Early language delay (ELD) is a warning sign that may presage the presence of a later language impairment (LI). In order to allow more targeted identification and earlier intervention for LI, better diagnostic measures for toddlers are needed. Development of accurate predictive/diagnostic models requires consideration of a set of complex interrelated questions around definition, causality, and theories of LI. A multifactorial model of language development and LI is essential to increase the accuracy of prediction. This article examines what is known about LI in the preschool years and language delay in toddlers, and examines these in relation to the Procedural Deficit Hypothesis (Ullman and Pierpont, 2005) Cortex 41:399-433 and the Statistical Learning Account (Stokes et al., [2012a] J Speech Lang Hear Res; Stokes et al., [2012b] J Child Lang 39:105-129) to suggest a new framework for characterizing ELD to better assist prediction of later LI.

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Key words: late talkers; preschool specific language impairment

Children with language disorders experience difficulties with communication which may impact adversely on social-emotional development, academic achievement, and vocational options if the disorder persists beyond the preschool period [Clegg et al., 2005]. During this period, a language impairment (LI) in the absence of significant sensory, psychiatric, neurological, or intellectual impairment/disorder is commonly referred to as specific language impairment (SLI). While the profile of specific linguistic abilities (e.g., in grammar, semantics, phonology, and pragmatics) varies across children with SLI and across time, English-speaking children with SLI generally exhibit problems with grammatical morphology relative to children of the same age without SLI [Leonard, 1998]. In an effort to understand this variability across linguistic abilities, researchers have identified subgroups of LI.

One attempt to delineate subgroups of children with SLI using cluster analysis found three underlying linguistic factors based on phonology/articulation, semantics/syntax, and pragmatics [Tomblin et al., 2004]. These factors dissociate to some degree to create different subgroups. Further, semantic/syntactic disorders may be expressive only or both receptive and expressive. While there is as yet no consensus regarding how many subgroups exist, it is clear that a child's subgroup membership and developmental profile can change over time [Conti-Ramsden and Botting, 1999]. There is evidence to suggest that these variations in how the disorder manifests itself may be characteristic of the core language disorder as it unfolds over time, rather than separate diagnostic entities [Bishop, 1994]. The heterogeneity of the SLI population is one of the challenges in early identification of this disorder. The focus of this article is the developmental period in which a core language disorder is identified. Language disorders in children are usually identified at four-to-five years of age. However, there are indications that SLI might be detectable earlier than the age at which it is usually diagnosed.

LATE TALKING AND LATER LANGUAGE IMPAIRMENT

Late onset of talking has long been thought to be an early manifestation of later LI [Olswang et al., 1998; Thal, 2000] and has been referred to variously as specific expressive language delay (SELD), early language delay (ELD), and late language emergence (LLE). It is identified between about 18 and 30 months of age, with the first indicators being early vocabulary delay, slow vocabulary growth, and/or delayed onset of word combinations. For research purposes, this group of children has been identified on the basis of parent-report measures such as the Language Development Survey [LDS; Rescorla, 1989], the MacArthur Communicative Development Inventory: Words and Sentences [CDI:WS; Fenson et al., 2007], and the Ages and Stages Questionnaire Communication scale [ASQ; Bricker and Squires, 1999]. Various operational definitions have been used to define the target population, including (1) fewer than 50 words of expressive vocabulary or no word combinations by 24 (± 6) months of age on the LDS [e.g., Rescorla, 1989; Klee et al., 1998]; (2) <10th percentile on the expressive vocabulary section of the CDI:WS [e.g., Reilly et al., 2007]; and (3) 1 SD or more below the mean on a composite score from the ASQ Communication scale [Zubrick et al., 2007]. These definitions typically result in between 10 and 20% of children being identified as late talkers (LTs).

While these criteria are commonly used, they appear to be at best only moderately indicative of ongoing language disorder. This is likely due to the wide variation in the development of early language skills such as vocabulary and grammar. Late talking has been associated with poorer developmental outcomes.

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such as SLI, dyslexia, autism spectrum disorders, and attention deficit hyperactivity disorder [Buschmann et al., 2008]. In general, children who start out as high achievers in language will continue to achieve well throughout childhood whereas those who start off slowly often, but not always, have poorer outcomes. However, the majority of children who start off slowly will accelerate in their development to come within normal range over the next 18 months [Paul, 1996; Rescorla and Roberts, 1997]. In comparison with the 50–70% recovery rate from late talking, a diagnosis of SLI at age 4 has a smaller degree of recovery in the short-term. Bishop and Edmondson [1987] found that of children with SLI at age 4 identified by a narrative measure, only 10% had resolved by age 5–6. In a longitudinal study with the same sample, Stothard et al. [1998] found that children who had resolved by age 5–6 differed from controls at ages 15–16 years on measures of phonological processing and literacy. However, those who had SLI or a general delay at age 5–6 had significant impairments in all aspects of spoken and written language functioning at outcome. This illustrates the changing nature of language delay and disorder across childhood. As a group, children with an early vocabulary delay fall within the range of typical development by 4–5 years but then delays in grammatical development become apparent. Conversely, there is some evidence that a portion of children start out in the normal range for language but slow in development, and fall in the disordered range at a later point [Dale et al., 2003; Rice et al., 2008]. Predicting which LTs will go on to manifest a significant language difficulty at age 4–5 poses a diagnostic challenge (see Rescorla, this issue), with part of the problem being what exactly constitutes SLI.

While the term specific is part of the definition, non-linguistic skills are also known to be impaired, with children having depressed nonverbal IQ scores [Johnston, 1994], and deficits in hypothesis testing [Kamhi et al., 1984], cross-modal processing [Montgomery, 1993], voice processing abilities [Creusere et al., 2004], sustained attention [Finneran et al., 2009], processing capacity and speed [Leonard et al., 2007], working memory [Montgomery et al., 2010], and motor coordination [Hill, 2001]. This begs the question of whether or not children with more general developmental delays are included in studies predicting SLI. Commonly, children with general developmental delays [indicated by nonverbal IQ scores below 85] have not been included. However, this practice is questionable for several reasons.

Firstly, the assumption behind this appears to be that children with low nonverbal scores do not have the cognitive underpinnings to support normal language development; that is, that an undetermined general factor which caused low nonverbal achievement will have also caused the problem with language development. However, groups of children with low nonverbal ability but language in the normal range have been identified [Rice et al., 2004]. The language ability of these children did not differ from age-matched controls. Secondly, it is debatable whether or not the language of children with SLI is qualitatively different from those with more general delays. Rice et al. [2004] reported qualitative differences while Tomblin et al. [2004] reported only quantitative differences. Children on the borderline between these two diagnoses shift between them over time, pointing to a degree of overlap in these clinical groups [Vig et al., 1987], leading some researchers to prefer the term primary language impairment (PLI) or language impairment (LI) rather than SLI. In this review, we adopt the term SLI as there is still significant debate about comorbidity and specificity of language-related disorders. Thirdly, studies of other developmental disorders are likely to be informative with respect to the definition of SLI. Boundaries between disorder types may not be distinct. For example, an overlap between dyslexia and SLI has been postulated, with the shared variance thought to be due to an underlying phonological processing disorder [Catts et al., 2004]. In addition, the categories of autism spectrum disorder and SLI may overlap to some extent because of an underlying auditory perceptual processing difficulty [Oram Cardy et al., 2008]. There is a moderate rate of comorbidity (or arguably overlap) with SLI and childhood apraxia of speech, attention deficit hyperactivity disorder (ADHD), and autism. Diagnosticians attempt to identify clusters of children who fit these categories; however there are always individuals who do not fit clear diagnostic categories, and who sit at the boundary between types. Children initially identified as LTs may later be diagnosed with SLI, dyslexia, ADHD, autism spectrum disorder (ASD), or general intellectual disability, moving in and out of diagnostic categories as they mature.

PREDICTING LATER SLI

We turn our attention now to factors that may contribute to an ELD (and thus, later talker status), as well as a preschool LI. A range of linguistic, demographic, medical, genetic, and environmental factors have been explored as predictors of later language ability [Zubrick et al., 2007; Ellis and Thal, 2008; Reilly et al., 2009; Stokes and Klee, 2009a]. While late emergence of expressive vocabulary alone is not a strong predictor of language ability at 4 or 5 years, other variables have some, but limited, predictive value. Language comprehension and gestural use have been shown to have some predictive value in small sample studies [e.g., Thal et al., 1991; Thal and Tobias, 1992]. Of the 10 LTs included in these studies, six were “late bloomers” (i.e., children who had caught up with their age-matched peers) by 30–35 months, while four were still delayed in language production. Examination of their gesture use and language comprehension at 18 months showed that LTs who continued to have poorer language skills had lower comprehension scores, and failed to use gestures as compensation for reduced expressive vocabulary ability, than the late bloomers. Ellis and Thal [2008], in a brief review of the relationship between ELD and later LI, suggested that there were three key likely predictors, a family history of language delay or impairment, delays in receptive as well as expressive vocabulary, and the lack of communicative gestures (the latter is supported by a longitudinal study [Rowe et al., 2012]). To these they add a history of otitis media, parent concern, high risk environments (e.g., poverty), and delays in symbolic play or social skills. Mother’s education [Dollaghan et al., 1999], a family history of speech/language delay [Bishop et al., 2003], the child’s sex, and early neurobiological growth [Zubrick et al., 2007], birth order/number of siblings [Stokes, 1997], a history of ear infections or parent concern [Klee et al., 2000], socio-emotional development [Borstein et al., 1998], and cognitive development [Price et al., 2003], have been shown to have some predictive value, in some, but not all, studies. Evidence for these factors as sole predictors is not consistent, and, as we show below, a multifactorial model of prediction is likely to be the way forward for improving our ability to predict the consequences of ELD.

A MULTIFACTORIAL MODEL

As we work toward a greater understanding of factors that contribute

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to late talking and SLI, we must consider multiple interrelated factors [Desmarais et al., 2008]. Klee and Stokes [2011] presented a multifactorial model of language development that draws on Gottlieb's model of epigenesis [Gottlieb, 2007], and Bishop and Snowling's [2004] model of relationships between observed behavior, cognitive processes, neurobiology, and etiology in development (see Fig. 1). The model considers the roles of genetic, environmental, neurobiological, and cognitive factors in language development. Here, we consider how these factors might serve as predictors of later SLI. For example, while the genotype is the starting point for development, identical genotypes can result in different phenotypic outcomes in response to differences in the environment (for example, the variations seen in monozygotic twin development).

**FACTORS POTENTIALLY PREDICTIVE OF SLI**

**Genetic Factors**

Genetic factors play an important role in the development of SLI. Verbal ability seems to be one of the most heritable of cognitive traits, as demonstrated by adoption studies [Plomin et al., 1997]. Twin studies have shown that monozygotic twins have higher rates of concordance of language disorders than dizygotic twins, indicating the impact of genes beyond that of the environment [Dale et al., 2003]. LTs are more likely to have a family history of language and learning disorders than children with non-affected language development [Zubrick et al., 2007; Reilly et al., 2009]. They are also more likely to score lower as a group on linguistic related measures [Flax et al., 2009] and to have SLI [Choudhury and Benasich, 2003]. In addition, boys are more likely to be LTs than girls [Zubrick et al., 2007; Reilly et al., 2009]. Boys show slower expressive vocabulary growth patterns than girls before 24 months, but there is no gender difference in overall vocabulary growth [Rowe et al., 2012].

The search for specific genes causing neurodevelopmental disorders has proven more complex than initially expected. The concept of a "language gene" is an appealing but simplistic one. Early studies of the genotype of the KE family, who have a high rate of childhood apraxia of speech in their family, yielded evidence that the FOXP2 gene was deficient for affected family members [Vargha-Khadem et al., 1998]. Recent research suggests that mutations of FOXP2, a transcription factor gene that encodes a regulatory protein, are rare and that it is not such mutations that are relevant for SLI, rather, it the endpoint of FOXP2 regulation, the target neural circuitry that is relevant [Fisher and Scharff, 2009].

**Environmental Factors**

While genetic factors are critical in the phenotypic outcome of disorders, environmental stimulation also plays a powerful role. Children learn language from social interaction with other people. Research into the effects of the environment on language acquisition has focused on two factors, conversational styles of parent–child interactions and variations in linguistic input. The role each plays is likely to differ as a function of age and also at the extreme ends of the ability and environment spectra.
Some aspects of parent–child interaction have been found to facilitate language development [Yoder and Warren, 2004]. Several studies have examined whether the interactions between LTs and their parents are different in a way that may negatively impact on child language development. However, the interaction styles of parents of typically developing [TD] children and parents of LTs appear to be more similar than different. Paul and Elwood [1991] reported that parents of LTs have a larger gap between their mean length of utterance and their child’s language, which may make language more difficult to learn. Vigil et al. [2005] found that parents of TD children used more responses, expansions, and self-directed speech than parents of LTs; however, these behaviors could be reflective of higher language abilities in TD children, which elicit different conversational styles, rather than style differences causing LLE. Although the amount of linguistic input experienced by children from different social groups has been shown to have a dramatic impact on the size of children’s developing lexicons [Hart and Risley, 1995], the more subtle differences in the features of parent–child interactions of late talking and TD children are not likely to play a causal role in developmental language disorders.

Nonetheless, it appears that the quality and quantity of linguistic input in the early years impacts on children’s linguistic achievements. Hart and Risley [1995] discussed the importance of the cumulative effect of linguistic input over time, with children with more enriched input having larger vocabularies at every time point. The main factor associated with the level of linguistic input in this study was socioeconomic status (SES). Subsequent authors [e.g., Hoff, 2003] proposed that variation in linguistic input was mediated by SES; however, two recent large-scale population studies found little relationship between SES and LLE [Zubrick et al., 2007; Reilly et al., 2009]. Conversely, SES has been associated with SLI in 4–6 year old children [Stanton–Chapman et al., 2002; Reilly et al., 2010]. However, Plomin et al. [1997] found that children adopted at birth became increasingly similar to their biological parents in cognitive and verbal abilities from infancy through adolescence than to their adoptive parents, confirming the importance of genetic influences on language abilities. It appears that associations between linguistic environment, SES, and language ability may be mediated by genetic influences.

There seems to be a differential effect of genes and environment on language development toward the extreme low ends of the spectrum in environment and ability. It is undisputed that environmental deprivation at the extreme end of the spectrum (poverty or severe neglect) is linked with depressed cognitive and language development [Turkheimer et al., 2003]. In a large twin study, Dale et al. [1998] reported that the genetic contribution accounted for 73% of the variance in vocabulary development for children in the lowest 5% of language ability at 2 years. For the entire sample, 25% of the variance was accounted for by genetic variables rather than environmental factors.

Unfolding Neurobiology

Having seen that genetic factors play a large role in late talking and SLI outcomes, we turn now to considering the neurobiology of language. Significant differences in the neurobiology of children with SLI have been demonstrated. For recent reviews, see Webster and Shevell [2011] and Badcock et al. [2012]. Brain structures in children with SLI have been found to differ in volume and symmetry from those of TD children. Cohen et al. [1989] also found differences in gyrification. Volume differences, relative to TD peers, include a smaller Broca’s area [Gauger et al., 1997], reduced surface area in the primary auditory cortex of the left hemisphere [Leonard et al., 2002], and increased gray matter in the right perisylvian region and the occipital petalia [Soriano-Mas et al., 2009]. Typically the dominant, left hemisphere language areas are larger in volume compared with the corresponding regions of the right hemisphere. However, for children with SLI the hemispheres may not differ in volume, or may show a rightward dominance. Reduced asymmetry of the perisylvian structures has been found [Plante et al., 1991], particularly in the temporal [Ors et al., 2005] and planum temporale regions [Gauger et al., 1997; Leonard et al., 2002]. At odds with the latter finding is one report of normal left–right asymmetry in children with SLI [Preis et al., 1998].

These volumetric (and possibly asymmetric) differences become important in their relationship with functional language organization and performance. However, as Badcock et al. [2012] point out, there are few studies of how these physiological differences relate to language function, and prior to Badcock et al., they were limited to investigations of the KE family [e.g., Vargha-Khadem et al., 2005]. Nonetheless, these studies do point to areas of the brain where neurolinguistic processing may be different for children with SLI, and potentially, children who are LTs.

Cognitive Mechanisms

Perception

Measures of perception have been found to correlate with language development. Speech perception plays an important role in language acquisition. Speech perception skills, including phoneme discrimination and learning to inhibit processing of non-critical sound contrasts, in infancy have been found to correlate with later language development [Conboy et al., 2008]. The assumption is that the sooner infants are able to discriminate between their native language phonemes, the sooner they can map phonological patterns and begin to learn words. Behavioral and electrophysiological measurements, such as event related potentials (ERP), have been used to show that infants’ native language speech discrimination abilities measured at seven months predict language outcomes at 14–30 months. Those with better native speech discrimination had significantly higher language abilities whereas those with better non-native phonetic discrimination had poorer language abilities (see Kuhl, [2010], for a review). Children who progress more quickly toward neural commitment to native sounds contrasts progress faster than those who retain the ability to discriminate between non-native contrasts for longer [Kuhl, 2010]. Children who advance in phonetic abilities faster should begin to detect phonotactic patterns and words more readily, leading to faster development of vocabulary and subsequent grammar. In a series of ERP studies, Molfese et al. found that infants who could discriminate consonants better between phonemic boundaries had better language development at ages 3, 5, and 9 years old [Molfese et al., 1999]. In addition, Tsao et al. [2004] found that infants who could discriminate between two non-native vowels at six months of age had better language development at 13, 16, and 24 months. Newman et al. [2006] found that in TD infants, the ability to detect a word in a stream of connected speech...
correlated with language outcomes at 2 and at 4–6 years of age. This ability was not related to general intelligence measures, but correlated with vocabulary and grammar.

**Speed of Processing**

Given the rapid transient nature of the acoustic speech signal, it is logical that efficient processing of brief auditory temporal stimuli may be an important cognitive process underlying the development of language. Difficulties with rapid auditory temporal processing have been associated in some children with SLI [Tallal et al., 1985; Bishop et al., 1999], and may potentially play a contributing role in the disorder. Studies have found correlations between rapid temporal processing of acoustic signals and early language development. For example, Benasich et al. [2008] found that children with a family history of language disorders were not as efficient at detecting very rapid stimuli (70 ms between stimuli) as those without a family history. The differences in latency of response to these rapid signals at six months of age predicted language outcomes at 24 months. These correlations have been found to hold through ages 3 and 4 for cognitive and language measures [Choudhury and Benasich, 2011].

The speed of spoken word recognition has also been investigated as a factor in language development. Fernald et al. [2006] demonstrated that efficient processing of spoken language correlates with concurrent and later vocabulary development in TD infants and toddlers. In addition, speed of spoken word recognition at 25 months of age contributes unique variance to language and cognitive skills at age 8 years [Marchman and Fernald, 2008]. Fernald and Marchman [2012] investigated the processing speed of LTs in relation to their language outcomes 18 months later. They found those who started out in the lowest quintile at 18 months of age, but had faster processing speeds, were more likely to show accelerated vocabulary growth over the next 18 months, compared with LTs with less efficient processing. Their data are consistent with a model of cascading influences of processing on later development. This is a promising factor to consider in the prediction of language disorders in LTs.

**Working Memory**

A complete review of working memory models is beyond the scope of this review (see Baddeley [2012] and Courage and Cowan [2009], for overviews). Here we briefly review why working memory skills are relevant for language learning, adopting Baddeley’s model. In this account, working memory, a cognitive system that “mediates between perception, long-term memory, and action” [Baddeley, 2012, p. 25] is considered to be a four-component cognitive system comprised of a central executive, an episodic buffer, a phonological loop, and a visuo-spatial sketchpad. All but the latter are relevant here. The central executive is an attention system, controlling directed, focused, and sustained attention, and attention shifts, while inhibiting irrelevant information. The episodic buffer acts as a temporary store for chunks of information that link working memory, perception, and long-term memory. The phonological loop is believed to be necessary for learning new word-form to referent mappings (learning new words). This sub-system acts as a temporary store of newly encountered phonological forms that are linked to long-term memory with increased experience. The loop is susceptible to rapid decay and capacity limitations, both of which improve with age and language experience.

Executive functioning and language usually show moderate correlations in the 3–6 year age range; however this is thought to be due to the role language plays in helping children regulate their attention and behavior purposefully, rather than vice versa [Carlson, 2005; Hughes and Ensor, 2007]. Working memory skills are strongly linked to vocabulary learning in preschool children and deficits in working memory have been identified in children with SLI [e.g., Gathercole and Baddeley, 1990]. Deficits in complex working memory, inhibition, and attention skills are associated with SLI at a group level throughout early-to-middle childhood [Alloway and Gathercole, 2006; Im-Bolter et al., 2006; Martin et al., 2008].

The use of working memory as a predictor factor is in its early stages of investigation. However, there is some research on this related to predicting language outcomes in young children. Conboy et al. [2008] used behavioral measures such as retrieving objects hidden or placed in boxes to estimate development of executive function. They found that in 11-month-old infants, scores on these tasks were correlated with the ability to ignore speech variations that were irrelevant to their native language. Infants’ native speech discrimination was also related to their concurrent receptive vocabulary skills. The authors suggested the role of inhibition in development of neural commitment to native speech contrasts.

Studies have measured neural activity in the frontal regions associated with cognitive processes such as attention, working memory, and associative learning in relation to predicting language. This brain activity, known as resting frontal gamma power, at 16, 24, and 36 months of age significantly correlated with sentence structure scores at 4 and 5 years of age [Gou et al., 2011]. In a similar study, children with a family history of language disorders have been found to have lower gamma power density functions [Benasich et al., 2008]. This research indicates that better cognitive control and working memory skills at critical points in development may facilitate language development.

**Phonological Short-Term Memory**

Phonological short-term memory (PSTM) is a linguistic processing component of working memory. PSTM is usually measured by repetition of a set of nonsense words (nonword repetition; NWR), increasing in complexity from one to four or five syllables in length. There are several published measures available [e.g., see Gathercole et al., 1994; Dollaghan and Campbell, 1998]. PSTM is believed to play an important role in early language development [Gathercole and Baddeley, 1990]. A reduced capacity to store and process incoming phonological information leads to weaker or incomplete phonological representations of words, which are then more difficult to access and retrieve for production, resulting in slowed vocabulary acquisition [Gathercole and Baddeley, 1990]. The ability to recall sequences of words relies principally on PSTM [Baddeley et al., 1998]. There is substantial evidence to suggest that a limitation in PSTM could be a factor in language disorders [Graf Estes et al., 2007]. Within the SLI population, few children have PSTM abilities within the normal range; however, when it does occur, better PSTM ability is correlated with higher language and literacy performance [Gathercole and Baddeley, 1990; Bottig and Conti-Ramsden, 2001; Alloway and Gathercole, 2006].

Recent research has indicated that LTs also perform poorly on a test of NWR relative to their age-matched
peers. For example, Stokes and Klee [2009a,b] found that NWR was strongly related to expressive vocabulary size in 2-year-old children. In addition, Chiat and Roy [2008] reported that early NWR skills at age 2 and 3 were predictive of morphosyntactic development at age 4 and 5. It should be noted though that LTs with very few words generally will not participate in a NWR task. Stokes and Klee [2009a,b] reported that children who did not cooperate for NWR testing scored significantly lower on cognitive and language tests than children who did cooperate. Noncompliance notwithstanding, NWR is promising as a predictive measure of later SLI for LTs.

THEORETICAL ACCOUNTS

The overview of the multifactorial model is reflective of our theoretical perspective of SLI and the relationship between late talker status and subsequent development of LI. We adopt a domain general, rather than a domain specific perspective. Domain specific theories include a specific grammatical deficit [van der Lely, 2005] and a delay in setting the parameters of the grammatical system [Rice et al., 1995]. However these grammatical theories are challenged by evidence of nonlinguistic impairments. Consequently, we do not discuss domain specific theories (or linguistic theories) in this review. Domain general theories implicate poor temporal auditory processing [Tallal et al., 1985; Bishop et al., 1999], reduced verbal working memory capacity, either specific to phonological processing [Gathercole and Baddeley, 1990; Chiat, 2001] or more broadly for linguistic processing [Ellis Weismer and Evans, 2002; Montgomery and Evans, 2009; Baird et al., 2010], a generalized slow speed of processing [Ellis Weismer and Hesketh, 1996], and weaknesses in the procedural memory system [Ullman and Pierpont, 2005].

The domain general theories emphasize a cascading effect of lower level processing difficulties impacting on the development of higher level skills. Investigations of how children with SLI learn in tasks that recruit the procedural memory system have contributed to the current focus on learning mechanisms in children with SLI. Ullman's [2001] declarative-procedural model of language postulated two separate (but possibly interactive) memory systems. It is postulated that the procedural memory system, anchored in the interactive network comprising the prefrontal cortex, basal ganglia, and cerebellum, is responsible for the learning and storage of rule-based behaviors, including some motor [e.g., bike-riding] and language behaviors (e.g., syntax and phonology). The declarative memory system, anchored in the medial temporal lobe network, particularly the hippocampus, is responsible for the learning and storage of arbitrary (non-rule-based) events, facts, and life experiences, such as form-referent mappings in word learning.

Ullman and Pierpont [2005] proposed the Procedural Deficit Hypothesis (PDH) as an account for SLI, suggesting that SLI arises from a deficit in the procedural memory system and underlying brain structures. (In addition to the above sections on genetics and neurobiology, see Ullman and Pierpont [2005] and Ullman [2001] for an overview of the neural correlates of SLI in relation to the PDH account.) In this account, a grammatical LI reflects difficulty with learning rule-based language knowledge (syntax, morphology, and phonology) due to deficits in the memory system responsible for rule-based learning, the procedural memory system.

Recent empirical evidence supports the PDH, highlighting the difficulties that children with SLI have with both nonverbal and verbal procedural learning tasks. Tomblin et al. [2007] reported poor learning by adolescents with SLI on a visual procedural learning task (the Serial Reaction Time task), as did Lum et al. [2010] in their study of 7-year-olds with SLI. However, Lum et al. [2010] also reported significance between group differences on a verbal declarative memory task (the number of semantically unrelated word pairs recalled in a list task), suggesting that memory deficits may underlie lexical learning in SLI.

As Lum and Kidd [in press] indicated, while the procedural memory system is principally concerned with learning information that is sequential and probabilistic/statistical (e.g., grammar), and the declarative memory system is principally concerned with the association between stimuli to store a memory (e.g., mapping a heard phonological form to a referent in the visual field), the two systems interact. Further, working memory and the declarative and procedural memory systems also interact, given shared neurological substrates, particularly the prefrontal cortex [Lum et al., in press].

Given the observed deficits in visual procedural learning, verbal declarative learning, and NWR, which together are indicative of deficits in procedural, declarative, and working memory, Lum and Bleses [2012] suggested that a primary deficit in the latter may underlie observed deficits in the former. They showed that the poor performance of school-aged children (mean age 7.6 years) with SLI on a verbal declarative memory task was mediated by poor working memory abilities. Overall, there is an emerging view that school-aged children with SLI show memory deficits and difficulties with statistical learning [e.g., Evans et al., 2009]. There is also evidence of a strong association between implicit statistical learning and grammatical ability in TD pre-school children [Kidd, 2012].

IMPLICIT STATISTICAL LEARNING OF LANGUAGE IN TODDLERS

The findings on the role of implicit learning in SLI prompted Stokes et al. [Stokes, 2010; Stokes et al., 2012a, 2012b] to explore whether implicit learning deficits or differences could be detected in very young children who are LTs and hence at risk of SLI. This research focused on probable statistical cues to language learning during infancy. The language input that infants hear is often a continuous string of language without word boundaries (theoglosiachthenca). This continuous string would seem to provide a significant challenge to infants who need to 'crack the code' of language learning [Kuhl, 2004]. To overcome this challenge, infants take advantage of statistical (probabilistic) cues in the input that help in the identification of words [Saffran, 2003; Saffran and Graf Estes, 2006]. These cues include cross-syllable transitional (sequential) probabilities of sound sequences [Aslin et al., 1998; Thiessen et al., 2005] and recurring clusters of syllables [Swingley, 2005]. None of this learning occurs with feedback and it requires repeated or sustained input, all of which are the hallmarks of procedural learning. The question that Stokes et al. addressed was whether there might be statistical cues in the language input at the lexical, rather than sub-lexical, level that might influence vocabulary acquisition in toddlers. Such lexical cues would include phonological neighborhood density and word frequency.
Toddler's first spoken words are influenced by the sound (phonological) and whole word (lexical) characteristics of words in the ambient language, and the statistical regularities with which they occur [Stokes, 2010]. For example, toddlers' first words are short words that sound similar to many other words (those that share phonological strings) in the ambient language (e.g. cat has 35 similar words, or neighbors, such as mat, pat, cap, kit). Such words have high phonological neighborhood density (ND). Words that have few phonological neighbors are said to "reside" in sparse neighborhoods [mouth has six neighbors: math, mouse, myth, moth, south, mouth (verb)]. Stokes et al. [Stokes, 2010; Stokes et al., 2012a, 2012b] showed that the relationship between phonological ND and vocabulary size was very strong in 2-year-old children. Figure 2 shows the strength of the relationship for English-speaking children. The figure shows that when vocabulary size is very small (lower than -1.25 SDs from the mean of 0; z-scores), children's words are, on average, drawn from words in the input that have very dense phonological neighborhoods (more than 2 SDs above the mean ND of 0; z-scores).

The finding is remarkably robust across languages. In three languages (English, French, and Danish), children who were poor vocabulary learners had expressive lexicons comprised of far fewer sparse words than TD children of the same age. The amount of variance in expressive vocabulary size accounted for by phonological ND was 47%, 53%, and 41% for English, French, and Danish, respectively [Stokes, 2010; Stokes et al., 2012a, 2012b].

**Extended Statistical Learning**

On the basis of these results, Stokes et al. proposed a theory of Extended Statistical Learning to account for slow vocabulary growth. This theory argues that poor learners have a prolonged period of use of high phonological density as a cue to word learning. This initially successful statistical cue effectively blocks subsequent learning of words from sparse neighborhoods. The idea of blocking the use of more effective statistical learning mechanisms was first proposed by Aslin and Newport [2008], although these authors did not suggest specific mechanisms, such as using ND as a cue to learning. The question for clinicians and researchers is why would LTs be learning words from dense neighborhoods? We believe the answer may lie in differences in working memory abilities, given that Stokes and Klee [2009a] found that children with small expressive vocabularies performed poorly on a test of NWR.

Poor working memory abilities may limit the ability to achieve form-referent mapping of words from sparse phonological neighborhoods because these phonological strings occur infrequently in the ambient language input [Stokes et al., 2012b]. Hsu and Bishop [2011] suggested that deficits in working memory ability could underpin the ability to use statistical cues in the input in children with SLI. Longitudinal studies are now needed to explore whether LTs who develop SLI, when compared with LTs who normalize, show different patterns of lexical learning arising from differing learning mechanisms, such as the use of the phonological cues inherent in word forms, and PSTM abilities.

**CLINICAL RECOMMENDATIONS**

We can draw several clinical recommendations from this literature review. There is strong evidence to suggest that at least three factors at 2 years of age should be considered in clinical assessments, a family history of late talking, being male, and being less than 85% of predicted birth weight. We suggest here that linguistic processing skills also be assessed. Two-year-old children with language delay should be screened on a test of NWR, and this should be repeated at six-month intervals. We predict that children who become late bloomers will be the children who score higher on a test of NWR than those LTs who will become language impaired in the preschool years. This idea is speculative at present, and awaits research. In addition, we have suggested that children who begin as LTs and develop into children with SLI may have quite different language learning mechanisms from their TD peers. We suggest that language clinicians explore teaching high and low density words to children who are identified as later talkers, in an effort to explore children's learning mechanisms.

**SUMMARY**

At the outset of this article, we stated that distinguishing between the LTs who do and do not develop a language disorder at 4–5 years of age poses a diagnostic challenge. SLI is a heterogeneous disorder, with various subgroups and a changing profile for each individual across development. Recent research on the genetic and neurobiological influences on language development and impairment, including SLI, suggest that there are likely to be genetic variants that are related to individual differences in neurobiological development in children that in turn contribute to the heterogeneity of SLI. This heterogeneity is one of the reasons why it is difficult to identify accurate predictors of development from late talker status to SLI status. Attempts to date to identify predictive factors have at best been moderately successful. In
this review, we suggested that it is time to widen the search for predictive factors, so as to include measures of language learning mechanisms. The last decade has produced a wealth of evidence on the relationships among memory systems and language behaviors. Very recent work has shown that this relationship is present even in 2-year-old children. Children with expressive language delays have poor short-term memory abilities and we suggest that this memory deficit contributes to the use of learning mechanisms that differ from those of TD 2-year-old children. We propose a new framework for thinking about ELD. We propose that ELD is characterized not only by the quantitative metric of number of words produced, or the presence/absence of word combinations, but also by short-term memory abilities that impact on children's learning mechanisms. Improving our ability to predict SLI outcomes from late talker status could depend on the careful combination of the most promising predictors, be they genetic, neurobiological, and/or cognitive learning mechanisms.

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