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Amelioration of the Acoustic and Speech Reception Deficits Underlying Language-Based Learning Impairments

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Introduction

A large percentage of language-impaired and dyslexic children have abnormal acoustic reception abilities that have been documented in several different classes of psychoacoustic experiments (see, Tallal and Piercy, 1973; 1974; 1975; Tallal and Stark, 1981; Bishop, 1992; Tallal *et al.*, 1993; Tomblin *et al.*, 1992; Anderson *et al.*, 1993; Kraus *et al.*, 1995, 1996; Farmer and Klein, 1995; Stein and McAnally, 1995; McAnally and Stein, 1996; Hari and Kiesila, 1996; Stark and Heinz, 1996a,b; Visto *et al.*, 1996; Tonnquist-Uhlen, 1996; Wright *et al.*, 1997a). Many language-impaired and dyslexic individuals have difficulties sequencing rapidly successive sound inputs because under the right conditions sounds destructively interfere with one another (Tallal and Piercy, 1973, 1974, 1975; Reed, 1989; see, Farmer and Klein, 1995). Many language-impaired children also have abnormal acoustic masking functions. For them, the detection of brief sounds is more strongly suppressed when those sounds are delivered nearby in time and within the same frequency channel as other ('masking') stimuli (Wright *et al.*, 1997a). In language-impaired children, these abnormal masking interference effects apply powerfully in the backward direction, that is, the detection of any brief sound is especially strongly suppressed by the occurrence of a rapidly following sound. At least some language-impaired and dyslexic children also appear to have abnormal frequency discrimination abilities (de Wierdt, 1988; McAnally and Stein, 1996; Stark and Heinz, 1996b; Wright *et al.*, 1997a; see, Farmer and Klein, 1995).

There is no dispute that aurally received speech is 'fuzzy' in language-learning impaired and dyslexic children (e.g. Liberman *et al.*, 1974; Vellutino and Scanlon, 1987; Steffens *et al.*, 1992; Watson and Miller, 1993; Hurford *et al.*, 1994; Vellutino *et al.*, 1995; Liberman, this volume; see, Brady and Shankweiler, 1991 for a review). For these children, the segmental features of speech are not consistently or often appropriately resolved (Liberman *et al.*, 1974; Wagner and Torgeson, 1987; Gowasmi and Bryant, 1990; Bird and Bishop, 1992; Hodgson 1992; Catts, 1993; Bird *et al.*, 1995; Stone and Brady, 1995; Stothard and Hulme, 1995; Mauer and Kamhi, 1996). As is predicted on the basis of abnormal masking interference patterns, language-learning impaired children have difficulty identifying the brief sound parts of words, especially with strong interferences occurring in the backward masking direction for the fine-grained acoustic features of speech that overlap spectrally (Tallal and Piercy, 1974; see, Wright *et al.*, 1997a). Consistent with this interpretation, just as a 'probe tone' in a masking experiment can be made audible by lengthening or amplifying it, so too can the initial or trailing acoustic events of syllables or words be relatively easily rendered more reliably distinguishable or recognizable in impaired children by simply differentially lengthening or strengthening them (Tallal and Piercy, 1975; Tallal and Stark, 1981; Tallal *et al.*, 1996; Merzenich *et al.*, 1996).

What is the origin of this striking impairment in complex acoustic signal/aural speech reception that so devastatingly limits language abilities in impaired children? What is actually wrong with the speech processing and learning machinery in the brains of these children? When, where and how does this problem arise? Why is this problem so resistant to change? How do the many other cognitive, pragmatic, emotional and other problems, that are commonly recorded in these children, originate from and relate to this fundamental signal reception/language processing problem? How do the language problems that spring from this acoustic signal reception problem relate to reading impairments? Perhaps most importantly, how can this problem be most effectively remediated in language-impaired children?

In this article, these important questions will be addressed briefly, to the extent that the current state of neuroscience, psychophysics and linguistics can answer them. The answers to these questions frame a large part of the logic that led us to develop a novel training method for overcoming this problem. The main principles that underlie this new approach to the remediation of the language-learning problems of these children will be briefly summarized. Some results from a trial conducted with 500 language-impaired children who were trained using this new methodology will be outlined. This large trial showed that this problem can be rapidly overcome in many children by a particular form of training. Finally, several important remaining theoretical and practical questions will be discussed.

Origins of the signal reception problems underlying language-learning impairments. There is growing evidence that language-learning impaired children form a separate language performance group, and do not simply fall within the tail of the normal distribution of language learners. By using a variety of perceptual, motor and cognitive assessment measures, specifically language-impaired children can be differentiated from normal children with no population overlap (Tallal *et al.*, 1985). In a study of the masking abilities of a small population of specifically language-impaired children, grossly impaired and consistent masking functions were recorded, with these children differing sharply, as a population, from matched normals (Wright *et al.*, 1997a). Dominant genetic factors clearly contribute to the genesis of language-learning impairments in at least a significant part of the impaired population (see, Tallal *et al.*, 1991; Lewis *et al.*, 1993; Bishop *et al.*, 1995; Pennington, 1995), again indicating that at least this large sector of the impaired population comprises a distinctly separate language performance group.

There is growing evidence that indicates that most language-learning impaired children are distinguished from the normal population in their acoustic signal processing abilities before or during the first six months of postnatal life. Benasich and Tallal (1996; and Benasich, this volume) have shown that about half of the children born of parents in families with genetic pedigrees that put them at risk for language-learning impairments are sharply differentiated from normal children when they are 6 months old (the age at which they were first assessed) in their ability to correctly discriminate rapidly successive sounds. Interestingly, the magnitudes of the signal processing deficits in these infants closely parallel the deficits recorded in school-age language-impaired children, i.e. once established, the dimensions of their impairment in the resolution of fast, successive acoustic signals appears to be remarkably stable. Not surprisingly, when these impaired children are tracked over time, they are consistently language delayed (Benasich, this volume).

These studies show that a 6-month-old infant destined for delayed language learning is still integrating sound inputs over relatively long time chunks (several hundred milliseconds), at a time when a normal infant is accurately making faster and more precise distinctions. Across this same period, normal children are progressively refining and deeply embedding their phonological representations, usually in the left hemisphere, i.e. are making fine-grained segmental distinctions about speech inputs on a heavy experiential schedule (see, Kuhl, this volume). A child destined for language-learning impairment does not appear to successfully employ the higher-rate neurological signal sampling and processing required to create reliable receptive language constructs. We have hypothesized that within any given frequency channel (but, it is important to note, not necessarily between channels), these children are constructing a language with an emphasis on prosodic-rate variations in acoustic inputs, and not on the basis of accurately distinguishing and representing fine-grained acoustic details of speech (Merzenich *et al.*, 1997a).

Some obvious questions about the neurological origins of language-learning impairments

Why do some children fail to develop normal temporally-refined complex acoustic signal processing capabilities in infancy? We now know that the integrative time constants that govern signal reception by the human forebrain are shaped by learning in normal children and adults (see, Merzenich *et al.*, 1993, 1995, 1997a,b, for a review) by the operation of increasingly well understood brain plasticity-learning mechanisms that underlie progressive perceptual, cognitive, metacognitive and motor skill learning in childhood and across life (see, Merzenich *et al.*, 1991; Merzenich and Jenkins, 1994; Merzenich and deCharms, 1996, for a review). In a juvenile or adult monkey or human, for example, progressively higher-speed signal processing can be acquired through appropriate behavioral practice (Karni and Sagi, 1990, 1993; Ahissar and Hochstein, 1992; Wright *et al.*, 1997b; Merzenich *et al.*, 1993, 1995a,b, 1997a,b). If (a) the learning machinery is intact; if (b) signal-to-noise conditions permit the resolution of spectral detail and spectral dynamics over time; and if (c) the child is selectively attentive to the appropriate speech details in learning, children should naturally develop the normal high-speed short-chunk processing that results in resolved, fine-grained acoustic signal treatment. The fact that these special children do not implies that one or more of the above-stated conditions for natural, learning-driven adaptive plasticity is not being met.

Perhaps the strongest evidence that a weakness in the learning machinery itself might contribute to this abnormal signal processing in at least some of these children comes from the studies of Galaburda and colleagues (Galaburda, 1994; Galaburda *et al.*, 1994; Rosen *et al.*, 1995), who showed that many language-impaired and dyslexic children have punctate cortical anomalies ('microgyria') in their temporal and/or frontal lobes. Creation of similar anomalies in the cerebral cortices of rats results in an impairment in the rat's ability to identify and sequence rapidly successive sounds (Fitch *et al.*, 1994). While we have no clear understanding as to how these focal cortical anomalies might lead to such a signal processing impairment, nonetheless, the evidence is growing that the occurrence of these defects in some impaired children may be contributing to their abnormal signal reception abilities.

Similarly, apparent degenerative changes in the magnocellular divisions of the mainline auditory and visual thalamic nuclei in dyslexic children as compared with normals (Livingstone, 1993; Galaburda and Livingstone, 1993) have been interpreted as representing a potential cause of language-learning impairments. This hypothesis is supported by the fact that the differentially degraded magnocellular nucleus subdivisions have been implicated in higher-speed signal processing in these two great sensory systems. Dyslexic individuals have receptive visual deficits that are consistent with magnocellular system deficiencies (e.g. see, Cornelissen *et al.*, 1995; Felmingham and Jakobson, 1995; Richardson *et al.*, 1995; Edwards *et al.*, 1996; Eden *et al.*, 1996)—although it might be noted that some investigators have argued that the visual impairments of dyslexics are not magnocellular system-specific (e.g. Evans *et al.*, 1994; Greatrex and Drasdoe, 1995; Johannes *et al.*, 1996).

There is also compelling evidence that grossly abnormal signal-to-noise conditions are a part of the history of a significant subpopulation of language-impaired children. For example, many impaired children have a history of very early chronic middle-ear disease. This means that some of them have hypothetically developed language in the first year of life under conditions of consistently muffled hearing. Learning/plasticity studies conducted in monkeys indicate that it is virtually impossible to develop normal high-speed input segmentation in a brain that has to make reliable distinctions about substantially muffled and, thereby, irresolvable, fine-grained acoustic features. It is not surprising, then, that a number of carefully conducted developmental studies have implicated very early chronic middle-ear disease as a risk factor for language-learning impairments (e.g. Teele *et al.*, 1984; Bishop and Edmundson, 1986; Luotonen *et al.*, 1995; Gravel and Ellis, 1995; Gravel *et al.*, 1996). Perhaps the most clearcut of these studies, from the perspective of the present arguments, are those in which children have middle-ear pathology that insures that they have continuous hearing conduction blocks bilaterally, throughout infancy, e.g. as occurs in children with cleft palates, or with Downs' syndrome (e.g. see, Pappas *et al.*, 1994; Schonweiler *et al.*, 1995). In such populations, a history of continuous middle-ear effusions from the time of birth can be unequivocal. In these children, delayed language learning and enduring language-learning impairments are nearly universal if middle-ear problems are uncorrected across the first postnatal year. However, when these middle-ear problems are aggressively corrected in these special infants, language delay and language-learning problems are commonly greatly ameliorated (e.g. Pappas *et al.*, 1994; Schonweiler *et al.*, 1995).

While chronic middle-ear infections across a crucial time window in early childhood is still an important suspect for a signal-to-noise based origin of abnormal signal reception/processing in some language-impaired children, it should be pointed out that defective signal-to-noise conditions that could forestall development of normal high-speed processing could also arise from a number of hypothetical central nervous system causes.

Finally, we know that there is a capacity to improve either temporal or spectral discrimination abilities selectively through learning, simply by attending to one cue set or the other in a task in which both cues are available. That fact alone suggests the possibility that a child's evolving attentive listening strategies in early infancy might contribute directly to the enduring adoption of slow-speed temporal 'chunking'.

Why does this problem endure in language-impaired children? We have hypothesized that the infant with impairments is being heavily rewarded for making distinctions about received speech using this alternative processing schema through a crucial window of time in language development in infancy, and that with heavy practice during this epoch, this processing mode simply comes to be *the* learned way for these children's brain to make reliable decisions about received speech (Merzenich *et al.*, 1993, 1995, 1997a,b). In the progressive, experience-based creation of receptive language constructs, the use of this alternative, suprasegmental feature-dominated processing strategy therefore ultimately comes to be a self-reinforcing developmental 'trap', resulting in syllable-length rather than normal higher-resolution, phoneme-based segmentation.

In the face of these potentially multiple causes of the signal reception problems recorded in these children, how can impaired children end up with such a consistent deficit? We have earlier hypothesized that the main fall-back position in signal processing in an impaired child that has problems with signal-to-noise conditions or with the fine-grained control of selective attention is to 'chunk' inputs over the mean duration of the main time-varying event in the speech stream, the syllable (Merzenich *et al.*, 1995, 1997a,b; Tallal *et al.*, 1997). Masking interference and temporal sequencing psychophysics as well as studies in which speech reception has been tracked in impaired infants across the first year of life (e.g. Donohue, 1993; Groenen *et al.*, 1996) indicate that the impaired child is integrating speech inputs over approximately the period of the syllable. With reception that is still predominantly limited to coarse-grained, prosodic-rate processing, the impaired child is making perceptual judgments based on integrated, relatively long-duration acoustic products. Because the statistics that apply for the temporal structure of speech are essentially common to all human speech receivers in any given language, whatever the cause of their particular weakness, deficits would be expected to be quantitatively similar from child to child and relatively stable across childhood, as a consequence of their adopting this common alternative 'fall-back' signal processing strategy applied to statistically predictable speech-input structures.

What is actually wrong with the brains of these children? Psychologists have identified long lists of specific receptive and expressive language problems that apply to language-impaired children. Children are defective in receptive phonology, in their uses of syntax and grammar, in their memory for speech, in their grammatical morphology, in their fluency, in their attentional behaviors, in their pragmatics, in their emotional behaviors, *et alia*. Each child presents a different idiosyncratic profile when their skills and abilities are assessed across this broad range, resulting in a rich basis for subdividing and categorizing language-impaired children following different complex schemata (e.g. see, Levine *et al.*, 1987; Shaywitz *et al.*, 1995; Rapin, 1996). At one time or another, nearly all of these 'specific impairments' have been defined as *the* core deficit and/or *the* probable cause of what could be termed the 'language-learning impairment syndrome'. Moreover, the panoply of problems that apply for these children have led a number of scientists to conclude that language-impaired children have fundamental, general defects in their brain machinery that directly result in specific, multiple impairments across a broad behavioral range (e.g. see, Locke, this volume).

In an effort to understand the neurobiological bases of these various deficits, a substantial literature has described differences between the brains of language-impaired vs. normal individuals. Beginning with the documentation of differences in the dimensions of the superior temporal plane resulting in a reduction of the normal physical hemispheric asymmetry that parallels the reduction of functional asymmetry for language representation in dyslexics, by Geschwind and Galaburda (1985), it has been repeatedly argued that the brains of language-impaired and dyslexic individuals differ from those of normals. Event-related potential and other electrophysiological studies, along with PET, fMRI and MEG imaging studies have also revealed clear functional brain differences in specifically language-impaired and dyslexic individuals, e.g. that apply for the representations of successive nonspeech and speech sounds, for hemispheric dominance, and for the reliable representations of a range of language abilities (e.g. Jernigan *et al.*, 1991; Hagman *et al.*, 1992; Rumsey *et al.*, 1994a,b; Fiez *et al.*, 1996; Llinas, this volume; see, Ingvar, this volume, for a review)

It should be pointed out that most of these physical and functional differences (the punctate 'microgyria' of Galaburda and colleagues being a probable exception) could be interpreted as either causes or effects of defective signal processing. Many basic neuroscience studies have documented changes in the morphological and functional status of brain centers resulting from heavy differences in their active engagement, as must occur on a massive scale in language-impaired children (see, Merzenich *et al.*, 1996, 1997a). That fact commonly obscures the definitive resolution of cause-effect issues that pertain to language impairment and dyslexia origin.

What, then, is inherited, when language impairments are attributed to inherited factors? The ability to identify rapidly successive sound events, e.g. the fine-grained, segmental acoustic events that are the basis of phoneme recognition for aural speech, are subject to powerful plasticity/learning effects. A human brain can be *trained* to more accurately receive non-speech acoustic and speech inputs at higher speeds and with greater precision (Steinbüchel and Poppel, 1993; Hurford *et al.*, 1994; Tallal *et al.*, 1996; Merzenich *et al.*, 1996; Wright *et al.*, 1997b). Studies of these powerful learning effects, as well as infant studies and brain plasticity/learning studies described earlier, argue that differences in learning progressions in infants can contribute directly to the emergence, consolidation and persistence, of the low-speed/poor resolution signal reception that distinguishes language-impaired individuals from normals. What triggers the initiation of the ball rolling downhill in the wrong direction in the developmental history of these impaired infants? At this point, all we can say is that some prenatal or early postnatal factor(s) prevents the brain from developing the normal, mature short-time chunk, fine-grained acoustic signal processing mode. When language-learning impairments are inherited, they could be based on an inherited physical anomaly in the brain that degrades temporal features of representation or that degrades signal-to-noise conditions in the auditory projection axis or cortex; or to an inherited, reliable genesis of muffled inputs from the cochleae; or due to an inherited epigenetic central nervous system developmental sequence delay; or to an inherited problem in the brain machinery or processes underlying selective attention; or to any one of a dozen other related possibilities. Understanding the interface between genetics and environment in perceptual and cognitive disabilities is clearly a main goal for future research.

It is important to note that, by definition, specific language-learning impairments in specifically language-impaired children are *not* correlated with nonverbal intelligence. Thus, the capacity of the cortical learning machinery to drive representational change (i.e. to learn) is not at issue in these individuals. The problem appears to lie with their appropriate reception and processing of rapidly successive inputs.

If the fundamental capacity to learn is still intact, how can the wide range of perceptual and cognitive and emotional impairments be accounted for in language-learning impaired children? Why is language-learning impairment so strongly related to the emergence of attentional problems? With problems of memory? With measurable deficits in emotional behaviors? Simple logic tells us that if the learning machinery of the brain is operational, a fundamental problem in signal reception could well account for the extraordinary range of deficits that apply for language-impaired children, much as is the case for children with hearing impairments.

We can understand the broad and varied impacts of a fundamental perceptual deficit by considering a simple analogy. Imagine the progression of development of visual perceptual and cognitive and visually-related metacognitive skills in a child that needs corrective eyeglasses. Measurements in almost every visual perceptual or cognitive or appropriate metacognitive task, or of any visually-guided motor task that the child is asked to perform would indicate impairment. Tests of the child's visual memory would record errors due to poor signal salience. Because the child must struggle to see and cope with blurred vision, she/he will be academically at risk, and undoubtedly also at risk for other aspects of their attentional and social and emotional development. Their confidence and self-image, as well as many other aspects of their metacognitive behaviors, can be expected to be affected by consistent negative feedback coming from their impaired visual operations. Many problems, individually expressed in complex ways as a function of the actual level of the signal reception deficit and as a consequence of numerous alternative compensating and coping behaviors, emerge in rich profusion in uncorrected visually-impaired individuals.

This is precisely the picture presented by language-impaired children.

There is a straightforward prediction that derives from this interpretation. If the signal reception problems that limit language-impaired children can be overcome by training, then impaired children should improve on a very wide range of receptive and even expressive language abilities. Moreover, effects on measures of serial memory, in pragmatics, and in other aspects of general real-world language use — and over time, in social and emotional development — should follow.

Why has this problem been so resistant to change with conventional speech therapy? Children with this acoustic signal/speech reception problem are processing speech information in a fundamentally different way than do normal children. As a rule, they are integrating information in 'chunks' that are roughly a syllable-length in duration, and making decisions about these integrated, coarse-grained inputs. Through this anomalous integration and analysis, briefer and weaker sound components in the speech stream are differentially suppressed. This is a massively practiced behavior, developed through the processing of several million speech inputs that the child's brain is making decisions about across early childhood. The young school-age child has constructed her/his entire receptive language construct on the basis of this alternative, heavily practiced signal processing strategy. For these children, this alternative processing mode has long since come to be *the* most effective way to derive meaning from aurally received speech. And while it is clearly not very effective or efficient, it is, nonetheless, strongly consolidated, and has come to be deeply embedded by practice.

Cortical plasticity studies indicate that the substitution of a more normal phonological processing for this suprasegmental processing-dominated mode will necessarily require very heavy repetitive stimulus presentation in a closely attended and highly rewarded behavioral context, akin to the level of practice that applies for accurately receiving the phonological elements of a second language in language learning. By this interpretation, the traditional therapies applied to these children greatly undershoot the requirements for heavy repetition on intense training schedules in highly motivated learning sessions, as would be necessary for changing and stabilizing the basic signal processing strategies of the brain to a fundamentally different processing mode.

The use of fast natural speech in the training of a system that cannot effectively process it represents a second inefficiency of the predominant contemporary approaches to remediating the receptive problems of language-impaired children. Efficient training also requires the application of acoustically modified speech stimuli that are appropriately discriminable by the learning-impaired child. From that initial starting point, the language-impaired child can be trained adaptively to more accurately process progressively faster and faster acoustic changes—i.e. those occurring in natural speech.

How do these language problems relate to reading impairments? Many longitudinal studies showed that preschool-age language-learning impaired children comprise at least a large cohort of those who later emerge in school as dyslexic (Stark *et al.*, 1984; Aram *et al.*, 1984; Tallal *et al.*, 1985; Silva *et al.*, 1987; Bishop and Adams, 1990; Rissman *et al.*, 1990; Catts, 1993; Hurford *et al.*, 1992; Bird *et al.*, 1995). Other studies have shown that dyslexic children and adults have acoustic reception problems as a rule (Tallal, 1980; Reed, 1989; Stein and McAnally, 1995; McAnally and Stein, 1996). Signal reception problems are manifested in a large proportion of language-learning impaired and dyslexic children by a fundamental difficulty in parsing words into their phonological parts (Liberman *et al.*, 1974; Tallal and Piercy, 1974; Wagner and Torgeson, 1987; Gowasmi and Bryant, 1990; Bruck, 1992; Steffens *et al.*, 1992; Hodgson 1992; Catts 1993; Bird *et al.*, 1995; Stone and Brady, 1995; Stothard and Hulme, 1995; Mauer and

Kamhi, 1996). This deficiency has been often described as a problem in 'phonological awareness' and identified as a cognitive problem.

However, as outlined earlier, growing evidence indicates that language-impaired children cannot process rapidly changing acoustic inputs with the resolution of within-channel, fine-grained acoustic inputs that is necessary for the creation of reliable representations of individual phomenes. Phonological awareness would be expected to be limited by this receptive deficit; you cannot have a normal 'awareness' of unformed or malformed aural representations of receptive phonology. The translation of aurally received speech to orthographic representation by letter therefore makes little sense because it is very difficult to associate visual symbols with sound percepts that are incorrectly or inconsistently perceived.

Development of a Novel Training Program for the Remediation of Language-Based Learning Disabilities

Our collaboration in developing a novel training program arose from the understanding that the accurate representation of rapidly successive inputs was subject to powerful learning effects in even the adult brain. If we could train a monkey to make progressively more reliable distinctions about spectro- (spatio-)temporally complex inputs delivered to the brain at progressively higher input rates, why would that not apply for children who have a fundamental problem in high-rate complex signal processing? If older human subjects can be trained to make progressively more accurate distinctions about rapidly successive stimuli, why would that not apply for children who have a problem in accurately receiving fast successive inputs?

Neuroscience, experimental psychology, linguistics and child psychology powerfully inform us about how a training program could be designed to shift the processing of children from a coarse- to a fine-grained acoustic mode. They tell us that training programs must be intense; must involve heavy stimulus repetition; must be conducted under conditions of high motivation and reward; must begin with problems that a child can solve successfully; must be adaptive; must drive changes that generalize to all of the contextual requirements of accurate speech reception; and must be conducted with the child under continuous behavioral control. To achieve those objectives, a series of training exercises disguised as 'computer games' were produced that extantiated these principles. Each 'game' was a carefully crafted exercise utilizing the principles of psychophysical training paradigms and cortical brain plasticity/learning models.

Computer-based training has a number of important advantages over one-on-one training. It permits the delivery of stimuli on a very intense schedule; permits the delivery of strong and consistent trial-by-trial and level-by-level rewards; allows for precise adaptive stimulus tracking to maintain a constant, relative difficulty level for the child as she/he progresses over time at their stimulus discrimination/recognition tasks; allows for the delivery of complex continua of synthetically produced stimuli that cannot be produced by natural voice; and allows for automatic performance tracking and documentation. In fact, for achieving the intense schedule of very closely controlled and strongly rewarded behaviors, and for the stimulus generation and control required to progressively drive a mode shift in impaired children, there is no practical substitute for computer-guided training.

With an acceptance that computer-based training was required for us to implement our training program, an automated internet/database system was also developed to provide a capability of remotely assessing and documenting subject performance. Our purpose was to facilitate the study of impaired populations in clinics and schools out in the field. The system created was designed to document daily performances of every child in training and to track them over time at all of the individual exercises that each child was working at. The database sends that information back to the therapist or teacher on a daily basis in various detailed and summary forms. The objectives in creating this system were to: (a) provide a continuous basis of assessment of the efficacy of training for all the children enrolled in the training program; (b) provide assistance for the therapist in monitoring children in training, by reconstructing for them each of their children's progress day-by-day over the course of training and by alerting them if a particular child in training was not complying or working 'by the rules' at his/her exercises; and (c) enable more flexibility and control for individualizing the training program based on the daily training achievements of individual children.

To assess the efficacy of this training method for clinical and classroom use, a comprehensive 6–8 week training program, called *Fast ForWord*, was developed. The *Fast ForWord* program includes seven training exercises that serve three main purposes. First, they were designed to adaptively train the child to make accurate, higher-speed distinctions about natural speech, i.e. to shift processing from the impaired slower-speed, suprasegmental to the normal higher-speed segmental mode. Second, training was designed to generalize this mode shift in signal reception to all of the contextual conditions that apply for accurate real-world speech reception. Third, training engaged the child in grammatical and syntactical training exercises designed to drive rapid improvements in these deficient language skills.

All of the 'games' are listening exercises. Some exercises employ nonspeech acoustic stimuli; others apply synthetically manipulated consonant vowel (CV), CVC or VCV stimuli; still others employ acoustically modified natural speech. Some games focus on the accurate identification of the confusable sound parts of words; still other game tasks are memory-loaded, requiring the child to accurately receive, comprehend and respond to aurally received instructions. In each of these exercises, brief and fast-changing acoustic events in the speech stream are initially disambiguated in training by (a) extending them in time, and/or (b) differentially amplifying them, and/or (c) by separating them in time from nearby interfering (masking) nonspeech acoustic or other speech inputs. The child is trained to distinguish between or identify stimuli or to follow aurally received instructions. As the child improves, these acoustically modified stimuli are presented in progressively less modified, i.e. more natural forms.

Preliminary Results of a Large-Scale Trial

Results of initial trials

Our initial evaluation of this training strategy was undertaken in a laboratory school setting at Rutgers University (Tallal *et al.*, 1996; Merzenich *et al.*, 1996). In studies conducted over two summers with 29 specifically language-impaired children (11 of whom served as treatment controls), children were trained for about 2 hr/day in a 3-hr classroom session held 5 days/week for 20 training days. Children worked at computer-based training exercises for about 40 to more than 60 min/day. In other daily training sessions, acoustically modified stimuli were mounted on audiotapes, and delivered by a therapist in a one-on-one format. Children made major gains in their ability to correctly identify and sequence rapidly successive sounds with training. They also very significantly improved in their ability to correctly identify the sound parts of words presented in natural form. Language skills tests administered before and after training showed that their receptive language age was advanced by these 20 days of training by about 1.5–2.0 years. These changes were very significantly greater than the small but significant gains achieved in carefully matched control children who underwent the same training program, but with (a) unmodified speech stimuli applied in training; and (b)with application of nonadaptive visual training games administered on an equally intense training schedule (Tallal *et al.*, 1996; Merzenich *et al.*, 1996).

Children were again tested 3 and 6 months after training had been completed. The main changes driven by the training were sustained across this post-training time epoch. Interestingly, receptive phonological abilities further improved markedly across this period, and at 6 months after training, the 18 trained children had performance abilities that were at, or above, the normal median for their age at this crucial skill. By contrast, the modest phonological reception gains of the control children continued to lag even further behind the phonological reception abilities of trained children.

Organization and parameters of a large-scale field trial

On the basis of these laboratory findings, we recognized what appeared to be potentially very powerful clinical and educational benefits of these new training procedures. However, to make them usable outside of the laboratory in different real-world classroom and clinical settings, we needed to reconstruct our training tools into a more practicable delivery form. Among many other changes, the language-training exercises were also mounted in the form of computer games. With this task completed by mid-1996, a large-scale trial of this training strategy was initiated. Our main purposes in this 500-child training trial were to: (a) document the efficacy of application of this new, fully computerized form of the training program, which was named 'Fast forWord'; (b) evaluate the effectiveness of application of this complex training program in the real world, i.e. in clinics and schools; and (c) assess efficacy in a wider range of language-impaired children. Other objectives in research have been to: (d) assess the effectiveness of training achieved in school or clinic vs. home-site training; (e) more completely document benefits for expressive as well as receptive language skills and abilities; and (f) document possible benefits for a wider range of real-world language-related cognitive and metacognitive skills and abilities.

Sixty-three professionals working at 35 sites in the US and Canada were educated on the use of this program in 2-day training courses held in San Francisco. Each professional group then set up training centers at their clinics and schools, or in some cases organized a home-training program. Every therapist was connected to us via the internet, as were all of the computers that were used to apply the training programs in 5–12-year-old language-impaired children.

Professionals selected children for training that performed below normal on standard language batteries and receptive phonology tests. Only children who (a) scored at least 1 SD (standard deviation) below the mean on at least one test or subtest; and/or (b) performed poorly on a new computerized version of the Tallal Repetition Test revealing problems in their reception of rapidly successive acoustic inputs; and (c) were judged by the professional to be receptively impaired were included in the trial. Out of approximately 500 children trained in the trial, the majority were identified as specifically language impaired. Significant subpopulations of pervasively developmentally disabled (PDD-autistic and PDD-NOS children) children who were comorbid for language impairments, for central auditory processing disabilities (CAPD) and for attentional deficit disorders (ADD), as well as a large number of dyslexic/language-impaired children were included in this sample.

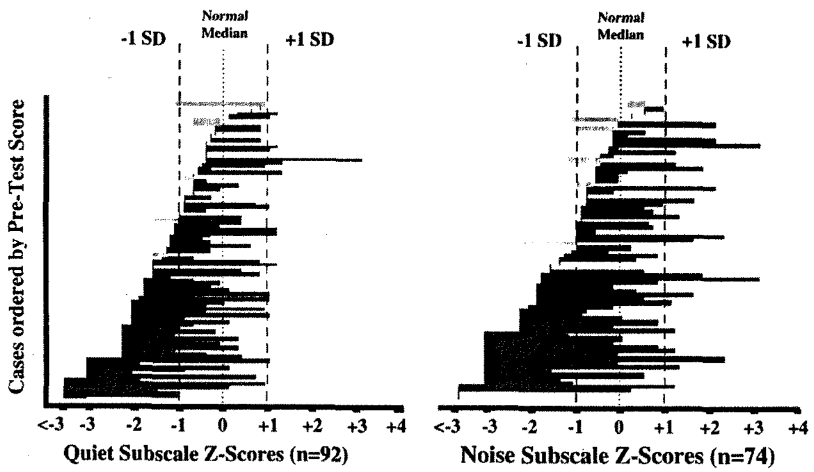
Children were trained for 1 hr and 40 min/day for an average period of about 35 days. The training program schedule for the 7 training games was predefined. Children worked at the games until they achieved nearly normal performance levels at the majority of game tasks, or until their performance assympoted, or in some children, until game-play compliance could no longer be reliably maintained. Compliance was excellent in nearly all children through at least a 4-week (20-day) training period, and in the great majority of children until some, or all, normal signal/speech/language reception performance targets were achieved.

Children received feedback and were rewarded during play by bells and lights and reward animations, and by visual performance barometers. Game trials all began with an observing response to assure that all behavioral exercise trials were attended. Children earned points that were proportional to game-play rates (effort), and not game-play performance. These points were converted to tokens, with most children being rewarded through the period of training in a token economy.

Some trial results

More than 75% of children reached high-performance levels on tasks that measured the ability to accurately receive rapidly successive acoustic inputs. As an apparent consequence, speech reception was clarified for the great majority (more than 90%) of trained children. For example, on the Goldman-Fristoe-Woodcock test of auditory discrimination, the average child jumped in ability by a Z score of about 1.4 in the quiet (advanced 1.4 SD; Fig. 1) ($t = -11.2$; $p < 0.0005$), and by about 1.8 in noise ($t = -10.0$; $p < 0.0005$). The average GFW performance in the quiet or in noise (simulating the background noise in a cafeteria) before training was more than 1 SD below the normal median. The average performance after training in the quiet subscale was at the normal median (Fig. 1, left). For distinguishing the sound parts of words in noise, the average post-training score advanced, the average, from an impaired level to one that was significantly *above* the normal median (Fig. 1, right). Note that for both tests, nearly all children who were impaired at this task moved into the normal range after training. Also note that a high number of children advanced to the above-the-normal-median performance ability levels, especially markedly for tests of phonological reception conducted in noise.

With more accurate acoustic signal and speech reception as indicated by the GFW results, it is not surprising that highly significant improvements in measures of language reception and comprehension were recorded. One measure of that ability applied in 255 children in this


Discrimination of Sounds in Words*

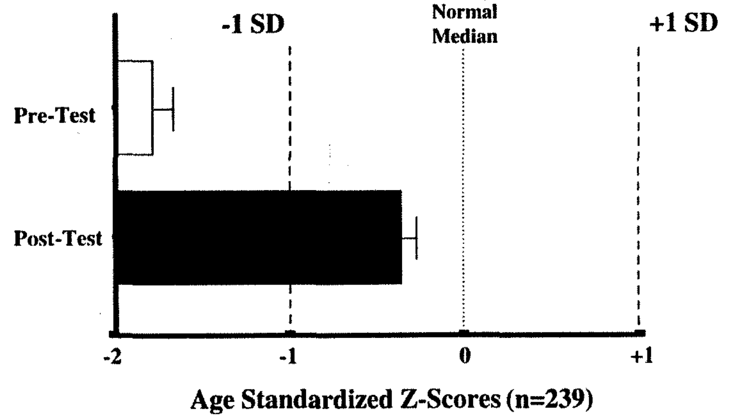
*Goldman-Fristoe-Woodcock Test of Auditory Discrimination

Fig. 1. Children trained on the *Fast ForWord* program had large gains in their ability to discriminate between and identify the sound parts of words. Here, before vs. after training results are shown for 92 and 74 children (all children in our series for which these measures were obtained up to this date) in which the Goldman-Fristoe-Woodcock Test of Auditory Discrimination was conducted in the Quiet (on the left) and Noise (at the right). The left end of each dark bar represents the pre-training performance level for an individual child; the right end represents their post-training performance level. Children are ordered as a function of their pretest scores, with results from the most impaired children displayed at the bottom of these two charts. Z-scores are computed from age-adjusted normalized T-scores. Note that the majority of children (see, text) improved at this basic ability in the Quiet and Noise by >1 SD. Note that about one-third of children advanced to >1 SD above the normal median on this test from an initial (pre-training) more-average or impaired performance level. Finally, note that the post-test scores of 6 children in both Quiet and Noise tests were lower than their pretest scores (light gray).

trial was the Token Test for Children. Token Test results from this trial are shown in Fig. 2. Here, we have subdivided the population into four equal-sized groups based on their initial performance scores. Note that very large gains—on average, about 2.6 SD—were made by very severely impaired children (in the first quartile in Fig. 2). In the second quartile of impaired children, gains were about 1.6 SD; in the third, about 1.1 SD; and in the fourth, about 0.4 SD. The Token Test has a ceiling effect that applies very strongly for older children, who make up most of this top quartile group. For children with any given initial performance level on this test, gains achieved with training were not correlated with the child's age.

The great majority of trained children who had initial impairments at accurately receiving and following instructions of variable length and

A. Language Reception & Comprehension*



*The Token Test for Children

B. Language Reception & Comprehension by Quartiles *re* Pre-Training Scores

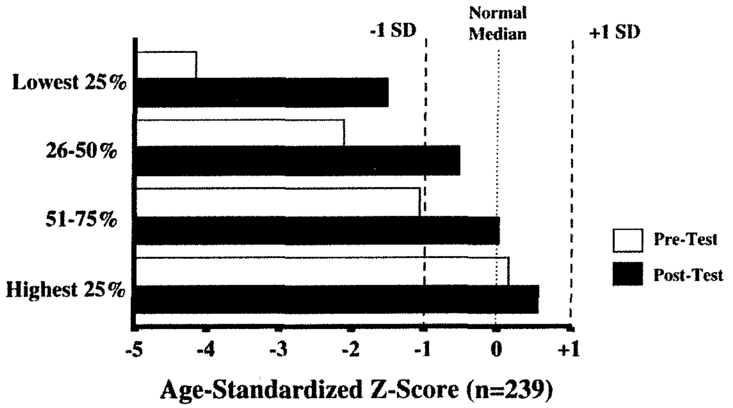
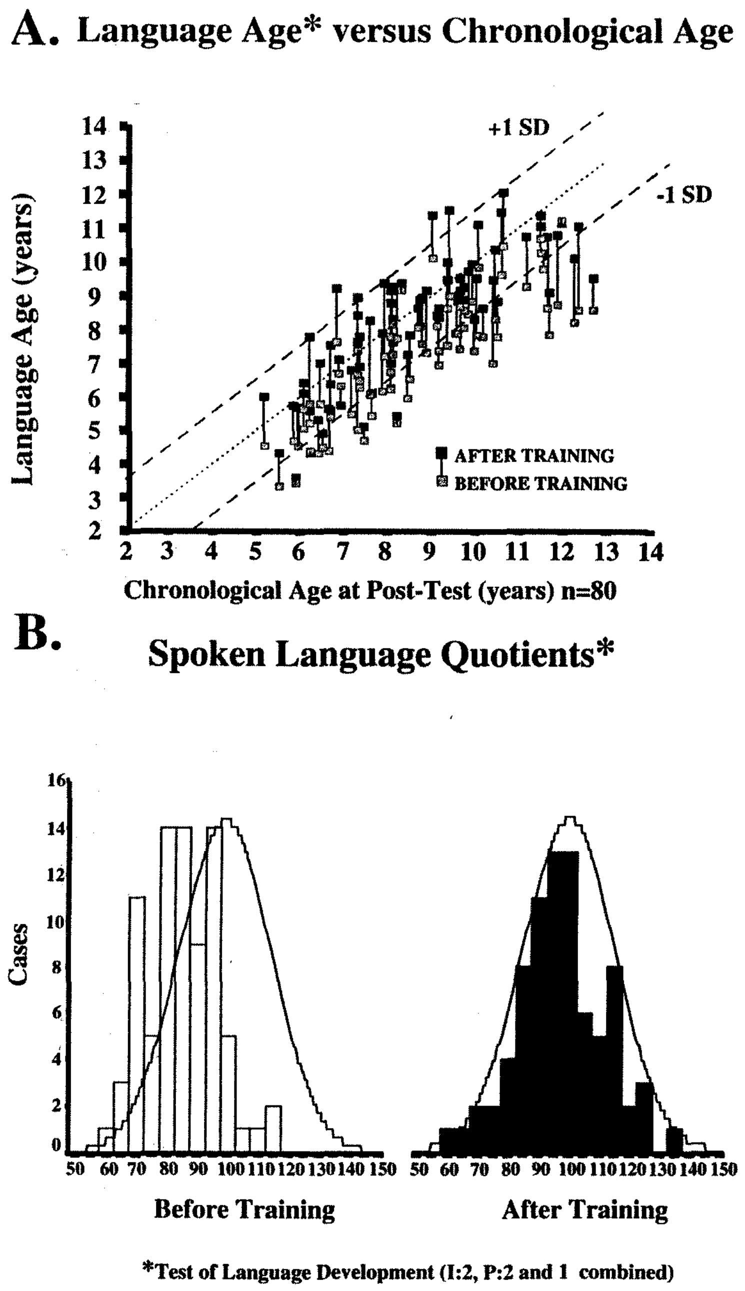


Fig. 2. Aural signal reception improvements were paralleled by sharp gains in measures of language comprehension. (A) Before vs. after training scores on the Token Test for Children for the 239 children for which data on this test were obtained in the *Fast ForWord* trial. Note that the average improvement with training was about 1.4 SD. Thin lines on bars are standard errors. T-tests showed that before and after training scores differed highly significantly ($t = -17.7$; $p < 0.0005$). (B) Token Test scores illustrated by population quartiles. Note the >2.6 SD (Z-score) advances for age-calibrated scores achieved, on the average, by the most impaired children in this series. The relatively modest gains of the least impaired children partly reflected a ceiling effect for the Token Test (see, test for further explanation). All quartile gains were highly significant.



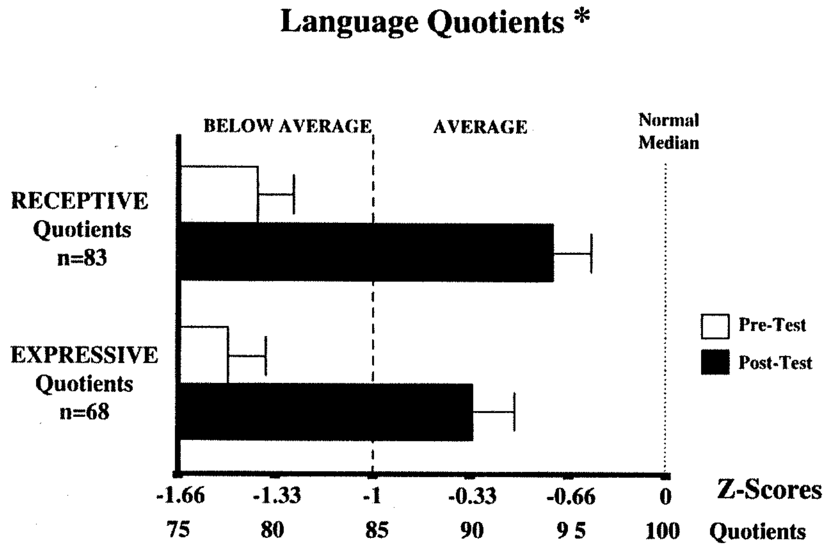
complexity, as measured by this task, performed in the normal range after training. More than 95% of children overall were within 1 SD of normal when tested after training, vs. 38% before training. Most children, among the 5% who did not achieve normal performance levels with training, nonetheless made a strong advance toward the normal range.

These positive changes in accurate speech reception and language comprehension were manifested in all other language tests administered to these children. In fact, therapists were encouraged to use the language batteries that they normally use to assess these impaired children. In our trial, 7 different language batteries were applied to independent subsamples of more than 10 children. Forty-three language subtests were conducted in the administration of these 7 batteries for which population n's were >10. Statistical comparison of before vs. after training test results showed that there were significant positive gains in performance at the $p < 0.001$ level for 29 of the 43 battery subtests for which n's were greater than about 10. In 12 of the 14 remaining tests, gains were statistically significant at the $p < 0.02$ level. The two exceptions were both vocabulary subtests.

In summary, gains were recorded in an extraordinarily wide range of measures of receptive and expressive language skills over a very short training period averaging about 7 weeks.

Overall pre- vs. post-training language scores are shown for two of the most widely used comprehensive language assessment batteries in Figs. 3 and 4. In Fig. 3A, all 80 children given different versions of the Test of Language Development (TOLD) battery are shown. The language age of each child defined before (light squares at the end of vertical lines) and after (dark squares) training are plotted as a function of the child's chronological age at the time of training completion. Note that nearly all children had a language age that was below their chronological age (by an average of 1.4 years) before training. The performance of nearly every child advanced with training. After training, children language quotients fell within a relatively normal language-age distribution (Fig. 3B), i.e. their language age now matched their chronological age, with an approximately normal distribution. In fact, for listening skills alone, an area of language skills that the *Fast ForWord* training program was designed to directly address, post-

Fig. 3. (A) The language age of all 80 children in this series given any one of three different versions of the Test of Language Development (TOLD) battery, shown before (light squares) and after (dark squares) training. ANOVA analysis indicated no significant differences between test performances and gains on the different TOLD versions ($F = 2.7$, n.s.) and thus justified the combining of data from different test versions. (B) Distributions of language quotients for 80 children (all children in our trial administered these particular tests before and after training), recorded before (left) and after (right) training.



***Clinical Evaluation of Language Fundamentals (Preschool, -3 & -R combined)**

Fig. 4. Receptive and expressive language quotients for the 83 and 68 children administered different forms of the Clinical Evaluation of Language Fundamentals battery. Thin lines are standard errors. As with children assessed with the TOLD battery, the average gains in the overall language quotient for a trained child were about 1 SD, or a change in their language quotients of about 15 points. Receptive language (or 'Listening Quotient') improvements exceeded expressive language (or 'Speaking Quotient') gains in both batteries.

training quotient distributions were biased toward the higher side of the normal distribution.

Similarly, Receptive and Expressive Language Quotients are shown before and after training for 83 and 68 children, respectively, after receiving different versions of the CELF language battery in Fig. 4. The open bars represent pre-training Quotients; the filled bars represent post-training Quotients. With only a few exceptions, the overall language performance abilities of children advanced with training, on the average by 1 SD for receptive language abilities, and by 0.9 SD for expressive language disabilities. Again, ANOVA showed that these before vs. after quotient differences were significant at the $p < 0.0005$ level (F = 80.7).

Post-training outcomes of attentional, emotional, pragmatic, reading and other related aspects of the 'language-learning impaired syndrome' were frequently reported by clinicians, teachers and parents to be markedly improved. These additional outcomes are now being quantitatively investigated. At this point, it is clear that there are substantial receptive and expressive language benefits of *Fast ForWord* training for at least a large proportion of trained children, with attentional and pragmatic improvements apparently occurring in many children as well.

It is clear from these studies that *Fast ForWord represents a major advance for language training for language-learning impaired children.*

Training impacts for different impaired populations

About half the children in this trial were specifically language-impaired children who failed at least one and usually multiple key subtest(s) on an appropriate language battery, or on the Tallal Repetition Test. Approximately 60 children had more severe impairments and other neurological diagnoses. Thirty-five of these children were classified as PDD. Nearly 100 children were also classified as having a Central Auditory Processing Disorder and almost 100 of trained language-impaired children were comorbid for Attentional Disorders.

Approximately equivalent results were obtained in all of these special populations. Although there were some subtest differences, overall Language Quotient gains for PDD-autistics, PDD-NOS, PDDs in general, language-impaired comorbid CAPDs, and ADDs, were not significantly different from the total population results when any one of these subject categories was excluded from it.

Remaining unresolved questions

We are now trying to document how improvements in signal reception abilities specifically relate to improvements in speech understanding, phonological awareness and language usage. Many more studies will have to be completed to determine how progressions at our seven exercises differentially account for the training gains recorded in studied children. Working cooperatively with the hundreds and soon thousands of speech therapists, special education teachers, audiologists, psychologists and other professionals in our internet-based collaborative network, those studies have now been initiated in earnest.

The documentation of precisely how abnormal masking accounts for speech reception errors in learning-impaired children is still incomplete. Again, it is a subject of current intense study in adult and child language-impaired and dyslexic individuals, because a deeper understanding of these issues could result in the development of even more effective training strategies.

Studies on the origins of language-learning impairments are being elaborated and extended in an attempt to understand more clearly what its true causes are in individual children. A major effort is being directed toward defining how very young children might be effectively trained to overcome their signal processing problems as infants or toddlers, and to determine whether, or not, and how very early intervention might ultimately prevent or reverse the establishment of language development problems.

Similarly, we are now conducting studies designed to show how we might more reliably and effectively initiate training in very severely impaired children. We are also elaborating training in these children to determine whether, or not, we can drive them still further into or toward the normal or above-normal language performance range with still more practice. We do not yet know whether, or not, the language abilities of these children will assymptote with training, or if we can drive further training improvements in at least some of them until even severely language-impaired children can achieve normal or high-normal performance abilities as a rule.

Finally, a series of follow-up studies must be completed to assess the longterm benefits of this new raining program. We must complete the documentation of pragmatic and emotional effects of intervention at an early age, and must quantitatively assess extended impacts on reading initiation and efficiency, and on academic and social achievements, all tracked across time.

Summary and Conclusion

The results of these studies argue strongly for our basic stated hypothesis: *The majority of language-learning impaired children have a fundamental deficit in their accurate reception of rapidly successive and fast-changing acoustic inputs*. Apparently, through increasing neural processing rates and in other ways refining acoustic signal reception, the aural reception of speech is clarified by training in these children. As a consequence, virtually every aspect of language reception and language use is significantly improved. The fault in these children, therefore, appears to lie at the base of language representation itself, i.e. with the salience of their aural speech inputs.

The fact that positive gains are recorded in the great majority of language-impaired children, ranging from severely impaired (autistic/PDD) to less severely impaired (comorbid CAPD, SLI) to more mildly impaired (dyslexics who may, or may not, have a strong documented record of language impairment) children, indicates that these populations share common neurological processing problems, as a large body of studies have long argued.

The fact that a very broad range of 'symptoms' and disabilities that apply ideosyncratically to different subpopulations of children appear to be positively impacted by this training indicate that they largely represent a rich panoply of compensating and coping strategies that are effects of the primary disability, and not a core part or cause of it. Indeed, at this point we are actively seeking component deficits that are decisively *unchanged* by training, as they might reflect either the irreversible deficits at the true core of these disabilities, or possibly other critical aspects of learning progression-based developmental disabilities that we can attack by other training strategies.

Acknowledgements

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