

OPINION

Improving language and literacy is a matter of time

Paula Tallal

Abstract | Developmental deficits that affect speech perception increase the risk of language and literacy problems, which can lead to lowered academic and occupational accomplishment. Normal development and disorders of speech perception have both been linked to temporospectral auditory processing speed. Understanding the role of dynamic auditory processing in speech perception and language comprehension has led to the development of neuroplasticity-based intervention strategies aimed at ameliorating language and literacy problems and their sequelae.

Concerns about language and literacy skills are increasing. Millions of students who start school with weak language skills fail to learn to read and drop out, with lowered prospects, before graduation. Despite the fact that US public schools spend twice as much money on educating special education students (a large proportion of whom have a language-based learning disability), reading scores in the United States have not improved over the past 20 years¹. National statistics show that 30% of youths with learning disabilities drop out of school, and 56% of these will be arrested². However, past experience has shown that increased funding³, educational focus^{1,2} and even political clout have failed to solve the literacy problem (FIG. 1). What is needed is a better scientific understanding of how the brain learns spoken and written language, and how to effectively transfer this scientific knowledge into practise.

Most scientific studies of reading have focused on dyslexia, a condition that is diagnosed when an otherwise normally developing child fails to learn to read, despite sufficient educational opportunity and normal intellect⁴. Dyslexia, originally called ‘word-blindness’, was thought to derive primarily from visual processing deficits, and to affect only written language. However, more recent research has focused on the role of acoustic, phonological and memory processes, as well as developmental language disorders, in the origins of reading disabilities^{4,5}. Furthermore, longitudinal studies have shown that more than 50 per cent of children who meet diagnostic criteria for specific language impairment (SLI — poor oral language skills but normal non-verbal abilities) subsequently or concurrently also meet the criteria for dyslexia^{5–8}, and many (but not all) people with dyslexia show deficits in aspects of oral language^{9–12}. Findings of similar (albeit not identical) patterns of deficits indicate that the difference between SLI and dyslexia might be quantitative rather than qualitative, and could be a question of maturation^{13–16}. To acknowledge the continuum between language and reading impairments in many children, I use the inclusive term ‘language learning impairment’ (LLI). However, although there are clear commonalities between SLI and dyslexia, there are also differences. Ultimately, we need a better understanding of within- as well as between-group individual differences before we can fully understand the neurobiological basis of the spectrum of these complex developmental disabilities.

Phonological processing deficits are not only characteristic of children with delayed language development¹⁷ — it is also possible to predict with considerable accuracy which children are going to struggle to read on the basis of their ability to manipulate phonemes within spoken words (for example, knowing that saying ‘plane’ without the /n/ sound is ‘play’)^{18–20}. The relationship between phonological deficits and language impairment seems obvious, but understanding the effects on reading requires a comprehension of the alphabetic principal that underlies reading. Learning to read alphabetic languages such as English depends on developing an awareness that printed characters (graphemes) correspond to phonemes, the smallest meaningful unit of sound that can change the meaning of a word. Proficiency in decoding words into their phonemic segments (phonemic awareness) is considered by many to be the core deficit in dyslexia⁴. Although other aspects of language (semantics, morphology, syntax and discourse) are also essential for reading fluency and comprehension^{9,11,12,21–24}, these have received considerably less research attention.

Although it is widely accepted that LLI is characterized by phonological deficits, the precise aetiology of these deficits remains the focus of intense research, and often heated theoretical debate. A central research question is whether phonological deficits are ‘speech-specific’, or whether they derive from more basic attention, perception, memory and/or motor constraints^{25–27}. Considerable research aimed at addressing this question has led to the development of several different models of LLI, including the rate-processing constraint hypothesis^{28–33}, the magnocellular deficit hypothesis³⁴, the cerebellar deficit hypothesis³⁵, the double-deficit hypothesis³⁶ and the attentional dwell time hypothesis³⁷. Interestingly, all of these hypotheses have in common a constraint in the speed of information processing and/or production that is proposed to disrupt essential components of language learning, beginning with the acquisition of phonological representations.

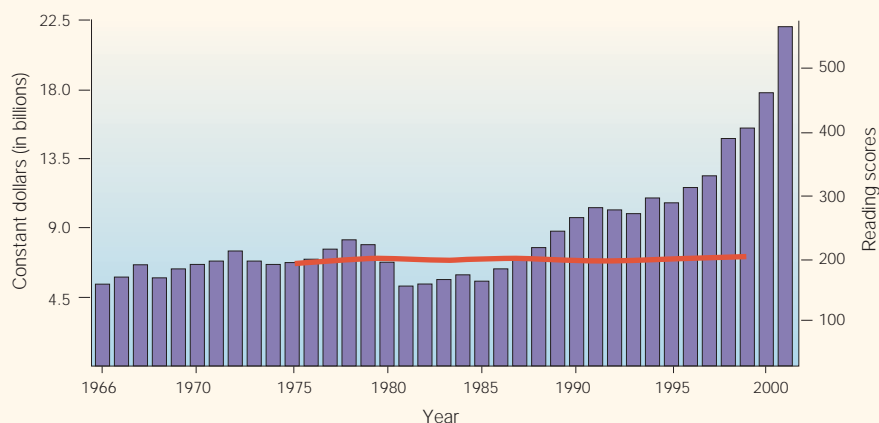


Figure 1 | History of low reading scores despite increased funding. Data on federal spending on education have been adjusted to provide consistency across time and to be comparable with the calculations of the 2005 US national budget³ (left axis, blue bars). Reading scores from the National Assessment of Educational Progress (NAEP) reflect the average performance of nine-year-old students who received the test in the years indicated in the graph (right axis, red line). Average scores 1975–1999 were below the score reflecting ‘partial mastery’ (208) as well as ‘complete mastery’ (238) of the knowledge and skills corresponding to proficient work for fourth grade students¹. During these years approximately only one third of fourth graders could read proficiently.

However, others have argued that these non-linguistic deficits occur in only a minority of individuals with LLI and, as such, are neither necessary nor sufficient to be causative^{38–41}. Thomas and Karmiloff-Smith⁴² recently suggested that much of this confusion derives from a failure to take a developmental neuroscience perspective. Although most early studies of developmental LLIs focused on young children who were failing to learn to talk, and subsequently to read, recent research has been dominated by studies of much older individuals (primarily college students) who have spent a lifetime developing alternative brain strategies to cope with their developmental disabilities. Unfortunately, key theories pertaining to dyslexia and other LLIs have rarely accounted for developmental and maturational brain changes that result from individual differences in early learning experience.

Auditory mapping of speech
How do phonemes come to be represented in the brain, and why do so many children with LLI have weak or imprecise phonological representation? An infant does not know which language(s) they will need to learn. Each language has its own set of phonemes that must be learned from experiencing the ongoing speech stream of the native language. FIGURE 2a shows the spectrogram (frequency-by-time display) of the acoustic energy that is produced when a sentence is spoken, and FIG. 2b highlights two distinct consonant-vowel (CV) syllables (/ba/ and /da/) segmented out of the sentence. Note that no time gaps or

explicit boundaries segment the ongoing speech stream into distinct phonemes or syllables. The acoustic waveform of speech is continuous, complex and characterized by rapid acoustic changes in frequency and intensity (known as formant transitions), followed rapidly in succession by steady-state vowels or other acoustic segments that are produced sequentially as we move our speech articulators. For example, in the syllables /ba/ and /da/, the only differentiating cues occur within the initial 40-millisecond formant transition.

Physiological mapping studies have shown that the detailed organization of the auditory cortex is driven by environmental input during critical periods of development⁴³. Exposure to altered acoustic input during critical periods of early development, such as continuous or pulsed noise, significantly disrupts the development of tonotopic representation in the primary auditory cortex, and these developmental changes persist into adulthood^{44,45}. Beyond the critical period, these sensory/neural maps can only be driven to change by intensive neuroplasticity-based training⁴⁶. In addition to tonotopic (frequency) representation in the primary auditory cortex⁴⁷, there are neurons that code selectively for temporal features of sound^{48–50}, as well as ‘inseparable’ temporospectral combinations, such as frequency sweeps similar to those that occur in formant transitions of speech^{51–53}. These studies show that the auditory cortex is shaped early in life by the features and statistical probability of occurrence of acoustic input during critical periods of development.

Considering the amount of speech that is directed to an infant, it is easy to understand the importance of speech in shaping the auditory cortex during critical periods of development^{54,55}. To aid this process, adult speech to infants (known as ‘parentese’) has been shown to exaggerate (extend in time and amplitude enhance) the acoustic changes that differentiate phonemes in syllables and words⁵⁶. We have previously suggested that during critical periods of development Hebbian learning⁵⁷ drives the auditory cortex to ‘represent’ each phoneme of the native language as a distinct neural firing pattern (‘cell assembly’) on the basis of its characteristic acoustic temporospectral features. First, however, the brain must segment the ongoing acoustic waveform of speech into chunks of time in which acoustic patterns occur frequently and consistently. Consistencies in the speech waveform can occur in ‘chunks’ of various durations. Chunking in the tens of milliseconds time window would allow the fine grain analysis that is needed to represent the acoustic differences between phonemes such as /b/ and /d/. However, chunking over longer periods of time (hundreds of milliseconds) would result in firing patterns that are consistent with syllable or word-length representations²⁸.

Language learning impairments

Infants as young as 6 months old require, on average, silent gaps of only tens of milliseconds to discriminate between two brief (70 msec) tones that differ in frequency⁵⁸. However, over 30 years of behavioural as well as electrophysiological research has shown that it is not uncommon for young children (5–9 years old) with LLI to require hundreds of milliseconds to perform this and other temporospectral acoustic tasks (for review, see REF. 28). On the basis of these findings, we proposed, and subsequently showed, that children with LLI are specifically impaired in both their ability to discriminate between^{59–61} and to produce⁶² speech sounds that are characterized by brief, rapidly successive acoustic changes, such as the brief formant transitions (40 msec) preceding the steady-state portion of the vowel, which are the sole differentiating feature between syllables such as /ba/ and /da/. However, they are unimpaired in processing speech contrasts that are based on acoustic cues of longer duration, such as 250 msec duration steady-state vowels (/E/ versus /ae/).

To demonstrate that the speech deficits of these children relate specifically to the duration of brief, rapidly successive acoustic cues in speech, two acoustic manipulations were used. First, the syllables /ba/ and /da/ were computer

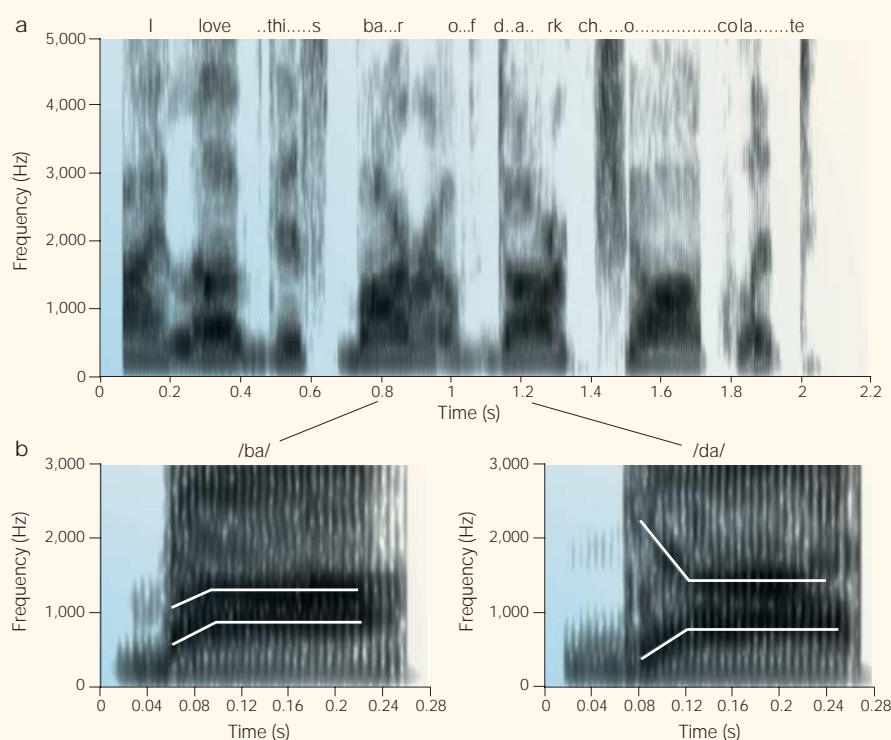


Figure 2 | **Acoustic energy produced by speech.** Spectrogram showing the frequency-by-time acoustic energy produced when **a** a sentence is spoken and **b** | for individual syllables (/ba/ and /da/) segmented out from the sentence.

synthesized and the duration of the formant transitions preceding the steady-state vowel segment extended from 40 msec to 80 msec, resulting in significant improvement in speech discrimination. Second, decreasing the duration of steady-state vowels (/E/ and /ae/) preceding the vowel /I/ from 250 msec to 40 msec (as would occur in the diphthongs /EI/ versus /aeI/) resulted in a significant decrement in discrimination⁶³.

The finding that speech perception could be significantly improved by temporospectral acoustic modification led to the development of an algorithm that could perform these manipulations in the acoustic waveform of ongoing speech⁶⁴. This formed the basis of a new intervention strategy called Fast For Word®, which combines acoustically modified speech with explicit phonological, language and reading intervention in a series of neuroplasticity-based training exercises disguised as computer games (see BOX 1).

Does acoustic speed predict verbal intelligence? [Au pls shorten to 1 line]
Is there any evidence that some infants require more acoustic chunking time than others? That is, is there evidence that what is chunked as 'nearly simultaneous in time' might differ between individuals, and, if so, is there any evidence that individual differences

in rapid auditory processing thresholds affect language development and/or verbal intelligence? To investigate these questions, A. Benasich and I developed a method for establishing individual rapid auditory processing (RAP) thresholds in infants. Language and cognitive development were then assessed prospectively and longitudinally in infants until they reached 36 months of age. As seen in FIG. 3, infants were operantly trained to look to a toy on their right when they heard one tone sequence (high–low) and to their left when they heard a different tone sequence (low–low). The training stimulus incorporated two 70 msec duration tones separated by a 500 msec silent inter-stimulus interval (ISI) between the end of the first tone and the beginning of the second tone in each sequence. Once the infant had learned this task to criterion, the ISI between tones was systematically decreased for correct responses or increased for incorrect responses, until an individual RAP threshold was established for each infant. In subsequent studies, an easier Go/No Go (oddball) variation on this task has been used in which the infant listens to a repeating tone sequence (low–low) and is trained to make a head turn when a different sequence (low–high) is detected. An infant performing this task can be seen online in [supplementary information S1](#) (movie).

Two groups of infants have been studied, one with a positive family history (FH+) for SLI, and one without (FH–). Familial genetic studies indicate that approximately 50% of infants born into FH+ families are at risk of developing similar problems¹⁴, so we predicted that if RAP differences in infancy were related to language outcomes, approximately 50% of the infants in the FH+ group would have elevated RAP thresholds. We further predicted that individual RAP thresholds in infancy (across both groups) would account for a significant degree of variance in subsequent language development and verbal intelligence. This is precisely the pattern that was found. Infant testing revealed a highly significant difference in RAP thresholds between the FH+ and FH– infant groups⁶⁵. On follow up, we found that among a large battery of sensory, perceptual and cognitive measures, infant RAP thresholds were the single best predictor of language outcomes at 2 years of age. By 3 years of age, two variables — RAP thresholds obtained at 6 months and male gender — together predicted 39–41% of the variance in language outcome. Furthermore, these two infant variables accurately classified 91.4% of 3-year-old children who scored in the 'impaired' range on the Verbal Reasoning scale of the Stanford-Binet intelligence scales. Importantly, none of the infant variables could discriminate between 3-year-old children on the non-verbal portions of the Stanford-Binet, demonstrating the specificity of the relationship between individual differences in infant RAP thresholds and subsequent individual differences in language and verbal intelligence⁶⁸.

These studies show that early individual differences in RAP both precede and predict subsequent language development and disorders. A similar predictive relationship between silent gap detection thresholds in infancy and subsequent language development is consistent with this⁶⁶. These results provide a valuable developmental perspective from which to discuss the role of individual differences in auditory processing — specifically, rapid temporospectral processing — in phonological and language development and disorders. This cohort is currently being followed longitudinally to determine the specific role of RAP, as well as early language development, in the acquisition of reading.

Not all studies have found such compelling evidence for RAP deficits in children with SLI and/or dyslexia, and, of those that do, some still question the causal relationship between elevated RAP thresholds and various aspects of phonological processing, language

Box 1 | The Fast ForWord® neuroplasticity-based training approach



Fast ForWord®, developed by Scientific Learning Corporation, is a series of neuroplasticity-based training programmes that are designed to improve fundamental aspects of oral and written language comprehension and fluency. The exercises incorporate two simultaneous approaches to intervention, disguised as a series of computer ‘games’. In one approach (Circus Sequence), subjects indicate the temporal order of tones that either rise or fall in pitch and that have been designed to cover the basic range of frequencies and speeds that typify the temporospectral changes that occur in formant transitions in consonants. The computer program adaptively changes on the basis of each subject’s trial-by-trial performance — correct responses are rewarded and errors corrected. The goal of the exercise is to increase the ability to attend to and sequence increasingly, rapidly changing acoustic stimuli, and to expand the memory span for rapidly successive events, until levels typically found in the acoustic changes that characterize ongoing speech are reached.

The second approach uses a computer algorithm to acoustically modify (temporally extend and amplify) the rapidly successive acoustic changes that occur in ongoing speech⁶⁴. This acoustic modification is intended to emphasize the rapid temporospectral acoustic changes that mark key differences between brief phonemes in the ongoing waveform of speech. This acoustically modified speech algorithm is used in a series of exercises to cross-train individual components of language and reading across multiple levels, from the phoneme (Phonic Match) to the word (Phonic Word) to grammatical sentences (Language Comprehension Builder). As a rule, as linguistic performance improves, the amount of acoustic modification adaptively decreases until all the exercises can be performed accurately with normal speech.

With the aid of computer technology, and the Internet for data transfer, the Fast ForWord® series of training programmes (which now incorporates both a language and reading series) has, to date, been applied to over 375,000 children in over 2,000 schools. See online links for data from clinical and school trials with multiple populations (Fast ForWord® results), to try an animated demo of Circus Sequence and Phonic Match, and to hear stimuli from various levels of each task (Fast ForWord® language exercises).

not homogeneous, and symptomatology can change markedly during development. The extent to which differences across studies reflect these methodological issues, or differences in the aetiological origins in subsets of these disorders, are areas in need of further research.

Longitudinal studies have demonstrated that many children have RAP deficits early in life that subsequently resolve⁷ or become difficult to assess using behavioural techniques. However, a recent study showed that RAP deficits that could only be documented in a minority of older individuals with LLI with behaviour measures could be uncovered in most of the same subjects using more sensitive electrophysiological techniques¹³. So, early auditory deficits, differences or experiences are likely to affect the sharpness of phonological representations that are established through experience-dependent learning in infancy, leaving a lasting effect of phonological impairment.

The data derived from prospective, longitudinal studies of ‘at risk’ infants give us perhaps the most compelling evidence of the developmental impact of RAP thresholds on language development and disorders⁵⁸. Other compelling data are coming from studies of animals with induced cortical neuromigrational or genetic anomalies that are designed to mimic those found in LLI^{67–70}. These ‘animal models’ show a strikingly similar pattern of thalamocortical magnocellular disruption^{70,71}, as well as behavioural RAP deficits²⁸, to those seen in people with LLI. These animal data not only add converging evidence to the human studies, but they also open the door for a more rigorous exploration of the neurobiological substrates that underlie LLI than is possible in humans.

Spike-timing dependent learning
Speech is not comprised of random chunks of acoustic information, but is organized in a rule-based sequential manner. One dominant function of the neocortex is prediction and sequence learning^{72–76}. At the cellular level, spike-timing dependent synaptic learning is a powerful mechanism for prediction plasticity (or what Hebb referred to as ‘temporally asymmetric learning’), capturing the influence of relative timing between input and output spikes in a neuron⁷⁶. Specifically, an input synapse to a given neuron that is activated slightly before the neuron fires is strengthened, whereas a synapse activated slightly after is weakened. This window of plasticity typically ranges from –40 to +40 msec. It has recently been shown that

or reading^{38–41}. Not all individuals with LLI demonstrate current RAP deficits, and, conversely, there are individuals who do poorly on measures of RAP, but who nonetheless develop adequate language and reading. However, there are important methodological

differences that might explain discrepancies across studies, notably the age of subjects, as well as the difficulty and age-appropriateness of the stimuli and tasks that were used. Furthermore, as is the case with all complex developmental disabilities, these disorders are



Figure 3 | Obtaining a rapid auditory processing threshold using a two-alternative forced choice paradigm. The infant's attention is maintained forward with colored slides while the infant listens to tone sequences. Infants are operantly conditioned to look to the electronic toy on their right for the tone sequence low-high and to their left for the sequence low-low. Correct responses (monitored by a video camera in the centre of the apparatus) activate the toy. The inter-stimulus-interval between tones is decreased after correct and increased after incorrect responses until a threshold is reached⁵⁸. For video, see online [supplementary information S1](#) (movie). FIGURE and video courtesy of A. Benasich, Rutgers University, Newark.

spike-based temporal difference learning causes neurons to become direction selective when exposed to moving visual stimuli⁷⁵. Could the same be true in the auditory system of humans?

Although speculative at this point, I propose that similar mechanisms might underlie important aspects of acoustic/phonetic perception and language learning. Such a mechanism, when exposed to the rapid temporospectral changes that characterize speech, would cause multiple neurons in the primary auditory cortex that fire nearly simultaneously to bind together. Not only would such temporally contiguous, frequent patterns of feature activation build cell assemblies representing discrete phonemes, but such activity in one cortical circuit could, through converging projections, activate other cortical areas, leading to a sequence of activations that Hebb called a 'phase sequence'^{57,76}. The theoretical application to speech processing would indicate that spike-based temporal difference learning would result in neurons becoming direction selective for the rapidly changing perceptual and motor patterns that characterize ongoing speech. Such a mechanism could account not only for the development and sharpening of neural representations for distinct phonemes that occur repeatedly in a language, but also for the elimination through regressive and inhibitory processes of phonemes and sequential patterns that do

not occur in an infant's native language⁷⁷. It is provocative that the critical time window for spike-timing dependent long-term potentiation is approximately 40 msec, as this also is the critical time window of the rapid temporospectral acoustic changes in formant transitions that are important for tracking temporal order across ongoing speech⁷⁸.

Sejnowski and colleagues suggest that the temporally asymmetric Hebbian learning rule is equivalent to the temporal difference learning algorithm in reinforcement learning, and that it can be used to make predictions and implement classical conditioning⁷⁹. The temporal window for classical conditioning is several seconds — much longer than the window for long-term potentiation/long-term depression that is observed at cortical⁸⁰ and hippocampal^{81,82} synapses. Therefore, they further predict that a circuit of neurons in the basal ganglia and frontal cortex might be needed to extend the computation of temporal differences to these long time intervals. This would indicate that the temporal order of input stimuli is a useful source of information about causal dependence, not only in many different learning contexts, but also over a range of timescales. There is ample evidence that children with LLI have difficulty with temporal order judgements over many timescales, ranging from the perception of brief, successive acoustic stimuli^{29,32,83,84}, to performing rapid sequential motor movements^{85,86}, to learning grammatical rules based on word order (such as 'the girl is chasing the boy')⁸⁷. There is also considerable evidence from physiological and neuroimaging studies that individuals with LLI have morphological, physiological and connectivity disturbances in the basal ganglia/cortical circuit that is thought to underlie Hebb's temporally asymmetric learning rules^{32,70,71,88,89}. Connectivity across this system could potentially drive the development of predictive rules that govern language-specific phoneme order, word segmentation and even grammatical rule systems. Deficits or delays in maturation¹³ or myelination in this system could disrupt or delay language development.

Current thinking on issues such as spike timing, spike synchrony and neural oscillations is at the forefront of theoretical and computational research into the role of the temporal cortical neuronal code^{90–92}. With the development of single-trial functional magnetic resonance imaging (fMRI) and other, more temporally relevant, neuroimaging technologies such as event-related potential and magnetoencephalography,

we might now be poised to extend such theories to the study of language development and disorders in humans.

Neuroplasticity and remediation

It has long been thought that sensory neural maps were established for a lifetime during early critical periods of development. However, more recent physiological studies, which show that sensory neural maps can be substantially altered at the cellular level by intensive behavioural training in adult animals, have challenged this perspective⁹³.

In the early 1990s, M. Merzenich, W. Jenkins, S. Miller and I began to discuss whether the results of neuroplasticity-based training studies in animals might be applied to children. Of particular interest were studies that showed that the grain of analysis for the rapid segmentation of sensory events could be significantly sharpened by intensive, adaptive behavioural training^{46,93}. We were eager to see whether similar methods could be developed with the aim of ameliorating the RAP deficits that are characteristic of many children with LLI, and, if so, whether improvements in phonological, language and reading skills might follow. These discussions led to a series of laboratory and field studies, and subsequently to the development of a new neuroplasticity-based training approach called Fast ForWord[®] (see BOX 1).

In our original laboratory studies, two matched groups of children with LLI participated in daily training for approximately 3 hours per day for 4 weeks^{94,95}. The experimental group was trained with the two approaches described in BOX 1. The treatment control group received precisely the same language intervention, but with speech that was not acoustically modified, and instead of the auditory tone sequencing exercise (Circus Sequence), which is aimed at speeding rapid auditory processing, the control group played non-temporally adapted visual computer games. After training the experimental group showed a substantial improvement in the rate of acoustic processing, with many (but not all) subjects moving from thresholds in the hundreds of milliseconds range to the tens of milliseconds range. In addition, many of the trained subjects showed significant improvements in speech discrimination and language comprehension⁹⁴. There was a highly significant correlation between decreases in RAP threshold and increases in language skills⁹⁵.

It is striking to note that expressive language also improved significantly, although no explicit training in motor speech or expressive language was provided. Recent brain imaging studies, which showed activation in primary

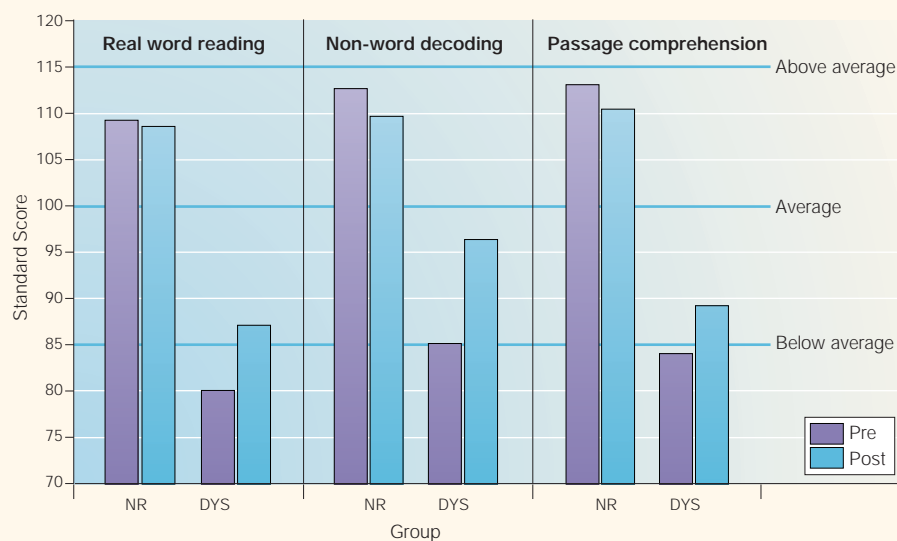


Figure 4 | Improvements in language and reading scores after neuroplasticity-based training. Standard scores on the three subtests of the Woodcock-Johnson Reading Mastery Test – Revised (AGS Publishing, Circle Pines, Minnesota), for normal readers (NR) as compared with readers with dyslexia (DYS), before (purple bars) and after (blue bars) Fast ForWord®-Language training. Mean = 100; standard deviation = 15; below average = 85. Adapted from data in REF. 104.

motor areas during speech perception, have renewed interest in the idea that motor areas are involved in perceptual processing⁹⁶. So, the finding of improved speech production after perceptual training is of considerable theoretical interest and is a fruitful area for future research.

As with any intervention, not all children improved to the same extent, and some did not improve at all. Furthermore, many children in the control group who received the same intensive language intervention, but without the benefit of the acoustically modified speech or RAP training, also showed improvements, leading to questions about the specificity of the results to the temporal manipulations *per se*. Aspects of this intervention share some features in common with many other successful treatment approaches, specifically the intensity and consistency of treatment, as well as explicit training of one or more components of language (phonology, semantics and/or syntax)^{97–101}. However, these factors were explicitly addressed in the randomized, treatment control group. Although both groups showed significant gains over baseline performance on language measures, the experimental group that received language training with acoustically modified speech, coupled with RAP training, showed a statistically significant advantage over the treatment control group. These laboratory results have been replicated in an independent study with French children with dyslexia, using a similar acoustic modification algorithm to train phonological awareness

abilities, therefore extending their potential relevance to reading intervention¹⁰². These results have been extended and replicated in numerous school and clinical settings with thousands of children (see online links box). Although these were not all randomly controlled trials, and with the caveat that similar results are not obtained in all cases, these widespread results show the practical application that is possible when scientifically-based methods are adequately implemented in schools and clinics with appropriate children.

Despite good overall success with these first-generation neuroplasticity-based training approaches, there remains a percentage of children who improve only slightly or not at all. Long-term follow up studies of trained children are needed, together with a better understanding of individual differences in outcomes that might be influenced by the clinical profile and learning environment of each child. Additional research is needed to reach a better understanding of which specific components of this and other intervention programmes drive which specific outcomes for which children.

Remediation and neuroimaging

We proposed that neuroplasticity-based intervention aimed at improving RAP thresholds and sharpening acoustic/phonetic processing would have an impact not only on oral language, but also on reading decoding and comprehension skills, and would do so by 're-mapping' brain areas that are important for these functions. Specifically, we suggested

that driving RAP thresholds from the hundreds of milliseconds into the tens of milliseconds time window would provide the neural substrate that is necessary for chunking the ongoing acoustic waveform of speech at the finer-grain level that is required to build distinct (categorical) phoneme representations. This should improve the ability of individuals to detect phonemes within words, and therefore the ability to learn letter-sound correspondences. In addition, we proposed that explicit training in the rules of English grammar, presented initially with acoustically modified speech, then individually adapted back to normal speech, should lead to improvements in reading comprehension and fluency. Finally, on the basis of research that shows evidence for deficits in auditory attention³⁷ and memory¹⁶, we proposed that adding intensive training components in these areas should be important for reaching the largest numbers of children, specifically those with multiple deficits who have historically been 'hard to remediate' with more traditional approaches.

Recently, researchers at Stanford University used behavioural as well as fMRI techniques to evaluate these proposals in people with dyslexia^{103–105}. In the most recent study¹⁰⁴, 20 children with dyslexia and 12 matched typical readers received a battery of language and reading tests, and 2 fMRI scans while performing a letter rhyming task, approximately 8 weeks apart. Between scans, the children with dyslexia completed the Fast ForWord®-Language training programme. After training, the children with dyslexia showed a significant improvement in performance on standardized language and reading tests (FIG. 4). Importantly, word-decoding scores (representing the core deficit of phonemic awareness) moved from one standard deviation below the mean before training to within the normal range after training. Comparable improvements in reading comprehension were also documented. In addition to significantly improved reading in the trained dyslexic group, fMRI results showed 'normalization' (increased metabolic activity) in left-hemisphere temporoparietal language regions (FIG. 5). The control group showed no significant behavioural or fMRI test-retest changes. These results replicate the pattern of results found in a similar study with adults with dyslexia¹⁰⁵, demonstrating that neuroplastic changes in the brain can be driven by behavioural training throughout an individual's lifespan.

Additional fMRI studies, including a matched control group of children with dyslexia who receive a different form of intervention, are currently underway to increase

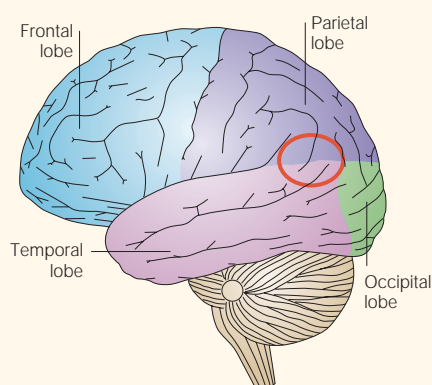


Figure 5 | **Normalization of brain activity after neuroplasticity-based training.** The red ring indicates the left-hemisphere temporoparietal language region, which is normally active during phonological processing, but is not active in children and adults with dyslexia. This area showed increased activity in individuals after participation in the Fast ForWord®-language programme. Adapted from data in REF. 104.

our understanding of the nature of the neuroplastic changes that occur with various forms of remediation. Specifically, we need to understand better the multiple changes that occurred in the brains of the trained children, not only in left-hemisphere language areas, but also in homologous areas of the right hemisphere, as well as other brain areas. Other studies have recently reported similar fMRI¹⁰⁶ or electrophysiological¹⁰⁷ changes after intensive, explicit phonological awareness training. These are among the first studies to show that fMRI and other neuroimaging modalities might become a useful adjunct to evaluating and comparing the efficacy of various treatment approaches. The long-term goal of this research is to elucidate the neurobiological signature of different forms of LLI, as well as various treatment approaches, so that more efficacious treatment approaches can be designed to meet the needs of each child.

Conclusion

The significant improvements in reading following Fast ForWord®-Language training provide strong support for the theoretical premise that initially drove the hypothesis linking rapid auditory processing, language and reading. It is important to remember that this series of training exercises does not incorporate any letters at all, but was designed to improve the rate of auditory sequential processing, attention, memory, phonological processing and grammatical skills. The finding of improved reading immediately after this training demonstrates the importance of these essential building blocks not only for language, but also for reading success. In turn, these results

highlight the importance of designing more neuroscience-informed intervention strategies for individuals who continue to struggle with language and reading when more traditional educational and clinical approaches are used.

These laboratory studies also have considerable practical implications for the development of neuroscience-based intervention strategies for use with other populations who are struggling with speech, language and literacy skills, including deaf children with cochlear implants¹⁰⁸, individuals with central auditory processing disorders¹⁰⁹ and patients with acquired aphasia resulting from brain injury¹¹⁰. It is also provocative to note that studies of older adults have implicated slowing of auditory processing speed as an important concomitant to language and cognitive decline^{111–113}. Public schools are struggling to meet the requirements of the 'No Child Left Behind' legislation, and increasing numbers of our population are experiencing the cognitive and language declines associated with ageing. However, this research offers new insights into the essential role of temporal processing in the development and maintenance of language functions, and how basic neuroscience research is leading to improved intervention strategies.

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- Campbell, J. R., Hombo, C. M. & Mazzeo, J. *NAEP 1999 Trends in Academic Progress: Three Decades of Student Performance* (US Department of Education, Office of Educational Research and Improvement, National Center for Education Statistics, Washington DC, 2000).
- Riley, R. W. Speech presented at the National Summit on Learning Disabilities, Washington DC [Online archive of talking points] <<http://www.ed.gov/Speeches/09-1994/learnsum.html>> (September, 1994).
- Whitehouse Office of Management and Budget. Historical tables, budget of the United States government, Fiscal Year 2005 (US Government Printing Office, Washington D. C. [Online] <<http://www.whitehouse.gov/omb/budget/fy2005/>> 2004).
- Lyon, G. R. Towards a definition of dyslexia. *Ann. Dyslexia* **45**, 3–27 (1995).
- Bishop, D. V. M. & Snowling, M. J. Developmental dyslexia and specific language impairment: same or different? *Psychol. Bull.* (In the press).
- Catts, H. W., Fey, M. E., Tomblin, J. B. & Zhang, X. A longitudinal investigation of reading outcomes in children with language impairments. *J. Speech Lang. Hear. Res.* **45**, 1142–1157 (2002).
- Stark, R. E. et al. Four-year follow-up study of language impaired children. *Ann. Dyslexia* **34**, 49–68 (1984).
- Tallal, P., Allard, L., Miller, S. & Curtiss, S. in *Dyslexia: Biology, Cognition and Intervention* (eds Hulme, C. & Snowling, M.) 167–181 (Whurr, London, 1997).
- Byrne, B. Deficient syntactic control in poor readers: is a weak phonetic memory code responsible? *Appl. Psycholinguistics* **2**, 201–212 (1981).
- Carroll, J. M. & Snowling, M. J. Language and phonological skills in children at high-risk of reading difficulties. *J. Child Psychol. Psychiatry* **45**, 631–640 (2004).
- Joanisse, M. F., Manis, F. R., Keating, P. & Seidenberg, M. S. Language deficits in dyslexic children: speech perception, phonology, and morphology. *J. Exp. Child Psychol.* **77**, 30–60 (2000).
- Scarborough, H. S. Very early language deficits in dyslexic children. *Child Dev.* **61**, 1728–1743 (1990).
- Bishop, D. V. M. & McArthur, M. Immature cortical responses to auditory stimuli in specific language impairment: evidence from ERPs to rapid tone sequences. *Dev. Sci.* **7**, 11–18 (2004).
- Flax, J. F. et al. Specific language impairment in families: evidence for co-occurrence with reading impairments. *J. Speech Lang. Hear. Res.* **46**, 530–543 (2003).
- McArthur, G. M., Hogben, J. H., Edwards, V. T., Heath, S. M. & Mengler, E. D. On the 'specifics' of specific reading disability and specific language impairment. *J. Child Psychol. Psychiatry* **41**, 869–874 (2000).
- Gathercole, S. E. & Baddeley, A. D. Phonological memory deficits in language disordered children: is there a causal connection? *J. Mem. Lang.* **29**, 336–360 (1990).
- Leitão, S., Hogben, J. & Fletcher, J. Phonological processing skills in speech and language impaired children. *Eur. J. Disord. Commun.* **32**, 73–93 (1997).
- Castles, A. & Coltheart, M. Is there a causal link from phonological awareness to success in learning to read? *Cognition* **91**, 77–111 (2004).
- Elbro, C., Borstrom, I. & Petersen, D. K. Predicting dyslexia from kindergarten: the importance of distinctness of phonological representations of lexical items. *Reading Res. Q.* **33**, 36–60 (1998).
- Lundberg, L., Olofsson, A. & Wall, S. Reading and spelling skills in the first school years predicted from phonemic awareness skills in kindergarten. *Scand. J. Psychol.* **121**, 159–173 (1980).
- Bishop, D. V. M. *Uncommon Understanding* (Psychology Press, Hove, 1997).
- Leonard, L. B. *Children with Specific Language Impairment* (MIT Press, Cambridge, 1998).
- Brady, S. A., Shankweiler, D. & Mann, V. A. Speech perception and memory coding in relation to reading ability. *J. Exp. Child Psychol.* **35**, 345–367 (1983).
- Wolf, M. Rapid alternating stimulus naming in developmental dyslexias. *Brain and Lang.* **27**, 360–379 (1986).
- Farmer, M. E. & Klein, R. M. The evidence for a temporal processing deficit linked to dyslexia: a review. *Psychonomic Bull. Rev.* **2**, 460–493 (1995).
- Habit, M. The neurological basis of developmental dyslexia: an overview and working hypothesis. *Brain* **123**, 2373–2399 (2000).
- Tallal, P., Galaburda, A., Von Euler, C. & Llinas, R. (eds) *Temporal Information Processing in the Nervous System* (New York Academy of Science, New York, 1993).
- Fitch, R. H. & Tallal, P. Neural mechanisms of language-based learning impairments: insights from human populations and animal models. *Behav. Cogn. Neurosci. Rev.* **2**, 155–178 (2003).
- Hari, R. & Klesla, P. Deficit of temporal auditory processing in dyslexic adults. *Neurosci. Lett.* **205**, 138–140 (1996).
- Talcott, J. B. et al. Dynamic sensory sensitivity and children's word decoding skills. *Proc. Natl Acad. Sci. USA* **97**, 2952–2957 (2000).
- Witton, C. et al. Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. *Curr. Biol.* **8**, 791–797 (1998).
- Kraus, N. et al. Auditory neurophysiological responses and discrimination deficits in children with learning problems. *Science* **273**, 971–973 (1996).
- Wright, B. A., Bowen, R. W. & Zecker, S. G. Nonlinguistic perceptual deficits associated with reading and language disorders. *Curr. Opin. Neurobiol.* **10**, 482–486 (2000).
- Stein, J. & Talcott, J. Impaired neuronal timing in developmental dyslexia: the magnocellular hypothesis. *Dyslexia* **5**, 59–77 (1999).
- Nicolson, R. I., Fawcett, A. J. & Dean, P. Developmental dyslexia: the cerebellar deficit hypothesis. *Trends Neurosci.* **24**, 508–511 (2001).
- Wolf, M. & Obregon, M. Early naming deficits, developmental dyslexia, and a specific deficit hypothesis. *Brain Lang.* **42**, 219–247 (1992).
- Hari, R., Viata, M. & Uutela, K. Prolonged attentional dwell time in dyslexic adults. *Neurosci. Lett.* **271**, 202–204 (1999).
- Bishop, D. V. M., Carlyon, R. P., Deeks, J. M. & Bishop, S. J. Auditory temporal processing impairment: neither necessary nor sufficient for causing language impairment in children. *J. Speech Hear. Res.* **42**, 1295–1310 (1999).
- Mody, M., Studdert-Kennedy, M. & Brady, S. Speech perception deficits in poor readers: auditory processing or phonological coding? *J. Exp. Child Psychol.* **64**, 199–231 (1997).
- Ramus, F. Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction? *Curr. Opin. Neurobiol.* **13**, 212–218 (2003).

41. Rosen, S. & Manganari, E. Is there a relationship between speech and nonspeech auditory processing in children with dyslexia? *J. Speech Lang. Hear. Res.* **44**, 720–736 (2001).
42. Thomas, M. & Karmiloff-Smith, A. Are developmental disorders like cases of adult brain damage? Implications from connectionist modelling. *Behav. Brain Sci.* **25**, 727–750 (2002).
43. Merzenich, M. M. & Schreiner, C. E. in *The Evolutionary Biology of Hearing* (eds Webster, D. B., Fay, R. F. & Popper, A. N.) 673–689 (Springer, New York, 1992).
44. Chang, E. F. & Merzenich, M. M. Environmental noise retards auditory cortical development. *Science* **300**, 498–502 (2003).
45. Zhang, L. I., Bao, S. & Merzenich, M. M. Disruption of primary auditory cortex by synchronous auditory inputs during a critical period. *Proc. Natl Acad. Sci. USA* **99**, 2309–2314 (2002).
46. Recanzone, G. H., Schreiner, C. E. & Merzenich, M. M. Plasticity in the frequency representation of primary auditory cortex following discrimination training in adult owl monkeys. *J. Neurosci.* **13**, 87–104 (1993).
47. Schreiner, C. E. Functional topographies in the primary auditory cortex of the cat. *Acta Otolaryngol.* **491** (Suppl.), 7–15 (1991).
48. Phillips, D. P., Mendelson, J. R., Cynader, M. S. & Douglas, R. M. Responses of single neurones in cat auditory cortex to time-varying stimuli: frequency-modulated tones of narrow excursion. *Exp. Brain Res.* **58**, 443–454 (1985).
49. Langner, G. & Schreiner, C. E. Periodicity coding in the inferior colliculus of the cat. I. Neuronal mechanisms. *J. Neurophysiol.* **60**, 1799–1822 (1988).
50. Blake, D. T., Strata, F., Churchland, A. & Merzenich, M. M. Neural correlates of instrumental learning in primary auditory cortex. *Proc. Natl Acad. Sci. USA* **99**, 10114–10119 (2002).
51. Eggermont, J. J. Spectrotemporal characterization of auditory neurons: redundant or necessary? *Hear. Res.* **5**, 109–121 (1981).
52. Linden, J. F., Liu, R. F., Sahani, M., Schreiner, C. E. & Merzenich, M. M. Spectrotemporal structure of receptive fields in areas A1 and AAF of the mouse auditory cortex. *J. Neurophysiol.* **90**, 2660–2675 (2003).
53. Orduna, I., Mercado, E., Guck, M. A. & Merzenich, M. M. Spectrotemporal sensitivities in rat auditory cortical neurons. *Hear. Res.* **160**, 47–57 (2001).
54. Jusczyk, P. W. How infants adapt speech processing capacities to native-language structure. *Curr. Dir. Psychol. Sci.* **11**, 15–18 (2002).
55. Kuhl, P. A new view of language acquisition. *Proc. Natl Acad. Sci. USA* **97**, 11850–11857 (2000).
56. Liu, H. M., Kuhl, P. K. & Tsao, F. M. An association between mothers' speech clarity and infants' speech discrimination skills. *Dev. Sci.* **6**, 1–10 (2003).
57. Hebb, D. O. *The Organization of Behavior: A Neuropsychological Theory* (Wiley, New York, 1949).
58. Benasich, A. A. & Tallal, P. Infant discrimination of rapid auditory cues predicts later language impairment. *Behav. Brain Res.* **136**, 31–49 (2002).
59. Stark, R. E. & Tallal, P. *Language, Speech, and Reading Disorders in Children: Neuropsychological Studies* (Little, Brown and Co. Inc., Boston, Massachusetts, 1988).
60. Tallal, P. & Piercy, M. Developmental aphasia: rate of auditory processing and selective impairment of consonant perception. *Neuropsychologia* **12**, 83–93 (1974).
61. Tallal, P. & Stark, R. Speech acoustic cue discrimination abilities of normally developing and language impaired children. *J. Acoust. Soc. Am.* **69**, 568–574 (1981).
62. Stark, R. & Tallal, P. Analysis of stop consonant production errors in developmentally dysphasic children. *J. Acoust. Soc. Am.* **66**, 1703–1712 (1979).
63. Tallal, P. & Piercy, M. Developmental aphasia: the perception of brief vowels and extended stop consonants. *Neuropsychologia* **13**, 69–74 (1975).
64. Nagarajan, S. S. *et al.* Speech modifications algorithms used for training language learning-impaired children. *IEEE Trans. Rehabil. Eng.* **6**, 257–268 (1998).
65. Benasich, A. A. & Tallal, P. Auditory temporal processing thresholds, habituation and recognition memory over the first year. *Infant Behav. Dev.* **19**, 339–357 (1996).
66. Trehub, S. E. & Henderson, J. L. Temporal resolution in infancy and subsequent language development. *J. Speech Hear. Res.* **39**, 1315–1320 (1996).
67. Galaburda, A. M. Developmental dyslexia and animal studies: at the interface between cognition and neurology. *Cognition* **50**, 133–149 (1994).
68. Rosen, G. D., Press, D. M., Sherman, G. F. & Galaburda, A. M. The development of induced cerebrocortical microgyria in the rat. *J. Neuropathol. Exp. Neurol.* **51**, 601–611 (1992).
69. Rosen, G. D., Sherman, G. F., Richman, J. M., Stone, L. V. & Galaburda, A. M. Induction of molecular layer ectopias by puncture wounds in newborn rats and mice. *Dev. Brain Res.* **67**, 285–291 (1992).
70. Galaburda, A. M., Menard, M. T., Rosen, G. D. & Livingstone, M. S. Evidence for aberrant auditory anatomy in developmental dyslexia. *Proc. Natl Acad. Sci. USA* **91**, 8010–8013 (1994).
71. Livingstone, M. S., Rosen, G. D., Drislane, F. W. & Galaburda, A. M. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc. Natl Acad. Sci. USA* **88**, 7943–7947 (1991).
72. Barlow, H. B. Cerebral predictions. *Perception* **27**, 885–888 (1998).
73. MacKay, D. M. in *Automata Studies* (eds Shannon, C. E., McCarthy, J. & Ashby, W. R.) 235–251 (Princeton Univ., Princeton, New Jersey, 1956).
74. Feldman, D. Timing-based LTP and LTD at vertical inputs to layer II/III pyramidal cells in rat barrel cortex. *Neuron* **27**, 45–56 (2000).
75. Rao, R. P. N. & Sejnowski, T. Self-organizing neural systems based on predictive learning. *Phil. Trans. R. Soc. Lond. A* **361**, 1149–1175 (2003).
76. Sejnowski, T. The book of Hebb. *Neuron* **24**, 773–776 (1999).
77. Kuhl, P. K., Williams, K. A., Lacerda, F., Stevens, K. N. & Lindblom, B. Linguistic experience alters phonetic perception in infants by 6 months of age. *Science* **255**, 606–608 (1992).
78. Dorman, M. F., Cutting, J. E. & Raphael, L. J. Perception of temporal order in vowel sequences with and without formant transitions. *J. Exp. Psychol. Hum. Percept. Perform.* **104**, 121–129 (1975).
79. Montague, P. R. & Sejnowski, T. J. The predictive brain: temporal coincidence and temporal order in synaptic learning mechanisms. *Learn. Mem.* **1**, 1–33 (1994).
80. Markram, H., Lubke, J., Frotscher, M. & Sakmann, B. Regulation of synaptic efficacy by coincidence of post-synaptic APs and EPSPs. *Science* **275**, 213–215 (1997).
81. Bi, G. Q. & Poo, M. M. Synaptic modifications in cultured hippocampal neurons: dependence on spike timing, synaptic strength, and postsynaptic cell type. *J. Neurosci.* **18**, 10464–10472 (1998).
82. Levy, W. B. & Steward, O. Temporal contiguity requirements for long-term associative potentiation/depression in the hippocampus. *Neuroscience* **4**, 791–797 (1983).
83. Hari, R. & Renvall, H. Impaired processing of rapid stimulus sequences in dyslexia. *Trends Cogn. Sci.* **5**, 525–532 (2001).
84. Tallal, P. & Piercy, M. Defects of non-verbal auditory perception in children with developmental aphasia. *Nature* **241**, 468–469 (1973).
85. Johnston, R. B., Stark, R., Mellits, D. & Tallal, P. Neurological status of language-impaired and normal children. *Ann. Neurol.* **10**, 159–163 (1981).
86. Tallal, P., Stark, R. & Mellits, D. The relationship between auditory temporal analysis and receptive language development: evidence from studies of developmental language disorder. *Neuropsychologia* **23**, 527–534 (1985).
87. Curtiss, S., Katz, B. & Tallal, P. Delay vs deviance in the language acquisition of language impaired children. *J. Speech Hear.* **35**, 373–383 (1992).
88. Hagman, J. *et al.* Cerebral brain metabolism in adult dyslexic subjects assessed with positron emission tomography during performance of an auditory task. *Arch. Neurol.* **49**, 734–739 (1992).
89. Jernigan, T., Hesselink, J., Sowell, E. & Tallal, P. Cerebral structure on magnetic resonance imaging in language-impaired children. *Arch. Neurol.* **48**, 539–545 (1991).
90. Buzsaki, G. & Draguhn, A. Neuronal oscillations in cortical networks. *Science* **304**, 1926–1929 (2004).
91. Llinas, R. R. *et al.* Thalamocortical dysrhythmia: a neurological and neuropsychiatric syndrome characterized by magnetoencephalography. *Proc. Natl Acad. Sci. USA* **96**, 15222–15227 (1999).
92. Munk, M. H., Roelfsema, P. R., König, P., Engel, A. K. & Singer, W. Role of reticular activation in the modulation of intracortical synchronization. *Science* **272**, 271–274 (1996).
93. Kilgard, M. P. & Merzenich, M. M. Plasticity of temporal information processing in the primary auditory cortex. *Nature Neurosci.* **1**, 727–731 (1998).
94. Tallal, P. *et al.* Language comprehension in language-learning impaired children improved with acoustically modified speech. *Science* **271**, 81–84 (1996).
95. Merzenich, M. *et al.* Temporal processing deficits of language learning impaired children ameliorated by training. *Science* **271**, 77–81 (1996).
96. Wilson, S. M., Saygin, A. P., Sereno, M. I. & Iacoboni, M. Listening to speech activates motor areas involved in speech production. *Nature Neurosci.* **7**, 701–702 (2004).
97. Gillam, R. B. & Van Kleeck, A. Phonological awareness training and short-term working memory: clinical implications. *Topics Lang. Disord.* **17**, 72–81 (1996).
98. Ehri, L. C. *et al.* Phonemic awareness instruction helps children learn to read: evidence from the National Reading Panel's meta-analysis. *Reading Res. Q.* **36**, 250–287 (2001).
99. Hatcher, P. J., Hulme, C. & Snowling, M. J. Explicit phonological training combined with reading instruction helps young children at risk of reading failure. *J. Child Psychol. Psychiatry* **45**, 338–358 (2004).
100. Torgesen, J. K. Individual differences in response to early interventions in reading: the lingering problem of treatment registers. *Learn. Disabil. Res. Pract.* **15**, 55–64 (2000).
101. Wise, B. W., Ring, J. & Olson, R. K. Training phonological awareness with and without explicit attention to articulation. *J. Exp. Child Psychol.* **72**, 271–304 (1999).
102. Habib, M. *et al.* Phonological training in children with dyslexia using temporally modified speech: a three-step pilot investigation. *Int. J. Lang. Commun. Disord.* **37**, 289–308 (2002).
103. Poldrack, R. A. *et al.* Relations between the neural basis of dynamic auditory processing and phonological processing: evidence from fMRI. *J. Cogn. Neurosci.* **13**, 687–697 (2001).
104. Temple, E. *et al.* Neural deficits in children with dyslexia ameliorated by behavioral remediation: evidence from functional MRI. *Proc. Natl Acad. Sci. USA* **100**, 2860–2865 (2003).
105. Temple, E. *et al.* Disruption of the neural response to rapid acoustic stimuli in dyslexia: evidence from fMRI. *Proc. Natl Acad. Sci. USA* **97**, 13907–13912 (2000).
106. Shaywitz, B. A. *et al.* Development of left occipitotemporal systems for skilled reading in children after a phonologically-based intervention. *Biol. Psychiatry* **55**, 926–933 (2004).
107. Tremblay, K. L. & Kraus, N. Auditory training induces asymmetrical changes in cortical neural activity. *J. Speech Lang. Hear. Res.* **45**, 564–572 (2002).
108. Schopmeyer, B., Mellon, N., Dobaj, H., Grant, G. & Niparko, J. K. Use of Fast ForWord to enhance language development in children with cochlear implants. *Ann. Otol. Rhinol. Laryngol.* **109**, 95–98 (2000).
109. Battin, R. R. & Young, M. Use of Fast ForWord in remediation of central auditory processing disorders. *Audiol. Today* **12**, 2 (2000).
110. Dronkers, N. F. *et al.* Lesion site as a predictor of improvement after Fast ForWord treatment in adult aphasic patients. *Brain Lang.* **69**, 450–452 (1999).
111. Frisina, D. R. *et al.* in *Functional Neurobiology of Aging* (eds Hoff, P. R. & Mobbs, C. V.) 565–579 (Academic, San Diego, California, 2001).
112. Pichora-Fuller, M. K. Cognitive aging and auditory information processing. *Int. J. Audiol.* **42**, 26–32 (2003).
113. Salthouse, T. A. Aging and measures of processing speed. *Biol. Psychol.* **54**, 35–54 (2000).

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Competing interests statement

The author declares competing financial interests see [web version](#) for details.

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P. Tallal is co-founder of Scientific Learning Corporation, the company that developed Fast ForWord®.

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Paula Tallal received her bachelor degree from New York University and her Ph.D. from Cambridge University. She is a Board of Governors' Professor of Neuroscience at Rutgers University, Newark, co-director of the Center for Molecular and Behavioral Neuroscience, and co-founder of Scientific Learning Corporation. A cognitive neuroscientist and board-certified clinical psychologist, Tallal is an active participant in many scientific advisory boards and has served on government and national policy committees for both developmental language disorders and learning disabilities. She is an expert on the neurobiological basis of language development and disorders, and an innovator of novel theories pertaining to brain development and disorders, as well as practical solutions to these disorders. Tallal has been awarded over two dozen US patents and recently won the Thomas Alvin Edison Patent Prize for her work leading to the development of Fast ForWord.