

# **A Multi-Level Synthesis of Dyslexia**

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Dyslexia has been studied from many angles. Researchers have obtained seemingly contradictory results and created widely varying theories and treatments. A complete understanding of dyslexia requires recognition of neurological and psychological components and their interaction, and could therefore benefit from a complex systems approach. This paper surveys and synthesizes results from many theoretical, experimental, and clinical approaches to dyslexia, including Galaburda, Davis, Geiger, and Merzenich. The magnocellular hypothesis combined with the Davis theory of "triggers" appear to explain nearly every experimental result, observation, and successful treatment of which the author is aware. Dyslexia can be understood as an accretion of simple symptoms in multiple sensory modalities, each symptom having the same neurological basis; each individual has a different combination of symptoms, and the symptoms are created and maintained through mental/psychological interaction with the individual's efforts to perform. There is strong observational evidence, confirmed by pilot studies carried out by the author, that the symptoms can change momentarily. Although such rapid change is not recognized by many dyslexia researchers, it has been demonstrated with PET scans in the case of stuttering; this finding is crucial to a full understanding of the interaction between neural function and mental state. The recognition of the diversity of symptoms, their common neurological basis, and their extreme plasticity in response to high-level mental state, may help to focus research and to develop increasingly effective and rapid treatments.

## **1 Introduction**

Over the past several decades, much work has been done to investigate the causes, symptoms, and treatments of dyslexia. Several opposing theories have been pursued [Ramus 2001], and several successful treatments have been developed and evaluated [McAnally, Castles, & Stuart 2000]. While each approach attempts to explain all the features of dyslexia, there has not been an attempt to synthesize the various approaches. At first sight, this would be an impossible task, since the theories contradict each other at many points. This paper presents an approach that attempts to cover all the observations, although it differs from current theories at several important points.

The present approach requires an understanding of dyslexia as a multi-level phenomenon: specifically, a set of high-level responses to low-level disruptions of perception. It is notable that several dyslexia correction methods, grounded in contradictory theories, claim success rates well over 50%. Also striking is the variety of symptoms and findings of dyslexics. In today's educational environment, a failure to read adequately is crippling, and thus forms a natural target for intensive study. However, the symptoms and syndromes associated with dyslexia are much broader than simple failure to read. These diverse symptoms can all be understood as arising from a common low-level cause, with the caveat that the low-level mechanism can affect, and be affected by, high-level mental state. This leads to a complexity that can be confusing or misleading to researchers attempting to study a single aspect of dyslexia.

There is no universally accepted definition of dyslexia. Most studies attempt to define it in terms of reading deficit vs. general intelligence, with various methods of establishing reading ability and intelligence level. Some studies require that remedial education has been unsuccessful. This paper will not attempt to establish a single, universal definition of dyslexia; instead, it will assume that when researchers intended to study dyslexia, they generally succeeded in forming a suitable study population. Another factor that is important in some papers is that of subtypes of dyslexia, for example, auditory and visual subtypes. As will be seen later, this paper recognizes the existence of auditory and visual syndromes, but does not support the idea of subtypes that must be studied separately (although the syndromes may need to be treated individually in clinical practice). Therefore, studies of any dyslexic subtype will be considered.

## **2 The Low-level Basis Of Dyslexia**

Many studies have found differences between dyslexics and non-dyslexics in low-level perception. For example, many researchers have found differences in visual perception such as the width and acuity of letter recognition field [Geiger & Lettvin 1987], or the sensitivity to contrast and motion under various conditions [Lovegrove, Garzia, & Nicholson 1990]. Tallal, Miller, and Fitch [1995] have found differences in auditory processing, such as a reduced sensitivity to rapid sound transitions. Any

theory of dyslexia must recognize and account for the multisensory nature of the disorder.

It is tempting to suggest that the magnocellular system is responsible for these sensory problems. Livingstone, Rosen, Drislane, and Galaburda [1991] have found physical malformation of the cells in the visual magnocellular pathway in dyslexics. In addition, many studies have found visual deficits consistent with a deficit in magnocellular processing. Unfortunately, the first proponents of the "magnocellular hypothesis" made a supposition that appears to be incorrect about how a magnocellular deficit could affect the visual system [Skottun 1999], and other studies have obtained inconsistent results [Skottun 2001].

Grossberg and Cohen [1997] have found clear evidence of distinct fast and slow processing channels in the auditory system. Tallal, Miller, and Fitch [1995] and Shaywitz and Shaywitz [1999], have found that dyslexics have a deficient ability to detect rapid transitions in normal speech. As noted by Stein [1993, p. 83], "The magnocellular component of visual processing that is impaired in dyslexics, does have anatomical counterparts in the somesthetic, auditory, and motor systems..."

Given the low-level nature of these symptoms, and (in some studies, at least) the unambiguous character of the difference between dyslexics and normals, it seems clear that dyslexia must involve a difference in the pre-cortical neural pathways of more than one sense. The best candidate seems to be the pathways responsible for maintaining information about timing and rapid change. In the visual system, this is the magnocellular pathway. This should not be read as support for the "magnocellular hypothesis", especially its original suggestion that the faulty mechanism was suppression of the parvocellular pathway by the magnocellular pathway. In hindsight, that suggestion was only one of many possibilities.

The claimed effectiveness of colored lenses (Ihrlen lenses) or colored overlays for books may provide additional evidence for a low-level visual basis in at least some cases. However, it should be noted that such claims are controversial [Evans & Drasdo 1991].

### **3 Evidence For Rapid Controllable Variability In Brain Function**

Dyslexia has been viewed historically as resulting from some form of brain damage. More recently, many researchers have found that even the low-level symptoms of dyslexia may be improved with appropriate training [Geiger & Lettvin 1997]. This improvement typically takes place over a period of weeks or months. A key point of the present approach is the idea that the symptoms can vary on a far more rapid time scale.

Geiger [1999, personal communication] has described a phenomenon of "conditional dyslexia", in which a person's ability to read depends on external factors, and may thus change rapidly. Davis has described his own variable dyslexia: "When I was at 'my artistic best', I was also at 'my dyslexic worst'" [1997, p. 127]. At this point, it seems unarguable that dyslexia is not a permanent condition. The only question is how rapidly it can change.

That aspects of language processing can change with extreme rapidity is demonstrated by the phenomenon of stuttering. Stuttering appears to involve faulty pathways between various parts of the brain; the activation of these pathways can be detected with PET scans. Notably, these pathways are not permanently damaged. When a person is not stuttering, they work normally. However, they show a deficit if the person even thinks about stuttering [Sobel 2001]. It is well known that stuttering is conditional--it depends on a person's emotional state and past experience. The recent PET studies show that these conditions, including imaginary exercises, can affect even low-level signal pathways in the brain [Ingham, Fox, Ingham, & Zamarripa 2000]. The studies also underscore the point that the low-level mechanisms of stuttering can change in a matter of seconds. It should be noted that stuttering is the result of an interaction between memory, rapid cognitive events, and rapid low-level changes.

An alternate approach to controllability of dyslexic symptoms is provided by Nicolson and Fawcett [2001] who hypothesize that dyslexia is based in cerebellar deficit. They note that "Difficulties in skill automatization correspond directly to the traditional role of the cerebellum." Also, as noted at "What is dyslexia?" [Internet], "The cerebellum plays a significant role in the timing system of the brain; the magnocellular system," and, "Dyslexics can only do acts, that normally are automatized, if they get the opportunity to consciously compensate for their deficit in automatization." This implies that dyslexics can perform these acts under the proper mental conditions, but that a condition such as confusion that interferes with conscious mental processing would degrade their performance more than that of non-dyslexics.

Many dyslexia researchers have not looked for extremely rapid changes. As mentioned above, Geiger is an exception. Another notable exception is Davis, who has described several exercises for teaching a dyslexic person to deliberately control their perceptual distortion [1997, pp. 149-177]. All of these methods take only a few minutes to teach, and the results are claimed to be immediate: a person doing these exercises will briefly have undistorted perception.

The author has tested these claims and has found support for them. Subjects were taught two variations of the exercise, one of which is designed to decrease perceptual distortion, and the other designed to increase it. Subjects were then presented with stimuli in a range, and asked to classify the stimuli to one of the endpoints. The point of division appears to vary depending on which exercise the subject has most recently done. Intervals between successive trial sets were usually less than 20 seconds. At this point only pilot studies have been done, but the effect was found for both auditory stimuli (Klatt-synthesized speech sounds) and visual stimuli (Phi stimulus).

## **4 The Dyslexic Feedback Loop**

Dyslexics manifest a multitude of symptoms. Different individuals may demonstrate primarily auditory [Schulte-Koerne, Deimel, Bartling, & Remschmidt 1999] or primarily visual symptoms, or a mix of the two. Dyslexia is often associated with other problems, such as ADD [Richards 1994], dyspraxia (clumsiness), and dyscalculia and dysgraphia [Temple 1992]. In addition, as noted above, the

symptoms may vary from time to time in a single individual. It seems clear that low-level neural damage, although present, is not the whole story.

Again we can take a clue from stuttering. It has been noted that stutterers frequently stutter on words they expect to have trouble with, and it has been suggested that the experience of previous failures acts to exacerbate the stutter on troublesome words. Given that dyslexia also involves a low-level deficit that can be controlled by high-level mental state, it seems clear that a similar mechanism is probably involved; in fact, this is suggested by Davis in his theory of "trigger words" [1997 p. 21]. According to this theory, a dyslexic person suffers an initial failure when confronted with a confusing task such as letter or word identification. The memory of this failure may persist, causing confusion to occur whenever the individual is confronted with the same symbol. This confusion then activates the perceptual distortion, causing repeated failures and reinforcing the confusion.

By this account, a dyslexic person will have an almost random set of "triggers" that cause the perceptual problem to occur. However, in a task as complicated as reading, the chance of encountering a trigger is very high. This accounts for the consistency of reading failure observed in many dyslexics. Conversely, the fact that the perceptual problem in fact depends on mental state accounts for the cases of conditional dyslexia.

Other tasks, such as handwriting and coordination, are also complicated enough to cause confusion and may suffer from faulty perceptions. Thus a person with variable perceptions may suffer from dyspraxia, dyscalculia, dysgraphia, etc. If early confusion associated with a task is resolved quickly, it is quite possible for a person to avoid a dysfunction on that task. However, if the confusion is allowed to persist and grow, the person will develop a disability in that area as a result of accumulated triggers.

As found by Nolander [1999], the expectation of improvement is an important factor in reading improvement in dyslexics. Certainly anyone who expects to improve will be less stressed at the thought of failure. Also, a change of context may reduce the strength of recall of former failures. The expectation factor should be considered in evaluating results reported for any treatment method. However, that does not damage the present argument; in fact, Nolander's observation that, "The interaction of treatment group and presence of dyslexia indicated that dyslexics are more sensitive to expectation," indicates the presence of a feedback loop, while the fact that, "Few dyslexics improve in the general, 'nonresearch' population regardless of the treatment," indicates a real underlying problem.

The feedback loop is strengthened by the fact that perceptual distortion is not always a bad strategy. As noted by Merzenich, Schreiner, Jenkins, and Wang [1993, p. 15], "Could dyslexia and developmental dysphasia arise because some infants adopt more global hearing or looking strategies? .... Note that very early visual practice with a wider field of view does not necessarily represent a 'deficient' or 'negative' or 'dysfunctional' behavior. To the contrary, it would probably represent a practice strategy that ... presents advantages for the rapid processing of relatively complex, spatially distributed inputs .... Once a 'bad' looking (or listening) strategy is in place, by this hypothetical scenario, visual scene representation at every cognitive level as well as eye movement representations would be powerfully reinforced by the

many tens or hundreds of thousands or millions of input repetitions." Davis [1997, pp. 72-74] describes a similar process, with imagination being used to supplement distorted vision: "Let's make little P.D. [Potential Dyslexic] three months old and put him in a crib. From his perspective, all little P.D. can see is ... a chest of drawers with someone's elbow .... If little P.D. happens to trigger the brain cells that alter his perception, he will no longer see what his eyes see, he will see something else .... little P.D. actually saw a face in his mind .... So here is little three-month-old P.D. recognizing things in his environment that he shouldn't be able to recognize for three more years. This ability he has for recognizing real objects in his environment will influence the rest of his early childhood development."

By the time dyslexics start learning to read, they will have been using a strategy of perceptual distortion quite successfully for many years. In particular, they will use it to resolve confusion when confronted with novel stimuli. This strategy is unsuitable for the task of reading, which involves many symbols that are meaningless without training and must be perceived accurately. However, as noted by both Davis and Merzenich, the strategy will be so ingrained that only a deliberate effort can countermand it. Absent that effort, the dyslexic will continue to be confused when trying to read, and the perceptual distortion strategy will only add to the problem.

## **5 Dyslexia Correction Methods**

There are several dyslexia correction methods in current use that have a demonstrably high degree of success. It is worth noting that these methods are based on incompatible theories and generally address only one sensory modality. Any theory of dyslexia should explain how they can all be successful.

Geiger and Lettvin [1997] achieved notable improvement in 100% of 27 children using simple visual and visual-kinesthetic exercises. Having observed that non-dyslexic readers have a narrow and asymmetric field of letter recognition while dyslexics have a broader and more symmetric field, they have students move a small window over the text they are reading. In a few months they read significantly better, and their letter recognition field became narrow and asymmetric.

Paula Tallal and Michael Merzenich, among others, have developed a program that trains subjects to listen to distorted speech sounds. Subjects improve in their ability to distinguish the sounds. Their reading also improves ["Fast ForWord Outcomes", Internet]; [Tallal, Miller, Bedi, Byma, Wang, Nagarajan, Schreiner, Jenkins, & Merzenich 1997].

These results cannot be reconciled unless there is an underlying mechanism that affects both the auditory and visual senses, so that training either sense improves the overall function. It is also worth mentioning that Delacato [1966] and others have claimed success with kinesthetic training, although the effectiveness of these programs has been questioned [Cummins & Coll 1988]. Although each result is inconsistent with the theory of the other approaches, all results are consistent with a timing-pathway theory. If a transitory timing-pathway deficit exists, then practicing any task that requires stable perceptions (without engendering additional confusion) will train the dyslexic to control the timing path problems to some extent.

Rather than focusing on any specific sensory pathway, the Davis program focuses on finding and correcting sources of confusion, after teaching the subjects how to temporarily control their perceptions. Davis claims a 97% success rate for his original program ["Davis Dyslexia Correction", Internet]; a variation for younger children in a school setting has also been found to be effective [Pfeiffer, Davis, Kellogg, Hern, McLaughlin, & Curry 2001]. A theory of controllable timing pathways would predict that such an approach could be successful, and in fact could be applied to other disabilities as well. The author has provided Davis programs to many children and adults, and observed rapid improvement in reading (within one week) in most cases. Informal followup after approximately two years found that 17 out of 18 clients were reading significantly better than expected, and 15 out of 18 attributed this to the Davis program. The author also observed rapid changes in handwriting, math, and physical skills, in cases where clients experienced difficulty in these areas.

## **6 Discussion**

This theory covers a lot of ground, so there is a lot of room for discussion and even speculation. I've arranged this section in several parts. Each of the parts stands on its own, and a failure of one speculation should not reflect on the others.

### **6.1 The Synthesis of "Real" and Imaginary Universes**

During normal vision, the eye takes repeated "snapshots" of the environment, and moves rapidly between the snapshots. The rapid movements are called saccades. The fragmented images are integrated to form a complete picture of the visual field. It seems plausible that one effect of a visual magnocellular deficit would be a loss of information about how far the eye moved during a saccade: a reduced ability to track the area the eye has swept across. This could cause difficulty in the integration of the images. Such integration is vital to the proper functioning of the visual system, so the brain would have to compensate. One method of compensation would be for the visual system to gain competence in rotating images and identifying images from fragments--a function performed by the parvocellular system. Davis [1997] and West [1997] describe visual thinking as a common concomitant of dyslexia. This parvocellular competence could account for the frequent observation that dyslexics are "creative."

Some authors have speculated on a link between dyslexia and ADD. One effect of a timing-pathway deficit would be a reduced ability to estimate the passage of time, or to keep one's internal "clock" stable. A person with a timing deficit would thus be susceptible to one of two dysfunctional modes in response to the experience of boredom, confusion, or frustration. If boredom caused the internal clock to speed up, then the person would perceive time as dragging even more. This would intensify the boredom, which would cause a further speedup. Conversely, if confusion or frustration caused the internal clock to slow down, then an overwhelming experience such as too-rapid presentation of knowledge would cause the clock to slow, which would make the experience even more overwhelming. In either case, a vicious cycle would develop, reinforced by the expectations built by previous cycles.

Davis [1997, p. 72] has speculated that earlier and more severe onset of the same perceptual distortions that lead to dyslexia may be the cause of autism. Merzenich, Saunders, Jenkins, Miller, Peterson, & Tallal [1999] have noted that their Fast ForWord training results in the same performance gains in children with PDD as in children with specific language impairments. Delacato [1974] has also noted that autism appears to involve severe perceptual distortion, and has applied similar treatments to autistics and dyslexics. Davis [personal communication, 1995] has described unpublished experiments in which a variation of his orientation procedure has produced marked improvement in some autistic children. It seems likely that methods which train dyslexics to stabilize their perceptions may also help children with autism--especially given the preliminary success by Merzenich, Davis, and Delacato with their widely varying methods.

## **6.2 Investigating the Mechanisms Of Dyslexia**

Dyslexia research is full of observations that are directly contradictory. Examples include [Livingstone, Rosen, Drislane, and Galaburda 1991] and [Johannes, Kussmaul, Munte, & Mangun 1996], and the results surveyed in [Skottun 2000]. Many good researchers have spent much time and effort only to find opposite results. In addition, such contradictions hamper the effort to develop consistent and useful theories, or worse yet, allow the development of theories that only account for fragments of the available evidence.

Any investigation of low-level brain function must take several new factors into account. First, individual dyslexics are likely to react differently to the same task. One dyslexic may be confused by a task and suffer perceptual distortions, while another finds it easy and approaches it with perceptions in "non-dyslexic" mode, and a third finds a way to use non-standard perceptions to make the task even easier. Second, some people who have no reading problem may nevertheless have a timing path deficit, and so should be included with the dyslexic population or excluded from the study. Third, when planning or attempting to replicate a study, the set and setting, and other factors affecting the emotional and cognitive state of the participants, may have a significant impact on the results obtained.

There are several ways to compensate for these difficulties. To detect a timing-path deficit in control subjects, subjects could be screened for multiple disabilities, not only dyslexia. It is likely that a timing path deficit will be apparent in at least one skill or activity. However, this method is difficult and ad-hoc. Another way to compensate would be to ensure that all dyslexics (and controls) are in the same mental state. The "orientation" exercise of Davis may be helpful here, although it may mask some dyslexic effects. Unfortunately, the effects of the exercise on mental state have not been well established; this is an area for further study. At this point, there is strong anecdotal evidence and some experimental evidence that the exercise has a marked effect on multiple perceptual mechanisms, including auditory, visual, and kinesthetic, in dyslexics. Effects on non-dyslexics are unknown.

The fact of "conditional dyslexia" needs to be definitively established and widely recognized. It seems likely that PET, fMRI, or possibly QEEG [Chabot, di Michele, Prichep, & John 2001] applied to dyslexics under appropriate conditions would be

able to detect a difference between a "dyslexic" and "non-dyslexic" mode, as has already been done for stuttering with PET. Again, the Davis "orientation" exercise may be helpful here.

### **6.3 Related Conditions**

There are a number of conditions (beginning with the syllable "dys") that appear to be explainable by the present theory. A complete survey is beyond the scope of this paper; however, a partial list may be instructive and may suggest further research directions. The author has observed the Davis method providing rapid improvement (a few days) in cases of each of these conditions.

Dyscalculia is difficulty with arithmetic or other mathematics. It seems obvious that the same conditions leading to reading difficulty could also cause a problem when perceiving or interpreting the symbols used in math. There is even more opportunity for confusion because of the increased abstractness and reduced contextual clues available in math as opposed to text.

Dyspraxia, or clumsiness, may result from visual perceptual distortion. Balance is partially dependent on accurate vision, and a visual distortion may magnify or obscure the effects of subtle head movements. In addition to the obvious problems created by a timing deficit, a feedback loop may appear, in which the stress of expectation of failure (e.g. falling off a balance beam) may cause increased perceptual distortion, resulting in new failure and reinforcing the expectation.

Handwriting involves many fine motions and much feedback between eye and hand. According to Davis, the confusion feedback can occur with regard to certain motions or letters. Dysgraphia, or poor handwriting, may result.

### **6.4 Improved Dyslexia Correction**

As noted previously, dyslexics frequently do poorly on a variety of tasks, and several programs have been designed that involve practicing a certain task; improvement in the task generally coincides with improvement in reading. Presumably the reading improvement depends on the subject first learning to do the task better and then practicing the new skill to "lock in" the improvement. A faster improvement on the task might translate into faster progress in reading, since the subject would be able to spend more time doing the task correctly. The Davis orientation exercise may provide such accelerated improvement in a variety of tasks.

Likewise, the confusion and frustration often felt by dyslexics could be ameliorated by directly addressing the confusion encountered by dyslexics during attempts to read. If dyslexia is based on a feedback loop between low-level perceptual distortion and high-level confusion, then explicit instruction in language symbols may be beneficial in any dyslexia program. Davis [1997, pp. 197-212] suggests a visual-kinesthetic learning method, echoing the observation [West 1997] that dyslexics frequently think visually.

Finally, it should be universally recognized that regardless of whether subtypes of dyslexia exist, a variety of types of exercises work for the vast majority of dyslexics. Every clinical dyslexia program should be willing to test and integrate exercises from a variety of approaches. For example, Merzenich uses a fine-motor exercise in

addition to the visual exercises, and Davis uses a large-motor exercise involving catching Koosh balls while balancing on one foot.

## 7 Summary

Dyslexia has been investigated from many angles. However, investigation has been hampered by contradictory theories and even contradictory results. The notable success of several treatment programs based in incompatible theories suggests an underlying mechanism that can be affected by a variety of interventions.

Much evidence has accumulated to suggest that the mechanism involves the timing pathways of the brain, probably at a cellular level. Many researchers have reported lasting results from interventions over a period of months, suggesting either that the problem is correctable or that dyslexics can learn to compensate with appropriate training (although they do not learn to compensate without training). Some researchers have gone farther and demonstrated improvement in a period of days, and perceptual changes requiring only a few seconds.

A theory in which the perceptions are unstable (flexible) and controllable by conscious or subconscious mental state on a time scale of seconds appears to account for all of the observations, including the contradictory results obtained by numerous experiments. Stuttering research appears to lead dyslexia research in this approach, having established that brain activity related to stuttering does in fact change rapidly depending on mental state.

The verification of rapid perceptual change in dyslexia, and the application to experiments and treatments, should be a high priority. A variety of new short-time-scale techniques for observing brain function, and the Orientation exercise of Davis which directly affects perceptual distortion in dyslexics, will be helpful. Dyslexia research, and treatments for dyslexia and many related conditions, will benefit greatly from the verification and control of this rapid change mechanism.

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