Memory, learning and language in autism spectrum disorder

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Abstract
Background and aims: The ‘dual-systems’ model of language acquisition has been used by Ullman et al. to explain patterns of strength and weakness in the language of higher-functioning people with autism spectrum disorder. Specifically, intact declarative/explicit learning is argued to compensate for a deficit in non-declarative/implicit procedural learning, constituting an example of the so-called see-saw effect. Ullman and Pullman extended their argument concerning a see-saw effect on language in autism spectrum disorder to cover other perceived anomalies of behaviour, including impaired acquisition of social skills. The aim of this paper is to present a critique of Ullman et al.’s claims and to propose an alternative model of links between memory systems and language in autism spectrum disorder.

Main contribution: We argue that a four-system model of learning, in which intact semantic and procedural memory are used to compensate for weaknesses in episodic memory and perceptual learning, can better explain patterns of language ability across the autistic spectrum. We also argue that attempts to generalise the ‘impaired implicit learning/spared declarative learning’ theory to other behaviours in autism spectrum disorder are unsustainable.

Conclusions: Clinically significant language impairments in autism spectrum disorder are under-researched, despite their impact on everyday functioning and quality of life. The relative paucity of research findings in this area lays it open to speculative interpretation which may be misleading.

Implications: More research is needed into links between memory/learning systems and language impairments across the spectrum. Improved understanding should inform therapeutic intervention and contribute to investigation of the causes of language impairment in autism spectrum disorder with potential implications for prevention.

Keywords
Autism, language, declarative/explicit memory, non-declarative/implicit memory, see-saw effect

Introduction
According to the dual-systems model of language acquisition, as argued for by Ullman (2001, 2004), certain facets of language, notably knowledge of the items and combinatorial rules of phonology and grammar, are acquired unconsciously and are not generally accessible to verbal report, i.e. they are ‘implicit’ and ‘non-declarative’. By contrast, lexical-semantic knowledge is available to conscious thought and verbal report, i.e. it is ‘explicit’ and ‘declarative’.

Based on this model, Ullman et al. have argued that structural language in people with autism spectrum disorder (ASD) reflects impaired non-declarative learning in combination with increased reliance on intact declarative learning (Walenski, Mostofsky, Gidley-Larson, & Ullman, 2008; Walenski, Mostofsky, & Ullman, 2014; Walenski, Tager-Flusberg, & Ullman, 2006). Ullman and Pullman (2015) have subsequently argued that declarative learning is used to compensate for deficiencies in non-declarative learning of various kinds in a number of neurodevelopmental and mental health disorders, including ASD, referring to this as a ‘see-saw effect’.

Ullman’s group has a well-earned reputation for scholarship in the field of neuropsychology, and their views are influential. Their interpretations of linguistic and other anomalies of learning in ASD are, however, in our opinion inconsistent with available evidence. In this paper, we first say why we think that their
views are untenable, critiquing each of four publications (two theoretical, two empirical) by this group in which their interpretations of anomalous learning in ASD are propounded. In the second part of the paper, we outline a more differentiated four-system model of language acquisition. We also summarise evidence relating to the intactness or otherwise of each of these learning systems in ASD, and suggest how the mixture of strengths and weaknesses links to language profiles across the spectrum. Finally, we utilise our preferred model of learning systems to offer a reinterpretation of findings from the two empirical papers on language in ASD published by Ullman’s group. We also propose a reformulation of the see-saw effect across language and other behaviours in ASD, using our preferred four-system model.

There are, of course, very many alternative models of normal language development (see Ambridge & Lieven, 2011, for discussion of key models). There is also a multiplicity of factors involved in language acquisition, other than the selective cognitive competences considered here. However, neither alternative models nor other contributory factors will be considered in this paper, the limited aims of which are first to offer a critique of Ullman et al.’s claims regarding ASD; and second to indicate how a more nuanced version of their dual-route model might provide a more accurate analysis of some linguistic anomalies in the case of autism.

The dual-systems model of language acquisition as applied to ASD by Ullman et al.

In this section, Ullman et al.’s four publications relating to language acquisition and learning in ASD are summarised, and our reasons for questioning the views expressed are stated.

Walenski, Tager-Flusberg and Ullman (2006)

Summary. In this theoretical paper, Walenski et al. argued that phonological and grammatical abilities tend to be impaired in ASD, whereas lexical-semantic knowledge is relatively unimpaired. Utilising the dual-systems model of language acquisition, Walenski et al. explained this uneven language profile in terms of a deficit in unconscious (‘implicit’/‘non-declarative’) learning, in combination with relatively spared conscious (‘explicit’/‘declarative’) learning ability. More specifically, they identified the deficit of unconscious learning with impaired procedural memory, citing the ‘procedural deficit hypothesis’ (PDH) as proposed by Mostofsky, Goldberg, Landa and Denkla (2000) as an explanation of motor impairments in ASD. Walenski et al. (2006) further proposed that intact declarative learning would be utilised to compensate for impaired procedural learning in what Ullman (2004) had referred to as a ‘see-saw effect’.

Critique. The claim that phonology and grammar are more impaired than lexical-semantic knowledge in ASD is inconsistent with evidence from most empirical studies. Specifically, grammatical abilities (syntax and morphosyntax) are largely if not entirely unimpaired in school-age children and adults with higher-functioning ASD (HF-ASD) (see reviews by Boucher, 2012; Kim, Paul, Tager-Flusberg & Lord, 2014; Williams, Botting & Boucher, 2008). Notably, Kim et al. conclude that ‘syntactic development is more similar than dissimilar to normal development’ (i.e. generally commensurate with developmental age) across the spectrum, suggesting that procedural memory is unimpaired not only in individuals with HF-ASD but also in those who are less able. Two caveats are relevant, however.

First, it is important to point out that intact grammar may only be observed in individuals with HF-ASD once they have reached junior school age (i.e. from c. age 7.0 years). During the preschool/infant school years, children with HF-ASD frequently have significant grammatical impairments, probably related to late language onset (Eigsti, Bennetto, & Dadlani, 2007; Rapin & Dunn, 2003; Rapin, Dunn, Allen, Stevens, & Fein, 2009). Second, some subtle syntactic anomalies have been observed in individuals with HF-ASD, especially in those whose language normalised following initial delay (Durrleman, Hippolyte, Zufferey, Iglesias & Hadjikhani, 2015). These anomalies may result indirectly from pragmatic or prosodic impairments, rather than from a procedural learning impairment (Prévost, Tuller, Barthez, Malvy, & Bonnet-Brilhaut, 2017; Terzi, Marinis, & Francis, 2016). However, a study by Perovic, Modyanova and Wexler (2013) demonstrated impaired comprehension of reflexive pronouns not easily explained in terms of impaired pragmatics or prosody. Perovic et al.’s groups were small, and the authors comment that further studies with larger groups are needed. However, even if their findings were confirmed, it is likely – given the weight of evidence of mental-age-appropriate syntactic development in ASD – that this (or other subtle syntactic impairments that may be demonstrated) are explicable in terms of one or other of the multiple factors contributing to the acquisition of syntax, rather than in terms of any generalised deficit in implicit learning. Additional factors contributing to syntactic development include hearing acuity, attentional factors, short-term memory, spatial and temporal processing, and concepts of self and other, to name just a few.

Regarding phonology, in particular phonotactics, i.e. the acquisition and use of the rules and regularities
governing combinations of phonemes, there is very little evidence relating to ASD. Two detailed studies of speech production by individuals with HF-ASD, including many with a diagnosis of Asperger syndrome, reported high rates of phonemic abnormalities which might constitute evidence of phonotactic impairment (Cleland, Gibbon, Peppe, O’Hare, & Rutherford, 2010; Shriberg, Paul, McSweeny, Klin, & Cohen, 2001). However, the data reported are more readily interpreted in terms of impaired phoneme acquisition, as is suggested in a later section of the present paper.

Regarding Walenski et al.’s (2006) explanation of perceived grammatical impairments in ASD, the PDH was – as noted above – first proposed as an explanation of motor impairments in autism (Mostofsky et al., 2000; see also Romero-Munguia 2008). However, more recent studies of motor learning do not support this explanation. On the contrary, studies by Barnes et al. (2008), Brown, Aczel, Jimenez, Kaufman and Grant (2010), Nemeth et al. (2010) and Travers, Klinger, Mussey and Klinger (2010) and a meta-analysis of such studies by Foti, De Crescenzo, Vivanti, Menghini and Vicari (2015) show that the ability to learn sequences of motor behaviour is completely normal in HF-ASD. Moreover, in recent authoritative reviews of studies delineating and attempting to explain patterns of motor ability and disability in ASD, neither Gowen and Hamilton (2013) nor Bodison and Mostofsky (2014) refer to the PDH.

In their study cited above, Brown et al. (2010) did not only assess procedural learning of motor sequences but they also assessed procedural memory using tests of artificial grammar learning and probabilistic learning. There were no impairments in the ASD group on any of these tasks relative to appropriately matched comparison groups. Moreover, Klinger, Klinger and Pohlig’s (2006) claim that lack of impairment on implicit procedural learning tasks in HF-ASD reflects high intelligence and compensatory use of explicit rule learning was assessed by Brown et al. and disconfirmed. So impressed were Nemeth et al. (2010) by the performance of their ASD group over a variety of implicit learning tasks that they entitled the paper reporting their findings: ‘Learning in autism: Implicitly superb’.

This conclusion has received recent support from two further meta-analyses. Obeid, Brooks, Powers, Gillespie-Lynch and Lum (2016) examined findings on six statistical learning tasks: serial reaction time, artificial grammar learning, contextual cueing, speech stream, observational learning and probabilistic classification. Performance by individuals with ASD on all these tasks was superior to that of individuals with specific language impairment (SLI), and unimpaired relative to other comparison groups. Clark and Lum (2017) carried out a meta-analysis of performance on the serial reaction time task across six disorders: ASD, developmental coordination disorder, dyslexia, Parkinson’s disease, schizophrenia and SLI. Procedural learning was spared in ASD in striking contrast to the five other disorders where there were clear impairments.

Finally, the conclusion from research studies that procedural learning is a notable strength in people with ASD across the spectrum is reinforced by observation of everyday behaviour. This is marked by an unusual degree of reliance on entrenched associations, habits and routines such as are generally acquired implicitly through experience, rather than by explicit learning (Schacter & Tulving, 1994; Ullman, 2004).

**Walenski, Mostofsky, Gidley-Larson and Ullman (2008)**

**Summary.** In this short empirical paper, Walenski et al. reported enhanced (speeded) picture-naming in boys with HF-ASD when compared to typically developing (TD) boys. However, speeded naming only occurred on low-frequency words. Moreover, there were no differences in naming speed when HF-ASD boys were compared with TD girls.

Walenski et al. explained the combination of enhanced speed of naming of low-frequency words with normal speed of naming high-frequency words in terms of an argument proposed by Levelt (2001) and Levelt, Roelofs and Meyer (1999). Levelt et al. argued that differences in word-finding speed for high- and low-frequency words derive from a sub-stage of lexical, i.e. declarative, processing. Using this argument, Walenski et al. suggested that the ASD boys’ habitual reliance on declarative learning systems to compensate for ‘impaired procedural memory’ (the so-called seesaw effect) would enhance naming speed selectively for low-frequency words. Walenski et al. further argued that the lack of a difference between the boys with HF-ASD and TD girls was consistent with the known female advantage, relative to males, in lexical and declarative memory (Ullman, 2004; Ullman et al., 2007).

**Critique.** The substantial body of evidence of intact procedural learning in ASD, cited above, argues conclusively against Walenski et al.’s assumption of impaired procedural learning, undermining their interpretation of the findings. And if procedural learning is unimpaired, as all the available evidence suggests, then one needs to ask what declarative memory is compensating for?

**Walenski, Mostofsky and Ullman (2014)**

**Summary.** In this empirical paper, Walenski et al. reported speeded grammatical processing in high-
functioning boys with ASD. Specifically, the boys with HF-ASD were significantly faster than TD boys of the same age and verbal ability at producing regular, rule-governed past tenses of English verbs, e.g. ‘slipped’ as the past tense of ‘slip’. They were also faster when asked to generate a regular, rule-governed past tense of a nonsense word, e.g. ‘plimmed’ as the past tense of ‘plim’. However, they were no faster than their TD peers at producing irregular past tense forms of English verbs, e.g. ‘brought’ as the past tense of ‘bring’. Nor were they faster when generating irregular past tense forms of nonsense words, e.g. ‘splam’ as the past tense of ‘splim’. Accuracy, i.e. the correctness or appropriacy, of the responses generated did not differ across the two groups.

Walenski et al. (2014) interpreted these findings in terms of normal declarative memory in HF-ASD underlying intact acquisition of irregular past tense verb forms, combined with an impairment of procedural learning manifesting in ‘unsuppressed and therefore speeded’ grammatical processing of the regular past tense verb form.

**Critique.** Walenski et al.’s suggestion that a procedural memory deficit could lead to unsuppressed and therefore speeded grammatical processing in HF-ASD is undermined by the substantial evidence of intact procedural processing in ASD across the spectrum, as summarised earlier. Moreover, the suggestion that a procedural learning deficit could lead to unimpaired but speeded grammatical processing is counter-intuitive. In support of their suggestion, Walenski et al. cite evidence of speeded grammatical processing in Tourette’s Syndrome (Walenski, Mostofsky, & Ullman, 2007). However, in their Tourette’s participants speeded grammatical processing occurred in the absence of a procedural memory deficit, consistent with what might be intuitively expected.

Walenski et al. are obviously aware that their procedural deficit explanation of unimpaired but speeded grammatical processing in HF-ASD is questionable because they allow that their findings might reflect ‘enhanced’ rather than defective procedural learning. However, they dismiss this possibility, stating ‘we are not aware of any independent evidence of enhanced procedural learning in autism’. Whilst we agree that there is no evidence of superior procedural learning in ASD, there is a wealth of evidence of intact procedural learning (as cited above). And later in this paper we argue for a pattern of learning strengths and weaknesses in HF-ASD such as might underlie enhanced reliance on, and hence practice in using, this form of learning, consistent with speeded grammatical processing.

**Ullman and Pullman (2015)**

**Summary.** Ullman and Pullman (2015) propose that a see-saw effect occurs in people with ASD in which declarative learning is used to compensate for deficiencies in non-declarative learning across a range of behaviours, not exclusively linguistic. In this paper, Ullman and Pullman specifically do not argue for a deficit in procedural learning, but refer instead to deficiencies in ‘implicit’ learning. Ullman and Pullman are also cautious in the claims they make concerning the intactness of declarative learning. Thus, they conclude from available evidence that individuals with HF-ASD have ‘intact memory for facts, percepts, and associations, with mild episodic memory impairments’ (see Gaigg, Bowler & Gardiner, 2014 for a particularly clear demonstration of the dissociation). They also accept (citing evidence tabulated in the review of memory abilities in ASD by Boucher, Mayes & Bigham, 2012) that declarative memory in lower-functioning people with ASD (LF-ASD) is probably more impaired than in people with HF-ASD.

Having established that declarative learning is predominantly intact – at least in HF-ASD – Ullman and Pullman cite behavioural evidence in support of their claim that declarative learning is used to compensate for certain behavioural impairments. In particular, they cite (i) the use of formulaic language ‘to compensate for linguistic and social deficits’; (ii) reliance on explicitly memorised rules, scripts and event schemas to compensate for deficits in the implicit (unconscious) acquisition of social behaviours; (iii) the use of explicit (conscious) problem-solving abilities to ‘hack out’ solutions to tests of theory of mind; (iv) the use of consciously formulated rules to establish categories and concepts such as are typically acquired unconsciously/implicitly; and (v) the reliance of therapeutic interventions on explicit teaching methods. References to the behavioural evidence cited by Ullman and Pullman can be found in their paper.

In sum, although the PDH is not invoked by name in the sections of Ullman and Pullman’s paper which relate to ASD, impairments of implicit knowledge and learning are identified as driving the need to compensate by utilising declarative learning.

**Critique.** Abandonment of the PDH is to be welcomed in view of the overwhelming evidence against it. However, use of the term ‘implicit’ to replace ‘procedural’ does not greatly help Ullman and Pullman’s argument for a ‘spared declarative-impaired non-declarative/implicit’ see-saw effect in the case of ASD.

In particular, deficits in the unconscious/implicit acquisition of social behaviours and capacities (points (ii) and (iii) above) are likely to result from primary social deficits, rather than from impaired implicit learning. Evidence of unimpaired – indeed ‘superb’ – implicit learning across a range of tasks has been referred to above (Brown et al., 2010, and Nemeth et al., 2010; also the reviews by Foti et al., 2015; Obeid, Brooks,
mental disorders, Happe´ and Frith conclude that in a review of atypical social development in neurodevelopmental disorders are discussed by Happe´ and Frith, but no specific causal links between these impairments and biological motion processing, emotion regulation and alexithymia and impairments of agent identification, the case of ASD, primary social deficits may include unimpaired procedural learning. Therefore, it may be concluded, therefore, that there is no generalised impairment of implicit learning or implicit knowledge in ASD. The selective impairments of implicit social learning ((iii) above) and implicit social cognition ((iii) above) are, on the other hand, readily explained in terms of primary social impairments in ASD (Frith, 2013; Happé & Frith, 2014). In their review of atypical social development in neurodevelopmental disorders, Happé and Frith conclude that in the case of ASD, primary social deficits may include alexithymia and impairments of agent identification, biological motion processing, emotion regulation and implicit mentalising. Possible causal links between these impairments are discussed by Happé and Frith, but no conclusions drawn.3

Similarly, impaired and anomalous category formation (point (iv) above) cannot be explained by a generalised impairment of implicit/non-declarative learning, in view of evidence of unimpaired procedural learning in ASD. On the other hand, anomalous category formation is entirely consistent with well-known and extensively researched anomalies of sensory-perceptual processing. Sensory-perceptual anomalies, like primary social impairments, are diagnostic of ASD (American Psychiatric Association, 2013). Originally characterised as reflecting ‘weak central coherence’ (Frith & Happé, 1994) or ‘enhanced perceptual functioning’ (Mottron & Burack, 2001), these anomalies are now thought to comprise superior discrimination abilities in combination with impaired generalisation as first proposed by Plaisted, O’Riordan and Baron-Cohen (1998) and most recently argued for by Davis and Plaisted-Grant (2014; see also Pellicano & Burr, 2012). More is said below concerning our preferred explanations of the causes of impaired social learning and impaired category formation in ASD.

These arguments do not invalidate Ullman and Pullman’s contention that explicit forms of learning are utilised compensatorily in the ways they suggest. However, they do underline the need for a clear and convincing account of exactly what deficits in ASD force an unusual degree of reliance on explicit, declarative learning.

A four-system model of language acquisition in ASD

In our own work on memory and language in ASD (Bott, Brock, Brockdorff, Boucher, & Lamberts, 2006; Boucher, Bigham, Mayes, & Muskett, 2008; Boucher, 2012) we have generally used a model of learning systems which subdivides both declarative and non-declarative memory into two functionally and neuroanatomically differentiable subsystems, as originally proposed by Tulving (1991) (see also Schacter & Tulving, 1994).4 Thus, perceptual memory and procedural memory are differentiable forms of non-declarative learning; and semantic memory and episodic memory are differentiable forms of declarative learning. It is important to point out, however, that although better differentiated than the declarative/non-declarative model of learning utilised by Walenski, Ullman et al. in their publications relating to ASD, the four-system model is in its turn an oversimplification of the multiple interactive and overlapping processes involved in individual instances of everyday learning (Cabeza & Moscovitch, 2013). Nevertheless, the model provides a useful framework, we believe, for understanding links between memory and language acquisition in the case of autism.

Definitions of each of these four subsystems and their roles in normal language acquisition are outlined below, followed by a summary of what is known about these forms of learning in ASD.

Non-declarative, implicit learning systems

Perceptual memory. This non-declarative learning system registers and briefly retains ‘snapshot’ records of single items such as a face in a crowd, a bird call, a whiff of scent (Kellman, 2002; Kellman & Garrigan, 2009). Occurring individually, such briefly retained percepts may influence how one feels or behaves at the time or subsequently, although experienced below the level of awareness. For example, in the phenomenon known as ‘perceptual priming’ a subliminally registered percept lowers the threshold for responding to a related stimulus. In the phenomenon known as ‘contextual cueing’, a subliminally registered percept may, if re-experienced, spontaneously cue recall of an object or event with which it originally co-occurred.

Perceptual memory contributes to language acquisition primarily through its role in category and concept formation. Precisely how categories and concepts are formed and organised is complex and controversial (Mareschal, Quinn, & Lea, 2010). However, according to one influential model which has been used in numerous tests of category formation in ASD, the repeated experience of a particular percept in slightly varying forms, referred to as ‘exemplars’, underlies the unconscious formation of individualised items of knowledge (Mayor & Plunkett, 2010). At the core of each item is a ‘prototype’ around which a prescribed degree of generalisability, or variation, can occur. For example, a prototypical bicycle has two large wheels, handlebars, a seat and pedals, but bicycles in general instantiate multiple variations.
around this prototype. In the case of language, category formation underlies the acquisition of concepts underlying lexical meaning; it also underlies the acquisition of first-language phonemes.

Available evidence indicates that whereas perceptual priming and contextual cueing are intact in people with ASD (Bowler, Matthews, & Gardiner, 1997; Brown et al., 2010; Renner, Klinger, & Klinger, 2000; Toichi, 2008), category formation is at best achieved anomalously and/or more slowly than normal, and in some circumstances is significantly impaired (Bott et al., 2006; Church et al., 2010; Froehlich et al., 2012; Gastgeb, Dundas, Minshew, & Strauss, 2012; Mercado & Church, 2016; Mercado et al., 2015; Molesworth, Bowlter, & Hampton, 2005; Soulières, Mottron, Giguère, & Larochelle, 2011; Vladushich, Olu-Lafe, Kim, Tager-Flusberg, & Grossberg, 2010).

Impaired or anomalous category formation is consistent with current models of primary sensory-perceptual anomalies in ASD (already referred to above) – in particular, Plaisted et al.’s (1998) ‘enhanced-discrimination reduced-generalisation theory’ and Pellicano’s ‘hypo-priors’ theory (Pellicano & Burr, 2012; see also Davis & Plaisted-Grant, 2014; Lawson, Rees & Friston, 2014). These well-supported theories converge on the conclusion that people with ASD have enhanced sensitivity to sensory detail associated with impaired ability to detect similarities between non-identical exemplars of a particular category, i.e. impaired generalisation (Church et al., 2015). Impaired generalisation in turn results in the formation of narrow or weakly defined prototypes – what Pellicano and Burr refer to as ‘hypo-priors’. So, for example, a child with ASD might be exceptionally sensitive to the differences between one brand of cornflakes and another, but insensitive to the similarities and therefore reluctant to eat cornflakes of an unfamiliar brand.

Anomalous category formation has clear implications for conceptual networks underlying linguistic meaning, helping to explain why words are often used with narrowed or idiosyncratic meaning by people with ASD across the spectrum (Asperger, 1944/1991; Eigsti, Bennetto, & Dadlani, 2007; Fay & Schuler, 1980; Mayes, Calhoun, & Crites, 2001). A study of semantic priming by Kamio and Toichi (2007) showing normal effects of closely semantically related primes but impaired effects of more peripherally related primes is also consistent with a narrowed semantic meaning base.

Anomalous category formation may also help to explain the subclinical phonemic anomalies in people with ASD noted above. However, there is no research evidence, to the best of our knowledge, relating to this possibility.

**Procedural memory.** This non-declarative learning system involves the unconscious perception, extraction and registration of regularities within sequences of sensory-perceptual experience and is involved in conditioning and the acquisition of associations, habits/routines and skills whether motor, social, cognitive or linguistic (Schacter & Tulving, 1994; Ullman, 2004; cf also Baron-Cohen, 2009, in which he articulates his concept of ‘systemising’).

With regard to language, procedural learning sub-serves the acquisition and use of the unconsciously internalised rules and regularities for combining words and word parts, i.e. syntax and morphosyntax. It also underlies the acquisition and use of implicit knowledge of the rules and regularities for combining phonemes, i.e. phonotactics. As noted in a previous section, grammar is generally mental-age appropriate in older children and adults with ASD across the spectrum, consistent with other evidence of intact procedural learning, as summarised earlier. Also as noted above, minor phonemic errors in autistic speech such as might theoretically result from impaired phonotactics are more readily explained by anomalous category formation.

**Declarative, explicit learning systems**

**Semantic/single item memory.** This declarative memory system sub-serves the acquisition of single items of decontextualised factual information, such as the fact that Paris is the capital city of France; that Shakespeare wrote a play called ‘Hamlet’. It also underpins acquisition of the link between a concept or item of knowledge with the word, or words, denoting the concept or item. So, for example, semantic memory enables one to learn that the word ‘Paris’ is the name for the capital city of France; that ‘dog’ refers to an animal with four legs, which barks and wags its tail; that the phrase ‘The sky’s the limit’ refers to high or unstinted ambition. The essential role of this memory system in the acquisition of a meaningful vocabulary of substantive single items or stock phrases is inherent in the term ‘semantic’ and highlighted in the term ‘lexical semantics’.

Semantic memory is most commonly assessed using recognition tests, performance on which is largely dependent on stimulus familiarity (Montaldi & Mayes, 2010; Yonelinas, 2002). Numerous empirical studies using recognition tests demonstrate conclusively that semantic memory is unimpaired in people with HF-ASD (see the review by by Boucher et al., 2012). This conclusion is reinforced by clinical and anecdotal observations of ‘encyclopaedic memory for facts’ (Wing, 1996) and by above-average performance on the Information subtest of the Wechsler Scales by people with HF-ASD (Koyama, Tachimori, Osada,
Takeda, & Kurita, 2007; Spek, Scholte, & van Berckelaer-Onnes, 2008). With regard to language, intact semantic/single item learning is evident in the large single-word vocabularies acquired by many individuals with HF-ASD. It is also consistent with the tendency to use formulaic language, i.e. repetitive use of stock phrases acquired as undifferentiated ‘chunks’. However, when learning is additionally dependent on social or emotional competences and understanding, learning may be impaired or anomalous. Effects on language include a relative paucity of words denoting mental states such as ‘think’, ‘believe’, ‘feel’ (Tager-Flusberg, 1995) or emotions (Hobson & Lee, 1989). Correct use of deictic terms, which involve comparing one’s own states such as ‘think’, ‘believe’, ‘feel’ (Tager-Flusberg, 1995) or emotions (Hobson & Lee, 1989). Correct use of deictic terms, which involve comparing one’s own with another person’s perspective, is also difficult to acquire (Hobson, García-Pérez, & Lee, 2010).

In lower-functioning (but formally testable) individuals with ASD, recognition tests tend to suggest that semantic/single item memory is impaired (Boucher et al., 2008; Boucher, 2012). Moreover, there is no evidence of a notable capacity to acquire factual knowledge in a field of special interest, and performance on the Information subtest of the Wechsler Scales is commensurate with sub-average performance on the Vocabulary and Similarities subtests, rather than constituting a relative peak of ability (Dawson, Soulieres, Gernsbacher, & Mottron, 2007). We have argued that impaired semantic memory plays a major role in the impairments of language seen in LF-ASD (Boucher, 2012; Mayes & Boucher, 2008). Similarities between scores on the Information, Vocabulary and Similarities subtests of the Wechsler scales are consistent with this hypothesis.

**Episodic/relational memory.** This declarative learning system subserves the acquisition and recall of memories of complex, multi-component stimuli including personally experienced events. In the case of memory for a personally experienced event, components of the memory might include where and when the event occurred, who one was with, how one felt at the time, what the weather was like, what happened just before or just after the event, and so on. In the case of complex stimuli that do not constitute personally experienced events, the individual components of such stimuli (e.g. a painting of a landscape; the characters and events in a novel) must be remembered as related to each other, and for this reason, episodic memory is sometimes referred to as ‘relational memory’. Because episodic memory is generally assessed using tests of recall, it is more dependent on processes associated with recollection, as opposed to familiarity (Montaldi & Mayes, 2010; Yonelinas, 2002).

Episodic/relational memory is important for the enrichment of word meanings. For example, the word ‘Paris’ has enriched meaning for anyone who has visited or lived there. Similarly, the word ‘dog’ acquires a rich set of connotations gleaned from the varied experiences of many different dogs in many different situations. It follows that although a large vocabulary may be acquired using semantic memory, in the absence of intact episodic/relational memory, word meanings will be impoverished, tending to ‘denote’ rather than to ‘connote’ (Fay & Schuler, 1980).

There is now overwhelming evidence that episodic/relational learning is impaired across the spectrum for people with HF-ASD, aggravating the effects of anomalous category formation. An unusual degree of reliance on semantic/single-item learning compensating for a weakness in episodic/relational learning is also consistent with the fact that whereas people with HF-ASD generally perform well on single-word vocabulary tests, they perform less well on tests of higher-order language processing (Dunn & Bates, 2005; Tager-Flusberg & Joseph, 2003). As noted by Kelley, Paul, Fein and Naigles (2006), vocabulary tests assess knowledge of the most basic identification function of words but do not probe conceptual networks underlying word meanings.

**Application of the four-system model to Ullman et al.’s empirical findings, and a reformulation of the see-saw effect in ASD**

**Enhanced production of low-frequency words in boys with HF-ASD (Walenski et al., 2008) – A reinterpretation**

The simplest explanation of enhanced (speeded) production of low-frequency picture names by boys with HF-ASD is that vocabulary was not matched across groups in this study; groups were matched on full-scale IQ, leaving open the possibility of a mismatch on language ability. It may therefore have been the case that the boys with HF-ASD had more advanced vocabularies than the TD boys. Any difference in vocabulary knowledge would have been more likely to affect speed of naming low-frequency words as opposed to high-frequency words. Failure to equate groups for vocabulary levels could also explain why the boys with HF-ASD were superior to TD boys but not to TD girls.

It is also relevant to note that a selective advantage in the production of low-frequency words is consistent
with clinical and empirical evidence of a tendency for able people with ASD to use unusual words or phrases (Asperger, 1944/1991; Dunn, Gomes, & Sebastian, 1996; Tantam, 1988; Volden & Lord, 1991). Thus, for these able individuals the usual distinction between low- and high-frequency vocabulary items may not apply: they will perform similarly – or somewhat similarly – across the two subsets of vocabulary items. By contrast, in neurotypical groups naming speed for lower-frequency words will drop in comparison to naming speed for higher-frequency words as shown in Figure 1(a) of Walenski et al.’s paper.

If, however, the groups were in fact well matched for language (using some test other than picture identification or naming), speeded naming might alternatively be explained in terms of a see-saw effect. Unlike Walenski et al., however, we would suggest that the effect occurs within declarative memory, with intact semantic/single-item memory compensating for impaired episodic/relational memory. Picture-naming is, in effect, a test of the basic identification function of words, which, as suggested above, capitalises on single-item learning.

Enhanced production of regular past tense verb endings in boys with HF-ASD (Walenski et al., 2014) – A reinterpretation

Our interpretation of this finding is straightforward, once it is accepted that procedural learning is intact in people with ASD across the spectrum, whereas declarative learning is partially impaired. In people with HF-ASD, this would result in an unusual degree of reliance on procedural learning and function, with enhanced ability to generate regular past tense verb forms.

The accuracy of the HF-ASD participants’ responses on tests of knowledge and use of irregular past tense verb forms is consistent with their intact semantic memory. However, in the previous subsection we suggested that in people with HF-ASD there is a within declarative memory see-saw effect in that intact semantic/single item memory is relied on to an unusual degree to compensate for impaired episodic/relational memory. We used this claim to explain speeded naming. Assuming that the acquisition of irregular past tense forms is also unusually dependent on intact lexical-semantic learning it is logical to ask why the ability to generate such forms was not also speeded. We suggest that the explanation lies in differences between substantive single-word vocabulary items such as have a simple identification function (as proposed by Kelley et al., 2006), as opposed to vocabulary items such as irregular past tense forms that do not have this basic identification function.

See-saw effects in ASD – A reformulation

As will be clear from preceding sections of this paper, we agree with Ullman and Pullman (2015) that see-saw

Table 1. The four-system model of learning abilities in ASD, including the concept of a ‘see-saw’ effect of distinct compensatory learning systems in HF-ASD and LF-ASD.

| UNEVEN LEARNING ABILITIES IN ASD | Non-declarative/implicit systems | Declarative/explicit systems |
|----------------------------------|----------------------------------|------------------------------|
|                                  | Perceptual memory                | Procedural memory            |
|                                  | Semantic memory                  | Episodic memory              |

HF-ASD Mainly intact (but primary sensory-perceptual anomalies impair category formation; and primary social deficits impair implicit social learning) Intact Impaired

See-saw effect Used to compensate for impaired episodic memory

LF-ASD Mainly intact (but primary sensory-perceptual anomalies impair category formation; and primary social deficits impair implicit social learning) Impaired Impaired

See-saw effect Used to compensate for across-the-board impairment of explicit learning
effects occur in people with ASD. However, as will also be clear from preceding sections, we consider that the claim that declarative memory compensates for impairments of non-declarative/implicit learning in ASD is inconsistent with the evidence.

Our view of where ‘see-saw’ effects, i.e. compensatory strategies, come into play in people with HF-ASD, and also – differently – in people with LF-ASD is summarised in Table 1.

We assume that intact learning systems (in green, in Table 1) will be used where possible to compensate for impaired systems (shown in red). On this assumption, and consistent with points made in previous sections of this paper, we argue that:

- In people with HF-ASD there is a within-declarative memory see-saw effect in which intact semantic memory compensates for impaired episodic memory.
- Also in people with HF-ASD, non-declarative/implicit learning is, overall, superior to declarative/explicit learning, manifesting in a secondary see-saw effect in which non-declarative learning is better practiced – and the products of this learning can be accessed with greater facility – than the products of declarative learning.
- In people with LF-ASD there is a see-saw effect in which non-declarative/implicit learning and knowledge is heavily relied on for all kinds of learning and behaviour to compensate for an across-the-board impairment of declarative/explicit learning and knowledge. The see-saw effect will be more marked in those with severe deficits in semantic memory additional to impaired episodic memory, than in those individuals with mild or moderate semantic learning impairments.

The autism-related behaviours that Ullman and Pullman seek to explain in terms of their formulation of a see-saw effect can be explained in terms of our reformulation, as follows:

(i) Excessive reliance on formulaic language in people with HF-ASD is consistent with an unusual degree of dependence on lexical-semantic/single-item learning in reaction to limited input from episodic/relational learning. Formulaic, or ‘stock’ phrases, constitute single items and, as such, are easily acquired.

(ii) We agree with Ullman and Pullman that people with HF-ASD – also those individuals with LF-ASD whose semantic memory is only mildly or moderately impaired – may benefit from being taught culture-appropriate social routines using explicitly memorised rules, scripts and event schemas. However, as argued above, this is not because implicit learning is impaired, as claimed by Ullman and Pullman, but because one or other of the genetically hardwired social interaction propensities/capacities on which unconscious acquisition of social behaviours also depends are not available.

(iii) Nor is the use of explicit (conscious) problem solving to ‘hack out’ solutions to tests of theory of mind attributable to impaired implicit learning per se, but rather to genetically determined impairments of one or more of the social propensities and capacities underlying normal social learning. These propensities and capacities may constitute prerequisites for the acquisition of mentalising ability; however, the possibility that mentalising ability is itself a genetically determined/preprogrammed capacity remains open (for discussion see Frith, 2013; Happe & Frith, 2014).

(iv) Similarly, the use of consciously formulated rules to establish categories and concepts is not attributable to impaired implicit learning, but rather to the peculiarities of sensory-perceptual processing in ASD.

(v) Finally, Ullman and Pullman’s claim that therapeutic interventions rely on explicit teaching methods is something of a half-truth. We agree (see above) that explicit instruction may be used to teach higher-functioning autistic individuals ‘how to behave’ socially. However, many psychosocial and educational interventions commonly used with people with ASD rely wholly or in part on ‘learning by doing’. An obvious example is the ‘TEACCH’ method a central tenet of which is to establish patterns of behaviour by repetition, using context-specific visual cues to support verbal instruction. And learning by doing (using implicit learning mechanisms) is essential to interventions for those people with LF-ASD in whom there is a total or near total lack of declarative learning ability, and little language. To give just one example: functional use of the Picture Exchange Communication System (Bondy & Frost, 2001; Frost & Bondy, 2002, see review by Sulzer-Azaroff, Hoffman, Horton, Bondy & Frost, 2009) is taught by a gradual process of behaviour training in which language and explicit teaching have a negligible role.

Conclusion

We have argued that most of the views expressed by Walenski, Ullman et al. concerning anomalies within the learning systems underlying language abilities and other capacities in ASD are incomplete or untenable. We have gone into considerable detail in making this
argument, not least because Ullman, the senior member of this group, has a well-deserved reputation for his work on language acquisition and language disorder (especially as manifested in specific language impairment and the aphasias). His work is influential, and if his views on the nature and origins of language impairment in ASD (and on the nature of the ‘see-saw’ effect as manifested in ASD) are not countered, they may lead some researchers and also practitioners down blind alleys. This would be greatly regrettable because the combination of ASD plus clinically significant language impairment and learning disability is a multifaceted load of ‘difference’, with heavy costs – emotional, practical and financial – for affected individuals and their families, and lifetime financial costs for States (Boucher, 2017).

There is, unfortunately, lamentably little research into the clinically significant language and learning disabilities that so commonly co-occur with ASD. The lack of knowledge and understanding of these major ‘specifiers’ (American Psychiatric Association, 2013) not only retards the development of rationale-based, evidence-based interventions. It also retards research into the etiological origins of these specifiers, such as might eventually contribute to reducing their incidence in association with ASD.

In the present paper, we have not only offered an evidence-based critique of Ullman et al.’s theoretical papers and empirical studies of language in ASD. We have also outlined our own alternative model of links between language outcomes and the patterns of strength and weakness that characterise learning and memory across the spectrum. We are all too aware, nevertheless, that there will be a need for revision and re-revision, as and when more evidence becomes available.

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Notes
1. This widely used term is descriptive and has no officially agreed definition, but generally refers to individuals without clinically significant learning disability or language impairment.
2. This descriptive term is generally used to apply to individuals with ASD who have clinically significant learning and language impairments.
3. Notably some of the earlier candidates for early manifesting primary social impairments – for example social orienting impairment – are now discounted (see, e.g. Johnson (2014)).
4. We do not cover the role of working memory in the acquisition of language by people with HF-ASD. This is not because working memory does not have a role; but rather that it is not centrally relevant to the discussion of Ullman et al.’s findings, nor to discussion of any see-saw effect relevant to language processing in HF-ASD.
5. The fact that generalisation occurs implicitly is incidental: impaired generalisation is what almost certainly causes impaired acquisition of categories and concepts, not the fact that generalisation is an implicit process.
6. It is worth noting that Baron-Cohen et al.’s claims that systemising is a peak ability in ASD supports our claim of unimpaired procedural learning.

7. A literature search shows that between 2000 and 2017, over 7500 papers with either ‘High-functioning autism/autistic/ASD’ or ‘Asperger(s)’ in the title were published compared with 174 papers with any of the terms ‘autism/langua ge impaired/language impaired’ plus any of the qualifying terms or phrases ‘low(er)-functioning/retarded/language impaired/language impairment/learning impaired/learning impairment/learning disabled/language impaired’ in their titles.

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