

Review



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From temporal processing to developmental language disorders: mind the gap

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The 'rapid temporal processing' and the 'temporal sampling framework' hypotheses have been proposed to account for the deficits in language and literacy development seen in specific language impairment and dyslexia. This paper reviews these hypotheses and concludes that the proposed causal chains between the presumed auditory processing deficits and the observed behavioural manifestation of the disorders are vague and not well established empirically. Several problems and limitations are identified. Most data concern correlations between distantly related tasks, and there is considerable heterogeneity and variability in performance as well as concerns about reliability and validity. Little attention is paid to the distinction between ostensibly perceptual and metalinguistic tasks or between implicit and explicit modes of performance, yet measures are assumed to be pure indicators of underlying processes or representations. The possibility that diagnostic categories do not refer to causally and behaviourally homogeneous groups needs to be taken seriously, taking into account genetic and neurodevelopmental studies to construct multiple-risk models. To make progress in the field, cognitive models of each task must be specified, including performance domains that are predicted to be deficient versus intact, testing multiple indicators of latent constructs and demonstrating construct reliability and validity.

1. Introduction

Developmental disorders of language and literacy are often understood as unexplained failures rather than as the result of specific causes. Many attempts to address this theoretical gap have proposed single distal causal factors leading via mediating proximal causes to the behavioural manifestation of the disorders. In this review, I discuss two prominent theories of developmental language disorders that have an auditory temporal processing deficit at their core. Both theories attribute language problems to faulty phonological processing resulting from underlying non-verbal auditory deficits. My emphasis is on reading impairment, but language impairment is also mentioned as the literatures overlap. After a brief overview of the main empirical support for each theory, additional findings are shown to complicate the empirical situation. Theoretical and methodological problems and limitations of the two proposals are discussed next, focusing on the experimental tasks used, their reliability and validity, the definition of constructs and mechanisms, and the theoretical causal chain accounting for the disorders. I consider the implications of inhomogeneity in the affected populations and end by sketching a speculative neurodevelopmental framework for reinterpreting the empirical situation, moving away from the single-cause approach and instead pointing towards a multiple-risk model of complex and variable behavioural phenotypes.

2. Temporal theories of developmental language disorders: an overview

(a) Rapid temporal processing

In the early 1970s, a series of studies by Tallal & Piercy [1–4] found that children failing to develop language normally were also impaired in sequencing

and discriminating brief auditory stimuli (75 ms or less) when the stimuli were closely spaced in time (i.e. with inter-stimulus intervals under 200 ms). No differences from the control group were observed with longer stimuli, with brief stimuli temporally separated by longer intervals, or with visual stimuli. Synthesized consonant–vowel syllables with stop consonants, which are characterized by rapid transitions of spectral peaks, also posed problems for the language-impaired children, but not when the transitional elements were extended in time. No difficulties were observed with vowel–vowel syllables composed of steady-state vowels, even brief ones. These findings were ascribed to the rate of auditory processing that was hypothesized to be too slow to keep up with rapidly incoming information. The presumed sensory deficit was non-verbal in nature. Its consequences were thought to be particularly devastating for language development because it impeded the discrimination of certain speech sounds, specifically consonants with rapid transitional elements. Even though vowels and suprasegmental prosodic features were unaffected, inaccurate perception of some speech sounds was thought to hold back language development.

Within the next decade, the same tasks were used in studies of children with reading impairment [5,6] (see also [7] for more recent data). These children showed a similar but less severe pattern of difficulties than that observed with language-impaired children. Individual performance in auditory tasks was correlated with pseudoword reading. These findings were interpreted as stemming from a primary perceptual deficit affecting ‘the rate of information processing essential for encoding simultaneous or rapidly occurring successive events’ [8, p. 168]. This deficit was hypothesized to cause difficulty in analysing speech at the phonemic level, resulting in the poor phonemic segmentation and recoding skills observed in children with reading difficulties. By the mid-1990s, the view that children with developmental language disorders were impaired in their ability to process rapidly occurring auditory events was well established [9] (though by no means universally accepted; see review in [10]). Tallal *et al.* [11, p. 33] addressed the ‘physiological basis of disorders in phonological awareness and decoding’ and discussed proposals regarding this link. At the same time, an intervention programme targeting temporal processing was developed. Intensive training was hierarchically structured from low-level perception of simple tones through the comprehension of spoken passages [12,13]. As a critical component of this programme, a speech processing algorithm was devised to slow down speech and enhance rapid transitions [14]. This programme was subsequently commercialized as remediation for language-learning impairments [15] though its efficacy has been strongly contested [16,17].

(b) Temporal sampling framework

More recently, a very different auditory timing issue has been implicated in the development of reading skill, focusing on slower rates and primarily affecting speech rhythm and stress. A non-verbal auditory processing deficit remains at centre stage. A new set of hypotheses has been put forward to link this deficit to speech processing and phonological development, ultimately aiming to account for poor reading and spelling skills. Goswami and colleagues [18–23] have documented group differences between children and adults diagnosed with dyslexia and control groups of typical readers (see Thomson & Goswami [24] for more references). The differences

concern the ability to discriminate and categorize amplitude-modulated sounds that differ in the abruptness of the amplitude increase, termed ‘rise time’. The core deficit was initially related to the perception of syllables, affecting their segmentation into onsets and rimes [18,25]. It was later recast as a deficit in slow brain oscillations [26]. In the tasks used most frequently, children are briefly trained to associate different sounds with cartoon dinosaurs. Then, they are asked to identify the sound with the sharper beat (two-alternative forced choice, with a slow-rising standard) or the odd-one-out in triplets (AXB discrimination, with a fast-rising standard).

Recent research on children with dyslexia has extended the findings to other languages [27] and established concurrent relations of rise time perception with prosodic sensitivity, phonological awareness, reading and spelling [28], speech perception [29], musical metrical sensitivity [30] and phonological learning [24], as well as longitudinal relations with the perception of syllable stress [31] and reading skills [32]. Rise time perception has been linked to reading skill in low-IQ readers [33] and in children with specific language impairment (SLI) [34]. In sum, rise time discrimination (or ‘beat perception’ as it is often termed) has been demonstrated to be associated with phonological awareness and reading skills, much like rapid temporal processing was two decades ago. Proposals for interventions have also appeared, suggesting general musical [35,36] or specifically rhythmic [37] training.

3. Presumed links in a causal chain: what do the data show?

Both these theories account for the poor development of oral or written language skills by providing a cause of poor phonological processing skills via impaired speech processing (figure 1). The representation of speech sounds presumably hinges on accurate speech discrimination and categorization from birth through infancy and throughout language acquisition. Therefore, any auditory processing deficit that causes speech to be misperceived might cause poorly specified, inaccurate or otherwise inadequate phonological representations. As phonological processing skills operate on phonological representations, they too are limited by auditory deficits. Subsequently, along the chain, poor phonological skills are associated with reading and oral language. Differences between languages and writing systems may modulate these relations across the auditory and linguistic domains [38,39].

To establish the viability of either temporal auditory processing theory, one would have to make specific predictions arising from the overall framework at each link of the causal chain. These predictions include differences that should occur as well as lack of differences where the theory offers no basis for an effect. That is, to ensure specificity, relative failure in tasks requiring temporal processing (‘indicator tasks’ in figure 1) should be accompanied by relative success in similarly structured tasks that do not hinge on temporal processing (‘control tasks’). Global testable predictions may also be possible (e.g. given the importance of rise time for rhythm perception, it follows that musicians with dyslexia cannot exist).

Despite considerable research, the empirical findings do not permit clear conclusions. They remain plagued by inconsistency [40] as they were 10 years ago [41,42]. Although both theories are supported by evidence of associations between language or reading and the signature task of the theory, there are also

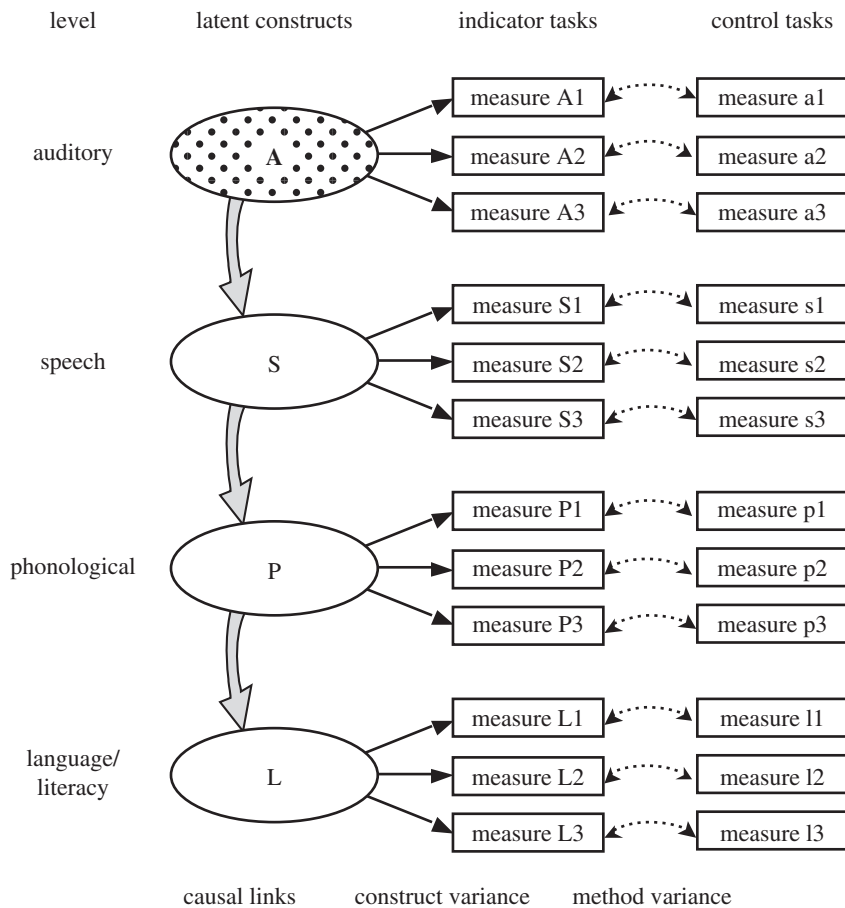


Figure 1. Simplified schematic diagram of hypothetical relations among theoretical and empirical constituents of the temporal processing approaches to developmental language disorders. Latent constructs are meant to be specifically defined by each particular theory. Constructs hypothesized to be causally related call for longitudinal evaluation. Constructs are defined by multiple indicators, i.e. tasks that require involvement or manipulation of the construct and therefore share variance with it, demonstrating convergent validity. Task variance unrelated to the construct is shared with other tasks using similar processes and methods. These control tasks assess skills not hypothesized to be involved in the theoretical causal chain; therefore they must not share variance with the latent constructs, demonstrating divergent validity. Large samples, well-defined constructs and multiple tasks boost reliability and help minimize sampling error and other sources of noise.

a host of findings that cannot fit within these frameworks. For example, there is evidence of impairments in other psychoacoustic tasks, such as duration, intensity or (especially) frequency discrimination, that do not require rapid processing or rise time perception [21,23,27,28,30–33, 43–50]. There are findings that fail to survive control for IQ [51] and failures to replicate (e.g. rapid processing: [41,52]; rise time: [39,53]). Reports of elevated backward masking thresholds in language-impaired children [54,55] have been contradicted by studies finding no differences from controls [56]. There are findings of children with developmental language disorders performing better than predicted on temporal processing tasks relative to grammatical [57] or phonological skills [58]. There are many inconsistent findings whereby one rise time task produced significant group differences but another rise time task, presumably equally dependent on beat perception, did not [22,28,34,45,50,59]. Importantly, children with auditory processing impairments do not necessarily have difficulties with language or reading [56,60].

Longitudinal data from infants at risk for developmental language disorders have shown deficits (or their brain signatures) that are linked to impairments in later years [61,62] but do not permit accurate prediction of future reading outcomes [63]. There are studies linking rapid processing [64–66] or beat detection [67] in pre-schoolers to future phonological awareness, as well as studies failing to find associations or producing unexpected findings [68]. There are also studies of infant and young children's brain responses to auditory

stimuli that are predictive of future language and literacy development but do not fit within temporal theories because their stimuli were neither rapid nor beat-related [63,69,70].

Several studies have examined the hypothesized deficits in the perception of speech sounds from birth on. Infant brain responses and later categorical perception of speech sounds have been linked to familial risk for dyslexia and reading outcomes [71–73] (see [74] for language outcomes). However, some of the predictive phonetic contrasts involved duration distinctions that require neither rapid processing nor rise time discrimination. Speech perception deficits have been reported for children with language impairment [75] and at least some children with dyslexia [76–78] or at risk for dyslexia [79], whereas others [55,80,81] have reported slight or no impairment. Crucially, the speech sounds on which differences have been reported range widely and, as for the infant studies, do not consistently require either rapid processing or rise time perception. Moreover, findings show little concordance between speech and non-speech distinctions that are based on similar acoustic cues [55].

Finally, both theories have garnered support from functional neuroimaging and neural oscillation studies (see [82–86] for rapid processing and [87,88] for temporal sampling). However, other recent neural oscillation data are inconsistent with temporal sampling at the syllable rate and call for a reinterpretation of data previously thought consistent with rapid processing [89,90].

Overall, and as remarked in previous reviews, the empirical landscape for theories of this type is fraught with inconsistency and contradiction [40–42,91]. To the extent that the two theories are seen as alternative contenders for the sole cause of developmental language disorders, then the data in support of one theory (as well as data in support of other sensory, motor or attentional theories not considered here; see Ramus & Ahissar [40] for references) constitute counterevidence for the other. As (i) the theories predict much more specific effects than typically observed, and (ii) the predicted effects do not emerge reliably and consistently in the relevant populations, none of the required links in a causal chain have been conclusively established. Although some of the discrepancies and inconsistencies in the literature might be ascribed to sampling error and noisy measures, the empirical picture is difficult to reconcile with either hypothesis when all of the available data are taken into account.

4. Theoretical and methodological issues

(a) On tasks and measures

Sensory theories rely on purportedly psychophysical tasks, to tap the hypothesized critical deficits, and on phonological awareness tasks, to document the mediated relationship from perception to reading through phonological representations. How satisfactory are these tasks? Although the association between phonological awareness and literacy development is well established (see Hulme & Snowling [92]), the precise nature of a phonological deficit as a causal theory of reading impairment remains vague—and is demonstrably false if taken to apply to the representation of the phonological identities of words or word parts: studies have failed to detect impaired or deficient phonological representations in tasks that require implicit use without explicit manipulation [80,93,94] or directly address phonological constraints [95]. The same observation applies to lexical stress: ‘stress impairments’ are observed in metalinguistic tasks involving explicit identification, comparison or manipulation of abstract stress patterns [28,31,47]. By contrast, studies that require participants to perform normal linguistic functions that implicitly rely on correct perception of stress fail to detect impairments [96–99] (but see Marshall *et al.* [100]).

Thus, ‘phonology’ is too crude a construct to permit theoretical progress. It must be properly dissected into representational and processing components that are involved in specific tasks, paying attention to the distinction between linguistic use and metalinguistic manipulation. Notwithstanding deficits in complex metalinguistic tasks, future studies might confirm that there is no deficit in phonological representations *per se* in those with language or literacy problems. In that case, the causal chain underpinning temporal processing theories would be fatally undermined, because these theories posit a mediation link from audition to reading via phonological representations. It seems of critical importance, then, to establish whether such representations are indeed deficient and to indicate the specific nature and extent of the deficiency.

Similar concerns apply to the psychoacoustic tasks. Obviously, rapid information processing is necessary to perform a two-tone repetition task with brief stimuli and intervals. Likewise, rise time perception is necessary to perform a rise time discrimination or identification task. Importantly, however, these tasks are complex and require more than perception of

the critical acoustic cues. The repetition task is particularly vulnerable to this type of criticism as it involves several cognitive components. Most psychoacoustic tasks are also laden with task demands other than sensory registration [101–103] (see Ramus & Ahissar [40] for a similar argument). For example, the requirement for explicit judgements introduces potential interference from auditory memory or even verbal processes that may be recruited to label the stimuli. In forced-choice tasks, decision criteria may be largely circumvented using signal detection techniques, but this does not mean that the same kind of sensitivity is measured across participants.

Anyone who has taken part in psychoacoustic experiments has experienced the progressive transformation of the task as the sounds approach threshold. The subjective impression on the basis of which judgements are made far from threshold is different from that near threshold. The ability to do well in such tasks is related to concentrating on the task and following the ever-more-subtle differences in the sounds through the sessions. To ensure reliability, in actual psychophysical studies, trained participants go through several sessions before their thresholds are measured. This familiarization involves some perceptual and procedural learning. By contrast, studies of individual differences in clinical populations or in the general population typically administer brief versions of psychoacoustic tasks with little or no training and few or no repetitions. Task reliability is rarely assessed (and may be too low, especially for the rise time tasks [53]). The resulting threshold is a measure of first impressions of naive participants [46,104] with different abilities in focusing on the task, identifying the critical acoustic elements and, crucially, adaptively tracking the progression of those elements towards threshold. It is unknown whether individual differences in concentration or perceptual learning ability contribute to the measured thresholds. However, it is clear that the extent to which repeated administration of auditory tasks leads to threshold improvement across sessions varies greatly among children with developmental language disorders [105]. This is an issue that warrants further scrutiny as it affects how findings from empirical studies should be interpreted. In the meantime, we may conclude that the flagship tasks of temporal theories are of at best unknown reliability. This is unfortunate as theoretical progress hinges on individually reliable processing deficits.

(b) From measures to constructs

To progress theory, we should be paying more attention to both measurement issues and cognitive mechanisms. Theoretical terms refer to unobservable latent constructs. Observable performance on specific tasks may partially index the construct to the extent the tasks recruit processes and representations that overlap with the construct. Well-defined constructs have precise roles to play within the context of a theory and must be clearly related to specific indicator tasks used to assess them. As illustrated in figure 1, multiple indicators are necessary to define any latent construct, abstracting away from task-specific features. In addition to convergent validity among putative indicator tasks, divergent validity must also be demonstrated. That is, tasks that are theoretically unrelated to the construct under consideration must not be linked to it once method variance is controlled.

Current theories of developmental language disorders remain underspecified. They posit associations between certain task outcomes but they fail to define specific constructs

linking putative underlying deficits to observed task performance. For example, we may speak of ‘phonology’ in the sense of an abstract concept, but, in practice, we equate this to a quantitative measure assessed by a single task that requires some sort of phonological operation. Little attention is paid to how the task is carried out, by what cognitive processes operating on what representations, in what order and under what constraints. Or we speak of ‘orthographic processing’ as if it were a single quantitative trait that can be equivalently assessed by any task involving letter strings. In this context, it is hardly remarkable that rapid processing has turned into a construct assessed with a ‘repetition test’ or that temporal sampling is quantified with a ‘beat detection’ task. Much more work will be required for the separation of theoretical and empirical components, and the comprehensive cognitive specification of tasks and measures.

The conflation of underlying skills with the tasks that are used as measures obscures important distinctions. To illustrate, consider why some tasks are more effective than others in bringing out differences. For example, in all but the least transparent orthographies, oral reading fluency is the single most useful task for the reliable identification of individuals with reading difficulties. It also exhibits remarkably high long-term longitudinal stability [106]. One might be tempted to conclude that fluency is such an effective task because it is a ‘pure’ measure of some critical domain. I submit that the opposite is the case, namely that fluency is so useful because it is an extremely complicated task with simultaneous demands on every component of the reading apparatus. In fluently reading aloud, each individual word is articulated while the next one is processed through the pipeline and another up ahead is fixated and its letters visually perceived. Coordination and integration demands are considerable; even minor weaknesses may be exacerbated by any vulnerability or inefficiency. Similar considerations apply for rapid serial naming tasks (termed ‘rapid automatized naming’ or RAN), which are strongly related to reading both concurrently and longitudinally [107,108]. The question ‘what does RAN measure?’ may be misguided insofar as RAN does not measure one single skill but, rather, is a complicated task requiring efficient coordination of multiple demanding processes.

These concerns also apply to psychophysical tasks. It is possible that the signature tasks heralded among proponents of sensory theories for developmental language disorders may turn out to make complex demands on the sensory-cognitive system in such a way that multiple weaknesses interact and become mutually exposed. Far from being detrimental to the temporal theories, such developments might lead to identification of specific areas of vulnerability. This approach may also explain why psychophysical tasks have proved less reliable than literacy tasks and have produced smaller effect sizes: Perhaps it is because they are less complicated, and therefore do not provide the opportunity of compounding weaknesses beyond the ability of the system to cope or compensate.

(c) From constructs to theories: the need for mechanisms

Figure 1 displays the structure of a set of constructs and measures that can be used, when fully specified, to examine associations and dissociations within an individual differences framework. This is still very far from a cognitive theory of

developmental deficits. To achieve that, one would need to define mechanisms underlying the constructs and measures, including specific representations and processes, as well as the precise locus and nature of impairment. Computational models might be the way to go, as they force full specification and commitment to implementation (see Mirman & Britt [109]). At present, available theories are grossly underspecified, and the tasks so poorly understood that it is unclear what is hypothesized, whether it concerns processes or representations and what the causal links are supposed to be.

Researchers often claim that they aim to identify brain mechanisms [26,110,111] underlying the observed perceptual and cognitive skills. Even though this may be premature and undoubtedly complicates the theoretical and empirical situation, it may be necessary, because only variation in brain development can ultimately explain variation in behaviour and performance. However, this does not free us from cognitive modelling. On the contrary, we will have to model the effects of differences in neuronal connectivity and functionality on the cognitive operations carried out by the affected networks. How else could we link individual differences at the level of neuronal networks to behavioural task performance and, eventually, to the diagnosis of language disorders?

The specification of cognitive and brain mechanisms to account for particular aspects of the data brings in the benefits of proper construct definition and the highly desirable peril of falsifiability. For example, a proposed link between auditory processing and literacy development has implicated neural oscillation entrainment to multiple timescales [26,89,90,112]. Although still vague, this approach connects with recent ideas about the implementation of speech perception in the brain [113,114]. Such speculation might lead to predictions regarding the development of phonological skills. The two temporal theories considered here need not be competitors but may concern complementary aspects of speech processing, acting on different timescales, independently or in unison (see evidence in [115] and discussion in [116]). Alternatively, neural entrainment may only be incidentally and not causally associated with language or literacy difficulties [46], by virtue of some shared physiological substrate. It may turn out that impairments in speech processing are not consistently found in individuals with language or literacy disorders. These theoretically provocative prospects are worth exploring, because the specification of a particular relationship to brain mechanisms of speech perception offers the possibility to test the temporal theories by forcing them to commit to specific predictions.

(d) Quantitative and qualitative individual variability

In her seminal article relating temporal processing to reading difficulties, Tallal [5] noted the substantial variability in individual performance observed in the experimental group, with 12 of 20 language-impaired children performing within the range of the control group in the auditory tasks. Despite the statistically significant group difference (indicating a difference in means, not lack of overlap), Tallal entertained the hypothesis that there might be distinct subgroups of children with reading difficulties and warned against treating reading-impaired populations as homogeneous. Indeed, substantial individual variability is a typical outcome of measuring any skill in children with reading difficulties. Invariably, a majority of children in the experimental group will perform within the range of the

control group, thus exhibiting by definition ‘unimpaired’ performance [42,117,118]. This pattern has been repeatedly observed [81,119] in studies exploring sensory and motor skills thought to be related to reading difficulties. Rise time tasks are no exception, as clearly demonstrated in a meta-analysis [91].

It is important to embrace this individual variability in the-oring about potential sensory causes of reading difficulties. Variability in routes to failure [120–122] is not surprising, because the multiple genetic and environmental factors underpinning language and literacy development, combined with the complex demands of reading and language, lead to multiple points of varying vulnerability across individuals. Note that variability does not imply distinct subtypes of dyslexia or language impairment: graded functional differences in a multitude of components can produce similar outcomes in processes requiring efficient integration of the components. Underlying causes need not form distinct clusters but may lie along continua, each, in turn, affected by a variety of genetic and environmental factors [123,124].

5. From genes to brain to behaviour

Theoretical accounts aiming to shed light on language and especially literacy problems are bounded by conflicting requirements. On the one hand, we need to account for clear and sizeable effects in literacy skills, which require clear and effective causes. On the other hand, there is no obvious anomaly or abnormality in these children outside literacy. They do not exhibit signs of aberrant development and their brains are not malformed or malfunctioning. Although there is some evidence of unusual grey matter formations, it concerns a very small sample that might not have been diagnosed with dyslexia today [125–127]. Even if replicated, these outdated findings cannot offer a solution as they are a far cry from the required clear-cut cause.

Genetic studies also point to a conundrum. On the one hand, behavioural genetics has consistently come up with heritability estimates exceeding 0.50, increasing with age, and occasionally approaching dizzying values around 0.80 for literacy skills such as fluency, decoding, spelling and comprehension, and for the diagnosis of reading or other disabilities [128–133] (but no comparable heritability for performance on the repetition test [134]). On the other hand, molecular genetics have failed to identify individual genes as singularly culpable for a diagnosis of dyslexia [112,126,127,135,136]. Instead, several genes are involved as contributing or ‘susceptibility’ factors in a multi-factorial and heterogeneous causal progression rather than as isolated causes of specific deficits [137,138]. The causal pathway from genes to brain and behaviour is unclear: non-human animal studies have linked these genes to neuronal migration and connectivity in brain development but their effects lie outside the range observed in humans [127], so their relevance remains to be established.

In sum, it seems that a multitude of minor deviations in neuronal networks, with no obvious gross anatomical or functional manifestation, causing unremarkable individual variation largely within the normal range, result in brain function that is somehow remarkably unsuited to learning to read. This realization is important because it affects the target of research: we cannot be searching for a specific cause of a specific condition if there is no specific condition. We are looking at the complexity of genetic and environmental interplay

leading to variety in brain development such that some outcomes lend themselves to effortless and efficient acquisition of literacy and others do not. The differences are likely to be miniscule, the causes many and each of negligible influence. The resulting effect on learning to read becomes so large perhaps because of compounded disadvantages rather than single identifiable factors.

This line of thinking brings out a major weakness in correlational research and highlights the need for specifically causal theories and targeted studies aimed at falsification rather than at confirmation. Consider minor variation in neuronal development, migration, connectivity, and so on, that underlies differences in brain structure with functional implications. To the extent that gene expressions are not restricted to single narrowly defined cortical areas or developmental time windows, such differences are likely to be diffuse, affecting multiple brain regions at various times. That is, whatever factors lead to brains unsuited for reading also cause additional variability that may not be involved in learning to read. It is almost inevitable that a variety of processes would be affected to some extent. This would lead to statistically significant, detectable differences in tasks not directly related to reading. Because of the developmental causes of the brain differences that make reading difficult, performance in many unrelated tasks would be associated with reading difficulties without having any causal or even contributing role in the reading process. And because of the multitude of causal factors contributing in various combinations and proportions to making reading difficult, different sets of tasks unrelated to reading might be collaterally affected in different individuals. If the reading difficulties are somehow owing to differences in brain wiring affecting the superior and lateral temporal cortex then we might also expect to see concomitant effects in auditory processing, which is also largely accomplished there. This does not mean that differences in auditory processing are directly related to differences in learning to read. It only signifies that both are affected by the function of the same or nearby cortical structures, perhaps in different subregions or in different ways. This argument shows that associations between tasks are moot with respect to specific causal theories regarding developmental disorders.

Moreover, the complexities of reading must be taken into account. We do not have adequate cognitive theories of the reading processes, let alone neuroscientific theories of the brain functions that implement those processes. Reading is not a skill humans have evolved to perform. This may go some way towards explaining why so many of us have difficulty with it, but the crucial point is that a number of pre-existing structures must be recruited and reassigned, functions appropriated and processes adapted [139,140]. From visual processing of letter arrays to fluent lexical access, phonological and semantic activation, and possibly articulation, it is clear that fluent reading requires not only coordination but also deep integration of a multitude of processes across brain regions and skill domains (see Woollams [141]). This brings an additional level of variability and complexity in the potential for failure. On the one hand, multiple genes at various times are implicated in the production of neuronal outcomes that might affect some component of the reading process. On the other hand, there are many ways in which the functional integration required for fluent reading might fail, because there are so many parts to it

and so many demands for flawless coordination. The complexity of the reading 'system', or even the potential for alternative reading systems to develop in response to written language exposure and instruction, implies that different people might struggle with reading for different reasons—whether distinctly different or lying along continua. If this is the case, then it is futile to search for a universal cause of reading problems. Instead, our efforts should be directed towards understanding the reading process and its neuronal instantiation well enough to be able to identify individual paths to failure. At least, we should be able to document the extent of heterogeneity at each of the various levels in a causal chain, from the neuronal through the cognitive and up to reading and spelling performance.

6. Routes to progress

There is no question that low performance in a variety of psychophysical tasks is associated with developmental language disorders when examined at the group level. Although part of the deficit may concern temporal processing, timing has not emerged as a prominent domain of weakness. The associations, which only hold for a minority of children, may be causally related to the difficulties in language and literacy, although clear evidence for this is lacking. Alternatively, they may reflect distinct and unrelated consequences of common physiological causes that originate in brain development, influenced by complex interactions of multiple genetic and environmental factors. Thus, correlations between distantly related tasks are no longer theoretically helpful. We know there are correlations, perhaps not very large or very tidy, but systematic enough to be interesting. We need to understand why these correlations exist. We need diverse and structured sets of tasks to understand associations and dissociations. We need longitudinal studies to understand causes, and models of individual differences to understand mechanisms and routes to success and failure.

Perhaps the thorniest issue in establishing a causal relationship between sensory processing and language deficits is that the phonological deficits theorized to mediate between the two are poorly specified. There is a wide theoretical gap between auditory processing and reading, as the putative theoretical links between them are not stated in sufficiently concrete and empirically testable terms. There is little (and inconsistent) empirical support for the notion that 'phonological deficits' concern the acoustic representations of phonemes: speech sound processing is hardly established to be deficient even in the children with low auditory processing performance. Moreover, deficits in phonological

awareness are common in children with reading difficulties, but the main problems seem to concern the metalinguistic level of performance and not the representation of speech units. The elephant still remains in the room: exactly how are problems in phonological awareness causally related to learning to read and why is poor phonological awareness an impediment to fluent reading and spelling?

It seems to me that the war that has been waged among proponents of sensory theories for developmental language disorders may be quixotic. It appears increasingly likely that SLI and dyslexia are not unitary, homogeneous conditions but, rather, multi-factorial expressions of complex developmental routes that share the common property of being unsuited to learning oral or written language efficiently. Thus, the holy grail of dyslexia (and perhaps SLI) research, namely the definition of the disorder itself, may prove as elusive as the grail of religious lore. In the absence of a single condition and therefore of the need to identify a single cause for it, there is no reason to consider alternative theories as competitive contenders. Perhaps they are complementary: both theories may apply to the same brains simultaneously, producing a double jeopardy that blocks compensatory routes and results in developmental language disorders. Or they may concern different subgroups of children to different extents, a possibility that can only be discerned if researchers apply wide testing batteries and focus on individual profiles of performance and development alongside theoretically predicted patterns of successful and impaired performance.

Finally, it is of primary importance to establish the reliability and validity of the measures applied, from neural oscillations and psychoacoustics through speech perception to indices of phonological components. Hypothesized latent constructs must be assessed by multiple indicators and must be empirically demonstrated to be properly defined by them. In addition, we need specification of the cognitive mechanisms involved in each task. It is hard to see how a theory of impairment can be specified in the absence of a well-defined theory of unimpaired performance. The ultimate goal is to specify every link in the chain from genetic and neural bases through sensory processing, the development of speech perception, phonological representations and processes, metalinguistic skills and the development of language and literacy.

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