The role of declarative and procedural memory in disorders of language

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Language is often assumed to rely on domain-specific neurocognitive substrates. However, this human capacity in fact seems to crucially depend on general-purpose memory systems in the brain. Evidence suggests that lexical memory relies heavily on declarative memory, which is specialized for arbitrary associations and is rooted in temporal lobe structures. The mental grammar instead relies largely on procedural memory, a system that underlies rules and sequences, and is rooted in frontal/basal-ganglia structures. Developmental and adult-onset disorders such as Specific Language Impairment, autism, Tourette syndrome, Parkinson's disease, Huntington's disease, and non-fluent aphasia each seem to involve particular grammatical deficits and analogous non-linguistic procedural memory impairments, as well as abnormalities of procedural memory brain structures. Lexical and declarative memory remain relatively intact in these disorders, and may play compensatory roles. In contrast, Alzheimer's disease, semantic dementia, fluent aphasia and amnesia each affect lexical and declarative memory, and involve abnormalities of declarative memory brain structures, while leaving grammar and procedural memory largely intact. Overall, the evidence suggests that declarative and procedural memory play critical roles in language disorders, as well as in language more generally.

Language is often claimed or assumed to rely on dedicated neural, psychological, and computational – i.e. neurocognitive – substrates (Chomsky 1995; Fodor 1983; Grodzinsky 2000; van der Lely 2005). However, evidence that directly supports such domain-specificity is lacking (Ullman, Lum & Conti-Ramsden 2014). Rather, increasing evidence suggests that language depends importantly on memory systems that also subserve a range of nonlanguage functions, and are moreover found in animals as well as humans (Ullman 2004, Under Review). Here we focus on two long-term brain memory systems, declarative and procedural memory, and explore their relations to language in a range of developmental and adult-onset disorders. Note that this paper is a modified and updated version of a previously published chapter (Ullman 2008), reprinted here with permission.

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1. The declarative and procedural memory systems

Evidence suggests the existence of multiple memory systems in the brain, including declarative and procedural memory (Ashby et al. 2010; Cabeza & Moscovitch 2013; Doyon et al. 2009; Eichenbaum 2012; Eichenbaum et al. 2012; Henke 2010; Squire & Wixted 2011; Ullman 2004, Under Review).

The *declarative memory system* subserves the learning, representation, and use of knowledge about facts ("semantic knowledge") and personally-experienced events ("episodic knowledge"), such as the fact that the permafrost in Alaska is melting, or that you ate fusilli with chili for dinner last night (for reviews, see Cabeza & Moscovitch 2013; Eichenbaum 2012; Eichenbaum et al. 2012; Henke 2010; Squire & Wixted 2011; Ullman 2004, Under Review). The system seems to be specialized for learning arbitrary pieces of information and the associations between them – indeed, declarative memory might be necessary for learning such knowledge. Knowledge is learned rapidly, with as little as a single exposure being necessary for learning. The learned information can be generalized and used flexibly across different contexts. The acquired knowledge is at least partly, but not completely, explicit – that is, available to conscious awareness.

The anatomical substrates of declarative memory have been well investigated in both humans and animals. The system is based on a network of brain structures that play complementary functional roles. The hippocampus and nearby medial temporal lobe structures are critical for learning and consolidating new memories. Over the course of years memories become largely independent of these structures and rely instead mainly on neocortical regions, especially but not only in the temporal lobes. Different neocortical regions subserve different kinds of knowledge. Declarative memory is closely related to the "ventral stream", which may feed visual and auditory representations into this long-term memory system (Ullman 2004, Under Review). Other brain structures also play roles in declarative memory. Specific portions of inferior frontal cortex, corresponding largely to Brodman's areas (BA) 45 and 47, as well as parts of the basal ganglia (presumably projecting to these frontal regions, and apparently distinct from those portions of the basal ganglia that underlie procedural memory; Ullman 2006b) subserve the selection or retrieval of declarative memories, while parts of the right cerebellum have been implicated in searching for this knowledge (Buckner & Wheeler 2001; Desmond & Fiez 1998). The molecular bases of declarative memory have also been investigated. For example, the gene for brain-derived neurotrophic factor (BDNF) plays an important role in declarative memory and hippocampal function, as does the neurotransmitter acetylcholine. The declarative memory system is also affected by estrogen, perhaps via the modulation of acetylcholine and/or BDNF. Estrogen

improves declarative memory in women and men, and strengthens the cellular and molecular correlates of long-term hippocampal learning.

The *procedural memory system* underlies the learning of new and the processing of established perceptual-motor and cognitive "skills" and "habits". The system may be specialized for learning rules and sequences. Learning requires repeated exposure to stimuli, or practice with the skill or habit. Neither the learning nor the retrieval of the skills or knowledge are accessible to conscious memory. Thus the system is referred to as an "implicit memory" system. Note that the term "procedural memory" is used here to refer only to one type of implicit "non-declarative" memory system, not to all such systems (some researchers have used the term "procedural memory" interchangeably with "implicit memory"). Note also that the declarative and procedural memory systems refer here to the entire neurocognitive systems involved in the learning, representation, retention and use of the relevant knowledge and skills, not just to the neural substrates underlying learning or consolidation, which is what some memory researchers refer to when they discuss the two systems.

Although the neurobiological bases of procedural memory are less well understood than those of declarative memory, a fair bit of progress has been made in elucidating the neural substrates of this system (for reviews, see Ashby et al. 2010; Doyon et al. 2009; Poldrack & Packard 2003; Ullman 2004, Under Review). Like declarative memory, this system is composed of a network of interconnected brain structures. The network is rooted in frontal/basal-ganglia circuits, including premotor regions and BA 44 within frontal cortex, and the caudate nucleus within the basal ganglia. The network may also include portions of inferior parietal cortex, superior temporal cortex, and the cerebellum. The mirror neuron system, which includes BA 44 and inferior parietal cortex, and underlies the execution and observation of well-learned motor skills, may be considered part of the procedural memory system (Ullman 2004). Procedural memory also seems to be closely related to the "dorsal stream", which has been implicated in perceptual-motor integration (Ullman 2004, Under Review). Different brain structures within the system appear to play different functional roles. For example, the basal ganglia (especially the caudate nucleus) may be important for the acquisition of new procedures, whereas frontal regions are more important for the computation or processing of those procedures once they have been learned and automatized. Note that within the frontal/basal-ganglia circuits that cut across these structures, parallel channels play analogous computational roles in different domains. For example, motor portions of the basal ganglia project (via the thalamus) to frontal motor cortex, whereas other portions of the basal ganglia play other functional roles and project to other frontal regions. Thus not all portions of frontal cortex or the basal

ganglia are expected to subserve the same domains within procedural memory, or even procedural memory at all, even though they may carry out similar computational roles (Ullman 2004; Ullman & Pierpont 2005; Ullman, Pullman, Lovelett, McQuaid, Pierpont & Turkeltaub Under Review). Finally, the neurotransmitter dopamine plays a particularly important role in aspects of procedural learning.

The declarative and procedural memory systems interact, yielding both cooperative and competitive learning and processing (Poldrack & Packard 2003; Ullman 2004, Under Review). First, the systems can complement each other in acquiring the same or analogous knowledge. The declarative memory system may acquire knowledge initially, including knowledge of sequences and rules, thanks to its rapid acquisition abilities, while the procedural system gradually learns analogous knowledge. Thus, at least to some extent, declarative and procedural memory can play *redundant* functional roles (Ullman 2004, 2007, Under Review). Second, animal and human studies indicate that the systems also interact competitively. This constitutes a "see-saw effect", such that a dysfunction of one system can enhance learning in the other, or that learning in one system may depress the functionality of the other (Ullman 2004, 2007, Under Review).

2. Language and the declarative and procedural memory systems

Although the declarative and procedural memory systems have not traditionally been thought of as underlying language, there is no *a priori* reason that they should not subserve aspects of language as well as other cognitive domains. Indeed, if the functions they subserve in nonlanguage domains share characteristics with language, it seems reasonable that the systems would play analogous roles across language and nonlanguage domains, whether these language functions developed phylogenetically (evolutionarily) and/or ontogenetically (developmentally, within an individual) within the memory systems (Ullman 2004, Under Review).

This perspective has led to the Declarative/Procedural (DP) theory, which posits that the two memory systems play critical roles in the learning, representation, and processing of language, and that these roles should be largely analogous to the roles they play in non-language domains. It is important to point out that the claim is *not* that language is subserved by "nonlanguage systems", but rather that the memory systems underlie language as well as nonlanguage functions, and that they subserve these different domains in a similar fashion. Because these two memory systems are quite well studied in animals and humans, including at functional, computational, anatomical, physiological, endocrine, biochemical, and genetic levels (e.g. see above), we can make a wide range of specific predictions about the neurocognition of language that might be unwarranted to make based

on the more circumscribed study of language alone. Thus it is a very powerful approach. Following are some of the key predictions that we will explore in developmental and adult-onset disorders (for more detail on the memory systems and their roles in language, see Ullman 2004, Under Review).

First, given the apparent importance of declarative memory for arbitrary relations, this system should be a critical memory store for such relations in language. Thus declarative memory should underlie what is traditionally thought of as the mental lexicon: all non-derivable word-specific linguistic knowledge, including in simple words (e.g. the sound pattern /kæt/ being associated with the furry pet), irregular morphology (e.g. that *dig* takes *dug* as its irregular past-tense form, however this knowledge is represented), and syntax (e.g. that *devour* requires a direct object).

As with other knowledge learned in declarative memory, linguistic knowledge should be rapidly learnable. Much but not all of this knowledge is expected to be explicit. The biological substrates of the learning, representation and use of the knowledge can also be predicted. For example, the hippocampus and other medial temporal lobe structures should underlie the learning and consolidation of the knowledge, which should eventually depend largely on neocortex, with different necortical regions, particularly in the temporal lobes, responsible for different types of knowledge (Ullman 2007, Under Review). Aspects of the learning or processing of the knowledge should be modulated by factors such as the hormone estrogen, the neurotransmitter acetylcholine, and the protein BDNF, to the extent that these play roles in this system in other domains. Inferior frontal cortex, in particular the region of BA 45/47, is expected to underlie lexical retrieval.

In contrast, the procedural memory system should subserve the gradual implicit learning of knowledge that underlies what is often thought of as the mental grammar – that is, the knowledge subserving the rule-governed sequential and hierarchical combination of complex linguistic representations. The system may be expected to subserve rule-governed knowledge and computations across linguistic domains, including in syntax, morphology (e.g. in regularly inflected forms) and phonology (e.g. in novel word forms, whose phonological elements must somehow be combined according to the phonotactics of the language). Portions of frontal/basal-ganglia circuits, including BA 44 and the caudate nucleus, should be critical in these linguistic functions. The caudate nucleus is predicted to play a crucial role in acquiring the knowledge, which should be modulated by dopamine, while BA 44 and premotor cortex may be more important in the computation or processing of that knowledge once it is learned. Given the existence of parallel functionally segregated frontal/basal-ganglia channels, there is no reason to assume that all grammatical domains or functions should depend on the same channels, or that language-subserving channels necessarily also underlie

nonlanguage functions. Rather it is an empirical question as to which segregated (sub)channels subserve which linguistic and/or nonlinguistic functions (Ullman 2004, 2006b), and whether there may be domain specific circuitry for language within the broader system.

The two memory systems are predicted to interact both cooperatively and competitively. Complex linguistic representations are expected not only to be computed by procedural memory, but also to be learned and stored in declarative memory. They may depend on declarative memory in various ways. For example, they may be memorized as chunks, generalized associatively across already-stored representations, or processed on the basis of explicit (or implicit) rules learned in declarative memory (Hartshorne & Ullman 2006; Ullman 2004; Ullman 2006a; Ullman Under Review). The likelihood of a grammatical reliance on declarative memory should depend on the various factors that affect learning or processing information in this system, including item-related variables such as the frequency of complex forms (higher frequency forms are more likely to be stored as chunks), and subject-related differences in the functionality of the system due to factors such as sex, estrogen levels, and genetic variability (Prado & Ullman 2009; Ullman 2004, Under Review; Ullman, Miranda, & Travers 2008). Additionally, the dysfunction of procedural memory should encourage a compensatory reliance on declarative memory. Finally, learning in one system may inhibit learning analogous knowledge in the other, while a dysfunction in one system may enhance the other, potentially facilitating a compensatory role for declarative memory in the use of complex linguistic representations following a dysfunction of the procedural memory system.

These and other predictions regarding the relations between the two memory systems on the one hand, and language on the other, have been investigated using a wide range of methods, in developmental, psycholinguistic, neurological, electrophysiological and neuroimaging studies (Ullman 2004, 2007, Under Review; Ullman et al. 1997). Across methodologies, the basic approach for testing the predictions laid out above has been to examine whether the expected language and nonlanguage functions both depend on one or the other memory system, and that they do so in a similar manner. For example, tasks involving lexical and nonlinguistic conceptual/semantic stimuli should elicit analogous fMRI activation patterns and ERP components, and should be similarly modulated by estrogen or BDNF.

Here we focus on evidence related to developmental and adult-onset disorders. As we will see, the evidence suggests the following. Disorders known to affect grammar seem to similarly affect non-linguistic functions of procedural memory, and involve the dysfunction of the neural substrates of this system. Conversely, disorders which involve abnormalities of procedural memory are associated with

analogous grammatical abnormalities. Thus impairments of grammar and procedural memory appear to co-occur, independently of whether the underlying disorder is traditionally thought of as affecting language or non-linguistic domains. Moreover, these disorders often leave lexical and declarative memory relatively intact. Indeed, in some disorders lexical/declarative memory seems to play a compensatory role for grammatical functions. In contrast, disorders generally thought of as affecting lexical memory similarly affect declarative memory, and vice versa. These disorders often leave grammar and procedural memory and their neural underpinnings largely intact. Thus across a number of disorders one finds double dissociations between declarative and procedural memory across both linguistic and non-linguistic functions.

3. Disorders of grammar and procedural memory

3.1 Developmental disorders

A number of developmental disorders seem to be associated with (different sorts of) procedural memory system dysfunctions and grammatical abnormalities, accompanied by relatively spared lexical and declarative memory. These include Specific Language Impairment, autism, Tourette syndrome, dyslexia, and Attention Deficit Hyperactivity Disorder (Lum, Conti-Ramsden, Morgan & Ullman 2014; Lum, Ullman & Conti-Ramsden 2013; Ullman, 2004; Ullman & Pierpont 2005; Ullman et al. Under Review; Ullman & Pullman Under Review; Walenski, Mostofsky & Ullman 2007, Under Review; Walenski, Tager-Flusberg & Ullman 2006). Here we focus on Specific Language Impairment, autism, and Tourette syndrome.

3.1.1 *Specific Language Impairment (SLI)*

SLI is usually defined as a developmental disorder of language that occurs in the absence of frank neurological damage, hearing deficits, severe environmental deprivation, and mental retardation (Leonard 1998). The disorder has generally been explained either as an impairment specific to grammar (Clahsen 1989; Rice, Wexler & Cleave 1995; van der Lely 2005) or as a processing deficit, for example of working memory or of briefly presented stimuli and rapidly presented sequences (Gathercole & Baddeley 1993; Leonard 1998; Merzenich, Schreiner, Jenkins & Wang 1993; Tallal & Piercy 1978). However, both classes of theoretical accounts have trouble explaining the pattern of language and non-language deficits, and the heterogeneity across individuals with the disorder (Ullman & Pierpont 2005). Moreover, these explanatory accounts have addressed SLI at a functional level.

However, SLI is clearly rooted in the brain, so a neurobiological might have more explanatory power.

According to the Procedural Deficit Hypothesis (PDH), SLI may be largely explained by abnormalities of procedural memory system brain structures, in particular of Broca's area (BA 44 and 45) within frontal cortex and the caudate nucleus within the basal ganglia (Ullman & Pierpont 2005; Ullman et al. Under Review). Thus the PDH is a neurobiological account, specifically positing that SLI can be best accounted for in terms of the pattern of neuroanatomical abnormalities, at least to some extent independent of genetic or environmental etiology (Ullman & Pierpont 2005; Ullman et al. Under Review).

Several lines of evidence support the PDH. First, frontal and basal ganglia abnormalities, specifically of Broca's region and the caudate nucleus, are consistently found in SLI and other developmental language disorders with similar phenotypes, such as disorders of the *FOXP2* gene (Ullman & Pierpont 2005). Indeed, a recent neuroanatomical meta-analysis revealed that these were the only structures to show consistent abnormalities in SLI (Ullman et al. Under Review). Second, the pattern of both language and non-language deficits is consistent with such abnormalities. Grammatical impairments are typical of the disorder – not only deficits of receptive and expressive syntax, but also of morphology and phonology. The procedural system dysfunction also clearly extends beyond language. Motor deficits are widely observed in children and adults with SLI. Individuals with SLI have particular difficulty on motor tasks involving complex sequences of movements, such as moving pegs, sequential finger opposition and stringing beads. Recent evidence has also revealed that SLI is associated with deficits of procedural memory itself, that is, learning and retention in this system (Hedenius et al. 2011; Lum, Conti-Ramsden, Page & Ullman 2012; Lum et al. 2014). Finally, the disorder may also be associated with deficits of other functions that depend on the brain structures underlying procedural memory, such as working memory, processing rapidly-presented sequences, and mental rotation (Leonard 1998; Ullman & Pierpont 2005).

In contrast, lexical and declarative memory are relatively spared in SLI, as evidenced by relatively intact word recognition and comprehension, word learning, lexical/semantic organization, and learning in declarative memory (Ullman & Pierpont 2005; Ullman & Pullman Under Review). Temporal lobe structures, including the medial temporal lobe, also seem to remain intact (Ullman et al. Under Review). However, the *retrieval* of lexical knowledge (word finding) is difficult for individuals with SLI (Rapin & Wilson 1978; Weckerly, Wulfeck & Reilly 2001), as might be expected if the frontal and basal ganglia structures underlying retrieval (e.g. BA 45) are dysfunctional.

Children and adults with SLI compensate for their deficit with lexical and declarative memory (Lum et al. 2012; Ullman & Pierpont 2005; Ullman & Pullman

Under Review). For example, unlike typically developing control subjects, individuals with SLI show consistent "frequency effects" on regularly inflected past-tense and plural forms – that is, correlations between the frequency of these forms and performance at producing them. This suggests that individuals with SLI, unlike typically developing subjects, generally retrieve regular past-tense forms from memory rather than combining them in the procedural memory-based mental grammar. Additionally, these individuals sometimes learn explicit grammar rules, such as "add -ed to make a past tense form". For example, one child reported that "at school, learn it at school. In the past tense put -e-d on it. If it's today it's -i-n-g. Like swimming: 'I went swimming today' and 'Yesterday I swammed'" (Ullman & Gopnik 1999).

It is important to emphasize that the PDH does *not* claim that all individuals identified as SLI are afflicted with a dysfunction of the procedural memory system (Ullman & Pierpont 2005). Given the broad definition of SLI that would clearly be too strong a claim. Nevertheless, it is predicted that many if not most individuals diagnosed with SLI will show abnormalities of brain structures underlying procedural memory, especially of the caudate nucleus and Broca's area – a prediction that is consistent with the results of the neuroanatomical meta-analysis of SLI discussed above. Moreover, much of the heterogeneity in the disorder may be explained by the particular combination of frontal/basal-ganglia channels or other procedural system structures that are affected (with the likelihood of SLI presumably being higher with greater or multiple dysfunctions; e.g. see Bishop 2006), as well as by the compensatory abilities of other systems, in particular of declarative memory (Ullman & Pullman Under Review).

3.1.2 *Autism*

Autism, also referred to as Autism Spectrum Disorder (ASD), is a developmental disorder associated with deficits of language and communication, as well as of social interaction, motor function and certain other domains. Roughly 20% of children with autism are essentially non-verbal, using fewer than five words per day (Lord, Risi, & Pickles 2004). Others acquire functional language to varying degrees, although the exact profile of language and communicative abilities is heterogeneous. Most research on language deficits in autism has focused on the pragmatic difficulties found in the disorder – that is, impairments in using and interpreting language appropriately for the social and real-world contexts in which utterances are made (Tager-Flusberg 2000).

However, evidence suggests that autism may also be associated with abnormalities of grammar, as well as non-linguistic functions that depend on the procedural memory system (for reviews, see Ullman 2004; Walenski et al. Under Review; Walenski et al. 2006). High-functioning individuals with autism may

show syntactic abnormalities in both receptive and expressive language. Inflectional morphology, and regular inflection in particular, has been found to be abnormal in both elicited and spontaneous speech. Whereas deficits are generally not observed in the processing of individual phonemes, impairments are often reported in processing sound combinations in nonwords. Both the acquisition and processing of both motor and non-motor sequences have been reported to be abnormal. Complex sequences seem especially problematic. Impairments of other functions that depend on the brain structures of the procedural memory system, such as rapid temporal processing and working memory, have also been observed. Although studies of the neurobiology of procedural and declarative memory brain structures in autism have produced a number of inconsistent results, some patterns are beginning to emerge. Of particular interest here, abnormalities of left frontal cortex, especially Broca's area, seem to be consistently found in studies that have examined this region. Abnormalities of cerebellar and as well as basal ganglia structures have also been observed (Amaral, Schumann & Nordahl 2008).

In contrast, lexical and conceptual knowledge appear to remain relatively intact in high-functioning autistics (Ullman & Pullman Under Review). In fact, object naming has even been found to be enhanced in ASD children, as compared to typically-developing children (Walenski, Mostofsky, Gidley-Larson & Ullman 2008) – consistent with the view that autism may be viewed not as a cluster of deficits, but rather a set of relative strengths and weaknesses across various domains (Bertone, Mottron, Jelenic & Faubert 2005; Frith & Happé 1994; Happe 1999). Additionally, tasks probing learning in declarative memory suggest normal rote learning of individual items such telephone numbers, as well as intact associative learning, such as remembering word pairs (Ullman & Pullman Under Review). However, learning personally experienced episodes seems to be impaired, perhaps due in part to the particular dependence of episodic memory on frontal structures.

Individuals with ASD may compensate for their grammatical deficits with lexical and declarative memory (Ullman & Pullman Under Review; Walenski et al. 2006). For example, children with autism rely much more than typically-developing children on "formulaic speech" (Lord & Paul 1997), that is, speech with prefabricated sequences of words that appear to be stored whole in memory (Wray & Perkins 2000). For example, ASD speech is marked by stereotyped utterances such as *thank you* or *you're welcome*. And some fMRI evidence suggests a compensatory increase in reliance on medial temporal lobe structures in autism (Dichter, Richey, Rittenberg, Sabatino & Bodfish 2012; Vaidya et al. 2011).

3.1.3 *Tourette syndrome*

Tourette syndrome is a developmental disorder characterized by the presence of verbal and motor tics, which are both fast and involuntary. The tics may be

explained by abnormal dopamine levels and structural abnormalities of the basal-ganglia, in particular of the caudate nucleus, resulting in decreased inhibition of frontal activity, a hyperkinetic behavioral profile, and an inability to suppress the tics (Albin & Mink 2006).

Given this frontal/basal-ganglia dysfunction, it is perhaps not surprising that procedural memory and related functions have also been reported to be abnormal in the disorder (Walenski et al. 2007). For example, the acquisition of implicit probabilistic rules (in the "weather prediction task"), which depends at least in part on the caudate nucleus, has been found to be impaired in Tourette syndrome (Marsh et al. 2004). However, deficits have not always been found in all tasks traditionally used to probe procedural learning, such as the serial reaction time task (Channon, Pratt & Robertson 2003), perhaps because of compensatory learning in declarative memory (Schendan, Searl, Melrose & Stern 2003; Ullman & Pullman Under Review).

Indeed, lexical and declarative memory are largely spared in Tourette syndrome (Ullman & Pullman Under Review; Walenski et al. 2007). For example, children with the syndrome have shown normal performance at both picture naming and stem completion, suggesting that lexical representations remain intact. Acquiring new information in declarative memory, such as list learning and remembering the location of objects, also seems unproblematic (Marsh et al. 2004). And whereas the implicit learning of procedural knowledge in the weather prediction task was found to be impaired in Tourette syndrome, normal performance was observed in a separate test of explicit knowledge in the same subjects (Marsh et al. 2004).

One study examined grammatical and lexical processing in a past-tense production task of regular and irregular forms, as well as the processing of previously-learned procedural and declarative knowledge in a picture naming task of objects that either involve procedural motor-skill knowledge (tools and other manipulated objects; e.g. *hammer*) or do not involve such knowledge (e.g. *elephant*) (Walenski et al. 2007). Children with Tourette syndrome were significantly faster than typically-developing control children at producing rule-governed past-tenses (*slip-slipped, plim-plimmed*) but not irregular and other unpredictable forms (*bring-brought, splim-splam*) thought to depend on lexical memory. They were also faster than controls at naming pictures of manipulated (*hammer*) but not non-manipulated (*elephant*) items. These data were not explained by a wide range of potentially confounding subject- and item-level factors, such as the age and IQ of the subjects, and the frequency and phonological complexity of the items. The results suggest that the processing of procedurally-based knowledge, both of grammar and of manipulated objects, is particularly *speeded* in Tourette syndrome. Thus the frontal/basal-ganglia abnormalities in the disorder may lead not

only to tics, but to a wider range of abnormally rapid behaviors, including in the processing of procedural knowledge both in the naming of manipulated objects and the production of rule-governed forms in language.

3.2 Adult-onset disorders

Various neurodegenerative and other adult-onset disorders affect grammar, non-linguistic aspects of procedural memory, and the neural substrates of this system. The different disorders are associated with damage to different portions of procedural memory system brain structures. This neuroanatomical variability may help explain the different types of dysfunctions, each of which seems to show similarities across language and non-language domains.

3.2.1 *Parkinson's disease*

Parkinson's disease (PD) is associated with the degeneration of dopamine-producing neurons, particularly in the basal ganglia (substantia nigra). This degeneration, which results in high levels of inhibition in the motor and other frontal cortical areas to which the basal ganglia project, is thought to explain why PD patients show suppression of motor activity (hypokinesia) and have difficulty expressing motor sequences (Jankovic & Tolosa 2007).

The degeneration has also been implicated in PD patients' impairments at procedural learning in a number of perceptual-motor and cognitive tasks, such as sequence learning in the serial reaction time task and probabilistic rule learning in the weather prediction task (Clark, Lum & Ullman In Press; Knowlton, Mangels & Squire 1996; Westwater, McDowall, Siegert, Mossman & Abernethy 1998). In contrast, temporal-lobe regions remain largely intact and lexical and declarative memory relatively spared in low- and non-demented PD patients (Knowlton et al. 1996; Ullman et al. 1997), although these patients often have word finding difficulties, consistent with a role for frontal/basal-ganglia circuits in retrieval.

Grammatical deficits are also found in Parkinson's disease. Even non-demented PD patients show impairments at both expressive and receptive syntax (Grossman et al. 2000; Ullman 2004). Non-demented severely hypokinetic PD patients have also shown impairments at the production of *-ed*-affixed past-tense forms (e.g. *walked*, *plagged*), relative to irregulars (Ullman et al. 1997). A similar though weaker pattern may be found in patients with lower levels of hypokinesia (Longworth, Keenan, Barker, Marslen-Wilson & Tyler 2005). However, it remains unclear whether the various grammatical deficits observed in PD can be attributed directly to the basal ganglia (perhaps even due to problems with presumably non-procedural functions, such as syntactic integration; Friederici, Kotz, Werheid, Hein & von Cramon 2003), or whether they can instead or additionally

be explained by excessive inhibition from the basal ganglia to frontal cortex, which seems to be responsible for rule-governed computation (Ullman 2006b).

3.2.2 *Huntington's disease*

Like Parkinson's disease, Huntington's disease (HD) results in early basal ganglia degeneration, in this case particularly in the caudate nucleus, with further progression to cortical regions as well. However, HD affects different basal ganglia circuits than PD, resulting in the disinhibition rather than the inhibition of frontal areas receiving basal ganglia projections (Jankovic & Tolosa 2007). This is thought to explain the unsuppressible movements – chorea, a type of hyperkinesia – found in patients with HD. HD patients have also been reported to show procedural learning deficits, as well as impairments in both expressive and receptive syntax (Murray 2000; Willingham, Koroshetz & Peterson 1996).

HD patients have been found to show the opposite pattern of PD patients not only in the type of movement impairment (the suppressed movements of hypokinesia vs. the unsuppressed movements of hyperkinesia), but also in the type of errors on *-ed*-suffixed forms. Ullman et al. (1997) observed that, unlike normal control subjects, HD patients produced many forms like *walkeded, plaggeded*, *dugged*, and *digged*. The patients did not produce analogous errors on irregulars like *dugug* or *keptet,* suggesting that the affixed error forms are not explained by articulatory deficits. Rather the data suggest unsuppressed *-ed*-suffixation. This conclusion is strengthened by the finding that the production rate of these over-suffixed forms correlated with the degree of chorea, across patients. Another study, which also found increased rates of over-regularization in HD patients (Longworth et al. 2005), did not examine the correlation between chorea and the production of these forms.

The contrasting patterns in PD and HD, linking movement and *-ed*-suffixation in two distinct types of impairments related to two types of basal ganglia damage, implicate frontal/basal-ganglia circuits in *-ed*-suffixation. They support the hypothesis that these circuits underlie the processing of grammatical rules as well as movement, and suggest that they play similar roles in the two domains. Moreover, given that disinhibition of frontal activity is implicated in the unsuppressed movements of HD, such disinhibition also seems likely to account for the unsuppressed affixation also observed in the disorder.

3.2.3 *Non-fluent aphasia*

The term "aphasia" generally refers to language impairments resulting from one or more focal lesions in the brain. Clusters of symptoms tend to co-occur in types (syndromes) of aphasia. Although there are a number of different adult-onset aphasia syndromes, several of these can be grouped into two larger categories,

which are often referred to as non-fluent and fluent aphasia (Alexander 1997; Feinberg & Farah 1997).

Non-fluent aphasia seems to reflect, at least in part, damage to brain structures of the procedural memory system (Ullman 2004). It is associated with lesions of left inferior frontal regions, in particular Broca's area and nearby cortex, as well as of the basal ganglia, inferior parietal cortex, and anterior superior temporal cortex (Alexander 1997; Feinberg & Farah 1997). It is also associated with agrammatism, in both expressive and receptive language. Agrammatic speech is characterized by abnormalities in the use of free and bound grammatical morphemes such as auxiliaries, determiners, and affixes. In receptive language patients have particular problems using the syntactic structure of sentences to understand their meaning. Non-fluent aphasics have also been found to have greater difficulty with regular than irregular morphology in both expressive and receptive language tasks, even holding constant word frequency, word length, and various other factors (Ullman et al. 1997; Ullman et al. 2005).

Non-fluent aphasia is also associated with impairments of non-linguistic functions that depend on the procedural memory system. These aphasics typically have a range of motor impairments, from articulation to the execution of complex learned motor skills, particularly those involving sequences (ideomotor apraxia) (Alexander 1997; De Renzi 1989; Feinberg & Farah 1997). Interestingly, they have also been found be impaired at learning new sequences, in particular sequences containing abstract structure (Dominey, Hoen, Blanc, & Lelekov-Boissard 2003; Goschke, Friederici, Kotz & van Kampen 2001). However, because the patients in these studies may also have had basal ganglia lesions, it is premature to conclude that the frontal regions alone are critical for learning sequences, even sequences containing abstract structure.

In contrast, non-fluent aphasics are relatively spared in their recognition and comprehension of content words, such as nouns and adjectives. Nevertheless, as would be expected with damage to Broca's area and the basal ganglia, they generally have lexical retrieval (word finding) difficulties. Interestingly, evidence also suggests that non-fluent aphasics can compensate for their grammatical impairments by memorizing complex forms in lexical memory, as evidenced by frequency effects for regular past-tense forms and other evidence (Drury & Ullman 2002; Ullman & Pullman Under Review).

4. Disorders of lexical and declarative memory

Several adult-onset disorders affect lexical and declarative memory, leaving grammar and procedural memory largely intact. Importantly, the particular type of

lexical and declarative memory impairments varies across the disorders, depending on the underlying neuropathology. Whereas disorders with neocortical temporal lobe lesions seem to particularly affect established (previously learned) lexical and conceptual-semantic knowledge (in Alzheimer's disease, semantic dementia, fluent aphasia), those with severe medial temporal damage instead or additionally show impaired learning of new lexical and non-linguistic declarative knowledge (in Alzheimer's disease and anterograde amnesia).

4.1 Alzheimer's disease

Alzheimer's disease (AD) particularly affects medial as well as neocortical temporal-lobe structures, leaving the basal ganglia and portions of frontal cortex, especially Broca's area and motor regions, relatively intact (Arnold, Hyman, Flory, Damasio & Van Hoesen 1991; Boller & Duyckaerts 1997; Feinberg & Farah 1997). Consistent with this degeneration, both the learning of new and the use of established lexical and conceptual-semantic knowledge is impaired in AD. In contrast, AD patients are relatively spared at acquiring and processing motor and cognitive skills, and at both expressive and receptive syntax (Ullman 2004). Additionally, patients with severe deficits at object naming or fact retrieval make more errors at producing irregular than *-ed*-affixed past-tense forms (Cortese, Balota, Sergent-Marshall, Buckner & Gold 2006; Ullman et al. 1997). A similar pattern has been found in Italian (Walenski, Sosta, Cappa & Ullman 2009). And across AD patients, error rates at object naming and fact retrieval correlate with error rates at producing irregular but not *-ed*-affixed past-tenses (Ullman et al. 1997). Overall, the data suggest that patients with AD have impairments both learning new and accessing established knowledge, both in linguistic and non-linguistic domains.

4.2 Semantic dementia

Semantic dementia is associated with progressive neurodegeneration, especially of inferior and lateral temporal lobe regions, and to a lesser extent medial temporal lobe structures, leaving inferior frontal, premotor, and basal ganglia structures largely intact (Grossman & Ash 2004; Mummery et al. 2000). The disorder results in impairments using established lexical and non-linguistic conceptual knowledge, but with relatively spared motor, syntactic and phonological abilities. These patients do not seem to have particular difficulty acquiring new knowledge in declarative memory, consistent with a relative sparing of medial temporal structures (Graham & Hodges 1997). However, like AD patients, and consistent with their deficits in established lexical/semantic knowledge, semantic dementia patients have more trouble producing and recognizing irregular than *-ed*-suffixed past-tenses; moreover, the degree of their impairment on irregulars has been

found to correlate with their performance on lexical memory tasks (Cortese et al. 2006; Patterson, Lambon Ralph, Hodges & McClelland 2001).

4.3 Fluent aphasia

Fluent aphasia is at least partly associated with damage to declarative memory brain structures, in particular left temporal and temporo-parietal neocortical regions, though the lesions often extend further into inferior parietal structures. Fluent aphasics have impairments in the production, reading, and recognition of the sounds and meanings of content words, as well as of conceptual knowledge (Alexander 1997; Feinberg & Farah 1997; Ullman 2004). In contrast, these aphasics tend to produce syntactically well-structured sentences, and to not omit morphological affixes. However, damage in and around inferior parietal cortex in fluent aphasia can lead to grammatical impairments, consistent with a role for this region in the mental grammar and procedural memory. In direct comparisons of regular and irregular morphology, fluent aphasics have been found to show the opposite pattern to that of non-fluent aphasics, with worse performance at irregular than regular forms (Ullman et al. 1997; Ullman et al. 2005).

4.4 Anterograde amnesia

Lesions of medial temporal lobe structures can lead to an inability to learn new information about facts, events, and words (Eichenbaum 2012; Squire & Wixted 2011). Neither phonological nor semantic lexical knowledge is acquired following such damage, supporting the hypothesis that these structures underlie the learning of word forms as well as word meanings (Gabrieli, Cohen & Corkin 1988; Postle & Corkin 1998; Ullman 2004, Under Review). This "anterograde amnesia" is typically accompanied by the loss of information for a period preceding the damage – that is temporally graded retrograde amnesia. However, knowledge acquired substantially before lesion onset is largely spared. Thus even though medial temporal lobe structures seem to underlie the learning of new lexical information, adult-onset amnesics should remember words learned during childhood. As expected, the well-studied amnesic H.M. does not seem to be particularly impaired at syntactic processing, or at the production of regular or irregular forms in past-tense, plural and derivational morphology (Kensinger, Ullman & Corkin 2001; Ullman 2004, Under Review).

5. Summary, challenges, and future directions

In sum, evidence from both developmental and adult-onset disorders suggests that the declarative and procedural memory systems play important roles in language, and that disorders of these systems similarly affect language and non-language

functions. However, many open questions remain (Ullman Under Review). First, some existing evidence is inconsistent. For example, procedural learning deficits have been found in some but not other studies in autism, Tourette syndrome, Parkinson's disease, and Huntington's disease. These inconsistent findings are not yet understood. Meta-analyses and meta-regressions may shed light on such variability (Clark et al. In Press; Lum et al. 2014; Lum et al. 2013). Second, various important experiments have not yet been carried out. For example, further work is needed examining the relationship between non-linguistic aspects of procedural memory and grammar, both in learning and processing. The nature of declarative memory based compensation also warrants further investigation (Ullman & Pullman Under Review). Finally, a number of particular issues have yet to be resolved. For example, the functional roles played by the basal ganglia and it substructures, and the precise nature of language deficits in basal ganglia disorders, are still not entirely clear (Ullman 2006b, Under Review).

Nevertheless, the evidence to date clearly supports the view that the two memory systems subserve language, and that many disorders that affect language can be profitably characterized as disorders that affect these systems. Importantly, the study of the two memory systems at many levels, in both humans and animals, should lead not only to a deeper understanding of language and the disorders that affect it, but also to advances in the diagnosis and therapy of these disorders (Ullman 2004, Under Review; Ullman & Pierpont 2005; Ullman & Pullman Under Review).

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