

Boucher, J., Mayes, A. & Bigham, S. (2012). Memory in Autistic Spectrum Disorder. *Psychological Bulletin*, 138(3), pp. 458-496. doi: 10.1037/a0026869



**CITY UNIVERSITY  
LONDON**

[City Research Online](#)

**Original citation:** Boucher, J., Mayes, A. & Bigham, S. (2012). Memory in Autistic Spectrum Disorder. *Psychological Bulletin*, 138(3), pp. 458-496. doi: 10.1037/a0026869

**Permanent City Research Online URL:** <http://openaccess.city.ac.uk/17791/>

### **Copyright & reuse**

City University London has developed City Research Online so that its users may access the research outputs of City University London's staff. Copyright © and Moral Rights for this paper are retained by the individual author(s) and/ or other copyright holders. All material in City Research Online is checked for eligibility for copyright before being made available in the live archive. URLs from City Research Online may be freely distributed and linked to from other web pages.

### **Versions of research**

The version in City Research Online may differ from the final published version. Users are advised to check the Permanent City Research Online URL above for the status of the paper.

### **Enquiries**

If you have any enquiries about any aspect of City Research Online, or if you wish to make contact with the author(s) of this paper, please email the team at [publications@city.ac.uk](mailto:publications@city.ac.uk).

RUNNING HEAD: Memory in ASD

## Memory in Autistic Spectrum Disorder

### Author Note

Jill Boucher, City University, London, UK.

Andrew Mayes, University of Manchester, Manchester, UK.

Sally Bigham, Bournemouth University, Bournemouth, UK.

Jill Boucher, Department of Psychology, City University, London, UK; Andrew Mayes, School of Psychological Sciences, University of Manchester, Manchester, UK;

Sally Bigham, Psychology Research Centre, Bournemouth University, Bournemouth, UK.

We are grateful to Sophie Lind for her advice and comments relating to studies of the relationship between self and memory in ASD. Correspondence concerning this article should be addressed to Jill Boucher, Autism Research Group, City University, Northampton Square, London, EC1V 0HB, UK, e-mail: Jill.Boucher.1@city.ac

## Abstract

Behavioral evidence concerning memory in high-functioning forms of autism (HFA) and in moderately low-functioning autism (M-LFA) is reviewed and compared. Findings on M-LFA are sparse. However, it is provisionally concluded that memory profiles in HFA and M-LFA (relative to ability-matched controls) are similar, but that declarative memory impairments are more extensive in M-LFA than in HFA. Specifically, both groups have diminished memory for emotion- or person-related stimuli. Regarding memory for non-social stimuli, both groups probably have mental-age appropriate nondeclarative memory; and within declarative memory, both groups have mental-age appropriate immediate free recall of within-span or supra-span lists of unrelated items, as well as cued recall and paired associate learning. By contrast, recognition is largely unimpaired in HFA but moderately impaired in M-LFA; and free recall of meaningful or structured stimuli is moderately impaired in HFA but more severely impaired in M-LFA.

Theoretical explanations of data on declarative memory in HFA identify problems in the integrative processing, or the consolidation and storage, of complex stimuli; or a specific problem of recollection. Proposed neural substrates include the following: disconnectivity of primary sensory and association areas; dysfunctions of medial prefrontal cortex, hippocampus or posterior parietal lobe; or combinations of these associated with neural disconnectivity.

Hypothetically, perirhinal dysfunction might explain the more extensive declarative memory impairments in M-LFA. Foreseeable consequences of uneven memory abilities in HFA and M-LFA are outlined, including possible effects on language and learning in M-LFA. Finally, priorities for future research are identified, highlighting the urgent need for research on memory in lower-functioning individuals.

[Type text]

Keywords: Autistic spectrum disorder; memory; connectivity; hippocampus; prefrontal cortex; parietal lobe

”It is impossible to separate the study of memory from that of autism”

DeLong (2003, p. 741)

Memory and learning are inseparable, and congenital or early acquired anomalies of memory will affect how and what an individual learns, which will in turn affect the course and outcomes of behavioral and brain development, including the ways in which an individual experiences and responds to the external world.

It is well established that certain memory impairments are present in all individuals with autistic spectrum disorder (ASD) (Boucher & Bowler, 2008). It is less clear, however, what differences there may be in the range and severity of memory impairments across the spectrum, from linguistically and intellectually able individuals with Asperger syndrome; through those high-functioning individuals in whom language is initially delayed but subsequently normalizes; through those with persistent, mild to moderate language impairment, usually accompanied by intellectual disability (ID); to the substantial subgroup of individuals with ASD who have little or no useful language and severe or profound ID. The main aim of this paper is to offer for the first time a systematic comparison of memory in groups selected from two different points within the spectrum: specifically, to compare memory in high-functioning as compared to moderately low-functioning individuals with ASD.

There has, moreover, been little discussion of possible differences in the underlying causes of anomalous memory in high-functioning as compared to lower-functioning individuals with ASD, nor have possible differences in the developmental consequences of anomalous memory abilities across the spectrum been considered. Subsidiary aims are, therefore, to consider possible explanations of any differences in memory profiles that may emerge; and to compare likely behavioral consequences of uneven memory abilities in high-functioning as

[Type text]

compared to lower-functioning groups. A final aim is to highlight lacunae in research in this area, and thereby to stimulate research into possible differences, as well as similarities, in memory abilities across the spectrum.

### Historical Background

The history of research into memory in ASD shows an uneven pattern of interest. Several early researchers included tests of memory amongst their experiments. Some of these, including Rimland (1964), Hauser, DeLong, and Rosman (1975), Boucher and Warrington (1976) and DeLong (1978), speculated that autism might derive at least in part from developmental amnesia associated with hippocampal or diencephalic brain abnormalities. It is important to note that diagnostic criteria for autism up to publication of DSM-III(R) (APA, 1987) included clinically significant structural language impairment (APA, 1980; Ritvo & Freeman, 1971; Rutter, 1968, 1974, 1978). The developmental amnesia hypothesis was therefore based on the study of individuals with what would now be diagnosed as autistic disorder (APA,2000).

From the publication of DSM-III(R) onwards, most behavioral research focused on high-functioning individuals with ASD on the grounds that these individuals have ‘pure autism’ uncontaminated by linguistic or intellectual impairments. Tests of theory of mind, central coherence, and executive functions dominated over the next two decades, and research into memory declined. Moreover, such investigations of memory as were carried out demonstrated predominantly normal abilities in high-functioning groups, indicating that a developmental amnesia hypothesis cannot explain autism *per se* (Bennetto, Pennington, & Rogers, 1996; Bowler, Matthews, & Gardiner, 1997; Renner, Klinger, & Klinger, 2000; Minshew & Goldstein, 1993; Rumsey & Hamburger, 1988). Nevertheless, all the foregoing studies demonstrated minor anomalies and impairments of memory. Moreover, further probing has confirmed and extended

[Type text]

the list of differences concerning ways in which high-functioning people with ASD remember and learn as compared to neurotypical (NT) individuals, as reviewed below.

### Theoretical Framework and Terminology

Memory may be analyzed in terms of psychological processes or in terms of underlying systems. Process-related distinctions have been made between deep and shallow encoding, item-specific and relational encoding; immediate (short-term) and long-term memory; rapid, single trial learning and slow, repetition-based learning; recollection and familiarity; effortful and automatic retrieval; verbal and visuospatial memory. All these distinctions have some relevance for characterizing and understanding memory in ASDs, and will be referred to in the reviews and discussions below. Brief definitions and discussion of process-related terms generally can be found in Gardiner (2008); and more extended discussions in Foster and Jelicic (1999) or Tulving and Craik (2000). Where the meanings of less familiar or more controversial process-related terms are critical to theoretical arguments being discussed, we provide definitions within the text, along with supporting references.

A further process-related distinction has been drawn between nondeclarative (or implicit) and declarative (or explicit) forms of memory and learning. This distinction is based in part on a continuum of levels of conscious awareness at retrieval, ranging from no conscious awareness of memory (in the case of, for example, learning to walk); through borderline cases where, for example, one might say of a memory “I don’t *think* I imagined it...”; to a fully conscious awareness that what is remembered is a ‘true’ memory rather than fantasy or déjà vu. Memory system theory overlaps with process theory at this point, in that system taxonomies also distinguish nondeclarative (aka ‘implicit’) and declarative (aka ‘explicit’) kinds of memory. Thus, within the well-known systems taxonomy developed by Tulving (1985; Schacter &

[Type text]

Tulving, 1994), perceptual memory (defined as automatic and unconscious memory for discrete single items, whether simple or complex) and procedural memory (which includes conditioning, habit memory, the acquisition of automatic sensorimotor and cognitive skills, and the acquisition of basic-level concepts) are characterized as *nondeclarative*. By contrast, episodic memory (which holds contextual information about personally experienced events) and semantic memory (which holds impersonal factual information, including word meanings) are characterized as *declarative*. These memory systems acquire and store information in the long-term. Tulving's taxonomy also includes *working memory* (WM), a system dedicated to the short-term maintenance and manipulation of information in thinking and reasoning (Baddeley, 2002).

Tulving's systems taxonomy has been widely used by ASD memory researchers, and his terminology will be used when reporting these researchers' work. However, Tulving's distinction between episodic and semantic declarative memory systems does not coincide with some process-oriented explanations of memory profiles in ASD. Neither does Tulving's distinction between episodic and semantic memory systems coincide accurately with findings from the most commonly used tests of declarative memory--i.e., recall and recognition tests. For these reasons, findings from memory studies of ASD will be presented and discussed under the broader headings italicized above, i.e. *nondeclarative memory*, *declarative memory*, and *working memory*. Evidence relating to specialized forms of memory such as autobiographical memory and prospective memory will not be reviewed, because of a lack of data.

## Plan

The remainder of the paper is in two main sections. The first of these consists of a review of behavioral evidence relating to memory abilities in high-functioning individuals with ASD and in lower-functioning ASD, presented separately. In the second main section, we

[Type text]



initially discuss possible causes of memory anomalies in higher-functioning and lower-functioning ASD, including possible neural substrates. We then discuss some foreseeable consequences of patterns of memory abilities and weaknesses in the two groups before closing the paper with a short section identifying future research priorities.

### Studies of Memory in ASD

Our central aim is to explore possible differences in memory abilities in higher- as compared to lower-functioning individuals with ASD. Studies reviewed in this section therefore include only those in which ASD groups can be clearly differentiated as either ‘high-functioning’ or ‘lower-functioning’, excluding studies of mixed-ability groups (those that include both high- and low-functioning participants) or borderline-ability groups. To achieve this differentiation, the following selection criteria have been used. Studies of ‘high-functioning autism’ (HFA) include only those in which ASD participant groups had mean verbal quotients (VQ) of 85 or above or, when information on verbal abilities was not available, nonverbal/performance quotients (PQ) or full-scale IQs (FSIQ) of 90 or above. Studies of ‘moderately low-functioning autism’ (M-LFA) include only those in which ASD participant groups had mean VQ below 70. Studies of memory in nonverbal individuals are not available, and M-LFA groups generally had VQs above 50. Thus, studies included in the review relate selectively to groups either at the top end or in the middle of an extended continuum of abilities.<sup>1</sup>

The term ‘HFA’ is used here to refer to all individuals with ASD and intellectual and linguistic abilities currently within the normal range, regardless of whether or not language was initially delayed. Thus it includes high-functioning individuals in whom language has normalized following initial delay (the ‘HF-LN’ group), as well as individuals with Asperger syndrome (AS) in whom language onset was not delayed. Evidence relating to individuals with

[Type text]

AS as opposed to HF-LN will not be considered separately because there are too few studies to make comparisons meaningful. There is in fact currently no robust evidence of persistent behavioral differences between high-functioning individuals with ASDs with or without a history of language delay (Frith, 2004; Macintosh & Dissanayake, 2004). Grouping them together may therefore be theoretically as well as pragmatically justifiable. However, the possibility of significant neuropsychological differences between the two subgroups has not been definitively ruled out. For this reason, participant groups consisting solely of individuals with AS or solely of individuals with HF-LN are identified within tables in an Appendix, where this information is available.

Decisions concerning whether or not a particular study focused on individuals with ‘HFA’ or with ‘M-LFA’ as defined above are not only on such information as is provided concerning participants’ verbal and nonverbal abilities but also on the diagnostic criteria and ascertainment methods used to select participants. Acceptable diagnostic criteria include those established by Rutter (1968, 1974, 1978) and by Ritvo and Freeman (1971), plus early versions of the Diagnostic and Statistical Manual (DSM) (American Psychiatric Association (APA), 1980), as well as DSM-III-R (APA, 1987), DSM-IV (APA, 1994; 2000) and International Classification of Diseases-10 (ICD-10) (World Health Association (WHO), 1992). The only important difference, as opposed to differences of emphasis, between the earlier and later sets of diagnostic criteria are the exclusion of structural language impairment as an obligatory criterion from DSM-III-R onwards; the abandonment of ‘early onset’ as a diagnostic criterion; and the acceptance of Asperger syndrome as a form of ASD in ICD-10. Regarding diagnostic ascertainment methods, it is only relatively recently that ‘gold standard’ methods of ascertainment have been developed, prior to which clinical judgment was necessarily relied on. Clinical judgment generally

[Type text]

corresponds well with the results of gold standard tests (Risi et al., 2006), and we consider it acceptable here. Reports of studies that do not state clearly either that (a) diagnosis was made by qualified psychiatrists or psychologists using one of the authoritative sets of diagnostic criteria listed above (b) diagnosis was made using the gold standard methods (or that an authoritative screening test was used to check diagnosis) are excluded the review.

Other exclusion criteria are the following: (i) the inclusion of a high proportion (15% or more) of participants with atypical autism, or pervasive developmental disorder not otherwise specified (PDD-NOS); (ii) inadequate or inappropriate group matching procedures, including failure to equate the ratio of males to females across experimental and comparison groups; or matching experimental and comparison groups only on digit span. In addition, (iii) studies with fewer than 10 participants per group that report negative findings are excluded. However small-scale studies that report positive findings are included, on the assumption that positive findings in small groups may relate to variables with large effect sizes.

Studies included in the review were identified from reference lists from other papers, supplemented by electronic searches of peer-reviewed journal articles. All identified studies meeting the stated criteria are included. Methodological details and main findings from these studies are tabulated in an Appendix. Within each table in the Appendix, studies are listed in order of the mean age of the ASD group tested. Ordering by age reveals that almost all studies of memory in M-LFA are of school-age children, and participant group age is only mentioned in the main text where studies were of adults. Studies of memory in HFA have, however, involved groups aged from approximately 5 to 40 years, and where inconsistent findings are reported, age may be a factor. Participant group age is therefore systematically indicated where findings are mixed (for example, across studies of free recall from declarative memory), but not elsewhere.

[Type text]

Finally, it is important to point out that because studies of mixed-ability groups were excluded, some well-known and important studies of memory in ASD are not cited in the main review. Findings from some such studies are, however, reported and discussed when they shed light on similarities or differences in memory abilities across the spectrum.

### Nondeclarative Memory

#### *Nondeclarative memory in HFA (see Table 1 in the Appendix)*

Many, but not all, of the various forms of nondeclarative, or implicit, learning have been shown to be intact in studies of individuals with HFA (as defined above) when memory for non-social stimuli is tested. Thus, normal perceptual and conceptual priming have been reported in studies using words or pictures or music as stimuli (Bowler, Matthews, & Gardiner, 1997; Gardiner, Bowler, & Grice, 2003; Heaton, Williams, Cummins, & Happé, 2007; Renner et al., 2000; Toichi, 2008). Implicit category formation was unimpaired in a study by Molesworth, Bowler, and Hampton (2005) and unimpaired, although possibly atypically achieved, in studies by Bott, Brock, Brockdorff, Boucher, and Lamberts (2006) and by Soulières, Mottron, Giguère, & Larochelle (2011). Implicit learning of spatial context was unimpaired in a study by Barnes et al. (2008). Implicit learning of motor sequences was also unimpaired in Barnes et al.'s study, and in studies by Travers, Klinger, Mussey, and Klinger (2010) and Nemeth et al. (2010). Classical conditioning was reported to be unimpaired in a study by Sears, Finn, and Steinmetz (1994). Finally, in a study comparing implicit and explicit learning in an HFA group, Brown, Aczel, Jimenez, Kaufman, and Plaisted-Grant (2010) showed unimpaired performance on four implicit learning tasks (contextual cueing, motor sequence learning, artificial grammar learning, and probabilistic learning) contrasting with mildly impaired performance on an explicit memory task (paired associate learning). By contrast, Gaigg and Bowler, (2007) demonstrated impaired

[Type text]

fear conditioning (in adults with AS). Impaired sequence learning was reported by Mostofsky, Goldberg, Landa, and Denkla (2000). However, the methodology used in this study was criticized in the report by Barnes et al. (2008) cited above, in which intact sequence learning was reported (see also the critique in Gordon & Stark, 2007). It has, in addition, been argued that impaired motor skills constitute evidence of impaired implicit learning (Romero-Mungía, 2008; Walenski, Tager-Flusberg, & Ullman, 2006). Motor skills are undoubtedly impaired across the spectrum. However, patterns of impaired and spared motor skills are heterogeneous in ASD, and it is certain that multiple factors are involved in shaping the different profiles that occur. Other causes would, therefore, have to be ruled out before concluding that procedural memory impairments contribute to motor impairments.

#### *Nondeclarative memory in M-LFA*

There are effectively no reliable studies of nondeclarative memory in M-LFA (as defined above). Klinger and Dawson (2001) reported impaired category formation in an M-LFA group. However, the methodology used in this study was criticized by Molesworth et al. (2005; see also Molesworth, Bowler, & Hampton, 2008) and by Bott et al. (2006), casting doubt on the reliability of this finding. Impaired motor skills might be indicative of impaired procedural learning across the spectrum, as argued by Walenski et al. (2006) and by Romero-Mungía (2008). However, other causes would have to be ruled out before reaching this conclusion, as argued above.

Observational and clinical evidence, however, suggests that most forms of nondeclarative memory are relatively unimpaired in M-LFA. For example, Miller (1999) and Pring (2008) have concluded that low-ability savants, many of whom have ASD, achieve their exceptional feats of calculation, drawing, or musical improvisation using implicit perceptual representations and

[Type text]

procedures. In addition, spontaneous behavior in individuals with M-LFA is dominated by habits and routines, suggesting that habit formation is also relatively unimpaired (Toal, Murphy, & Murphy, 2005). Daily living skills involving appropriate use of everyday objects may be relatively spared even in some nonverbal individuals (Carter et al., 1998; Kraijer, 2000), suggesting that implicit knowledge of basic-level categories can be acquired. Behavioral training is used successfully in educational programs and is the intervention of choice for low ability individuals with challenging behavior, implying that instrumental – if not classical - conditioning is also at least relatively intact.

### Declarative Memory

Declarative memory may be assessed using tests of *recognition*, *free recall*, or *cued recall*, and we review studies of declarative memory in ASD under these three subheadings. *Source memory* tasks constitute a specialized set of declarative memory tasks, and we review studies of source memory in a fourth subsection. Some comments concerning the processing requirements of cued recall and source memory are made prior to reviewing relevant studies in these subsections.

#### *Recognition in HFA (see Table 2 in the Appendix)*

Numerous studies show that performance on recognition tests using a variety of non-social stimuli is unimpaired and occasionally superior in HFA. Thus: intact recognition of spoken words was shown in studies by Beversdorf et al. (2000), by Hillier, Campbell, Keillor, Phillips, and Beversdorf (2007), and by Salmond et al. (2005); intact recognition of written words was shown by Boucher et al. (2005), by Bowler, Gardiner, and Grice (2000a), and by Bowler, Gardiner, Grice and Saavalainen (2000b); intact recognition of spoken sentences was shown by Kamio and Toichi (2007) and of heard stories by Salmond et al. (2005) and Williams,

[Type text]

Goldstein and Minshew (2006a); intact recognition of pictures of common objects was shown by Ambery, Russell, Perry, Morris and Murphy (2006), Boucher et al. (2005), Joseph, Steele, Meyer, and Tager-Flusberg (2005), Lind (2008)<sup>2</sup>, Renner et al. (2000), and Salmond et al. (2005); intact recognition of meaningless patterns or shapes was shown by Buitelaar, van der Wees, Swaab-Barneveld and van der Gaag (1999), Bigham, Boucher, Mayes and Anns (2010) and by Boucher, Bigham, Mayes, and Muskett (2008a); and intact recognition of pictures of common objects, of locations, and of colors was shown by Bowler, Gaigg and Gardiner (2010). Superior recognition of geometric shapes and symbols of various colors and number was shown by Hillier et al. (2007); and superior word recognition following phonological encoding was shown in an unexpected recognition test by Toichi et al. (2002).

Some studies have investigated the susceptibility of individuals with HFA to making false positive responses on recognition tasks, and – in particular – to false positive responses when a set of semantically related targets are been presented in so-called ‘memory illusions’. Susceptibility to memory illusions was either typical or reduced in verbal recognition tasks reported by Beversdorf et al. (2000), Hillier et al., 2007, and Kamio and Toichi (2007). Susceptibility to false positive responses more generally was typical in a word recognition experiment by Bowler et al. (2000b), and reduced in a shape recognition experiment by Hillier et al. (2007). These observations are consistent with intact or superior recognition abilities.

In contrast to the overwhelming majority of studies showing unimpaired or superior recognition of non-social stimuli by people with HFA, one study reported mildly impaired recognition of written words (Bowler, Gardiner, & Berthollier, 2004). Given the modest significance level reported ( $p < 0.05$ ) this finding may have occurred by chance. However, group sizes were quite large, and the finding might reflect a weak tendency towards impairment,

consistent with the likelihood that memory abilities in ASD lie along a continuum. In the study by Bowler et al. (2010) in which recognition of objects, locations, and colors was unimpaired when tested individually, recognition of object-location or object-color combinations was impaired on a forced choice recognition task in which foils consisted of previously seen colors and objects in novel combinations. Similarly, Williams, Goldstein, & Minshew (2006a) reported impaired recognition of complex scenes when foils were of scenes resembling but not identical with the studied item.

A handful of studies have investigated the effects of different encoding conditions on recognition memory. Bowler, Gaigg, and Gardiner (2008a) assessed recognition of written words presented in the context of a second word that was either semantically related or unrelated to the target word. They reported normally enhanced recognition of words presented in a meaningful context. Similarly, in the study by Toichi et al (2002) that showed superior recognition of written words encoded phonologically, words encoded semantically were recognized normally. By contrast, whereas participants in the comparison group in this study showed enhanced recognition of descriptive words such as ‘generous’ or ‘shy’ that they had judged to apply, or not to apply, to themselves, the HFA participants did not show this effect. This observation was replicated in studies by Henderson et al. (2009) and by Lombardo, Barnes, Wheelwright and Baron-Cohen (2007). Both these latter studies showed normal recognition of words encoded in terms of physical features (numbers of letters or numbers of syllables), but impaired recognition of words encoded self-referentially.

Recognition of previously unfamiliar faces by people with HFA was shown to be impaired in studies by Boucher et al. (2005) and by Williams, Goldstein, and Minshew (2005a). In the former study, impaired face recognition contrasted with intact written word recognition (as was



also shown in a small-scale study by Ellis, Ellis, Fraser, & Deb, 1994). However, Salmond et al. (2005) reported unimpaired face recognition in a relatively small group, most of whom were diagnosed with AS.

*Recognition in M-LFA (see Table 3 in the Appendix)*

Compared with the number of studies of recognition in HFA, there are relatively few methodologically robust studies of recognition in M-LFA, and findings from those are mixed. Four studies of delayed recognition reported impairment. Thus, Boucher and Warrington (1976) reported impaired recognition of pictures of everyday objects, relative to age-matched NT and age- and ability-matched ID groups. Summers and Craik (1994) reported impaired recognition of words used to name objects, relative to younger, ability-matched NT children. Lind (2008)<sup>2</sup> reported impaired recognition of named pictures of common objects relative to children with HFA or ID and relative to younger, ability-matched NT children. Boucher et al. (2008a) reported impaired recognition of colored shapes relative to younger, ability-matched HFA and NT groups, but not relative to an age- and ability-matched ID group. In the studies by Summers and Craik, and by Lind, recognition -- although impaired in M-LFA groups -- was better for stimuli the child had handled than for those only handled or named by the experimenter, reflecting a self-enactment effect similar to that in NT children.

Three studies have reported unimpaired recognition. In one of these (assessing recognition of pictures), testing was immediate rather than delayed (Boucher & Lewis, 1992). In the other two studies, unexpected recognition tests were given: Hill and Russell (2002) used recognition of common objects the child had handled in a previous task now unexpectedly assessed; Hauck, Fein, Maltby, Waterhouse, and Feinstein (1998) investigated recognition of pictures of common objects used in a preceding matching task now unexpectedly tested.

Regarding recognition of social stimuli, the study by Boucher and Lewis (1992) cited above found impaired immediate recognition of previously unfamiliar faces in the same children whose immediate recognition of pictures of houses was unimpaired. Unfamiliar face recognition was also shown to be impaired in M-LFA in a study by Klin et al. (1999). However, in a study by Wilson, Blades, Coleman and Pascalis (2009), unfamiliar face recognition was only mildly impaired relative to a young NT group, and unimpaired relative to an ability-matched NT group. Similarly, recognition of familiar faces (teachers at children's schools) was shown to be impaired relative to an ID group in a study by Boucher, Lewis, & Collis (1998), but not in a study Wilson, Blades, and Pascalis (2007).

*Free recall in HFA (see Table 4 in the Appendix)*

Findings on free recall in HFA are mixed, depending largely on the nature of the stimuli. Studies of unrelated items are considered first, followed by studies of related items, of visual material, and finally of emotion- or person-related material.

Free recall of supraspan sets of unrelated words or pictures of everyday objects, or of words from a single category, has been reported to be unimpaired in most studies of individuals with HFA. Intact performance is reported regardless of age groups assessed, and regardless of whether recall is immediate or delayed (e.g. Ambery et al., 2006; Bowler et al., 1997; Bowler, Gaigg, & Gardiner, 2008b; Minshew, Goldstein, Muenz, & Payton, 1992; Mottron, Morasse, & Belleville, 2001; Renner et al., 2000; Smith, Gardiner, & Bowler, 2007; Williams et al., 2006a). The ability to learn long lists of unrelated words over repeated trials is also reported to be unimpaired in most studies (Bowler, Gaigg, & Gardiner, 2008b; Bowler, Limoges, & Mottron, 2009a; Buitelaar, van der Wees, Swaab-Barneveld, & van der Gaag, 1999; Salmond et al., 2005).

However, mildly impaired recall of unrelated words on trial 1 and also over trials was reported in adults by Minshew and Goldstein (2001).

Despite the preponderance of studies reporting intact recall of unrelated words, there is some evidence suggestive of anomalous learning in single trial as well as multiple trial tests. In a study of children, Renner et al. (2000) showed a lack of the usual primacy effect on single-trial recall; and Bowler et al. (2009a) noted that the primacy effect increases atypically slowly in HFA adults over repeated trials. In addition, Bowler et al. (2008b) reported that the subjective organization of unrelated words in recall was idiosyncratic in HFA adults, but convergent in an NT comparison group, even though there was no difference in overall recall.

Free recall of semantically related word lists by individuals with HFA is less robustly intact than free recall of unrelated items. Studies by Bowler et al. (1997) and by Smith et al. (2007) showed that adults whose free recall of unrelated words was unimpaired had impaired recall of word lists composed of semantically related words. In another study of adults, Bowler, Gaigg, and Gardiner (2008a) showed that whereas recall of words presented in the context of an unrelated word was unimpaired, recall of words presented in the context of a semantically related word was impaired. In addition, Bowler et al. (2000b) showed that impaired recall of semantically related words by an adult group was characterized by excess false positive responses, although these did not qualify as memory illusions. Minshew and Goldstein (2001) reported impaired immediate recall of a list of related words by adults, although the impairment resolved over subsequent trials. Similarly, Salmond et al. (2005) reported a trend toward impaired immediate recall of related words by adolescents, which resolved over trials. Bowler, Gaigg, and Gardiner (2009b) showed that free recall of lists of words from different categories

organized under superordinate category headings was unimpaired in HFA adults; however, analyses of items recalled showed reduced use of semantic relations between words.

Some evidence suggests that people with HFA are able to utilize semantic meaning to enhance verbal memory but do not spontaneously do so. Thus, Gaigg, Gardiner, and Bowler (2008) showed that adults with HFA are impaired if words selected from a limited set of categories are presented without any encoding instruction but unimpaired when participants are instructed to encode by category during presentation. However, in the study by Smith et al. (2007) in which impaired recall of semantically related words was shown to be impaired, training participants to use categorical information to improve recall was not effective. Most interestingly, Smith et al. also showed that when words could be organized by phonological similarity, phonological relatedness was neither spontaneously used nor used more effectively following training. It may be the case, therefore, that it is relatedness amongst items generally, rather than semantic relatedness in particular, that is not readily utilized in recall by people with HFA.

Because semantic relations and possibly inter-item relations more generally are not spontaneously utilized to aid recall, it is unsurprising that immediate free recall of sentences has been shown to be impaired relative to standardized norms in several studies (Botting & Conti-Ramsden, 2003; Iwanaga, Kawaski, & Tsuchida, 2000; Williams et al., 2006a). In the study by Iwanaga et al. (2000), preschool children with HF-LN were reported to be significantly more impaired than preschool children with AS. However, sentence repetition was unimpaired in a study of children by Whitehouse, Barry, and Bishop (2008). Findings on story recall in groups with HFA are similarly mixed. Three studies of adults have reported unimpaired story recall (Ambery et al., 2006; Boucher et al., 2005; Williams et al., 2005a). However, another study of

[Type text]

adults reported marginally impaired immediate story recall with a strong trend toward impairment following a delay (Salmond et al., 2005). Also, Minshew and Goldstein (2001) reported that both immediate and delayed story recall were impaired in a large-scale study of slightly younger and less able individuals than those tested by Williams et al. (2005a). In a later study of children, Williams et al. (2006a) also reported impairment in both immediate and delayed conditions. These mixed findings may reflect not only differences in stories used in the various studies, some of which may have required more social understanding than others; but also the age and ability of the HFA participants and the size of the groups tested (see Table 4 in the Appendix).

Free recall of visual material has less often been assessed, and findings are mixed. In tests of figure reproduction by HFA adults, Ambery et al. (2006) and Boucher et al. (2005) reported unimpaired ability, where Minshew and Goldstein (2001) reported impaired performance. Minshew and Goldstein additionally reported impaired ability to retrace complex mazes, although performance on a simple maze was unimpaired. Studies of children by Verté, Geurts, Roeyers, Oosterlaan, and Sergeant (2006) and by Williams et al. (2006a) reported impaired ability to reproduce pictures and geometric designs after a short delay.

Free verbal recall of passively observed naturalistic events involving people was reported to be impaired in adolescents with HFA (McCrorry, Henry, & Happé, 2007). Free recall of words with emotionally arousing connotations was impaired in a study of adults (Gaigg & Bowler, 2008).

*Free recall in M-LFA (see Table 5 in the Appendix)*

Findings on free recall of unrelated as opposed to related material to some extent resemble those for HFA. In studies carried out before diagnostic criteria for autism were firmly

[Type text]

established, Hermelin and O'Connor (1970) showed that children with 'infantile autism' were not impaired on immediate free recall tests of supraspan unrelated word lists but were impaired when semantic relatedness or syntactic structure were introduced into the material for recall. Several methodologically acceptable studies subsequently confirmed and extended these observations. In particular, unimpaired immediate recall of supraspan lists of unrelated words was shown in studies by Boucher (1978, 1981a), Fyffe and Prior (1978) and Tager-Flusberg (1991). Boucher (1978, 1981a) showed in addition that the recency effect in children with M-LFA is equivalent to that of age-matched NT children and that unimpaired recall of unrelated words is achieved atypically by children with M-LFA, with enhanced recency compensating for a reduced primacy effect. Regarding immediate free recall of structured or meaningful stimuli: Fyffe and Prior (1978) showed impaired recall of sentences in the same children whose immediate recall of unrelated words was unimpaired, noting in addition that impaired recall of sentences was associated with an exaggerated recency effect. The disruptive effect of enhanced recency on serial recall had earlier been noted by Frith (1970) in an experiment assessing immediate serial recall of structured but non-meaningful supraspan word lists, such as 'spoon horse horse spoon horse horse.' Tager-Flusberg (1991) reported impaired recall of semantically related words in the same group of M-LFA children<sup>3</sup> whose immediate recall of unrelated words was unimpaired; she also reported that impaired recall of semantically related words was associated with reduced use of category clustering. One study of lower-functioning children reported impaired immediate recall of unrelated words, in this instance the names of common objects (Summers & Craik, 1994).

Turning to tests of delayed recall, Boucher and Warrington (1976) reported impaired delayed recall of supraspan lists of unrelated words; and Boucher and Lewis (1989) showed

[Type text]

impaired ability to carry out spoken or demonstrated instructions with or without an intervening delay (with intact ability to carry out written instructions not requiring memory). In the latter study, Boucher and Lewis also showed that participants with M-LFA asked more repeat questions than non-autistic participants in a simplified '20 questions' game, suggesting impaired memory for their own recent utterances. Boucher and Lewis (1989) also replicated the finding from an earlier study by Boucher (1981b) showing that delayed free recall of activities in which children with M-LFA had actively participated was impaired relative to recall by non-autistic children with ID. Millward, Powell, Messer, and Jordan (2000) reported a similar finding in a study comparing children with M-LFA with younger NT children (no ID group was included). These authors also reported that although the autistic children's recall of their own activities was severely impaired relative to recall by the young NT group, recall of what another child had been observed doing was not impaired – a 'reverse enactment effect'. The methodology underlying this latter finding has, however, been questioned (Hare, Mellor, & Azmi, 2007; Lind, 2010). Moreover, in an attempted replication by Hare et al. (2007), no reverse enactment effect was observed in very low ability adults with M-LFA compared with non-autistic ID adults. On the other hand, there was a trend towards a self-enactment effect in both groups. The M-LFA adults performed consistently less well than the ID group in this study, although none of the group comparisons reached significance. However, this negative finding should be treated with caution, given that group sizes were relatively small and some participants in both groups performed at floor.

#### *Cued recall: Introductory remarks*

All experimental tests of recall are, of necessity, at least minimally cued. Thus, tests of 'free recall' typically identify a set of stimuli to be recalled by providing information about the

[Type text]

study context, such as ‘the words you heard just now’ or ‘the pictures I showed you yesterday.’ This kind of cue requires the participant to generate their own further cues before individual target stimuli can be directly and automatically retrieved.

In contrast, in tests of ‘cued recall’ as commonly understood, cues usually bear some well-established relationship to targets such as may lead directly and automatically to the reactivation of an individual target stimulus. For example, if a list of studied words included ‘pig,’ provision of the category cue ‘farm animal’ may automatically and directly reactivate the target response ‘pig,’ which the participant can identify as feeling correct – i.e., as having been in the study word list. However, if automatic reactivation does not occur, this kind of cue may be used to generate a set of possible targets from semantic memory, from which the target response can be recognized as recently experienced (e.g. ‘sheep..? cow..? hen..? PIG!’): a ‘generate-recognize’ strategy. There is considerable variation, however, in how informative cues of this kind may be. For example, whereas the cue ‘farm animal’ enables the participant to generate a relatively small set of candidate responses amongst which ‘pig’ is quite likely to appear, the cue ‘animal’ has the potential to generate a very large set of targets within which ‘pig’ may not occur. Thus, different cues offer different amounts of what Bowler, Gardiner, and Berthollier (2004; Bowler et al., 2009b) refer to as ‘task support’.

In paired associate learning (PAL), which constitutes a type of cued recall, a novel and arbitrary relationship is generally established between two stimuli during study, and one stimulus is then used to cue recall of the other stimulus at test. For example, one word may be used to cue another (e.g., ‘What word went with ‘book’?’); or an object to cue a location (e.g., ‘Where was the triangle on the grid?’); or a face to cue a name (‘What was this person called?’).



Source memory tests constitute a specialized type of cued recall. However, because there are several subtypes of source memory tests, studies of source memory are reviewed in a separate section.

*Cued recall in HFA (see Table 6 in the Appendix)*

Performance by participants with HFA on standard tests of cued recall or PAL is generally unimpaired. In a study by Mottron et al. (2001), category names were used to cue delayed recall of category exemplars, and initial syllables to cue recall of polysyllabic words. Low-frequency targets were used, to reduce successful guessing. So, for example, ‘broccoli’ rather than ‘peas’ or ‘carrots’ was the target cued by the category name ‘vegetable’. Delayed recall of words in response to written word-fragment cues was unimpaired in studies by Bowler et al. (1997) and Gardiner et al. (2003). Delayed recall of unfamiliar proper names in response to cues relating to occupations was unimpaired in a study of adults by Ambery et al. (2006).

Paired word associate learning following a delay was unimpaired in the study by Gardiner et al. (2003), and was unimpaired regardless of whether testing was immediate or delayed in the study by Ambery et al. (2006), as well as in studies by Minshew and Goldstein (2001) and by Williams et al. (2005a). Sound-symbol PAL and object-location PAL were also reported to be unimpaired regardless of whether recall was immediate or delayed (Williams, Goldstein, & Minshew, 2006b). Similarly, studies by Caron, Mottron, Rainville, and Chouinard (2004) and by Salmond et al. (2005) showed unimpaired route learning; it may be assumed that successive features along the route acted as cues for recall. The study by McCrory et al. (2007) in which free recall of passively observed naturalistic events was found to be impaired showed that when leading or direct questions were supplied as recall cues (e.g., ‘What were they wearing?’), performance was unimpaired. Similarly, a study of eye-witness testimony by adults

[Type text]

with HFA showed that non-informative prompts and direct questions can, if sensitive to the needs of individuals, also elicit unimpaired recall relative to ability-matched NT adults (Maras & Bowler, 2010).

Contrasting with these findings, PAL using word pairs was reported to be mildly impaired in a study by Brown et al. (2010). Two studies have reported severe impairment of face-name associate learning (Ambery et al., 2006; Salmond et al., 2005) and impaired cued recall of pictures depicting family scenes has also been reported (Williams et al., 2005a).

*Cued recall in M-LFA (see Table 7 in the Appendix)*

Cued recall and PAL are largely intact in M-LFA, as in HFA. Four studies using standard tests of cued recall with M-LFA groups have been reported. Boucher and Warrington (1976) reported that the ability to use phonological cues (e.g. 'Fr..' to cue 'fruit') was not only unimpaired relative to an age and ability-matched group without autism but also unimpaired relative to an age-matched NT group. Both the comparison groups performed somewhat less well than the M-LFA group in terms of numbers of items recalled, and it therefore seems unlikely that this striking negative finding occurred by chance, although group sizes were small. In a second study reported in the same paper, Boucher and Warrington assessed the ability to use semantic cues (e.g., 'Something you sit on' to cue the relatively low-frequency target 'stool') and again reported intact performance relative to age-matched NT and ability-matched ID groups. Similarly, Tager-Flusberg (1991) assessed the ability of children with M-LFA to use rhyme cues (e.g., 'box' to cue the target word 'fox') and also category cues (e.g., 'fruit' to cue recall of 'cherry') and reported no impairment, nor was there any difference between the ability to use rhyme as compared to category cues in any of the three groups tested. Farrant, Boucher, and Blades (1999) assessed the spontaneous use of visually available category cues (e.g., a picture of

[Type text]

a bathroom to cue verbal recall of ‘toothbrush’, ‘soap’ etc.), and reported no impairment in an M-LFA group. Klin et al. (1999) showed intact ability (relative to a group of children with non-ASD-related developmental or psychiatric disorders) to recall the location of a picture on a page when cued with the picture. Finally, verbal PAL was shown by Boucher and Warrington (1976) to be at an age-appropriate level and superior to that of ability-matched controls. No other methodologically acceptable tests of PAL in M-LFA have been reported, and Boucher and Warrington’s striking finding is in clear need of replication.

Two studies that reported impaired free recall of participants’ own or others’ activities showed significantly improved recall when informative cues were provided (e.g., ‘What did you buy in the shop?’) (Boucher & Lewis, 1989; Millward et al., 2000). Nevertheless, the M-LFA group was impaired relative to a young NT group in the Millward et al. study, with a strong trend toward impairment relative to an ID group in the Boucher and Lewis study. In their study of M-LFA adults’ ability to recall their own or others’ activities, Hare et al. (2007) also reported that cueing significantly improved recall of actions, especially those that had been carried out by the participant, and no impairment was reported relative to individuals with ID without autism. Similarly, Hill and Russell (2002) reported unimpaired recall of actions children had previously carried out with two common objects (for example, placing a pig on a box), when later given the objects and instructed to ‘Show me what you did with these’.

#### *Source memory: Introductory remarks*

Source memory tests constitute a specialized type of cued memory task in which stimuli that have been correctly identified in a standard recognition test are used as cues to information (generally referred to as ‘contextual information’) associated with that stimulus when it was presented in the study phase. Memory for contextual information may be assessed by cued

[Type text]

recognition or by cued recall. For example, a participant who has correctly recognized a word presented during the study phase might be shown a set of colors and asked ‘Which of these colors was this word printed in?’ -- a cued recognition task. Alternatively, the participant might be asked: ‘What color was this word printed in?’ -- a cued recall task. Associations between cues and targets may be established either intentionally (‘Try to remember the word and the color it is printed in’) or incidentally (‘Try to remember the words’).

Source memory recall tests vary in difficulty in the same ways as other kinds of cued recall. In the example above, the recognized word (e.g., ‘dog’) may directly trigger a memory that it was printed in purple when seen at study. If direct activation does not occur, then a generate-recognize strategy may be used with a relatively high chance of success, provided that the set of color names constitutes well-established semantic knowledge, is clearly delimited, and relatively small. By contrast, in the ‘remember-know’ source memory paradigm (Gardiner & Java, 1993), participants are asked to say whether they recall anything they personally experienced when a recognized word was presented at study. In this task, the contextual information to be recalled may sometimes have arisen from an established association (e.g., the word ‘dog’ conjured up an image of the participant’s own dog) but is more likely to involve a novel and arbitrary association (e.g., a bell rang just when the word ‘dog’ was presented; or ‘dog’ came after the word ‘bird’). The more arbitrary the association between a recognized stimulus and the item of contextual information to be recalled, the less source memory tasks resemble ‘standard’ cued recall tasks and the more they resemble free recall tasks or most PAL tasks.

Memory for contextual information related to people is assessed in ‘source monitoring’ tasks (Johnson, Hashtroudi, & Lindsay, 1993). In source monitoring tasks, interest focuses on the

[Type text]

external or internal sources of recognized stimuli, where internal refers to the participants themselves and external refers to some other person or persons. So, for example, a participant who has correctly recognized a previously heard word might be asked ‘Was it spoken by a man or a woman?’ (two external sources); or ‘Did I say it, or did you?’ (one external one internal source – sometimes referred to as reality monitoring); or ‘Did you say it, or did you just think it?’ (two internal sources). The non-specific memory processing demands of source monitoring tasks are low: recognition tests are widely used (e.g., ‘Did this person say the word, or did that person?’), and recall tests generally probe two alternatives (e.g., ‘Did you say it or think it?’). Attention therefore focuses on the social nature of the tasks.

*Source memory in HFA (see Table 8 in the Appendix)*

Findings on source memory in HFA are mixed. A study of adults by Bowler et al. (2004) showed unimpaired ability to recognize (from a written list) a description of what they had been asked to do when a particular word had appeared on a screen in the study phase (e.g. find a rhyme, or think of a related word). However, the same participants were impaired on a recall test of the same contextual information, even though only four alternatives were involved. Using the remember-know paradigm (also with adults), Bowler et al. (2000a) further showed impaired recall of self-experienced contextual information associated with remembered words. In a study of children using a task designed to replicate the processing demands of the remember-know paradigm, Bigham et al. (2010) demonstrated impaired recall of manual actions that had been arbitrarily associated with a meaningless shape in the study phase. In contrast, Salmond et al. (2005) reported that adolescents had unimpaired recall of temporal source in a word recognition test, conflicting with findings from a well-known study of children by Bennetto et al. (1996), in which ASD participants were of mixed ability.

[Type text]

To the best of our knowledge, no tests of source monitoring have been carried out with HFA groups. However, O’Shea, Fein, Cillessen, Klin, and Schultz (2005) assessed source memory for impersonal and person-related contextual information in a mixed-ability, predominantly high-functioning group, reporting intact memory for impersonal, but impaired memory for person-related, contextual information.

*Source memory in M-LFA (see Table 9 in the Appendix)*

Only two studies of source memory (as opposed to source monitoring) in M-LFA have been reported, one of source recognition and one of source recall. In the study of source recognition (Russell & Jarrold, 1999), picture cards were taken from, and returned to, one of four differently colored boxes during study. In one condition this was done by the experimenter and in another condition by the child. Source memory was tested by asking children to return correctly recognized picture cards to their appropriate boxes, visible on the table in front of the child. Children with M-LFA were not impaired on this task. However, whereas a reverse enactment effect occurred in ID and younger NT comparison groups—with children in these groups recalling color-source more accurately in the experimenter-performed than the self-performed condition—this effect did not occur in the M-LFA group, in which participants performed similarly in both conditions. In the study of source recall (Bigham et al., 2010) participants were shown a set of everyday objects individually, one of which – presented toward the middle of the sequence – was a banana. At test, participants were asked whether correctly recognized objects had been presented before or after the banana, in a test of temporal source memory. Teenagers with M-LFA were impaired relative to non-autistic ID and young NT comparison groups on this task.

Two studies of reality monitoring in M-LFA have been reported.<sup>4</sup> In a second experiment reported in the paper by Russell and Jarrold (1999), cited above, the participant and the experimenter took turns to place a picture card on a grid, either on their own behalf or on behalf of a doll partner. Following each successful response on a subsequent picture recognition test, participants were asked who had placed the card on the grid, the participant themselves, the experimenter, or (nominally) one of the dolls. Children with M-LFA were impaired on this task, relative to ID and NT comparison groups. Moreover, the children with M-LFA differed from both comparison groups in failing to show a positive self-enactment effect. In the experiment by Hill and Russell (2002), which included a test of cued recall of an action such as placing a pig on a box, participants took turns with the experimenter in carrying out the action. Participants were subsequently asked to recall whether they, or the experimenter, had carried out the action. Children with M-LFA were not impaired relative to ID and younger NT comparison groups in the main statistical comparison, though mild impairment emerged when selected subsets of participants were compared.

### Working Memory

Two components of working memory (WM) may be distinguished: First the so-called “slave” systems (Baddeley, 2002) that maintain information in an activated on-line state for the purposes of further cognitive processing; and second the set of executive functions that control this further cognitive processing. The “slave” systems may hold internally generated information, and/or information from immediate (short term) memory. In the reviews that follow, studies of immediate memory/ “slave” system capacity are considered first, followed by consideration of studies assessing executive functions in WM.

*Working memory in HFA (see Table 10 in the Appendix)*

[Type text]

Findings on WM in HFA are mixed. Tests of the immediate, serial-order free recall of a sequence of unstructured items such as digits, spatial locations, or single words generally show normal capacity in groups with HFA, as assessed using standardized tasks (e.g., Cui et al., 2010; Joseph et al., 2005; Minshew, Turner, & Goldstein, 2005; Siegel, Minshew, & Goldstein, 1996; Verté et al., 2006; Williams, Goldstein, Carpenter, & Minshew, 2005b; Zinke et al., 2010). Most of these studies assessed children. Nonword repetition in children with HFA is also reported to be intact (Whitehouse et al., 2008). In a study of adolescents, Ozonoff and Strayer (2001) reported intact ability to recall the spatial locations of geometric shapes that had been presented simultaneously on a screen prior to a short delay, indicating unimpaired spatial memory capacity.

In contrast, Manjiviona and Prior (1999) reported mildly impaired digit span in children, as assessed by the Wechsler Intelligence Scale for Children (WISC). In a study of adults using more sensitive scoring procedures than those in standardized tests, Poirier, Martin, Gaigg, and Bowler (2011) reported impaired immediate recall of both digits and words, associated with diminished recall of order rather than of items. Williams, Goldstein, Carpenter, and Minshew (2005b; see also Williams et al., 2006a) reported impaired sequential spatial span in both children and adults, although immediate serial order recall of non-spatial items was intact for both groups (as noted above). In their test of adults, Williams et al. (2005b) used a version of the Corsi blocks task that included a backwards as well as a forwards recall condition. Separate scores for the two conditions were not, however, reported.

These discrepant findings are hard to reconcile. Age does not appear to be a factor. The WISC Digit Span subtest includes a test of backwards as well as forwards recall, and Manjiviona and Prior ascribe their finding of impaired digit span on the WISC backward digit span, with

[Type text]



forward span unimpaired. Backward digit span requires some executive control and recall length is generally somewhat shorter than forward digit span in NT populations (Wilde & Strauss, 2002). However, WISC norms for this subtest are based on the combined forwards and backwards scores, and the majority of studies of HFA groups show normal performance on this subtest contrary to reports of unimpaired digit span from other studies using the Wechsler tests (see Siegel et al., 1996 for a review). With regard to findings on spatial span, it is similarly tempting to suggest that the introduction of a backwards condition in the Corsi blocks test can explain Williams et al.'s (2005b) finding of impairment in adults. However, problems with backward spatial span cannot explain Williams et al.'s (2005b) finding on children, which relates to a forward only task. Poirier et al.'s (2011) report of diminished memory for serial order may have potential explanatory power but is currently an isolated observation.

Working memory tests requiring the manipulation and control of verbal or visual-spatial representations in addition to their maintenance on-line produce similarly discrepant findings, possibly associated with age. Thus, whereas adults' performance on a task involving the re-organization of verbal material was reported to be intact in the study by Williams et al. (2005b), performance on a task involving verbal mediation and rehearsal was impaired in a study of children by Joseph et al. (2005). Similarly, performance on visual spatial tasks with an executive function component was reported to be intact in the study of adolescents by Ozonoff and Strayer (2001), although other studies assessing executive functions in visual-spatial WM (all of children) have reported impairments (Cui et al., 2010; Morris et al., 1999; Steele, Minshew, Luna, & Sweeny, 2007; Williams et al., 2005b). A study by Goldberg et al. (2005) typifies the scope for inconsistent findings on complex WM tasks: HFA children in their study who made

more errors than NT children on a self-ordered search task were nevertheless unimpaired in their ability to generate and use an appropriate search strategy.

*Working memory in M-LFA (see Table 11 in the Appendix)*

WISC subtest profiles reviewed by Siegel et al. (1996) show that digit span (combined forwards and backwards scores) is relatively spared in individuals with M-LFA, where ‘relatively’ indicates that scores on this subtest are higher than on other verbal subtests, even if below standardized norms. Frith (1970) and Farrant et al. (1999) have, amongst others, reported relatively intact performance on the WISC Digit Span subtest. Unimpaired or superior forward digit span relative to ability-matched comparison groups has also been confirmed in studies by Boucher and Warrington (1976), Fein et al. (1996), and Fyffe and Prior (1978). In the study by Boucher and Warrington (1976) forward digit span was reported to be chronological age-appropriate. Russell, Jarrold, and Henry (1996) assessed word span using a verbal repetition task and a nonverbal task in which children were instructed to point to previously named pictures in the order in which the pictures had been presented. No impairments relative to ability-matched comparison groups were reported. Farrant et al. (1999) also used a picture naming task to assess word span, reporting no impairment. Kjelgaard and Tager-Flusberg (2001) reported a mild impairment of nonword repetition, relative to standardized norms, with the M-LFA children’s standard scores being just over 1 sd below the mean.

There are very few studies assessing executive components of WM in lower-functioning groups. However, when children with M-LFA in Farrant et al.’s (1999) study were asked to describe any strategies used to help them to recall digit strings, responses included references to cumulative rehearsal and number grouping. Similarly, Russell et al. (1996) reported evidence that verbal mediation and rehearsal were used to a greater extent by children with M-LFA than

[Type text]

by children with ID in their nonverbal memory span task. In a second experiment reported in the same paper, Russell et al. (1996) assessed WM capacity, defined as the amount of information that can be maintained off-line during concurrent completion of a second, related task. These authors reported that WM capacity in children with M-LFA was impaired relative to ability-matched young NT children but comparable to capacity in non-autistic ID children. Thus, no autism-specific impairment was reported. Griffiths, Pennington, Wehner, and Rogers (1999) also reported no impairment in a group of very young children with M-LFA relative to a non-autistic ID group on a self-ordered box search task.

#### Summary of Findings: Similarities and Differences in Memory Abilities Across the Spectrum

The evidence reviewed above suggests that memory profiles in HFA and in M-LFA (as defined here) show a largely, but not entirely, similar profile of strengths and weaknesses relative to appropriate comparison groups, although HFA groups perform at a higher level than M-LFA groups. Similarities and differences are summarized separately below.

##### *Similarities*

There are probably shared strengths in most forms of nondeclarative memory. However, research-based evidence on nondeclarative memory in M-LFA is almost completely lacking, and nondeclarative memory strengths in M-LFA are inferred from clinical evidence. Experimental evidence of intact semantic priming was, however, demonstrated in a predominantly lower-functioning, mixed-ability group by Hala, Pexman, and Glenwright (2007), providing some research-based support for the conclusion of similarities across the spectrum.

Within declarative memory there is a shared strength in the immediate free recall of supraspan sets of items that are familiar but unrelated, such as random word lists or named pictures of everyday objects, with evidence that enhanced recency compensates for reduced

[Type text]

primacy. There is also a shared strength on standard cued recall and PAL tasks. Within WM there is a shared strength in immediate memory span for digits or unrelated words.

There are shared weaknesses in free recall of semantically related stimuli and possibly on source memory tests in which cues provide contextual rather than target-specific information. However, research evidence on source memory in M-LFA groups is sparse. Performance on WM tasks involving central executive control has more often been shown to be impaired than unimpaired in HFA, as well as in predominantly high-functioning mixed-ability groups across verbal tasks (e.g., Bennetto et al., 1996) and visual-spatial tasks (Loveland, Bachevalier, Pearson, & Lane, 2008). It may be inferred that executive-related WM impairments extend across the spectrum. Here again, however, evidence relating to M-LFA is notably lacking.

There is almost certainly a shared weakness in all kinds of memory for emotion-related or person-related materials (see also the reviews of social memory abilities in ASD by Webb, 2008, and by Lind, 2010). However, vulnerability to impaired memory for social stimuli is not absolute. Face recognition has not always been reported as impaired, and recognition of face parts may be spared, as in the studies of M-LFA by Wilson et al. (2007, 2009)<sup>5</sup>. Moreover, although free recall of observed or personally experienced events has consistently been shown to be impaired, cued recall of events – at least in response to certain types of cue – may be spared in M-LFA as well as in HFA groups. In addition, self-enactment effects have been shown to be typical in most relevant studies of M-LFA as well as HFA groups, as noted at various points in this review (see also the studies of mixed-ability groups by Farrant, Blades, & Boucher, 1998; Hala, Rasmussen, & Henderson, 2005; Lind & Bowler, 2009). Consistent with observations of spared self-enactment effects, tests of reality monitoring ('Did you say/do it, or did I?') have not invariably shown impairment, even in M-LFA groups.

[Type text]

*Differences*

Differences between memory profiles in HFA as compared with M-LFA groups are, on the basis of current evidence, confined to certain facets of declarative memory. Specifically: recognition of non-social stimuli is robustly intact in HFA with the possible exception of the ability to recognize stimuli composed of specific combinations of features, such as complex scenes or object-location/object-color combinations (see Table 2). In contrast, recognition of non-social stimuli was impaired in 4 out of 7 studies of M-LFA (see Table 3). Notably, in two of these 4 studies (those by Lind, 2008, and by Boucher et al., 2008a) recognition was impaired in an M-LFA but not an HFA group. Recognition was also impaired (at chance) in an M-LFA group but not in a higher-functioning autism group in a large-scale study by Barth, Fein, and Waterhouse (1995), not cited in the main review because it did not meet the study inclusion criteria. Of the three studies showing intact recognition of non-social stimuli in M-LFA, one assessed immediate rather than delayed recognition, allowing for an effect of enhanced recency. The other two studies in which recognition was not impaired used unexpected tests, thus assessing incidental rather than intentional learning. In one of these studies, incidental learning was significantly superior to intentional learning in children with M-LFA, whereas the opposite was true of a younger NT group, with the group x condition interaction highly significant (Russell & Jarrold, 1999). Thus, discrepancies between findings on studies of recognition of non-social stimuli by M-LFA groups are likely to be explicable in terms of differences in methodology used.

Differences between HFA and M-LFA groups on tests of free recall of non-social stimuli also emerged. Specifically: free recall was impaired in 9 out of 13 studies of M-LFA with only the immediate free recall of unrelated words being spared in some studies (see Table 5). In

[Type text]

contrast, free recall of unrelated items by HFA groups was unimpaired in 9 out of 10 studies, regardless of whether recall was immediate or delayed; and recall of semantically related word lists, sentences, stories and visual stimuli was unimpaired in 13 out of 31 individual tests reported, with performance least impaired in conditions where verbatim recall could be utilized (see Table 4). Thus, although there are undoubted free recall impairments in higher-functioning individuals with ASD, free recall impairments are more pervasive in moderately low-functioning individuals with ASD.

## Discussion

### Causal Models

Findings from the review raise two major theoretical questions. First, how may the mainly shared profile of memory strengths and weaknesses across the autism spectrum be explained? and second, how may such differences as occur between memory profiles in HFA and in M-LFA be explained? Shared features of the profile are probably associated with autism *per se* and therefore emerge most clearly from studies of HFA. We therefore consider causal models of the memory profile in HFA first.

#### *Explaining the shared memory profile as manifested in studies of HFA*

Causal models proposed by the two research groups that have studied memory in HFA most intensively over recent years, namely the groups headed by Minshew and by Bowler, are considered first.

Minshew and colleagues explain uneven memory abilities in HFA in terms of an impairment of complex information processing, leaving the processing of simple information unimpaired (e.g., Minshew et al., 1997; Williams et al., 2006b). ‘Complex information processing’ is

[Type text]

variously identified with the detection or use of organizational strategies, high processing load, or a requirement for the integration of information. Conversely, ‘simple information processing’ is identified with basic perceptual processes or a low information-processing load. Impaired complex information processing is ascribed by Minshew and colleagues to neural disconnectivity, the pattern of spared and impaired abilities being explained in terms of “a generalized dysfunction of the association cortex, with sparing of primary sensory and motor cortex” (Minshew & Williams, 2007, p. 946). This model of the immediate causes of memory anomalies in ASD is consistent with evidence of a generalized bias in favor of local as opposed to global processing in ASD (Happé & Frith, 2006; Mottron, Dawson, Soulières, Hubert, & Burack 2006). It is also consistent with models of ASD as a disconnection syndrome (Belmonte et al., 2004; Courchesne, 2004; Rippon, Brock, Brown & Boucher, 2007). Notably, Minshew and Williams (2007) explicitly distance themselves from localisationist explanations.

Bowler and colleagues are mainly concerned with the theoretical interpretation of declarative memory strengths and weaknesses in HFA. They note a ‘subtle but persistent’ impairment of episodic memory combined with anomalies in the processing of semantically meaningful information. They ascribe these impairments to diminished ability to bind diverse elements of complex stimuli such as events or stories into memory representations, referring to this as ‘impaired relational encoding’ (Bowler, Gaigg, & Lind, 2011; Gaigg et al., 2008). Bowler and colleagues further argue that single-item or simple associative item-item encoding is unimpaired, consistent with intact free recall of unrelated items, and intact cued recall, and PAL.

Bowler and colleagues have been more concerned than Minshew and colleagues with identifying specific brain structures underlying the declarative memory impairment. In the past, Bowler and colleagues have suggested that prefrontal cortex (PFC) may be the critical site of

dysfunction (Bowler et al., 2004). More recently they have argued for the hippocampus (HC) as the critical neural substrate, specifically excluding the involvement of perirhinal and entorhinal MTL cortices subserving single-item memory (Bowler et al., 2011). Behavioral predictions from both the PFC and HC hypotheses were tested in a study by Bowler, Gaigg, and Gardiner (2010; see also Gaigg et al., 2008), with results favoring the HC hypothesis. A hippocampal explanation of impaired relational processing entails a shift of emphasis away from problems in the initial processing of complex associations (as argued for by Minshew's group) and towards a failure of consolidation and storage according to the dominant view of HC function (see Aggleton & Brown, 2006).

In an influential review of memory in ASD, Ben Shalom (2003) concluded that episodic memory is selectively impaired, leaving perceptual, procedural, and semantic memory mainly intact (WM was not discussed). In this review, Ben Shalom hypothesized that the episodic memory impairment derives from impaired co-ordination of neural activity within PFC-HC circuitry, consistent with the regions of interest to Bowler and colleagues. More recently, however, Ben Shalom (2009) has argued more specifically that medial-PFC may be the critical site of dysfunction underlying not only memory anomalies but also social, emotional and perceptual processing anomalies in ASD.

Like Bowler and colleagues (2004) as well as Ben Shalom (2003), Toichi and Kamio (2002, 2003) conclude that individuals with HFA have mild episodic memory impairment. Toichi and Kamio point out in addition that single-trial free recall tests constitute tests of memory for personally experienced episodes and that impaired performance on such tasks may reflect the episodic memory impairment. They further suggest that the episodic memory impairment in ASD may itself result from a lack of auto-noetic awareness (Wheeler, Stuss, &



Tulving, 1998). ‘Autonoetic awareness’ relates to that component of episodic memory that involves a sense that ‘I was there’ and thus self-concept and ways in which self is experienced, both of which may be abnormal in ASD (see Lind & Bowler, 2008 for a review). Two studies of autonoetic awareness and memory in ASD support Toichi and Kamio’s suggestion (Bowler, Gardiner & Gaigg, 2007; Lind & Bowler, 2010). However, impaired episodic memory would itself impair self-concept, and the initial direction of cause and effect is uncertain.

Joseph et al. (2005) discuss findings on memory in ASD in terms of a dual process model that cuts across Tulving’s systems-based distinctions. According to this model, all declarative forms of memory are partially dependent on two distinct but interactive memory processes, namely recollection and familiarity (Aggleton & Brown, 2006; Jacoby, 1991; Mayes, Montaldi, & Migo, 2007; Yonelinas, 2002). Recollection is defined as a kind of recall in which a recognized stimulus cues recall of diverse kinds of contextual information experienced within the episode in which the stimulus was encountered. It contributes to rapid, single-trial learning, and is almost certainly dependent on intact HC function and connectivity<sup>6</sup>, and possibly also on intact function and connectivity of dorsolateral and other regions of PFC (Aggleton & Brown, 2006; Kirwan et al., 2008; Mayes et al., 2007). Familiarity is defined as a feeling that one has experienced a stimulus before without recalling contextual detail. It generally relates to single percepts or items, including complex items such as scenes, but may also contribute to recognition of certain kinds of association between items (Mayes et al., 2007). Familiarity increases with stimulus repetition, building on perceptual representations that may initially be implicit, and is important for slow, incremental learning. It is thought to be dependent at least in part on intact function and connectivity of perirhinal and entorhinal MTL cortex (Aggleton & Brown, 2006; Montaldi, Spencer, Roberts, & Mayes, 2006). Joseph et al. (2005) hypothesized that recollection

[Type text]

may be impaired in HFA, leaving familiarity intact. They argued in particular that their hypothesis is consistent with impaired performance on recall tasks (which rely mainly on recollection) in combination with good performance on recognition tests (which rely mainly on familiarity). Like other theorists in the field, Joseph and colleagues hypothesize that the pattern of strengths and weaknesses in declarative memory in ASD may be associated with dysfunction within PFC-HC circuitry, leaving perirhinal and entorhinal MTL cortices unaffected.

Boucher, Mayes, and Bigham (2008b; see also Bigham et al., 2010) argue for the same process-based interpretation of the declarative memory profile in HFA as Joseph and colleagues. Boucher and colleagues note in addition that impaired recollection impoverishes the lexical-semantic knowledge-base (Holdstock, Mayes, Isaac, Gong, & Roberts, 2002a), which may help to explain the subtle semantic impairments (Kamio, Robins, Kelley, Swainson, & Fein, 2007; Kelley et al., 2006) as well as the verbal memory anomalies present in HFA. Boucher and colleagues further suggest, following the arguments of Ullman (2001) and Thomas and Karmiloff-Smith (2005), that some anomalies of memory in HFA including instances of superior performance may be understood in terms of unusually well-developed compensatory use of intact memory processes.

*Controversies and uncertainties.* None of the authors whose theoretical interpretations are considered above would claim that their interpretations are either complete or fully proven, and all would recognize that many challenges remain. Outstanding amongst these is the question of the brain substrates of memory impairments in HFA, bearing in mind that the large majority of memory functions are unaffected.

Hippocampal pathology or dysfunction has most often been proposed as the likely source of declarative memory impairments in ASD. However, as early as 1976, Boucher and

Warrington (1976) noted that their findings on children with M-LFA differed from the pattern of memory impairments in HC-related acquired amnesia. Specifically: whereas cued recall and PAL have almost invariably been reported to be intact across the spectrum (see Tables 6 and 7 in the Appendix), this is not the case in HC-related acquired amnesia (Holdstock et al., 2002b; Shimamura & Squire, 1988; Winocur & Weiskrantz, 1976). Bowler et al. (2011) hypothesize that intact cued recall and PAL in HFA reflects compensatory learning, utilizing memory for single items and item-item associations, subserved by intact perirhinal/entorhinal cortices. This argument is, however, weakened by the fact that the individuals with developmental amnesia of HC origin studied by Vargha-Khadem and colleagues (Baddeley, Vargha-Khadem, & Mishkin, 2001; Isaacs et al., 2003; Vargha-Khadem et al., 1997) do not make the suggested compensation, despite having intact perirhinal and entorhinal cortices. Rather, they resemble patients with acquired HC-related amnesia in having impaired PAL and cued recall, additional to impaired free recall. However, another group of individuals with HC-related free recall impairments, namely children with low birthweight, do not always have impaired cued recall and PAL (Isaacs, Edmonds, Chong, Lucas, Morley, & Gadian, 2004; Isaacs, Lucas, Chong, Wood, S. et al., 2000; Taylor, Klien, Minich, & Hack, 2000). Notably, in Taylor et al.'s study cued recall impairment was significantly greater in very low birthweight children than in children with less low birthweights and, by implication, better-preserved HC function. This observation suggests that there may be a continuum of ability to utilize cues, associated with lesser or greater degrees of HC dysgenesis and dysfunction. Consistent evidence of HC pathology in individuals with ASD has proved elusive (for reviews see Cody, Pelphrey, & Piven, 2002; Palmen, Durston, Nederveen, & van Engleland, 2006; Rojas et al., 2004; for recent positive evidence from brain studies see Dager et al., 2007; Groen, Teluij, Buitelaar, & Tendolkar, 2010; Nicolson, DeVito,

[Type text]

Vidal, Sui, Hayashi, et al., 2006). The inconsistency of the evidence suggests that any structural anomalies of HC in ASD may, like those in mildly affected individuals of low birthweight, be selective and difficult to detect.

A hippocampal explanation of the memory profile in HFA might be defended on other grounds. For example, Bowler (personal communication) has suggested that tests of PAL and cued recall in groups with ASD have been insufficiently sensitive to demonstrate a mild impairment that may be present. This possibility receives preliminary support from Brown et al.'s (2010) finding that PAL was unimpaired in an HFA group when data were analyzed by ANOVA but impaired on an equivalence analysis. Alternatively, uncertainty concerning the precise functions of the HC (or regions of HC; or neurotransmitters within the HC) leaves open the possibility that HC anomalies are different in kind from those resulting in developmental or acquired amnesia, with subtly different effects on memory. In sum, although a hippocampal explanation of the declarative memory profile in HFA faces significant challenges, the hypothesis has not been disproven.

The most commonly suggested alternative critical sites of pathology or dysfunction are regions of PFC, especially medial, dorsolateral, and ventrolateral regions as argued for in theoretical papers by Minshew and Williams (2007), Bowler et al. (2004), Ben Shalom (2009), and Joseph et al. (2005). Medial PFC is known to be implicated in the socio-cognitive impairments diagnostic of ASD (Mundy, 2003) and is thus consistent with problems of self-referenced forms of memory, as well as helping to explain impaired memory for person-related stimuli. Similarly, dorsolateral and ventrolateral PFC have been shown to be implicated in executive impairments in ASD (e.g. Gilbert, Bird, Brindley, Frith, & Burgess, 2008), consistent with certain WM impairments. Moreover, as pointed out by Joseph et al. (2005), dorsolateral

[Type text]

and ventrolateral regions of PFC contribute to recollection as well as to facets of WM in neurotypical populations (Ranganath, Johnson, & D'Esposito, 2003). Finally, it is known that frontal efferents show limited development during adolescence in individuals with ASD (Courchesne, 2004), consistent with Minshew and Williams' (2007) disconnectivity explanations of impaired encoding of complex stimuli.

A further possibility, recently proposed on theoretical grounds by Boucher and Mayes (in press), is that the pattern of spared and impaired declarative memory in HFA might be explained in terms of parietal abnormalities. There is a growing body of evidence showing that posterior regions of parietal cortex (PPC) are involved in retrieval from episodic memory (Svoboda, McKinnon, & Levine, 2006; Wagner, Shannon, Kahn, & Buckner, 2005). Moreover, studies by Berryhill et al. (2007) and by Cabeza, Ciaramelli, Olson, and Moscovitch (2008) showed that whereas free recall of episodic memories is impaired in individuals with bilateral PPC lesions, cued recall of event detail is intact, paralleling findings on HFA reported by McCrory et al. (2007) and by Maras and Bowler (2010). In addition, Cabeza et al. (2008) and Davidson et al. (2008) reported impaired performance by patients with PPC lesions on the 'remember' component of the remember-know task, paralleling findings reported by Bowler et al. (2000a). Finally, Ally, Simons, McKeever, Peers and Budson (2008) reported evidence that impaired retrieval of episodic memories in patients with PPC lesions is associated with a loss of autonoetic awareness, consistent with findings reported by Bowler et al. (2007) and by Lind and Bowler (2010). There have been no brain studies focusing specifically on posterior parietal cortex structure or function in HFA. However, there is evidence suggestive of abnormalities affecting other parietal regions (e.g. McAlonan et al., 2005; Perez-Velazquez et al., 2009; Wallace et al.,

2010), especially in the literature on the mirror neuron system in ASD (reviewed in Rizzolatti & Fabbri-Destro, 2010).

Hippocampus, PFC, and PPC are all components of the ‘default network’ thought to subserve internally-focused thinking as opposed to the performance of externally-directed activity (Buckner, Andrews-Hanna, & Schacter, 2008; Buckner & Carroll, 2007; Raichle et al., 2001). Cingulate cortex also forms part of the default network, and there is evidence of abnormal function in ASD within cingulate cortex (e.g., Chiu et al., 2008; Haznedar et al., 2000; Oner et al., 2007; Silk et al., 2006) and in fronto-parietal networks more generally (Thakkar et al., 2008). Buckner and colleagues have explicitly argued that a dysfunctional default system may help to explain behaviors diagnostic of ASD; and it would be parsimonious to explain the selective memory impairments in HFA in this way, also.

Finally, it is possible (and indeed likely) that memory impairments in HFA have different brain correlates in different individuals or subgroups, with subtle behavioral differences that have not been detected to date.

#### *Explaining the differences between memory profiles in HFA and M-LFA*

It might be assumed that the more extensive and severe declarative memory impairments in lower-functioning as compared to high-functioning individuals with ASD result from differences in IQ. Differences in IQ can explain why performance across all kinds of memory task is generally better in HFA than in M-LFA. However, verbal abilities were controlled for by group matching in all studies of M-LFA included in the review; moreover, nonverbal abilities in M-LFA groups in almost all of these studies were either comparable or superior to those in comparison groups (see Tables 3 and 5 in the Appendix). If IQ significantly influences performance on tests of declarative memory, as suggested by Brown et al. (2010),

[Type text]

superior PQ, unless statistically controlled for, might have been expected to bias findings toward superior performance by M-LFA groups in some studies. Instead, significant impairments emerged on some declarative memory tasks, but not on others. Factor(s) other than IQ must therefore be driving such differences as emerge between memory in M-LFA and ability-matched comparison groups. By extension, these factors must also be driving differences between M-LFA and HFA groups. Possible additional factors are considered next.

Hermelin and O'Connor (1970), whose pioneering studies were the first to demonstrate uneven memory abilities in M-LFA groups, interpreted their findings in terms of impaired ability to process meaning, resulting in a habitual preference for processing the physical characteristics of inputs. Habitual reliance on perceptual encoding and memory can explain relatively intact immediate memory span in M-LFA, good rote memory abilities, and also enhanced recency in free recall of unrelated word strings. Impaired ability to process meaning was not, however, explained by these researchers.

Boucher and colleagues (Boucher et al., 2008a, 2008b; Boucher & Mayes, 2011) have hypothesized that the more extensive declarative impairments in M-LFA as compared to HFA result from impaired familiarity additional to impaired recollection. Impaired familiarity would mainly affect semantic as opposed to episodic memory, which could explain the failure to process verbal meaning, first noted by Hermelin and O'Connor (1970).

Mayes and Boucher (2008) tentatively ascribed a combined impairment of recollection and familiarity to a combination of HC and perirhinal and/or entorhinal MTL dysfunction. As noted in the Introduction, several early researchers had hypothesized that hippocampal pathology might explain impaired memory in M-LFA. Of these researchers, DeLong has most consistently continued to make this argument (DeLong, 1978; 1992; 2003; 2008). In DeLong (2003) it was

[Type text]

argued that increases in the extent of pathology, from unilateral to bilateral lesions of the HC, to pervasive bilateral MTL lesions, result in a continuum of memory and learning disabilities from the mild memory anomalies in AS to the global impairments of language and learning in very low-functioning autism. This is an attractive hypothesis in that it attempts to explain the continuum of memory and learning abilities across the whole spectrum.

However, as noted above, the robust sparing of cued recall and PAL – typical of M-LFA as well as HFA groups -- is problematic for a hippocampal hypothesis and more consistent with PPC dysfunction (Boucher & Mayes, in press). In the present state of knowledge, a parietal explanation of impaired recollection in ASD cannot, however, explain differences in the extent of declarative memory impairments in HFA and M-LFA. There is a suggestion that different regions of PPC subserve recollection and familiarity respectively (Cabeza et al., 2008). However, there is no direct evidence to support this claim. In contrast, evidence linking familiarity/recognition/single-item memory to perirhinal cortex in non-autistic populations is strong (Aggleton & Brown, 2006; Montaldi, Spencer, Roberts, & Mayes, 2006). It seems more likely, therefore, that any impairment of familiarity in M-LFA is associated with perirhinal dysgenesis or with abnormal connectivity disrupting perirhinal function.

Of other researchers, only Minshew and Williams (2007) have, to the best of our knowledge, speculated concerning the brain correlates of the memory profile in M-LFA as distinct from brain correlates of the memory profile in HFA. These researchers suggested that additional impairments of connectivity between primary sensory and association areas underlie the more severe cognitive impairments in low-functioning autism including, by implication, impairments of memory. Although this explanation lacks specificity, impaired connectivity



could still underlie cognitive and mnestic limitations in M-LFA. The issue is which neural circuits are disconnected, given the selective nature of memory impairments, even in M-LFA.

The lack of hypothesis-driven brain studies of lower-functioning individuals with ASD means that all the above hypotheses are speculative (but see the review of brain studies by Bauman & Kemper, 2005, and the large-scale structural MRI study by Bonilha et al., 2008).

#### Consequences of Uneven Memory Abilities Across the Spectrum

If, as a reasonably large body of research suggests, high-functioning or 'pure' autism involves an uneven memory profile, then those facets of memory that are intact will, from the earliest months and years, be relied on to compensate for facets of memory that are impaired. For example, in the absence of reliable memory for individual episodes, high-functioning individuals will capitalize on their intact abilities to acquire habits, associations, routines, and also verbal and factual knowledge. An example of this is given in the firsthand account of the exceptionally able individual 'JS' (reported by Boucher, 2007), who described how, after attending a lecture, the content of which he wishes to retain, he converts his notes into a narrative, which he then rehearses using phonological memory, until the narrative is established as an extended fact within semantic memory. Self-organization based on habits and routines, plus intact or superior semantic memory, may predispose individuals with HFA to academic and occupational success, especially when accompanied by above average intelligence. The effects of anomalous memory and learning abilities in HFA are not, therefore, by any means all negative.

However, compensation of the kind reported by JS is arduous and probably stressful. Moreover, default reliance on factual memory to compensate for impaired memory for personal experience distorts autobiographical memory (Crane & Goddard, 2008) and self-concept (Lind,

[Type text]

2010). The peaks and troughs in WM in HFA are less well established than the pattern of strengths and weaknesses in nondeclarative and declarative memory. However, if some kinds of WM are impaired, as available evidence suggests, then certain facets of thinking and reasoning will also be impaired. Certain negative effects of anomalous memory and learning abilities in HFA should therefore also occur. Moreover, the long term effects on brain development of uneven memory abilities and their behavioral consequences in HFA, although unknown, are likely to be cumulative and significant.

There is a much smaller and less reliable body of evidence relating to memory in M-LFA. However, if, as available evidence suggests, groups with M-LFA have the uneven memory profile associated with 'pure' autism, plus the additional disadvantage of an impairment of single item (semantic) memory, the likely behavioral consequences would be overwhelmingly negative. In the first place, the acquisition of word meanings would be impaired, although phonology and syntax, which are products of implicit learning, would be relatively spared. This prediction is consistent with evidence on language abilities in M-LFA, reviewed by Boucher and Anns (in press). In the second place, the acquisition of factual information would be impaired, in contrast to good memory for facts in HFA. The contrasting performance by HFA and M-LFA groups on the Information subtest of the Wechsler tests supports this prediction (Manjiviona & Prior, 1999; Ozonoff, South, & Miller, 2000; Minshew et al., 2005; Siegel et al., 1996). Impaired acquisition of language and of factual information would in turn explain the VQ < PQ discrepancy characteristic of IQ profiles in low-functioning but not high-functioning groups with ASD (Lord & Paul, 1997; Minshew et al., 2005; Siegel et al., 1996). In sum, an additional impairment of semantic memory would explain the language and learning disabilities that distinguish lower-functioning from high-functioning forms of ASD. On the positive side, unimpaired habit learning

[Type text]

and conditioning may be utilized adaptively in the acquisition of everyday habits and skills and may also underlie responsiveness to behavioral interventions.

#### Directions for Future Research

This brief consideration of some foreseeable effects of uneven memory abilities across the spectrum and, at the same time, our recurrent references to a lack of available data, underscores the need for further investigation of memory abilities in ASD.

The most striking lacunae in ASD memory research relate to memory in lower-functioning individuals. As already noted, there are almost no studies of adults with M-LFA<sup>7</sup>. There are no methodologically rigorous studies of nondeclarative, or implicit, learning in M-LFA despite the fact that there are almost certainly strengths here that can be utilized in remedial learning. There are half the number of studies of declarative memory in M-LFA as there are of declarative memory in HFA. There are no hypothesis-driven brain studies of M-LFA, although brain studies relating to memory in high-functioning groups are relatively common. The imbalance between research into memory in M-LFA as compared to research into memory in HFA occurs despite the fact that the foreseeable consequences of a selective declarative memory impairment in HFA are subtle and in some ways advantageous; whereas the foreseeable consequences of a more pervasive declarative memory impairment in lower-functioning forms of ASD are potentially catastrophic.

This is not to argue that more research into memory in HFA is not needed, and (as noted earlier) there are arguments in favor of understanding 'pure' autism before more complex forms of the disorder. Hypothesis-driven brain research is the main priority in the case of HFA. However, behavioral comparisons of cued recall and PAL in groups with HFA or HC-related

developmental amnesia would also be of critical theoretical interest. A better understanding of WM strengths and weaknesses in HFA would be of practical as well as theoretical importance.

More generally, there is a need for longitudinal studies of memory in ASD, preferably starting from infancy, and ideally including clinical, neuropsychological, and brain measures. Normal memory development follows a reasonably well documented trajectory (Cowan, 1998). Autism is a developmental disorder and it was argued at the outset of this paper that congenital or early acquired abnormalities of memory will have cumulative consequences. To date, there have been no attempts to chart even selective aspects of this developmental process in groups with ASD. There is also a need for cross-sectional studies in which a broad range of memory tasks is administered to the same participant group. This practice would enable patterns of memory strengths and weaknesses to emerge directly, rather than by inference from many individual studies as at present (although some of Minshew's studies of HFA partially achieve this aim). There is also a need for more studies directly comparing memory in higher- and lower-functioning groups, obviating the need to make comparisons across diverse individual studies, as we have done herein. Finally, if subsequent research were to prove that individuals with moderately low-functioning autism have somewhat more pervasive declarative memory impairments than groups with HFA, with consequences for language and learning, the possibility that very low-functioning, nonverbal individuals with ASD have a total loss of declarative memory should be investigated.

Because memory weaknesses may be circumvented to at least some extent, and because memory strengths are used compensatively in day to day care as well as in intervention and remedial education, this research is of considerable practical urgency as well as theoretical importance. Caregivers and practitioners already circumvent memory impairments in many

[Type text]

ways, having learned by experience ‘what works.’ For example, visual timetables and instructions are widely used in classrooms to obviate the need to remember a succession of activities; family photographs and videos are used to cue and to reinforce memories of past events; and good habit memory is commonly utilized in remedial teaching, whether to establish self-care routines or in classroom learning. However, increased understanding of some of the blocks to learning and ways around such blocks, should enhance good practice and improve quality of life for individuals, their families, and caregivers.

## References

- Aggleton, J. & Brown, M. (2006). Interleaving brain systems for episodic and recognition memory. *Trends in Cognitive Sciences*, *10*, 455-463. DOI: 10.1016/j.tics.2006.08.003
- Ally, B., Simons, J., McKeever, J., Peers, P., & Budson, A. (2008). Parietal contributions to recollection: Electrophysiological evidence from aging and patients with parietal lesions. *Neuropsychologia*, *46*, 1800-1812. DOI: 10.1016/j.neuropsychologia.2008.02.026
- Ambery, F., Russell, A., Perry, K., Morris, R., & Murphy, D. (2006). Neuropsychological functioning in adults with Asperger syndrome. *Autism*, *10*, 551-564. DOI: 10.1177/1362361306068507
- American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders*. 3rd edition (DSM III). Washington D.C.: APA.
- American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders*, 3<sup>rd</sup> edition revised (DSM III-R). Washington D.C.: APA.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> Edition) (DSM-IV). Washington DC: APA.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> Edition, text revised) (DSM-IV-TR). Washington DC: APA.
- Baddeley, A. (2002). Is working memory still working? *European Psychologist*, *7*, 85-97. DOI: 10.1027//1016-9040.7.2.85
- Baddeley, A., Vargha-Khadem, F., & Mishkin, M. (2001). Preserved recognition in a case of developmental amnesia: Implications for the development of semantic memory? *Journal of Cognitive Neuroscience*, *13*, 357-369. DOI: 10.1162/08989290151137403
- Barnes, K., Howard, J., Howard, D., Kenworthy, L., Vaidya, C., Gaillard, W., et al. (2008).

- Intact implicit learning of spatial context and temporal sequences in childhood autism spectrum disorder. *Neuropsychology*, 22, 563-570. DOI: 10.1037/0894-4105.22.5.563
- Barth , C. , Fein , D. , & Waterhouse , L. ( 1995). Delayed match-to-sample performance in autistic children. *Developmental Neuropsychology*, 11 , 53 – 69. DOI: 10.1080/87565649509540603
- Bauman, M.L., & Kemper, T.L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, 23, 183-187. DOI: 10.1016/j.ijdevneu.2004.09.006
- Belmonte, M., Cook, E., Anderson, G., Rubenstein, R., Greenough, W., Beckel-Mitchener, A., ... Tierney, E. (2004). Autism as a disorder of neural information processing: Directions for research and targets for therapy. *Molecular Psychiatry*, 9, 646-663. DOI: 10.1038/sj.mp.4001499
- Bennetto, L., Pennington, B., & Rogers, S. (1996). Intact and impaired memory functions in autism. *Child Development*, 67, 1816-1835. DOI: 10.1111/j.1467-8624.1996.tb01830.x
- Ben Shalom, D. (2003). Memory in autism: Review and synthesis. *Cortex*, 39, 1129-1138. DOI: 10.1016/S0010-9452(08)70881-5
- Ben Shalom, D. (2009). The medial prefrontal cortex and integration in autism. *The Neuroscientist*, 15, 589-598. DOI: 10.1177/1073858409336371
- Berryhill, M., Phuong, L., Picasso, L., Cabeza, R., & Olson, I. (2007). Parietal lobe and episodic memory: Bilateral damage causes impaired free recall of autobiographical memory. *The Journal of Neuroscience*, 27, 14415-14423. DOI: 10.1523/JNeurosci.4163-07.2007
- Beversdorf, D., Smith, B.W., Crucian, G., Anderson, J., Keillor, J., Barrett, A., ... Heilman, K. (2000). Increased discrimination of “false memories” in autism spectrum disorder.

- Proceedings of the National Academy of Sciences, USA 97*, 8734-8737. DOI: 10.1073/pnas.97.15.8734
- Bigham, S., Boucher, J., Mayes, A., & Anns, S. (2010) Assessing recollection and familiarity in autistic spectrum disorders: methods and findings. *Journal of Autism and Developmental Disorders*, 40, 878-889. DOI: 10.1007/s10803-010-0937-7
- Bonilha, L., Cendes, F., Rorden, C., Eckert, M., Dalgalarrrondo, P., Min Li, L., & Steiner (2008). Gray and white matter imbalance--Typical structural abnormality underlying classic autism? *Brain & Development*, 30(6), 396-401. DOI: 10.1016/j.braindev.2007.11.006
- Bott, L., Brock, J., Brockdorff, N., Boucher, J. & Lamberts, K. (2006). Perceptual similarity in autism. *Quarterly Journal of Experimental Psychology*, 59, 1237-1254. DOI: 10.1080/02724980543000196
- Botting, N., and Conti-Ramsden, G. (2003). Autism, primary pragmatic difficulties and Specific Language Impairment: can we distinguish them using psycholinguistic markers? *Developmental Medicine and Child Neurology*, 45, 515-545. DOI: 10.1111/j.1469-8749.2003.tb00951.x
- Boucher, J. (1978). Echoic memory capacity in autistic children. *Journal of Child Psychology and Psychiatry*, 19, 161-166. DOI: 10.1111/j.1469-7610.1978.tb00457.x
- Boucher, J. (1981a). Immediate free recall in early childhood autism: Another point of behavioral similarity with the amnesic syndrome. *British Journal of Psychology*, 72, 211-215. DOI: 10.1111/j.2044-8295.1981.tb02177.x
- Boucher, J. (1981b). Memory for recent events in autistic children. *Journal of Autism and Developmental Disorders*, 11, 293-302. DOI: 10.1007/BFO1531512
- Boucher, J. (2007). Memory and generativity in very high functioning autism. *Autism*, 11, 255-



264. DOI: 10.1177/1362361307076863

Boucher, J. & Anns, S. (in press). Structural language in autistic spectrum disorder:

Characteristics and causes. *Journal of Child Psychology and Psychiatry*,

Boucher, J., Bigham, S., Mayes, A., & Muskett, T. (2008a). Recognition and language in lower-functioning autism. *Journal of Autism and Developmental Disorders*, 38, 1259-1269.

DOI: 10.1007/s10803-007-0508-8

Boucher, J. & Bowler, D. M. (Eds.) (2008), *Memory in Autism*. Cambridge: Cambridge University Press.

Boucher, J., Cowell, P., Howard, M., Broks, P., Mayes, A., & Roberts, N. (2005). A combined clinical neuropsychological and neuroanatomical study of adults with high-functioning autism. *Cognitive Neuropsychiatry*, 10, 165-214. DOI:10.1080/13546800444000038

Boucher, J. & Lewis, V. (1989). Memory impairments and communication in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, 30, 99-122. DOI: 10.1111/j.1469-7610.1989.tb00771.x

Boucher, J. & Lewis, V. (1992). Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, 33, 843-860. DOI: 10.1111/j.1469-7610.1992.tb01960.x

Boucher, J., Lewis, V., & Collis, G. (1998). Familiar face and voice matching and recognition in children with autism. *Journal of Child Psychology and Psychiatry*, 39, 171-181. DOI: 10.1111/1469-7610.00311

Boucher, J. & Mayes, A. (in press). Memory in ASD: Have we been barking up the wrong tree? *Autism*,

Boucher, J. & Mayes, A. (2011). Memory in autistic spectrum disorders. In D. Fein (Ed.). *The*

- Neuropsychology of Autism* (pp....). Oxford: Oxford University Press.
- Boucher, J., Mayes, A., & Bigham, S. (2008b). Memory, language, and intellectual ability in low functioning autism. In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism: Theories and Evidence* (pp. 268-290). Cambridge: CUP.
- Boucher, J. & Warrington, E. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, *67*, 73-87. DOI: 10.1111/j.2044.8295.1976.tb01499.x
- Bowler, D. M., Gaigg, S. B. & Gardiner, J. M. (2008a). Effects of related and unrelated context on recall and recognition by adults with high-functioning autism spectrum disorder. *Neuropsychologia*, *46*, 993-999. DOI:10.1016/j.neuropsychologia.2007.12.004
- Bowler, D. M., Gaigg, S. B. & Gardiner, J. M. (2008b). Subjective organisation in the free recall of adults with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, *38*, 104-113. DOI: 10.1007/s10803-007-0366-4
- Bowler, D. M., Gaigg, S. B. & Gardiner, J. M. (2009b). Free recall learning of hierarchically organized lists by adults with Asperger's syndrome: Additional evidence for diminished relational processing. *Journal of Autism and Developmental Disorders*, *39*, 589-595. DOI: 35400060172328.0006
- Bowler, D. M., Gaigg, S. B. & Gardiner, J. M. (2010). Multiple list learning in adults with autistic spectrum disorder: Parallels with frontal lobe damage or further evidence of impaired relational encoding? *Journal of Autism and Developmental Disorders*, *40*, 179-187. DOI: 10.1007/s10803-009-0845-x
- Bowler, D.M., Gaigg, S., & Gardiner, J. (2010). Diminished episodic binding of elements of complex stimuli in Autism Spectrum Disorder. Paper presented at the 9th International

Meeting for Autism Research. Philadelphia.

Bowler, D. M., Gaigg, S., & Lind, S. (2011). Memory in autism: Binding, self and brain. In I. Roth & P. Rezaie (Eds.), *Researching the Autistic Spectrum: Contemporary Perspectives* (pp. 316–347). Cambridge: Cambridge University Press.

Bowler, D. M., Gardiner, J., & Berthollier, N. (2004). Source memory in adolescents and adults with Asperger syndrome. *Journal of Autism and Developmental Disorders*, *34*, 533-542. DOI: 10.1007/s10803-004-2548-7

Bowler, D., Gardiner, J., & Gaigg, S. (2007). Factors affecting conscious awareness in the recollective experience of adults with Asperger's syndrome. *Consciousness and Cognition*, *16*, 124–143. DOI:10.1016/j.concog.2005.12.001

Bowler, D.M, Gardiner, J., & Grice, S. (2000a). Episodic memory and remembering in adults with Asperger syndrome. *Journal of Autism and Developmental Disorders*, *30*, 295-304. DOI: 10.1023/A:1005575216176

Bowler, D. M., Gardiner, J. M., Grice, S., & Saavalainen, P. (2000b). Memory illusions: false recall and recognition in high functioning adults with autism. *Journal of Abnormal Psychology*, *109*, 663-672. DOI: 10.1037/0021-843X.109.4.663

Bowler, D. M., Limoges, E. & Mottron, L. (2009a). Different verbal learning strategies in high-functioning autism: Evidence from the Rey Auditory Verbal Learning Test. *Journal of Autism and Developmental Disorders*, *39*, 910-915. DOI: 10.1007/s10803-009-0697-4

Bowler, D.M., Matthews, N.J., & Gardiner, J.M. (1997). Asperger's syndrome and memory: Similarity to autism but not amnesia. *Neuropsychologia*, *35*, 65-70. DOI: 10.1016/S0028-3932(96)00054-1

- Brown, J., Aczel, B., Jimenez, L., Kaufman, S.B., Plaisted-Grant, K. (2010). Intact implicit learning in autism spectrum conditions. *Quarterly Journal of Experimental Psychology* 63, 1789-1812. DOI: 10.1080/17470210903536910I
- Buckner, R., Andrews-Hanna, J., & Schacter, D. (2008). The brain's default network: Anatomy, function, and relevance to disease. *The Year in Cognitive Science: Annals of the New York Academy of Sciences*, 15, 589-598. DOI: 10.1196/annals.1440.011
- Buckner, R. & Carroll, D. (2007). Self-projection and the brain. *Trends in Cognitive Neurosciences*, 11, 49-57. DOI: 10.1016/j.tics.2006.11.004
- Buitelaar, J., van der Wees, M., Swaab-Barneveld, H., & van der Gaag, R. (1999). Verbal memory and performance IQ predict theory of mind and emotion recognition ability in children with autistic spectrum disorders and in psychiatric control children. *Journal of Child Psychology and Psychiatry*, 40, 869-882. DOI: 10.1111/1469-7610.00505
- Cabeza, R., Ciaramelli, E., Olson, I., & Moscovitch, M. (2008). The parietal cortex and episodic memory: An attentional account. *Nature Reviews Neuroscience*, 9, 613-625. DOI: 10.1038/nrn2459
- Caron, M., Mottron, L., Rainville, C., & Chouinard, S. (2004). Do high functioning persons with autism present superior spatial abilities? *Neuropsychologia*, 42, 467-481. DOI: 10.1016/j.neuropsychologia.2003.08.015
- Carter, A., Volkmar, F., Sparrow, S., Wang, J., Lord, C., Dawson, G., et al. (1998). The Vineland Adaptive Behavior Scales: Supplementary norms for individuals with autism. *Journal of Autism and Developmental Disorders*, 28, 287-302. DOI: 10.1023/A:1026056518470
- Chiu, P. H., Kayali, M. A., Kishida, K. T., Tomlin, D., Klinger, L. G., Klinger, M. R., Montague, P. R. (2008). Self responses along cingulate cortex reveal quantitative neural phenotype

- for high-functioning autism. *Neuron*, 75, 463-73. DOI: 10.1016/j.neuron.2007.12.020
- Cody, H., Pelphrey, K. & Piven, J. (2002). Structural and functional magnetic resonance imaging of autism. *International Journal of Developmental Neuroscience*, 20, 421-438. DOI: 10.1016/S0736-5748(02)00053-9
- Courchesne, E. (2004). Brain development in autism: Early overgrowth followed by premature arrest of growth. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 106-111. DOI: 10.1002/mrdd.20020
- Cowan, N. (1998) *The Development of Memory in Childhood*. Hove, UK: Psychology Press.
- Crane, L. & Goddard, L. (2008). Episodic and semantic autobiographical memory in adults with autism spectrum disorder . *Journal of Autism and Developmental Disorders*, 38, 498-506. DOI: 10.1007/s10803-007-0420-2
- Cui, J., Gao, D., Chen, Y., Zou, X., & Wang, Y. (2010). Working memory in early-school-age children with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 40, 958-967. DOI: 10.1007/s10803-010-0943-9
- Dager, S., Wang, L., Friedman, S., Shaw, D., Constantino, J., Artru, A., Dawson, G., & Csernansky, J. (2007). Shape mapping of the hippocampus in young children with autism spectrum disorder. *American Journal of Neuroradiology*, 28, 672-677. DOI: 10.1001/archgenpsychiatry.2010.148
- Davidson, P., Anaki, D., Ciaramelli, E., Cohn, M., Kim, A., Murphy, K., ... Levine, B. (2008). Does lateral parietal cortex support episodic memory? Evidence from focal lesion patients. *Neuropsychologia*, 46, 1743-1755. DOI:10.1016/j.neuropsychologia.2008.01.011
- DeLong, G. R. (1978). A neuropsychological interpretation of infantile autism. In M. Rutter & E. Schopler (Eds.), *Autism* (pp. 207-218). New York: Plenum Press.

- DeLong, G. R. (1992). Autism, amnesia, hippocampus, and learning. *Neuroscience and Biobehavioral Reviews*, *16*, 63-72. DOI: 10.1016/S0149-7634(05)80052-1
- DeLong, G. R. (2003). Disorders of memory in childhood with a focus on temporal lobe disease and autism. In S. Segalowitz & I. Rapin (Eds.), *Handbook of Neuropsychology (2<sup>nd</sup> edition), Vol 8, Part II: Child Neuropsychology, Part II* (pp. 731-751). Amsterdam: Elsevier.
- DeLong, G.R. (2008). Dysfunction and hyperfunction of the hippocampus in autism? In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism: Theories and Evidence* (pp. 103-121). Cambridge: CUP.
- Dunn, L. (1965) *Peabody Picture Vocabulary Test*. Minnesota: American Guidance Service.
- Ellis, H.D., Ellis, D. M., Fraser, W., & Deb, S. (1994). A preliminary study of right hemisphere cognitive deficits and impaired social judgments among young people with Asperger syndrome. *European Child and Adolescent Psychiatry*, *3*, 255-266. DOI: 10.1007/BF01978114
- Farrant, A., Blades, M., & Boucher, J. (1998). Source monitoring in children with autism. *Journal of Autism and Developmental Disorders*, *28*, 43-50. DOI: 10.1023/A:1026010919219
- Farrant, A., Boucher, J., & Blades, M. (1999). Metamemory in children with autism. *Child Development*, *70*, 107-131. DOI: 10.1111/1467-8624.00009
- Fay, W. & Schuler, A. (1980). *Emerging Language in Autistic Children*. Baltimore: Edward Arnold.
- Fein, D., Dunn M., Allen, D. A., Aram, D. M., Hall, N. Morris, R., & Wilson, B. C. (1996). Neuropsychological and language data. In Rapin, I. (Ed.), *Preschool Children with*

- Inadequate Communication: Developmental Language Disorder, Autism, Low IQ* (pp.123-154). London: Mac Keith Press.
- Foster, J.K. & Jelicic, M. (1999). *Memory: Systems, Process, or Function?* Oxford, Oxford University Press.
- Frith, U. (1970). Studies in pattern detection in normal and autistic children. *Journal of Abnormal Psychology, 76*, 413-420. DOI: 10.1037/h0020133
- Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*, 672-686. DOI: 10.1111/j.1469-7610.2004.00262.x.
- Fyffe, C. & Prior, M. (1978). Evidence for language recoding in autistic, retarded and normal children: A re-examination. *British Journal of Psychology, 69*, 393-402. DOI: 10.1111/j.2044-8295.1978.tb01672.x
- Gaigg, S.B., & Bowler, D.M. (2007). Differential fear conditioning in Asperger's syndrome: Implications for an amygdala theory of autism. *Neuropsychologia, 45*, 2125-2134. DOI:10.1016/j.neuropsychologia.2007.01.012
- Gaigg, S.B., & Bowler, D.M. (2008). Free recall and forgetting of emotionally arousing words in autistic spectrum disorder. *Neuropsychologia, 46*, 2336-2343. DOI: 10.1016/j.neuropsychologia.2008.03.008
- Gaigg, S. B., Gardiner, J. M., & Bowler, D.M. (2008) Free recall in autism spectrum disorder: The role of relational and item-specific encoding. *Neuropsychologia, 46*, 986-992. DOI: 10.1016/j.neuropsychologia.2007.11.011
- Gardiner, J. M. (2008). Concepts and theories of memory. In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism: Theory and Evidence* (pp. 3-20) Cambridge: CUP.

- Gardiner, J. M., Bowler, D. M. & Grice, S. (2003). Perceptual and conceptual priming in autism: An extension and replication. *Journal of Autism and Developmental Disorders*, *33*, 259-269. DOI: 10.1023/A:1024450416355
- Gardiner, J. M., & Java, R. I. (1993). Recognising and remembering. In A. F Collins, S. E. Gathercole, M. A. Conway, & P. E Morris (Eds.), *Theories of Memory*. Hove, U.K: Psychology Press.
- Gilbert, S., Bird, G., Brindley, R., Frith, C., & Burgess, P. (2008). Atypical recruitment of medial prefrontal cortex in autism spectrum disorders: An fMRI study of two executive function tasks. *Neuropsychologia*, *46*, 2281–2291. DOI: 10.1016/j.neuropsychologia.2008.03.025
- Goldberg, M., Mostofsky, S., Cutting, L., Mahone, E., Astor, B., Denckla, M. & Landa, R. (2005). Subtle executive impairments in children with autism and children with ADHD. *Journal of Autism and Developmental Disorders*, *35*, 279-293. DOI: 10.1007/s10803-005-3291-4
- Gordon, B. & Stark, S. (2007). Procedural learning of a visual sequence in individuals with autism. *Focus on Autism and Other Developmental Disabilities*, *22*, 14-22. DOI: 10.1177/10883576070220010201
- Griffiths, E., Pennington, B., Wehner, E., & Rogers, S. (1999). Executive functions in young children with autism. *Child Development*, *70*, 817-832. DOI: 10.1111/1467-8624.00059
- Groen, W., Teluij, M., Buitelaar, J., & Tendolkar, I. (2010). Amygdala and hippocampus enlargement during adolescence in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*, 552-560. DOI: 10.1016/j.jaac.2009.12.023
- Hala, S., Pexman, P., & Glenwright, M. (2007). Priming the meaning of homographs in typically developing children and children with autism. *Journal of Autism and Developmental*



- Disorders*, 37, 329-340. DOI: 10.1007/s10803-006-0162-6
- Hala, S., Rasmussen, C., & Henderson, A. (2005). Three types of source monitoring in children with and without autism: The role of executive function. *Journal of Autism and Developmental Disorders*, 35, 75-89. DOI: 10.1007/s10803-004-1036-4
- Happé, F. & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 36, 5-23. DOI: 10.1007/s10803-005-0039-0
- Hare, D.J., Mellor, C., & Azmi, S. (2007). Episodic memory in adults with autistic spectrum disorders: Recall for self- versus other-experienced events. *Research in Developmental Disabilities*, 28, 317-329. DOI: 10.1016/j.ridd.2006.03.003
- Hauck, M., Fein, D., Maltby, N., Waterhouse, L., & Feinstein, C. (1998). Memory for faces in children with autism. *Child Neuropsychology*, 4, 187-198. DOI: 10.1076/chin.4.3.187.3174
- Hauser, S., DeLong, G. R., & Rosman, N. (1975). Pneumographic findings in the infantile autism syndrome. *Brain*, 98, 667-688. DOI: 10.1093/brain/98.4.667
- Haznedar, M., Buchsbaum, M., Wei, T., Hof, P., Cartwright, C., Bienstock, C., et al. (2000). Limbic circuitry in patients with autism spectrum disorders studied with positron emission tomography and magnetic resonance imaging. *The American Journal of Psychiatry*, 157, 1994-2001.
- Heaton, P., Williams, K., Cummins, O., & Happé, F. (2007). Beyond perception: Musical representation and on-line processing in autism. *Journal of Autism and Developmental Disorders*, 37, 1355-1360. DOI: 10.1007/s10803-006-0283-y
- Henderson, H. A., Zahka, N. E., Kojkowski, N. M., Inge, A. P., Schwartz, C. B., Hileman, C. M.

- et al. (2009). Self-referenced memory, social cognition, and symptom presentation in autism. *Journal of Child Psychology and Psychiatry*, *50*, 853-861. DOI: 10.1111/j.1469-7610.2008.02059.x
- Hermelin, B. & O'Connor, N. (1970). *Psychological Experiments with Autistic Children*. Oxford: Pergamon Press.
- Hill, E. & Russell, J. (2002). Action memory and self-monitoring. *Infant and Child Development*, *11*, 159-170. DOI: 10.1002/icd.303
- Hillier, A., Campbell, H., Keillor, J., Phillips, N. & Beversdorf, D. Q. (2007). Decreased false memory for visually presented shapes and symbols among adults on the autism spectrum. *Journal of Clinical and Experimental Neuropsychology*, *29*, 610-616. DOI: 10.1080/13803390600878760
- Holdstock, J., Mayes, A., Isaac, C., Gong, Q., & Roberts, N. (2002a). Differential involvement of the hippocampus and temporal lobe cortices in rapid and slow learning of new semantic information. *Neuropsychologia*, *40*, 748-768. DOI: 10.1016/S0028-3932(01)00192-0
- Holdstock, J., Mayes, A., Roberts, N., Cezayirli, C., Isaac, C., O'Reilly, R., & Norman, K. (2002b). Under what conditions is recognition spared relative to recall after selective hippocampal damage in humans? *Hippocampus*, *12*, 341-351. DOI: 10.1002/hipo.10011
- Isaacs, E., Lucas, A., Chong, W., Wood, S., Johnson, C., Marshall, C., Vargha-Khadem, F., & Gadian, D. (2000). Hippocampal volume and everyday memory in children of very low birthweight. *Pediatric Research*, *47*, 713-720.
- Isaacs, E., Vargha-Khadem, F., Watkins, K. E., Lucas, A., Mishkin, M. & Gadian, D. (2003) Developmental amnesia and its relationship to degree of hippocampal atrophy. *Proceedings of the National Academy of Sciences USA*, *100*, 13960-13063. DOI:

10.1073/pnas.1233825100

- Iwanaga, R., Kawasaki, C., & Tsuchida, R. (2000). Comparison of sensory-motor and cognitive function between autism and Asperger syndrome in preschool children. *Journal of Autism and Developmental Disorders*, *30*, 169-174. DOI: 10.1023/A:1005467807937
- Jacoby, L. L. (1991). A process dissociation framework: Separating automatic from intentional uses of memory. *Journal of Memory and Language*, *30*, 513-541. DOI: 10.1016/0749-596X(91)90025-F
- Johnson, M., Hashtroudi, S., & Lindsay, D. (1993). Reality monitoring. *Psychological Bulletin*, *114*, 3-28. DOI: 10.1037/0033-2909.114.1.3
- Joseph, R., Steele, S., Meyer, E. & Tager-Flusberg, H. (2005). Self-ordered pointing in children with autism: Failure to use verbal mediation in the service of working memory? *Neuropsychologia*, *43*, 1400-1411. DOI: 10.1016/j.neuropsychologia.2005.01.010
- Kamio, Y., Robins, D., Kelley, E., Swainson, B., & Fein, D. (2007). Atypical lexical/semantic processing in high-functioning autism spectrum disorders without early language delay. *Journal of Autism and Developmental Disorders*, *37*, 1116-1122. DOI: 10.1007/s10803-006-0254-3
- Kamio, Y., & Toichi, M. (2007). Memory illusion in high-functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders*, *37*, 867-876. DOI: 10.1007/s10803-006-0214-y
- Kelley, E., Paul, J., Fein, D., & Naigles, L. (2006). Residual language deficits in optimal outcome children with a history of autism. *Journal of Autism and Developmental Disorders*, *36*, 807-828. DOI: 10.1007/s10803-006-0111-4
- Kirwan, C.B., Wixted, J.T. & Squire, L.R. (2008) Activity in the medial temporal lobe predicts

[Type text]

- memory strength, whereas activity in the prefrontal cortex predicts recollection. *Journal of Neuroscience*, 28, 10541-10548. DOI: 10.1523/JNEUROSCI.3456-08.2008
- Kjelgaard, M. & Tager-Flusberg, H. (2001). An investigation of language profiles in autism: Implications for genetic subgroups. *Language and Cognitive Processes*, 16, 287-308. DOI: 10.1080/01690960042000058
- Klin, A., Sparrow, S., de Bildt, A., Cicchetti, D.V., Cohen, D., & Volkmar, F. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29, 499-508. DOI: 10.1023/A:1022299920240
- Klinger, L. & Dawson, G. (2001). Prototype formation in autism. *Development and Psychopathology*, 13, 111-124.
- Kraijer, D. (2000). Review of adaptive behavior studies in mentally retarded persons with autism/pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, 30, 39-48. DOI: 10.1023/A:1005460027636
- Lind, S. (2008). Episodic memory, theory of mind, and temporally extended self-awareness in autism spectrum disorder". Unpublished doctoral thesis, City University, UK.
- Lind, S. (2010). Memory and the self in autism spectrum disorder: A review and theoretical framework. *Autism*, 14, 430-456. DOI: 10.1177/1362361309358700
- Lind, S. & Bowler, D.M. (2008). Episodic memory and autoegetic consciousness in autism spectrum disorders. In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism* (pp. 166-187). Cambridge UK: CUP.
- Lind, S. & Bowler, D. (2009). Recognition memory, self-other source memory, and theory-of-mind in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 1231-1239. DOI: 10.1007/s10803-009-0735-2

- Lind, S. & Bowler, D. (2010). Episodic memory and episodic future thinking in adults with autism. *Journal of Abnormal Psychology, 119*, 896-905. DOI: 10.1037/a0020631
- Lombardo, M. V., Barnes, J. L., Wheelwright, S. J., & Baron-Cohen, S. (2007). Self-referential cognition and empathy in autism. *Plos One, 2*, e883. DOI: 10.1371/journal.pone.0000883
- Lord, C. & Paul, R. (1997). Language and communication in autism. In D. Cohen & F. Volkmar (Eds.), *Handbook of Autism and Pervasive Developmental Disorders (2nd edition)* (pp. 195-225). New York: John Wiley.
- Loveland, K., Bachevalier, J., Pearson, D., & Lane, D. (2008). Fronto-limbic functioning in children and adolescents with and without autism. *Neuropsychologia, 46*, 49-62. DOI: 10.1016/j.neuropsychologia.2007.08.017
- Macintosh, K. & Dissanayake, C. (2004). The similarities and differences between autistic disorder and Asperger's disorder: A review of the empirical evidence. *Journal of Child Psychology and Psychiatry, 45*, 421-434. DOI: 10.1111/j.1469-7610.2004.00234.x
- Manjiviona, J. & Prior, M. (1999). Neuropsychological profiles of children with Asperger syndrome and autism. *Autism, 3*, 327-356. DOI: 10.1177/1362361399003004003
- Maras, K. & Bowler, D. (2010). The Cognitive Interview for eyewitnesses with autistic spectrum disorder. *Journal of Autism and Developmental Disorders, 40*, 1350-1360. DOI: 10.1007/s10803-010-0997-8
- Mayes, A. R. & Boucher, J. (2008). Acquired memory disorders in adults: implications for autism. In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism: Theories and Evidence* (pp. 43-62). Cambridge: CUP.
- Mayes, A.R., Montaldi, D. & Migo, E. (2007). Associative memory and the medial temporal lobes. *Trends in Cognitive Sciences, 11*, 126-135. DOI: 10.1016/j.tics.2006.12.003

McAlonan, G., Cheung, V., Cheung, C., Suckling, J., Lam, G., Tai, K., ... Chua, S. (2005).

Mapping the brain in autism. A voxel-based MRI study of volumetric differences and intercorrelations in autism. *Brain*, *128*, 268-276. DOI: 10.1093/brain/awh332

McCrorry, E., Henry, L., & Happé, F. (2007). Eye-witness memory and suggestibility in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, *48*, 482-489.

DOI: 10.1111/j.1469-7610.2006.01715.x

Miller, L.K. (1999). The savant syndrome: Intellectual impairment and exceptional skill.

*Psychological Bulletin*, *125*, 31-46. DOI: 10.1037/0033-2909.125.1.31

Millward, C., Powell, S., Messer, D., & Jordan, R. (2000). Recall for self and other in autism:

Children's memory for events experienced by themselves and their peers. *Journal of Autism and Developmental Disorders*, *30*, 15-28. DOI: 10.1023/A:1005455926727

Minshew, N. & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the

California Verbal Learning Test. *Neuropsychology*, *7*, 209-216. DOI: 10.1037/0894-4105.7.2.209

Minshew, N. & Goldstein, G. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry*, *42*, 1095-1101. DOI:

10.1017/S0021963001007867

Minshew, N., Goldstein, G., Muenz, L. R. & Payton, J. (1992). Neuropsychological functioning in nonmentally retarded autistic individuals. *Journal of Clinical and Experimental*

*Neuropsychology*, *14*, 749-761. DOI: 10.1080/01688639208402860

Minshew, N., Goldstein, G., & Siegel, D. (1997). Neuropsychologic functioning in autism:

Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society*, *3*, 303-317.

- Minschew, N., Turner, C., & Goldstein, G. (2005). The application of short forms of the Wechsler Intelligence Scales in adults and children with high functioning autism. *Journal of Autism and Developmental Disorders*, *35*, 45-52. DOI: 10.1007/s10803-004-1030-x
- Minschew, N. & Williams, D. (2007). The new neurobiology of autism. *Archives of Neurology*, *64*, 945-950. DOI: 10.1001/archneur.64.7.945
- Molesworth, C., Bowler, D., & Hampton, J. (2005). The prototype effect in recognition memory: Intact in autism? *Journal of Child Psychology and Psychiatry*, *46*, 661-672. DOI: 10.1111/j.1469-7610.2004.00383.x
- Molesworth, C., Bowler, D., & Hampton, J. (2008). When prototypes are not best: Judgements made by children with autism. *Journal of Autism and Developmental Disorders*, *38*, 1721-1730. DOI: 10.1007/s10803-008-0557-7
- Montaldi, D. & Mayes, A.R. (2010). The role of recollection and familiarity in the functional differentiation of the medial temporal lobes. *Hippocampus*, *20*, 1291-1314. DOI: 10.1002/hipo.20853
- Montaldi, D, Spencer, T.J., Roberts, N. & Mayes, A.R. (2006) The neural system that mediates familiarity memory. *Hippocampus*, *16*, 504-520. DOI: 10.1002/hipo.20178
- Morris, R., Rowe, A., Fox, N., Feigenbaum, J., Miotto, E., & Howlin, P. (1999). Spatial working memory in Asperger's syndrome and in patients with focal frontal and temporal lobe lesions. *Brain and Cognition*, *41*, 9-26. DOI: 10.1006/brcg.1999.1093
- Mostofsky, S., Goldberg, M., Landa, R., & Denkla, M. (2000). Evidence for a deficit in procedural learning in children and adolescents with autism: Implications for cerebellar contribution. *Journal of the International Neuropsychological Society*, *6*, 752-759. DOI: 10.1017/S1355617700677020

- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, *36*, 27-43. DOI: 10.1007/s10803-005-0040-7
- Mottron, L., Morasse, K., & Belleville, S. (2001). A study of memory functioning in individuals with autism. *Journal of Child Psychology and Psychiatry*, *42*, 253-260. DOI: 10.1017/S0021963001006722
- Mundy, P. (2003). The neural basis of social impairments in autism: The role of the dorsal medial-frontal cortex and anterior cingulate system. *Journal of Child Psychology and Psychiatry*, *44*, 793-809. DOI: 10.1111/1469-7610.00165
- Nemeth D, Janacsek K, Balogh V, Londe Z, Mingesz R, et al. (2010). Learning in autism: Implicitly superb. *PLoS ONE* 5: e11731. DOI: 10.1371/journal.pone.0011731
- Nicolson, R., DeVito, T., Vidal, C., Sui, Y., Hayashi, K., Drost, D., ... Thompson, P.C. (2006). Detection and mapping of hippocampal abnormalities in autism. *Psychiatry Research: Neuroimaging*, *148*, 11-21. DOI: 10.1016/j.psychresns.2006.02.005
- Oner, O., Devrimci-Ozguven, H., Oktem, F., Yagmurlu, B., Baskak, B., Munir, K. M. (2007). Proton MR spectroscopy: Higher right anterior cingulate *N*-acetylaspartate/choline ratio in Asperger syndrome compared with healthy controls. *American Journal of Neuroradiology*, *28*, 1494-1498. DOI 10.3174/ajnr.A0625
- O'Shea, A., Fein, D., Cillessen, A., Klin, A., & Schultz. R. (2005). Source memory in children with autism spectrum disorders. *Developmental Neuropsychology*, *27*, 337-360. DOI: 10.1207/s15326942dn2703\_3
- Ozonoff, S., South, M., & Miller, J. (2000). DSM-IV defined Asperger syndrome: Cognitive, behavioral, and early history differentiation from autism. *Autism*, *4*, 29-46. DOI:



10.1177/1362361300041003

Ozonoff, S. & Strayer, D. (2001). Further evidence of intact working memory in autism. *Journal of Autism and Developmental Disorders*, 31, 257-263. DOI:

10.1023/A:1010794902139

Palmen, S., Durston, S., Nederveen, H., & van Engleland, H. (2006). No evidence for the preferential involvement of medial temporal lobe structures in high-functioning autism.

*Psychological Medicine*, 36, 827-834. DOI: 10.1017/S0033291706007215

Perez Velazquez, J.L., Barcelo, F., Hung, Y., Leshchenko, Y., Nenadovic, V., Belkas, J., ...Garcia Dominguez, L. (2009). Decreased brain coordinated activity in autism spectrum disorders during executive tasks: Reduced long-range synchronization in the fronto-parietal networks. *International Journal of Psychophysiology*, 73, 341-349.

DOI: 10.1016/j.ijpsycho.2009.05.009

Poirier, M., Martin, J.S., Gaigg, S., & Bowler, D.M. (2011). Short-term memory in autism spectrum disorder. *Journal of Abnormal Psychology*, 120, 247-252. DOI:

10.1037/a0022298

Pring, L. (2008). Memory characteristics in individuals with savant skills. In J. Boucher & D.M. Bowler (Eds.), *Memory in Autism*, (210-230). Cambridge: CUP.

Raichle, M., MacLeod, A., Snyder, A., Powers, W., Gusnard, D., & Shulman, G. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences*, 98,

676-682. DOI: 10.1073/pnas.98.2.676

Ranganath, C., Johnson, M., & D'Esposito, M. (2003). Prefrontal activity associated with working memory and episodic long-term memory. *Neuropsychologia*, 41, 378-389.

DOI:10.1016/S0028-3932(02)00169-0

- Renner, P., Klinger, L., & Klinger, M. (2000). Implicit and explicit memory in autism: Is autism an amnesic disorder? *Journal of Autism and Developmental Disorders*, *30*, 3-14. DOI: 10.1023/A:1005487009889
- Rimland, B. (1964). *Infantile Autism*. New York: Appleton-Century-Crofts.
- Rippon, G., Brock, J., Brown, C. & Boucher, J. (2007). Disordered connectivity in the autistic brain: Challenges for the new psychophysiology. *International Journal of Psychophysiology*, *63*, 164-172. DOI:10.1016/j.ijpsycho.2006.03.012
- Risi, S., Lord, C., Gotham, K., Chrysler, C., Corsello, C. et al. (2006). Combining information from multiple sources in the diagnosis of autistic spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*, 1094-1103. DOI: 10.1097/01.chi.0000227880.42780.0e
- Ritvo, E. & Freeman, B. (1971). National Society for Autistic Children definition of autism *Journal of Pediatric Psychology*, *2*, 146-148. DOI: 10.1093/jpepsy/2.4.146
- Rizzolatti, G. & Fabbri-Destro, M. (2010). Mirror neurons: From discovery to autism. *Experimental Brain Research*, *200*, 223–237. DOI 10.1007/s00221-009-2002-3
- Rojas, D., Allegra-Smith, E., Benkers, T., Camou, S., Reite, M., & Rogers, S. (2004). Hippocampus and amygdala volumes in parents of children with autistic disorder. *American Journal of Psychiatry*, *161*, 2038-2044.
- Romero-Mungía, M. (2008). Mnestic imbalance: A cognitive theory about autistic spectrum disorders. *Annals of General Psychiatry*, *7*, 20. DOI:10.1186/1744-859X-7-20
- Rumsey, J. & Hamburger, S. (1988). Neuropsychological findings in high-functioning men with infantile autism, residual state. *Journal of Clinical and Experimental Neuropsychology*, *10*, 201-221. DOI: 10.1080/01688638808408236

- Russell, J. & Jarrold, C. (1999). Memory for actions in children with autism: self versus other. *Cognitive Neuropsychiatry*, 4, 303-331. DOI: 10.1080/135468099395855
- Russell, J., Jarrold, C., & Henry, L. (1996). Working memory in children with autism and with moderate learning difficulties. *Journal of Child Psychology and Psychiatry*, 37, 673-687. DOI: 10.1111/j.1469-7610.1996.tb01459.x
- Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry*, 9, 1-25. DOI: 10.1111/j.1469-7610.1968.tb02204.x
- Rutter, M. (1974). The development of infantile autism. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, 4, 147-163. DOI: 10.1017/S0033291700041982
- Rutter, M. (1978). Diagnosis and definitions of childhood autism. *Journal of Autism & Childhood Schizophrenia*, 8(2), 139-161. DOI: 10.1007/BF01537863
- Salmond, C., Ashburner, J., Connelly, A., Friston, K., Gadian, D., & Vargha-Khadem, F. (2005). The role of the medial temporal lobe in autistic spectrum disorders. *European Journal of Neuroscience*, 22, 764-772. DOI: 10.1111/j.1460-9568.2005.04217.x
- Schacter, D. & Tulving, E. (1994). What are the memory systems of 1994? In D. Schacter & E. Tulving (eds.), *Memory Systems 1994* (pp. 1-38). Cambridge, MA: MIT Press.
- Sears, L.L., Finn, P., Steinmetz, J. (1994). Abnormal classical eye-blink conditioning in autism. *Journal of Autism and Developmental Disorders*, 24, 737-751. DOI: 10.1007/BF02172283
- Shimamura, A.P. & Squire, L.R. (1988). Long-term memory in amnesia: Cued recall, recognition memory and confidence ratings. *Journal of Experimental Psychology: Learning, Memory and Cognition*, 14, 763-770. DOI: 10.1037/0278-7393.14.4.763

- Siegel, D., Minshew, N., & Goldstein, G. (1996). Wechsler IQ profiles in diagnosis of high functioning autism. *Journal of Autism and Developmental Disorders*, 26, 389-407. DOI: 10.1007/BF02172825
- Silk, T., Rinehart, N., Bradshaw, J., Tonge, B., Egan, G., O'Boyle, M., & Cunnington, R. (2006). Visuospatial processing and the function of prefrontal-parietal networks in autism spectrum disorders: A functional MRI study. *American Journal of Psychiatry*, 163, 1440-1443. DOI: 10.1176/appi.ajp.163.8.1440
- Smith, B., Gardiner, J., & Bowler, D.M. (2007). Deficits in free recall persist in Asperger Syndrome despite training in the use of list-appropriate learning strategies. *Journal of Autism and Developmental Disorders*, 37, 445-454. DOI: 10.1007/s10803-006-0180-4
- Soulières, I., Mottron, L., Giguère, G., & Larochelle, S. (2011). Category induction in autism: Slower, perhaps different, but perfectly possible. *Quarterly Journal of Experimental Psychology*, 64, 311-327. DOI: 10.1080/17470218.492994
- Squire, L.R., Wixted, J. & Clark, R. (2007). Recognition memory and the medial temporal lobe: A new perspective. *Nature Reviews Neuroscience*, 8, 872-883. DOI: 10.1038/nrn2154
- Steele, S., Minshew, N., Luna B., & Sweeney, J. (2007). Spatial working memory deficits in autism. *Journal of Autism and Developmental Disorders*, 37, 605-612. DOI: 10.1007/s10803-006-0202-2
- Summers, J. & Craik, F. (1994). The effect of subject-performed tasks on the memory performance of verbal autistic children. *Journal of Autism and Developmental Disorders*, 24, 773-783. DOI: 10.1007/BF02172285
- Svoboda, E., McKinnon, M. C., & Levine, B. (2006). The functional neuroanatomy of autobiographical memory: A meta-analysis. *Neuropsychologia*, 44, 2189–2208.

DOI:10.1016/j.neuropsychologia.2006.05.023

Tager-Flusberg, H. (1991). Semantic processing in the free recall of autistic children: Further evidence of a cognitive deficit. In G. Dawson (Ed.), *Autism: Nature, Diagnosis, and Treatment*. (pp. 92-109). New York: Guilford Press.

Taylor, H. G., Klien, N., Minich, N. M., & Hack, M. (2000). Verbal memory deficits in children with less than 750 g birthweight. *Child Neuropsychology*, *16*, 49–63. DOI:10.1076/0929-7049(200003)6:1;1-B;FT049

Thakkar, K., Polli, F., Joseph, R., Tuch, D., Hadjikhani, N., Barton, J., Manoach, D. (2008). Response monitoring, repetitive behavior and anterior cingulate abnormalities in autism spectrum disorders (ASD). *Brain*, *131*, 2464-2478. DOI: 10.1093/brain/awn099

Thomas, M., & Karmiloff-Smith, A. (2005). Can developmental disorders reveal the component parts of the human language faculty? *Language Learning and Development*, *1*, 65-92. DOI: 10.1207/s15473341lld0101\_5

Toal, F., Murphy, D., & Murphy, K. (2005). Autistic spectrum disorders: Lessons from neuroimaging. *British Journal of Psychiatry*, *187*, 395-397. DOI: 10.1192/bjp.187.5.395

Toichi, M. (2008). Episodic memory, semantic memory and self-awareness in high-functioning autism. In J. Boucher & D. Bowler (Eds.), *Memory in Autism: Theory and Evidence* (pp. 143-165). Cambridge: CUP.

Toichi, M. & Kamio, Y. (2002). Long-term memory and levels-of-processing in autism. *Neuropsychologia*, *40*, 964-969. DOI: 10.1016/S0028-3932(01)00163-4

Toichi, M. & Kamio, Y. (2003). Long term memory in high functioning autism: Controversy on episodic memory reconsidered. *Journal of Autism and Developmental Disorders*, *33*, 151-161. DOI: 10.1023/A:1022935325843

- Toichi, M., Kamio, Y., Okada, T., Sakihama, M., Youngstrom, E. A., Findling, R. L. et al. (2002). A lack of self-consciousness in autism. *American Journal of Psychiatry*, *159*, 1422-1424.
- Travers, B., Klinger, M., Mussey, J., & Klinger, L. (2010). Motor-linked implicit learning in persons with autistic spectrum disorders. *Autism Research*, *3*, 68-77. DOI: 10.1002/aur.123
- Tulving, E. (1985). How many memory systems are there? *American Psychologist*, *40*, 385-398. DOI: 10.1037/0003-066X.40.4.385
- Tulving, E. & Craik, F. (2000). *Oxford Handbook of Memory*. New York: OUP.
- Ullman, M. (2001). The declarative/procedural model of lexicon and grammar. *Journal of Psycholinguistic Research*, *30*, 37-69. DOI: 10.1023/A:1005204207369
- Vargha-Khadem, F., Gadian, D., Watkins, K., Connelly, A., van Paesschen, W. & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science*, *277*, 376-380. DOI: 10.1126/science.277.5324.376
- Verté, S., Geurts, H., Roeyers, Oosterlaan, & Sergeant, J. (2006). Executive functioning in children with an autism spectrum disorder: Can we differentiate within the spectrum? *Journal of Autism and Developmental Disorders*, *36*, 351- 362. DOI: 10.1007/s10803-006-0074-5
- Wagner, A., Shannon, B., Kahn, I., & Buckner R. (2005). Parietal lobe contributions to episodic memory retrieval. *Trends in Cognitive Sciences*, *9*, 445–453. DOI: 10.1016/j.tics.2005.07.001
- Walenski, M., Tager-Flusberg, H., Ullman, M. (2006). Language in autism. In Moldin S. & Rubenstein J. (Eds.). *Understanding Autism: From Basic Neuroscience to Treatment* (pp. 175–203). Boca Raton, FL: Taylor and Francis Books.

- Wallace, G., Dankner, N., Kenworthy, L., Giedd, J., & Martin, A. (2010). Age-related temporal and parietal cortical thinning in autism spectrum disorders. *Brain, 133*, 3745-3754. DOI: 10.1093/brain/awq279
- Webb, S. (2008). Impairments in social memory in autism? Evidence from behavior and neuroimaging. In J. Boucher & D. M. Bowler (Eds.), *Memory in Autism: Theories and Evidence* (pp. 188-209). Cambridge: CUP.
- Wheeler, M., Stuss, D., & Tulving, E. (1998). Toward a theory of episodic memory: The frontal lobes and autonoetic consciousness. *Psychological Bulletin, 121*, 331-354. DOI: 10.1037/0033-2909.121.3.331
- Whitehouse, A. J. O., Barry, J. G., & Bishop, D. V. M. (2008). Further defining the language impairment of autism: Is there a specific language impairment subtype? *Journal of Communication Disorders, 41*, 319-336. DOI: 10.1016/j.jcomdis.2008.01.002
- Wilde, N., & Strauss, E. (2002). Functional equivalence of WAIS-III/WMS-III Digit and Spatial Span under forward and backward recall conditions. *The Clinical Neuropsychologist, 16*, 322-330. DOI: 10.1076/clin.16.3.322.13858
- Williams, D.L., Goldstein, G., Minshew, N.J., (2005a). Impaired memory for faces and social scenes in autism: Clinical implications of memory dysfunction. *Archives of Clinical Neuropsychology, 20*, 1-15. DOI: 10.1016/j.acn.2002.08.001
- Williams, D.L., Goldstein, G., Carpenter, P., & Minshew, N. (2005b). Verbal and spatial working memory in autism. *Journal of Autism and Developmental Disorders, 35*, 747-756. DOI: 10.1007/s10803-005-0021-x
- Williams, D.L., Goldstein, G., Minshew, N.J., (2006a). The profile of memory function in children with autism. *Neuropsychology, 20*, 21-29. DOI: 10.1037/0894-4105.20.1.21

- Williams, D.L., Goldstein, G., Minshew, N.J., (2006b). Neuropsychologic functioning in children with autism: Further evidence for disordered complex information-processing. *Child Neuropsychology*, *12*, 279-298. DOI: 10.1080/09297040600681190
- Wilson, R., Blades, M., Coleman, M., & Pascalis, O. (2009). Unfamiliar face recognition in children with autistic spectrum disorders. *Infant and Child Development*, *18*, 545-559. DOI: 10.1002/icd.638
- Wilson, R., Blades, M., & Pascalis, O. (2007). Familiar face recognition in children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *37*, 314-320. DOI: 10.1007/s10803-006-0169-z
- Winocur, G. & Weiskrantz, L. (1976). An investigation of paired associate learning in amnesic patients. *Neuropsychologia*, *14*, 97-110. DOI: 10.1016/0028-3932(76)90011-7
- World Health Organisation (1992). *International Classification of Mental and Behavioral Disorders: Clinical Descriptions and Diagnostic Guidelines*, (10<sup>th</sup> edition) (ICD-10). Geneva: WHO.
- Yonelinas, A. (2002). The nature of recollection and familiarity: A review of 30 years research. *Journal of Memory and Language*, *46*, 441-517. DOI: 10.1006/jmla.2002.2864
- Zinke, K., Fries, E., Altgassen, M., Kirschbaum, C., Dettenborn, L., & Kliegel, M. (2010). Visuospatial short-term memory explains deficits in tower task planning in high-functioning children with autism spectrum disorder. *Child Neuropsychology*, *16*, 229-241. DOI: 10.1080/09297040903559648



## Endnotes

<sup>1</sup>The inclusion criteria relating to ability in HFA and M-LFA groups respectively do not rule out some overlap between the abilities of individuals included in some 'HFA' or 'M-LFA' groups. However, given the 15 point difference between the VQ minimum for HFA groups and VQ maximum for M-LFA groups, and given also that in the majority of studies reviewed 'HFA' participant groups had VQs in the 90s or 100s whereas 'M-LFA' participant groups had VQs in the 50s or 60s (see Appendix), the extent of any overlap can only be small.

<sup>2</sup>Lind (2008), cited in the sections on recognition in HFA and in M-LFA, refers to an unpublished Doctoral Thesis in which data on recognition in HFA and M-LFA groups were reported separately. In a published paper in which data from these HFA and M-LFA subgroups were combined and compared with data from a combined ID + NT group (Lind & Bowler, 2009a), unimpaired recognition was reported. This observation underlines the fact that studies of mixed ability groups can conceal differences between higher and lower-functioning individuals, a possibility first noted by Boucher and Warrington (1976).

<sup>3</sup>Tager-Flusberg describes her participants as 'high-functioning'. Other researchers working in the 1970s and 1980s also sometimes described their ASD participants as 'relatively able' or 'relatively high-functioning'. However, it must be remembered that at the time almost all children with a diagnosis of autism had language impairments and ID, and such terms were used only to imply that participants had some useful language and were able to co-operate with formal testing.

<sup>4</sup>A frequently-cited study of source memory by Farrant, Blades, and Boucher (1998), not included in this review because it assessed a mixed-ability (albeit predominantly low-

[Type text]

functioning) group, reported unimpaired performance on what was ostensibly a reality-monitoring task. However, in this experiment the tester and the participant each held a different colored block, eliminating the need to encode social information to achieve successful performance.

<sup>5</sup>The face recognition studies by Wilson et al. (2007, 2009) used Yes/No as opposed to forced choice recognition tests, which might also have contributed to their somewhat unexpected findings. It would be interesting to compare Yes/No with forced choice recognition in people with ASD, not least because Yes/No recognition may rely more on recollection than on familiarity at least when foils are very similar to targets, whereas the reverse may be true of forced choice recognition (Holdstock et al., 2002b; Yonelinas, 2002).

<sup>6</sup>The view that whereas the HC is important for recollection, perirhinal, and entorhinal cortices are important for familiarity is disputed by Squire, Wixted, and Clark (2007). These authors have argued that recollection/recall and familiarity are both affected in the same way whether the damage/dysfunction is to the hippocampus, entorhinal cortex, perirhinal cortex, or parahippocampal cortex, although damage/dysfunction to the last three structures has more serious effects on recognition memory. Nevertheless, there is growing evidence that selective HC pathology causes more selective as well as perhaps milder declarative memory disorders (Holdstock et al., 2002b; Vargha-Khadem et al., 1997) as opposed to the global and more serious effects of HC pathology extending into adjacent MTL cortex (Montaldi & Mayes, 2010).

<sup>7</sup>This lack of studies results partly from the fact that adults with ASD were not commonly identified until the later years of the 20<sup>th</sup> century. At just this time, diagnostic criteria for ASD changed to include high-functioning individuals with intact language, and most research attention switched to this group. The fact that adults with LFA continued to be largely ignored by

[Type text]

researchers probably results from practical difficulties relating to access, ethics, and the greater difficulties in devising appropriate methods for testing less able people.

## APPENDIX: Tables giving study details

**Abbreviations used in the Tables:**

AS = Asperger syndrome. HF-LN = High-functioning individuals with ASD whose language has normalized after initial delay. HFA = High-functioning autism as defined in this paper to include individuals with either AS or HF-LN. M-LFA = Moderately low-functioning autism as defined in this paper. NT = Neurotypical. ID = Intellectual disability (idiopathic unless otherwise stated). DLD = Developmental language disorder. SLI = Specific language impairment.

DSM (various versions) = Diagnostic and Statistical Manual. ICD-10 = International Classification of Diseases. ADI-R = Autism Diagnostic Interview – Revised (Lord et al., 1994). ADOS = Autism Diagnostic Observation Schedule (DiLavore et al., 1995). AQ = Autism Spectrum Quotient (Baron-Cohen et al., 2001). ASSQ = Autism Spectrum Screening Questionnaire (Ehlers et al., 1999). CARS = Childhood Autism Rating Scale (Schopler et al., 1988). WADIC = Wing Autism Diagnostic Interview Checklist (Wing, 1996). ASAS = Australian Scale for Asperger Syndrome (Garnett & Attwood, 1997). [Diagnostic criteria/ascertainment methods used for ASD participant selection are given in square brackets in the column headed ‘Groups’.]

BPVS = British Picture Vocabulary Scale. CELF = Clinical Evaluation of Language Fundamentals (various versions). CLCT = Carrow Language Comprehension Test. CMS = Children’s Memory Scale. CPM = Coloured Progressive Matrices. CVLT = California Verbal Learning Test. EOWPVT = Expressive One-Word Picture Vocabulary Test. ITPA = Illinois Test of Psycholinguistic Abilities. K-ABC = Kaufman Assessment Battery for Children. K-BIT = Kaufman Brief Intelligence Test. Mullen = Infant - Mullen Scales of Early Development. NEPSY = A developmental NEuroPSYchological assessment. PPVT = Peabody Picture Vocabulary Test. RAPT = Renfrew Action Picture Test. RBMT = Rivermead Behavioral Memory Test. RO = Rey Osterrieth (complex figure task). RPM = Ravens Progressive Matrices. RWFT = Renfrew Word Finding Test. S-B = Stanford Binet. TROG(-E) = Test for the Reception of Grammar(-Electronic). WAIS = Wechsler Adult Intelligence Scales (various editions). WASI = Wechsler Abbreviated Scale of Intelligence. WECHS = WAIS/WISC. WISC = Wechsler Intelligence Scale for Children (various editions). WMTB = Working Memory Test Battery.

VQ = Verbal (Intelligence) Quotient. PQ = Performance (Intelligence) Quotient (WAIS/WISC). NVQ = Nonverbal (Intelligence) Quotient (tests other than WAIS/WISC). FSQ = Full Scale (Intelligence) Quotient. SS = Standard score. VMA = Verbal Mental Age. NVMA = Nonverbal Mental Age.

I = Immediate. D = Delayed

MI = Memory illusion i.e. false positive response on tests of related materials

FP = False positive response on tests of unrelated stimuli

N/A = Not available

N/T = Not tested

PAL = Paired associate learning

In the right-hand column of all Tables, **bold** type is used to indicate findings of impaired or anomalous performance and *italics* are used to indicate findings that may be questionable, although the studies cited satisfy methodological criteria for inclusion in this review.

In all Tables, studies are listed in order approximating to the age of the ASD participant group tested.

[Type text]

**Table 1 Nondeclarative memory in HFA**

Author(s)	Groups	No.	Age (yrs) $\mu$ (sd/range)	Ability $\mu$ (sd/range)	Stimuli/task	Main findings
Renner et al. (2000)	HFA [DSM-IV]	14	10.2 (2.4)	IQ 99.3 (11.2) [K-BIT]	Perceptual priming	Unimpaired
	NT	14	9.4 (2.0)	IQ 110.7(8.1) [K-BIT]		
Heaton et al. (2007)*	HFA [DSM-IV]	20	12.6 (7-19)	NVQ 92 (55-125) [RPM]	Implicit musical learning	Unimpaired
	NT	20	11.6 (9.6-15.9)	NVQ 87 (67-131) [RPM]		
Molesworth et al. (2005)	HFA [DSM-IV]	15	11.7 (1.7)	VMA 11.7(3.0) [BPVS] NVQ >100 (RPM)	Implicit category formation	Unimpaired
	NT	15	11.7 (1.8)	VMA 11.5(3.0) [BPVS] NVQ c.100 (RPM)		
Barnes et al. (2008)	HFA [DSM-IV +ADI-R+ADOS]	14	11.6 (1.7)	FSQ 110(12.6) [WISC]	Implicit sequence learning	Unimpaired
	NT	14	11.0 (1.8)	FSQ 116 (13.8) [WISC]		
Brown et al. (2010)	HFA [DSM-IV +ADI-R]	26	11.5 (1.2)	VQ 102.2 (13.5) [WISC] PQ 102.2 (15.7) [WISC]	Contextual cueing Motor sequence learning Artificial grammar learning Probabilistic learning	Unimpaired
	NT	26	11.8 (1.6)	VQ 104.3 (10.5) [WISC] PQ 104.1 (10.9) [WISC]		
Nemeth et al. (2010)	HFA [DSM-IV +ADI+ADOS]	13	11.8 (3.1)	FSIQ 93.2 (20.7) [WISC]	Alternating serial reaction time task	Unimpaired (relative to both groups)
	NT IQ-matched	13	9.2 (2.6)	FSIQ 96.5 (17.7) [WISC]		
	NT age-matched	14	11.6 (3.3)	FSIQ 109.1 (12.8) [WISC]		
Sears et al. (1994)	HFA [DSM-III(R)]	11	12.2 (7-22)	PQ 106 (N/A) (WECHS)	Classical conditioning	Unimpaired
	NT	11	12.7 (6-23)	PQ 115 (N/A) (WECHS)		
Soulières et al. (2011)	HFA [DSM-IV +ADI-R+ADOS]	16	17.8 (11-29)	VQ 109.4 (81-132) [WISC] PQ 105.1 (77-126) [WISC]	Implicit category formation	Unimpaired
	NT	15	16.7 (11-27)	VQ 109.2 (91-128) [WISC] PQ 109.8 (87-128) [WISC]		
Travers et al. (2010)	HFA [DSM-IV +ADI-R+ADOS]	15	19.0 (2.11)	FSQ 103 (17.8) [K-BIT]	Implicit sequence learning	Unimpaired
	NT	15	19.0 (2.1)	FSQ 100 (14.1) [K-BIT]		
Toichi (2008)	HFA [DSM-IV +CARS]	18	23 (5.2)	VQ 95.3(17.9) [WAIS] PQ 92.1(14.8) [WAIS]	Semantic priming	Unimpaired
	NT	18	24.5 (7.9)	VQ 97.2(19.5) [WAIS] PQ 91.2(19) [WAIS]		
Bowler et al. (1997)	HFA[ICD-10]	16	31.2 (11.0)	VQ 99.4 (16.7) [WAIS] PQ 96.3 (13.2) [WAIS]	Perceptual/ conceptual priming	Unimpaired
	NT	16	33.3 (11.4)	VQ 86.3 (19.2) [WAIS] PQ 96.1 (10.3) [WAIS]		
Gardiner et al. (2003)	AS [ICD-10]	16	31.6 (8.9)	VQ 95.8 (17.4) [WAIS] PQ 84.6 (13.6) [WAIS]	Perceptual priming	Unimpaired
	NT	14	31.3 (7.1)	VQ 94.5 (12.9) [WAIS] PQ 88.3 (17.1) [WAIS]		
Gardiner et al. (2003)	AS [ICD-10]	10	28.3 (5.3)	VQ 89.9(16.8) [WAIS] PQ 86 (18.0) [WAIS]	Conceptual priming	Unimpaired
	NT	10	29.1 (4.6)	VQ 93.1 (13.4) [WAIS] PQ 86.1 (11.0) [WAIS]		
Bott et al., (2006)	HFA	10	30 (20-62)	VMA 27.1 (17-36) [WAIS] NVMA 19.5 (7-31) [WAIS]	Implicit category formation	Unimpaired
	NT	17	21 (19-48)	VMA 27.1 (20-35) [WAIS] NVMA19.5(17-31) [WAIS]		
Gaigg & Bowler (2007)	AS [ICD-10]	14	29.7 (10.2)	FSQ 111 (17.3) [WAIS]	Fear conditioning	<b>Impaired</b>
	NT	14	30.4 (12.2)	FSQ 109 (12.7) [WAIS]		

\* Three participants out of 20 in each group had NVQs < 70.

**Table 2 Declarative memory in HFA as tested by recognition**

Author(s)	Groups	No	Age (yrs) $\mu$ (sd/range)	Ability $\mu$ (sd/range)	Stimuli/task	Main findings
Boucher et al. (2008a)	HFA [DSM-IV +ASSQ]	28	8.3 (1.4)	VMA 9.11 [BPVS] PQ 110.3 (20.7) [WASI]	Pictures of coloured shapes - D	Unimpaired
	NT	23	8.0 (1.5)	VMA 8.0 [BPVS] PQ 100.2 (15.3) [WASI]		
Joseph et al. (2005)	HFA [DSM-IV +ADI-R +ADOS]**	24	8;11 (2;4)	VQ 94 (19) [DAS] NVQ 99 (20) [DAS]	Pictures (common objects: same category; different categories)	Unimpaired (both conditions)
	NT	24	8;11 (2;2)	VQ 89 (12) [DAS] NVQ 94 (14) [DAS]		
Bigham et al. (2010)	HFA [DSM-IV +CARS]	18	9.2 (6;4-13;4)	VQ 103.7 (10.6) [BPVS]	Meaningless shapes - D	Unimpaired
	NT	29	8.0 (5;0-10;4)	VQ 109.4 (11.6) [BPVS]		
Lind (2008)	HFA	25	9.4 (2.7)	VQ 84.9 (8.1)	Pictures (common objects) - D	Unimpaired
	NT	25	9.0 (3.8)	VQ 87.6 (15.0)		
Williams et al. (2006a)	HF-LN [DSM-IV +ADI+ADOS]	38	11.7 (2.5)	VQ 106.4 (16.0) [WISC] PQ 100.6 (14.2) [WISC]	Story content – D Complex scenes – I (WRAML)	Unimpaired <b>Impaired</b>
	NT	38	12.2 (2.2)	VQ 107.3 (8.1) [WISC] PQ 106 (10.4) [WISC]		
Renner et al. (2000)	HFA [DSM-IV]	14	10.2 (2.4)	IQ 99.3 (11.2)[K-BIT]	Pictures (common objects) - D	Unimpaired
	NT	14	9.4 (2.0)	IQ 110.7(8.1) [K-BIT]		
Salmond et al. (2005)	HFA [DSM-IV +ASAS]	14	12.9 (0.7)	VQ 102 (4.0) [WISC]	Spoken words (unrelated)	Unimpaired
	NT	18	12.6 (0.7)	VQ 104 (2.0) [WISC]	Word pairs (unrelated) Stories (CMS) Pictures of common objects (RBMT) Faces (RBMT)	Unimpaired Unimpaired Unimpaired Unimpaired
Buitelaar et al. (1999)	HFA [DSM-IV +CARS]	20	12;6 (3;2)	VQ 104.1 (15.4) [WISC] PQ 99.5 (22.8) [WISC]	Meaningless patterns - I (Benton Vis. Rec. Test)	Unimpaired
	Non-ASD psychiatric	20	12;4 (3;2)	VQ 100.1 (13.8) [WISC] PQ 99.9 (15.1) [WISC]		
Henderson et al. (2009)	HFA [DSM-IV +ASSQ]	28	12.5 (2.8)	VQ 101.1 (14.8) [WASI] PQ 105.2 (17.8) [WASI]	Written words (various encoding conditions; unexpected test) - I	Unimpaired (physical feature encoding) <b>Impaired</b> (self-ref. encoding)
	NT	31	13.3 (2.1)	VQ 105.0 (14.8) [WASI] PQ 99.2 (14.4) [WASI]		
Bowler et al. (2004)	Young AS [ICD-10]	16	13.5 (1.1)	VQ 100.8 (20.7) [BPVS]	Written + spoken words (unrelated; various encoding conditions) - D	<b>Impaired</b> (combined AS groups - scores corrected for FPs)
	Young NT	16	13.4 (0.7)	VQ 94.6 (18.3) [BPVS]		
	Adult AS [ICD-10]	16	34.5 (6.7)	VQ 100.1 (14.9) [WAIS]		
	Adult NT	16	33.4 (4.6)	VQ 97 (15.5) [WAIS]		
Kamio & Toichi (2007)	HF-LN [ICD-10]	13	16.4 (4.4)	VQ 102.5 (17.9) [WAIS] FSQ 97.8 (16.3)[WAIS]	Spoken sentences - D	HF-LN unimpaired ( <b>fewer MIs</b> ) AS unimpaired (same MIs)
	AS [ICD-10]	15	19.0 (5.6)	VQ 105.4 (16.0) [WAIS] FSQ 99.5 (15.6)[WAIS]		
	NT	15	20.3 (7.0)	VQ 102.9 (14.3)[WAIS] FSQ101.9 (14.5)[WAIS]		
Hillier et al. (2007)	HFA [DSM-IV +ADI-R]*	14	21.7 (6.4)	VQ 116.4 (17.4) [WAIS] PQ 104.8 (16.2) [WAIS]	Geometric shapes - I	<b>Superior (fewer FPs)</b> Unimpaired (same MIs)
	NT	14	23.3 (3.5)	VQ 112.3 (8.5) [WAIS] PQ 109 (14.4) [WAIS]	Spoken words (semantically related) - I	
Boucher et al. (2005)	HFA [DSM-IV +WADC]	10	23;9 (7;9)	VQ 105.5 (20.2) [WAIS] PQ 90.3 (19.3) [WAIS]	Written words (unrelated) – D	Unimpaired Unimpaired <b>Impaired</b>
	NT	10	24.2 (8;1)	VQ 104.4 (13.2) [WAIS] PQ 97.5 (16.9) [WAIS]	Pictures of common objects - D Unfamiliar faces - D	
Bowler et al. (2000b) Expt. 2	AS [ICD-10]	10	28.5 (8.6)	VQ 89.2 (9.7) [WAIS] PQ 82.7 (8.9) [WAIS]	Written words (related) - I	Unimpaired (same FPs)
	NT	10	26.1 (9.0)	VQ 92.8 (15.4) [WAIS] PQ 88.5 (18.1) (WAIS		

[Type text]

Beverdort et al. (2000)	HFA [DSM-IV +ADI-R]	8	31.8 (8.6)	VQ 114 (19.7) [WAIS] PQ 106.1 (16.0) [WAIS]	Spoken words (semantically related) – I	Unimpaired (fewer MIs)
	NT	16	31.4 (12.1)	VQ 114.9 (16.4) [WAIS] PQ 107.4 (10.3) [WAIS]		
Bowler et al. (2000a)	HFA [ICD-10]	16	30.9 (6.3)	VQ 96.5 (14.4) [WAIS] PQ 90.1 (11.8) [WAIS]	Written words (unrelated) - D	Unimpaired
	NT	15	31.1 (5.6)	VQ 96.5 (14.4) [WAIS] PQ 90.1 (11.8) [WAIS]		
Ambery et al. (2006)	AS [ICD-10 +ADI-R+ADOS]	27	37.6 (14.6)	VQ 106.1 (15.7) [WAIS] PQ 103.7 (19.2) [WAIS]	Pictures of doors Proper names (Doors and People test)	Unimpaired Unimpaired
	NT	20	33.5 (12.0)	VQ 107.1 (13.1) [WAIS] PQ 109.4 (18.5) [WAIS]		
Bowler et al. (2010)	HFA [DSM-IV-TR + ADOS]***	19	36.5 (13.7)	VQ 107.3 (14.5) [WAIS] PQ 100.9 (14.2) [WAIS]	Objects, locations, colors Separately - D Object-location/object-color combinations - D	Unimpaired <b>Impaired</b>
	NT	19	37.5 (12.4)	VQ 107.5 (14.0) [WAIS] PQ 106.1 (13.0) [WAIS]		
Bowler et al. (2008a)	HFA [DSM-IV]	20	31.8 (11.2)	VQ 100.4 (17.9) [WAIS] PQ 94.8 (19.0) [WAIS]	Written words (unrelated; in context of semantically related/unrelated words) - D	Unimpaired (both conditions)
	NT	20	34.5 (11.9)	VQ 101.1 (12.4) [WAIS] PQ 101.1 (13.3) [WAIS]		
Toichi et al. (2002)	HFA [DSM-IV +CARS]	18	23.0 (5.2)	VQ 95.3 (17.9) [WAIS] PQ 92.1 (14.8) [WAIS]	Written words (various encoding conditions; unexpected test) - I	<b>Superior</b> (phonological encoding) Unimpaired (semantic encoding) <b>Impaired</b> (self-ref. encoding)
	NT	18	24.5 (7.9)	VQ 97.2 (19.5) [WAIS] PQ 91.2 (19.0) [WAIS]		
Lombardo et al. (2007)	HFA [DSM-IV + AQ]	30	29.1 (7.4)	VQ 116.1 (12.8) [WAIS] PQ 114.2 (14.2) [WAIS]	Written words (various encoding conditions; unexpected test) - D	Unimpaired (physical feature encoding) <b>Impaired</b> (self-ref. encoding)
	NT	30	29.9 (7.8)	VQ 116.5 (8.7) [WAIS] PQ 114.4 (10.1) [WAIS]		
Williams et al. (2005a)	HF-LN [DSM-IV +ADI+ADOS]	29	28.7 (10.4)	VQ 108.8 (14.9) [WAIS] PQ 100.8 (13.9) [WAIS]	Unfamiliar faces – I & D	<b>Impaired</b> (both conditions)
	NT	34	26.5 (10.2)	VQ 108.1 (10.1) [WAIS] PQ 109.8 (12.5) [WAIS]		

\* This group included individuals with AS, with HF-LN, and also PDD-NOS, but numbers of each diagnosis were not given.

\*\* Ability ranges given in the paper show that a small number of children in both groups had VQs < 70

\*\*\* Groups assessed for recognition of objects, locations, and colours separately, as opposed to object-location-colour combinations, overlapped and had similar ages and abilities, but were not identical. Details given here are for the groups tested for recognition of object-location-colour combinations.

**Table 3 Declarative memory in M-LFA as tested by recognition**

Author(s)	Groups	No.	Age (yrs) $\mu$ (sd)/range)	Ability $\mu$ (sd)/range)	Stimuli/task	Main findings
Klin et al. (1999)	M-LFA	34	7.4 (2.9)	VMA 3.7 (1.0) [K-ABC] NVMA 4.5 (1.5) [K-ABC]	Unfamiliar faces [K-ABC]	<b>Impaired</b>
	Non-ASD psychiatric	34	6.3 (2.2)	VMA 4.9 (1.5) [K-ABC] NVMA 4.7 (1.4) [K-ABC]		
Wilson et al. (2007)	M-LFA	17	8;7(6;11-10;10)	VMA 5;6 (3;6 – 8;9) [APT]	Familiar faces (whole, inner, outer)	<b>Impaired</b> (relative to age- matched NT group) Unimpaired (relative to ID group)
	ID	17	8;9 (6;1–10.5)	VMA 5;6 (3;0-8;10) [APT]		
	NT	17	8;3 (6;6–10.7)	N/T		
Boucher et al. (1998)	M-LFA [DSM-IV]	19	8;8 (1.3)	VMA 4;1 (3;6 – 6;4) [APT]	Familiar faces	<b>Impaired</b>
	Mixed ID/SLI*	20	7;10 (1;6)	VMA 4;9 (3;6 - 6;0) [APT]		
Hill & Russell (2002)	M-LFA[DSM-IV]	20	9.8 (1.9)	VQ 61.9 (16.8) [BPVS]	Object pairs (unexpected test) - I	Unimpaired
	ID	20	9.6 (1.1)	VQ 62.3 (13.6) [BPVS]		
	NT	20	6.0 (0.7)	VQ 99.4 (26.2) [BPVS]		
Summers & Craik (1994)	M-LFA [DSM-III-R]	8	11;5 (2;3)	VMA 5;4 (0;11)	Spoken words - D	<b>Impaired</b>
	NT	8	5;0 (1;2)	VMA5;0 (1;2)		
Hauck et al. (1998)	M-LFA [DSM-III-R]	24	9.6 (1.7)	VMA 5.3 (2.3) [PPVT]	Pictures of common objects - D	Unimpaired
	NT	34	4.7 (0.8)	VMA 5.5 (1.5) [PPVT]		
		M-LFA [DSM-IV]	19	9;11 (1;3)	VMA 6;11 (1;6) [BPVS]	Unfamiliar faces (parts only)
ID	15	10;4 (1;3)	VMA 6;6 (1;6) [BPVS]			
NT 1	20	6;7 (0;6)	N/T			
NT 2	21	10;5 (0;6)	N/T			
Boucher & Warrington (1976)	M-LFA [Rutter, 1968]	10	12;8 (8;5 – 16;11)	VMA 5;5(3;9– 6;3) [PPVT] NVQ 9;3 (5;0–11;0) [CPM]	Pictures of common objects - D	<b>Impaired</b> (relative to both groups)
	ID	10	9;3(7;1 – 13;8)	VMA 5;7(3;8– 7;6) [PPVT] NVQ 8;9 (8;3–10;9) [CPM]		
	NT	10	12;8(8;2 – 16;1)	N/T N/T		
Lind (2008)	M-LFA	11	12.3 (3.6)	VQ 53.8 (11.2)	Pictures of common objects - D	<b>Impaired</b> (relative to all other groups)
	ID	11	12.2 (3.6)	VQ 55.9 (13.4)		
	HFA	25	9.4 (2.7)	VQ 84.9 (8.1)		
	NT	25	9.0 (3.8)	VQ 87.6 (15.0)		
Boucher et al. (2008a)	M-LFA [DSM-IV + CARS]	30	14.6 (1.8)	VMA 7.6 [BPVS] PQ 71.9 (16.7) [WASI]	Pictures of coloured shapes - D	<b>Impaired</b> (relative to NT and HFA groups) Unimpaired (relative to ID group)
	NT	23	8.0 (1.5)	VMA 8.0 [BPVS] PQ 100.2 (15.3) [WASI]		
	HFA [DSM-IV +ASSQ]	28	8.3 (1.4)	VMA 9.11 [BPVS] PQ 110.3 (20.7) [WASI]		
	ID	24	14.3 (1.2)	VMA 7.7 [BPVS] PQ 68.0 (12.2) [WASI]		
Boucher & Lewis (1992)	M-LFA [Rutter, 1968]	16	13;6 (2;8)	VMA c. 6;11 [TROG]	Pictures of buildings - I	Unimpaired
	ID	16	11;2 (1;11)	VMA c. 6;1 [TROG]	Unfamiliar faces - I	<b>Impaired</b>

\* The M-LFA, ID and SLI children taking part in this experiment attended one or other of two schools for children with communication disorders, and diagnoses were made by relevant clinicians. Ages and ability levels were calculated by combining means and sds for participants from the two schools.



**Table 4 Declarative memory in HFA as tested by free recall**

Author(s)	Groups	No.	Age (yrs) μ(sd)/range	Ability μ(sd)/range	Stimuli/task	Main findings
Iwanaga et al. (2000)	AS [DSM-IV] HF-LN [DSM-IV] Comparison with standardized norms	10 15	Range 4;10-6;2 Range 5;1-6;2	IQ 93.3 (10.2)[Tanaka-Binet] IQ 91.7 (13.9)[Tanaka-Binet]	Sentence repetition - I	<b>Impaired</b> (HF-LN more severely than AS)
Verté et al. (2006)	AS [DSM-IV-TR +ADI-R] HF-LN [DSM-IV-TR+ADI-R] NT	37 50 47	8.7 (1.9) 8.5 (2.1) 9.4 (1.6)	VQ 105.2 (16.3) [WISC] PQ 104.0 (17.8) [WISC] VQ 93.1 (18.0) [WISC] PQ 104.0 (15.9) [WISC] VQ 113.6 (10.4) [WISC] PQ 108.5 (11.9) [WISC]	Meaningless patterns - D (Benton Vis. Retention Test)	<b>Impaired</b> (both groups, covarying for IQ)
Renner et al. (2000)	HFA [DSM-IV] NT	14 14	10.2 (2.4) 9.4 (2.0)	IQ 99.3 (11.2)[K-BIT] IQ 110.7(8.1)[K-BIT]	Pictures of common objects - D	Unimpaired <b>(reduced primacy)</b>
Botting & Conti-Ramsden (2003)	HFA [DSM-IV + CARS] SLI*** Comparison with standardized norms	13 29	10;10 (10;2-12;6) 10;10 (10;2-11;9)	PQ 90 (76-107) [WISC] PQ 85 (76-90) [WISC]	Sentence repetition (from CELF) - I	<b>Impaired</b> (relative to norms) Unimpaired (relative to SLI group)
Whitehouse et al. (2008)	HFA [DSM-IV + ADOS] SLI Comparison with standardized norms	16 34	10;8 (2;7) 11;10 (2;3)	VQ 101.8 (9.6) [TROG-E] PQ 110.3 (14.9) [WASI] VQ 91.7 (13.9) [TROG-E] PQ 100.4 (13.2) [WASI]	Sentence repetition (from NEPSY) - I	Unimpaired (relative to norms) <b>Superior</b> (to SLI group)
Williams et al. (2006a)	HF-LN [DSM-IV +ADI+ADOS] NT	38 38	11.7 (2.5) 12.2 (2.2)	VQ 106.4 (16.0) [WISC] PQ 100.6 (14.2) [WISC] VQ 107.3 (8.1) [WISC] PQ 106 (10.4) [WISC]	Number-letter lists – I Unrelated words – I & D Sentence repetition – I Story recall – I & D Figure reproduction – D Geometric shape reproduction - D	Unimpaired Unimpaired <b>Impaired</b> <b>Impaired</b> <b>Impaired</b> <b>Impaired</b>
Buitelaar et al. (1999)	HFA [DSM-IV +CARS] Non-ASD psychiatric	20 20	12;6 (3;2) 12;4 (3;2)	VQ 104.1 (15.4) [WISC] PQ 99.5 (22.8) [WISC] VQ 100.1 (13.8) [WISC] PQ 99.9 (15.1) [WISC]	Written words (unrelated) over trials	Unimpaired
Salmond et al. (2005)	HFA [DSM-IV +ASAS] NT	14 18	12.9 (0.7) 12.6 (0.7)	VQ 102 (4.0) [WISC] VQ 104 (2.0) [WISC]	Spoken words (unrelated) - I over trials (CMS) Story recall (RBMT) – I & D	Unimpaired <b>Impaired</b> <i>Unimpaired (trends)</i>
McCrory et al. (2007)	AS [ICD-10] NT	24 27	13.0 (1.2) 12.6 (1.1)	VQ 103.0 (17.3) [WASI] VQ 101.7 (12.1) [WASI]	Naturalistic events involving other people	<b>Impaired</b>
Mottron et al. (2001)	HFA [DSM-IV +ADI-R] NT	14 14	17.1 (7.3) 16.0 (6.8)	VQ 103.8 (15.0) [mixed]* PQ 106.7 (12.3) [mixed]* VQ 108.9 (7.3) [mixed]* N/T	Written-spoken words (unrelated; various encoding conditions) - D	Unimpaired (all encoding conditions)
Bowler et al. (2009a)	HFA [DSM-IV-TR +ADI+ADOS] NT	21 21	19 (8.7) 16 (3.7)	VQ106 (16.0) [WAIS] PQ 111 (12.8) [WAIS] VQ110 (10.2)[WAIS] PQ 108 (10.7) [WAIS]	Written words (unrelated) over trials	Unimpaired all trials (typical subjective organization over trials; <b>reduced primacy</b> )
Minshew et al. (1992)	HF-LN [DSM-IV +ADI+ADOS] NT (matched pairs)	15 15	21.1 (8.0) 21.3 (8.3)	VQ 98.5 (21.6) [WAIS] PQ 92.9 (10.7) [WAIS] VQ 99.1 (14.2) [WAIS] PQ 96.5 (12.3) [WAIS]	Unrelated words (CVLT) Trial 1 – I over trials	Unimpaired Unimpaired
Minshew & Goldstein (2001)	HF-LN [DSM-IV + ADI+ADOS] NT	52 40	22.3 (0.6) 21.6 (9.9)	VQ 95.0 (17.6) [WAIS] PQ 91.5 (13.0) [WAIS] VQ 97.2 (14.6) [WAIS] PQ 95.8 (12.1) [WAIS]	Unrelated words Trial 1 – I over trials (CVLT)	<b>Impaired</b> <b>Impaired</b>
Boucher et al. (2005)	HFA [DSM-IV + WADC] NT	10 10	23;9 (7;9) 24.2 (8;1)	VQ 105.5 (20.2) [WAIS] PQ 90.3 (19.3) [WAIS] VQ 104.4 (13.2) [WAIS] PQ 97.5 (16.9) [WAIS]	Story recall – I & D Figure reproduction – I and D (RO)	Unimpaired Unimpaired (both conditions)
Bowler et al. (1997)	HFA [ICD-10]	16	31.2 (11.0)	VQ 99.4 (16.7) [WAIS] PQ 86.3 (19.2) [WAIS]	Unrelated words – I	Unimpaired

[Type text]

	NT	16	33.3 (11.4)	VQ 96.3 (13.2) [WAIS] PQ 96.1 (10.3) [WAIS]	Semantically related words) - I	<b>Impaired</b>
Bowler et al. (2000b) Expt. 1	HFA [ICD-10]	10	29.4 (8.3)	VQ 90.8 (78-121) [WAIS] PQ 90.1 (59-130) [WAIS]	Spoken words (semantically related) - I	<b>Impaired (excess FPs, but not MIs)</b>
	NT	15	34.0 (8.1)	VQ 94.3 (72-119) [WAIS] PQ 90.1 (76-106) [WAIS]		
Ambery et al. (2006)	AS [ICD-10]	27	37.6 (14.6)	VQ 106.1 (15.7) [WAIS] PQ 103.7 (19.2) [WAIS]	Meaningless shape reproduction ('Doors and People') Story recall- I & D (WMS)	Unimpaired Unimpaired
	NT	20	33.5 (12.0)	VQ 107.1 (13.1) [WAIS] PQ 109.4 (18.5) [WAIS]		
Smith et al. (2007)	AS [ICD-10]	12	40.1 (10.8)	VQ 103.6 (18.3) [WAIS] PQ 104.2 (18.4) [WAIS]	Written-spoken words unrelated - I + rehearsal semantically related I + rehearsal phonologically related I + rehearsal	Unimpaired <b>Impaired</b> <b>Impaired</b> <b>Impaired</b> <b>Impaired</b>
	NT	12	39.9 (12.4)	VQ 104.6 (17.2) [WAIS] PQ 106.3 (12.9) [WAIS]		
Bowler et al. (2008a)	HFA [DSM-IV]	20	35.7 (13.7)	VQ 107.4 (18.1) [WAIS] PQ 108.6 (21.4) [WAIS]	Written words (unrelated: in semantically related/unrelated word contexts) - D	Unimpaired (umrelated contexts) <b>Impaired</b> (related contexts)
	NT	20	34.4 (12.2)	VQ 107.0 (14.3) [WAIS] PQ 106.3 (18.6) [WAIS]		
Bowler et al. (2008b)	AS [ICD-10]	16	39 (13.1)	VQ102 (13.1) [WAIS] PQ 99 (17.4) [WAIS]	Written words (unrelated) over trials	Unimpaired all trials <b>(but atypical subjective organisation)</b>
	NT	16	34 (12.3)	VQ103 (11.7) [WAIS] PQ 103 (10.0) [WAIS]		
Bowler et al. (2009b)	HFA [DSM-IV-TR + ADOS]	20	33.0 (13.1)	VQ 107.8 (15.5) [WAIS] PQ 107.8 (16.6) [WAIS]	Categorically organized word lists	Unimpaired <b>(reduced categorical organization)</b>
	NT	20	30.4 (10.0)	VQ 107.4 (14.7) [WAIS] PQ 105.1 (12.5) [WAIS]		
Gaigg et al. (2008)	HFA [DSM-IV]	20	34.1 (12.9)	VQ 101 (17.0) [WAIS] PQ 98 (20.0) [WAIS]	Written words from various categories - D As above + category encoding at study - D	<b>Impaired</b> Unimpaired
	NT	20	33.6 (10.8)	VQ 103 (15.5) [WAIS] PQ 103.5 (13.0) [WAIS]		
Williams et al. (2005a)	HF-LN [DSM-IV +ADI+ADOS]	29	28.7 (10.4)	VQ 108.8 (14.9) [mixed]* PQ 100.8 (13.9) [mixed] *	Story recall - I & D	Unimpaired (both conditions)
	NT	34	26.5 (10.2)	VQ 108.1 (10.1) [mixed]* PQ 109.8 (12.5) [mixed] *		
Gaigg & Bowler (2008)	HFA [DSM-IV +ADOS]	18	32.8 (12.4)	VQ 105.2 (14.7) [WAIS] PQ 106.4 (17.5) [WAIS]	Emotionally arousing words (on screen)	<b>Impaired</b>
	NT	18	33.2 (13.6)	VQ 105.3 (12.8) [WAIS] PQ 104.8 (10.8) [WAIS]		

\* Participants were variously tested on the WISC, WAIS and S-B tests in Mottron et al.'s study, and on the WAIS and Kaufman Test of Educational Achievement in the Williams et al. study.

**Table 5 Declarative memory in M-LFA as tested by free recall**

Author(s)	Groups	No.	Age (yrs) μ(sd/range)	Ability μ(sd/range)	Stimuli/task	Main findings
Fein et al. (1996)	M-LFA [DSM-III-R + WADC] ID	125	5;0 (1;4)	Vocab. score:27.6(6.0) [S-B]** Comp. score: 27.0(4.8) [S-B]** NVQ 45.6(19.4) [S-B/Bayley]	Sentence repetition (from S-B) - I Story recall (McCarthy Verb. Mem. II)	<b>Impaired</b>  <b>Impaired</b>
		110	4.7 (1.1)	Vocab. score:32.9(6.0) [S-B]** Comp. score: 33.2(8.0) [S-B]** NVQ 55.5 (19.9) [S-B/Bayley]		
Frith (1970)	M-LFA [Rutter,1968] ID NT	10	Range: 7;0 – 13;0	VMA 4;6 [PPVT]	Spoken word strings (structured, non-meaningful) Serial recall - I	<b>Impaired</b> (relative to both groups) <b>(enhanced recency)</b>
		10	Range: 10; - 16;0	VMA 4;6 [PPVT]		
		10	Range: 4;0 – 5;0	VMA 4;6 [PPVT]		
Boucher & Warrington (1976)	M-LFA [Rutter,1968] ID NT	11	10;9 (7;5–14;4)	VMA 5;4 (4;0–7;1) [PPVT] NVMA 9;4 (7;6–11;6) [CPM]	Spoken words (unrelated) - D	<b>Impaired</b> (relative to both groups)
		11	10;8 (7;2–15;4)	VMA 5;9 (4;6–7;6) [PPVT] NVMA 8;6 (6;9–10;3) [CPM]		
		11	10;8 (7;7–15;4)	N/T		
Summers & Craik (1994)	M-LFA [DSM-III-R] NT	8	11;5 (2;3)	VMA 5;4 (0;11)	Spoken words (unrelated) - I	<b>Impaired</b>
		8	5;0 (1;2)	VMA5;0 (1;2)		
Fyffe & Prior (1978)	M-LFA [Rutter,1974] ID NT	14	11;3 (2;4)	VQ 58 (13) [mixed]* PQ 66 (16) [mixed]*	Spoken words (unrelated) free recall – I Spoken sentences serial recall - I	Unimpaired (relative to both groups)  <b>Impaired</b> (relative to both groups) <b>(enhanced recency)</b>
		14	12;2 (3;8)	VQ 59 (12) [mixed]* PQ 66 (12) [mixed]*		
		14	6;9 (0;3)	N/T		
Boucher (1981a)	M-LFA [Rutter,1974] ID	11	11.6 (7.3–14.0)	VMA 6.0 (4.0–7.0) [CLCT] NVMA 9.3 (5.6–11.6) [CPM]	Spoken words (unrelated) - I	Unimpaired <b>(reduced primacy; enhanced recency)</b>
		11	11.4 (8.0–15.4)	VMA 6.0 (3.6–7.0) [CLCT] NVMA 9.3 (6.6–11.3) [CPM]		
Tager-Flusberg (1991)	M-LFA [DSM-III] ID NT	15	11.2 (3.3)	VMA 5.2 (1.4) [PPVT] NVMA 9.6 (3.0) [CPM]	Spoken words (unrelated) – I  Spoken words (semantically related) - I	Unimpaired (relative to both groups)  <b>Impaired</b> (relative to both groups; <b>limited clustering)</b>
		15	10.8 (3.0)	VMA 5.0 (1.8) [PPVT] NVMA 6.0 (2.0) [CPM]		
		15	4.7 (0.10)	VMA 4.9 (1.3) [PPVT] NVMA N/T		
Boucher & Lewis (1989)	M-LFA [Rutter,1978] ID	12	13.1 (10;11–15;0)	VMA 7;6 *** [RWF] NVMA 11;6*** [CPM]	Instructions: spoken I & D demonstrated I & D written: no memory	<b>Impaired</b> <b>Impaired</b> Unimpaired
		12	13;2 (10;11–15;4)	VMA 7;1 *** [RWF] NVMA 11;3*** [CPM]		
Boucher (1981b)	M-LFA [Rutter,1974] ID	10	13;2 (10;10–16;0)	VMA 7;2 (4;8 – 10;7) [PPVT] VMA 6;0 (4;0 – 7;0) [CLCT] NVMA 10;6 (5;0–11;0) [CPM]	Past activities own - D	<b>Impaired</b>
		10	13;3 (9;10–16;2)	VMA 9;0 (5;11–12;3) [PPVT] VMA 6;4 (6;0 – 7;0) [CLCT] NVMA 10;3 (5;0–11;0) [CPM]		
Millward et al. (2000)	M-LFA [DSM-III-R] NT	12	13.1 (11.8–15.8)	VMA 6.3 (4.5 – 10.3) [BPVS]	Past activities: own - D others' - D	<b>Impaired</b> <i>Unimpaired</i> (see text)
		12	6.3 (4.5–10.3)	VMA 6.2 (4.9 – 7.1) [BPVS]		
Boucher (1978)	M-LFA [Rutter,1968] NT	10	14;2 (9;10–18;0)	VMA 5;9 (4;7 – 9;5) [PPVT] NVMA 8;9 (5;6–11;0)[CPM]	Written/spoken words (unrelated) - I	Unimpaired <b>(age-appropriate recency)</b>
		10	13;11 (9;5–16;7)	N/T		
Hare et al. (2007)	M-LFA [DSM-IV/ICD-10] ID	12	27.7 (6.3)	VMA 6.1 (1.9) [BPVS] VMA 5.3 (1.2) [TROG]	Past activities: own - D others' - D	<i>Unimpaired</i> <i>Unimpaired</i> (floor effects)
		14	49.6 (10.2)	VMA 6.8 (1.5) [BPVS] VMA 4.5 (0.5) [TROG]		

\* IQ scores were obtained from WISC, Binet, Leiter performance scale, and PPVT.

\*\* Mean score on these tests is 50 (sd 8.0).

\*\*\* Ranges are given in terms of raw scores in the report, and are closely similar in the two groups.

**Table 6 Declarative memory in HFA as tested by cued recall**

Author(s)	Groups	No.	Age (yrs) μ(sd/range)	Ability μ(sd/range)	Stimuli/task	Outcomes
Williams et al. (2006b)	HF-LN [DSM-IV +ADI+ADOS]	38	11.4 (2.2)	VQ 105.5 (16.1) [WISC] PQ 102.1 (14.6) [WISC]	Sound-symbol associations Design-location associations	Unimpaired Unimpaired
	NT	38	11.8 (2.2)	VQ 107.9 (8.2) [WISC] PQ 106 (8.4) [WISC]		
Brown et al. (2010)	HFA [DSM-IV +ADI-R]	26	11.5 (1.2)	VQ 102.2 (13.5) [WISC] PQ 102.2 (15.7) [WISC]	PAL (visual presentation, typed response)	Unimpaired (ANOVA) <b>Impaired</b> (equivalence analysis)
	NT	26	11.8 (1.6)	VQ 104.3 (10.5) [WISC] PQ 104.1 (10.9) [WISC]		
Salmond et al. (2005)	HFA [DSM-IV +ASAS]	14	12.9 (0.7)	VQ 102 (4.0) [WISC]	Route recall – I & D	Unimpaired
	NT	18	12.6 (0.7)	VQ 104 (2.0) [WISC]	Face-name associations	<b>Impaired</b>
McCrary et al. (2007)	AS [ICD-10]	24	13.0 (1.2)	VQ 103.0 (17.3) [WASI]	Naturalistic events involving other people, cued by questioning	Unimpaired
	NT	27	12.6 (1.1)	VQ 101.7 (12.1) [WASI]		
Mottron et al. (2001)	HFA [DSM-IV +ADI-R]	14	17.1 (7.3)	VQ 103.8 (15.0) [mixed]* PQ 106.7 (12.3) [mixed]*	Written-spoken words (unrelated: semantic/syllabic encoding) semantic/syllabic cues - D	Unimpaired <b>(Atypical advantage of syllabic over semantic encoding)</b>
	NT	14	16.0 (6.8)	VQ 108.9 (7.3) [mixed]* N/T		
Ambery et al. (2006)	AS [ICD-10]	27	37.6 (14.6)	VQ 106.1 (15.7) [WAIS] PQ 103.7 (19.2) [WAIS]	Proper names (cued by occupation)	Unimpaired
	NT	20	33.5 (12.0)	VQ 107.1 (13.1) [WAIS] PQ 109.4 (18.5) [WAIS]	PAL (Doors and People test)	Unimpaired
Minshew & Goldstein (2001)	HF-LN [DSM-IV +ADI+ADOS]	52	22.3 (0.6)	VQ 95.0 (17.6) [WAIS] PQ 91.5 (13.0) [WAIS]	PAL	Unimpaired
	NT	40	21.6 (9.9)	VQ 97.2 (14.6) [WAIS] PQ 95.8 (12.1) [WAIS]		
Bowler et al. (1997)	HFA[ICD-10]	16	31.2 (11.0)	VQ 99.4 (16.7) [WAIS] PQ 96.3 (13.2) [WAIS]	Written words (unrelated; word fragment cues) - D	Unimpaired
	NT	16	33.3 (11.4)	VQ 86.3 (19.2) [WAIS] PQ 96.1 (10.3) [WAIS]		
Gardiner et al. (2003)	AS [ICD-10]	16	31.6 (8.9)	VQ 95.8 (17.4) [WAIS] PQ 84.6 (13.6) [WAIS]	Written words (unrelated; word fragment cues) - D	Unimpaired
	NT	14	31.3 (7.1)	VQ 94.5 (12.9) [WAIS] PQ 88.3 (17.1) [WAIS]		
Gardiner et al. (2003)	AS [ICD-10]	10	28.3 (5.3)	VQ 89.9(16.8) [WAIS] PQ 86 (18.0) [WAIS]	PAL - D	Unimpaired
	NT	10	29.1 (4.6)	VQ 93.1 (13.4) [WAIS] PQ 86.1 (11.0) [WAIS]		
Williams et al. (2005a)	HF-LN [DSM-IV +ADI+ADOS]	29	28.7 (10.4)	VQ 108.8 (14.9) [mixed]* PQ 100.8 (13.9) [mixed]*	PAL - I & D  Family scenes cued by picture title	Unimpaired  <b>Impaired</b>
	NT	34	26.5 (10.2)	VQ 108.1 (10.1) [mixed]* PQ 109.8 (12.5) [mixed]*		
Caron et al. (2004)	HFA [DSM-IV +ADI+ADOS]	16	17.6 (6.3)	VQ 102.2 (21.2) [mixed]* PQ 112.3 (12.9) [mixed]*	Route recall - I	Unimpaired
	NT	16	18.9 (5.7)	VQ 111.1 (10.4) [mixed]* PQ 107.3 (12.1) [mixed]*		
Maras & Bowler (2010)	HFA	26	38;8 (12;5)	VQ 111.2 (13.2) [WAIS] PQ 109.1 (15.9)[WAIS]	Naturalistic events involving other people, cued by questioning	Unimpaired
	NT	26	41;3 (11;7)	VQ 112.1 (14.1) [WAIS] PQ 109.1 (15.0)[WAIS]		

\* Participants were variously tested on the WISC, WAIS and S-B tests in Mottron et al.'s study, on the WAIS and Kaufman Test of Educational Achievement in the Williams et al. study, and on the WAIS and the WISC in the Caron et al. study.

**Table 7 Declarative memory in M-LFA as tested by cued recall**

Author(s)	Groups	No.	Age (yrs) μ(sd/range)	Ability μ(sd/range)	Stimuli/task	Main findings
Klin et al. (1999)	M-LFA	34	7.4 (2.9)	VMA 3.7 (1.0) [K-ABC] NVMA 4.5 (1.5) [K-ABC]	Locations on a grid cued by previously pictured common objects	Unimpaired
	Non-ASD psychiatric	34	6.3 (2.2)	VMA 4.9 (1.5) [K-ABC] NVMA 4.7 (1.4) [K-ABC]		
Hill & Russell (2002)	M-LFA [DSM-IV]	20	9.8 (1.9)	VQ 61.9 (16.8) [BPVS]	Self- or other-performed actions - D cued by common objects	Unimpaired (both conditions)
	ID	20	9.6 (1.1)	VQ 62.3 (13.6) [BPVS]		
	NT	20	6.0 (0.7)	VQ 99.4 (26.2) [BPVS]		
Boucher & Warrington (1976)	M-LFA [Rutter, 1968]	11	10;9 (7;5-14;4)	VMA 5;4 (4;0-7;1) [PPVT] NVMA 9;4 (7;6-11;6) [CPM]	Spoken words (unrelated): semantic cues - D	Unimpaired (relative to both groups)
	ID	11	10;8 (7;2-15;4)	VMA 5;9 (4;6-7;6) [PPVT] NVMA 8;6 (6;9-10;3) [CPM]		
	NT	11	10;8 (7;7-15;4)	N/T		
Tager-Flusberg (1991)	M-LFA [DSM-III]	15	11.2 (3.3)	VMA 5.2 (1.4) [PPVT] NVMA 9.6 (3.0) [CPM]	Spoken words (unrelated): rhyme cues - D category cues - D	Unimpaired (relative to both groups) (rhyme-cued=category cued all groups)
	ID	15	10.8 (3.0)	VMA 5.0 (1.8) [PPVT] NVMA 6.0 (2.0) [CPM]		
	NT	15	4.7 (0.10)	VMA 4.9 (1.3) [PPVT] NVMA N/T		
Boucher & Warrington (1976)	M-LFA [Rutter, 1968]	10	12;8 (8;5-16;11)	VMA 5;5(3;9- 6;3) [PPVT] NVMA 9;3 (5;0-11;0) [CPM]	Pictures of common objects: phonological cues - D	Unimpaired (relative to both groups)
	ID	10	9;3 (7;1-13;8)	VMA 5;7(3;8- 7;6) [PPVT] NVMA 8;9 (8;3-10;9) [CPM]		
	NT	10	12;8 (8;2-16;1)	N/T N/T		
Farrant et al. (1999)	M-LFA [DSM-IV]	17	13.1 (3.1)	VMA 8.3 (3.9) [BPVS]	Named pictures of common objects: visual category cues - D	Unimpaired (relative to both groups) (ID group impaired relative to NT group)
	ID*	17	13.2 (2.8)	VMA 8.2 (3.7) [BPVS]		
	NT	17	8.7 (3.8)	VMA 8.3 (3.9) [BPVS]		
Boucher & Warrington (1976)	M-LFA [Rutter, 1968]	12	13;7 (8;11-18;4)	VMA 5;2 (3;3-7;1) [PPVT] NVMA 9;2 (5;0-11;6)[CPM]	PAL over trials	<b>Superior</b> (relative to NVMA- matched ID group) Unimpaired (relative to age-matched NT group)
	ID	12	13;0 (9;7-16;5)	VMA N/T NVMA 9;0 (5;3-11;6) [CPM]		
	NT	12	13;7 (9;7-17;0)	VMA N/T NVMA N/T		
Boucher & Lewis (1989)	M-LFA [Rutter, 1978]	12	13.1 (10;11-15;0)	VMA 7;6 [RWF] NVMA 11;6 [CPM]	Own past activities: cued by questioning - D	<i>Unimpaired</i> (but trend in small groups)
	ID	12	13;2 (10;11-15;4)	VMA 7;1 [RWF] NVMA 11;3 [CPM]		
Millward et al. (2000)	M-LFA [DSM-III-R]	12	13.1 (11.8-15.8)	VMA 6.3 (4.5-10.3) [BPVS]	Own past activities: cued by questioning - D Others' observed activities - cued by questioning - D	<b>Impaired</b>  Unimpaired
	NT	12	6.3 (4.5-10.3)	VMA 6.2 (4.9-7.1) [BPVS]		
Hare et al. (2007)	M-LFA [DSM-IV]	12	27.7 (6.3)	VMA 6.1 (1.9) [BPVS] VMA 5.3 (1.2) [TROG]	Self- or other-performed actions- D (cues not specified)	Unimpaired (both conditions)
	ID	14	49.6 (10.2)	VMA 6.8 (1.5) [BPVS] VMA 4.5 (0.5) [TROG]		

\* This group consisted of 6 children with Down syndrome, 6 with idiopathic ID, and 5 with speech and language delay.

**Table 8 Source memory in HFA**

Author(s)	Groups	No.	Age (yrs) $\mu$ (sd/range)	Ability $\mu$ (sd/range)	Stimuli/task	Main findings
Bigham et al. (2010)	HFA [DSM-IV + CARS] NT	18	9.2 (6;4-13;4)	VQ 103.7 (10.6) [BPVS]	Recall of an unrelated action	<b>Impaired</b>
		29	8.0 (5;0-10;4)	VQ 109.4 (11.6) [BPVS]		
Salmond et al. (2005)	HFA [DSM-IV +ASAS] NT	14	12.9 (0.7)	VQ 102 (4.0) [WISC]	Recall of temporal source	Unimpaired
		18	12.6 (0.7)	VQ 104 (2.0) [WISC]		
Bowler et al. (2004)	Young AS [ICD-10] Young NT	16	13.5 (1.1)	VQ 100.8 (20.7) [BPVS]	Recognition of encoding condition	Unimpaired (combined AS group)
		16	13.4 (0.7)	VQ 94.6 (18.3) [BPVS]		
	Adult AS [ICD-10] Adult NT	16	34.5 (6.7)	VQ 100.1 (14.9) [WAIS]	Recall of encoding condition	<b>Impaired</b> (combined AS groups)
		16	33.4 (4.6)	VQ 97 (15.5) [WAIS]		
Bowler et al. (2000a)	HFA [ICD-10] NT	16	30.9 (6.3)	VQ 96.5 (14.4) [WAIS] PQ 90.1 (11.8) [WAIS]	Recall of episodic experience	<b>Impaired</b>
		15	31.1 (5.6)	VQ 96.5 (14.4) [WAIS] PQ 90.1 (11.8) (WAIS		

**Table 9 Source memory in M-LFA**

Author(s)	Groups	No.	Age (yrs) $\mu$ (sd/range)	Ability $\mu$ (sd/range)	Stimuli/task	Main findings
Hill & Russell (2002)	M-LFA [DSM-IV]	20	9.8 (1.9)	VQ 61.9 (16.8) [BPVS]	Reality monitoring	Unimpaired (whole-group comparisons) <b>Impaired</b> (selected subgroups)
	ID	20	9.6 (1.1)	VQ 62.3 (13.6) [BPVS]		
	NT	20	6.0 (0.7)	VQ 99.4 (26.2) [BPVS]		
Russell & Jarrod (1999)	M-LFA [DSM-III-R]	22	13.2 (2.6)	VMA 7.1 (1.0) [BPVS]	Recognition of color source	Unimpaired
	ID	22	11.3 (1.4)	VMA 7.2 (1.2) [BPVS]	Reality monitoring (expected/unexpected tests)	<b>Impaired (better in unexpected than expected test)</b>
	NT	22	6.8 (0.2)	VMA 7.1 (1.0) [BPVS]		
Bigham et al. (2010)	M-LFA [DSM-IV +CARS]	29	14.5 (1.8)	VMA 7.6 (4.4-8.4) [BPVS] NVMA 8.7 [WASI]	Recall of temporal location (before/after)	<b>Impaired</b> (relative to both groups)
	ID	24	14.3 (1.2)	VMA 7.7 (4.5-8.3) [BPVS] NVMA 8.7 [WASI]		
	NT	23	8.0 (1.5)	VMA 8.0 (4.8-9.0) [BPVS] NVMA 7.8 [WASI]		

**Table 10 Working memory in HFA**

Author(s)	Groups	No.	Age (yrs) $\mu$ (sd)/range	Ability $\mu$ (sd)/range	Stimuli/task	Main findings
Cui et al. (2010)	AS	12	7.5 (0.8)	FSIQ 100.0 (17.1) [WISC]**	Auditory span forwards + backwards; visual-spatial span (WMTB) N-back tasks	<b>Superior</b> Unimpaired <b>Impaired</b> <b>Impaired</b>
	NT	29	7.4 (0.5)	FSIQ 108.3 (14.1) [WISC]		
Verté et al. (2006)	AS [DSM-IV-TR +ADI-R]	37	8.7 (1.9)	VQ 105.2 (16.3) [WISC] PQ 104.0 (17.8) [WISC]	Spatial span forwards (Corsi blocks)	Unimpaired (both groups)  <b>Impaired</b> (both groups)
	HF-LN [DSM-IV-TR +ADI-R]	50	8.5 (2.1)	VQ 93.1 (18.0) [WISC] PQ 104.0 (15.9) [WISC]		
	NT	47	9.4 (1.6)	VQ 113.6 (10.4) [WISC] PQ 108.5 (11.9) [WISC]		
Joseph et al. (2005)	HFA [DSM-IV +ADI-R+ADOS]*	24	8;11 (2;4)	VQ 94 (19) [DAS] NVQ 99 (20) [DAS]	Word span forwards (DAS) Self-ordered visual-spatial pointing task: (i) nonverbal; (ii) verbal mediation available	Unimpaired  (i) Unimpaired <b>(ii) Impaired</b>
	NT	24	8;11 (2;2)	VQ 89 (12) [DAS] NVQ 94 (14) [DAS]		
Zinke et al. (2010)	HFA [DSM-IV +ADI-R+ADOS]	15	9.0 (1.5)	V-SS 10.7 (3.0) [WISC vocab.] P-SS 10.7 (2.8) [WISC blocks]	Spatial span forwards (Corsi blocks)	Unimpaired (despite lower IQ)
	NT	17	9.8 (1.7)	V-SS 12.8 (1.6) [WISC vocab.] P-SS 13.0 (3.5) [WISC blocks]		
Goldberg et al. (2005)	HFA [DSM-IV +ADI-R+ADOS]	17	10.3 (1.8)	VQ 99.2 (16.6) [WISC] PQ 98.8 (15.8) [WISC]	Self-ordered visual-spatial search task	<b>Impaired</b> (errors measure) Unimpaired (on strategy use)
	NT	31	10.4 (1.5)	VQ 116.9 (11.9) [WISC] PQ 105.8 (11.9) [WISC]		
Manjiviona & Prior (1999)	AS [DSM-IV/ICD-10]	35	10.8 (2.6)	VQ 101.4 (15.7) [WISC] PQ 102.0 (19.9) [WISC]	Digit span forwards (WISC)  Digit span backwards (WISC)	<i>Unimpaired</i> (both groups – see text) <b>Impaired</b> (both groups)
	HF-LN [DSM-IV/ICD-10] Comparison with standardized norms	21	10.4 (2.7)	VQ 89.2 (19.2) [WISC] PQ 89.6 (14.6) [WISC]		
Whitehouse et al. (2008)	HFA [DSM-IV +ADOS]	16	10;8 (2;7)	VQ 101.8 (9.6) [TROG-E] PQ 110.3 (14.9) [WASI]	Nonword repetition (NEPSY) - I	Unimpaired (relative to norms) <b>Superior</b> (to SLI group)
	SLI Comparison with standardized norms	34	11;10 (2;3)	VQ 91.7 (13.9) [TROG-E] PQ 100.4 (13.2) [WASI]		
Siegel et al. (1996)	HF-LN children [DSM-IV+ADI]	36	10.1 (3.5)	VQ & FSQ >70	Digit span forwards+backwards (WISC/WAIS)	Unimpaired
	HF-LN adults [DSM-IV+ADI] Comparison with standardized norms	45	26.5 (9.2)	VQ & FSQ > 70		
Williams et al. (2005b)	HF-LN children [DSM-IV+ADI-R+ADOS]	24	11.8 (2.4)	VQ 112.5 (16.5) [WISC] PQ 106.4 (14.2) [WISC]	Number-letter span forwards (WRAML) Spatial span forwards (finger windows-WRAML) Letter-number sequencing verbal task (WMS-III) Spatial span forwards+backwards (WMS-III)	Unimpaired <b>Impaired</b>  Unimpaired <b>Impaired</b>
	NT children	44	12.4 (2.2)	VQ 110.3 (9.8) [WISC] PQ 108.1 (11.1) [WISC]		
	HF-LN adults [DSM-IV +ADI-R+ADOS]	31	26.6 (8.7)	VQ 111.1 (16.5) [WAIS] PQ 103.1 (16.6) [WAIS]		
	NT adults	25	26.8 (9.1)	VQ 108 (10.2) [WAIS] PQ 110.2 (12.8) [WAIS]		
Ozonoff & Strayer (2001)	HF-LN [DSM-IV +ADI-R+ADOS]	25	12.9 (3.2)	VQ 94.6 (18.5) [WISC] PQ 99.3 (19.9) [WISC]	Spatial span (nonserial) – D N-back visual memory task Self-ordered visual-spatial search task	Unimpaired (all three tasks, covarying PQ)
	NT	15	11.8 (3.2)	VQ 103.8 (10.9) [WISC] PQ 110 (14.5) [WISC]		
Steele et al. (2007)	HF-LN [DSM-IV +ADI+ADOS]	29	14.8 (5.5)	VQ 107.5 (13.0) [WISC/WAIS]	Self-ordered visual-spatial search task	<b>Impaired</b> (at higher memory loads)
	NT	29	16.9 (5.4)	PQ 106.2 (11.8) [WISC/WAIS] VQ 110.1 (9.5) [WISC/WAIS] PQ 110.1 (9.9) [WISC/WAIS]		
Minshew et al. (2005)	HF-LN [DSM-IV +ADI+ADOS] Comparison with standardized norms	215	Range 8;0-55	VQ & PQ > 70 [WISC/WAIS]	Digit span forwards+backwards (WISC/WAIS)	Unimpaired

[Type text]



Morris et al. (1999)	AS [ICD-10]	15	29.5 (19–49)	VQ 99 (81-129) [WAIS] PQ 100 (84-137)[WAIS]	Self-ordered visual-spatial search task	<b>Impaired</b> (at higher memory loads)
	NT	18	29.4 (19–45)	VQ 106.3 (96-137) [WAIS] PQ 105.8 (78-136) [WAIS]		
Poirier et al. (2011)	HFA DSM-IV [ADOS]	22	37.6 (13.3)	VQ 109.8 (18.3) [WAIS] PQ 108 (19.4) WAIS]	Word span forwards+ backwards (not WAIS)	<b>Impaired</b> (item recall unimpaired; <b>order recall Impaired</b> )
	NT	22	37.3 (11.3)	VQ 110.5 (13.9) [WAIS] PQ 110.1 (11.8) [WAIS]		

\*Ability ranges given in the paper show that a small number of children in both groups had VQs < 70

\*\* A short form of the WISC developed for use with Chinese children was used in this study.

**Table 11 Working memory in M-LFA**

Author(s)	Groups	No.	Age (yrs) μ(sd/range)	Ability μ(sd/range)	Stimuli/task	Main findings
Griffiths et al. (1999)	M-LFA [DSM-IV+ADI-R] ID/DLD****	18	4.2 (0.6)	VMA 2.2 (1.0) [Mullen]	Self-ordered search (i) by location + design; (ii) by design only	(i) Unimpaired (ii) Unimpaired
		17	4.3 (0.8)	NVMA 3.1 (0.7) [Mullen] VMA 2.3 (0.9) [Mullen] NVMA 2.7 (1.2) [Mullen]		
Fein et al. (1996)	M-LFA [DSM-III-R + WADC]	125	5;0 (1;4)	Vocab. score: 27.6 (6.0) [S-B]*** Comp. score: 27.0 (4.8) [S-B]*** NVQ 45.6 (19.4) [S-B or Bayley]	Digit span forwards+backwards (S-B)	Unimpaired (relative to ID group)  <b>Impaired</b> (relative to norm)
	ID  Comparison with standardized norms	110	4.7 (1.1)	Vocab. score: 32.9 (6.0) [S-B]*** Comp. score: 33.2 (8.0) [S-B]*** NVQ 55.5 (19.9) [S-B or Bayley]		
Kjelgaard & Tager-Flusberg (2001)	M-LFA [DSM-IV +ADI+ADOS] Comparison with standardized norms	21	6;11 (4;0 - 14;0)	VQ < 70 [CELF]	Nonword repetition (NEPSY) - I	<b>Mild impairment</b>
Frith (1970)	M-LFA [Rutter, 1968] ID NT	10	Range 7;0 -13;0	VMA 4;6 [PPVT]	Digit span forwards+backwards (WISC)	Unimpaired (relative to both groups)
		10	Range 10;0-16;0	VMA 4;6 [PPVT]		
		10	Range 4;0-5;0	VMA 4;6 [PPVT]		
Farrant et al. (1999)	M-LFA [DSM-IV] ID NT	12	11.1 (2.8)	VMA 6.3 (2.2) [PPVT]	Named picture span forwards	Unimpaired (relative to both groups)
		12	10.9 (2.9)	VMA 6.1 (1.9) [PPVT]		
		12	6.4 (2.0)	VMA 6.1 (1.8) [PPVT]		
Fyffe & Prior (1978)	M-LFA [Rutter, 1974] ID NT	14	11;3 (2;4)	VQ 58 (13) [mixed]** PQ 66 (16) [mixed]**	Digit span Forwards (ITPA)	Unimpaired (relative to both groups)
		14	12;2 (3;8)	VQ 59 (12) [mixed]** PQ 66 (12) [mixed]**		
		14	6;9 (0;3)	N/T		
Russell et al. (1996)	M-LFA [DSM-III-R] ID NT	33	12.4 (3.0)	VMA 6.3 (1.2) [BPVS]	Word span forwards: (i) verbal repetition (ii) picture pointing	Unimpaired (relative to NT group, both tasks) <b>Superior</b> (relative to ID group, both tasks)
		33	10.8 (1.9)	VMA 6.2 (1.2) [BPVS]		
		33	6.3 (1.2)	N/T		
Russell et al. (1996)	M-LFA [DSM-III-R] ID NT	22	12.5 (2.8)	VMA 6.9 (1.5) [BPVS]	Concurrent storage+processing tasks	<b>Impaired</b> (relative to NT group) Unimpaired (relative to ID group)
		22	11.1 (2.0)	VMA 6.9 (1.6) [BPVS]		
		22	6.8 (0.5)	VMA 6.9 (1.5)		
Farrant et al. (1999)	M-LFA [DSM-IV] ID* NT	17	13.1 (3.1)	VMA 8.3 (3.9) [BPVS]	Digit span forwards+backwards (WISC)	Unimpaired (relative to both groups)
		17	13.2 (2.8)	VMA 8.2 (3.7) [BPVS]		
		17	8.7 (3.8)	VMA 8.3 (3.9) [BPVS]		
Boucher & Warrington (1976)	M-LFA [Rutter, 1968] ID NT	12	13;7 (8;11-18;4)	VMA 5;2 (3;3-7;1) [PPVT] NVMA 9.2 (5;0-11;6)[CPM]	Digit span forwards	Unimpaired (relative to age-Matched NT group) <b>Superior</b> (relative to NVMA -matched ID group)
		12	13;0 (9;7-16;5)	VMA N/T NVMA 9;0 (5;3 - >11;6) [CPM]		
		12	13;7 (9;7-17;0)	VMA N/T NVMA N/T		

\* This group consisted of 6 children with Down syndrome, 6 with idiopathic ID, and 5 with speech and language delay.

\*\* IQ scores were obtained from WISC, Binet, Leiter performance scale, and PPVT.

\*\*\* Mean score on these tests is 50 (sd 8.0).

\*\*\*\* This group consisted of 6 children with Down syndrome, 5 with specific speech/language delays, and 6 with general cognitive delays.

[Type text]