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Grammar predicts procedural learning and consolidation deficits in children with Specific Language Impairment

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ABSTRACT

The Procedural Deficit Hypothesis (PDH) posits that Specific Language Impairment (SLI) can be largely explained by abnormalities of brain structures that subserve procedural memory. The PDH predicts impairments of procedural memory itself, and that such impairments underlie the grammatical deficits observed in the disorder. Previous studies have indeed reported procedural learning impairments in SLI, and have found that these are associated with grammatical difficulties. The present study extends this research by examining consolidation and longer-term procedural sequence learning in children with SLI. The Alternating Serial Reaction Time (ASRT) task was given to children with SLI and typically developing (TD) children in an initial learning session and an average of three days later to test for consolidation and longer-term learning. Although both groups showed evidence of initial sequence learning, only the TD children showed clear signs of consolidation, even though the two groups did not differ in longer-term learning. When the children were re-categorized on the basis of grammar deficits rather than broader language deficits, a clearer pattern emerged. Whereas both the grammar impaired and normal grammar groups showed evidence of initial sequence learning, only those with normal grammar showed consolidation and longer-term learning. Indeed, the grammar-impaired group appeared to lose any sequence knowledge gained during the initial testing session. These findings held even when controlling for vocabulary or a broad non-grammatical language measure, neither of which were associated with procedural memory. When grammar was examined as a continuous variable over all children, the same relationships between procedural memory and grammar, but not vocabulary or the broader language measure, were observed. Overall, the findings support and further

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specify the PDH. They suggest that consolidation and longer-term procedural learning are impaired in SLI, but that these impairments are specifically tied to the grammatical deficits in the disorder. The possibility that consolidation and longer-term learning are problematic in the disorder suggests a locus of potential study for therapeutic approaches. In sum, this study clarifies our understanding of the underlying deficits in SLI, and suggests avenues for further research.

1. Introduction

Specific Language Impairment (SLI), which appears to affect about 7% of children (Tomblin et al., 1997), is a developmental disorder of language that cannot be accounted for by hearing problems, environmental deprivation, mental retardation, gross neurological insults, or physical abnormalities that lead to speech problems (Bishop, 1992; Leonard, 1998). However, despite the standard use of exclusionary criteria to diagnose SLI, the disorder does not appear to be limited to language. Rather, the linguistic deficits tend to co-occur with various non-linguistic problems, including impairments of motor skills and working memory (Hill, 2001; Leonard, 1998; Montgomery, Magimairaj, & Finney, 2010; Ullman & Pierpont, 2005), which have complicated the understanding of SLI (Leonard, 1998; Stromswold, 2000).

Among the various explanatory hypotheses of SLI, two broad competing perspectives can be distinguished. One perspective suggests that the disorder is caused by a deficit or delay that is specific to certain aspects of language, in particular to grammar (Clahsen, 1989; Gopnik & Crago, 1991; Rice, Wexler, & Cleave, 1995; Rice, Wexler, Marquis, & Hershberger, 2000; van der Lely, 2005). The other broad perspective posits that the underlying cause of SLI is some sort of non-linguistic processing deficit, either of a general sort (Bishop, 1994a; Kail, 1994; Leonard, McGregor, & Allen, 1992; Norbury, Bishop, & Briscoe, 2001), or one that is relatively specific, such as of working memory (Archibald & Gathercole, 2007; Montgomery, 1995) or of briefly presented stimuli or rapidly presented sequences of items (Tallal, Miller, & Fitch, 1993; Tallal & Piercy, 1973).

Hypotheses within both perspectives have been able to account for certain deficits observed in SLI. Those arguing for a purely grammatical deficit have, naturally, been successful in explaining many of the grammatical impairments observed in the disorder. However, such hypotheses cannot account for the non-linguistic problems commonly found in SLI, nor can they easily explain the full range of linguistic deficits, which typically include phonological, morphological and syntactic impairments, as well as difficulties with lexical retrieval. Conversely, hypotheses positing a general or specific processing deficit may be able to explain a number of the non-linguistic (and even some linguistic) deficits, but cannot easily account for the specific pattern of spared and impaired linguistic and non-linguistic functions observed in SLI (Ullman & Pierpont, 2005).

Crucially, all of these previously proposed hypotheses have attempted to explain SLI at a functional rather than neural level, even though it is indubitably the case that the dysfunctions in the disorder originate in the brain. It is possible that an explanatory hypothesis that takes the brain as well as behaviour into account might be better able to tie together the various apparently disparate problems of SLI by linking them to common neural substrates. The Procedural Deficit Hypothesis (PDH) takes just this approach (Ullman, 2004; Ullman & Pierpont, 2005).

According to the PDH, the pattern of impaired linguistic and non-linguistic functions associated with SLI can be largely explained by abnormalities of brain structures that constitute the procedural memory brain system (Ullman & Pierpont, 2005). This memory system is one of several brain systems involved in the implicit acquisition, storage and use of knowledge (Gabrieli, 1998; Squire & Zola, 1996; Willingham, 1998). The system involves a network of brain structures that includes the basal ganglia, the cerebellum and portions of parietal and frontal cortex, including premotor cortex and posterior parts of Broca's area (e.g., BA 44) (Eichenbaum & Cohen, 2001; Gabrieli, 1998; Knowlton, Mangels, & Squire, 1996; Ullman, 2004; Ullman & Pierpont, 2005). The system underlies a range of perceptual, motor and cognitive skills. In particular, a large literature suggests that it subserves sequencing (Fletcher et al., 2005; Hikosaka et al., 1999; Wilkinson & Jahanshahi, 2007; Willingham, Salidis, & Gabrieli, 2002). However, it also appears to play a role in other functions (Ullman, 2004), including in the learning of probabilistic rules and categorization (Knowlton et al., 1996; Poldrack et al., 2001). Finally, accumulating evidence indicates that procedural memory also subserves the learning and use of rule-governed aspects of grammar, across syntax, morphology and phonology (Ullman, 2001, 2004; Ullman & Pierpont, 2005).

The PDH posits that abnormalities of brain structures underlying procedural memory, in particular portions of frontal/basal-ganglia circuits (especially the caudate nucleus and the region around Broca's area) and the cerebellum, lead to impairments of the various domains and functions that depend on these structures. Most importantly, procedural memory itself is expected to be impaired, leading to deficits in implicit sequence learning, grammar, and various other tasks and functions that depend on procedural memory. Additionally, other, non-procedural, functions that depend at least in part on these brain structures should tend to be problematic, including working memory, aspects of temporal processing, and lexical retrieval (Ullman, 2004; Ullman & Pierpont, 2005).

Ullman and Pierpont (2005) accompanied their theoretical proposal with a comprehensive review of the brain and behavioural evidence regarding SLI. Overall, the data largely supported the pattern of predictions of the PDH. However, at that time no studies of *learning* in procedural memory in SLI had yet been published. Rather the PDH was argued on the basis of a wide range of other evidence, and it *predicted* impairments of procedural learning (that is, learning in procedural