thank you for remembering to do this survey on 30th June 2017. If you have any questions about the survey, please email me after that date.

Best wishes,
Amanda

Follow the link to opt out of future emails:
\${l://OptOutLink?d=Click here to unsubscribe}

Note: A systematic email was sent to Participants who had completed lab tasks, together with unique de-identified code, 7 days before required completion date.

Figure 7.10b Survey for Naturalistic TBPM-OR Task (other-relevant)



Default Question Block

To start this survey, please enter the code that was emailed to you with this survey link:

☐ I have a code

☐ I forgot my code (please provide your email address)

Thank you for being involved in the Ageing with Autism Research Project.

We would be grateful for your feedback about being involved in this research. Please select one response for each short statement below. This will help to improve staff training and how we approach future projects.

Your responses are de-identified and confidentially stored.

With sincere thanks,

Amanda Roestorf
The Autism Research Group
City, University of London
email: [REDACTED]
web: www.city.ac.uk

1. The researcher kept the appointments to the time that was specified.

☐ Always

☐ Most of the time

☐ About half the time

☐ Sometimes

☐ Never

continued

2. The researcher was professional and friendly.

- ☐ A great deal
- ☐ A lot
- ☐ A moderate amount
- ☐ A little
- ☐ Not at all

3. Taking part in this research was an overall good experience for me.

- ☐ Strongly agree
- ☐ Somewhat agree
- ☐ Neither agree nor disagree
- ☐ Somewhat disagree
- ☐ Strongly disagree

4. Did you use any reminders to help you complete the survey? (Please select all that apply)

- ☐ No, I just remembered
- ☐ Diary or Calendar note
- ☐ Alarm
- ☐ Memo
- ☐ Other (please provide more information)

5. Please enter today's date (numerical format e.g. 24th March 2017 = Day 24 ; Month 03 ; Year 2017)

Day	
Month	
Year	

Figure 7.10c End of Survey Message and Link to Naturalistic TBPM-SR Task (self-relevant)

 **Ageing with Autism - TBPM-SR link** Edit Select Language ▾

Thank you for taking the time to complete this survey. To thank you for your involvement in the Ageing with Autism Project, we would like to offer a £10 gift card. You will need to enter the password code: **ageing_TPM**

This gift card can be claimed on **14th July 2017**, by visiting this web page:
[Ageing with Autism - Thank You](#)

Remember the web page will only be open on 14th July 2017.

Best wishes,
Amanda Roestorf
The Autism Research Group

email: [REDACTED]
web: www.city.ac.uk

Note: On completion of the Naturalistic other-relevant survey, participants were presented a ‘Thank you’ message with a link to the Naturalistic self-relevant survey with the required date of completion. Since this link would only be accessible electronically, participants who completed the Naturalistic other-relevant task were also emailed this message and survey link.

Figure 7.10d Email to Participants for Naturalistic TBPM-SR Task (self-relevant), after omissions on Naturalistic TBPM-SR task

 TBPM_SR Email no-OR

Edit

English ▾

Dear \${m://FirstName},

In your last research appointment, I mentioned that I would be in contact by email regarding the Ageing with Autism Research Project.

To thank you for your involvement in the Ageing with Autism Project, we would like to offer a £10 gift card.

This gift card can be claimed on **24th July 2017**, by visiting the **Ageing with Autism Thank You** web page. You will need to enter your unique code: **\${m://ExternalDataReference}**

Follow this link to the Survey:

[\\${l://SurveyLink?d=Take the Survey}](#)

Or copy and paste the URL below into your internet browser:
[\\${l://SurveyURL}](#)

Remember the web page to claim your gift card will only be available on **24th July 2017**.

Best wishes,
Amanda

Follow the link to opt out of future emails:
[\\${l://OptOutLink?d=Click here to unsubscribe}](#)

Figure 7.10e Survey for Naturalistic TBPM-SR Task (self-relevant)



Default Question Block

To start this survey, please enter the code that was emailed to you with this survey link:

☐ I have a code

☐ I forgot my code (please enter your email address)

Thank you for being involved in the Ageing with Autism Research Project.

Over the past 4 years, I have worked with more than 120 younger and older adults, across several research appointments. This research aimed to better understand the potential changes that occur with growing older, and the effects of these changes on quality of life of autistic adults. Over the next few months, I am writing up the research and look forward to sharing this with you soon.

I would like to thank you once again for being involved in this project, and for your company over the past 4 years.

As an additional gesture of thanks, I am offering a **One4All gift card**, that can be redeemed at various retailers including book stores, food and drink, department stores, entertainment, restaurants and many more*.

continued



Please let me know if you would like the gift card, OR if you would rather not be sent a gift card:

- ☐ Yes, please send me the voucher. I have provided my address below.
- ☐ No, thank you. I do not want to receive the gift card.

Great, now please provide your address below, to make sure the gift card is sent to the correct address for you. Please also provide your email address so I can update our records:

First Name	
Last Name	
Address	
Post code	
Email address	

Thanks, the gift card will be posted to you in the next few days together with more information about where you can use it.

continued

Finally, please let me know how you remembered to visit this survey link today (select all that apply):

- ☐ I just remembered
☐ Diary or Calendar note

- ☐ Memo
☐ Other (please provide more information)

- ☐ Alarm

Please provide your email address so that I can update our records:

Email

Thanks, I will update our records.

Finally, please let me know how you remembered to visit this survey link today (select all that apply):

- ☐ I just remembered
☐ Diary or Calendar note

- ☐ Memo
☐ Other (please provide more information)

- ☐ Alarm

Thank you for your responses. I look forward to sharing more news about this research in the new year.

With sincere appreciation,

Amanda Roestorf, Dermot Bowler and Patricia Howlin
The Autism Research Group
City, University of London

email: 

web: www.city.ac.uk

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Thanks. One last question...

You may recall seeing an email on **23 June 2017**, with a link to a survey about your participation in the Ageing with Autism Project.

We noticed that you had not completed that survey and would like to know a little bit more about that.

Were you unable to complete the survey for any of the following reasons:

☐ I just forgot	☐ I did not want to
☐ My diary or calendar reminder failed to alert me	☐ I did not see the email
☐ I was busy	☐ I prefer not to say
☐ I got distracted	☐ Other (please provide more information)
☐ I was ill or unwell	

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