



Observations on the temporal correlates of reading failure

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Abstract. The definitions of developmental dyslexia and temporal processing are discussed and several construals of what a temporal processing deficit might entail are illustrated. Then, using a framework developed by Farmer & Klein (1995), the proposal that a temporal processing deficit (in vision, audition, or both) might be the root cause of some proportion of cases of developmental dyslexia is introduced and various research strategies for testing this proposal are identified. The symposium papers which address this general question using a range of these strategies are then critically reviewed. It is noted that whereas reading is a recent artifact not yet subject to any direct selection pressure, its normal performance requires the use of, and connections between, the highly evolved modules for visual pattern recognition and language. Within vision and language redundant coding, which permits performance in the face of injuries and degraded input, also makes it difficult to detect subtle deficits (whether temporal, or not) unless precise tests are used. Yet such deficits may degrade performance on tasks (such as reading) which require the non-copious connections between vision and language. The efficacy of this pathway, I suggest, is measured by performance on the rapid automatized naming (RAN) test.

Keywords: Dyslexia, Modules, Rapid automatized naming (RAN), Reading, Temporal processing deficit

The core issues that interest reading researchers and practitioners can be captured in two questions: How do people read and why do some people fail to read well? The symposium upon which this special issue is based was devoted to exploring the more specific issue of the role of temporal processing in reading and in failures to acquire the reading competence necessary for success in the modern world. To lay the groundwork for my discussion of the papers presented at the symposium I will first briefly introduce the key topics of dyslexia, temporal processing, and the temporal processing deficit hypothesis.

Developmental dyslexia(s)

Unexpected reading failure is perhaps the most commonly used definition of dyslexia. This is captured in the World Federation of Neurology's definition which requires that the dyslexic should have no sensory impairment

(e.g., they should be neither blind nor deaf!), no cognitive impairment (e.g., normal IQ, no language impairment), no emotional disturbance, no educational deprivation and no gross acquired neurological impairment. Suppose you were considering a language impaired child with poor reading ability. According to this definition such a child would not be considered dyslexic because their reading problem would not be unexpected. To make this point using an example about which there would be little disagreement, we would not use the term dyslexia to refer to reading difficulties associated with deafness or blindness. Another example: According to this definition the young student who can't read because they are educationally deprived is not dyslexic, they are illiterate. It is sad to note (as Richard Olson did in the discussion), however, that one might be a poor reader because of the low quality of training whose quantity might otherwise have been adequate.

Interpreted strictly, the exclusive definition of dyslexia as an 'unexpected' reading failure points rather narrowly to a possible locus for the deficit: a cognitive system (or systems) needed for reading and nothing else. The acquired module that computes grapheme-phoneme correspondences is just such a system. Practically speaking, however, such a strict interpretation is not particularly useful because it is clear that children who have problems learning to read present with an array of difficulties in the component processes required for, or associated with, reading (e.g., see Boder 1971; Castles & Coltheart 1993) and thus their difficulty may be rooted in different causes.

In any event, scientists and scholars of reading do not agree on a definition of dyslexia. It is not essential that they do agree. Indeed, maybe it is not even desirable, because a universally adopted definition might marginalize some groups of poor readers. What is essential, however, and what we have learned from this symposium, is that we must provide the definition we are using.

Temporal processing deficit

Temporal processing refers to the extraction of information presented over time or the production of a sequence of behaviors. Focusing on the encoding dimension, Farmer & Klein (1995) described a hierarchy of information processing achievements that were pertinent to temporal processing. At the lowest level, and in a way a prerequisite for all the remaining ones, is that the target stimuli are detected and, when necessary, identified. In almost none of the studies we reviewed was it shown that dyslexics required a longer or more intense stimulus to achieve detection or identification of non-speech stimuli. The next most simple achievement is individuation. When two identical stimuli are presented in rapid succession in the same location, how much

time is required between them for the observer to correctly discern that two presentations rather than one has occurred? In both the visual and auditory modality, dyslexics have been shown to require a longer ISI to achieve stimulus individuation. Alternatively, one can describe this finding as showing that the minimal detectable gap separating two otherwise identical stimuli is larger for dyslexics than for normal readers. With identification and individuation established, it is possible to briefly present two different stimuli while varying the interval between them and ask the observer to report their order. In the performance of this elementary temporal order judgment (TOJ) task, our review revealed, in agreement with the findings of Tallal (1980), a consistent deficit, in both the auditory and visual modalities, in dyslexics. Finally there are more complex sequencing tasks requiring perception and production of sequences of more than 2 events. There are many studies in both modalities showing that dyslexics are worse at such tasks than controls, but the results are not as consistent as with individuation and sequencing judgments (TOJs) with just two events.

Merely stating that dyslexics have a temporal processing deficit on such broadly defined tasks does not tell us much about the nature of the deficit. Klein & Farmer (1995) described three construals of 'temporal processing deficit' which are illustrated in Figure 1.

One construal (Figure 1A) is that there is a problem with the rate of information processing (this is Tallal's construal). Under this view when events or stimuli are presented at some, relatively slow, rates, there will be no problem; however, when the rate of input is increased, a bottleneck is achieved sooner for individuals with a temporal processing deficit of the 'throughput' variety. At rates which are too fast in relation to one's throughput ability individual elements may be misordered or even lost. A second construal (Figure 1B) is that there is temporal smudging and that individuals differ in the degree to which stimuli are smudged over time. Finally (Figure 1C), there may be 'jitter' in the assignment of events to points in the temporal stream and individuals may differ in the amount of this jitter. This is loosely analogous to Treisman's proposal (Treisman & Gelade 1980) that visual attributes are not well attached to their correct locations (unless attended) except that we have replaced locations in space with moments in time. Under each of these construals, all other things being equal, performance will degrade as the interval between stimulus onsets decreases, and the degradation should be seen sooner (at larger intervals) for individuals with a temporal processing deficit.

The caveat 'all other things being equal' is an important one, because it is a condition that is rarely achieved. Psychologists can measure performance but we can only infer the operation of stages of processing. And when we mani-

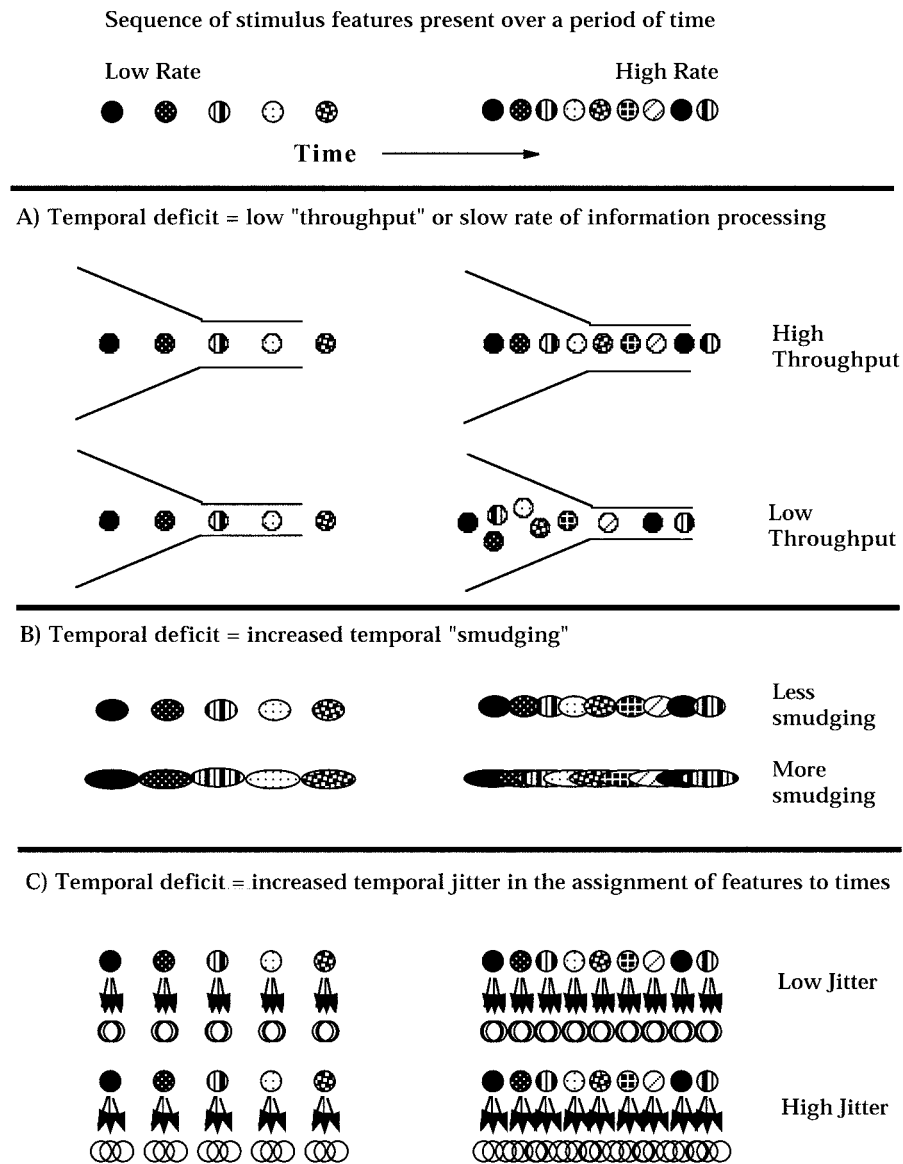


Figure 1. A sequence of events with distinct features is illustrated at a slow (left) or fast (right) presentation rate at the top of the figure. In the three panels below three construals of a temporal processing deficit are illustrated. Normal processing is illustrated at the top portion of each panel; the temporal processing deficit is illustrated in the bottom portion. Throughout, slow sequences are shown on the left and fast ones on the right. (A) Temporal processing deficit is related to throughput or rate of information processing. (B) Temporal processing deficit involves increased smudging of signals over time. (C) Temporal processing deficit involves increased jitter in the assignment of features to moments in time.

pulate a variable, such as the interval between stimuli, and even when we are correct in our assumption that this may affect a particular mental operation, it is still possible that the manipulation may have other unanticipated effects that may also impact performance of a task. To make this point concrete, consider that we present first a 50 ms stimulus to the left or right of fixation, then an identical one to the other side and that we vary the interval between these two stimuli while asking the observer which was presented first – a simple TOJ task. In this case, an emergent property – motion – may confound our pure manipulation of the time between events. As we shorten the interval, possibly approaching a point where the TOJ may become difficult, the motion processing system begins to be activated by the temporal difference and the observer will see not two separate stimuli, but rather one stimulus moving through space from the first stimulated location to the second one. Even if order information is not explicitly available, it can now be inferred from the direction of motion experienced.

Linking a temporal processing deficit to dyslexia

In 1995 Farmer & I (Farmer & Klein 1995) reviewed the literature revealing that many dyslexics have difficulties performing a wide range of temporal processing tasks. The association was strong enough that we suggested it would be worthwhile for scholars to explore whether there was a causal link. The present Symposium and this special issue conform to this suggestion. We also described how either a visual or an auditory temporal processing deficit might lead to reading problems and why the nature of these problems would depend on which modality was temporally deficient, and we raised the possibility that temporal deficits in different modalities might co-occur in some individuals (Figure 2). We viewed the causal pathways we delineated as plausible, but didn't assert that there was a causal link from temporal processing deficits to dyslexia (hereafter I will abbreviate this idea, the temporal processing deficit hypothesis, as TPDH). Rather we proposed a variety of methods that could be used to determine if there were one, some of which were used by presenters at this meeting. It is important to note that even if there were a causal link from, say, an auditory temporal processing deficit to the core phonemic deficit and hence to dyslexia, this would not exclude the possibility of other non-temporal causes of a phonemic deficit.

In considering the plausibility of the causal link from auditory temporal processing to a problem with phonemic processing, Klein & Farmer (1995) emphasized the following line of reasoning. Audition provides the inputs to the information processing stages which yield the phonemic categories that are so important to speech perception. Hence, if there is damage to

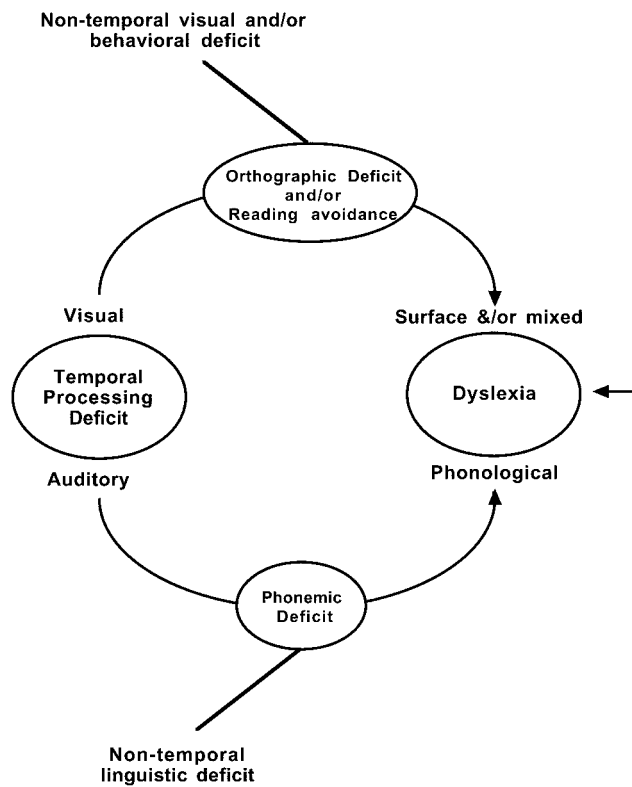


Figure 2. Flow chart illustrating hypothetical causal pathways leading to dyslexia from visual and auditory temporal processing deficits (adapted from Farmer & Klein 1995, Figure 1).

the early auditory pathway the quality of the phonemic representations extracted from its degraded signals will be correspondingly degraded and a language problem with its roots in speech perception and/or production will be observed.

Among the strategies we suggested for assessing the causal link were:

1. multivariate analytic methods (which were very well illustrated at this meeting);
2. anatomical and electrophysiological data (although none of the participants presented any neuroimaging data, Breznitz presented ERP data that is very pertinent);
3. behavioral genetic approach (which has been expertly exploited by Olson and colleagues in this issue; see also, Olson, Datta, Gayan & DeFries 1999);
4. cross-cultural/cross-linguistic (For example, different languages have different mapping systems from orthography to phonology and meaning,

and this provides a powerful tool for addressing questions about the nature of the encoding operations used in reading. Although none of the special issue studies used this methodology it was briefly reviewed in M. Wolf et al. (2002, this issue);

5. remediation and educational efficacy – Although this approach was not directly employed, it was implicit in some special issue (M. Wolf et al. and P. Wolff, 2002, this issue) papers and has been emphasized in the work of Lovett (1999), Levy (1999), and Tallal, Miller, Bedi, Byma, Wang, Nagarajan, Schreiner, Jenkins & Merzenich (1996);
6. longitudinal approach (which was exploited by David Share and colleagues).

Reflections on the symposium studies

All of the studies which have been presented in this symposium required their subjects to perform one or more information processing tasks. In every task we present some information to the observer and we require some output from them. The inputs may be visual, auditory, tactile or some combination. Responses may be verbal, manual, discrete or continuous. Sometimes the inputs, outputs and translation rules (task) are very complex; sometimes very simple. To understand how performance on a particular task may be related to reading ability we need to consider what mental operations or stages of processing may underlie performance differences that are correlated with reading ability. Therefore, in my discussion of some studies, I will include a brief task analysis (see Figure 3), asking: What are the inputs, what are the output requirements, and what mental representations and operations might be required to perform the task?

Rapid Automatized Naming (RAN)

As a group dyslexic readers are well known to be slower than normal readers on the Rapid Automatized Naming (RAN) task originally developed by Denckla & Rudel (1974). In this task, featured in the research of Patricia Bowers (Bowers & Newby-Clark, this issue) and Maryanne Wolf (M. Wolf et al., this issue), the input is an extrinsic (experimenter produced) visual stimulus (object, color patch, alphanumeric character) and the output is a speech act which, not incidentally, generates intrinsic feedback in the form of hearing one's own vocal response. Correct performance of the task requires visual pattern recognition, memory retrieval of the correct name of the stimulus, and pronunciation of the name (which, itself, can be decomposed further into more elementary mental operations).

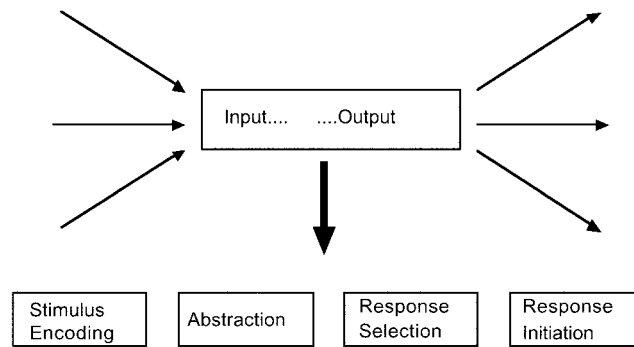


Figure 3. A typical task is schematized in the top of this figure where varied inputs (e.g., auditory, visual, tactile) may, after internal mental operations (illustrated at the bottom), lead to varied outputs (manual, verbal, etc.).

One question that naturally arises from a task analysis is what stage or stages of performance are slower in dyslexics thus causing their ubiquitous disability in this task. Maryanne was quite excited by Peter Wolff's demonstration (see below) that dyslexics are deficient in rhythm production and make unusually long pauses when talking, and she thought that this might map onto the RAN deficit. And if it did, this would probably mean that the deficit is more on the output (response selection, initiation and/or execution) end of the continuum. Patricia Bower's finding of a deficit in the ability of dyslexics to encode four identities when given only a 250 msec exposure duration as well as her finding of a significant correlation between continuous and discrete RAN, suggest to me that the deficit is more on the input side. To be sure, however, cognitive studies aimed at this issue would be useful.

Several findings merit consideration. One of these is that performance on the RAN predicts reading ability even when the contribution of phonological processing ability has been removed. So whereas there is shared variance, it is also the case that the two deficits, RAN and phonological processing, can occur separately. If these deficits occur independently in the population, then the probability of someone having both deficits should be the product of the incidence rates of the two deficits. Pertinent data from three studies is shown in Table 1.

There is good agreement between the observed incidence rate of each deficit and the rate predicted on the basis of independence of the two deficits in the Wolf and Lovett (Lovett, Steinbach & Frijters 2000) studies, where the population was restricted to individuals with reading difficulties. In the third study, by Bowers, there appears to be a discrepancy between the observed

Table 1. Incidence rates of phonological and RAN deficits in three studies. Predicted rates of their co-occurrence based on independence and the observed rate of co-occurrence

Relative incidence of	Wolf (dysl only)	Lovett (dysl only?)	Bowers (unselected sample)
Phono ^a	0.79	0.64	0.39
RAN ^a	0.75	0.65	0.34
Both:			
Expected	0.59	0.42	0.13
Actual	0.60	0.46	0.24

^aThe incidence rate of a deficit (e.g. phono) reported on these lines is the sum of the incidence rates of the deficit alone and the incidence rate of 'both' (as reported in the target studies). (Data from Wolf et al., this issue; Lovett et al., 2000, as cited in Wolf et al.; and Bowers & Newby-Clark, this issue).

and predicted rate of overlap. Unlike the other two studies which sampled only dyslexic students, Bowers' sample is unselected.

Wolf asserts that RAN is a better predictor of reading ability than is discrete naming because – like reading – it is sequential. Two properties of the RAN that may also be important in mediating its predictive power are that it requires selection, possibly via suppression, in the face of competition (from amongst the most recently activated codes) and coordination (rapid switching between encoding and output), both of which are also involved in reading.

Bowers makes the interesting proposal that the slow encoding of letters in individuals with RAN deficits prevents chunking and the consequent poor orthographic coding is the chief component of reading tapped by RAN. In support of this interpretation, Bowers describes her own data and that of Levy & Bourassa (1998) showing that the efficacy of training (print exposure and decoding practice) depends on RAN.

As noted above, Bowers' clever study showing that reading disabled children had difficulty identifying four letters presented in 250 msec appears to localize the deficit to input rather than output mechanisms. It would be interesting to know if this finding generalizes to four objects or colors? Or if it is specific to the operations needed to process alphanumeric? Also, what happens to performance (of both good and poor readers) when the duration of the 4-item array is increased beyond 250 msec – i.e., is the problem with working memory capacity (à la Jorm 1983) or with the rapidity of entry of name codes into STM. It is possible that the brief exposure of four items taps

Table 2. Latency of ERP components (P200 and P300) in dyslexic and control subjects in simple tasks using linguistic auditory (A) and visual (V) stimuli and in more complex tasks requiring homophonic (P) and homographic (G) processing of letter strings

	ERP component	Dyslexic			Control			Dyslexic-control	
		A	V	A-V	A	V	A-V	A	V
Simple linguistic	P200	286	238	49	241	205	35	45	33
	P300	518	405	112	352	327	25	166	78
		P	G	P-G	P	G	P-G	P	G
Letter strings	P200	298	252	46	193	223	30	105	29
	P300	548	402	146	386	350	36	162	52

The latency difference between modalities and tasks are shown in the columns labeled A-V and P-G. The latency difference between groups for each modality and task are shown in the columns labeled Dyslexic-control. All numbers are in msec.

Data from Breznitz (this issue).

two independent functions (and disabilities in poor readers): rapid extraction of identities and working memory capacity.

The presence of the term 'automatized' in RAN (rapid automatized naming) carries the implication that poor readers have difficulty with this task because they have difficulty automatizing the connections between names and the things named (Nicolson & Fawcett 1990). This may be the case, but merely saying it is doesn't make it so. Claims for automaticity, in my opinion, should be based not merely on speed of response but on converging operations which include the absence of dual task interference. It should also be kept in mind that the standard RAN involves colors and objects as well as digits and letters. As shown by Wolf, reading disabled children are poor with all types of stimuli. Although the deficit with alphanumeric is more strongly correlated with reading ability, it is possible that one reason for this is the contribution of print exposure to both reading ability and RAN performance. One way to test this possibility would be to see if print exposure is correlated with the difference between RAN for letters and digits minus RAN for colors and objects.

Asynchronous code extraction inferred from recordings of brain electrical activity

In Zvia Breznitz's study (this issue) visual and auditory stimuli varying in complexity, and letter strings requiring various judgments were presented to normal and mildly disabled readers (minimum of 1.66 years behind their cohorts). Her focus is not so much on performance, as on the relative time of

occurrence of different stages of processing or the achievement of different codes as indexed by averaged electrical responses recorded from the surface of the scalp (ERPs). Her major finding is a strong relationship between reading ability and the time difference in the appearance of ERP components (P200 and P300) evoked by visual and auditory signals or, correspondingly, orthographic and phonological tasks. As seen in Table 2 (which provides a few examples from Breznitz' study), the time difference in the appearance of these components is larger in dyslexics than in normal readers (see Bigsby 1985, for a similar demonstration with regard to the time difference to perform physical and name matches with pairs of visually presented letters). Breznitz's creative speculation is that reading problems are caused by an asynchrony between the timing of activation of orthographic and phonological codes during reading. However, as can be seen in the table, the larger differences in dyslexics are due more to slower auditory/phonological processing in dyslexics than to faster visual/orthographic processing in normal readers.

Thus, one can legitimately ask whether it is the temporal asynchrony per se that is critical (A-V column) or just the extra time dyslexics require to compute auditory and phonological representations (Dyslexic-control: Aud. and P column). That Breznitz finds a larger correlation between reading ability and the time difference between auditory and visual code activation's than she does between reading ability and the time of auditory code activation, would seem to argue against this alternative, but it might not for the following reason. The effect of subtracting a within-subject measure of processing speed from the auditory/phonological activation times may be to reduce noise and therefore increase amount of variance that can be accounted for by the critical variable. One way to test this proposal, and to provide direct support for or against Breznitz's asynchrony/coordination proposal would be to collect ERPs and RTs to stimuli whose codes have nothing to do with reading, say a tactile detection or discrimination task. If my proposal is correct that the critical variable is the speed of extraction of auditory/phonological codes and that the larger correlation when the A-V subtractions were used was due to noise reduction, then a similarly high correlation should be observed when Auditory-Tactile subtractions are used. In contrast, if Breznitz's proposal that the critical variable is the asynchronous extraction of auditory and visual codes, then subtracting tactile encoding time (estimated from RT or the timing of ERP components, should improve the correlation with reading ability significantly less than should subtracting visual encoding time).

The visual transient system in dyslexics: Heritability and efficacy

Richard Olson has been involved in a large scale, twin-based, behavioral genetics research program in Colorado (Olson et al. 1999). Recently Olson & Datta (2002, this issue) have added to this program a test battery designed to measure the efficacy of the visual sustained and transient systems. As described by David Share in his introductory remarks, William Lovegrove, in a series of studies dating back to 1980, has repeatedly demonstrated that severely reading disabled readers have deficits on visual tasks thought to assess the functioning of the transient system (e.g., Lovegrove, Martin & Slaguis 1986). As proposed by Breitmeyer (1989; for a discussion, see Farmer & Klein 1995: 483–485) a transient system deficit might interfere with the visual extraction of information during reading because as the eyes saccade to new regions of text the transient system is thought to be responsible for erasure of the visual persistence from the preceding fixation. A deficient transient system would entail ineffective and/or delayed erasure and hence, until full erasure is accomplished, impoverished visual signals.

Olson's purpose was twofold: first, to see if he could replicate Lovegrove's finding of a transient system deficit in many dyslexics, and second, to determine the degree to which hereditary vs. environmental factors were responsible for this deficit. In Olson's test battery the inputs were oscillating gratings of high and low spatial and temporal frequency, and the output was the subject adjusting the contrast until the oscillation was just barely visible. Based on Lovegrove's work and our understanding of the transient channel it was expected that dyslexics would show a selective deficit for low spatial frequency gratings oscillating at high temporal frequency. Instead, dyslexics showed a relatively uniform increase in contrast threshold on all 4 combinations, suggesting a deficit with the detection task in general, but not necessarily a transient system deficit. Comparison of twin and cotwin scores on a measure reflecting this overall deficit did not reveal any evidence of heritability. Although the ANOVA didn't reveal the selective deficit that would have been expected on the basis of Lovegrove's work, it would probably still be warranted, based on this expectation, to have assessed the heritability of performance on the one condition that by hypothesis should have selectively tapped the transient system (1cpd/10Hz).

How might Olson's non-replication of Lovegrove's findings be explained? The first place to look is for methodological differences. Olson discusses three: subject differences, task differences and luminance differences. Olson notes that the definition he used to classify subjects as dyslexic was not as stringent as Lovegrove's, and, therefore, Lovegrove's subjects were, as a group, much more disabled. To address this difference Olson sorted his

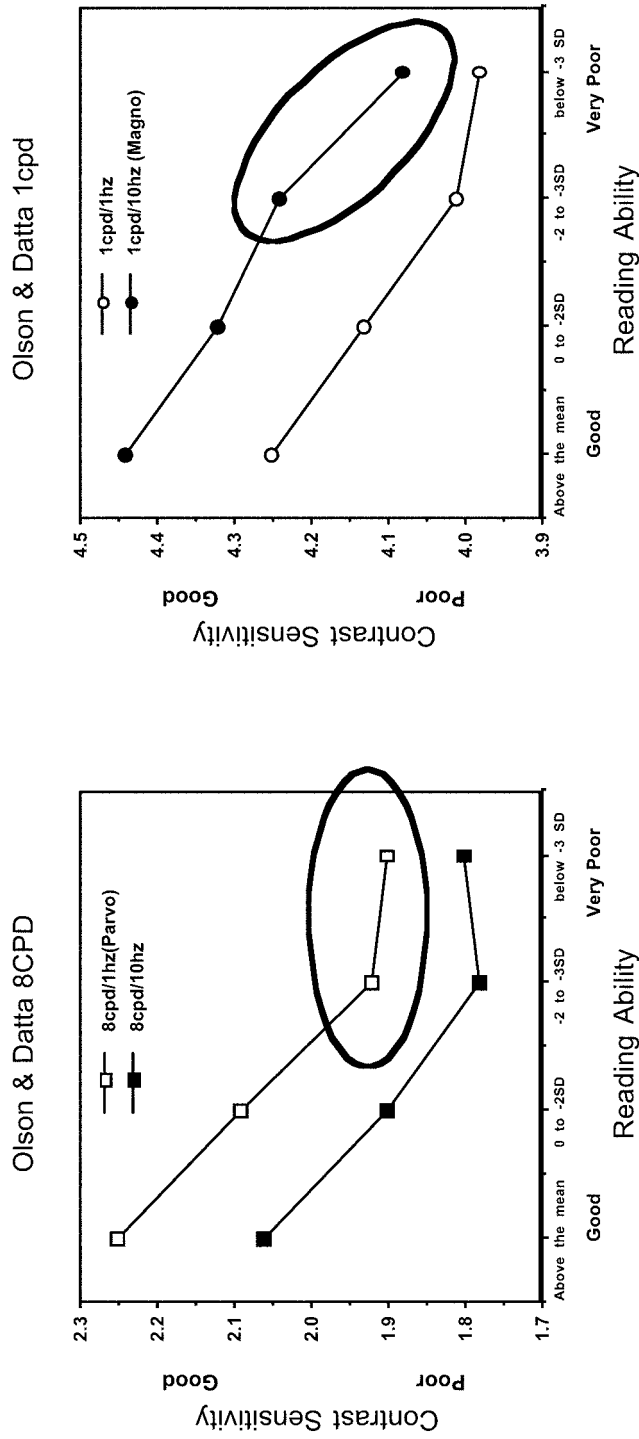


Figure 4. Contrast sensitivity as a function of reading group, temporal frequency (open symbols, low; closed symbols, high) and spatial frequency (left panel, high; right panel, low). The ellipses highlight the data from the two poorest reading groups and from the conditions that ought to best tap the parvocellular (left panel) and magnocellular (right panel) pathways. Adapted from Olson & Datta (this issue).

poor readers into subgroups, with the poorest ones beginning to approach the degree of deficit of Lovegrove's subjects. Although statistical analysis of the data provided no support for the possibility of a *selective* magnocellular (transient system) deficit in the most disabled group, comparison of the 4 conditions (see Figure 4) suggests that had even worse readers been tested by Olson a selective transient system deficit might have been obtained. Lovegrove's testing takes place under scotopic visual conditions, whereas the displays used by Olson were bright enough to allow the participation of photopic vision. According to Lovegrove the selective participation of the magnocellular pathway in detecting the 1cpd/10Hz oscillating stimuli is not guaranteed under photopic viewing conditions. Finally, it is possible that the method of adjustment and smaller number of observations which Olson's battery entails generated noisier and less stable data, making it difficult to obtain significant differences. Arguing against each of these last two points is the fact that Olson's test battery did yield a tell-tale pattern (which can be seen in Figure 4): with the low spatial frequency stimuli sensitivity is higher at high temporal frequencies; in contrast, with the high spatial frequency stimuli performance is better with low temporal frequencies. This is precisely what should be seen if the stimuli are assessing selectively the transient and sustained pathways.

Is there a temporal processing deficit in adult dyslexics?

Chiappe, Stringer, Siegel & Stanovich (2002, this issue) subjected normal adult readers, adult dyslexics (reading at or below the 25th percentile) and reading matched controls to a battery of tests designed to assess various aspects of temporal processing (not unreasonably, the reading matched controls were subjected to a subset of the tests used with the adult subjects). The inputs were auditory and visual stimuli and the outputs were reaction time and accuracy in most tasks. Each measure used in their battery could be subjected to its own task analysis. The major methodological approach was multivariate analysis, particularly, multiple regression. This study allows us to begin to answer the question 'Is there a temporal processing deficit in adults?', and if so, to assess whether its nature would support the claims of Tallal and suggestions of Farmer & Klein.

One general finding from Chiappe et al. is that the adult dyslexics perform worse than their age matched controls on nearly every task assessing temporal processing. Although this is precisely what might be expected if dyslexics suffered from a temporal processing deficit, there are several further findings which Chiappe et al. use to argue against the proposition that a deficit in *rapid* temporal processing *causes* (even indirectly) the reading problem.

One reason Chiappe et al. reject the TPDH is that on no task for which data was available from the reading matched control group, except the one assessing phonological processing, were the dyslexics worse than their reading matched controls. I find this reason unconvincing. The main purpose for testing a reading matched group is to address the concern that a deficit in comparison to the more typical age matched controls might be caused by a difference in print exposure. Poor readers will read less than good ones of the same age. Hence if a deficit is due to differences in print exposure it will not be present when print exposure is better equated by testing reading matched controls. However, there is a logical problem with insisting that to show a 'real deficit' dyslexics must be worse than reading matched controls. Whereas it is true that if a deficit is caused by print exposure it may not be observed when this confound is controlled for, it is also the case that a failure to find a difference between a dyslexic group and its reading matched control group does not mean that the deficit that is observed between the dyslexic and age matched controls is due to print exposure. Age is a powerful variable when performance is measured on almost all tasks. Hence, in nearly any performance based comparison involving children and adults, the older group would be expected to do better. Thus, if a dyslexic group has a true deficit on task A (one not due to print exposure) and, as with most tasks, performance on task A improves with age, then it is likely that when compared to a younger group of reading matched controls the older dyslexics' deficit may not be apparent. One response to this caution is to note that the dyslexic adults are worse than the reading matched children on phonological tasks. The rebuttal, would not deny that there is a genuine deficit here, but would note that the cognitive demands of the tasks used to assess phonological awareness and processing are generally low, hence age differences are unlikely to be such a significant factor as might counteract the genuine deficit.

A second reason leading Chiappe et al. to reject the TPDH is that in several temporal processing tasks (CV Same/Different, Auditory Gap detection) the deficit shown by the dyslexics did not get progressively worse as the ISI decreased. This pattern raises the possibility that the deficit has more to do with attention or working memory capacity than with extraction of information from a rapid sequence. However, the results from several tasks (CV matching, visual gap detection; 2- and 3-syllable repetition) did show (numerically, at least) the appropriate sensitivity to rate. Moreover, the temporal processing differences that were observed in reaction time (RT) in several tasks were not separated by ISI (TV matching, CV Same/Different, AudGap, VisGap). Because RT cannot be interpreted with confidence without a joint consideration of accuracy, and vice versa, the failure to report RTs for

each accuracy score makes it impossible to evaluate the possibility of a rapid temporal processing deficit on these tasks.

Finally, in regression analyses after the variance in reading scores attributable to phonological coding and the RAN was controlled for, none of the temporal measures accounted for significant variance. Similarly, commonality analyses demonstrated that while phonological processing and RAN performance contributed unique variance (16.6% and 6.3%, respectively) very little unique variance (1.3%) was attributed to a combined timing-perception measure. However, according to the causal path:

temporal processing deficit → phonological processing deficit → reading deficit

one would not expect temporal processing to contribute unique variance to reading once the variation due to phonological processing is removed. Moreover, as noted in Farmer & Klein (1995) several studies suggest that a temporal processing deficit that is apparent in young dyslexics may resolve (perhaps through compensatory mechanisms that are not yet understood) as they get older. If this were the case, then the residual temporal processing deficits seen by Chiappe et al. may not be reflective of the nature or severity of the deficits these individuals might have experienced when they were younger.

Like Chiappe et al., Farmer (Farmer 1993; see also Farmer & Klein 1993) administered a battery of temporal processing tasks to good and poor readers (for some measures from this thesis, see Table 3a). However, in contrast to Chiappe et al.'s participants who were adults, Farmer's were children with a mean age of 14. Also, more like Lovegrove's than Olson's, Farmer's participants severely dyslexic with a reading level that ranged from 3 to 7 years below grade level (mean 5.75). First, I want to draw your attention to the findings that on click fusion and auditory TOJs the dyslexics were worse than both their age and reading matched controls. Whether this difference between our study and Chiappe et al.'s is because the temporal processing deficits in our subjects hadn't had a chance to resolve or merely because all our subjects were children, cannot be determined. Using Tallal's task, Farmer tested TOJs using different ISIs. Her data from this task are shown in Table 3b. Farmer found that performance declined with ISI for all groups about equally, though on trials where the two tones were different (i.e., on those trials where most errors would be order errors), the dyslexic deficit was statistically more reliable for the shortest interval.

Table 3a. Reading, rhyming & auditory temporal processing performance by dyslexics and controls

	Controls		
	Dyslexics	Age matched	Reading matched
WATT (40 max.)	20.6	33.1	23.9
Rhyming	17	22.9	18.2
Click fusion (msec)	6.5	2.9	3.9
Auditory TOJ (%C)	80	97	87

Data from Farmer (1993). WATT is the Woodcock Word Attack. Auditory TOJ is collapsed across all ISIs and stimulus pairs.

Table 3b. Auditory TOJ performance for trials with different stimulus elements (high/low or low/high) as a function of ISI

Interstimulus intervals (ISI)	Controls		
	Dyslexics	Age matched	Reading matched
40	65	92.5	70
120	75	98.75	78.75
360	80	98.75	90

Data from Farmer (1993).

Early temporal processing deficits and later reading: A longitudinal analysis

As suggested by Farmer & Klein (1995: 486) one of the most direct and powerful strategies for assessing whether one deficit causes another is to examine performance longitudinally. Astutely (and courageously – as longitudinal studies are resource demanding and risky) David Share and his colleagues began just such a study way back in the early 1980s (Jorm, Share, Matthews & Maclean 1986). Recent excitement about the possible links between reading and temporal processing encouraged Share to conduct some reanalyses, and the paper in this special issue is the result (Share, Jorm, Maclean & Matthews 2002, this issue). In brief, Tallal's TOJ task was administered to many children entering kindergarten and then over 500 of them were followed through their first few years of school. This study thus provides an assessment of temporal processing in pre-literate children together with follow up reading scores collected during the course of reading acquisition. Several predictions which follow from the view that temporal processing deficits play a causal role in reading difficulty encountered by some children can be assessed using such longitudinal data.

This is an outstanding study whose biggest weakness is, ironically, the rigid adherence to Tallal's task! Following a protocol provided by Tallal's laboratory, Share et al. collected only 12 TOJ judgments when the interval between the tones was long (ISI = 428 msec) and 12 further trials consisting of a varied mix of shorter SOAs (8 to 305 msec). Moreover, 1/2 of all trials involved reporting the order of two tones of identical pitch. Although identification errors are possible on such trials, and single responses might indicate a failure to individuate two identical tones, order errors are not possible on such trials. In any case, when so few trials are collected from a task with a simple, 'correct/incorrect' dependent variable, the performance measure (% or number correct) is liable to be unstable.

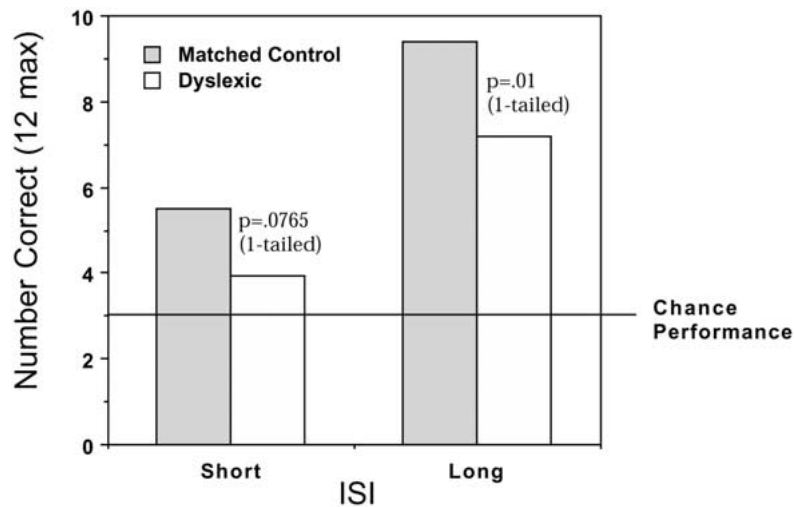


Figure 5. Temporal order judgment performance at kindergarten entry by students later classified as reading disabled and matched controls (from Share et al., this issue).

Using the number of correct responses, Share et al. that showed that children subsequently classified as poor readers had poorer TOJ performance at school entry than did a carefully matched control group (Figure 5). The deficit in TOJ performance, however, was significant at the long SOA but not at the short SOA, which might be interpreted as negative evidence for Tallal's proposal. It should be noted that performance at the short SOAs was very close to chance and that the trend was similar similar in magnitude on a %age basis for both SOAs. These facts suggest that the deficit was in temporal order judgments generally and not concentrated at either short or long SOAs.

In what should be a crucial test of the TPDH Share et al. examined temporally disabled individuals on entry to school during the following 2 years to see if they would develop reading problems. Although nearly all of the expected

Table 4. Reading and spelling performance in Grade 2 by children whose auditory TOJ performance on entry to kindergarten was normal (Control) or poor (TPD)

	TPD	Control	<i>p</i>
Word reading accuracy	32.1	40.5	0.117
Pseudoword accuracy	36.9	47.1	0.063
Comprehension	10.2	14.8	0.021
Spelling	29.5	33.7	0.152

Data from Share et al. (this issue). Group differences were assessed using t-tests, reported as *p*.

differences in grade 2 were in the right direction (see Table 4) (using the group with the long ISI deficit), only one was significant. If a temporal processing deficit causes dyslexia then the incidence of dyslexia should be higher in those children who performed poorly on the TOJ task. This prediction was not confirmed: The incidence of reading problems was no higher in the group showing temporal processing deficits (at the slow ISI) (10%) than the remainder of the sample (7.5%). In contrast to this negative evidence for the TPDH, performance on the TOJ task (long ISI) was highly related to phoneme segmentation in a sample of approximately 290 kindergarten children ($r = 0.34$).

Production of temporal sequences

In a classic paper Lashley (1951) highlighted the problem of serial order in behavior, and this issue is revisited in the work of Peter Wolff. In many of his experiments subjects are required to stay in beat with a metronome or use a memory guided strategy to produce a repeating pattern with the metronome providing only the tempo. Poor readers were not particularly poor at producing a regular beat in time with the metronome, but they did lag normal readers when required to switch to a new rate. More interestingly, poor readers were dramatically unable to perform a non-uniform rhythmic pattern (e.g.: | | | | |) with the metronome setting the beat, even though they could do so when the pattern was performed endogenously (that is without the metronome). Thus the dyslexics were unable to produce a pattern in the presence of a strong exogenous entraining tempo. Wolff noted that this problem with motor timing and sequencing carries over to the dyslexics' verbal behavior where it is apparent in occasional, unusually long, pauses.

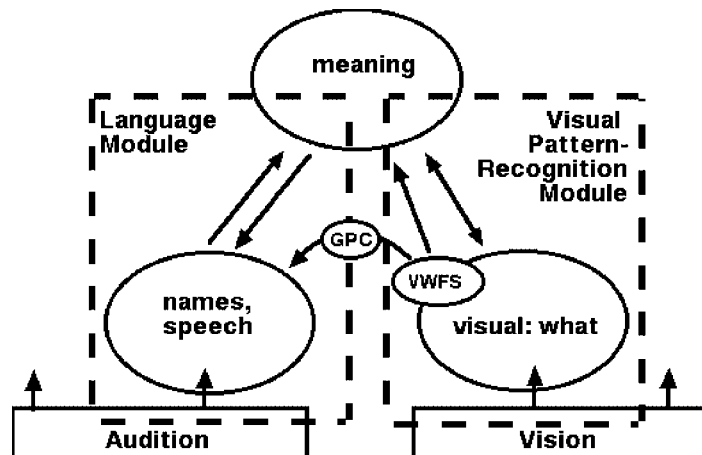


Figure 6. Schematic illustrating the major functional modules thought to be involved in reading.

Components of the Brain's reading machine

Reading involves the translation of visual symbols into propositions, often (though not necessarily) through the mediation of speech (subvocal or not), and hence is sometimes referred to as “visible language”. Some of the major functional systems that might be used in reading are illustrated in schematic form in Figure 6. This diagram is presented as a heuristic device that I hope will be useful for organizing some of the summational remarks which follow. In contemporary analyses of reading explicit computational modeling of architectures like that represented here have become a powerful scientific tool. But it is the interdisciplinary strategy of combining such computational modeling with evidence linking these abstract representations to actual modules in the brain – evidence that can be generated by modern neuroimaging techniques and by studies of the acquired dyslexia's – that yields the most fruitful and exciting contributions to our understanding of how the brain reads. Of course, the application of this interdisciplinary approach to the problem of normal reading acquisition and its failure in developmental dyslexia, will go a long way toward answering the questions posed at the beginning of this paper.

Modules crafted by evolution and refined by experience

It is generally accepted that humans are evolutionarily ‘prepared’ for language. Of course, this is an abstract sort of preparation, one that allows for the acquisition of any human language depending on the experiences we have as infants and young children. We are also evolutionarily prepared for visual

pattern recognition. This point is often not emphasized, however, perhaps because we do not differ from all other species in this regard. Communication and vision are of great importance to human survival, thus the modules that mediate language and visual pattern recognition are characterized by highly redundant coding schemes enabling them to function reasonably well even in the presence of small insults or degraded inputs. In contrast, written language is a human artifact of recent vintage, and therefore the mechanisms for decoding it (viz. reading) have not been used by the species long enough for evolution to have improved them via natural selection. The natural function of naming the objects and events we experience visually is mediated by pathways linking the language and visual pattern recognition modules. Reading, attaching names and meanings to visual symbols and groups of symbols (lexical access), involves the opportunistic use of these pathways. Through repeated reading experiences, the modules and pathways involved in this artificial collaboration become relatively more and more efficient. In the normal reader this will be reflected in the development of a visual word form system, as well as a representation (either explicit or implicit) of the grapheme-phoneme correspondences in an alphabetic writing system. Skilled reading of prose for comprehension depends on a coordination of parallel activities: voluntarily guided visual scanning, visually driven lexical access, and the natural processes of spoken language comprehension and production. Breakdowns in the acquisition of this complex coordination (skill) are referred to as developmental dyslexia.

Where (when) is processing defective?

As noted by Klein & McMullen (1999), because of the redundant coding that characterizes the language and visual pattern recognition modules, subtle deficiencies in these modules might not, unless measured with very sensitive and artificial tasks, cause an obvious deterioration in language or pattern recognition performance. However, such subtle deficits might be multiplied when the codes produced must be transferred to and analyzed by other modules using pathways with sparse connections.

According to the auditory/phonological TPDH as put forward by Tallal and Farmer & Klein (1995) a deficit in processing rapidly occurring auditory events (such as characterize the speech signal) will cause (proximally) a deficient phonological representation of the sound pattern of one's language. This deficient phonological representation causes (proximally) a reading problem because the complex, and somewhat artificial task of mapping orthography onto phonology is made even more difficult when the phonological representation is degraded. Thus, the auditory temporal processing deficit causes (distally) a difficulty with reading. There is evidence for and against the

propositions upon which this hypothesis depends in this special issue. In several cases (Share et al.; Chiappe et al.) the findings were ambiguous enough that (as with the famous Necker cube) scientists looking at the evidence with different expectations could interpret it in quite opposite ways. In both Chiappe et al. and Share et al. a temporal processing deficit was associated with poor reading by adults and by children; however, the TOJ deficit was NOT always more severe as the rate of stimulus presentation was increased. Although this finding would appear to flatly contradict the TPDH which assumes that the deficit is specific to *rapid* processing (see Figure 1), it is possible to present explanations for the pattern in each study that allow the TPDH to survive. How plausible these 'escape hatches' are perceived to be will likely depend on how committed one is to the TPDH.

One study (Olson & Datta) focuses on the visual side of the TPDH. Olson's diligent effort to obtain evidence for a visual transient system deficit in developmental dyslexia, and then to apply behavioral-genetic analysis to determine the heritability of this deficit, is laudable. As with Share et al's longitudinal study, this is precisely the kind of research that is needed to test the TPDH. Unfortunately, Olson's failure to replicate Lovegrove's finding of a selective deficit in the transient visual system, frustrated the more interesting effort to obtain an index of its heritability. Further research will be needed to determine the source of the discrepancy, which may be attributed to empirical and conceptual inadequacies of the magnocellular deficit theory of dyslexia (e.g. Skottun 2000).

The evidence presented by Wolfe and Bowers showing that RAN performance makes a unique contribution to reading ability even after controlling for the variance associated with phonological processing was compelling and important. Other evidence converges with the conclusion that phonological processing ability and RAN performance tap independent components of the reading machine. The RAN test involves rapidly naming colors and objects as well as letters and digits, and poor readers are poor on all 4 types of material, not just the alphanumerics. The deficit in RAN performance shown by many reading disabled individuals is, as Patricia Bowers demonstrated, likely to be more dependent on the encoding than on the output side of the processing stream. For these reasons, I propose that the RAN is tapping the efficacy of the pathways connecting the visual pattern recognition module with the auditory language module. This is one pathway (the other might go from vision to meaning to names) that would be used to name objects and events in literate adults as well as in children and pre-literate hominids and it is likely the pathway upon which print to sound (pronunciation) conversion and hence for reading acquisition critically depends (see Figure 6).

Caveats

When considering the evaluation of the hypothesis that there is a causal link from some deficit (e.g., in temporal processing) to dyslexia, there are two caveats to consider: heterogeneity and the direction of causality. If there are different classes of developmental dyslexia, then it is unlikely that they will all be caused by a common mechanism. Thus we are less likely to find the cause of developmental dyslexia than we are to find the causes of the developmental dyslexia's (cf. Farmer & Klein 1995: 460–461). I believe that this point is generally well accepted, and therefore does not require further development. Second, some temporal processing deficits may co-occur with dyslexia because poor readers read less than good readers do and frequent, rapid and successful print-to-sound conversion may tune some systems for other forms of rapid processing. Were this the case, then some temporal processing deficits could be the effect rather than the cause of dyslexia.

As this claim may appear counter-intuitive, let me try to buttress it by example. The papers in this special issue didn't emphasize the physiological differences between good and poor readers very much. Such differences may exist, however, and those who believe they do usually interpret them as strong evidence supporting one's favorite causal explanation (e.g. Breznitz, this issue). So, for example, when evidence is presented that the magnocellular system appears less well endowed in the brains of dyslexics than in normal readers (Livingstone, Rosen, Drislane & Galaburda 1991), the typical interpretation is that this difference in neural efficacy is responsible for the different reading levels. However, "... the possibility must be considered that the differences in brain structure found in studies [of dyslexic and normal brains] may be the result of a reading disability or speech perception problem, rather than the cause of such deficits" (Farmer & Klein 1995: 478). Indeed, it has been a fundamental principle of modern neuropsychological theory at least since Hebb (1949) that the experiences we have can cause long-lasting changes in the brain. The early experiences a child has being read to, the strategies emphasized in training the young reader and the strategies naturally selected by the reader will strengthen some pathways and modules and reciprocally weaken others. Similarly, the amount of reading experience (print exposure) might dramatically affect brain processes and structures. Spending several hours a day making 4–5 deliberately guided saccades per second, with each one bringing a new, high contrast, high spatial frequency array into the fovea while erasing the previous one, ought to do something to the brain (see Merzenich, Nelson & Stryker 1984, for a demonstration of such influences in somatosensory cortex). Therefore, if one person reads much less than another person, all other things being equal, we should expect to see differences in their brains just because of their different experiences. Of course, this is not

to deny that brain differences can be the cause of performance differences. Rather, my purpose was to illustrate that discovery of an association between a neuroanatomical and/or neurophysiological measure and reading ability is not sufficient for inferring that a brain difference causes a reading ability difference. A multidisciplinary effort to generate converging evidence (Klein & McMullen 1999) is the recommended strategy to confidently determine why some people fail to read well.

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