SPECIAL ISSUE

SPECIFIC LANGUAGE IMPAIRMENT IS NOT SPECIFIC TO LANGUAGE: THE PROCEDURAL DEFICIT HYPOTHESIS

Michael T. Ullman and Elizabeth I. Pierpont

(Departments of Neuroscience, Linguistics, Psychology and Neurology Georgetown University, USA)

ABSTRACT

Specific Language Impairment (SLI) has been explained by two broad classes of hypotheses, which posit either a deficit specific to grammar, or a non-linguistic processing impairment. Here we advance an alternative perspective. According to the Procedural Deficit Hypothesis (PDH), SLI can be largely explained by the abnormal development of brain structures that constitute the procedural memory system. This system, which is composed of a network of interconnected structures rooted in frontal/basal-ganglia circuits, subserves the learning and execution of motor and cognitive skills. Crucially, recent evidence also implicates this system in important aspects of grammar. The PDH posits that a significant proportion of individuals with SLI suffer from abnormalities of this brain network, leading to impairments of the linguistic and non-linguistic functions that depend on it. In contrast, functions such as lexical and declarative memory, which depend on other brain structures, are expected to remain largely spared. Evidence from an in-depth retrospective examination of the literature is presented. It is argued that the data support the predictions of the PDH, and particularly implicate Broca's area within frontal cortex, and the caudate nucleus within the basal ganglia. Finally, broader implications are discussed, and predictions for future research are presented. It is argued that the PDH forms the basis of a novel and potentially productive perspective on SLI.

Key words: Specific Language Impairment (SLI), procedural memory, declarative memory, language grammar, lexical memory, syntax, morphology, phonology, working memory, mental imagery, motor function, temporal processing, compensation, basal ganglia, caudate nucleus, Broca's area, fMRI, MRI, ERP

INTRODUCTION

Specific Language Impairment (SLI) is generally defined as a developmental disorder of language in the absence of frank neurological damage, hearing deficits, severe environmental deprivation, or mental retardation (for diagnostic definitions and prevalence of SLI, see Bishop, 1992; Leonard, 1998; Tomblin et al., 1997). Other terms have also been used to label such children, including developmental dysphasia, language impairment, language learning disability, developmental language disorder, delayed speech and deviant language (Leonard, 1998; Ahmed et al., 2001). Several factors have complicated attempts to provide a unified theory of SLI, or even of subgroups of SLI. First, despite the standard use of exclusionary criteria to diagnose SLI, the disorder is clearly not limited to language. Rather, the linguistic impairments co-occur with a number of non-linguistic deficits, including impairments of motor skills and working memory, and with other disorders, such as Attention Deficit Hyperactivity Disorder (Hill, 2001; Leonard, 1998; Tirosh and Cohen, 1998). Second, even though SLI must be a consequence of some sort of neural dysfunction, the neural correlates of the disorder have been largely ignored. This potentially valuable information could provide important constraints on explanatory accounts of the disorder. Third, SLI is a classification that is quite heterogeneous (Leonard, 1998; Stromswold, 2000). Surveys document variation within and across subgroups in the particular aspects of language that are affected and in the types of co-occurring non-linguistic deficits, as well as in the severity with which these linguistic and non-linguistic deficits are found (Aram and Nation, 1975; Miller, 1996; Rapin and Allen, 1988; Stark and Tallal, 1981). Although some previous research has focused on apparently distinct SLI sub-types (Bishop, 2000; Gopnik and Crago, 1991; van der Lely et al., 1998), most studies have paid little attention to variation across individuals with *similar* types of deficits. However, the nature of this type of variability may be important, and may even provide clues to the nature of SLI itself.

Two broad competing theoretical perspectives have attempted to explain SLI. One perspective posits that people with SLI, or at least certain subgroups of individuals with the disorder, suffer from a deficit or delay that is specific to the domain of language, specifically to grammar – that is, to the mental capacity that underlies the rule-governed combination of words into complex structures. This viewpoint has been espoused in numerous flavors, many of which identify particular grammatical operations, mechanisms, or types of knowledge that are problematic. For example, it has been proposed that children with SLI have a selective impairment in establishing structural relationships such as agreement (Clahsen, 1989) or specifier head-relations (Rice and Oetting, 1993). Alternatively, it has been posited that at least certain people with

developmental language impairments may be missing linguistic features (Gopnik and Crago, 1991). Another view is based on the observation that normal children pass through a period in the development of language during which they fail to consistently mark tense in main clauses which require it (Wexler, 1994). According to this "extended optional infinitive" account, children with SLI remain in this stage for a much longer period than normal children, with their language deficits reflecting an incomplete specification of the obligatory tense markings that are normally represented in grammar (Rice et al., 1995).

Some grammar-deficit hypotheses posit that the dysfunction in SLI is quite broad within grammar, rather than being highly specific to a particular grammatical function or operation such as agreement or tense-marking. Thus it has been proposed that a broad range of language impairments may be explained by a deficit that affects the mechanisms underlying the learning and/or computation of implicit grammatical rules (Ullman and Gopnik, 1994). Another account claims that wide-ranging grammatical difficulties can be explained, at least in some children with SLI, by a representational deficit of grammatical relations (van der Lely, 1994; van der Lely et al., 1998).

Grammar-deficit hypotheses have, not surprisingly, been quite successful in accounting for many of the grammatical impairments observed in SLI. However, these hypotheses are also somewhat problematic. Few if any of them – particularly those that posit a highly specific dysfunction – can explain the full range of linguistic deficits, either within a given language or cross-linguistically (Leonard, 1996, 1998). For example, a purely grammatical deficit cannot easily account for the word-finding difficulties often observed in SLI. Even within grammar, such hypotheses may not fully explain the combination of syntactic, morphological and phonological deficits that occur. Moreover, hypotheses positing *only* grammatical deficits cannot account for the non-linguistic difficulties prevalent in SLI.

According to the second broad theoretical perspective, SLI is caused by a non-linguistic processing deficit. Some processing-deficit hypotheses claim that the problems are quite *general* in nature, such as a reduced processing rate, or capacity limitations on processing (Bishop, 1994; Kail, 1994; Leonard et al., 1992b; Norbury et al., 2001). This helps to account for some of the breadth of linguistic *and* non-linguistic impairments observed in SLI. In particular, such hypotheses can explain why children with SLI have difficulties processing verbal and nonverbal stimuli that are rapidly presented or of brief duration, and problems with cognitive tasks such as word retrieval, simultaneous task execution, and phonological discrimination (Leonard, 1998). However, the view that impairments in SLI can be captured by a general processing deficit is also somewhat problematic. First of all, it has been argued that this perspective cannot easily account for certain types of linguistic impairments observed in SLI (Gopnik and Crago, 1991; Rice and Oetting, 1993; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001). In addition, because these hypotheses claim that the deficits are quite general, they have difficulty explaining the apparently selective nature of the non-linguistic impairments (see below, and Leonard, 1998). Finally, a limited processing capacity account does not lend itself well to specific predictions or testable hypotheses, because nearly *any* kind of impairment could potentially be explained by processing limitations or generalized slowing.

Not all processing-deficit hypotheses posit a general deficit. Impairments of a *specific* cognitive or processing mechanism have also been proposed. Some investigators attribute the language impairments in SLI to the dysfunction of phonological working memory (Gathercole and Baddeley, 1990; Montgomery, 1995b), or to an "information processing deficit affecting phonology" (Joanisse and Seidenberg, 1998). Others have argued that the impairments in SLI can be explained by a perceptual or temporal processing impairment, particularly of briefly presented stimuli or rapidly presented sequences of items (Merzenich et al., 1993; Tallal et al., 1993; Tallal and Piercy, 1973b, 1974). On the one hand, these hypotheses can explain certain specific deficits observed in SLI, such as difficulties on tasks involving working memory, phonological processing, or the perception of rapidly presented stimuli. However, it is not clear that *all* children with SLI suffer from these problems (Bishop et al., 1999; Tallal et al., 1991; Tomblin et al., 1995; van der Lely and Howard, 1993). Moreover, such hypotheses cannot easily account for the specific pattern of spared and impaired linguistic and non-linguistic functions in SLI (Hill, 1998; Leonard, 1998; Ullman and Gopnik, 1999; van der Lely and Stollwerck, 1996; van der Lely and Ullman, 2001).

In sum, although previously proposed explanatory hypotheses can individually capture specific aspects of the empirical data, none of them can easily account for either the range or the variation of the particular impaired linguistic and non-linguistic functions found across SLI, and even within SLI subgroups. Indeed, we believe that any purely *functional* account of SLI will have difficulty explaining the variety of impairments that occur even within individuals with SLI. Moreover, few hypotheses have seriously attempted to link the cognitive impairments in SLI to the brain, or to account for the range of neural abnormalities observed in the disorder.

Here we propose that a substantial number of individuals with SLI are afflicted with

abnormalities of brain structures that constitute a well-studied brain system known as the *procedural memory system*. We argue that this Procedural Deficit Hypothesis (PDH) can explain the neural abnormalities found in SLI, and can account for – apparently paradoxically – much of the consistency *and* heterogeneity in the linguistic and non-linguistic deficits found in the disorder, both within and across individuals and subgroups. Moreover, *independent* knowledge of these well-studied brain structures and their functions allows us to make testable predictions regarding SLI that would not be possible on the basis of more restricted explanatory accounts. The remainder of this paper is structured as follows. First, we discuss the neuroanatomy and functions of the procedural memory system. Second, we introduce the PDH in some detail. Third, we present a wide range of neural and behavioral empirical evidence, and argue that it provides converging evidence for the PDH. We conclude by examining clinical and other implications, and presenting a specific research program and further predictions.

The Procedural Memory System

The "procedural memory system" is the brain system that is implicated in "procedural memory" – that is, in the learning of new, and the control of long-established, motor and cognitive "skills", "habits," and other procedures, such as typing, riding a bicycle, and skilled game playing (Mishkin et al., 1984; Schacter and Tulving, 1994; Squire et al., 1993; Squire and Knowlton, 2000). (For the sake of simplicity, below we also refer to the system as the "procedural system".) The system underlies aspects of rule-learning (Knowlton et al., 1996; Poldrack et al., 1999), and is particularly important for acquiring and performing skills involving sequences – whether the sequences are serial or abstract, or sensori-motor or cognitive (Aldridge and Berridge, 1998; Boecker et al., 2002; Graybiel, 1995; Jenkins et al., 1994; Saint-Cyr et al., 1988; Willingham, 1998). Acquisition of the procedures is gradual, in that learning occurs on an ongoing basis during multiple trials. In contrast, the learned procedures generally apply quickly and automatically. The system is closely related to the "dorsal" stream (also referred to as the "how" or "where" pathway, Goodale, 2000; Goodale and Milner, 1992; Ullman, 2004; Ungerleider and Mishkin, 1982). It is commonly referred to as an implicit memory system because both the learning of the procedural memories and the memories themselves are generally not available to conscious access. Note that we use the term "procedural memory system" to refer *only* to one type of implicit, non-declarative, memory system (Squire and Knowlton, 2000), *not* all non-declarative or implicit memory systems. Moreover, we use the term to refer to the *entire system* involved in the learning, representation and use of procedural memories, not just to those parts of the system underlying the acquisition of new procedural knowledge (also see Ullman, 2004).

The procedural system is composed of a network of several interconnected brain structures (De Renzi, 1989; Heilman et al., 1997; Hikosaka et al., 2000; Jenkins et al., 1994; Mishkin et al., 1984; Rizzolatti et al., 2000; Schacter and Tulving, 1994; Squire and Zola, 1996). Within the cerebrum, the system depends especially on structures in the left hemisphere (De Renzi, 1989; Heilman et al., 1997; Schluter et al., 2001). Frontal/basal-ganglia circuits play a particularly important role in the system (Eichenbaum and Cohen, 2001; Mishkin et al., 1984; Schacter and Tulving, 1994; Squire and Zola, 1996).

The basal ganglia are a set of highly interconnected subcortical structures, including the neostriatum, globus pallidus, sub-thalamic nucleus, and substantia nigra (Cote and Crutcher, 1991; Wise et al., 1996). In primates, the neostriatum (also simply called the "striatum") is composed of two structures: the putamen and the caudate nucleus. Studies of non-human primates have revealed much about the connectivity of the basal ganglia. These structures are closely linked to cortical regions, particularly in the frontal lobes, via parallel and largely functionally segregated "channels" (also referred to as "circuits" or "loops") (Alexander and Crutcher, 1990; Alexander et al., 1986; Middleton and Strick, 2000a). Each channel receives projections at the neostriatum – some channels primarily at the caudate, others at the putamen – from a particular set of cortical and subcortical structures. Each channel then follows the same set of internal connections within the basal ganglia, and then projects outward via the thalamus to a particular cortical region (from which it also receives input), primarily in frontal cortex.

Within the basal ganglia, the circuitry underlying the channels splits into the "direct" and "indirect" pathways. These two pathways have opposing effects on the basal ganglia's influence on frontal cortex. Via a series of inhibitory and excitatory projections, the direct pathway ultimately disinhibits frontal cortical activity, whereas the indirect pathway ultimately inhibits it. Imbalances between the two pathways can lead to the excessive inhibition or disinhibition of functions that depend on the frontal cortical regions to which the basal ganglia project (Albin et al., 1989; Young and Penney, 1993). This is thought to explain the inhibited/suppressed ("hypo") and disinhibited/unsuppressed ("hyper") motor and other behaviors found in Parkinson's disease, Huntington's disease, Tourette syndrome, Obsessive-Compulsive disorder (OCD), Attention Deficit Hyperactivity Disorder (ADHD), and other adult-onset and developmental disorders affecting

the basal ganglia (Bradshaw, 2001; Middleton and Strick, 2000a; Young and Penney, 1993).

The channels are topographically organized at each level of the circuit, including within the direct and indirect pathways. That is, the parallel channels are at least partly anatomically separable in having a topographic organization that is maintained from the neostriatum throughout the basal ganglia to the thalamus and frontal cortex. The different basal ganglia-thalamocortical channels project to a heterogeneous set of frontal regions, and subserve a heterogeneous set of functions. Thus, distinct channels project to primary motor cortex, ventral premotor cortex (a region that contains F5, the likely macaque homologue of Brodmann's Area (BA) 44, Rizzolatti et al., 1996), the Supplementary Motor Area (SMA), and dorsolateral prefrontal cortex, among other regions (Alexander et al., 1986; Middleton and Strick, 2000a). Each channel underlies those functions that are associated with the cortical region to which it projects (Middleton and Strick, 2000a). For example, the channel projecting to primary motor cortex subserves motor functions. Channels passing through the putamen play a particularly important role in movement, whereas those passing through the caudate seem to be especially important for aspects of cognition – although it should be emphasized that both striatal structures likely play at least some sort of role in both motor and cognitive functions (Alexander et al., 1986; Middleton and Strick, 2000a; Poldrack et al., 1999). The different basal ganglia channels appear to have a similar synaptic organization, suggesting that similar neuronal operations are performed at comparable stages of each channel (Alexander et al., 1990; Middleton and Strick, 2000b). So, the basal ganglia appear to perform analogous computations that are applied to different sets of information from different domains, depending on the particular set of input regions and frontal cortical output destinations of a given channel (Middleton and Strick, 2000b).

Within frontal cortex, certain regions play especially crucial roles in procedural memory: most importantly, pre-motor cortex (particularly the region of the Supplementary Motor Area; i.e., SMA and pre-SMA) and cortex within Broca's area (part of inferior frontal cortex, containing BA 44 and 45, Amunts et al., 1999). Intriguingly, evidence suggests that Broca's area is critical for abstract, cognitive aspects of procedural memory, although the region also subserves motor functions (Conway and Christiansen, 2001; Dominey et al., 2003; Goschke et al., 2001; Ullman, 2004). Thus, despite the apparent computational equivalence of the basal ganglia across channels, not all of the frontal regions to which the channels project subserve procedural memory, and those regions that do underlie this function play a variety of roles. This functional heterogeneity can be explained by several factors, including the differential cytoarchitectonic and computational specialization of different frontal regions (Amunts et al., 1999), and their differential connectivity to posterior regions, such that each frontal region selects or inhibits a different type of information or response (Passingham, 1993; Shimamura, 1995). Importantly, because not all frontal regions are involved in procedural memory, it follows that not all topographically organized regions of the neostriatum (or other structures within the basal ganglia) should be involved in this function. Rather, only those basal ganglia regions whose circuitry projects to frontal regions that subserve procedural memory are predicted to play an important role in this function.

In addition to the frontal/basal-ganglia circuits, several other structures play likely roles in the procedural memory system (For further discussion, see Ullman, 2004): parietal cortex, particularly the supramarginal gyrus (BA 40); aspects of the superior temporal cortex, including the superior temporal sulcus; and the cerebellum, including the dentate nucleus (one of the deep cerebellar nuclei, and an important output nucleus of the cerebellum) (Carey et al., 1997; Chao et al., 1999; Jellema and Perrett, 2001; Martin et al., 2000; Perrett et al., 1989, 1990; Rizzolatti et al., 2001). Interestingly, the cerebellum is similar to the basal ganglia in that parallel topographically organized channels are maintained from the cerebellum through the thalamus to frontal cortex, including to ventral premotor and dorsolateral prefrontal regions (Middleton and Strick, 2000a).

The different structures of the procedural system provide distinct and complementary computational and functional contributions. For example, the basal ganglia are particularly important for learning new procedures, but may be less so for the normal processing of already-learned procedures (Mishkin et al., 1984; Squire and Knowlton, 2000; Ullman, 2003, 2004). In contrast, Broca's area appears to underlie both the learning and the processing of procedures (Dominey et al., 2003; Goschke et al., 2001; Ullman, 2004). So, abnormalities of different structures in the system should lead to different types of impairments of procedural memory.

Finally, the brain structures that constitute the procedural system subserve not only motor and cognitive skills, but also a number of other functions, including grammar, lexical retrieval, dynamic mental imagery, working memory, and rapid temporal processing. Moreover, whereas procedural memory appears to be directly related to some of these functions (e.g., grammar), its relation to others is still not clear. Indeed, other functions might plausibly depend on portions of these brain structures that do not subserve procedural memory (e.g., other channels within frontal/basal-ganglia circuits). Crucially however, whether or not these

other functions are related to procedural memory, they should be compromised by abnormalities of the structures that subserve them. Therefore these functions should be impaired in brain disorders that affect the procedural memory system. Here we focus on those functions that are discussed again below in the context of SLI. However, others (presumably including some that have yet to be discovered) also likely depend on these brain structures, and so would also be expected to be impaired in individuals with abnormalities of these structures.

Language

The involvement of the procedural system in language appears to be largely based upon the distinction between rule-governed and idiosyncratic linguistic mappings. Specifically, it has been posited that important aspects of the difference between these two types of mappings can be captured by the distinction between procedural memory and declarative memory, another well-studied memory system (Ullman, 2001a, 2001c, 2004; Ullman et al., 1997). According to this view – referred to as the Declarative/Procedural (DP) model – idiosyncratic mappings are stored in a memorized "mental lexicon" that depends on declarative memory, whereas the learning and use of rule-governed computations involves a "mental grammar" that depends on procedural memory.

Thus, like other "dual-system" views of language (Chomsky, 1995; Pinker, 1994; Pinker and Ullman, 2002b), the DP model critically assumes a categorical distinction between lexicon and grammar ("Single-mechanism" models are discussed in the Conclusion). The lexicon is the repository of all arbitrary word-specific knowledge, such as the fact that *cat* refers to the warm furry thing on your bed. It also holds other irregular – i.e., not entirely derivable – information, such as the particular arguments that must accompany a given verb (i.e., verb complements, as reflected by the fact that *hit* has a direct object), and any unpredictable forms that a word takes (e.g., *teach* takes the irregular past-tense *taught*). The lexicon may also comprise other distinctive information, such as bound morphemes (e.g., the *-ed* or *-ness* suffixes, as in *walked* and *happiness*) and representations of complex linguistic structures whose meanings cannot be transparently derived from their parts (e.g., idiomatic phrases, such as *kick the bucket*) (Di Sciullo and Williams, 1987; Halle and Marantz, 1993). In contrast, the rules of the mental grammar underlie the regularities of language. The rules constrain how lexical forms can combine sequentially and hierarchically to make complex representations. Such rule-governed behavior is found in various language domains, including in phrases and sentences (syntax), and in complex words such as *walked* (morphology). The rules are a form of mental knowledge in that they underlie our individual capacity to produce and comprehend complex forms. The learning and use of the rules and operations of grammar are generally implicit (non-conscious). At least certain aspects of grammatical processing are fast as well as automatic (Fodor, 1983; Friederici, 2002; Friederici and Mecklinger, 1996). Finally, although complex representations (e.g., the regular past-tense *walked*) could be composed anew each time (e.g., *walk* + *-ed*), and must be if they were not previously encountered (e.g. *glicked*), they could in principle also be stored in the mental lexicon.

In order to understand the claims of the DP model, and the role of the procedural memory system in language, it is also important to briefly discuss the declarative memory system. This brain system underlies the learning, representation, and use of knowledge about facts ("semantic knowledge") and events ("episodic knowledge") (Mishkin et al., 1984; Schacter and Tulving, 1994; Squire and Knowlton, 2000). (Analogous to our use of the term "procedural memory system", we use the term "declarative memory system" to refer to the entire system involved in the learning, representation and use of the relevant information (Eichenbaum, 2000), not just to those brain structures underlying the learning of new memories.) The system may be particularly important for learning arbitrary relations (Eichenbaum and Cohen, 2001). The learned information can, at least in part, be consciously ("explicitly") recollected. The system is closely related to the "ventral" stream or "what" pathway (Goodale, 2000; Ungerleider and Mishkin, 1982). The system crucially involves medial temporal lobe regions (in particular the hippocampus), which are connected extensively with temporal and parietal neocortical regions (Suzuki and Amaral, 1994). The medial temporal structures underlie the consolidation (and possibly the retrieval) of new memories. Memories eventually become independent of these structures, and dependent on neocortical regions, particularly in the temporal lobes (Hodges and Patterson, 1997; Martin et al., 2000).

The DP model posits that each of the two memory systems plays an analogous role in its non-linguistic and linguistic functions (for additional details and discussion, see Ullman, 2001c, 2004). Thus medial temporal lobe structures are predicted to underlie the learning of new words, which eventually depend largely on neocortical regions, particularly in temporal and temporo-parietal regions. Middle and inferior portions of the temporal lobe are posited to be especially important for storing word meanings, whereas superior temporal and temporo-parietal regions are more important for storing word sounds, and possibly also for stored complex morphological and syntactic structures. These latter

regions may thus serve as an interface between the declarative and procedural systems. The procedural system is claimed to underlie aspects of a symbol-manipulating grammar, across grammatical sub-domains, including not only morphology and syntax, but likely also phonology (in the sequencing of sounds). The procedural system may be particularly important in the learning and use of rules that underlie grammatical structure building – that is, the sequential and hierarchical combination of stored lexical forms (e.g., *walk* + *-ed*) and abstract representations (e.g., noun phrase) into complex structures. It is expected that learning rules of grammar, like learning other rules within the procedural system, should depend especially on the basal ganglia, in particular the caudate nucleus, whereas rule-governed compositional aspects of grammar should depend largely on Broca's area. It is an empirical question as to whether the same or distinct basal ganglia-thalamocortical channels (e.g., within the caudate and Broca's area) subserve grammatical and non-linguistic procedures. Similarly, the different sub-domains of grammar (e.g., phonology, morphology, syntax) may rely on the same or distinct circuitry within the procedural memory system.

Thus the DP model predicts important associations between lexical and declarative memory, and between aspects of grammar and procedural memory, as well as dissociations between lexicon and grammar that parallel dissociations between the two memory systems. However, these dissociations are somewhat complicated by the fact that certain brain structures underlying the procedural system also play specific roles related to declarative memory. Ventro-lateral pre-frontal cortex, which corresponds to the inferior frontal gyrus and BA 44, 45 and 47 (Damasio, 1995), underlies the encoding of new declarative memories and the selection or retrieval of declarative knowledge (Buckner, 2000; Buckner and Wheeler, 2001; Thompson-Schill et al., 1997; Wagner et al., 1998). Moreover, evidence suggests that portions of the basal ganglia and the cerebellum are also involved in selecting, retrieving or searching for declarative memories (Desmond and Fiez, 1998; Ivry and Fiez, 2000; Ullman, 2004; Wise et al., 1996). Thus the DP model predicts that these structures should play analogous functional roles for lexical memory – namely, in the selection, retrieval or search for lexical knowledge (Ullman, 2004). Dissociations between lexicon and grammar should therefore be particularly striking when these functions are *not* required, such as in lexical recognition tasks.

We have argued elsewhere that a broad range of converging evidence, from psycholinguistic, neurological, and neuroimaging studies, supports the DP perspective (Ullman, 2001c, 2004; Ullman et al., 1997). Therefore this evidence will not be presented or discussed here.

Mental Imagery

The procedural system also subserves aspects of mental imagery. Two types of mental imagery have previously been identified (Farah, 1989). "Dynamic" or "spatial" mental imagery involves spatial relations (e.g., location) and the mental manipulation or transformation of images. This type of mental imagery, which is found in tasks entailing mental rotation or orientation discrimination, is strongly linked to motor processes (Vingerhoets et al., 2002; Wexler et al., 1998c), and seems to depend on the dorsal stream pathway (Farah, 1989; Podzebenko et al., 2002) and on the brain structures underlying the procedural system: Broca's area (Jordan et al., 2001; Podzebenko et al., 2002); premotor regions, including both lateral premotor cortex and the SMA (Cohen et al., 1996; Jordan et al., 2001; Kosslyn et al., 1998; Podzebenko et al., 2002; Richter et al., 2000; Tagaris et al., 1997); the basal ganglia (Podzebenko et al., 2002); the cerebellum (Ivry and Fiez, 2000; Podzebenko et al., 2002); and parietal cortex (Bestmann et al., 2002; Harris et al., 2000; Podzebenko et al., 2002), including the supramarginal gyrus (Harris et al., 2000; Podzebenko et al., 2002). In contrast, "static" or "visual" imagery, which involves imaging static objects or their features (e.g., color, form), is linked to the perception and processing of this type of information, to occipital and temporal regions, and to the ventral stream pathway (which is closely related to declarative memory; see above) (Farah, 1989, 1995; Goodale, 2000).

Working Memory

Working memory is strongly linked to brain structures that underlie the procedural system: Broca's area and premotor regions, including both lateral premotor cortex and the SMA (Ivry and Fiez, 2000; Smith and Jonides, 1999); the basal ganglia, (Menon et al., 2000); the cerebellum (Desmond and Fiez, 1998; Desmond et al., 1998); and parietal cortex, including the supramarginal gyrus (Ivry and Fiez, 2000). Working memory appears to involve the dorsal stream system (Hickok and Poeppel, 2000), and seems to be functionally related both to the retrieval of declarative knowledge and to procedural memory itself (for further discussion, see Ullman, 2004). Thus portions of frontal cortex that subserve the selection or retrieval of declarative information are closely tied to working memory (Buckner and Wheeler, 2001; Moscovitch, 1992). For example, neuroimaging studies show that ventro-lateral pre-frontal cortex is consistently activated while working memory is engaged (Smith and Jonides, 1998, 1999), and is activated, within the same subjects, during both retrieval and working memory tasks (Braver et al., 2001). It has been

suggested that Broca's area, and perhaps ventro-lateral pre-frontal cortex more generally, may be specialized for the selection and comparison of maintained information (Petrides, 1996; Petrides et al., 1995), or for the maintenance of information over a delay (D'Esposito et al., 1998; Smith and Jonides, 1997). Similarly, it has been argued that the role of this cortex in working memory may be to recall or select and maintain information that is actually stored in temporal and temporo-parietal regions (Cowan, 1996, 1999; Ruchkin et al., in press). A recent study suggests that posterior Broca's area subserves the manipulation of sequential information, independent of the type of information that is manipulated (Gelfand and Bookheimer, 2003). The basal ganglia have been implicated in similar functions, including context-dependent rule-based selection, and the maintenance in working memory of – and the real-time shifting between – sets, functions or programs (Menon et al., 2000; Peigneux et al., 2000; Wise et al., 1996). Moreover, these basal-ganglia functions seem to be quite intimately related (Meck and Benson, 2002; Wise et al., 1996). In sum, working memory clearly depends on the brain structures underlying the procedural system, and moreover may be closely related to their other functions.

Temporal Processing

Finally, evidence also suggests that important aspects of timing and rapid temporal processing depend on brain structures that underlie the procedural system – including Broca's area (Fiez et al., 1995; Hickok and Poeppel, 2000; Muller et al., 2001; Schubotz and von Cramon, 2001), the basal ganglia (Meck and Benson, 2002; Rammsayer and Classen, 1997), and the cerebellum (Hazeltine et al., 1997; Ivry et al., 1988). This is not surprising, given that the procedural system underlies on-line rapid processing, including rapid sequencing functions (Eichenbaum and Cohen, 2001; Schacter and Tulving, 1994; Squire and Knowlton, 2000), and that the dorsal stream is crucial for real-time interaction with the world (Goodale, 2000; Goodale and Milner, 1992).

Neuropharmacological evidence suggests that temporal processing of intervals in the range of milliseconds depends on the level of dopaminergic activity in the basal ganglia (Brooks et al., 1990; Rammsayer, 1999). Behavioral and fMRI evidence also closely ties the basal ganglia to the processing of temporal discrimination thresholds (the perception of rapidly presented stimuli as either simultaneous or sequential) (Fiorio et al., 2003); to motor timing (Grasso et al., 1999; Harrington et al., 1998; Malapani et al., 2002) and to duration estimation (Ferrandez et al., 2003; Harrington et al., 1998; Nenadic et al., 2003). Moreover, just as the left hemisphere is particularly important for procedural memory, so it also appears to be crucial for rapid temporal processing: Evidence suggests that the left hemisphere has better temporal resolution than the right, and may be specialized for much shorter periods of temporal integration (Allard and Scott, 1975; Hickok and Poeppel, 2000; Ivry and Robertson, 1998; Nicholls, 1996; Zatorre et al., 2002).

Intriguingly, the timing and rapid processing functions of the brain structures underlying the procedural system appear to be linked to other functions of this system. Thus it has been suggested that the real-time set shifting and sequence coordination roles of frontal cortex may depend upon timing functions subserved by this region (Knight and Grabowecky, 2000; Meck and Benson, 2002; Wise et al., 1996). Similarly, the basal ganglia's role in real-time motor control may be especially important for motor skills that involve precise timing (Penhune et al., 1998). Moreover, the basal ganglia also play an important role in interval timing and rhythm (Meck and Benson, 2002; Rammsayer and Classen, 1997). Thus timing, rhythm and rapid temporal processing depend on the brain structures underlying the procedural system, and may be closely related to their other functions.

The Procedural Deficit Hypothesis

According to the Procedural Deficit Hypothesis, many if not most individuals with SLI are afflicted with procedural system brain abnormalities that result in grammatical impairments and/or lexical retrieval deficits (also see Gopnik, 1999; Paradis and Gopnik, 1997; Ullman and Gopnik, 1994, 1999). These individuals may be characterized as having Procedural Language Disorder, or PLD. Importantly, these individuals should also have impairments of the non-linguistic functions that also depend on the affected brain structures of the procedural system.

The PDH leads to several testable and falsifiable predictions, both at the population and individual level. Abnormalities of the brain structures that underlie the procedural system, and impairments of grammar, lexical retrieval, and the non-linguistic functions that depend on these structures, should all be widespread in SLI. Moreover, these brain abnormalities and linguistic and non-linguistic impairments should be strongly associated with each other – that is, they should co-occur. This association should be reflected within individuals with SLI. People with SLI who have impairments of grammar and/or of lexical retrieval should have abnormalities of brain structures underlying the procedural system, and impairments of the non-linguistic functions that depend on them.

Grammatical impairments and lexical-retrieval deficits could in principle arise from abnormalities

of any of the brain structures in the procedural system. Moreover, the dysfunction of different structures in the system should lead to different types of impairment. For example, abnormalities of structures thought to underlie the acquisition of procedural knowledge, such as the basal ganglia, should yield different behavioral phenotypes than abnormalities of those that subserve the execution of procedural skills, which might be the case for the cerebellum (Seidler et al., 2002). We expect that a portion of the heterogeneity of SLI is explained by variation across individuals as to which structures are affected, and to what degree. Importantly, a variety of methods can be used to determine which particular structures are abnormal (see below).

Despite the likely existence of such variation across SLI, we posit that grammatical and lexical-retrieval impairments are particularly strongly associated with dysfunctions of the basal ganglia, especially the caudate nucleus, and of frontal cortex, especially Broca's area. As we have seen above, frontal/basal-ganglia circuits play a core role in procedural memory. Moreover, the caudate nucleus and Broca's area are especially important for cognitive functions, including aspects of grammar and lexical retrieval (see above, and Ullman, 2004).

Just as abnormalities of different structures within the procedural system should lead to behavioral heterogeneity, so should the dysfunction of different *portions* of structures – especially of those structures that constitute the frontal/basal-ganglia circuitry. First of all, different *types* of deficits should be associated with the dysfunction of the direct and indirect pathways. Indeed, neurodevelopmental as well as neurodegenerative disorders affecting the basal ganglia have been characterized as primarily "hyper" or "hypo" in nature (Bradshaw, 2001; Jankovic and Tolosa, 1993; Middleton and Strick, 2000a, 2000b). It remains to be seen whether or not SLI is associated primarily with one or the other type of deficit. Note that the demonstration of either deficit in SLI would strongly implicate the basal ganglia, and would support the predictions of the PDH.

Second, it is unlikely that *only* those channels that subserve grammar or lexical retrieval will be affected in a given individual with PLD. Both in neurodegenerative and neurodevelopmental disorders that involve the basal ganglia, multiple domains are generally impaired, moreover with analogous impairments (e.g., suppressed, "hypo" behaviors) across domains (Bradshaw, 2001; Jankovic and Tolosa, 1993), including grammar. Thus Huntington's disease patients show unsuppressible grammatical rule use (e.g., in affixation) as well as unsuppressible movements, whereas Parkinson's disease patients show the suppression of both grammatical rule use and of movement (Ullman, in press-b; Ullman et al., 1997). Crucially, it is unlikely that exactly the same channels will be affected across all individuals with a particular frontal/basal-ganglia disorder. Indeed, the correlations between motor and grammatical impairments across Parkinson's or Huntington's disease patients are not perfect (Ullman, in press-b; Ullman et al., 1997). We therefore expect variability across individuals with SLI with respect to the combination of channels that are affected, and the severity of their dysfunction.

Despite this variability, we do not expect that all channels have an equal probability of being affected in SLI. Rather certain channels should be more likely than others to be dysfunctional. Evidence from well-studied basal ganglia disorders suggests that in a given disorder certain portions of the circuitry are more problematic than others. For example, Parkinson's disease primarily disturbs channels passing through the putamen, whereas Huntington's disease tends to affect the caudate nucleus (Jankovic and Tolosa, 1993). Therefore, we predict that in SLI, grammatical and lexical-retrieval deficits should be more strongly associated with certain impairments than with others. Functions that depend on circuitry involving the caudate nucleus and Broca's area should be especially likely to be impaired. So, although variability in which channels are affected should lead to some functional heterogeneity across subjects, an important degree of similarity among individuals with the disorder is expected.

Although it is unlikely that any abnormality will be restricted only to those portions of the frontal/basal-ganglia circuitry that subserve grammar or lexical retrieval, such a circumscribed dysfunction is possible in principle. However, this would not necessarily lead to a deficit limited to language – that is to "pure" SLI. As discussed above, it is an empirical question as to whether the circuitry subserving grammar and lexical retrieval is dedicated to these functions. Only if this turns out to be true could frontal/basal-ganglia abnormalities lead to pure SLI. Conversely, the demonstration of pure SLI with frontal/basal-ganglia abnormalities would suggest the existence of such domain-specific circuitry. It should be emphasized, however, that demonstrating domain-specificity – that is, showing that a deficit is restricted to a particular domain – is a very difficult endeavor (also see Karmiloff Smith, 1998). In the absence of a theory that posits particular deficits in other domains, it is not even clear which other domains to test. Because there are in principle an infinite number of tests that could be given, one can never completely rule out the presence of other deficits. Even with a guiding hypothesis such as the PDH, there is the danger that a failure to find a predicted deficit – that is, a null result – may be due to a failure to "look hard enough". Therefore one must be very cautious in

accepting claims of domain-specificity, including claims of deficits that are restricted to grammar in SLI.

Note also that if non-linguistic deficits co-occur probabilistically with language impairments in SLI, the selection of SLI subjects based on the exclusion of co-occurring deficits would severely bias the subject sample, resulting in the false impression that SLI is a distinct disorder circumscribed to language. Indeed, it appears that divergent results from different studies are at least partly due to the criteria used to select or eliminate participating subjects (Stromswold, 2000).

The etiology of the disorder is expected to be diverse. That is, there could be numerous etiologies resulting in the atypical development of one or more of the structures underlying the procedural system. However, we hypothesize that many cases of the disorder are attributable to etiologies affecting the basal ganglia, in particular the striatum – that is the caudate nucleus and/or the putamen. The basal ganglia, especially portions of the striatum, are implicated in many developmental disorders, including ADHD, Tourette syndrome, OCD, and Sydenham's chorea (Bradshaw, 2001; Garvey et al., 1998; Giedd et al., 2000; Middleton and Strick, 2000a, 2000b). Intriguingly, among these disorders, at least ADHD is highly co-morbid with SLI (Cohen et al., 2000; Tirosh and Cohen, 1998). More generally, the striatum appears to be extremely vulnerable to damage, both in adult-onset and developmental disorders (Mitchell et al., 1999). Different types of biochemical and other insults differentially affect the different striatal cell types, and lead to distinct behavioral phenotypes, including contrasting "hypo" and "hyper" behaviors (Mitchell et al., 1999). For example, whereas amphetamines affect dopaminergic terminals in the striatum, other striatal neurons are susceptible to mitochondrial inhibitors (Mitchell et al., 1999). Increasing evidence also suggests that certain streptococcal bacteria can trigger antibodies that cross-react with the basal ganglia of genetically susceptible children, leading to a constellation of developmental disorders, including Tourette syndrome and OCD (Garvey et al., 1998; Swedo et al., 1997). Thus the basal ganglia are also vulnerable to auto-immune reactions.

Recent studies of rodents, monkeys and humans that have examined the regional brain expression of the *FOXP2* gene during different stages of development also implicate the striatum – especially the caudate nucleus – as well as other procedural system brain structures (Bruce and Margolis, 2002; Lai et al., 2003; Takahashi, 2003; Takahashi et al., 2003). This research is of particular interest because mutations of *FOXP2* are found in certain individuals with speech and language impairments which we claim are explained by the PDH (i.e., the members of the KE family; see below). One study examined the regional brain expression of rat *Foxp2* throughout development (Takahashi et al., 2003). High levels of *Foxp2* expression were found in the striatum, particularly in striatal elements that are prominent in the caudate nucleus. Levels of expression were higher in the developing tissue than in the adult tissue, demonstrating the importance of the gene in the developing striatum. The gene was also expressed in portions of the cerebral cortex and the cerebellum, but not in the hippocampus. Similar results were obtained in a study of *FOXP2/Foxp2* expression in prenatal humans and mice, and in newborn mice (Lai et al., 2003): The gene was found to be expressed in several structures, including the caudate nucleus, substantia nigra, cerebellum (including the deep cerebellar nuclei), and certain cortical regions, but not in the hippocampus. *FOXP2* expression in the caudate nucleus was also reported in an investigation of human adult tissue (Bruce and Margolis, 2002). Finally, preliminary data from non-human primates suggests the expression of *Foxp2* in prenatal striatum (both in the caudate and the putamen) but not in adult striatum (Takahashi, 2003). Together, these data suggest that *FOXP2* is important for the striatum and certain other brain structures of the procedural system, especially the caudate nucleus, particularly in the developing brain. Moreover, *FOXP2* does not appear to play any significant role in the hippocampus, which underlies the learning of declarative memories.

Thus we posit that basal ganglia abnormalities in PLD can and do arise from a variety of sources, including anomalies of genes that are expressed in the developing caudate nucleus, and a variety of early-onset intrinsic and extrinsic insults to the basal ganglia (e.g., see Tallal et al., 1994). So, PLD may arise not only from the problematic *development* of certain brain structures, but also from early neural *insults*, which in turn lead to atypical brain development. It is important to emphasize that the source of the disorder is expected to vary across individuals. Some may have mutations of the *FOXP2* gene, while many others show no evidence of such mutations (Meaburn et al., 2002; Newbury et al., 2002), and instead suffer from other etiologies. Moreover, *FOXP2* is not expected to be the only gene involved in PLD – although a recent study found that SLI was strongly associated with genetic markers adjacent to *FOXP2*, suggesting that "genetic factors for regulation of language impairment reside in the vicinity of *FOXP2*" (O'Brien et al., 2003). Note that even if a genetic component *can* lead to the disorder, an environmental component may *also* be necessary. Thus, as discussed above, auto-immune reactions resulting in basal-ganglia abnormalities might occur only in individuals with a *genetic pre-disposition*.

Even if the abnormality in an individual with SLI is initially circumscribed to specific brain

structures, other structures may also be affected as development ensues. Thus evidence suggests that a dysfunction which is at first restricted to one structure can lead to problems in others during development, partly due to their inter-connectivity (Levitt, 2000; Neville and Bavelier, 2000; Rakic et al., 1991; Sur and Leamey, 2001). In PLD, this atypical development may be concentrated in the procedural system, thanks to the high inter-connectivity among its structures. However, other structures connected to this system could in principle also be affected. Thus it is important to emphasize that although we explicitly predict that structures in the procedural system will develop atypically, abnormalities will not necessarily be restricted to these areas.

Given the highly plastic nature of the developing brain, compensation is likely to occur. In particular, it has been shown that the functions of abnormal neural tissue can be taken over by similar or proximate intact tissue (Merzenich et al., 1988). Thus abnormalities of specific portions of the striatum or frontal cortex may be compensated for by other portions of these structures. This may partly explain the gradual improvements in language abilities that are often observed in SLI as the child matures (Aram, 1984; Leonard, 1998).

In addition, if a function can be performed by more than one computational mechanism, it could be taken over by a brain structure whose computational role is distinct from that of an abnormal structure. We posit that in PLD the declarative memory system can and will take over certain grammatical functions from the procedural memory system. In particular, complex structures that can be composed by the grammatical/procedural system (e.g., *walk* + *-ed*) in normally developing individuals may simply be stored as chunks (e.g., *walked*) in lexical/declarative memory by individuals with PLD. Moreover, these individuals should be able to compensate for their grammatical deficit by learning explicit rules in declarative memory, such as "add -ed to the end of the verb when the event has already happened". Importantly, evidence suggests that such declarative-memory compensation takes place in other populations who appear to be afflicted with a grammatical/procedural dysfunction, such as normal adult learners of a second language, and agrammatic aphasics (for discussions and supporting evidence, see Drury and Ullman, 2002; Ullman, 2001b, in press-a). At least some of the improvement that is often observed in SLI as the child matures should be explained by such compensation. Thus the extent to which language deficits resolve over time in individuals with SLI is predicted to depend at least in part on extant lexical/declarative abilities. Grammatical impairments should be less apparent over time in individuals with superior lexical/declarative abilities, because greater compensation should take place.

The specific profile of impaired and spared linguistic abilities should be affected by declarative-memory compensation. Particular types of complex structures (e.g., those that are shorter and of higher frequency) and particular types of rules (e.g., those that are simpler or more "salient") should be especially easy to learn in declarative memory. This should lead not only to longitudinal improvements within individuals with SLI, but also to group differences. The phenotype (i.e., the expression) of SLI should vary across languages as a function of the extent to which the languages' grammatical systems lend themselves to one or the other strategy. Thus in principle the nature of the grammatical system of a particular language may lead to higher or lower observable levels of SLI.

Importantly, the composition of complex structures by the grammatical/procedural system can be empirically distinguished from the retrieval of such forms from lexical/declarative memory, and from the use of explicit rules learned in declarative memory. These different neurocognitive strategies can be teased apart using a range of methods, including the following: testing for frequency effects (only memorized complex forms should show frequency effects, Drury and Ullman, 2002; Ullman et al., 2002; Ullman and Gopnik, 1994, 1999; van der Lely and Ullman, 2001); performing detailed phonetic analyses (explicit rules may yield anomalous phonological forms, Ullman and Gopnik, 1994, 1999); examining the electrophysiological correlates of complex forms (in ERP studies, violations of grammar should elicit Left Anterior Negativities, whereas forms that are retrieved from memory, or are constructed by explicit rules, should yield N400s, Ullman, 2001b; Ullman et al., 2002); and examining the hemodynamic responses to linguistic forms (in PET or fMRI studies, grammatical composition should yield activation in procedural system structures, whereas stored chunks and explicit rules should elicit activation in declarative memory brain structures, Ullman, 2001b). Moreover, the role of the procedural system in lexical/declarative tasks can be experimentally minimized, allowing one to test for clean dissociations between the two systems. Thus lexical recognition tasks allow one to sidestep the need for lexical retrieval, which depends on brain structures underlying the procedural system. Similarly, word-learning tasks can be designed to minimize involvement of the procedural system – for example, by avoiding complex syntactic contexts and the rapid presentation of items.

Of course compensation by the declarative memory system can only take place if this system remains relatively normal. Although in principle the PDH makes strong predictions only about the procedural system, and is agnostic as to whether other structures and their functions remain intact, we hypothesize that in PLD the declarative

memory system generally remains largely normal, and may even be enhanced. This hypothesis is motivated by three factors. First, in the absence of any specific reason to believe that the system should be impaired, it is reasonable to take the position that it is likely to be spared. Second, if both declarative and procedural memory *were* largely dysfunctional, the resulting linguistic and cognitive profile should be more severely affected than that is generally observed in SLI. Such doubly afflicted individuals would be expected to have serious deficits of word-learning as well as of grammar, rendering them almost without language. Moreover, impairments in non-linguistic aspects of the two memory systems should leave such individuals largely dysfunctional. Third, evidence from animals and humans suggests that the declarative and procedural memory systems interact competitively (Jaskiw et al., 1990; Lipska et al., 1992; Packard and Knowlton, 2002; Poldrack and Packard, 2003; Ullman, 2004). This competition is expressed in a variety of ways, including in the enhancement of one system following the dysfunction of the other. Thus procedural system abnormalities could actually enhance declarative memory function.

It is important to emphasize, however, that – unlike abnormalities of the procedural system – a spared or enhanced declarative memory is *not* a *sine qua non* of PLD. That is, declarative memory could in principle be abnormal in PLD, perhaps due to the same factors that directly affect the procedural system brain structures, or perhaps as a result of abnormal development thanks to the close connections between the declarative and procedural systems (Ullman, 2004). Nevertheless, based on the arguments presented just above, we hypothesize here that declarative memory generally remains largely intact in PLD.

In sum, the Procedural Deficit Hypothesis posits that many cases of SLI, which can be grouped together under the PLD umbrella, can be explained by abnormalities of brain structures underlying the procedural memory system. Moreover, we expect declarative memory to be largely spared. So, if PLD is at all specific, it is specific to the procedural memory system. It is certainly *not* specific to language, and does *not* affect all aspects of language. Thus, at least for those cases of SLI that can be grouped under PLD, we believe that the term "Specific Language Impairment" does not accurately represent the nature of the disorder.

We should emphasize that we do *not* claim that *all* subgroups and individuals identified as SLI (or developmental dysphasia and related language disorders) are afflicted with a dysfunction of the procedural system. That is, not all individuals with SLI are expected to have PLD. Given the broad definition of SLI, and the exclusionary nature of the definition, that would be far too strong a claim. Nevertheless, we do hypothesize that many if not most individuals and subgroups with developmental language impairment can be accounted for by the PDH; that is, they can be classified as PLD. As will be seen below, we posit that the individuals and subgroups that are encompassed by PLD range from those with apparently pure linguistic deficits (Hill, 1998; van der Lely and Stollwerck, 1996) to those with clear accompanying motor and other non-linguistic deficits (Bishop, 2002; Hill, 1998; Leonard, 1998; Goorhuis-Brouwer and Wijnberg-Williams, 1996), including the well-studied KE family (Fisher et al., 1998; Hurst et al., 1990; Lai et al., 2001; Ullman and Gopnik, 1999; Vargha-Khadem et al., 1995, 1998). Thus the PDH provides an explanatory framework that may lead to a useful classification process.

The PDH both overlaps with and differs from the two main classes of competing explanatory hypotheses. On the one hand, the PDH is similar to perspectives that argue for a grammar-specific deficit, in that it acknowledges the claim that grammar itself is directly affected. However, the PDH diverges from this view in its assertion that the impairment extends not only to all domains of grammar, but beyond grammar to lexical retrieval and to particular non-linguistic functions. On the other hand, like processing-deficit hypotheses, the PDH acknowledges certain non-linguistic deficits that affect processing, including those of working memory and rapid temporal processing. However, unlike those hypotheses, the PDH argues that these impairments are reflections of an underlying dysfunction which also leads directly to other impairments, including of grammar, lexical retrieval and motor functions.

Finally, the PDH differs from *both* competing perspectives in at least three important ways. First, the PDH is highly predictive, in that it provides the means to make novel predictions about SLI on the basis of *independent* sources of knowledge. That is, the types of neural and functional deficits that are expected in SLI can be predicted on the basis of what is independently known about the procedural memory system. Second, the PDH purports to explain, within a single theoretical framework, a large amount of the SLI data, both within and across subjects. Unlike other perspectives, it makes claims about broad patterns of the impaired and spared development of aspects of both brain and behavior, for a substantial set of cognitive functions. Moreover, unlike other hypotheses, it may explain much of the heterogeneity of SLI. Third, the PDH is essentially a hypothesis about both the brain and behavior, rather than focusing largely on linguistic or cognitive functions. Given that brain abnormalities necessarily underlie SLI, we believe that any theory of the disorder must seriously consider not only behavior, but also its underlying neural correlates.

Empirical Evidence

Studies of the Neural Correlates of SLI

Anatomical Studies

Converging evidence from structural neuroimaging, metabolic neuroimaging, post-mortem brain examination, and functional neuroimaging indicates the following. Despite the wide range of neural regions examined across studies, and the likelihood of etiological heterogeneity in the SLI populations across these studies, there appears to be consistent evidence that SLI is strongly associated with abnormalities of at least two structures: frontal cortex and the basal ganglia. To our knowledge, every study that has explicitly examined frontal regions has reported frontal abnormalities (Clark and Plante, 1998; Cohen et al., 1989; Denays et al., 1989; Gallagher and Watkin, 1997; Gauger et al., 1997; Jernigan et al., 1991; Kabani et al., 1997) (KE family: Liegeois et al., 2002; Vargha-Khadem et al., 1998). This seems to hold especially for inferior frontal regions (Clark and Plante, 1998; Cohen et al., 1989; Gauger et al., 1997; Jernigan et al., 1991; Lou et al., 1984) (KE family: Liegeois et al., 2002; Vargha-Khadem et al., 1998), in particular for Broca's area (Gauger et al., 1997; Lou et al., 1984) (KE family: Liegeois et al., 2002; Vargha-Khadem et al., 1998). In addition pre-motor frontal regions, including SMA, have been implicated (KE family: Belton et al., 2002; Vargha-Khadem et al., 1998; Watkins et al., 2002). Similarly, all studies that we are aware of that have examined the basal ganglia in developmental language impairment have reported abnormalities of these structures (Jernigan et al., 1991; Tallal et al., 1994) (KE family: Liegeois et al., 2002; Vargha-Khadem et al., 1998; Watkins et al., 1999). The caudate nucleus is particularly likely to be affected (Jernigan et al., 1991; Tallal et al., 1994) (KE family: Vargha-Khadem et al., 1998; Watkins et al., 1999). There is also some indication of an association between SLI and abnormal cerebellar structures (Oki et al., 1999; but see Cohen et al., 1989) (KE family: Belton et al., 2002; Watkins et al., 1999). Finally, studies have reported abnormalities or atypical (a)symmetries in inferior parietal and superior temporal regions (Cohen et al., 1989; Gauger et al., 1997; Jernigan et al., 1991; Lou et al., 1984) (KE family: Belton et al., 2002; Liegeois et al., 2002), as well as in perisylvian regions more generally (Jernigan et al., 1991; Plante, 1991; Plante et al., 1991). See Table I for detailed descriptions of all studies that we are aware of that have examined regional differences in the neural substrates of individuals with developmental language disorders.

As we have outlined above, the PDH predicts that frontal regions (especially Broca's area) and the basal ganglia (especially the caudate nucleus) should be particularly affected in SLI. This prediction appears to be confirmed by the empirical evidence. Moreover, the same brain regions are implicated in different sets of individuals with developmental language impairments, including in affected members of the KE family, whose impairments have been linked to *FOXP2* mutations (Lai et al., 2001). This lends credence to our claim that the impairments of the KE family (and presumably other individuals with similar *FOXP2* mutations) can also be explained by the PDH.

The abnormalities in posterior perisylvian regions, including the planum temporale, are intriguing. (See the second to last column of Table I). It is not clear whether these are directly related to the frontal/basal-ganglia abnormalities, either through connectivity or compensation, or whether they have an independent origin. One possible explanation is that at least some of the abnormalities in this region reflect the posited compensatory shift from the dysfunctional procedural memory system to the declarative memory system. Such a shift might reasonably be expected to result in anatomical or functional changes in the temporal/temporo-parietal structures underlying declarative memory, either because of the increased use of this system, or due to competitive interactions between the two systems (see above). This may explain findings that in developmental language impairment these structures show atypical asymmetry (Cohen et al., 1989; Gauger et al., 1997; Jernigan et al., 1991), in particular due to volume increases in the right hemisphere (Plante, 1991; Plante et al., 1991). It might also explain changes in the amount of gray matter or in the gray matter density in this region (Belton et al., 2002; Vargha-Khadem et al., 1998; Watkins et al., 2002). However, these and other types of abnormalities in these regions, such as hypoperfusion (Denays et al., 1989; Lou et al., 1984), or changes in the level of functional activity (Liegeois et al., 2003; Vargha-Khadem et al., 1998), are quite difficult to interpret, and future research is clearly needed to examine this issue.

Event-Related Potential Studies

Event-Related Potentials (ERPs) reflect the real-time electrophysiological brain activity of cognitive processes that are time-locked to the presentation of target stimuli. The few ERP studies of subjects with developmental language impairments have revealed the following pattern. Function words, which are critical for grammatical processing, have been shown to elicit a left-lateralized negativity in normally developing children, but a more bilateral and even somewhat right-lateralized negativity in language-impaired children with syntactic impairments (Neville et al., 1993). The distribution of this latter negativity was quite similar (even in the same subjects) to that of the N400, which we

TABLE I

Regional Brain Abnormalities in Neuroanatomical Studies of Developmental Language Impairment

Reference	Subjects	Method	Abnormalities of SLI brain structures			
			Frontal cortex and the insula	Basal ganglia and diencephalon/thalamus	Posterior cortex: temporal, parietal and occipital lobes	Cerebellum
STRUCTURAL/RESTING ACTIVATION STUDIES						
Lou et al., 1984	3 dysphasic children with verbal dyspraxia, and 9 normal controls	SPECT with analyses of rCBF at rest	"frontal lobe anomalies"	Not Reported	"posterior left perisylvian hypoperfusion" in one dysphasic	Not Reported
	4 dysphasic children with phonologic-syntactic deficits, and 9 normal controls		Hypoperfusion symmetrically in anterior perisylvian regions	3 dysphasics (with co-morbid ADHD) showed hypoperfusion of the "caudate nuclei region"	Hypoperfusion symmetrically in posterior perisylvian regions	Not Reported
Cohen et al., 1989	7-year-old girl with developmental dysphasia, with co-morbid ADHD	Postmortem analysis	Dysplastic microgyrus (additional small gyrus) on the inferior surface of the left frontal cortex along the sylvian fissure, due to abnormal neuronal migration	Hypoxic neuronal shrinkage in the basal ganglia, attributed to the acute mononucleosis infection causing the death of the child	Symmetrical planum temporale	Normal cerebellum
Denays et al., 1989	12 children with global dysphasia (expressive and comprehension deficits)	SPECT	Hypoperfusion in "upper and middle areas of the right frontal lobe" for 9 of 12 dysphasic children	Not Reported	Hypoperfusion in the left tempoparietal region for 9 of 12 dysphasic children	Not Reported
	2 children with expressive dysphasia		Hypoperfusion in the inferior frontal convolution of the left hemisphere, involving Broca's area	Not Reported	Not Reported	Not Reported
Jernigan et al., 1991	20 language-impaired children; 12 age-matched normal control subjects	Structural MRI, with volumetric analyses of regions of interest	• "Inferior anterior frontal cortical" region (defined as inferior to the plane above the frontal operculum, including orbitofrontal, dorsolateral and mesial frontal) volume reduction on the left relative to the right, as compared to controls, due to a reduction of structures in the left • Region including inferior posterior frontal cortex (with frontal operculum), insula and anterior temporal cortex reduced in the left (approaching significance; $p = .07$), but not the right. • No LI/control volume difference in region containing the frontal lobes above the frontal operculum	• Bilateral reduction in the volume of the caudate nucleus • Reduction of diencephalic gray matter (i.e., which is primarily the thalamus) on the right side • Lenticular regions normal bilaterally	• Parietal region (all cortex above but not including parietal operculum) larger in right than left in control children, but not in LI, who show a trend in the opposite direction • Reduced volume bilaterally of inferior posterior region that includes perisylvian parietal cortex, the parietal operculum, and most of the temporal lobe, with more LI than control subjects showing larger right than left volumes • Occipital region reduced volume in right but not left	Not Reported
Plante et al., 1991	8 boys with SLI, and 8 male control subjects	Structural MRI, with volumetric analyses or regions of interest	Not Reported	Not Reported	Abnormal asymmetry of the perisylvian region (defined to include the frontal and parietal operculae, the STS and the planum temporale), with the right but not left region larger than in the controls	Not Reported

(continued in the next page)

TABLE I

Regional Brain Abnormalities in Neuroanatomical Studies of Developmental Language Impairment

Reference	Subjects	Method	Abnormalities of SLI brain structures			
			Frontal cortex and the insula	Basal ganglia and diencephalon/thalamus	Posterior cortex: temporal, parietal and occipital lobes	Cerebellum
Plante, 1991	• Parents and siblings of 4 males with SLI, from 4 different families; most of the parents and siblings also had developmental language impairments • 19 control subjects without any history of language impairment	Structural MRI, with volumetric analyses focusing on the perisylvian region (defined as in Plante et al., 1991)	Not Reported	Not Reported	• Abnormal asymmetry of the perisylvian region (mostly R > L or R = L) in the family members, as compared to the controls (mostly L < R) • As compared to the controls, the family members showed increased volume in both hemispheres, but particularly in the right	Not Reported
Tallal et al., 1994	10-year old boy with history of delayed speech and language development	Structural MRI with visual inspection of the scans	Not Reported	Bilateral damage to the head of the caudate nucleus	Not Reported	Not Reported
Jackson and Plante, 1996	10 children with developmental language disorder, their parents (n = 20) and their siblings (n = 10); control group of 20 adult subjects with no history of language impairment	Structural MRI, with visual evaluation of gyral morphology in the posterior Sylvian fissure	Not Reported	Not Reported	Higher frequency of atypical type Sylvian fissure morphology families associated with developmental language disorder	Not Reported
Gallagher and Watkin, 1997	Four fetuses, one with a positive history of Familial Language Impairment, and the other three without any such history	Three dimensional ultrasonic fetal neuroimaging, with regions of interest analyses; scanning at 24, 28 and 32 weeks gestational age	Growth of the left inferior anterior region (frontal BA 45, 47, 11, 12, 10, anterior 44 and inferior 46, plus the temporal pole) and left inferior middle region (posterior BA44 extending back through the supramarginal gyrus, and down through the temporal lobe) occurred later for the fetus with a history of familial impairment than for the control fetuses	Not Reported	No significant differences found in the growth of posterior regions	Not Reported
Gauger et al., 1997	11 children with SLI; 19 age- and sex-matched controls	Structural MRI, with analyses of cortical surface area	Left pars triangularis (BA 45) smaller in SLI than controls	Not Reported	No SLI/control difference in the size of the planum temporale or the PAR, though the combination of the two yielded a greater rightward asymmetry in SLI as compared to controls	Not Reported
Kabani et al., 1997	6 children and 5 adults with Familial Language Impairment. 53 normal controls for the adults. No control children	Structural MRI, with volumetric analyses	• In the adults, visual inspection suggested atrophy, particularly in "the anterior region of the brain including the frontal and temporal lobes" • No regional comparisons reported for the children	Not Reported	Not Reported	Not Reported

(continued in the next page)

TABLE I

Regional Brain Abnormalities in Neuroanatomical Studies of Developmental Language Impairment

Reference	Subjects	Method	Abnormalities of SLI brain structures			
			Frontal cortex and the insula	Basal ganglia and diencephalon/thalamus	Posterior cortex: temporal, parietal and occipital lobes	Cerebellum
Clark and Plante, 1998	20 parents of developmentally language-disordered children; 21 controls, with no known history of language impairment	Structural MRI	Brain morphology types that included an extra sulcus in the inferior frontal gyrus were associated with behavioral classification of subjects as language-impaired, but not with a positive family history for language disorders	Not Reported	Not Reported	Not Reported
Vargha-Khadem et al., 1998	10 affected members of the KE family; 7 unaffected family members as controls	Structural MRI, with volumetric analyses	• Reduced gray matter in Broca's left area (BA 45 and BA 9/44) and left preSMA/cingulate cortex • Increased gray matter in left anterior insular cortex and right sensorimotor cortex (BA 4/3)	• Bilaterally reduced volume of the head of the caudate nucleus • Increased volume of lentiform nucleus (putamen) and the posterior thalamus	Bilaterally increased gray matter in the posterior temporal cortex (STG/STS), angular gyrus	Not Reported
Oki et al., 1999	Boy with developmental dysphasia	SPECT scanning performed twice, at 4 and 9 years old	Not Reported	Not Reported	Not Reported	• Age 4 years: Low cerebellar activation, particularly in the vermis • Age 9 years: Increase in cerebellar activation, concurrent with significant improvement in expressive language skills and verbal intelligence
Leonard et al., 2002	21 Children with specific language impairment and 14 children with reading disabilities. Comparisons with normally developing control children were not reported	Structural MRI, with measures of cerebral volume and asymmetry, focusing on Heschl's gyri, planum temporale and parietale, and anterior cerebellum	Not Reported	Not Reported	The children with SLI had a smaller left Heschl's gyrus than the reading disabled children. The planum temporale were asymmetric in the reading disabled children (L > R), but symmetric in the children with SLI	• No cerebellar differences were reported between SLI and reading disabled children
Belton et al., 2002	10 affected KE family members compared to 7 unaffected members and 17 sex- and age-matched unrelated control subjects	Structural MRI, with morphometric bilateral conjunction analyses	Bilaterally reduced gray matter density in the dorsal inferior frontal gyri (BA 45) and precentral gyri (BA 6/4; lower premotor/motor cortex)	Bilaterally reduced gray matter density in the head of the caudate nucleus	• Bilaterally reduced gray matter density of temporal poles • Bilaterally increased gray matter density in the posterior superior temporal gyrus (BA 22) and the angular gyrus	Bilaterally reduced gray matter density in the cerebellum (ventral cerebellar lobules VIIB and VIIIB)
Watkins et al., 2002	10 affected KE family members and 17 sex- and age-matched unrelated control subjects	Structural MRI, with voxel-based volumetric analyses	• Less gray matter bilaterally in sensorimotor cortex (BA 4/3) • More gray matter in the left inferior frontal gyrus (BA 45), and bilaterally in the precentral	• Less gray matter bilaterally in the head of the caudate nucleus • More gray matter bilaterally in the putamen	• Less gray matter bilaterally in the inferior temporal gyrus (BA 37) • More gray matter in medial occipito-parietal	• Less gray matter bilaterally in the posterior lobe of the cerebellum (lobule VIIIB) • More gray matter in the

(continued in the next page)

TABLE I

Regional Brain Abnormalities in Neuroanatomical Studies of Developmental Language Impairment

Reference	Subjects	Method	Abnormalities of SLI brain structures			
			Frontal cortex and the insula	Basal ganglia and diencephalon/thalamus	Posterior cortex: temporal, parietal and occipital lobes	Cerebellum
			gyrus (BA 4) and the anterior insular cortex		cortex (BA 19/7)	right posterior lobe of the cerebellum (lobule VI)
	10 affected and 7 unaffected members of the KE family		• Less gray matter in the left inferior frontal gyrus (BA 9/44 and BA 46) and left SMA (BA 6) • More gray matter in the left inferior frontal gyrus (BA 45), right sensorimotor cortex (BA 4/3), and left anterior insular cortex	• Less gray matter bilaterally in the head of the caudate nucleus • More gray matter bilaterally in the putamen, and in the tail of the right caudate nucleus	More gray matter bilaterally in the superior temporal gyrus (BA 22)	No significant differences found in cerebellar regions
			FUNCTIONAL STUDIES			
Lou et al., 1984	3 dysphasic children with verbal dyspraxia	SPECT. Comparison of rCBF during object naming as compared to rest	Object naming "failed to increase the perfusion of Broca's area" in the dysphasics, unlike in the controls	Not Reported	Not Reported	Not Reported
	4 dysphasic children with phonologic-syntactic deficits		Not Reported	Not Reported	Object naming in one dysphasic child did not yield increased left perisylvian perfusion, unlike in the controls	Not Reported
Vargha-Khadem et al., 1998	2 affected members of the KE family; 4 normal control subjects	PET with H_20^{15}. Experimental condition: Repeating aurally-presented words Baseline condition: Say one specified word repeatedly while listening to reversed words	• Underactivation in left SMA/preSMA/cingulate cortex left sensorimotor cortex (BA 4/3) • Overactivity in Broca's area (BA 44), left ventral prefrontal cortex (BA 47/45) • Overactivity in left premotor cortex (BA 6/8/9)	Overactivation in the head and tail of the left caudate nucleus	• Underactivation in left posterior middle temporal cortex (very posterior BA 21) • Overactivation of left angular gyrus (BA 39/19)	Not Reported
Liegeois et al., 2002	5 affected and 5 unaffected members of the KE family	FMRI Task: covert (silent) generation of verbs in reponse to hearing nouns	• Underactivation in left and right inferior frontal gyrus (BA 44/45) • Underactivation in upper part of left precentral gyrus (BA 4) • Overactivation in upper left and right postcentral gyrus (BA 3) • Overactivation in left and right precentral gyrus (BA 4)	Underactivation in the right putamen/globus pallidus	• Underactivation in left supramarginal gyrus (BA 40) • Overactivation in left and right posterior superior temporal gyrus and middle temporal gyrus (BA 22 and 22/39) • Overactivation in right temporal pole (BA 38)	Not reported
		FMRI Task: overt generation and repetition of verbs	• Underactivation in left inferior frontal cortex (BA 45) • Underactivation in upper part of left precentral gyrus (BA 4/6) • Overactivation in left anterior insula	Underactivation in left and right putamen	No significant difference in activation reported between affected family members and controls	Not Reported

LI: Language Impaired; PAR: Posterior Ascending Ramus (sulcus in the supramarginal gyrus); RCBF: regional cerebral blood flow; STG: superior temporal gyrus; STS: superior temporal sulcus

have previously claimed reflects lexical/declarative memory processes (Ullman, 2001b, 2004). These data are consistent with dysfunctional grammatical processing, and a compensatory shift to lexical/declarative memory – although exactly how the use of function words might increase their dependence on lexical/declarative memory requires further investigation. In contrast, content words, which are critical for conveying conceptual meaning, elicited the *same* type of waveform – that is, N400s – in children with SLI and control subjects (Neville et al., 1993). This strengthens the view that lexical/declarative memory remains intact in SLI. Intriguingly, this SLI pattern of grammatical contexts failing to elicit left anterior negativities (which instead are displayed as N400-like effects), whereas lexical/conceptual contexts elicit intact N400s, is similar to the pattern shown by late second language learners, who also appear to suffer from a dysfunctional grammatical/procedural system (Ullman, 2001b). Finally, it is noteworthy that the N400s elicited by second language learners and by individuals with SLI differ slightly from those elicited by their respective control subjects. Thus second language learners show a somewhat later and smaller amplitude N400 than first language speakers (Hahne, 2001; Ullman, 2001b; Weber-Fox and Neville, 1996), while the amplitudes of the N400s are larger in children with SLI and in their parents (especially their fathers) than in normal control subjects (Neville et al., 1993; Ors et al., 2001). The significance of these differences is still not clear, and invites further investigation.

Behavioral Evidence from SLI

Studies of Language in SLI

The PDH predicts a specific pattern of impaired and spared language functions. Aspects of grammar that depend on the procedural system, in particular those that involve rule-governed combinatorial operations, should be abnormal. Impairments are predicted to be found across domains of grammar, including in aspects of syntax, in regular morphology, and in phonology – especially with the use of novel (as opposed to existing) words, whose phonological sequences have not been memorized, and thus are expected to be combined (Ullman, 2004). In contrast, linguistic knowledge that is normally stored in the lexicon – i.e., that which is idiosyncratic – need not be impaired, and in fact may develop normally. Lexical knowledge should remain largely intact not only for simple words (e.g., *cat*), but also for lexicalized grammatical representations, such as memorized knowledge about argument structure and irregular morphological forms. The sparing of lexical knowledge should be particularly evident in tasks which do not depend on the brain structures of the procedural system. Thus normal performance is expected on receptive lexical tasks. Finally, individuals with SLI could compensate for their grammatical/procedural deficit by increasing their reliance on lexical/declarative memory, especially in the use of complex linguistic representations that could depend on either system.

The Grammatical Profile of SLI

Syntax. Numerous studies have reported syntactic impairments in SLI, in both expressive and receptive language tasks, across languages. (See morphology subsection below for a discussion of morpho-syntax.) First of all, children with SLI show a variety of syntactic deficits in production tasks (Leonard, 1995, 1998; Clahsen et al., 1997; Hansson and Nettelbladt, 1995; Johnston and Kamhi, 1984; Rothweiler and Clahsen, 1993; Thordardottir and Weismer, 2002; van der Lely, 2003). For example, children with SLI have been claimed to have difficulty following appropriate word order patterns (Hansson and Nettelbladt, 1995), using adjuncts (Leonard, 1998; Johnston and Kamhi, 1984), producing wh-questions (van der Lely and Battell, 2003), and representing grammatical elements that depend on functional categories (Leonard, 1995).

Children and adults with SLI also show abnormal patterns in receptive tasks probing syntax. In comprehension tasks, children with SLI have difficulty assigning thematic roles in passive sentences (van der Lely, 1994) and assigning reference to pronouns or reflexives (van der Lely, 1996). Children with SLI also have trouble judging the syntactic acceptability of sentences (Kamhi and Koenig, 1985; Liles et al., 1977; Rice et al., 1999). Importantly, some studies have documented that children with SLI have more difficulty, compared to controls, at identifying errors of syntax (including morpho-syntax), such as of word order and syntactic agreement, than those which violate conceptual semantics (e.g., "The dog writes the food") (Kamhi and Koenig, 1985; Liles et al., 1977). In fact, Kamhi and Koenig (1985) reported that children with SLI performed significantly worse than controls in identifying and correcting syntactic errors, but performed *normally* at identifying and correcting not only conceptual semantic violations (see example above), but also violations of the phonological representations of real words (e.g., "John has two tig cars" vs. "John has two big cars"). Consistent with the PDH, this suggests the impairment of the grammatical functions posited to depend on the procedural system, but not of conceptual knowledge and stored lexical knowledge posited to depend on declarative memory.

Importantly, not all aspects of syntax are impaired. In particular, syntactic knowledge which

is likely to be lexicalized – that is, stored in lexical memory – appears to be relatively spared in SLI. Thus it has been observed that children with SLI tend to rely disproportionately on high frequency phrases (i.e., those that are often encountered in the language) (Thordardottir and Weismer, 2002; Whitehurst et al., 1972), which are particularly likely to be memorized. Further, the use of argument structure, which depends heavily on memorized knowledge, is generally correct in the speech of children with SLI (Leonard, 1998; Rice and Bode, 1993; Thordardottir and Weismer, 2002). Intriguingly, in one study children with SLI were able to use a verb with its appropriate arguments after they *observed* events for which the verb was suitable (van der Lely, 1994). However, when the children in this same study were tested on verb argument structure learning *without* this visual information about the verb's meaning, and only heard the word in a sentence context, they performed more poorly than the control children. This suggests that they had difficulty selecting argument structure from a syntactic frame. More generally, the results imply that learning aspects of language by using one's knowledge of syntax (i.e., syntactic bootstrapping) may be particularly difficult for children with SLI, whereas they can learn the same knowledge with the help of non-linguistic semantic contexts, suggesting a dependence on declarative memory.

Morphology. SLI is strongly associated with impairments of morphology: both of morpho-syntax and of regular morpho-phonology. Children and adults with SLI have been shown to be abnormal in their production of complex words in both verbal and nominal inflectional morphology, including past tense formation (Leonard et al., 1992a; Norbury et al., 2001; Oetting and Horohov, 1997; Ullman and Gopnik, 1994, 1999; van der Lely and Ullman, 2001), agreement (Bortolini et al., 2002; Gopnik and Crago, 1991; Rice and Oetting, 1993; Rothweiler and Clahsen, 1993), pluralization (Clahsen, 1995; Goad and Rebellati, 1994; Gopnik and Crago, 1991), and morphological case (Wexler et al., 1998b). In addition to their difficulties producing inflected forms, people with SLI deviate from controls in compounding (van der Lely and Christian, 2000) and derivational morphology (Gopnik and Crago, 1991). Although most of this research has focused on English, morphological deficits have also been found in German (Clahsen, 1995; Rothweiler and Clahsen, 1993), Italian (Bortolini et al., 2002; Leonard et al., 1992a), Hebrew (Dromi et al., 1993), Japanese (Fukuda and Gopnik, 1994; Fukuda and Fukuda, 1999, 2001), Inuktitut (Crago and Allen, 1994, 1996), Swedish (Hansson and Nettelbladt, 1995), Finnish (Niemi, 1999), Dutch (Wexler et al., 1998a) and Greek (Dalalakis, 1994). These data clearly show that SLI is strongly associated with impairments of morphology, independent of whether the deficits are of morpho-syntax and/or morpho-phonology.

Some studies directly implicate regular morpho-phonology. English-speaking children and adults with SLI do not show the normal pattern of producing regular past-tense forms more accurately than irregular past-tense forms (Gopnik and Crago, 1991; Leonard et al., 1992b; Oetting and Horohov, 1997; Rice et al., 1995; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001); they have particular difficulty producing regular past-tenses of novel verbs (e.g., *plag-plagged*), with less difficulty producing irregularizations of novel verbs (e.g., *crive-crove*) (which are posited to depend on lexical memory, Pinker and Ullman, 2002b; Ullman, 2001a) (Ullman and Gopnik, 1999; van der Lely and Ullman, 2001); and they over-regularize past tense forms (e.g., *swimmed*) less than control subjects (Leonard, 1989; Marchman et al., 1999; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001). Similarly, abnormally low over-regularization rates in SLI have been shown in German pluralization (Clahsen et al., 1993), and in the production of complex forms in Japanese (Fukuda and Fukuda, 2001).

These deficits suggest that the use of regular morphologically complex forms, which tends to rely on the procedural system (Ullman, 2001c; Ullman et al., 1997), is impaired relative to the use of morphological forms hypothesized to depend on lexical memory. Importantly, although the production of regular past-tense forms is particularly impaired (thus implicating regular morpho-phonology), both regulars *and* irregulars are produced less successfully by language-impaired children and adults than by control subjects (Ullman and Gopnik, 1994, 1999; van der Lely and Ullman, 2001; Vargha-Khadem et al., 1995). This can be explained by impairments of morpho-syntax, which would affect both past-tense types (Ullman and Gopnik, 1999). However, the pattern could also (in addition or instead) be accounted for by lexical retrieval deficits, which would depress performance at the production of irregulars, and also of regulars if these are memorized as a compensatory strategy (see below). Consistent with this latter view, the judgment of both past-tense types, unlike their production, has been shown to be spared (Gopnik, 1994; Ullman and Gopnik, 1999; van der Lely and Ullman, 1996).

Some investigations of morphology in SLI have employed "language-learning" tasks. These studies use invented morphemes to assess whether the morphological difficulties are be caused by an inability to induce grammatical rules on the basis of exposure to language stimuli. Participants are exposed to linguistic stimuli containing invented inflectional morphemes that are used according to specific rules of a made-up grammar. Subjects are subsequently tested on their comprehension and

production of these morphemes in appropriate contexts. Several such studies have shown that children with SLI do not generalize trained bound morphemes onto untrained words as readily as controls do (Anderson, 2001; Bellaire et al., 1994; Connell and Stone, 1992; Roseberry and Connell, 1991). One study employed a version of this task with a visual symbolic morpheme instead of spoken morphemes. As in the tasks with spoken morphemes, the SLI group generalized less than age-matched peers (Stone and Connell, 1993; but see Kiernan et al., 1997), suggesting a rule-induction dysfunction that extends across perceptual modalities.

Phonology. Children and adults with SLI also exhibit impairments of phonological processing. However, not all aspects of phonology are impaired. Phonological representations that are unlikely to be memorized, in particular novel forms, are especially difficult to process. In contrast, phonological representations that are likely to be stored in lexical memory, such as those of frequent words, are relatively spared. Several distinct lines of evidence demonstrate this pattern. First, the repetition of non-words, as compared to real words, is a notoriously difficult task for children with SLI (Montgomery, 1995b; Bishop et al., 1996; Botting and Conti-Ramsden, 2001; Edwards and Lahey, 1998; Gathercole and Baddeley, 1993; Weismer et al., 2000). In fact, this pattern, which suggests phonological processing difficulties and/or deficits of phonological working memory (Montgomery, 1995a; Edwards and Lahey, 1998; Gathercole and Baddeley, 1990; Kamhi and Catts, 1986), has even been claimed to be a behavioral indicator or diagnostic marker for language impairment (Gray, 2003; Bishop et al., 1996; Conti-Ramsden et al., 2001). Second, a study of the KE family documented that the affected subjects acquired the phonological inventory of English at an extremely delayed rate, and never achieved the competence of adults at reproducing complex syllable patterns (Fee, 1995). Third, a study of a well-documented phonological operation in Japanese compounding, *rendaku*, suggested that Japanese SLI subjects did not apply the appropriate voicing to novel compounds as readily as controls (Fukuda and Fukuda, 1999). The authors argue that instead of using a productive "procedural" voicing rule, the SLI subjects relied on metalinguistic declarative knowledge (searching for stored forms) to produce a few of the most frequent compounds. Finally, several investigators have argued that the phonological system of children with specific language impairment may not be as highly adaptive as that of normally developing children (Leonard, 1989; Kamhi and Catts, 1986; Kamhi et al., 1985): children with SLI have difficulty using the phonetic properties of a word to categorize, differentiate and generalize among words and their parts.

Some investigators have argued that deficits related to phonological processing may be responsible for the higher-level morphological and syntactic impairments found in children with SLI (Joanisse and Seidenberg, 1998, 2003; McGregor and Leonard, 1994). While we do not deny the possibility that phonological impairments (from deficits of phonological processing and/or of phonological working memory) may contribute to such higher level impairments, we maintain that such a causal relationship between linguistic levels is not necessary. Rather, we suggest that impairments of syntax, morphology and phonology are largely a direct consequence of an underlying dysfunction of the procedural memory system.

A compensatory shift between systems. Evidence suggests that children and adults with SLI rely on lexical/declarative memory for processing at least some types of complex forms that tend to be computed by the grammatical/procedural system in unimpaired individuals. In particular, people with SLI can use declarative memory either to memorize complex forms (Gopnik and Crago, 1991; Ullman and Gopnik, 1994, 1999; van der Lely and Ullman, 2001) and/or to learn and use explicit rules (Paradis and Gopnik, 1997; Ullman and Gopnik, 1994, 1999).

Several lines of evidence support the claim that individuals with SLI memorize complex forms as a compensatory strategy. First, as discussed above, children with SLI rely disproportionately on high frequency phrases, suggesting that they are relying heavily on phrases that are memorized rather than composed. Second, whereas control subjects produce regular past-tenses more accurately than irregular past-tenses, children and adults with SLI show equivalent performance on the two verb types, controlling for factors such as frequency, in both expressive (Gopnik and Crago, 1991; Leonard et al., 1992b; Oetting and Horohov, 1997; Rice et al., 1995; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001) and receptive (Gopnik, 1994; Ullman and Gopnik, 1999; van der Lely and Ullman, 1996) language tasks. This suggests that the two past-tense types are processed similarly in SLI; i.e., both are stored. Third, as discussed above, it has been shown that people with SLI are impaired at producing, but not at judging, both types of past-tense forms (Gopnik, 1994; Ullman and Gopnik, 1999; van der Lely and Ullman, 1996) (for a discussion, see Ullman and Gopnik, 1999). This suggests that regular and irregular past tense forms may both be stored, given that retrieving both forms is problematic but recognizing them is not. Fourth, whereas in normally developing children and adults, frequency effects for regular inflected forms are absent, inconsistent or weak (Pinker, 1999; Ullman, 2001a, 2001c), they are consistently demonstrated in children and adults with SLI, both in past-tense production (Oetting and Horohov, 1997; Ullman and Gopnik, 1994,

1999; van der Lely and Ullman, 2001) and in plural production (Oetting and Rice, 1993). Fifth, van der Lely and Christian (2000) found that children with SLI produced compounds with regular as well as irregular plurals inside them (e.g., *rats-eater* and *mice-eater*). In contrast, normal children only produce compounds with irregular plurals (e.g., *mice-eater* vs. *rat-eater*) (Gordon, 1986; van der Lely and Christian, 2000). This suggest that whereas normal children mainly retrieve irregular past-tense forms from memory (Gordon, 1986; Pinker, 1999), children with SLI memorize both types of past-tense forms.

Individuals with developmental language impairments can also compensate for grammatical deficits by learning and using explicit rules, such as "add -ed to make a past tense form" (Paradis and Gopnik, 1994, 1997; Ullman and Gopnik, 1994, 1999). This view is supported by evidence showing that some language-impaired individuals in the KE family incorrectly appended suffix-like past-tense endings to verbs, as revealed by detailed phonetic analyses (Ullman and Gopnik, 1999). Errors included the use of an incorrect allomorph (e.g., *scour-scourt, spuff-spuffid*), the insertion of a pause between the verb and the suffix (*wring…d*), and the inappropriate stressing of the suffix. These errors were found on existing regulars, over-regularizations and novel verbs. No such errors occurred with irregular past-tense forms, even when they were phonologically similar to regular past-tense forms (e.g., *made*). Moreover, one subject reported that he explicitly learned this strategy: "at school [I] 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'." Some affected members of the KE family also make analogous errors on pluralization (Goad and Rebellati, 1994). Interestingly, such explicit rules are not learned by all language-impaired subjects, and not necessarily in all inflectional contexts. For example, one subject used such a strategy in plural but not past tense contexts (Ullman and Gopnik, 1999). Additionally, the explicit training of grammatical rules in children with SLI does not always guarantee that they will be able to productively generalize novel forms in all contexts (Swisher et al., 1995). Nevertheless, explicit rule-learning, including in the context of explicit training, does appear provide one method whereby individuals with developmental language impairments can compensate for grammatical deficits.

Both the memorization of complex forms as chunks and the learning and use of explicit rules can help to account for some of the language patterns observed in SLI. These two strategies, particularly in combination (Ullman and Gopnik, 1999), may explain findings that children with SLI can perform relatively normally at the production of some complex forms (Dromi et al., 1993; Leonard et al., 1992a; Oetting and Rice, 1993). Similarly, observations that SLI subjects produce over-regularizations, both in the past tense (Eyer and Leonard, 1995; Leonard et al., 1992a; Oetting and Horohov, 1997; van der Lely and Christian, 2000) and in pluralization (Rice and Oetting, 1993), could be explained not only by the use of explicit rules, but also by the associative-memory based generalization of patterns across stored regulars (Hartshorne and Ullman, submitted; Pinker, 1999; Ullman, 2001a). Importantly, the use of explicit rules and the memorization of complex forms can be empirically distinguished from each other and from grammatical/procedural rule-computation (see PDH section).

The Lexical Profile of SLI

According to the PDH, lexical/declarative memory is not likely to be impaired (see PDH section). However, this system does not operate in isolation. In particular, a number of functions and tasks that involve lexical/declarative memory also depend on the brain structures underlying the procedural system. These tasks and functions should therefore be at least somewhat impaired. Thus language-impaired individuals should have particular difficulty with word learning when meaning can be inferred only by analyzing a grammatical structure, when large amounts of information are required to be held in working memory, or when information is presented rapidly. These are conditions which presumably confront all children in the course of daily interaction, and therefore vocabulary acquisition may be vulnerable for language-impaired children in normal learning situations. However, these variables can be experimentally manipulated, and so the relative sparing of lexical/declarative memory can be empirically tested. Word learning should be quite easy when items are presented slowly and in a rich semantic context, facilitating memorization in declarative memory. Additionally, tasks that involve lexical retrieval or selection are predicted to be more impaired than receptive tasks like lexical comprehension or recognition. Performance at lexical retrieval tasks should be particularly impaired when rapid responses are expected, but should be considerably improved or even normal when ample time is given for subjects to respond. In sum, we argue that (apparent) lexical impairments will be explained not by problems with lexical/declarative memory itself, but rather by the predicted dysfunction of the procedural system, which can affect specific aspects of the learning and use of words.

Lexical organization. Evidence suggests that lexical organization, and in particular lexical-*semantic* organization, is similar in SLI and control children (Freedman and Carpenter, 1976; Kail et

al., 1984; Kirchner and Klatzky, 1985; Schwartz and Leonard, 1985). For example, it appears that children with SLI are not atypical in the type and variety of semantic relations used in their speech. In one study, investigators collected a corpus of two-word utterances from language-impaired children and from language age-matched normally-developing children, and categorized them as expressing certain semantic relations, independent of their grammatical accuracy (Freedman and Carpenter, 1976). The type-token ratio computed for each of the 10 semantic-relation classifications was not significantly different between the two groups for 9 of the 10 relations, and in fact the language-impaired group showed *better* diversity than the control children in the remaining category. Thus, the children with SLI used at least as wide a variety of semantic relations as the control children. These data suggest that important aspects of lexical/declarative memory remain relatively spared in SLI.

Word learning. Children with SLI are often quite capable of learning new words, and can even reach normal levels of performance (Whitehurst et al., 1972), especially under conditions which are expected to depend less on the brain structures underlying the procedural system. Thus word learning in SLI has been reported to be *un*impaired under conditions of focused or repetitive input, or when the child can obtain the meaning by direct observation of an object or action (Leonard, 1982; Dollaghan, 1987; Rice et al., 1994). Similarly, there is strong evidence that children with SLI can form new word-referent associations as readily as their age-matched peers, as long as they have adequate input – that is, that the input is either presented frequently or with strong contextual support (Dollaghan, 1987; Rice et al., 1994; Weismer and Hesketh, 1996). Indeed, children with SLI appear to rely disproportionately on high frequency words and phrases (Rice and Bode, 1993; Rice et al., 1994; Watkins et al., 1993; Whitehurst et al., 1972).

Not every study has reported normal word-learning. In two studies, Rice and colleagues found that children with SLI had lower mean comprehension scores than control children on tasks of quick incidental word learning (i.e., fast-mapping) tasks (Rice et al., 1990, 1992). However, the items in these experiments were presented with few repetitions, and with little contextual support other than grammatical cues. Moreover, they were presented in television programs, which are unlikely to provide as rich a context as naturalistic interactive scenarios. Indeed, Kiernan (1998) argues that word learning problems "may occur when children with SLI are exposed only briefly to target items as in fast-mapping paradigms … or are left much on their own to learn words from more impersonal media sources as in quick incidental-learning paradigms". In her own study, in which the target words were presented in a rich context, children with SLI did not demonstrate word learning impairments (Kiernan, 1998).

Expressive vs. receptive tasks. Tests of lexical abilities that involve word *retrieval*, such as object naming and word or list recall, are often impaired in SLI (Bishop, 1997; Fazio, 1998, 1999; Kamhi and Catts, 1986; Katz et al., 1992; McGregor and Appel, 2002; Weckerly et al., 2001). In contrast, tasks probing *receptive* lexical abilities, such as comprehension and especially recognition tasks, are often relatively spared (Clarke and Leonard, 1996; Dollaghan, 1987; Kiernan, 1998; Vargha-Khadem et al., 1995; Weismer and Hesketh, 1996). This pattern holds for investigations of vocabulary (Clarke and Leonard, 1996), for tasks probing the recognition of "gated" words (Montgomery, 1999), and in controlled studies of word learning (Dollaghan, 1987; Kiernan, 1998; Weismer and Hesketh, 1996). The pattern is found even when the same subjects and/or the same items are tested on both types of tasks (Dollaghan, 1987; Weismer and Hesketh, 1996). Performance at receptive tasks is sometimes completely normal, as compared to that of control children (Dollaghan, 1987; Montgomery, 1999; Weismer and Hesketh, 1996). Moreover, this lack of a difference in performance between SLI and control children does not appear to be attributable to ceiling effects (Dollaghan, 1987; Leonard, 1982; Weismer and Hesketh, 1996). In some cases SLI performance at word learning is even *superior* to that of vocabulary-matched control children (Leonard, 1982; Weismer and Hesketh, 1996). Even when the control children are *age-matched* to the SLI subjects, SLI performance on receptive lexical tasks can be identical (Dollaghan, 1987; Clarke and Leonard, 1996; Weismer and Hesketh, 1996).

Lexical retrieval deficits are particularly striking when the necessity of lexical look-up is combined with a rapid presentation rate of items (Menyuk, 1975; Rapin and Wilson, 1978; Weismer and Hesketh, 1996). For example, the children with SLI in one word-learning study were more impaired than vocabulary-matched control children only on the *production* of novel words that had been presented at a *fast* rate (Weismer and Hesketh, 1996). The SLI subjects' production of novel words that had been presented at a slow rate, as well as comprehension abilities for both types of word presentation conditions, were equivalent for SLI and control children. Moreover, these children with SLI demonstrated significantly *better* accuracy than the vocabulary-matched controls on word recognition for both types of word presentation conditions.

If lexical/declarative *retrieval* abilities really are a particular source of difficulty for children with SLI, we would expect that language-impaired subjects might rely more than control subjects on

external cues as recall strategies. Indeed, the subjects with SLI in one study were reported to benefit significantly from using hand gestures in order to remember poems, whereas cognitive- and language-matched controls recalled the same percentage of the poems across the conditions (i.e., with and without gesture cues) (Fazio, 1997). Similarly, children with SLI have been shown to utilize counting strategies to remember math facts (which are also expected to depend on declarative memory) significantly more than age- and language-matched controls (Fazio, 1999).

Even when SLI subjects' performance at tasks requiring lexical retrieval is as accurate as control subjects', it tends to be slower. Thus children and adults with SLI have been reported to name words as accurately as control subjects (Lahey and Edwards, 1996; Vargha-Khadem et al., 1995, 1998), even when the control subjects are age-matched (Leonard et al., 1983). However, in these cases either the responses were untimed, or, if they were timed, the SLI subjects took *longer* than the controls. These data suggest that in both experimental and naturalistic expressive language contexts, children with SLI are likely to be particularly slow at word-finding, even when they have intact lexical knowledge. Interestingly, fast automatic retrieval is also difficult for children with SLI in non-linguistic domains like counting numbers and reciting math facts (Fazio, 1996, 1999), suggesting a similar pattern for other types of information in declarative memory, as predicted by the PDH.

It is important to point out, however, that it has also been argued that word retrieval and word knowledge cannot be as easily dissociated as has often been assumed (McGregor et al., 2002). In studies that have probed the relation between lexical-semantic representation and naming (McGregor and Appel, 2002; McGregor et al., 2002), not only were children with SLI impaired at naming, but in addition there was a significant correlation, across items, between naming errors and an inability both to create accurate drawings of the words and to produce their definitions. It was argued that this correlation suggests that the naming impairment was attributable to missing and/or sparse semantic representations of words. However, the correlation between naming abilities and drawing or definition accuracy was *also* found in the normally developing group. Moreover, the children with SLI were found *not* to be atypical with respect to the types of semantic information that they drew or produced. Thus, the difficulty that the children with SLI displayed on the naming task could plausibly be attributed to problems acquiring the words in the first place. Future research of this type may shed light on these issues.

Nouns vs. verbs. Evidence suggests that children with SLI may have greater difficulty with verbs than nouns, as compared to control children (Rice and Bode, 1993; Rice et al., 1994; Watkins et al., 1993). Moreover, it has been well-documented that children with SLI rely on a highly restricted set of verbs, especially those of high frequency (Rice and Bode, 1993; Rice et al., 1994; Watkins et al., 1993; Whitehurst et al., 1972). This particular difficulty with verbs in SLI is analogous to the particular difficulty with verbs in patients with adult-onset lesions to structures underlying the procedural system, especially inferior frontal cortex, including Broca's area (Cappa and Perani, 2003; Gainotti, 1998; Marshall, 2003). Indeed, verb deficits are associated with agrammatic aphasia, which is linked to lesions of these frontal regions and the basal ganglia. The verb impairments in such adult-onset lesions may be due to the particular grammatical complexity of verbs, and/or the strong association of verbs with actions, and hence with procedural knowledge (Cappa and Perani, 2003; Gainotti, 1998; Marshall, 2003). The same explanations may hold for SLI. Indeed, it has also been claimed that difficulties with verbs in SLI may be related to their grammatical roles (Rice et al., 1994).

Studies of Non-Language Domains in SLI

Co-Occurring Non-Linguistic Deficits in SLI: Impairments of the Procedural Memory System

A range of studies has shown that many if not most individuals with SLI exhibit one or more non-linguistic deficits in addition to their language impairments (Bishop, 2002; Stromswold, 2000; Goorhuis-Brouwer and Wijnberg-Williams, 1996; Johnston and Ramstad, 1983; Stark and Tallal, 1988). (For some reviews of non-linguistic deficits in SLI, see Bishop, 1992; Hill, 2001; Leonard, 1998). Although in some cases non-linguistic deficits have not been found in individuals with SLI (van der Lely, 1993), it may be that in these cases not all deficits were probed for, or that the deficits are subtle and hard to detect (see discussion in the PDH section). Moreover, if, as we claim, there may be variation in the degree to which various linguistic and non-linguistic deficits co-occur in SLI, subjects with obvious non-linguistic impairments would likely be excluded from studies of SLI, despite a language profile that might be characteristic of the disorder. In this section we show that SLI is strongly associated with impairments of motor function (especially motor sequences), dynamic mental imagery, working memory, and rapid temporal processing (especially of sequences).

Motor impairments. Numerous studies have shown the existence of motor deficits in children and adults with SLI (for a wide-ranging review, see Hill, 2001). Moreover, these deficits appear to be of a type that would be expected from impairments

of procedural memory, or from a dysfunction of frontal/basal-ganglia circuits or the cerebellum — that is, impairments of sequencing, speed, timing, and balance.

Several studies have demonstrated impairments in children and adults with developmental language impairments on tests of oral or facial praxis (Alcock et al., 2000; Dewey and Wall, 1997; Katz et al., 1992; Noterdaeme et al., 2002; Vargha-Khadem et al., 1995; Wiznitzer et al., 1986). These impairments have been found in both speech *and* non-speech movements (Alcock et al., 2000; Dewey and Wall, 1997; Noterdaeme et al., 2002; Vargha-Khadem et al., 1995). Interestingly, studies of the affected members of the KE family have shown that the oromotor impairments are particularly severe for combinations or sequences of movements, as compared either to single movements or to accuracy in positioning of the tongue and lips (Alcock et al., 2000; Hurst et al., 1990; Vargha-Khadem et al., 1995). Moreover, rapid oral movements are particularly problematic for children with SLI (Tallal et al., 1985a).

Motor deficits in SLI are not, however, restricted to face and mouth movements. Numerous studies have documented that children with SLI exhibit deficits in various tests of fine and gross motor function (Hill, 1998; Goorhuis-Brouwer and Wijnberg-Williams, 1996; Gross-Tsur et al., 1996; Noterdaeme et al., 2002; Powell and Bishop, 1992; Wiznitzer et al., 1986), limb praxis and/or coordination (Bishop, 2002; Hill, 1998; Dewey and Wall, 1997; Powell and Bishop, 1992; Schwartz and Regan, 1996), and fine motor skills (Gross-Tsur et al., 1996; Schwartz and Regan, 1996; Trauner et al., 2000; Wiznitzer et al., 1986) such as cutting out a circle, copying shapes, and tracing a maze without crossing lines (Schwartz and Regan, 1996). Children with SLI have also been reported to perform more poorly than control subjects on tests of balance (Gross-Tsur et al., 1996; Noterdaeme et al., 2002; Powell and Bishop, 1992), which may depend especially upon the cerebellum (Ivry and Fiez, 2000). Note that Stark and Tallal (1981) did not observe balancing deficits in children with SLI. However, the length of the time that the children were asked to balance may not have been sufficient to differentiate between the SLI and control groups (see Powell and Bishop, 1992).

Tasks involving complex sequences of movements appear to be particularly difficult for language-impaired subjects. We have seen above that this holds for orofacial movements. In addition, children with SLI are impaired, compared to control children, on complex sequential motor tasks such as peg-moving (Bishop, 2002; Owen and McKinlay, 1997; Powell and Bishop, 1992; Preis et al., 1997; Wiznitzer et al., 1986), sequential finger opposition (Johnston et al., 1981; Katz et al., 1992; Wiznitzer et al., 1986), and stringing beads (Owen and McKinlay, 1997; Schwartz and Regan, 1996). Even less complex sequential motor tasks such as rapid finger-tapping (in which the same movements are being repeated successively) are often impaired (Bishop, 2002; Goorhuis-Brouwer and Wijnberg-Williams, 1996; Preis et al., 1997; but see Archer and Witelson, 1988).

Many (though not all) of the motor tasks that have been found to be impaired in SLI require speeded movements (see above). In fact, rate of motor performance may be an especially significant feature in distinguishing children with SLI from control children on motor tasks (Johnston et al., 1981). Moreover, and analogously to naming tasks (see above), children with SLI may be as accurate as control subjects on some motor tasks, but perform them significantly more slowly (Archer and Witelson, 1988).

The evidence presented above makes a strong case that SLI is associated with motor impairments, particularly in tasks involving rapid movements or complex sequential motor skills. This supports the PDH prediction that individuals with SLI have procedural system motor deficits. Moreover, the claim that an aberrant procedural system leads not only to these motor deficits, but also to language impairments, is strengthened by a study reporting a strong correlation between scores at auditory language comprehension, including of complex sentences, and performance on fine motor tasks (Schwartz and Regan, 1996).

It might be argued that SLI subjects with motor deficits actually suffer from an independent concomitant motor disorder, rather than from a common underlying abnormality. A study published by Hill, Bishop and Nimmo-Smith (1998) addressed this issue. The SLI subjects in this study were divided into two subgroups, based on their performance on the Movement ABC battery (Henderson and Sugden, 1992). Those SLI subjects who scored in the range necessary for classification as Developmental Coordination Disorder were placed into a "clumsy" SLI group, while the rest were categorized as having "pure" SLI. However, the two SLI groups performed almost identically on most of the motor praxis tests, and both groups performed significantly worse than controls on all of the motor praxis tests. Thus, even those children assumed to have a "pure" language disorder have been shown to exhibit subtle motor deficits as well, as predicted by the PDH. This finding, as well as those presented above, also strengthens the view that groups previously considered exceptional or non-SLI because of obvious motor impairments, such as the affected members of the KE family, may in fact display a non-atypical SLI profile. Indeed, one study by Bishop (2002) that examined motor deficits in SLI subjects included a heritability analysis which pointed to a possible shared genetic influence for motor and

speech/language development, consistent with the predictions of the PDH.

Impairments of dynamic mental imagery. Many studies have shown that SLI is associated with impairments of mental imagery (Inhelder, 1976; Johnston and Ramstad, 1983; Johnston and Weismer, 1983; Kamhi, 1981; Kamhi et al., 1984; Montgomery, 1993; Savich, 1984) (for a review, see Leonard, 1998). However, most if not all mental imagery tasks yielding impaired performance in SLI likely involve some sort of dynamic real-time mental manipulation of images, such as mental rotation, anticipatory imagery, or the outright processing of moving images (Inhelder, 1976; Johnston and Weismer, 1983; Kamhi, 1981; Kamhi et al., 1984; Savich, 1984). In contrast, children with SLI do *not* seem to experience the same difficulty in tasks involving the visual perception of static figures, or in tasks which require no mental generation, manipulation, or rotation of images (Johnston, 1982; Kamhi, 1981; Kamhi et al., 1984, 1990; Leonard et al., 1997; Savich, 1984; Wyke and Asso, 1979) (also see Leonard, 1998). Thus children with SLI appear to be impaired at "dynamic" mental imagery, which is linked to the procedural memory system, but not at "static" visual imagery, which is linked to the declarative memory system.

Working memory impairments. SLI is strongly linked to working memory impairments (Botting and Conti-Ramsden, 2001; Fazio, 1996, 1998; Gathercole and Baddeley, 1993; Kirchner and Klatzky, 1985; Montgomery, 1995a, 1995b; Montgomery, 2000, 2003; Sininger et al., 1989; Weismer, 1996). In one study children with SLI took almost four times as long to scan a sequence of items in short-term memory (measured by response speed on a Sternberg task) as compared to control children (Sininger et al., 1989). Children with SLI have particular difficulty with serial order in working memory tasks (Fazio, 1996; Gathercole and Baddeley, 1990; Gillam et al., 1995; Kirchner and Klatzky, 1985). In addition, non-word repetition, a task whose performance is highly dependent on phonological working memory, and requires the maintenance of a sequence of phonological segments, is notoriously difficult for children with SLI (see above). Intriguingly, non-word repetition has been found to correlate, across subjects, with performance at several tasks probing grammatical processing: sentence repetition (Bishop et al., 1996; Kamhi and Catts, 1986), past tense and third person singular production tasks (Botting and Conti-Ramsden, 2001), and a test of receptive grammar (Botting and Conti-Ramsden, 2001). Similarly, other measures of working memory abilities have also been found to correlate with performance at sentence comprehension (Montgomery, 2000). These data suggest a common neural basis for working memory and grammar, as predicted by the PDH.

As discussed above, it has been argued that phonological working memory impairments may be largely or completely responsible for language and other deficits in children with developmental language disorders (Gathercole and Baddeley, 1990, 1993; Montgomery, 1995b). However, other investigators dispute this claim (Howard and van der Lely, 1995; Rice et al., 1994; van der Lely and Howard, 1993). Indeed, it has been argued that the impairments shown by children with SLI at tasks probing phonological working memory might be explained by deficits of phonological processing rather than of working memory (Chiat, 2001; Gillam et al., 1998). Although we acknowledge that working memory deficits may aggravate the language impairments in SLI, including in tasks probing grammatical processing, we argue that they are not necessary for the presence of SLI. Rather, as with other non-linguistic functions that depend on the brain structures of the procedural system, we expect a strong statistical association between grammatical and working memory abilities in this disorder. Further research will be required to elucidate the exact nature of the association between working memory and language processing, both in general and in SLI (Montgomery, 2003).

Temporal processing deficits. One of the most commonly documented deficits in the SLI literature is a difficulty in the perceptual processing of a sequence of stimuli presented in rapid succession, or of stimuli of brief duration (for reviews, see Leonard, 1998; Tallal et al., 1993). These problems have been observed in a variety of auditory tasks, including in speech discrimination (Leonard et al., 1992b; Tallal and Piercy, 1974; Uwer et al., 2002) and word learning (Weismer and Hesketh, 1996). The deficits occur not only with language stimuli such as syllables (Leonard et al., 1992b; Tallal and Piercy, 1974) and words (Weismer and Hesketh, 1996), but also with non-language stimuli such as tones (Tallal and Piercy, 1973a; Tallal et al., 1981; Tomblin et al., 1995; but see Uwer et al., 2002). Similar perceptual deficits are found in vision (Fazio, 1998; Tallal et al., 1981, 1985b), and in the somatosensory modality (Kracke, 1975; Tallal et al., 1985b). Temporal processing difficulties do not appear to be limited to perception. As we have discussed above, impairments in production tasks across domains are aggravated when they require fast responses, such as in rapid naming (Katz et al., 1992; Lahey and Edwards, 1996; Leonard et al., 1983), or in the execution of rapid sequential movements (Bishop, 2002; Johnston et al., 1981; Preis et al., 1997). Moreover, affected members of the KE family have problems both discriminating and producing rhythms (which likely require precise timing and sequencing), while being spared at discriminating and producing pitch (Alcock et al., 2000). We expect similar dissociations in other SLI sub-groups. Indeed, children with SLI have been shown to have greater difficulty than age-

matched controls in identifying rhythmic sequences (Kracke, 1975).

These data suggest that the temporal processing deficits in SLI are quite broad in the domains they affect, and may be closely linked to other impairments in the disorder. Indeed, rapid or sequential auditory processing deficits have been shown to correlate, across subjects, with linguistic deficits (Leonard et al., 1992b; Tallal et al., 1976), suggesting a common neural basis for these impairments. It has been argued that perceptual temporal processing deficits are directly responsible for the language problems of children with SLI (Tallal et al., 1993). However, perceptual processing deficits do not necessarily accompany SLI (Bishop et al., 1999; Neville et al., 1993; Tomblin et al., 1995), and cannot easily explain certain linguistic impairments found in the disorder (Ullman and Gopnik, 1999; van der Lely and Ullman, 2001). Thus the data suggest that perceptual processing deficits are strongly *associated* with language impairments, but do not *necessarily* co-occur with them, and do not seem to cause them. This pattern is consistent with the view that the observed perceptual and language problems are at least partly explained by abnormalities of related but separate portions of the procedural system (e.g., distinct frontal/basal-ganglia channels). Future studies may elucidate this issue.

Preserved Abilities of the Declarative Memory System

Whereas tasks and functions that depend on the procedural memory system are impaired in SLI, those that depend instead on the ventral stream and the declarative memory system seem to remain normal. As we have seen above, children with SLI are good at static as opposed to dynamic mental imagery tasks (Kamhi, 1981; Kamhi et al., 1984; Leonard et al., 1997; Savich, 1984; Wyke and Asso, 1979). Importantly, children with SLI also appear to be relatively normal at learning new information in declarative memory. First, as discussed above, they can detect semantic anomalies, they are relatively proficient at word learning, and their lexical-semantic organization appears normal. Second, evidence suggests that verbal episodic memory remains largely intact. Dewey and Wall (1997) tested the performance of children with SLI on a variety of memory skills. They found that although the SLI group was impaired on tests of short-term verbal memory, no deficits were found on the verbal memory task that included a learning component. Third, there is also evidence that children with SLI do not have difficulty learning new conceptual or factual knowledge in long-term memory. That is, acquiring new information in semantic memory also appears to be normal. Merrit and Liles (1987) tested language-impaired and unimpaired children on the generation and retelling of stories. Crucially, the two groups of children did not differ in their memory of the factual details of the stories. Fourth, visual episodic memory has also been reported to be spared in SLI (Dewey and Wall, 1997; Williams et al., 2000). In sum, evidence suggests that declarative memory abilities often remain intact in SLI.

Conclusion

We have presented an in-depth examination of previous studies, which have used multiple techniques to probe both the neural basis of SLI and its linguistic and non-linguistic correlates. We have argued that the Procedural Deficit Hypothesis accounts for much of this data. In particular, the PDH explains abnormalities of seemingly disparate functions and structures, in the face of others that remain largely normal. As discussed above, other explanatory hypotheses cannot account for many of these findings. This holds not only for hypotheses positing grammatical or processing deficits, but also for the view that children with SLI are simply worse at those tasks and functions that are "harder"; importantly, such a perspective cannot easily account for impairments of procedural but not declarative functions, particularly since the latter are often *more* problematic for normal individuals (e.g., regulars vs. irregulars; see above). Moreover, no other explanations of SLI that we are aware of expect the particular brain abnormalities in frontal and basal ganglia structures that are specifically predicted by the PDH, and appear to be so strongly associated with SLI.

The PDH has a number of implications. First, the hypothesis has clinical significance. Very early detection or confirmation of SLI may be possible by examining the neuroanatomical structures posited to underlie the disorder (e.g., with volumetric analyses of structural MR data). Thus early abnormalities of the caudate nucleus or of Broca's area may be indicative of developmental language impairments, allowing for early intervention. Moreover, SLI should be susceptible to pharmacological and behavioral therapies that are motivated by our independent knowledge of the two memory systems and their neural correlates. For example, the neuropharmacology of declarative memory and its underlying neural substrates (Curran, 2000) should pertain to language as well. Thus it is plausible that cholinergic interventions, which can enhance declarative memory (Freo et al., 2002; Packard, 1998), may facilitate the compensatory shift to this system. Conversely, dopaminergic interventions, which have been successful at treating other developmental and adult-onset disorders that affect the neural substrates of the procedural system (e.g., ADHD and Parkinson's disease) (Gerfen, 1995; Jankovic

and Tolosa, 1993), may be helpful in directly enhancing grammatical and non-linguistic procedural function (see Tallal et al., 1994). Behavioral therapies can also be guided by the predictions of the PDH. For example, one should be able to exploit the functional characteristics of declarative memory, such as promoting learning in rich semantic contexts. Additionally, learning environments can be manipulated to reduce demands on the procedural memory system. Thus breaking down complex sequences into their component parts, and presenting new information frequently, should also be expected to facilitate language acquisition.

Second, the PDH may help explain patterns of co-morbidity between SLI and other developmental disorders: If other disorders also involve abnormalities of the brain structures underlying the procedural system, then at least some degree of co-morbidity with SLI would be expected. Here we briefly examine the relation between SLI and ADHD. These two disorders are highly co-morbid. Some studies document as high as a 45% rate of language impairment (Tirosh and Cohen, 1998) among children with ADHD. Conversely, the most frequent psychiatric diagnosis among children with language impairments is ADHD (Cohen et al., 2000). Goorhuis-Brouwer and Wijnberg-Williams (1996) documented that among 14 children assumed to have 'pure' SLI, attention problems were noted in eleven of them in a four-year follow-up assessment. Additionally, like children with SLI, those with ADHD commonly have impairments of working memory (Barkley, 1997; Denckla, 1996; Paule et al., 2000b; Pennington and Ozonoff, 1996) and motor functioning (Diamond, 2000; Denckla and Rudel, 1978). Children with ADHD have also been shown to have perceptual deficits of timing discrimination (Paule et al., 2000a; Smith et al., 2002) and an impaired capacity to reproduce time intervals (Barkley et al., 1997). In contrast, long-term memory abilities tend to be normal in ADHD (Kaplan et al., 1998; Paule et al., 2000a). Studies of the neural bases of ADHD have implicated several structures, including prefrontal cortex, the basal ganglia (in particular the caudate nucleus), and the cerebellum (Aylward et al., 1996; Castellanos, 2001; Castellanos et al., 1996; Filipek et al., 1997; Paule et al., 2000a). However, evidence suggests that SLI and hyperactivity can appear not only as comorbid syndromes (or symptom complexes), but also as separate disorders (Williams et al., 2000). These findings suggest that the disorders have distinct but overlapping cognitive and neural correlates. We suggest that SLI and ADHD may both be considered disorders affecting the brain structures of the procedural system, in particular frontal/basal-ganglia circuits (especially the caudate), and that they involve overlapping but partially distinct neural structures (e.g., within frontal/basal-ganglia circuitry), and therefore overlapping but partially distinct cognitive functions.

Third, the PDH, and the evidence supporting it, have implications for neurocognitive models of language. The PDH is largely motivated by the Declarative/Procedural model of language, and evidence supporting the PDH also supports this perspective. The PDH predicts that SLI is associated with non-linguistic functions that are subserved by the same brain system that also underlies grammar; this outcome is clearly not expected by the view that domain-specific modules subserve distinct aspects of grammar (Chomsky, 1995; Fodor, 1983; Frazier and Fodor, 1978; Grodzinsky, 2000). Likewise, at least previously proposed "single-mechanism" models of grammatical and lexical phenomena (Bates and MacWhinney, 1989; Joanisse and Seidenberg, 1999; MacDonald et al., 1994; Rumelhart and McClelland, 1986) do not appear to make the same claims and prediction as the PDH, or to explain the PDH, or with the grammatical/lexical dissociations found in SLI (see Pinker and Ullman, 2002a; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001). However, it must be noted that the single-mechanism (connectionist) computational perspective is not in principle incompatible with the PDH and the findings described above, since distinct declarative and procedural systems can and have been modeled by connectionist simulations (Dominey et al., 2003; McClelland et al., 1995). In any case, the PDH and associated empirical evidence should provide useful constraints for neurocognitive models of language.

Fourth, the PDH suggests a specific research program. In this paper we have focused on predictions and relevant data at the population level. That is, we have examined the claim that impairments of a particular set of linguistic and non-linguistic functions, and abnormalities of a particular set of brain structures, should be commonly found in SLI. We have not focused much on research that allows us to examine the claims of the PDH at the level of individual subjects. That is, we have not discussed many experiments which test the prediction that *within subjects*, a particular set of structural abnormalities and functional impairments should co-occur, while other structures and functions are spared. Such experiments are crucial for testing the PDH, and should form the basis of any research program which examines this hypothesis. Unfortunately, most previous empirical studies have restricted themselves to a small number of tasks, or to the underlying neuroanatomy, in a given set of subjects. Those studies that have examined multiple functions and/or structures in the same set of subjects have reported promising results. We have seen above that a number of correlations have been found, within a given groups of SLI individuals, between procedural system functions. Moreover, a number of reports suggest that the affected members of the KE family show the expected set of

functional and structural abnormalities. Other in-depth investigations of this sort, examining other groups of SLI subjects, are needed.

Finally, the PDH leads to a number of testable predictions, both at the population and individual levels. SLI is predicted to be associated not only with the structural and functional patterns discussed above, but also with the following outcomes, both across and within subjects. Frontal/basal-ganglia circuits, especially the caudate nucleus and Broca's area, are expected to be abnormal, whereas the hippocampus and other medial and inferior/lateral temporal lobe structures may remain largely normal. The grammatical deficits should not be restricted to syntax, morphology and phonology. Rather they are expected to encompass all rule-governed combinatorial aspects of grammar. Thus compositional semantics (the interpretive – i.e., semantic – aspects of the composition of words into complex structures, Portner and Partee, 2002) is expected to be impaired. Learning grammatical rules in artificial language studies should also be difficult, and should yield abnormal ERP and fMRI/PET patterns of activity (see Friederici et al., 2002; Opitz and Friederici, 2002). All non-linguistic functions that depend on the brain structures of the procedural system, in particular those functions that rely on the caudate nucleus and Broca's area, should be problematic. This includes functions and tasks not yet examined in SLI. For example, deficits are expected in procedural memory learning, as tested with Serial Reaction Time tasks and other tasks that probe the acquisition of motor and cognitive skills and rules that depend on procedural memory (Eichenbaum and Cohen, 2001; Schacter and Tulving, 1994; Squire and Knowlton, 2000). In contrast, declarative memory should be largely spared. Thus not only tests of word learning, but also those probing episodic and semantic memory learning, should yield normal performance in circumstances that do not require procedural memory and its underlying brain structures – for example, where learning takes place in contexts with slowly presented items and with adequate time for responses, and where knowledge is probed with recognition rather than retrieval tasks.

In sum, in this paper we have presented the PDH as a novel alternative to previously proposed explanatory accounts of SLI. We have argued that the PDH explains a substantial amount of previously reported brain and behavioral data. Moreover, the hypothesis has a number of implications and makes a range of testable predictions, allowing it to be both falsified and further specified. Thus the PDH may provide a useful paradigm for the study of SLI.

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Michael T. Ullman, Department of Neuroscience, Georgetown University, Box 571464, Washington, DC 20057-1464, USA. e-mail: michael@georgetown.edu.