14   Memory, language and intellectual ability in low-functioning autism

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Introduction

Over the last two decades most psychological and neuropsychological research into autism has focused on individuals with Asperger syndrome or high-functioning autism (HFA),[1] rather than on individuals with low-functioning autism (LFA) or what is termed autistic disorder in the *Diagnostic and Stastical Manual of Mental Disorders*, 4th edition (DSM-IV; American Psychiatric Association, 1994). The core symptoms of autism, namely impairments of social interaction, communication and behavioural flexibility, are more likely to occur in pure form in people with HFA than people with LFA, and it makes sense, therefore, to focus on HFA to improve understanding of the core impairments.

A consequence of this strategy, however, has been a relative neglect of the impairments of language and intellectual ability that distinguish LFA from HFA. This is regrettable for both practical and theoretical reasons. From a practical point of view the combined effects of cognitive and linguistic impairments with autism are devastating for individuals themselves, and for their families and carers. Better understanding of the additional impairments is needed to provide optimal interventions and care. From a theoretical point of view, familial and genetic studies indicate that vulnerability to language impairment is related to vulnerability to autism (e.g. Bolton *et al.*, 1994; Piven & Palmer, 1997; Folstein *et al.*, 1999; Tomblin, Hafeman & O'Brien, 2003; Bartlett *et al.*, 2004). Understanding the bases of the language impairment in LFA should therefore contribute to understanding autism as a whole.

There is, however, no corresponding evidence of intellectual impairment in families that include someone with autism, and it is often

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[1] 'High-functioning autism' is used here to refer to individuals with the triad of autism-diagnostic impairments whose current levels of cognitive functioning and language are normal. Thus the term includes people with 'Asperger syndrome'. This usage is adopted because to date there is no clear evidence that Asperger syndrome is a discrete disorder.

assumed that intellectual disability (ID) co-occurs with autism for reasons unconnected with autism itself. However, this assumption leaves unexplained why autism and ID are so strongly associated, as pointed out by Bailey, Phillips and Rutter (1996). These authors also noted that verbal intelligence, but not nonverbal intelligence, shows a substantial association with the severity of autism symptomology and with familial loading (Bolton *et al.*, 1994), and suggested that attempts to explain the association between autism and ID should focus on verbal abilities.

In this chapter we pursue Bailey *et al.*'s suggestion by developing the hypothesis outlined towards the end of Mayes and Boucher, this volume, Chapter 3, that a pervasive impairment of declarative memory is a critical cause of both the language impairment and the impairment of verbal intelligence (and hence overall intellectual disability) in people with low-functioning autism. This hypothesis builds on suggestions by Bachevalier (1994; also this volume, Chapter 2) and Bauman and Kemper (2004), and is consistent with the model of autism proposed in Boucher *et al.* (2005; see also Ben Shalom, 2003; Faran and Ben Shalom, this volume, Chapter 5). According to this model, the socio-emotional impairments of autism are associated with the disruption of co-ordinated activity between amygdala and prefrontal structures, and the additional language and learning impairments in LFA are associated with a disruption of co-ordinated hippocampal–parahippocampal and prefrontal activity. The relative sparing of procedural memory, and the resulting over-dependence on its use, can help to explain some of the repetitive behaviours diagnostic of autism – a suggestion also made by Bauman and Kemper (2004).

The chapter is in three main sections. The first section covers what has to be explained in terms of the typical profiles of linguistic and intellectual abilities in individuals with LFA. In the second main section we present our hypothesis, and suggest how it may explain the linguistic and intellectual ability profiles described, finishing with a short review of evidence relating to declarative memory in LFA. In the third main section we consider other explanations of the language and intellectual impairments in LFA, and suggest how our hypothesis may relate to these theories. The chapter concludes with a short summary.

What has to be explained

The linguistic profile is considered first, because we argue that declarative memory impairment affects language acquisition in the first instance, and intellectual ability secondarily.

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The typical language profile in people with low-functioning autism

Language profiles in individuals with LFA are diverse. This diversity has numerous causes, including the fact that it varies with the severity of the language impairment; that the profile changes with age (Rapin & Dunn, 2003); and that the incidence of comorbid conditions including hearing loss, dyspraxia, and specific language impairment (SLI) is higher in people with autistic spectrum disorders (ASDs) than in the general population (Rapin, 1996; Kjelgaard & Tager-Flusberg, 2001).

Despite the diversity of language profiles at the level of individuals, a typical profile emerges from group studies (see Lord & Paul, 1997, for a detailed review). This profile is described next, features of the profile being enumerated in the order in which they will be discussed in the section headed 'Explaining the language impairment in low-functioning autism', later in the chapter.

1. The severity of the language impairment in lower-functioning autism ranges from mild to profound, and a high proportion of individuals never acquire language, or acquire at most a few words or signs used communicatively.[2] Individuals with no language, or negligible communicative language, may be described as having nonverbal LFA (NV-LFA). Correspondingly, those with some useful language may be described as having verbal LFA (V-LFA).

2. Language comprehension in people with V-LFA is invariably impaired: no instances of expressive language impairment in the absence of comprehension impairment have been found in large-scale studies (Rapin & Dunn, 2003). The universal comprehension impairment reflects a problem in relating linguistic symbols to an underlying knowledge-base. Thus, in an early review of studies of language in LFA, Fay and Schuler (1980) described word learning as consisting of 'the assignment of concrete labels rather than rule-linked conceptual units .... Memorised words denote but fail to connote' (p. 84). Similarly, in their review of language in autism, Lord and Paul (1997, p. 212) comment on the 'limited ability to integrate linguistic input with real-world knowledge'.

3. The language impairment in V-LFA is amodal: spoken, signed and written language are all affected, although there may be minor differences in the facility with which language can be acquired in one or

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[2] As the diagnosis of able individuals with ASDs increases, it becomes harder to put a figure on the proportion of individuals with no language. At one time a figure of 50 per cent would have been accepted, but that decreases with the extension of the diagnostic capsule upwards.

other modality (especially where some comorbid condition such as hearing loss is present).

4. Phonology (sometimes referred to – inaccurately – as articulation) and grammar (sometimes referred to as syntax) in people with V-LFA are less reliably and persistently impaired than semantics.[3] Early studies of verbal children of school age suggested that phonology and grammar were appropriate for mental age (Bartolucci *et al.*, 1976; Boucher, 1976; Tager-Flusberg, 1981). Recent studies that include preschool children suggest that clinically significant phonological and grammatical abnormalities are commonly present in younger children but tend to resolve, especially in the more able, whereas difficulties in the processing of meaning persist (Rapin & Dunn, 2003; see also Kjelgaard & Tager-Flusberg, 2001).

5. There is a tendency to reproduce rote-learned chunks of grammatically well-formed language in echolalic and formulaic language (Kanner, 1946; Prizant, 1983a; Dobbinson, Perkins & Boucher, 2003). The use of echolalic and formulaic language can give the impression that expressive language is superior to comprehension, masking a paucity of productive expressive language (Dobbinson, 2000).

6. Finally, expressive language in V-LFA is characterized not only by echolalic utterances and excessive formulaicity, but also by the use of idiosyncratic words or phrases, and neologisms (Kanner, 1946; Volden & Lord, 1991). There are also problems with deictic terms, especially personal pronouns (Lee, Hobson & Chiat, 1994).

The intellectual ability profile in people with low-functioning autism

As in the case of language profiles, individual intelligence test profiles in LFA show considerable variation.[4] This is not surprising given the multi-factorial nature of intelligence (Mackintosh, 1998); the profile also changes with age (Mayes & Calhoun, 2003a). Across groups, however, an LFA-specific profile emerges. The profile is first described utilizing the distinction between verbal and nonverbal intelligence as exemplified in the Wechsler intelligence scales (Wechsler, 1974, 1981). This is followed

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[3] Pragmatics is always impaired in individual with ASDs, even those who are high functioning. However, pragmatics relates to the *use of* language, and is more to do with communication than with structural language (which is a *means* of communication), which is why the pragmatic impairment is not discussed here.

[4] Intelligence theory and testing are not, of course, the only framework that can be used for examining intellectual abilities, but they provide the most useful framework for present purposes – not least because of the availability of research data based on standardized measures.

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Table 14.1. *Summary of findings from studies using the Wechsler intelligence tests (from Siegel, Minshew & Goldstein, 1996, with permission)*

	Age status of participants	FSIQ	VQ	PQ	VQ < PQ difference
Ohta, 1987	Children[*]	72	65	85	20
Wassing, 1965	Children[*]	71	59	88	29
Lincoln *et al*., 1988	Children[*]	69	60	84	24
Allen *et al*., 1991	Children[*]	68	57	85	28
Bartak *et al*. 1975	Children[*]	67	NR (<67)	97	>30
Narita & Koga, 1987	NR	66	61	78	17

FSIQ = Full-Scale Intelligence Quotient; VQ = Verbal Quotient; PQ = Performance Quotient
NR = not reported
[*]Pre-adolescent

by a short section utilizing the distinction between fluid and crystallized intelligence (Horn & Cattell, 1966; Cattell, 1971).

Profile based on the verbal–nonverbal distinction

Bailey *et al*. are not the only authors who have noted that overall intellectual disability in LFA owes more to a decline in verbal intelligence than a decline in nonverbal intelligence (see for example Rumsey, 1992; Lincoln, Allen & Kilman, 1995; Siegel, Minshew & Goldstein, 1996; Lord & Paul, 1997; however, see Mayes & Calhoun, 2003b for some contrary evidence). Evidence in support of this suggestion is summarized in Table 14.1, which shows findings from studies using versions of the Wechsler intelligence scales to assess Verbal and Performance (nonverbal) abilities in individuals whose Full-Scale IQ is below 75.

Notably, the discrepancy between verbal and nonverbal intelligence in low-ability individuals with autism does not extend to high-functioning individuals, in whom no consistent pattern of verbal or nonverbal superiority occurs (Minshew, Turner & Goldstein, 2005).

More detailed examination of the intelligence test profiles of groups of individuals with verbal LFA assessed using the Wechsler scales shows the following. Of the six Verbal subtests, the Comprehension subtest is least well performed, with performance on the Information, Vocabulary, Arithmetic and Similarities subtests also low in relation to Full-Scale IQ, leaving performance on the Digit Span subtest constituting a relative peak of ability, though not necessarily within the normal range (Lincoln, Allen & Kilman, 1995; Siegel, Minshew & Goldstein, 1996). Performance across the five Performance (nonverbal) subtests is also

uneven, with scores on the Picture Arrangement subtest and the Digit Symbol (adult version) or Coding (children's version) subtest consistently lower than those on the Block Design, Picture Completion and Object Assembly subtests (Lincoln, Allen & Kliman, 1995; Siegel, Minshew & Goldstein, 1996).

Notably, individuals with high-functioning autism differ from groups with V-LFA in their Verbal subtest profile, performing well on the Information, Vocabulary, Arithmetic and Similarities subtests (although performing less well on the Comprehension subtest) (Klin *et al.*, 2000; Minshew, Taylor & Goldstein, 2005). Performance on Digit Span does not emerge as a peak ability in individuals with HFA because they perform well on most of the other Verbal subtests. Notably, also, individuals with high-functioning autism do not differ from groups with V-LFA in their Performance (nonverbal) subtest profile, although achieving higher scores than low-functioning individuals.

Individuals with nonverbal LFA, who are not testable on formal scales, may be profoundly and pervasively mentally impaired. However, some individuals with NV-LFA are well oriented within familiar environments, with daily living skills that are superior to either their social or their communication abilities (Carter *et al.*, 1998; Kraijer, 2000). It may be inferred that these individuals have acquired at least some implicit, *preverbal* knowledge of basic-level categories: they put on shoes, open doors, turn on taps, eat with spoons, etc. These mute, or nearly mute, individuals also have some relatively spared nonverbal abilities, generally to do with fitting and assembly skills (DeMyer, 1976).

Profile based on the fluid–crystallized intelligence distinction

A widely accepted distinction made in the literature on intelligence is that between fluid and crystallized intelligence (Horn & Cattell, 1966; Cattell, 1971). Fluid intelligence reflects the ability to solve novel problems not dependent on acquired knowledge, and may be thought of as corresponding to a general reasoning factor 'g', reflecting genetic potential. Crystallized intelligence, on the other hand, corresponds to acquired knowledge and is more dependent on verbal ability, experiential opportunities and education.

Individuals with V-LFA tend to perform better on Raven's Matrices (Raven, Court & Raven, 1986), which is generally considered to reflect fluid intelligence, than they do on the Comprehension, Information, Similarities, Arithmetic and Vocabulary subtests of the Wechsler tests, all of which reflect verbally mediated, crystallized intelligence (Mackintosh, 1998). This suggests that fluid intelligence is generally less impaired than crystallized intelligence in people with V-LFA. The relative sparing of fluid

intelligence is also suggested by the finding that speed of processing (widely considered to correlate with 'g') is not significantly impaired in lower-functioning individuals with autism (Scheuffgen *et al.*, 2000).

Pervasive declarative memory impairment as an explanation of language impairment and intellectual disability in low-functioning autism

The hypothesis

We hypothesize that the language and intellectual ability profiles of individuals with low-functioning autism, outlined above, derive in large part from impairments of long-term declarative memory. This hypothesis was introduced in Mayes and Boucher (this volume, Chapter 3) in terms of combined impairments of recollection and familiarity, affecting recall and recognition of both personally experienced events and impersonal facts. In that chapter we briefly presented neurobiological evidence consistent with this hypothesis. Here we flesh out the hypothesis, and review the behavioural evidence relating to it. First, however, we briefly recapitulate the distinction between declarative and nondeclarative, or procedural, memory.

The distinction between declarative and nondeclarative memory

Memory that is accompanied by a conscious feeling of memory is generally described as declarative, or explicit. Memory not so accompanied is generally described as nondeclarative, or implicit. Memory for personally experienced events (episodic memory) and memory for factual information (semantic memory) are generally accompanied by a conscious feeling of memory, and may therefore be described as declarative. By contrast, the heterogenous kinds of learning covered by procedural memory (see this volume, Gardiner, Chapter 1, and Mayes & Boucher, Chapter 3) are not usually accompanied by a conscious feeling of memory, and may be described as nondeclarative, or implicit.[5]

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[5] The distinctions between declarative and nondeclarative, explicit and implicit, are not clear cut. Memories that we actually use often comprise differently weighted mixtures of declarative and nondeclarative memory with feelings of memory correspondingly varying in strength.

Explaining the language profile

Key features of the language profile in people with LFA as described earlier in the chapter are considered in turn, and explained in terms of the hypothesis.

1. According to the hypothesis, the complete or near-complete absence of language in people with nonverbal LFA results from a total loss, or near total loss, of declarative memory, comparable to that seen in severe forms of adult-acquired global amnesia as described in Chapter 3. The language impairment in people with verbal LFA is hypothesized to result largely from diminished, but not total loss of, declarative memory ability, with procedural memory and immediate memory relatively intact.

2. The universal impairment of comprehension and meaning in V-LFA is hypothesized to result from impaired access to memory for preverbal categorical knowledge of the world. Access to such information is required for the acquisition of fully meaningful linguistic symbols. For example, the typically developing infant or young child who hears the word *dog* spoken in the presence of an actual dog, or picture of a dog, sees the dog in front of her, accesses her memory for information relating to the category of 'dogs-in-general', and learns that *dog* refers to (this particular dog and) dogs-in-general (see Figure 14.1a). A child who is unable to access her categorical knowledge of 'dogs-in-general' will hear the word *dog*, and learn only that *dog* refers to this particular dog (see Figure 14.1b).
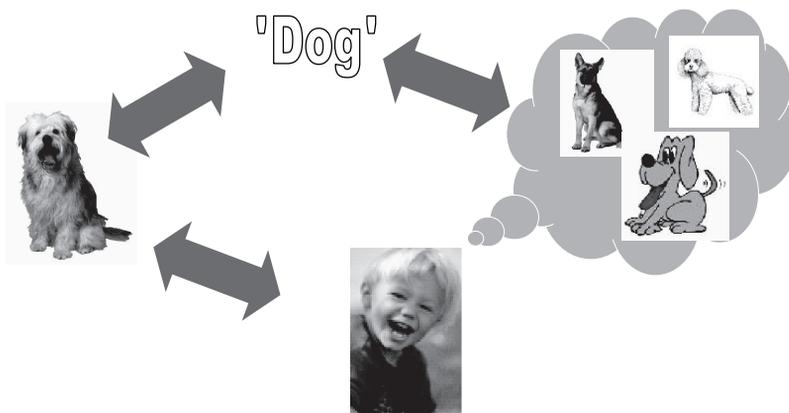

Figure 14.1a Learning that a word refers to a particular referent and to the category to which the referent belongs
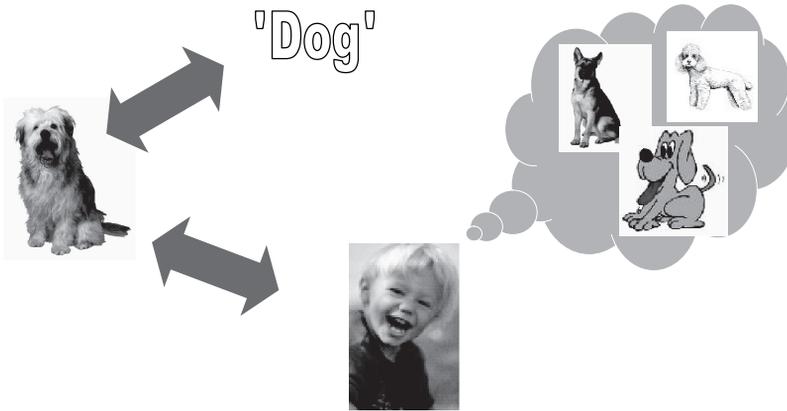
Figure 14.1b Learning to label a particular referent

Thus, single-word naming of basic-level categories (whether by spoken word or manual sign) *can* proceed in the absence of declarative memory. However, the linguistic symbols that are acquired will tend to operate like proper names with fixed meanings rather than with rich, flexible and generalizable meanings. An impairment of declarative memory would therefore cause early word learning to have the character of 'the assignment of concrete labels rather than rule-linked conceptual units', as noted by Fay and Schuler (1980) in their early review. An impairment of declarative memory would also cause precisely the 'limited ability to integrate linguistic input with real-world knowledge' noted by Lord and Paul (1997, p. 212). Moreover, recent work by Preissler (2006) confirms that children with autism tend to make the kind of symbol-referent association (underlying denotation), rather than the normal symbol-reference association (underlying connotation), as illustrated in Figures 14.1b and 14.1a, respectively.

Lord and Paul's comment that linguistic *input* – i.e. heard speech, seen manual signs or seen written language – fails to connect with real-world knowledge, highlights the problems of comprehension that would result from the limited and inflexible meanings that words have for individuals with V-LFA. Thus, impaired comprehension can be traced back to the declarative memory impairment underlying the acquisition of linguistic meaning.

It seems likely that those categories and concepts that are normally acquired via language – for example, superordinate terms or abstract words – would be cumulatively affected both by the overall delay in language acquisition, but also by the limited meaning of such linguistic symbols as have been acquired. There is some evidence which might

suggest that acquisition of superordinate terms, at least, is not impaired (e.g. Tager-Flusberg, 1985a, b; Boucher, 1988). However, these studies compared participant groups matched for verbal mental age using a vocabulary comprehension test that included both basic-level and superordinate-level items, biasing towards negative findings.

3. A pervasive declarative memory impairment will affect language acquisition in all modalities, as outlined in the previous paragraph.

4. In contrast to the effect of declarative memory impairment on the acquisition of linguistic meaning, the sparing of procedural memory in people with V-LFA will leave the acquisition of phonology and grammar relatively intact. This is because the items and combinatorial rules of phonology and grammar are learned unconsciously (Ullman, 2004) – we only gain explicit access to this knowledge if we study linguistics. However, the development of grammar will not be completely spared, because it is partly dependent on linguistic meaning, via the process sometimes referred to as semantic bootstrapping. For this reason, the acquisition of grammatical items and rules will be most affected in younger or less able individuals with V-LFA, whose lexical development is most impoverished.

5. Spared procedural memory and spared immediate memory will also enable the individual with declarative memory impairment to acquire perceptual representations of chunks of heard speech or seen writing. According to Ullman's (2004) 'see-saw' effect, such selectively spared abilities will be utilized to an unusual extent to compensate for diminished declarative memory. The phonologically and grammatically correct language reproduced will give the impression that expressive language is superior to comprehension, masking the impairment of truly productive expressive language.

6. Use of idiosyncratic language and impaired understanding and use of deictic terms can be explained mainly in terms of the social impairments of people with ASDs, and specifically by a lack of understanding of other minds (see below), rather than by our hypothesis. However, spared associative learning of the kind that is included within the set of procedural memory abilities (Mayes and Boucher, this volume, Chapter 3) may contribute to the tendency to use idiosyncratic terms (cf. Kanner's, 1946, well-known anecdote about an individual's use of 'Don't throw the dog off the balcony' to mean 'No').

Explaining the intellectual ability profile

Verbal versus nonverbal abilities

The lower scores of individuals with verbal LFA on Verbal as opposed to Performance (nonverbal) subtests of the Wechsler scales

can be understood in terms of the effect that a partial impairment of declarative memory, combined with spared procedural and immediate memory, would have on the performance of individual subtests.

In particular, those subtests most likely to be adversely affected by declarative memory impairment are all in the Verbal group, namely: the Comprehension, Information, Vocabulary, Arithmetic and Similarities subtests, all of which are heavily dependent on linguistic knowledge *per se*, on language-mediated learning, and to a greater or lesser extent on the ability to access factual knowledge from semantic memory. At the same time, most of those subtests least likely to be affected by declarative memory impairment are in the Performance group, namely Block Design, Picture Completion, and Object Assembly – all subtests probing perceptual and constructive visuo-spatial abilities, with limited reliance on language, verbal mediation, or access to factual knowledge in semantic memory. This, we argue, is sufficient to explain the pattern of $VQ < PQ$ observed in individuals with low-functioning autism (see Table 14.1).

Of the remaining subtests, performance on the Digit Span Verbal subtest is – according to the hypothesis – relatively spared because it tests immediate memory, with minimal dependence on either language or long-term declarative memory. Relatively poor performance on the Picture Arrangement and Digit Symbol/Coding nonverbal subtests may be explained in terms of their partial dependence on language mediation and on declarative memory (for events, in the case of Picture Arrangement; for the symbols/codes provided in the case of Digit Symbol/Coding). The fact that the $VQ < PQ$ discrepancy reliably occurs in groups of individuals with V-LFA, despite the relatively good performance on Digit Span and the relatively poor performances on Picture Arrangement and Digit Symbol/Coding, underlines the extent to which the contrasting performances on the other Verbal and Performance subtests drives VQ and PQ apart.

It is important to stress that we are not claiming that declarative memory impairments are the sole cause of impaired performance on certain intelligence subtests in lower-functioning individuals with ASDs. In terms of the hypothesis, individuals with HFA perform well on most of the Verbal subtests precisely because they do not have diminished declarative memory leading to the impairments of language and semantic memory that affect lower-functioning individuals. However, individuals with HFA, as well as those with LFA, perform consistently less well on the Comprehension subtest than on other Verbal subtests; and less well on Picture Arrangement and Digit Symbol/Coding than

on other Performance subtests (Siegel, Minshew & Goldstein, 1996; Minshew, Turner & Goldstein, 2005). This suggests that other, autism-specific impairments are depressing performance on these particular tests. In the case of Comprehension, which assesses understanding and knowledge of social situations and conventions, autism-related social impairments are clearly contributory. In the case of Picture Arrangement and Digit Symbol/Coding, impaired episodic memory (which affects individuals across the spectrum) may be a contributory factor. Impaired sequential processing may also be involved – a possibility that is discussed towards the end of the chapter.

Finally, the fact that not all those individuals who have nonverbal LFA are profoundly intellectually impaired is of particular relevance to the hypothesis. According to the hypothesis, these individuals have total, or near total, but *selective* loss of declarative memory, leaving procedural memory at least relatively intact. Non declarative, procedural forms of memory are sufficient for the acquisition of categorical knowledge based on sensory-perceptual experience, as is evident from observation of preverbal typically developing infants. Implicit learning would therefore proceed in these individuals, including the acquisition of basic-level categorical knowledge, and some daily living skills and routines. Similarly, visuo-spatial abilities, including certain fitting and assembly skills of the kinds assessed in formal intelligence tests would be relatively spared, leading to the uneven patterns of ability that have been observed (Carter *et al.*, 1998; Kraijer, 2000; DeMyer, 1976). By contrast, those individuals with NV-LFA who are profoundly and pervasively mentally impaired are hypothesized to have total or near total loss of both declarative and procedural memory, probably associated with extensive bilateral medial temporal lobe dysgenesis or damage (DeLong & Heinz, 1997).

The fluid versus crystallized intelligence distinction

The discrepancy between relatively spared performance on tasks mainly dependent on fluid intelligence, as opposed to impaired performance on tasks that are largely dependent on language and other acquired knowledge and skills, can be explained in terms of the problems of language acquisition and of access to memory for factual or episodic information entailed by a pervasive impairment of declarative memory. Specifically, impaired declarative memory will have negative effects on measures of crystallized intelligence which, by definition, assess abilities that are dependent on language and acquired knowledge and skills. Assuming that the declarative memory impairment is selective, general reasoning ability need not be affected, leaving performance on measures of fluid intelligence, such

as Raven's Matrices, relatively spared. Speed of processing would also be unaffected, as has been observed by Scheuffgen *et al.* (2000).

Evidence relating to the hypothesis

Published findings

A pervasive impairment of long term declarative memory would be man-ifested in impaired performance on tests of delayed free recall and recog-nition extending across memory for factual information as well as memory for personally experienced episodes. Consistent with the tendency to focus on people with high-functioning autism or Asperger syndrome in recent neuropsychological research, there have been relatively few studies of memory in lower-functioning autism over the last two decades. The results of early studies and a few that are more recent are summarized next.

Free recall in people with LFA has generally been found to be either impaired or anomalous. Impairments have been shown for recall of meaningful verbal material (e.g. O'Connor & Hermelin, 1967; Hermelin & O'Connor, 1967; Fyffe & Prior, 1978; Tager-Flusberg, 1991), and also events (Boucher, 1981a; Boucher & Lewis, 1989; Millward *et al.*, 2000). Anomalies have been demonstrated in tests of the recall of unrelated words, where recency effects tend to make a greater than normal contribution to overall performance (Boucher, 1978, 1981b; Fyffe & Prior, 1978). Free recall impairments and anomalies in people with LFA are unsurprising, given the evidence of impaired recollection and associated impairments of episodic memory in people with HFA, as documented in other chapters in this book.

The more critical test of our hypothesis concerns the predictions that (a) recognition memory will be impaired; and (b) impaired recognition will relate to levels of conceptual, lexical and factual knowledge, including performance on specific verbal intelligence subtests.

Regarding prediction (a), there is some evidence suggestive of impaired recognition memory in people with LFA. However, the evidence is sparse and inconclusive. For example, Boucher and Warrington (1976), using a picture recognition task with a mixed-ability group, noted wide variation in the scores of the children with autism, although there was no overall group impairment. These authors suggested that the lower-functioning children in their study, but not the higher-functioning children, may have had a recognition impairment. In another early study, Ameli *et al.* (1988) reported impaired recognition of nonmeaningful, but not meaningful, visual stimuli in a mixed-ability group; however, the control group was not matched for verbal ability in this study. Summers and Craik (1994) reported impaired word recognition; and Barth, Fein and Waterhouse

(1995) reported visual recognition impairment, although the impairment was not evident when differences in nonverbal ability were partialled out. Dawson *et al*. (1998, 2001) showed impaired performance on a test of delayed nonmatching to sample, but interpreted this finding in terms of impaired reward-association mechanisms, rather than as a recognition impairment *per se*.

Regarding prediction (b), there are no published studies assessing relations between recognition and conceptual-lexical and factual knowledge in LFA. In a recent study (papers in preparation) we set out to obtain additional data concerning recognition abilities in individuals with LFA, and to test the prediction that recognition relates to conceptual and lexical abilities in this group, but not in comparison groups. This study is briefly described next.

Unpublished findings

The aim of the study was to test predictions (a) and (b) as outlined above. A group of teenagers with V-LFA was compared with a young, ability-matched group of typically developing (TD) children, a group of children with HFA matched with the TD group for age, and an age- and language-ability-matched group of teenagers with intellectual disability (ID) without autism. To test the prediction that recognition memory will be uniquely impaired in individuals with LFA, we administered two visual recognition tasks, using nonmeaningful materials. To test the prediction that recognition will correlate with conceptual-lexical knowledge in individuals with LFA, but not in other groups, we gave the participants four tests assessing access to, and explicit use of, conceptual and lexical knowledge. We used analyses of covariance, controlling for differences in nonverbal abilities, to compare recognition memory in the four groups, and also to compare conceptual-lexical knowledge in the four groups. Bivariate correlation tests were used to assess relations between recognition and conceptual-lexical knowledge in each of the four groups.

The results of the study were in line with the predictions in so far as recognition was more impaired in the LFA group than in the other three groups, though only consistently significantly impaired relative to the TD group. There was, in addition, a strong positive correlation between recognition scores and scores on measures of conceptual and linguistic knowledge in the LFA group, but not in the TD or ID groups, although there was a trend towards positive correlation in the HFA group.

Summary

In sum, firm evidence of a pervasive declarative memory impairment in LFA, affecting recognition as well as recall, is currently lacking, although

the weight of the evidence is positive. Regarding the prediction that recognition and conceptual-linguistic knowledge will be related in people with the profile of autism-related language impairments, the evidence from our recent study supports the prediction, and is hard to explain except in terms of the hypothesis.

Other suggested explanations of language (and intellectual) impairments in low-functioning autism

It is certain that more than one causal factor contributes to structural language impairments in LFA, even before considering the additional effects of comorbid conditions which modify linguistic profiles in individuals and subgroups. It is therefore important to set our own theory into the context of other major theories, and to consider how other theories may relate to our own.

Several explanations of the language impairment in LFA have been proposed in the past, a few of which share with our own hypothesis the potential to explain the intellectual impairment, also, although this is rarely emphasized. In this section, the theories presented all relate primarily to the language impairment. Where they may have some potential to explain intellectual disabilities, this is mentioned.

Mindblindness

Mindblindness (Baron-Cohen, 1995) resulting from reduced empathy (Baron-Cohen, 2005) cannot but affect the way in which language as a shared conventional symbol system is acquired by people with ASDs (Hobson, 1993; Bloom, 2000). In particular, impaired mindreading would contribute to abnormal lexical development, given that typically developing children routinely infer the speaker's intention when forming an association between a novel object or action and a novel word – something that children with autism do not generally do (Baron-Cohen, Baldwin & Crowson, 1997; Preissler & Carey, 2005). It can be assumed, therefore, that mindblindness contributes to the abnormalities of lexical development that are so marked in people with LFA and which remain in subtle form in higher-functioning individuals (Happé, 1994). Mindblindness can also explain the problems that younger and less able individuals have in understanding and using deictic terms – i.e. terms whose meaning depends on the identity of the speaker ('you'/'me'), or the speaker's location ('here'/'there').

However, as pointed out by Bloom (2000), mindblindness cannot offer a sufficient explanation of language impairment in LFA because individuals with HFA/Asperger syndrome have impaired joint attention and

theory of mind but nevertheless develop clinically normal language. As Bloom argues, there must be other routes into language that people with HFA are able to utilize, but which are unavailable to people with LFA. According to the declarative memory hypothesis, the critical difference is that people with HFA, unlike people with LFA, have normal access to implicitly acquired knowledge-of-the world, and are therefore able to acquire a predominantly normal word-meaning system (as illustrated in Figure 14.1a).

Impaired symbol formation and use; impaired ability to form semantic categories

An early suggestion of 'asymbolia' (Ricks & Wing, 1975) is broadly compatible with the declarative memory hypothesis, so long as asymbolia is interpreted as a description of individuals with NV-LFA, with anomalous symbol formation characterizing V-LFA. Another, somewhat similar, early theory was that individuals with 'Kanner's syndrome' / 'early infantile autism' have impaired ability to form semantic categories which are integrated into an underlying conceptual system (Menyuk, 1978; Fay & Schuler, 1980; Tager-Flusberg, 1981). This theory is also compatible with our hypothesis, if understood as a difficulty in forming *explicit* semantic categories that fully connote *implicit* conceptual knowledge. Both these early theories have some potential to explain the intellectual impairments, as well as the language impairments, in LFA.

Specific language impairments

The early theory that specific language impairments (SLI) contribute to low-functioning autism (Churchill, 1972) has been reinvigorated by recent studies showing that the commonalities between SLI and language impairments in autism are more frequent (Rapin & Dunn, 2003) and more fine-grained (Kjelgaard & Tager-Flusberg, 2001; Roberts, Rice & Tager-Flusberg, 2004) than was previously thought. These findings pose challenges for understanding the relationship between autism and specific language impairments (SLIs) at all levels of causal analysis. However, we concur with Bishop (2004) and with Botting and Conti-Ramsden (2003) who conceptualize the relation between SLI and language impairment in autism in terms of a continuum of language-related impairments, features of which can occur in the various subtypes of SLI and also in association with autism, as a result of shared genetic risk factors. According to this view, SLI cannot by itself explain the language impairment in LFA, although the two conditions share some linguistic

features, and mixed forms of autism and SLI can occur at all levels of ability (Bartak, Rutter & Cox, 1975; Kjelgaard & Tager-Flusberg, 2001).

Impairments of sequencing and segmenting

Impaired sequencing has been suggested as a cause of language impairment and IQ troughs in LFA (Lincoln, Allen & Kilman, 1995; see also Tanguay, 1984). Sequencing is generally identified with the processing of transient or successive inputs such as heard speech, or seen sign language, that occur through time; and also with analytic as opposed to holistic processing. The suggestion of impaired analysis of transient or successive inputs is implicit in the impaired segmenting hypothesis proposed by Prizant (1983b) as an explanation of language impairment in LFA (see also Boucher, 2000).

Consistency between the explanations of language and intellectual impairments in LFA in terms of impaired declarative memory and in terms of impaired sequencing and segmenting may be achieved by building on insights into psychological processes underlying autism, some of which are presented in this book. Specifically, it was early suggested (Frith, 1989) that weak central coherence in autism might result from abnormalities of integrative neural binding. This hypothesis was argued for in greater detail by Brock *et al.* (2002), who suggested that whereas activity in local neural networks functions normally, the synchronization, or binding together, of activity across networks may be dysfunctional in autism. The notion of impaired binding as a contributory cause of various aspects of the behavioural abnormalities in autism is now increasingly invoked, including as an explanation of the declarative memory impairments and anomalies that occur in HFA/Asperger syndrome (e.g. in this volume: DeLong, Chapter 6; Webb, Chapter 10; and Bowler & Gaigg, Chapter 17). If, as seems intuitively likely, the synchronization of neural activity across disparate local networks is dependent on the same oscillatory or cyclic mechanisms that subserve the temporal analysis of transient inputs, then the memory impairments emphasized in this and other chapters in this book, and the sequencing–segmenting impairments noted by others, can be seen as having the same root cause.

Other explanatory theories

Ullman's (2004) model of the prerequisites for language acquisition is relevant to our hypothesis, in that he argues that lexical development is dependent on declarative memory, whereas phonological and grammatical development are dependent on procedural memory. Ullman

proposes, albeit very briefly, that procedural memory impairment under-lies language impairment in autism (also in SLI). This proposal appears to be based on a limited knowledge of the language profile in autism. However, his model may be important for an understanding of the relationship between autism and SLI.

Baron-Cohen (2006) has recently argued that an excessive tendency to systemize could cause, or contribute to, the language impairment in LFA and also the intellectual impairment. This theory has not been well developed, and cannot therefore be meaningfully assessed. However, in seeking a common explanation for the language and intellectual impair-ments together, Baron-Cohen is, as we are, following the precept of Bailey *et al*. referred to at the outset of this chapter, and this is to be welcomed.

Summary

Little attempt has been made to explain why language impairment and intellectual disability occur together in people with low-functioning autism, and this is regrettable for both practical and theoretical reasons. In this chapter we present behavioural arguments and evidence support-ing the hypothesis that both the language impairment and the ID derive in part from a pervasive impairment of declarative memory, affecting memory for factual information as well as personally experienced events, and manifested in impairments of recognition as well as recall. We describe the profiles of language ability and disability, and of intellectual ability and disability, most characteristic of people with LFA, and argue that diminished declarative memory, leaving proce-dural memory and immediate memory unimpaired, can explain the profiles seen in groups of individuals with V-LFA; whereas total, or near total, loss of declarative memory, leaving procedural and immedi-ate memory relatively intact in some but not all individuals, can explain the profiles associated with NV-LFA. We present evidence, including some from a recent study of our own, which provides some support for the hypothesis, although more investigation is needed. Finally, we con-sider alternative or additional explanations of the language impairment in LFA, and suggest how our own theory relates to other possible causal factors.

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References

Allen, M., Lincoln, A. & Kaufman, A. (1991). Sequential and simultaneous processing abilities of high-functioning autistic and language-impaired children. *Journal of Autism and Developmental Disorders*, *21*, 483–502.

Ameli, R., Courchesne, E., Lincoln, A., Kaufman, A. & Grillon, C. (1988). Visual memory processes in high functioning individuals with autism. *Journal of Autism and Developmental Disorders*, *18*, 601–615.

American Psychiatric Association (APA) (1994). *Diagnostic and statistical manual of mental disorders*, 4th edn (DSM-IV). Washington, DC: American Psychiatric Association.

Bachevalier, J. (1994). Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia*, *32*, 627–648.

Bailey, A., Phillips, W. & Rutter, M. (1996). Autism: towards an integration of clinical, genetic, neuropsychological, and neurobiological perspective. *Journal of Child Psychology and Psychiatry*, *37*, 89–126.

Baron-Cohen, S. (1995). *Mindblindness*. London: MIT Press.

Baron-Cohen, S. (2005). The empathising system: a revision of the 1994 model of the mindreading system. In B. Ellis & D. Bjorkland (eds.), *Origins of the social mind*, pp. 468–492. New York: Guilford Press.

Baron-Cohen, S. (2006). Two new theories of autism: hyper-systemising and assortative mating. *Archives of Diseases of Childhood*, *91*, 2–5.

Baron-Cohen, S. Baldwin, D. & Crowson, M. (1997). Do children with autism use the speaker's direction of gaze strategy to crack the code of language? *Child Development*, *68*, 48–57.

Bartak, L., Rutter, M. & Cox, A. (1975). A comparative study of infantile autism and specific developmental receptive language disorder: I. The children. *British Journal of Psychiatry*, *126*, 127–145.

Barth, C., Fein, D. & Waterhouse, L. (1995). Delayed match-to-sample performance in autistic children. *Developmental Neuropsychology*, *11*, 53–69.

Bartlett, C., Flax, J., Logue, M., Brett J., Smith, D., Vieland, V., Tallal, P. & Brzustowicz. L. (2004). Examination of potential overlap in autism and language loci on chromosomes 2, 7, and 13 in two independent samples ascertained for specific language impairment. *Human Heredity*, *57*, 10–20.

Bartolucci, G., Pierce, S., Streiner, D. & Tolkin-Eppel, P. (1976). Phonological investigation of verbal autistic and mentally retarded subjects. *Journal of Autism and Childhood Schizophrenia*, *6*, 303–315.

Bauman, M. L. & Kemper, T. L. (2004). Structural brain anatomy in autism: what is the evidence? In M. L. Bauman & T. L. Kemper (eds.), *The neurobiology of autism*, pp. 119–145. Baltimore: Johns Hopkins Press.

Ben Shalom, D. (2003). Memory in autism: review and Synthesis. *Cortex 39*: 1129–1138.

Bishop, D. V. M. (2004). Autism and specific language impairment: categorical distinction or continuum? In G. Bock & J. Goode (series eds.), *Autism: neural bases and treatment possibilities*. London: Novartis Foundation.

Bloom, P. (2000). *How children learn the meaning of words*. Cambridge, MA: MIT Press.

Bolton, P., Macdonald, H., Pickles, Arowson, M., Bailey, A. & Rutter, M. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry*, *35*, 877–900.

Botting, N. & 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*, 515–524.

Boucher, J. (1976). Articulation in early childhood autism. *Journal of Autism and Childhood Schizophrenia*, *6*, 297–302.

Boucher, J. (1978). Echoic memory capacity in autistic children. *Journal of Child Psychology and Psychiatry*, *19*, 161–166.

Boucher, J. (1981a). Memory for recent events in autistic children. *Journal of Autism and Developmental Disorders*, *11*, 293–301.

Boucher, J. (1981b). Immediate free recall in early childhood autism: another point of behavioural similarity with the amnesic syndrome. *British Journal of Psychology*, *72*, 211–215.

Boucher, J. (1988). Word fluency in high-functioning autistic children. *Journal of Autism and Developmental Disorders*, *18*, 637–647.

Boucher, J. (2000). Time parsing, normal language acquisition, and language-related developmental disorders. In M. Perkins & S. Howard (eds.), *New directions in language development and disorders*. London: Kluwer Academic/Plenum Publishers.

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

Boucher, J. & Lewis, V. (1989). Memory impairments and communication in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, *30*, 99–122.

Boucher, J. & Warrington, E. K. (1976). Memory deficits in early infantile autism: some similarities to the amnesic syndrome. *British Journal of Psychology*, *67*, 73–87.

Brock, J., Brown, C. C., Boucher, J. & Rippon, G. (2002). The temporal binding deficit hypothesis of autism. *Development and Psychopathology*, *14*, 209–224.

Carter, A. S., Volkmar, F., Sparrow, S., Wang, J-J., Lord, C., Dawson, G., Fombonne, E., Loveland, K., Mesibov, G. & Schopler, E. (1998). The Vineland Adaptive Behaviour Scales: supplementary norms for individuals with autism. *Journal of Autism and Developmental Disorders*, *28*, 287–302.

Cattell, R. B. (1971). *Abilites: their structure, growth and action*. Boston: Houghton Mifflin.

Churchill, D. (1972). The relation of infantile autism and early childhood schizophrenia to developmental languge disorders of childhood. *Journal of Autism and Childhood Schizophrenia*, *2*, 182–197.

Dawson, G., Meltzoff, A., Osterling, J. & Rinaldi, J. (1998). Neuropsychological correlates of early autistic symptoms. *Child Development*, *69*, 1247–1482.

Dawson, G., Osterling, J., Rinaldi, J., Carver, L. & McPartland, J. (2001). Recognition and stimulus-reward association: indirect support for the role

of ventromedial prefrontal dysfunction in autism. *Journal of Autism and Developmental Disorders*, *31*, 337–341.

DeLong, R. & Heinz, E. R. (1997). The syndrome of early-life bilateral hippocampal sclerosis. *Annals of Neurology*, *43*, 11–17.

DeMyer, M. K. (1976). Intellectual disabilities of autistic children. In L.Wing (ed.), *Early childhood autism*, pp. 169–193. Oxford: Pergamon Press.

Dobbinson, S. (2000). Repetitiveness and productivity in the language of adults with autism. Unpublished PhD thesis, University of Sheffield.

Dobbinson, S., Perkins, M. & Boucher, J. (2003). The interactional significance of formulas in antistic language. *Clinical Linguistics and Phonetics*, *17*, 299–307.

Fay, W. & Schuler, A. L. (1980). *Emerging language in autistic children*. London: Edward Arnold.

Folstein, S., Szantangelo, S., Gilman, S., Piven, J., Landa R., Lainhart, J., Hein, J. & Wzorek, M. (1999). Predictors of cognitive test patterns in autism families. *Journal of Child Psychology and Psychiatry*, *38*, 667–683.

Frith, U. (1989). *Autism: explaining the enigma*. Oxford: Blackwell.

Fyffe, C. & Prior, M. (1978). Evidence for language recoding in autistic, retarded and normal children. *British Journal of Psychology*, *69*, 393–402.

Happé, F. (1994). *Autism: an introduction to psychological theory*. London: UCL Press.

Hermelin, B. & O'Connor, N. (1967). Remembering of words by psychotic and normal children. *British Journal of Psychology*, *68*, 213–218.

Hobson, R. P. (1993). *Autism and the development of mind*. Hove: Erlbaum.

Horn, J. & Cattell, R. (1966). Refinement and test of the theory of fluid and crystalised general intelligences. *Journal of Educational Psychology*, *57*, 253–270.

Kanner, L. (1946). Irrelevant and metaphorical language in early infantile autism. *American Journal of Psychiatry*, *103*, 242–246.

Kjelgaard, M. & Tager-Flusberg, H. (2001). An investigation of language profiles in autism: implications for genetic subgroups. *Language and Cognitive Processes*, *16*, 287–308.

Klin, A., Sparrow, S., Marans, W., Carter, A. & Volkmar, F. (2000). Assessment issues in children and adolescents with Asperger syndrome. In A. Klin, F. Volkmar & S. Sparrow (eds.), *Asperger syndrome*. New York: Guilford Press.

Kraijer, D. (2000). Review of adaptive behaviour studies in mentally retarded persons with autism/pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, *30*, 39–48.

Lee, A., Hobson, R. P. & Chiat, S. (1994). I, you, me, and autism: an experimental study. *Journal of Autism and Developmental Disorders*, *24*, 155–176.

Lincoln, A., Allen, M. & Kilman, A. (1995). The assessment and interpretation of intellectual abilities in people with autism In E. Schopler & G. Mesibov (eds.), *Learning and cognition in autism*, pp. 89–118. New York: Plenum Press.

Lincoln, A., Courchesne, E., Kilman, B., Elmasian, R. & Allen, M. (1988). A study of intellectual abilities in high-functioning people with autism. *Journal of Autism and Developmental Disorders*, *18*, 505–523.

Lord, C. & Paul, R. (1997). Language and communication in autism. In D.Cohen & F.Volkmar (eds.), *Autism and pervasive developmental disorders*, 2nd edn, pp. 195–225. New York: Wiley.

Mackintosh, N. (1998). *IQ and human intelligence*. Oxford: Oxford University Press.

Mayes, S. D. & Calhoun, S. (2003a). Ability profiles in children with autism: influence of age and IQ. *Autism*, *7*, 65–80.

Mayes, S. D. & Calhoun, S. (2003b). Analysis of WISC-III, Stanford–Binet IV, and Academic Achievement Test scores in children with autism. *Journal of Autism and Developmental Disorders*, *33*, 329–342.

Menyuk, P. (1978). Language: what's wrong and why. In M. Rutter and E. Schopler (eds.), *Autism: a reappraisal of concepts and treatment*, pp. 105–116. New York: Plenum Press.

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.

Minshew, 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.

Narita, T. & Koga, Y. (1987). Neuropsychological assessment of childhood autism. *Advances in Biological Psychiatry*, *16*, 156–170.

O'Connor, N. & Hermelin, B. (1967). Auditory and visual memory in autistic and normal children. *Journal of Mental Deficiency Research*, *11*, 126–131.

Ohta, M. (1987). Cognitive disorders of infantile autism: a study employing the WISC, spatial relationship conceptualisation, and gesture imitations. *Journal of Autism and Developmental Disorders*, *17*, 45–62.

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

Preissler, M. (2006). Symbolic representation of pictures and words in children with autism. Paper presented at the Scottish Autism Research Group seminar series at Glasgow Caledonian University in September 2006.

Preissler, M. A. & Carey, S. (2005). The role of inferences about referential intent in word learning: evidence from autism. *Cognition 97*: B13–B23.

Prizant, B. (1983a). Echolalia in autism: assessment and intervention. *Seminars in speech and language*, *4*, 63–77.

Prizant, B. (1983b). Language acquisition and communicative behaviour in autism: towards an understanding of the 'whole' of it. *Journal of Speech and Hearing Disorders*, *48*, 296–307.

Rapin, I. (1996). *Preschool children with inadequate communication*. London: Mac Keith Press.

Rapin, I. & Dunn, M. (2003). Update on the language disorders of individuals on the autistic spectrum. *Brain and Development*, *25*, 166–172.

Raven, J. C., Court, J. & Raven, J. (1986). *Raven's Progressive Matrices*. London: H. K. Lewis.

Ricks, D. & Wing, L. (1975). Language, communication, and the use of symbols in normal and autistic children. *Journal of Autism and Childhood Schizophrenia*, *5*, 191–219.

Roberts, J., Rice, M. & Tager-Flusberg, H. (2004). Tense marking in children with autism. *Applied Psycholinguistics*, *25*, 429–448.

Rumsey, J. M. (1992). Neuropsychological studies of high-level autism. In E. Schopler & G. Mesibov (eds.), *High functioning individuals with autism*, pp. 41–64. New York: Plenum Press.

Scheuffgen, K., Happé, F., Anderson, M. & Frith, U. (2000). High 'intelligence', low 'IQ'? Speed of processing and measured IQ in children with autism. *Development and Psychopathology*, *12*, 83–90.

Siegel, D., Minshew, N. & Goldstein, G. (1996). Wechsler IQ profiles in diagnosis of high-functioning autism. *Journal of Autism and Developmental Disorders*, *26*, 389–406.

Summers, J. A. & Craik, F. I. M. (1994). The effects of subject-performed tasks on the memory performance of verbal autistic children. *Journal of Autism and Developmental Disorders*, *24*, 773–783.

Tager-Flusberg, H. (1981). On the nature of linguistic functioning in early infantile autism. *Journal of Autism and Developmental Disorders*, *11*, 45–56.

Tager-Flusberg, H. (1985a). Basic level and superordinate level categorisation by autistic, mentally retarded, and normal children. *Journal of Experimental Child Psychology*, *40*, 450–469.

Tager-Flusberg, H. (1985b). The conceptual basis for referential word meaning in children with autism. *Child Development*, *56*, 1167–1178.

Tager-Flusberg, H. (1991). Semantic processing in the free recall of autistic children: further evidence for a cognitive deficit. *British Journal of Developmental Psychology*, *9*, 417–430.

Tanguay, P. (1984). Toward a new classification of serious psychopathology in children. *Journal of American Academy of Child Psychiatry*, *23*, 374–384.

Tomblin J., Hafeman, L. & O'Brien, M. (2003). Autism and autism risk in siblings of children with specific language impairment. *International Journal of Language and Communication Disorders*, *38*, 235–250.

Ullman, M. (2004). Contributions of memory circuits to language. *Cognition 92*, 231–270.

Volden, J. & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders*, *21*, 109–130.

Wassing, H. (1965). Cognitive functioning in early infantile autism: an examination of four cases by means of the Wechsler intelligence scale for children. *Acta Paedopsychiatrica*, *32*, 122–135.

Wechsler, D. (1974). *Wechsler Intelligence Scale for Children – revised manual*. New York: Psychological Corporation.

Wechsler, D. (1981). *Wechsler Adult Intelligence Scale – revised manual*. New York: Psychological Corporation.