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Developmental language learning impairments

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Abstract

Developmental language learning impairments (LLI) are one of the most prevalent of all developmental disabilities, can occur in children for a wide variety of reasons, and have been shown to co-occur frequently with other developmental social, emotional and behavioral disorders, as well as with academic achievement problems. Research pertaining to developmental LLI of unknown origin, with an emphasis on the continuum between oral and written language impairment, is the focus of this review. Given the complexity of language learning, research has focused on multiple levels of analysis, including linguistic, neuropsychological, genetic, neurobiological, and remediation studies. To date, the vast majority of data on LLI derive from studies focused on a single level of analysis. Although attempts have been made to integrate data across studies and multiple levels of analysis, this has proven to be problematic, given the heterogeneity of the subject populations used to study LLI, as well as the differences in ages, degree of impairment, and types of impairment included in each study. Given that LLI is a complex developmental disability, it is suggested that future research would benefit from taking a multiple levels of analysis approach with the same individuals, incorporating mathematical models designed to analyze dynamically changing complex systems, and studying individual differences in language learning, prospectively and longitudinally, throughout the most dynamic stages of the process.

Oral language is expected to develop naturally and effortlessly, without any need for formal instruction. One of the great wonders of parenthood is to observe a child's progress as language appears to automatically blossom and springs forth rapidly over the course of the first few years of life. Reading, on the other hand, does not develop naturally, but must be explicitly taught. Reading and writing are invented systems for translating aural language to a visual, written form. Individuals raised in cultures without access to formal education do not learn to read and remain illiterate throughout their lives.

Developmental language learning impairments (LLI) can occur in children for a wide variety of reasons and, as such, are one of the most prevalent of all developmental disabilities. Many developmental cognitive disorders, such as mental retardation, pervasive developmental disability (including autism), Attention Deficit disorder, Central Auditory Processing disorder, or specific syndromes, such as Down syndrome, Fragile X syndrome, and Kleinfelter syndrome, may include delay in language development. It has been estimated that approximately 20% of all children have some form of language learning impairment (Beitchman, Hood, Rochon, & Peterson, 1989; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990). The most common known causes of developmental LLI are hearing loss (including intermittent hearing loss resulting from chronic

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otitis media), general mental retardation, autism, and motor or structural defects affecting the oral musculature. However, even when these known causes of developmental LLI are excluded, over 7% of school aged children have significantly below average language development of unknown origin (Tomblin, Records, Buckwalter, Zhang, Smith, & O'Brien, 1997). Throughout this paper when we refer to individuals with LLIs, we are referring to this latter group for whom the origin of their disability is unknown.

Historically, oral and written LLIs (including dyslexia) have been viewed as separate disabilities. However, longitudinal research has shown that children with oral language impairments are at very high risk for reading and other academic failure and that early oral language abilities are among the best predictors of subsequent reading abilities (Rissman, Curtiss, & Tallal, 1990). Furthermore, many, but not all, children struggling to learn to read have been shown to have concurrent difficulty with one or more aspects of oral language, which includes phonology, morphology, semantics, and/or syntax (see Snow, Burns, & Griffin, 1998, for a review). Current research has also focused on similarities in the profile of nonlinguistic sensory, perceptual, motor, and cognitive deficits that characterize individuals with oral and/or written developmental LLIs. Although we will primarily address research that has investigated developmental oral language impairments of unknown origin, where appropriate, we will also discuss research that has focused on the continuum between oral and written language impairments. To incorporate the increasing evidence that there is, in many cases, a continuum of language and learning deficits that includes both oral and written language, we use the term LLI.

For unknown historical reasons and unlike reading, language development is not routinely formally assessed in young children. Unless a child has a distinctive articulation disorder or a profound expressive language delay, few children will ever receive any kind of formal speech or language assessment, much less ongoing testing similar to that done to access reading achievement. It is not surprising, therefore, that the majority of children with developmental oral language impairments are overlooked. Similarly, when a child begins to struggle in school for the first time, the focus is almost always on assessing and remediating reading per se, rather than on the possible precursors in oral language development. The concern with reading failure is understandable, given that lower reading ability has been linked to lower academic achievement, higher adjudication rates, higher school dropout rates, greater incidence of social and emotional problems, and lower economic and occupational outcomes (Snow et al., 1998). However, if we are going to make progress in improving reading outcomes, we may need to begin by better understanding the foundation in oral language upon which reading must be developed, specifically, the continuum between oral and written language learning disabilities.

Given the complexity of language learning, it is likely that multiple levels of analysis will be needed to better understand the various potential causes and determinants of this prevalent developmental disability. In this paper we will review the most commonly used approaches and methodologies that have been employed to study LLI, including linguistic, neuropsychological, genetic, neurobiological and remediation studies. Given the developmental nature of LLI, the majority of research in this field has been done using either cross-sectional or longitudinal designs, which can evaluate individuals with LLI across various ages.

Definition

The first published report of children who had language impairments dates back to Gall (1835). Over the remainder of the nineteenth and early twentieth centuries, cases of children with "infantile aphasia" or "congenital aphasia" were reported. In a classic paper, Benton (1964) described children who seemed to be developing normally in every respect except for an unexpected delay in their oral language development. He classified these children as "developmentally dysphasic," noting several similarities with the pattern of both receptive and expressive language deficits that

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are generally seen in patients with acquired brain damage leading to aphasia. However, neurological examination did not demonstrate evidence of any frank neurological deficits, such as stroke, brain lesion, hemiplegia, or seizure disorders. Furthermore, this developmental language delay occurred in the absence of hearing impairment and oral motor or structural deficits such as cleft palate. Other than what appeared to be a relatively pure deficit affecting language development, the only other common problem shared by these children was difficulty with what Benton (1964) described as "higher order central auditory processing." Specifically, Benton noted that developmentally dysphasic children commonly had difficulty with nonlinguistic auditory sequencing and memory, as well as with language per se.

The term developmental dysphasia has not been widely used for many years. It was replaced by the term specific language impairment (SLI), which is still most often used in research studies, despite growing evidence that the deficits of most SLI children are not specific to language (Leonard, 1998). More recently, the term LLI has begun to be used to take into account the longitudinal evidence that early language impairments generally lead to more widespread learning disabilities, specifically, reading, writing, and spelling problems.

Although the terminology may have changed, the symptomatology that most commonly classifies children with developmental language impairment of unknown origin has not. Diagnosed primarily by the exclusion of known causes for language delay, this heterogeneous group of children is primarily classified based on a significant expressive and/or receptive and expressive language delay as assessed by standardized language tests. The degree of language delay required for this diagnosis has varied from study to study and clinically, but it is generally ≤1 *SD* from the mean of normal. A discrepancy between nonverbal intelligence and verbal abilities used to be included in the diagnostic criteria for specific developmental language impairment, at least for inclusion in research studies. However, as little difference has been found in the profiles of children with LLI with or without this IQ discrepancy, this criterion is rarely used today. For the purpose of this article, the term LLI is intended to be inclusive of other terms commonly used in the literature, such as SLI, developmental language impairment, and developmental dysphasia. Furthermore, when we speak here of LLIs, unless otherwise specified, we are referring to those developmental language disabilities that are of unknown origin (i.e., not related to other primary deficits or known syndromes).

Prevalence

Epidemiological research studies have shown that nearly 20% of all children have some form of language deficit, the majority of which are related to other primary deficits, such as hearing loss, mental retardation or frank neurological deficits. (Beitchman et al., 1989; Shaywitz et al., 1990). Recently it was shown that even when language impairments that can be accounted for by these other primary deficits are excluded, 8% of boys and 6% of girls enter school with LLI of unknown origin, a much higher number than previously thought (Tomblin et al., 1997). More surprising, however, was the finding that over 70% of these children had never been identified by their parents, physicians, or teachers as having developmental language learning difficulties, despite the finding that formal testing demonstrated that they were 1.25 *SD* or more below the mean of normal, meeting the clinical diagnostic criteria for SLI (Tomblin et al., 1997).

Diagnosis

The differential diagnosis of developmental LLI is based on behavioral evaluations that include audiological, neurological, psychological, educational, speech, and language assessments. *The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association, 1994) uses the term Developmental Language disorders, which it divided into two basic categories: Expressive Language disorder, affecting language production, and Mixed Receptive–Expressive Language disorder, a disorder en-

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compassing both language comprehension and production deficits. Comprehension and/or production problems may each occur within one or more of the components of language, including phonology, morphology, semantics, and/or syntax. Problems with pragmatics (interactive conversational skills) also occur frequently. A high proportion of children with developmental LLI also have difficulty with speech articulation, that is, difficulty clearly and correctly producing one or more of the speech sounds of their language. However, speech articulation defects and LLI can occur independently of each other.

Children with developmental LLI have been shown to be at risk for childhood behavioral disorders as well. For example, clinical research studies showed that children referred to child guidance clinics for a variety of behavioral and emotional problems were found to have a higher than expected incidence of developmental language learning disorders (Chess & Rosenberg, 1974; Grinnell, Scott–Hartnet, & Glasier, 1983; Gualtieri, Koriath, Van Bourgondein, & Saleeby, 1983). Conversely, children diagnosed with developmental language learning disorders have been found, on examination, to have a preponderance of long-lasting behavioral and emotional problems (Baker & Cantwell, 1987a, 1987b; Beitchman, Wilson, Johnson, Atkinson, Young, Adlaf, Escobar, & Douglas, 2001). Moreover, at least one study suggests that an increasing number of emotional problems may emerge over time in children diagnosed as LLI. A well-selected sample of LLI children, followed longitudinally from ages 4 through 8 years, showed a significantly greater proportion of behavior problems than a matched control group (Benasich, Curtiss, & Tallal, 1993; Tallal, Ross, & Curtiss, 1989). At the conclusion of the study, the percentage of children with LLI whose behavior problem scores were in the clinical range had increased significantly (from 11% at 4 years to 32% at 8 years); however, this was not the case for the age-matched control children (from 2 to 9%).

Additional evidence for the long-term co-occurrence of LLI and behavioral problems comes from data reporting that approximately 80% of juvenile delinquents who were tested had LLIs (Beitchman, Nair, Ferguson, & Patel, 1986; Hall & Moats, 1999). Taken together, the research cited clearly illustrates the high rates of co-occurrence seen between LLI and a variety of behavioral, psychiatric, and social problems. However, little is known about how these developmental disabilities interrelate or the precise neural mechanisms that might drive the observed comorbidity.

Longitudinal Outcomes

Longitudinal research studies that have followed children with early developmental language impairments, either retrospectively or prospectively from the preschool years, are consistent in demonstrating that developmental oral language impairments generally persist, albeit in weaker form, into later childhood, adolescence, and, in many cases, adulthood. Although most of the subjects who were studied showed improvement in language over time, with some reaching normal limits, a very high percentage continued to have significant oral language problems at follow-up, even into adulthood (Aram, Ekelman, & Nation, 1984; Aram & Nation, 1980; Bishop & Edmundson, 1987; Hall & Tomblin, 1978; Stark, Bernstein, Condino, Bender, Tallal, & Catts, 1984). In the largest prospective longitudinal study to date, Tallal, Curtiss, and Kaplan (1988) assessed 101 children with specific developmental language impairment, including both chronological age and language age matched control groups. All groups were assessed annually for 5 years. The majority of the children with language impairments were receiving speech therapy and/or special services in school and thus made language gains across the years of the study. Nonetheless, they continued to score significantly lower than expected for their age on all aspects of language throughout the study period. Moreover, no evidence emerged, across the 5 years of the study, that these children were catching up to their age matched controls. Children diagnosed as SLI remained, on average, 1 to 2 years behind their peers throughout the study.

All longitudinal studies to date have demonstrated that a majority of children with oral

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language deficits identified in early childhood subsequently develop substantial academic achievement problems, specifically dyslexia, which is characterized by deficits in reading, writing, and spelling. Samuel Orton (1937) was the first to speculate that the basis of specific reading disabilities (dyslexia) may lie in the linguistic system. Over the past two decades, evidence supporting a continuum between oral and written language impairments has continued to mount. This research compared children classified with oral language impairments with those classified based on reading deficits and showed that both groups are characterized by a variety of sensory, perceptual, and motor problems, as well as oral language deficits, particularly in the areas of phonology and grammatical morphology (see Leonard, 1998, for a review; Catts, 1989; Perfetti, 1985; Stanovich, 1988, Tallal et al., 1988; Vellutino, 1979). Whether these common deficits derive from speech-specific mechanisms or more basic neural processing deficits has been the focus of considerable research and is currently the center of theoretical debate in this field (see Farmer & Klein, 1995; Nicolson, Lenane, Singaracharlu, Malaspina, Giedd, Hamburger, Gochman, Bedwell, Thaker, Fernandez, Wudarsky, Hommer, & Rapoport, 2000; Stein & Talcott, 1999; Tallal, 1998; Tallal, Miller, & Fitch, 1993, Wright, Bowen, & Zecker, 2000, for reviews).

Etiology

Considerable research attention has been focused on trying to understand the causes and determinants of LLI. Given the lack of heterogeneity among children showing delayed oral and written language development and the diversity of symptomatologies that can accompany LLI, it is unlikely that a single etiology will be found. Etiological research to date has focused on linguistic, neuropsychological, genetic, and neurobiological studies. Virtually all the data pertain to group differences. Because of the heterogeneity of the LLI population, the reported results generally apply to many but not all, individuals with LLI. Similarly, differences across studies likely result from different methods and criteria used for ascertaining subjects with LLI, the matching variables of which are chosen for control populations, and differences in the age of subjects participating in each study.

It must also be emphasized that LLI is a developmental disability. The neural mechanisms necessary for developing higher cortical functions such as speech, language, and reading are likely to be different from those necessary or sufficient to maintain these skills, once developed. Unfortunately, there is a growing trend to use older subjects, especially adults with dyslexia, in studies aimed at addressing issues of the etiology of LLI. Older subjects are not only easier to ascertain, but they are also better able to participate in complex studies that require sustained attention or the ability to remain motionless during brain imaging procedures. However, given that we now know that experience-dependent brain plasticity can significantly alter brain morphology, physiology, and behavior (see Merzenich & Jenkins, 1995), caution must be exercised when interpreting patterns observed in older children or adult subjects with LLI, who have had many years to compensate for their developmental deficits. Many differences across studies, as well as theoretical disagreements, may likely relate to differences in the age of subjects studied. Moreover, the pattern and severity of oral and/or written language deficits, not only at the time of study but also earlier in development, are likely to exert a highly significant effect on the results.

Longitudinal studies have demonstrated that the severity, as well as the pattern, of disability change significantly across development for most individuals with LLI. Therefore, data pertaining to the etiology of developmental cognitive disabilities such as LLI become increasingly more difficult to investigate and interpret with increasing age. What we are likely to observe in adults with a life-long history of developmental LLI are the outcomes of compensatory strategies on brain organization, rather than the primary causes of these deficits. A failure to keep the developmental nature of LLI clearly in mind when interpreting data pertaining to etiology has contributed to considerable confusion and controversy in this field.

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Linguistic studies

Considerable advances have been made in understanding the specific linguistic deficits of children with LLI. Research has demonstrated particular patterns of difficulty within the linguistic components of phonology, syntax, and morphology. Specifically, deficits in both the understanding and use of grammatical morphology have been demonstrated (see Bishop, 1997, and Leonard, 1998, for a review). One of the major research questions in the past focused on whether the pattern of linguistic deficits of these children was more consistent with delay or with deviance in language learning. The results of the majority of longitudinal studies have shown that children with developmental language impairments develop the linguistic structures of language along a similar linguistic trajectory to that observed in normally developing younger children, albeit considerably more slowly (Curtiss, Katz, & Tallal, 1992). As such, there is little evidence at this time that children with developmental LLI make linguistic errors that are deviant or substantially different from younger children at the same stage of language development. Rather, children with LLI appear to take far longer to progress through the stages of normal language development, requiring approximately 2 years to develop the equivalent of 1 year of language, and some language components may continue to be deficient even into adulthood (Curtiss, Katz, & Tallal, 1992; Rissman et al., 1990). It is important to note that a similar pattern occurs across most populations of children experiencing developmental LLIs, regardless of clinical diagnosis.

One of the most fruitful research approaches that have been used to explore the linguistic specific aspects of LLI has been to look cross-linguistically at children who are struggling to learn their native language in different countries. The most important theoretical reason to do cross-linguistic research is that different languages have different structures and, as such, one language can have linguistic properties useful for testing hypotheses based on another. If there are linguistic specific deficits that are hypothesized to cause specific developmental LLIs, then we should expect to see these same linguistic impairments occurring across languages, regardless of the structure of the native language the child is attempting to learn. Leonard (1998), who is one of the major contributors to this research approach, exhaustively reviewed the outcomes of decades of cross-linguistic research looking for evidence for specific linguistic "universals" that are impaired in children around the world experiencing LLIs. Contrary to expectation, surprisingly few consistent patterns of linguistic deficits were found in cross-linguistic studies. Rather, the most consistent findings appear to support a perceptual, rather than linguistic, basis for developmental language disorders. What has been shown across all the languages studied is that those linguistic structures that are brief, unstressed, or of weak phonetic substance and that occur less frequently in a particular language are the most difficult for children to learn and the most delayed in children with LLI (Leonard, 1998). That the same order of linguistic development occurs across populations and language environments gives strong support to the premise that there is a potent metric of sheer difficulty (whether representational or perceptual) that is imposed on the learning of linguistic structures and contents.

Neuropsychological studies

Neuropsychological studies have focused on those sensory, perceptual, motor, attention, and memory substrates presumed to be requisite components of higher cortical functions, including language, reading, writing, and spelling. The most extensive studies in this domain to date have utilized behavioral methodologies. Recently, however, newer imaging methodologies also have been employed to address the neural basis of these functions. One of the most prominent debates in the field of language learning disabilities is whether these disabilities are linguistic specific or rather result from constraints in more basic neural functions on which language learning depends. Although some authors continue to question the existence of nonlinguistic deficits in individuals with oral and/or written language impairment (Mody, Studdert–Kennedy, & Brady,

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1997), these deficits have been so well documented at this point that there seems little question that they occur. After extensively reviewing the literature on nonlinguistic processing deficits, Leonard (1998) states that:

> The conclusion that children with SLI have difficulty processing brief or rapidly presented stimuli seems indisputable. These findings are so consistent and demonstrable across tasks and stimulus variations that it is difficult to imagine that they are not an important piece of the SLI puzzle. (p. 145)

What is less well agreed upon is the prevalence of nonlinguistic deficits, the precise nature of these deficits, the modalities affected, the neurobiological basis of these deficits, and whether nonlinguistic deficits are causal to, or merely co-occur with, developmental linguistic deficits. Attempts to integrate this rapidly expanding body of research have spanned studies of both oral and written language impairments (particularly dyslexia) and it is currently one of the central areas of research in this field. Such studies have resulted in the formulation of several prominent hypotheses, including the rate processing constraint hypothesis (see Tallal, 1998, for a review), the magnocellular deficit hypothesis (see Stein & Talcott, 1999, for a review), the cerebellar deficit hypothesis (see Nicolson et al., 2000, for a review), the double deficit hypothesis (see Wolf, 1986, for a review), and the attentional dwell time hypothesis (Hari, Saaskilahti, Helenius, & Uutela, 1999). It is interesting that all of these hypotheses have a component in common that includes a constraint in the speed of information processing and/or production that is postulated to disrupt various components of language learning, beginning with the acquisition of phonological representations. These hypotheses are not mutually exclusive but rather focus on different levels of analysis, including cognitive, anatomical, and physiological.

The rate processing constraint hypothesis (Tallal, 1998) assumes a direct impact on the learning of the linguistic system, specifically, on the precision of categorical representation of individual phonemes, as well as on grammatical morphology. Rate processing deficits have been shown in various sensory modalities (auditory, visual, and tactile, as well as cross-modally), and in the motor system (Laasonen, Tomma–Halme, Lahti–Nuuttila, Service, & Virsu, 2000; Stark & Tallal, 1988). Considerable data have now accumulated supporting a pansensory/motor rate processing constraint in infants, children and adults with LLIs (see Benasich & Tallal, in press, for review; see also Tallal, 1998). Specifically, individuals with oral and/or written language impairments have been shown to be slower in processing and producing brief, rapidly successive events, generally in the 10s to 100s of millisecond time range, regardless of sensory modality. It has been hypothesized that this dynamic processing rate constraint has a particularly detrimental effect on phonological processing, as many acoustic changes occur in rapid succession within this time frame within syllables and words. In support of this hypothesis, research has demonstrated that perception, as well as the production of those speech syllables that incorporate brief, rapidly successive acoustic cues, are the most problematic for these children (Stark & Tallal, 1979; Tallal & Stark, 1981; Tallal, Stark, & Curtiss, 1976). Moreover, Tallal and Piercy (1975) demonstrated that speech discrimination significantly improved when the rapidly successive acoustic cues within speech syllables were extended in duration using computer synthesis. This finding has led to the development of novel remediation strategies using acoustically modified speech for training a variety of speech, language, and reading skills (Merzenich, Jenkins, Johnston, Schreiner, Miller, & Tallal, 1996; Tallal, Miller, Bedi, Byma, Wang, Nagarajan, Schreiner, Jenkins, & Merzenich, 1996;), as discussed in the section on remediation.

Another line of evidence providing perhaps the strongest support for a causal role of rate processing constraints on language development comes from prospective, longitudinal studies that follow infants born into families with a positive history for LLI. These studies showed that, as a group, infants born into families with a positive history of developmental LLI demonstrate longer processing times for both nonverbal and verbal acoustic

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stimuli than matched infants born into families with a negative family history (Benasich & Tallal, 1996, 1998; Leppänen, Pihko, Eklund, & Lyytinen, 1999). Furthermore, when followed prospectively, nonverbal auditory temporal integration thresholds identified at 6 months of age were shown to be highly predictive of the rate of language development; the infants showing the slowest processing rates were subsequently the most likely to be delayed in language learning (Benasich & Tallal, in press; Trehub & Henderson, 1996).

In older children and adults, highly significant positive correlations between nonverbal auditory and visual dynamic sensory sensitivity and reading decoding (phonological and orthographic, respectively) were found across a number of studies (see Farmer & Klein, 1995, and Stein & Talcott, 1999, for reviews). It is important that robust relations were shown between dynamic auditory and visual sensitivity and literacy skills in normally developing children as well (Talcott, Witton, McClean, Hansen, Rees, Green, & Stein, 2000). These recent findings provide strong, empirical evidence demonstrating that individual differences in dynamic sensory sensitivity play an important role in the development of both oral and written language, across the entire range of development.

The proponents of the "linguistic-specific" hypothesis strongly dispute these data. They exclude any causal role for nonlinguistic processing constraints as a possible etiology for LLIs. Rather, they support a nativist view that proposes that language emerges within a discrete neural module as a function of an innate and domain-specific ability that needs only to be triggered by verbal input from the environment, that is, by normal childhood exposure to native language (Chomsky, 1971, 1975; Crain, 1991; Liberman, 1966; Liberman, Cooper, Shankweiler, & Studdert–Kennedy, 1967; Liberman, & Mattingly, 1985; Pinker, 1994).

However, even if this were strictly the case, acoustic processing, adaptive learning, and experience-dependent brain plasticity must somehow play a role if children are to learn the aural phonology of their own native language (Brown, 1958; Jusczyk, 1993; Skinner, 1957). For example, prelingually deaf children have great difficulty learning aural phonology, despite their ability to learn sign language. They also are deficient in learning to decode written language, presumably because they have never developed the acoustic phonetic representations that need to be paired with orthographic representations when learning to read. Jusczyk (1997) suggests a middle road through such controversies. He posits that language development is neither "hard-wired nor totally unrestricted," but rather a constrained process termed "innately guided learning," involving general perceptual mechanisms in conjunction with a set of innate biases to attend closely to acoustic signals with specific properties (Jusczyk, 1997, p. 198). The cognitive neuroscience perspective posits a similar resolution, albeit based more tightly on nonspecies-specific sensory motor processes and innate biases, as well as progressive, experience-dependent shaping of the neural circuitry underlying language learning (Benasich & Read, 1998).

Genetic studies

Over the past decade, evidence was collected in support of the hypothesis that developmental language impairments aggregate in families and may contain a genetic component (Bishop, North, & Donlan, 1995; Brzustowicz, 1996; Gopnik & Crago, 1991; Lahey & Edwards, 1995; Lewis & Thompson, 1992; Rice, Haney, & Wexler, 1998; Spitz, Tallal, Flax, & Benasich, 1997; Tallal et al., 1989; Tomblin, 1989; van der Lely & Stollwerck, 1996; see Stromswald, 1998, 2000, for a comprehensive review of family aggregation studies). Initial reports of family aggregation derived from individual family case histories (Byrne, Willerman, & Ashmore, 1974; Ingram, 1959; Luchsinger, 1970). Large group reports (Neils & Aram, 1986; Tallal et al., 1989; Tomblin, 1989) and case-control family studies (Neils & Aram, 1986; Tallal et al., 1989; Tomblin, 1989) followed. Other studies focused on examining the concordance of language impairment in twins as a means of looking at the rate of heritability (Bishop et al., 1995; Lewis & Thompson, 1992; Tomblin & Buckwalter,

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1998). To date, most studies were based on retrospective questionnaire data collected from family members of language-impaired probands (the ascertained individual with the disorder that identifies a family).

Very early family studies were based on questionnaire data and looked at family histories of children identified as language impaired. Such studies reported that between 28 and 63% of these children had at least one other family member who was impaired. Unfortunately, none of these studies provided enough information to determine the nature of the language problem(s). The wide range of results most likely reflects the fact that very different criteria were used across studies for the classification of language impairment. Based on case history profiles, Hurst, Baraitser, Aiger, Graham and Norell (1990) reported the pedigree of a three-generation family containing 30 members. More than 50% (16) of the family members had a history of speech and/or language difficulties, the pattern of which was consistent with an autosomal dominant mode of inheritance. Gopnik and Crago (1991) tested 20 members of this same family and found that only 7 were within the normal range of language development at the time of testing. This family was widely reported as demonstrating a highly specified, genetically transmitted defect in the use of grammatical suffixation rules (Gopnik, 1990; Gopnik & Crago, 1991; Pinker, 1991, 1994), thus supposedly supporting the existence of genes specific to grammar. However, more detailed study showed that the phenotype is quite broad, encompassing virtually every aspect of language. Furthermore, in affected family members, deficits in language are accompanied by severe orofacial dyspraxia, mental retardation, impairments in memory, and unintelligible speech (Vargha–Khadem, Watkins, Alcock, Fletcher, & Passingham, 1995). As such, this family is not representative of the vast majority of cases of LLI.

More recent studies provide evidence of family aggregation in quantitatively assessed populations of children with developmental language impairments. Bishop and Edmundson (1986) demonstrated a significantly increased frequency of affected primary and secondary relatives for a group of children with language impairment, compared to a group of normally developing control children. Similarly, Robinson (1991) found that 28% of the language impaired children he studied had first-degree relatives with a history of speech delay and 20% had first-degree relatives with a history of learning problems. Similarly, Neils and Aram (1986) reported that 20% of the language impaired children they assessed had family members with a spectrum of speech and language problems compared to a control group in which the rate was only 3%. Tomblin (1989) found similar results when he studied the family histories of 51 language impaired second grade children and 146 control children. In this study, family members were only considered affected if they reported having received speech–language intervention. Using these criteria, 23% of the proband families, but only 3% of the control families, reported a significant family history for language impairment.

Tallal and colleagues (1989) explored the relationship between language impairment and learning problems, using a family history design. They evaluated the incidence of language and/or learning problems in the family members of children with SLI as compared with matched controls. Based on the self-report of either language or learning problems, the families of language impaired children were more likely to have a positive family history, higher average frequency of impairment, a higher rate of affected siblings, and a higher rate of affected mothers and fathers. Approximately 40% of the primary family members of children with language impairment were found to have some kind of language and/or learning problem, including general academic failure.

Only a few studies have included concurrent test data of probands and all of their primary relatives from families in which a child has been identified as language impaired (Tallal, Hirsch, Realpe–Bonilla, Miller, Brzustowicz, & Flax, 2001; Tomblin & Buckwalter, 1994). Tallal et al. (2001) were the first to employ a case-control family study design in which the current language-related abilities of all biological, primary relatives (mother, fa-

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ther, siblings) of probands with specific developmental language impairment, as well as matched controls and their primary relatives, were assessed to investigate familial aggregation for language disorders. Current test data from each family member showed the rate of language impairment for the mothers, fathers, sisters, and brothers of the impaired probands to be significantly higher (31%) than for control family members (7%). Impairment rates for fathers and mothers were approximately equal, whereas the rates for brothers were significantly higher than for sisters. In the families of the probands with language impairment, language impairment occurred in 13% of offspring (excluding the proband) with neither parent affected, 40% of offspring with one parent affected and 71% of offspring in families with both parents affected.

Family aggregation research designs can also be used to investigate hypotheses pertaining to the incidence of co-occurrence of language impairments and reading impairments, as well as the incidence of rapid auditory processing impairments. In a recent multigenerational family study, children identified as having LLI were ascertained as probands (Flax, Realpe–Bonilla, Hirsch, Nawyn, & Tallal, 2000). All subjects (probands as well as their family members) were classified based on current testing, using a battery of language, reading, and auditory rate processing tests. Results showed that the incidence of language impairment in these families were comparable to those previously reported in the literature. In addition, results revealed a very high degree of co-occurrence within individuals of language, reading and auditory rate processing impairment. This pattern of co-occurrence was found for probands as well as their nuclear and extended family members. Surprisingly, only 24% of family members diagnosed as language impaired met the diagnostic classification for language impairment alone. The other 76% also met the classification for reading impairment and/or rapid auditory processing impairment. Likewise, only 18% of family members diagnosed as reading impaired based on standardized reading tests met the diagnostic classification for reading impairment alone, whereas the other 82% showed a co-occurrence of reading, language and auditory rate processing impairment. Males consistently scored lower than females on measures of language and reading while females scored lower on measures of rapid auditory processing. Results from this multigenerational family study, based on LLI probands, demonstrate that when language impairments occur within families, these impairments are rarely specific or confined to oral language functions. Rather, they generally co-occur with reading and/or rapid auditory processing impairment, with males demonstrating impairments more often than females.

Quantitative behavioral genetics offers researchers the opportunity to partition phenotypic variance occurring across families (as measured by behavioral tests) into two main variance components, those of genetic and environmental influences. The genetic (heritable) component can be calculated in a variety of ways, including classical twin, adoptive twin, sibling pairs, parent–offspring pairs, and extended family designs, with the remaining variance not accounted for in heritability estimates attributed to environmental influences.

Twin studies take advantage of the fact that monozygotic twins should have identical genetic material and dizygotic twins share an average of 50% of their genetic material. Therefore, monozygotic twins should be concordant for inherited traits approximately twice as often as dizygotic twins. In line with this expectation for genetic transmission, Lewis and Thompson (1992) looked at 57 sets of same sex twins (32 monozygotic, 25 dizygotic) and found a concordance rate of language impairment of .86 for monozygotic twins and .48 for dizygotic twins. The impairment rate for other family members ranged from 23% (monozygotic) to 27% (dizygotic), which is consistent with affected rates found in other family aggregation studies. Bishop and colleagues (1995) reported a concordance rate of .70 for monozygotic twins and .46 for dizygotic twins, and concordance was stronger for male twins than for females. Tomblin and Buckwalter (1998) found the highest rate of concordance reported across studies for language impairment, a concordance rate of .96 in monozygotic twins and .69 in dizygotic twins.

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Family aggregation studies can determine whether specific disorders aggregate in families and the incidence and pattern of occurrence, whereas twin studies can estimate the degree of heritability of a disorder. However, neither of these approaches can determine whether a disorder is actually genetic or, more specifically, identify which gene(s) may be responsible. For this purpose, gene linkage studies that aim to link the phenotype with the genotype are necessary. Gene linkage studies of complex behavioral disorders such as LLI rely on first correctly identifying the phenotype (behavioral profile) of interest in multiple related individuals and then linking this phenotype to the genotype (from DNA usually acquired from a blood test or cheek swab). For gene linkage studies, either multiple family members from large extended families or a large number of smaller families or sibling pairs need to be ascertained, based on an affected proband. Although specific genes have been linked for dyslexia, as well as for speech impairments related to orafacial dyspraxia, to date, no specific genes have been linked for LLI. The data currently reported certainly do suggest a potential genetic component underlying at least a subset of individuals with LLI. However, it should also be kept in mind that across all studies to date there are between 30 and 60% of probands with language impairments that have been found to be the only member of their family with LLI.

Neurobiological studies

Individuals with LLI have been shown to have neuroanatomic differences compared to normally developing controls. Volumetric and asymmetry differences were described in brain imaging studies, including a lack of the normal left-greater-than-right pattern in the planum temporale (located in Wernicke's area) and abberant asymmetry in the parietal and frontal regions (Cowell, Jernigan, Denenberg, & Tallal, 1995; Jernigan, Hesselink, Sowell, & Tallal, 1991; Larsen, Hoien, Lundberg, & Odegaard, 1990; Leonard, Voeller, Lombardino, Morris, Hynd, Alexander, Anderson, Garofalakis, Honeyman, & Mao, 1993; Tallal, Wood, Buchsbaum, Flowers, Brown, & Katz, 1990). It is hypothesized that such anomalies in anatomic asymmetry in the frontal and posterior temporal regions may be related to the decreased metabolic activation in these same areas seen in individuals with LLI (Hagman, Wood, Buchsbaum, Flowers, Katz, & Tallel, 1992; Neville, Coffey, Holcomb, & Tallal, 1993; Rumsey, Andreason, Zametkin, Aquino, King, Hamburger, Pikus, Rapoport, & Cohen, 1992). Group differences for anatomical and functional measures were found to be significantly correlated in several studies with performance differences on tasks requiring rapid auditory processing, irrespective of whether the stimuli were verbal or nonverbal. Moreover, Jernigan and colleagues reported reduced overall cortical and subcortical volume in children with SLI (Jernigan et al., 1991), as well as in children with more global cognitive deficits (Jernigan, Bellugi, Sowell, Doherty, & Hesselink, 1993).

A number of neuropathological studies support the hypothesis that brain abnormalities associated with LLI (e.g., ectopias and microgyric lesions) may occur very early in prenatal life. Focal cortical anomalies that may result from errors in neuronal migration were identified in postmortem analysis in many, but not all, brains from dyslexic individuals (Galaburda & Kemper, 1979; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Humphreys, Kaufmann, & Galaburda, 1990). In addition, Galaburda and colleagues showed that there are alterations in the large neuron system (i.e., magnocellular neurons) in the visual processing area of the thalamus (lateral geniculate nucleus, or LGN), which is responsible for processing fast, low contrast stimuli, as well as in the auditory processing area of the medial geniculus nucleus (MGN; Galaburda & Livingstone, 1993; Galaburda, Menard, & Rosen, 1994; Livingstone, Rosen, Drislane, & Galaburda, 1991). Specifically, there are fewer magnocellular neurons, and this class of neurons is also smaller in both the LGN and MGN of the thalamus in dyslexic as compared to control brains. Psychophysical studies with dyslexic individuals showed that the ability to process fast, low contrast visual stimuli, as well as dynamic auditory events,

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is impaired relative to controls (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Stein & Talcott, 1999; Stein & Walsh, 1997). Evidence in support of a magnocellular deficit is evident in behavioral assessment and is also reflected in neurophysiological measurements that show that event-related potentials (ERPs) to rapidly presented visual stimuli have longer latencies in dyslexics than controls, reflecting a slower neural response (see Leppanen et al., 1999). Additional evidence in support of a system-wide rate processing constraint derive from studies using functional magnetic resonance imaging (fMRI) in dyslexics. Dyslexics were found to have significantly decreased activation of cortical area middle temporal in comparison to controls when processing dynamically changing visual stimuli (Eden, VanMeter, Rumsey, Maisog, Woods, & Zeffiro, 1996), as well as a failure to activate the frontal operculum in response to rapidly changing acoustic stimuli (Temple, Poldrack, Salidis, Deutsch, Tallal, Merzinch, & Gabrieli, 2001). Findings of magnocellular-specific deficits in both the auditory and visual modalities were sustained by experimental tests in many, but not all, studies and are currently the focus of active research (see Habib, 2000, and Wright et al., 2000, for reviews).

Stein (in press) suggests an intriguing model for a possible neural–immune interaction that may underlie both the magnocellular-specific deficits, as well as cerebellar deficits, that are widely reported in individuals with LLIs (see Nicolson et al., 2000, for a review). Stein hypothesized that a subset of neurons, including those most responsible for timing in the 10s of millisecond time range (e.g., magnocellular neurons in the visual and auditory thalamic regions, large cells in the dorsal column somaesthetic nuclei, and the dense magnocellular input to the cerebellum), comprise a unique population of neurons. These neurons arise from a separate developmental lineage with common surface antigens, heavy mylelination, and rapid membrane dynamics, which contribute to their exquisite temporal resolution (Hockfield & Sur, 1990; Stein & Glickstein, 1992). However, these common characteristics may also render these neurons, as a class, more vulnerable to immunological attack. It is proposed that the development of these common surface antigens is regulated by class 1 major histocompatibility complex (MHC) molecules (Corriveau & Shatz, 1998). This intriguing hypothesis may provide an important link across several prominent behavioral, physiological, neurobiological, and genetic hypotheses, given that both phonological and orthographic impairments in dyslexics were linked to a site on the short arm of chromosome 6 near the tumor necrosis factor and MHC immunological sites (Cardon, Smith, Fulker, Kimberling, Pennington, & DeFries, 1994; Fisher, Marlowe, Lamb, Maestrinin, Williams, Richardson, Weeks, Stein, & Monaco, 1999; Grigorenko, Wood, Meyer, Hart, Speed, Shuster, & Pauls, 1997).

Given that LLI may involve basic sensory, perceptual, and motor abilities that support language but are not speech specific, it is possible to examine such processes in animal models (Fitch, Tallal, Brown, Galaburda, & Rosen, 1994). Galaburda and his colleagues developed rodent models with induced neocortical microgyric lesions designed to mimic those seen in the brains of dyslexics. They recently collaborated with Fitch and her colleagues, who developed a rodent behavioral paradigm modeled after tasks used to identify rapid auditory processing thresholds in humans. This team demonstrated that rats with induced cortical microgyric lesions, like humans with LLI, showed significant deficits in rapid auditory processing, but normal processing of the same stimuli presented more slowly. The pattern of performance seen in these rats was strikingly similar to that seen in humans with LLI (Fitch et al., 1994).

Tract tracing studies showed that animals with induced cortical microgyric lesions also show anomalies in the magnocellular regions MGN of the thalamus (Herman, Galaburda, Fitch, Carter, & Rosen, 1997). Further, surface electrodes and electrodes implanted in either the primary auditory cortex or MGN of mice with induced ectopias, were used to show that evoked responses to auditory stimuli were delayed. A strong and specific relationship was shown between alterations in auditory evoked potentials and the number of

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ectopias observed in the brains of these mice (Frenkel, Sherman, Bashan, Galaburda, & Lo-Turco, 2000).

An examination of electrophysiological recordings from human subjects, with or without language impairments, was made during auditory and visual sensory processing tasks and during a visually presented sentence processing task, and the results are consistent with these animal studies. Neville et al. (1993) reported finding abnormal patterns of hemispheric activation in language impaired children. Moreover, components of the auditory ERPs considered to represent activity in the perisylvian area (particularly the superior temporal sulcus) were abnormal in the subset of children who showed behavioral difficulties in rapid auditory processing (Neville et al., 1993).

Given Galaburda's proposal that some of the neuropathologies implicated in language impairment arise early in prenatal development, it may be possible to identify aberrant neurophysiological responses in newborns who are at genetic risk for LLI. Lyttinen and colleagues in Finland tested this hypothesis with newborn infants with a family history of dyslexia. Neurophysiological results show different patterns of ERPs to consonant–vowel (CV) pairs that differ solely by temporal acoustic cues, at 1 week and also at 6 months of age, in infants with a positive family history of dyslexia as compared to matched controls (Leppanen et al., 1999; Pihko, Leppanen, Eklund, Cheour, Guttorm, & Lyytinen, 1999). It remains to be seen whether these differences relate to later language and reading development. However, even within a normally developing population, early neurophysiologic responses were found to relate to later language outcome. Molfese and Molfese (1997) reported that factors derived from ERPs to CV combinations within 36 h of birth are remarkably accurate in identifying children whose verbal IQ is above or below the normal mean at 5 years of age. Using three ERP factors, 8 of 9 children who were subsequently found to be below the mean in verbal IQ and 60 out of 62 children found to be above the mean were correctly identified. In reviewing this literature, Benasich and Read (1998) suggest that this ERP data reflects a mechanism that may underlie the perception of phonemic boundaries, which is possibly inherent within the mammalian auditory system. Children whose acoustic or phonetic perception deviates from the norm in early infancy may well have difficulty building discrete phoneme representations from the language input that they receive from the environment. This early difficulty learning to represent phonemes, the building blocks on which both oral and written language depend, will result in lower verbal skills later in life.

With the recent advent of in vivo brain imaging technologies, such as ERP, structural MRI, fMRI, position emission tomography (PET), and magnetoencephalography (MEG), as well as the development of animal models that mimic the neuropathology seen in humans with LLIs, significant progress has been made in understanding some of the potential neurobiological substrates of developmental LLI. Electrophysiological studies support the results of behavioral studies showing specific deficits in phonological processing as well as acoustic analysis (Leppanan & Lyytinen, 1997). Results from structural MRI show that the pars triangularis (Broca's area) is significantly smaller in the left hemisphere of children with LLI and that these children are also more likely to have a rightward asymmetry of language structures. The opposite pattern is seen in children developing language normally (Gauger, Lombardino, & Leonard, 1997). Studies using functional neuroimaging, such as MEG have the advantage of providing both the spatial and temporal resolution most compatible with studying the real time neural processing involved in language in vivo. Recently, MEG was used in an attempt to help resolve the hotly debated "linguistic-specific" versus "rate processing constraint" hypotheses. MEG studies uniformly demonstrated significant differences in physiological processing rate for a variety of dynamic nonlinguistic acoustic stimuli in individuals with a history of LLI (including dyslexia), compatible with the rate processing constraint hypothesis (Habib, 2000; Hari & Koivikko, 1999; Hari, Vlata, & Uutela, 1999; Nagarajan, Mahncke, Salz, Tallal, Roberts, & Merzenich,

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1999; see Wright et al., 2000, for a review). Determining the precise neural mechanisms that link these physiological rate processing constraints with the phonological and/or other linguistic deficits that characterize individuals with language learning disabilities remains a very active area of current research.

Remediation

The remediation of LLIs is generally provided by speech–language pathologists, psychologists, or special education teachers. Traditionally, speech and language therapy has been guided by a detailed linguistic assessment of the pattern of receptive as well as expressive errors made by an individual child, using both standardized testing instruments and language sampling methodologies. Remediation has focused on helping the child to use age-appropriate linguistic structures in a variety of contexts. Reading remediation has concentrated on explicitly training children to recognize and manipulate phonemes within words (called phonological awareness) in an attempt to improve reading decoding skills, as well as on methods aimed at improving fluency (reading rate) and reading comprehension. Longitudinal outcomes research has shown that children with developmental LLIs generally require approximately 2 years of traditional intervention to acquire the equivalent of 1 year of gain in oral and written language development (see Leonard, 1998, and Lyon & Moats, 1997, for reviews).

Until very recently, remediation strategies have not been based on what we know about the neural basis of language learning. However, exciting new approaches to enhancing oral and written language skills have been developed that integrate in a novel way what is now known about the neural basis of learning in general (called neuroplasticity) with what has been learned about language development and disorders (see Merzenich & Jenkins, 1995, for a review).

Considerable research (reviewed above) has shown that rather than deriving from primarily linguistic or cognitive impairment, the phonological and language deficits of many individuals with LLI may result from a more basic constraint in the rate of processing and producing rapidly successive stimuli. In particular, these individuals are impaired in identifying elements occurring within 10s of milliseconds embedded in ongoing speech, a critical time frame over which many phonetic contrasts are signaled (see Tallal, 1998, for a review). Interestingly, it was shown that speech discrimination in children with LLI significantly improved when the rates of change of the critical formant transitions within syllables were synthetically extended in time (Tallal & Piercy, 1975). This finding led to the hypothesis that if the critical acoustic cues within the context of fluent, ongoing speech could be modified in such a way as to differentially emphasize and extend them in time, then the phonological and on-line language comprehension abilities of individuals with LLI should significantly improve.

To test this hypothesis a speech processing algorithm was developed to create more salient versions of the rapidly changing elements in the acoustic waveform of speech. The modification of fluent speech was achieved using a two-stage processing algorithm in which the duration of the speech signal was first prolonged by 50% while preserving its spectral content and natural quality. Next, fast (3–30 Hz) transitional elements of speech were differentially enhanced by as much as 20 dB. This acoustic modification process was applied to speech and language listening exercises that were presented as "games" as part of an intensive, daily training program. By amplifying and extending the rapidly changing elements within speech, the goal was to render them more salient, and thus more reliably temporally integrated. As previous research had demonstrated marked deficits in both forward and backward acoustic masking in individuals with LLI (Wright et al., 2000), a further goal of the acoustic modification procedure was to make the dynamically changing components less susceptible to masking by neighboring, slowly modulated speech elements. Cortical plasticity experiments in animals have indicated that a more sharply modulated signal should be more likely to drive complex signal learning (Merzenich & Jenkins, 1995).

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It has also been hypothesized that the phonological deficits evident in many individuals with LLI derive from a more basic rate processing constraint (Tallal, 1998). Experimental studies with humans and primates have demonstrated that the capacity to segment successive sensory events can be sharpened both behaviorally and physiologically by behavioral training (Ahissar & Hochstein, 1993; Recanzone, Merzenich, & Screiner, 1992; Wang, Merzenich, Sameshima, & Jenkins, 1995). Based on these studies, Merzenich et al. (1996) and Tallal et al. (1996) developed two audiovisual computer training "games" to engage young children with LLI in highly repetitive learning tasks, while maintaining a high level of attention and motivation. The first game was a perceptual identification task in which a correct response was the reproduction of the temporal order of two-stimuli sound sequences made by mouse click responses to two symbols on the computer screen. The nonverbal stimuli used in training were 16 octaves/s upward- or downward-gliding frequency-modulated (FM) tonal pairs. These stimuli were designed to cover the range of sweep frequencies and speeds that apply for the formant transitions of English consonants. Both the interstimulus intervals and FM frequencies were adaptive parameters, driven by an individual subject's trial-by-trial performance. The second game was a two-alternative forced-choice task in which the subject was presented with two CV stimuli, which were contrasted by a rapidly changing temporal spectral acoustic feature. Both games began with end point stimuli that each child could easily discriminate and then were individually adapted, trial by trial, to drive performance progressively toward normal levels, through corrective feedback and rewards.

The results of 4 weeks of daily language training with acoustically modified speech and the two adaptive computer games have been reported for two independent studies with children with LLI (Merzenich et al., 1996; Tallal et al., 1996). The second study directly compared a matched group of children with LLI who received the same training, but without the acoustically modified speech and temporally adaptive computer games. The results showed that the LLI children who received the experimental training with acoustically modified speech and temporal adaption showed significantly decreased temporal integration thresholds. A comparison of pretraining to posttraining thresholds showed markedly improved abilities to recognize brief and fast sequences of nonspeech and speech stimuli by the trained children. Standardized tests of speech discrimination, language processing, and grammatical comprehension were also given pre- and posttraining. The results showed that the group that received extensive daily training over a 4-week period with the temporally adaptive computer games and with listening exercises in which all speech was translated into acoustically modified form improved the equivalent of 2 years after only 1 month of training. This group also significantly outperformed the control group who received the same listening exercises with normal speech. Tallal et al. (1996), concluded that "This study demonstrates that providing LLI children with access to an acoustically modified signal that they can adequately process, coupled with reducing their temporal processing deficit, achieved through adaptive training, significantly improved LLI children's subsequent processing of natural 'on line' speech" (p. 83). Similar results were replicated in an independent study with French-speaking children with LLI (Habib, Expesser, Rey, Giraud, Bruas, & Gres, 1999). The Fast ForWord® training program was subsequently developed for school and clinic application based on these laboratory studies. Results from studies conducted in over 60 schools and clinics and with 500 children demonstrated that a similar level of efficacy can be achieved by teachers and clinicians (Merzenich, Saunders, Jenkins, Peterson, & Tallal, 1999; Miller, Linn, Tallal, Merzenich, & Jenkins, 1999; Tallal, 2000). These new remediation strategies have led to the development of a growing number of computer/internet based training programs. The success of these and other new research-based approaches have stimulated a new generation of remediation research that aims to develop more scientifically validated approaches for the treatment of oral and written LLI (Kraus, McGee, Car-

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rell, & King, 1995; Leonard, 1998; see also Lyon & Moats, 1997).

Conclusion

Research into the causes, determinants, and remediation of LLIs has led, not only to a better understanding of deficient language learning, but also to advances in our understanding of normal language development. Specifically, this body of research has demonstrated that there is considerable variability in the rate and quality of language learning and that a much larger number of children struggle with language learning than was once thought. It is not likely that a single etiology will be found to represent all individuals with LLI or that the same constellation of deficits will characterize individuals with LLI throughout their lifetime. Unfortunately, many children's difficulties in learning language are not recognized until they have experienced school failure, which has been shown to lead to reading and other academic achievement problems, an increased incidence of social and emotional problems, higher adjudication rates, and increased high school dropout. These data clearly support earlier, more routine assessment of oral language development than is in current practice.

Longitudinal research, as well as genetic family studies, have revealed a striking degree of co-occurrence between oral and written language impairments. Furthermore, oral and written language abilities have both been linked to more basic components of sensory, perceptual, motor, and cognitive development, particularly, those affecting the development of phonological systems. Specifically, constraints in systems that process transient sensory information in the nervous system appear to be linked to both oral and written language impairments. Precision in processing rapidly successive, nonlinguistic acoustic events within the first months of life has also been shown to significantly predict the rate of oral language learning in toddlers, across the entire continuum from normal to impaired linguistic development. In addition, individual differences in dynamic sensory sensitivity, in both auditory and visual modalities, have now been shown to be highly correlated with children's individual differences in developing orthographic, phonological, reading, and spelling precision. The finding that this seems to be the case for normally developing children, as well as for those experiencing significant difficulty acquiring literacy skills, has broad implications for the development of more efficacious methods for teaching reading to all children, not just those who are impaired. Explicit training in dynamic sensory sensitivity, combined with explicit training in phonological, morphological, and language comprehension skills, is proving to be the most successful strategy to drive rapid improvements in oral as well as written language development. The use of new technologies to provide individually adaptive computer and/or Internet based strategies offer the potential to offer early intervention to larger numbers of affected individuals.

For a variety of reasons, increasing numbers of students are coming to school without sufficient oral language skills to support the development of literacy. The impact of inadequate language skills is further amplified because the vast majority of education, especially in the early grades, is provided through oral instruction. Thus, poor oral language skills place such students at a significant disadvantage as compared to their peers. The cascading effects of deficient linguistic skills not only affects the acquisition of reading proficiency but also negatively impacts the ability to benefit from all aspects of the curriculum. Individuals with weak communication and literacy skills will inevitably find it difficult to participate in an economy requiring increasingly more sophisticated communication skills. Thus, there is a growing public interest in, and indeed increasing demand for, scientific research into the causes and determinants of reading failure, as well as for the development of research-based methods for enhancing literacy. We now know that if we are going to be more effective in developing strategies for addressing reading failure, we must begin with a better understanding of the prerequisites to reading success, much of which derives from the development of strong oral language skills.

Historically, research in this field has moved

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from a focus on single case studies to multiple case studies and then to well-controlled group studies. Further, research has moved from a focus on a single dimension of the problem to an understanding that language learning is a complex, dynamic system and, as such, multiple levels of analysis will ultimately be needed to gain a fuller understanding of LLI. Given the complexity of the neural substrates engaged in language learning, the multiple genetic and environmental influences affecting language learning, and the individual differences and heterogeneity of children with LLI, it is now widely accepted that a multiple levels of analysis approach, which can integrate data across all of these areas, will be required. Considerable progress has been made during the past century in explicating the multiple causes and determinates of LLI. However, the vast majority of studies in this field to date have focused on a single level of analyses (for example, linguistic, neurobiological, or genetic). Although some attempts have been made to integrate data across multiple studies and levels of analysis (Leonard, 1998), this has proven to be problematic, given the heterogeneity of the subject populations in each study, as well as the vast differences in ages, degree of impairment, and types of impairment included in each study. To make more rapid progress in the future it will be beneficial to design research studies that aim to take a multiple levels of analysis approach with the same individuals, as well as to use mathematical models designed to analyze dynamically changing, complex systems. Given that LLIs are developmental disabilities, it will be important not only to evaluate multiple levels within individuals, but also to do so prospectively and longitudinally throughout the most dynamic stages of language learning.

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