Dyslexia, Learning, and the Brain

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1 Introduction

The traditional formal definition of developmental dyslexia is ''a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities'' (World Federation of Neurology, 1968, p. 26). Dyslexia is the most prevalent of the developmental disorders, and the most researched, with the U.S. National Institute for Child Health and Human Development funding its dyslexia program at $10 to 20 million per year since the late 1980s. Despite this intensive research, lively and unresolved controversies remain as to the underlying cause, the appropriate methods of diagnosis, and the optimal means of support for dyslexic children; many influential researchers question the very concept of dyslexia as a coherent syndrome.

It is important to note that dyslexia is traditionally defined in terms of a discrepancy between actual reading performance and what would be expected based on the child's intelligence. A central problem with this definition is that a child must fail to learn to read for two years or so before a formal diagnosis is considered valid. It is, of course, very destructive for a child to have the crucial early years at school blighted by failure to acquire one of the fundamental skills. In the later school years, there is also the danger of a vicious circle of poor reading leading to poor motivation, avoidance of text-based school work, emotional trauma, and adoption of maladaptive strategies such as clowning around, disruptive behavior, or truancy. Even in adulthood, many dyslexic people still feel intensely angry about the way they were treated at school. Nevertheless, many dyslexic children turn out to be creative and successful, and it has been suggested that a disproportionate number of our most creative artists and scientists were dyslexic (West, 1991).

A brief historical review demonstrates both the range of possible explanations and the surprising swings in fashion that characterize dyslexia

research. Recognition of developmental dyslexia is credited to Pringle Morgan (1896), who identified a 14-year-old boy called Percy, who despite adequate intelligence was unable to even write his name correctly. The concept was taken up by James Hinshelwood (1917), a Glasgow eye surgeon, who used the term *word blindness*, and the American neurologist Samuel Orton (1937), who advocated use of the term *strephosymbolia* to indicate that the problem was not one of word blindness per se but of ''symbol twisting.'' Working from 1925 onwards, Orton studied over 1000 children. His work inspired many, including the neurologist Norman Geschwind, and led to the foundation of the Orton Dyslexia Society (now the International Dyslexia Association).

One may see from this brief history that early work on dyslexia derived from a medical perspective and was strongly influenced by clinical insights. Moreover, when the Word Blind Centre was set up in the United Kingdom (UK) in the early 1960s to study the diagnosis and teaching of dyslexic children, the terminology adopted was clearly influenced by the U.S. research. In this center, Sandhya Naidoo was the first researcher to publish quantified differences between dyslexic boys and controls in terms of late speech and articulation difficulties, identifying a specific pattern known as the ACID profile within a group identified by ''exclusionary'' criteria, namely, ''difficulty in learning to read and spell in physically normal intelligent children'' (Naidoo, 1972). Margaret Newton, Michael Thomson, and Ian Richards at Aston University undertook similar theoretical work and developed the Aston Index, a comprehensive diagnostic battery for dyslexia (Newton, Thomson, & Richards, 1976). Tim Miles (e.g., 1983b) adopted a similar approach in his analysis of what he called the syndrome of dyslexia, derived from his clinical caseload of 223 children in the early 1970s, which formed the basis of the Bangor Dyslexia Test (Miles, 1983a). In the UK in the 1970s dyslexia was also studied from an epidemiological perspective, because it is after all in educational settings that the problem first shows up. The definitive early work in the UK derived from a large-scale study in the Isle of Wight (Rutter & Yule, 1975) that identified an unexpected ''hump'' of around 4% in the normal distribution of low achievers. This 4% showed specific retardation in reading despite adequate intelligence, and, surprisingly, had a poorer prognosis than children who were more generally backward. Although the existence of a ''hump'' was not supported by subsequent work, the general incidence level of 4% still provides a representative estimate of the prevalence.

A significant change of focus in dyslexia research arose following a seminal analysis by Frank Vellutino (1979), when it was realized that the deficit

was not just in visual processing, but also, and perhaps primarily, in processing of language. One of the major achievements of dyslexia researchers in the 1980s was to refine this concept of a linguistic deficit, developing the *phonological deficit theory* that remains the consensus view of much of the dyslexia research community to this day. The preeminent status of the phonological deficit hypothesis derives from findings in the early 1980s that dyslexic children had particular difficulty in hearing the individual sounds in words. For instance, at the age of 5 years, children who would later turn out to be dyslexic had considerable difficulty in hearing that, say, *cat*, *mat*, and *bat* rhyme. In general, they seem to have limited ''phonological awareness'' (sensitivity to the sound structure in words). This phonological deficit leads to difficulties in learning to read and spell because one of the early stages in learning to spell is to split a word into its component sound chunks, each of which then has to be spelled in order.

In an article commemorating the centenary of the discovery of developmental dyslexia, Sally Shaywitz (1996) explained that the key assumption of the phonological deficit hypothesis is that a deficit in the speech/language phonological module in the brain leads to specific problems in learning to read (and in remembering linguistic information), without otherwise affecting higher-level reasoning. She illustrated the fundamental paradox of dyslexia—the discrepancy between reading ability and other skills—in the example of Gregory, a dyslexic medical student who ''excelled in those areas requiring reasoning skills. More problematic for him was the simple act of pronouncing long words ... perhaps his least well-developed skill was rote memorization'' and went on to outline an impressive range of multidisciplinary evidence consistent with the phonological deficit hypothesis. She concluded that ''The phonological model crystallizes exactly what we mean by dyslexia: an encapsulated deficit often surrounded by significant strengths in reasoning, problem solving, concept formation, critical thinking and vocabulary'' (p. 84).

Interestingly, although there is no doubt that difficulties in processing phonological information are a characteristic feature of dyslexia, phonological difficulties can arise from a wide range of causes. Furthermore, new discoveries of abnormalities in the processing of visual and auditory information, allied to findings of subtle difficulties in a wide range of skills, have cast doubt on the phonological deficit as the *only* cause of dyslexia (see section 2.1).

Arguably, therefore, the key theoretical priority for dyslexia research is to identify the underlying cause(s) of the phonological deficits. For these purposes, it is important to establish the full range of symptoms of dyslexia

(whether or not they are related to reading) and to consider the possible neural mechanisms that might underlie these symptoms. Recent research has suggested that phonological difficulties may be just one piece, albeit a central one, in the jigsaw puzzle.

In short, researchers from different backgrounds have identified a range of apparently unrelated problems in dyslexia. It is hard not to get confused. The more one reads, the more confusing it gets. At this stage, therefore, we think it is useful to take a step back, to find a suitable vantage point for surveying the entire picture.

1.1 Explanation in the Developmental Sciences

In common with that in many Western countries, dyslexia research in the United States and the UK has been remarkably successful in its political objectives over the past decade. Dyslexia is now established as a key disability, and hence dyslexic children and adults benefit fully from increasingly powerful disability legislation. In the United States, the 2004 Disabilities Education Improvement Act (IDEA) introduced the concept of identifying children ''at risk'' and intervening early, recognizing the importance of preliteracy skills in the development of the young child. IDEA advocates an inclusive approach whereby support is provided early for children with an at-risk profile, so that intervention is more effective and cost-effective. Furthermore, the UK Code of Practice for Children with Special Educational Needs (UK Department for Education, 1994; DfES, 2002) explicitly requires schools to diagnose and support dyslexic children (and children with any special need) from the very start of schooling. Nonetheless, the principles of teaching dyslexic children date back to work in the 1980s, and there is currently no theoretically informed link between the individual child and the individual support provided. Of course, theoretically informed links depend on having theoretical frameworks that map explicitly onto diagnosis and support.

One of the fascinating aspects of dyslexia research is that, whatever one's speciality as a researcher—reading, phonology, writing, spelling, education, memory, speed, creativity, hearing, vision, balance, learning, skill, genetics, brain structure, or brain function—dyslexic children will show interesting and unusual differences in that domain. Given the need for specialization in science, many researchers have undertaken incisive and insightful studies in their specific domain of expertise. This explains why, on the one hand, there is an unrivaled wealth of research on dyslexia, and, on the other hand, the research fails to cumulate in or to build toward a

''grand'' theory of dyslexia. In an analogy much loved by psychologists, it is like the Hindu fable of the four blind men attempting to describe an elephant. One touches the trunk, another the leg, another the side, another the tail, leading to descriptions of ''a pipe,'' ''a tree,'' ''a house,'' and ''a rope,'' respectively. If one wants to describe the whole elephant, one needs a range of perspectives. Let us start the tour of the elephant by identifying some potent causes of confusion in the area.

One of the greatest challenges for theoretical research in dyslexia is to find an explanatory framework sufficiently general to accommodate the diversity of the deficits in dyslexia while sufficiently specific to generate testable predictions, to support better diagnostic procedures, and to inform remediation methods.

A major source of confusion in theoretical dyslexia research derives from the different motivations of different researchers. In particular, many applied theoreticians are concerned with educational attainment, and in particular literacy. Consequently, they analyse the different components of reading, investigate the differential effects of various interventions, and often stress (correctly) the need for support for any child who is at risk of reading failure, whether or not he or she is dyslexic. By contrast, pure theorists are interested primarily in the underlying cause(s) of dyslexia (rather than literacy per se), and so they undertake theoretically motivated tests, often in domains not directly related to literacy. We (e.g., Nicolson, 2002) have termed this divergence of perspectives with a similar overall goal the *dyslexia ecosystem*, and we argue that much of the confusion in the dyslexia world derives from this confusion of perspectives. Consequently, it is particularly important to be clear about what one is trying to achieve.

In most areas of science, the distinction between cause, symptoms, and treatment is clearcut; in medicine, for instance, the causes, symptoms, and treatment of malaria are quite different. Indeed, several diseases may have similar symptoms. Influenza and meningitis may lead to symptoms of fever, aching, and nausea similar to those of malaria; but, of course, the underlying causes (and treatments) are quite different. In dyslexia, this distinction is much less clearcut, and it is therefore particularly important to maintain the distinctions between cause, symptom, and treatment. Figure 1.1 shows a schematic of the starting point of our analysis. Our research program was designed to determine the unknowns in this schematic.

Phonological difficulties are certainly an important symptom, but only one symptom. Phonological support is certainly an important aspect of treatment, but it may be only one aspect of treatment. Abnormalities in the language areas of the brain may or may not be the underlying cause of

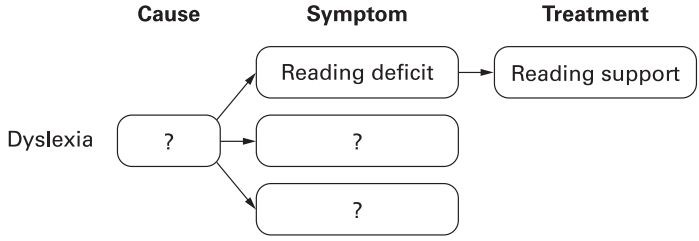

Figure 1.1
Symptoms as cues to the underlying cause(s).

the symptoms. Many possible neurological substrates could lead to the symptoms of poor reading and poor phonology. It may be that in five years it will become clear that dyslexia in fact has several subtypes, each corresponding to abnormality in a different brain region, each leading to phonological difficulties, but also to further and more distinctive symptoms (such as visual difficulties, auditory difficulties, motor difficulties, speed difficulties, etc.). It is likely that these brain-based diagnoses will also reveal commonalities between specific types of dyslexia and other developmental disorders, including attention deficit/hyperactivity disorder (ADHD), specific language impairment, dyspraxia, and generalized learning disability. It may also be that the appropriate treatment for a given child depends critically on the specific underlying cause(s) of their difficulties, rather than just the general reading symptoms displayed.

In particular, if one can identify the underlying cause of a child's potential difficulties *before* they are manifested, it should be possible to give proactive support, to the extent that the child will not fail to learn to read, and will not suffer the concomitant emotional and educational devastation. This, then, is the big applied challenge for pure theorists—to fill in the question marks in figure 1.1. This will facilitate early diagnosis and support for dyslexic children (and other children with special educational needs).

Having made the case for pure theoretical research aimed at identifying the underlying cause(s) of dyslexia, we now turn to the requirements for a causal theory in general, and a causal theory of dyslexia specifically.

1.2 Stages in Scientific Explanation

In this section we outline the general research approaches that have been suggested as good practice in pure science generally. This rather basic schematic (figure 1.2) has been somewhat overlooked in much dyslexia research

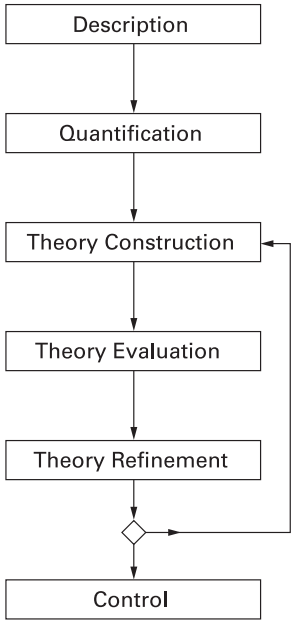

Figure 1.2
Stages in scientific explanation.

(including our initial work), which has led to considerable confusion in the literature.

1.2.1 The Pure Science Model

Figure 1.2 illustrates the standard stages in scientific explanation—description, quantification, theory construction, and control. The two initial stages involve data gathering: first developing a clear description of the phenomena involved, and then developing methods for quantifying them, possibly introducing new technical terms and new measurement devices. Failure to undertake this initial exploratory work may result in *premature specificity*, in which theories are based on incomplete knowledge, and therefore do not cover the full range of phenomena. The next stage involves theory construction, which means inventing an economical characterization of the data to be handled in terms of some underlying regularities. Once constructed, the theory must be tested, in terms of, first, its sufficiency (to explain the known data), and then its ability to make novel predictions that may then be subjected to empirical tests. Those theories whose predictions are confirmed are then worthy of further development

(and the more specific the prediction, or the lower the likelihood of the results being attributable to chance or to other theoretical interpretations, the greater the support for the theory under investigation). Once a theory can make reliable and correct predictions of what will happen under various conditions, the final stage may well be control, that is, manipulating the conditions such that the desired results are obtained. Of course, this bland description of the stages hides the often tortuous and recursive nature of the process. In most areas of scientific endeavor there is usually a period of disconfirmation, when theories' predictions are not supported. This leads to modification and refinement of theories or, in some cases, scientific "revolutions" (Kuhn, 1962), when a completely different perspective is adopted.

As Chomsky (1965) has noted, it is also important to stress the difference between a descriptive theory (such as Mendeleyev's theory of the underlying patterns in the periodic table, or Kepler's theory that the planets travel along ellipsoidal paths around the sun) and a causal, explanatory theory (for the periodic table, the theory of atomic structure; for the planets, Newton's theory of gravitation). While the development of an adequate descriptive theory is often the appropriate initial target, true understanding depends on developing a causal theory that relates the facts to underlying theoretical knowledge. Until recently it has been rather difficult to determine whether or not a given theory should be deemed explanatory, but in a contribution to this rather contentious area of scientific metatheory, Seidenberg (1993b, p. 231) argues that one important requirement for an explanatory theory is that it should "explain phenomena in terms of independently motivated principles." This distinguishes explanatory theories, such as the atomic weights explanation, from ad hoc descriptive theories, such as Mendeleyev's original theory. A further important criterion introduced by Seidenberg (p. 233) is that "an explanatory theory shows how phenomena previously thought to be unrelated actually derive from a common underlying source."

1.2.2 Levels of Explanation in Medicine

We have already highlighted the importance of distinguishing clearly between cause, symptom, and treatment (see figure 1.1). This analysis is normally thought of as a medical model, but of course it is equally applicable, say, to an engineering problem or, specifically, to an educational or psychological issue. Nonetheless, it is worth noting that in medicine and engineering, the expectation is that there is a single cause, and that cause leads

directly to symptoms. In education and psychology, there can be multiple causes, and a primary cause can lead to primary and secondary symptoms. Over time, the symptoms themselves can lead to further symptoms (for instance, in dyslexia, failure at reading may lead to avoidance of reading, and perhaps adoption of some coping strategies that in themselves lead to further difficulties or advantages). Consequently, though valuable, the medical analysis needs to be augmented by further explanatory methods.

1.2.3 Levels of Explanation in the Life Sciences

A related case is well made by Morton and Frith (1995), who distinguish between three levels of explanation—biological, cognitive, and behavioral—with the biological providing the deepest level of explanation (though one needs to add an even deeper level of description, namely, the genetic level). For example, in the case of a patient with amnesia, the behavioral symptoms might be difficulty in remembering events or people's names. At the cognitive level, this might be described as an inability to transfer information from short-term store to long-term memory stores, and at the biological level this might be the result of damage to the hippocampus.

It is important to stress that each level of description has its strengths and its weaknesses. In the amnesia example, description at the behavioral level is useful in terms of identifying the problems suffered by the patient (and hence perhaps the basis for accommodations that address these problems). Very often, the description at the cognitive level is based on administration of sophisticated tests of memory function, thereby allowing much greater precision in describing the problem suffered and possibly pointing toward both cause and treatment. A cognitive-level description, however, does not uniquely identify the biological-level problem—damage to one or more of several brain areas can lead to the same cognitive and behavioral symptoms. Finally, the brain level in some sense gives the "true" underlying problem, but it is important to note that such reductionism does not necessarily help. Because of the interplay between different brain regions, the between-individual differences in brain organization, and the multiple roles each part of each brain region plays, it is difficult to specify precisely the effect even of a clear brain lesion. Unfortunately, with acquired disorders (typically the result of head injury, stroke, or degeneration) damage to several brain regions, and perhaps to the connectivity between regions, is often involved.

Things are even more difficult with developmental disorders (attributable typically to abnormal brain development). It is most likely that brain

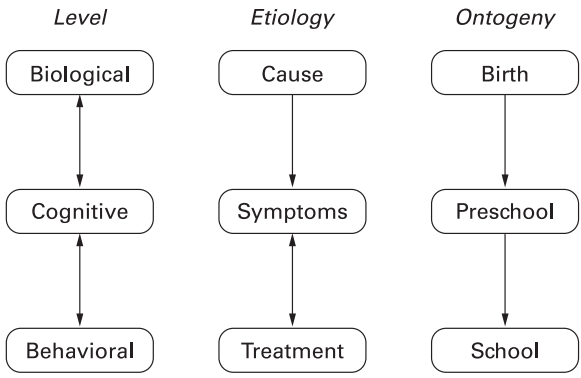

Figure 1.3
Levels of analysis.

regions are just less efficient than normal, rather than nonfunctioning. Furthermore, brain development is driven by the experiences it receives, and so all brains are different. It is currently difficult to be sure what is within the normal range of individual variation, and what is ''abnormal.'' Finally, given the way the brain is designed to achieve important performance targets regardless of its organization, it is likely that performance may apparently be little different from normal. In light of these strengths and weaknesses of each level of description, the wisest approach is to attempt to develop a theory that covers all three levels. In this way, even though no individual finding may be conclusive in itself, the ''converging operations'' provided by a range of findings help us to identify the most likely causes of problems. Note the bidirectionality of the arrows on some of the links between levels (figure 1.3).

1.2.4 Levels of Explanation in the Developmental Sciences

It may be seen that psychologists find a three-level analysis quite attractive. A further such analysis (figure 1.3) is in terms of what is called the *ontogenetic* framework (Waddington, 1966). Put simply, this just means the way that the symptoms develop and change as a function of a child's development. Clearly this developmental framework is valuable for understanding developmental disorders, diagnosing developmental disorders, and supporting children with developmental disorders. It is logically independent of the other frameworks, and we suspect that many researchers have failed to take seriously enough the issue of how the disorder develops.

1.3 Descriptions of Developmental Dyslexia

In the spirit of scientific investigation, then, let us consider how to describe developmental dyslexia. There are several formal definitions.

1.3.1 Definitions of Developmental Dyslexia

Consider the following attempts to define developmental dyslexia:

1. Developmental dyslexia is a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing, and spelling commensurate with their intellectual abilities (World Federation of Neurology, 1968).
2. Developmental dyslexia, or specific reading disability, is defined as an unexpected, specific, and persistent failure to acquire efficient reading skills despite conventional instruction, adequate intelligence, and sociocultural opportunity (American Psychiatric Association [APA], 1994).
3. Developmental dyslexia is a specific language-based disorder of constitutional origin, characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities (Orton Society, 1995).
4. Dyslexia is evident when accurate and fluent word reading and/or spelling develops very incompletely or with great difficulty (Reason [BPS], 1999).
5. The term learning disability refers to a class of specific disorders. They are due to cognitive deficits intrinsic to the individual and are often unexpected in relation to other cognitive abilities. Such disorders result in performance deficits in spite of quality instruction and predict anomalies in the development of adaptive functions having consequences across the life span (U.S. Office for Special Education Programs [USOSEP], 2002).
6. Dyslexia is a specific learning disability that is neurological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede the growth of vocabulary and background knowledge (International Dyslexia Association [IDA], 2002).

It is evident that these definitions are, at best, a compromise. On the one hand, they are not specific enough to allow a definitive diagnosis, whereas

on the other hand, they describe only a symptom of dyslexia—the problem in terms of reading. The original (1968) definition highlights the discrepancy between actual reading performance and expected reading performance. This distinction is abandoned in the 1994 Orton Society definition, which emphasizes the basis in terms of language and phonology. The recent USOSEP definition attempts to pin learning disability at the cognitive level (rather than brain or symptom level) but, like the Orton Society and BPS definitions, it does not explicitly include the concept of discrepancy as a defining characteristic. The IDA definition (2002) does represent a reasonable compromise, broadening the deficits to include fluency, and retaining an element of discrepancy. Nonetheless, it is clear that the definition leaves considerable scope to the interpreter. Perhaps more important, in common with all the other definitions, it makes no attempt to pin down the underlying cause, preferring to leave the ''neurological origin'' unspecified.

We shall return at length to the definition of dyslexia and the issue of discrepancy. For the present, we note the one common factor among these definitions, namely, poor reading. Unfortunately, poor reading is a particularly unsatisfactory criterion from a theoretical perspective, as we discuss in the following section.

1.3.2 Problems with Reading

Reading is arguably the most complex cognitive skill routinely acquired by humans. Unlike language, reading is clearly not innately predetermined and indeed, until the Renaissance, hardly anyone could read at all. Fluent reading requires the blending of a large number of components: semantic knowledge, letter knowledge, phonological knowledge, eye control, and so on. It is a miracle that anyone manages to learn to read, and in a sense it is hardly surprising if anyone has difficulty. Consequently, failure to learn to read could be attributable to a wide range of possible causes, any one of which could lead to ''dyslexia.'' If we take the analogy of pollution, the place to look for pollution is at a confluence of rivers, such as London or New Orleans. Finding evidence of pollution in London is only the first step in identifying the source. One needs to trace back the possible sources until one finds the one (or more) tributaries that carry the pollution, and then trace each tributary back until the point of ingress of pollution is identified. Indeed, in a sense more information is provided by *not* finding pollution in London—it indicates that all the tributaries are unpolluted. Similarly, normal acquisition of reading surely indicates that most of the underlying processes are working fine.

In short, poor reading per se tells us little or nothing about the underlying cause; it is good for screening but not for understanding. Furthermore, unlike our rather simplistic pollution analysis, one can not necessarily identify single ''tributaries.'' Good reading requires the fluent interplay of several cognitive skills, all at high speed. It may well be that problems arise not from an individual skill but in blending different skills.

Second, the absence of poor reading does not necessarily indicate absence of dyslexia. Fortunately, given the appropriate learning environment and enough time, dyslexic children will learn to read adequately. One should beware the danger of concluding, as did one headmaster, in the words of Jean Augur (1991), ''Well, you taught him to read Jean, so he's not dyslexic.'' This flawed conclusion (which is all too prevalent) confuses symptom (poor reading) and cause (dyslexia).

Third, the prevalence of dyslexia in Western school populations is around 5% (Badian, 1984b; Lyon, 1996). Traditionally, roughly four times as many boys as girls were diagnosed. Relaxing the discrepancy criterion, and allowing for potential gender-based referral bias, leads to considerably higher prevalence estimates of 5 to 17.5% and a gender ratio closer to unity (Olson, 2002; Shaywitz, 1998). Given that there are, therefore, around 15 to 50 million dyslexic individuals in the United States and 3 to 10 million in the UK, it seems unlikely that there will be a single underlying cause, convenient though this would be for theorists.

In summary, the study of the cause(s) of dyslexia is fraught with difficulty. Diagnostic criteria are based on symptoms rather than causes, and the primary symptom—poor reading—is a learned skill that is not only very dependent upon the learning environment provided but might also reflect any of a large number of possible underlying causes.

1.4 Applying Theory

If it were not the case that dyslexia is both prevalent and debilitating, a researcher might be excused for choosing a more convenient research area, one not confounded by so many uncontrollable factors.

In persevering, we were inspired by the approach of the late Donald Broadbent, the foremost British cognitive psychologist of his time, who extolled the virtues of doing ''real world'' applied theoretical research. Broadbent argued that the world ''kept one honest'' (Broadbent, 1973). Applying theory in the real world mercilessly exposes its limitations!

Moreover, there was undoubtedly work to do. Certainly when we first started investigating dyslexia, the educational system was such that

dyslexia could not be diagnosed formally (and hence a dyslexic child not given special help) before the appropriate discrepancy criterion (typically an 18-month discrepancy between reading age and chronological age) was reached. In practice, this meant that a child had to be over 7 years old before diagnosis. He or she had to fail at reading for the first two, crucial, years of school before support was available. This failure was corrosive and cumulative, scarring psyche and stunting skill.

Clearly any theory based solely on analyses of reading could not, even in principle, address this ''catch-22.'' By contrast, a causal theory, one that was able to predict the precursors of dyslexia, would lead to the identification of potential problems before a child started to learn to read, allowing proactive support, and avoiding reading failure—even if the child were ''really'' dyslexic.

1.5 Our Agenda for Dyslexia Research

Early in our research program, we were commissioned to prepare a report on how best to diagnose dyslexia in adults. This proved to be an outstanding learning opportunity for us. We first interviewed 12 acknowledged UK dyslexia experts—theorists and practitioners—as to their views on how this should be done. Based on the rich interview transcripts we devised a questionnaire, which was then circulated to all those in Britain and internationally whom we knew to be in the area. This was, and as far as we know still is, the only systematic international survey of this type that has been undertaken. This led to a very clear set of recommendations (Nicolson, Fawcett, & Miles, 1993). However, for our purposes here, having to make sense of the rich mix of practical, diagnostic, and theoretical views that we obtained turned out to be pivotal for us, and has informed all our subsequent work. In particular, we realized that for cumulative progress to be made it was vital for researchers, diagnosticians, and practitioners to work in collaboration rather than independently. A viable strategy has to see the system as a whole and to be ''joined up'' so that each component works at its own problems but in the context of an overall blueprint (see Nicolson, 2002, for an article that expands this idea).

The blueprint we developed is shown in figure 1.4. Bearing in mind the dangers of premature theoretical specificity (see figure 1.2), the schematic is designed to be pragmatic rather than theory-bound, and explicitly includes the need for cost-effectiveness as well as effectiveness. It is heartening that subsequent UK approaches (UK Department for Education, 1994) adopted

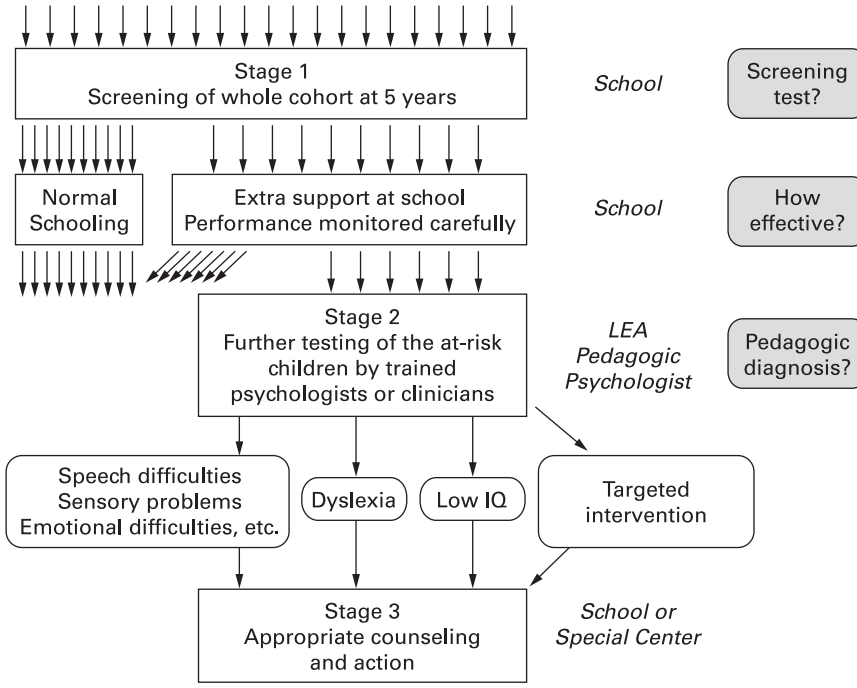

Figure 1.4
A dyslexia blueprint.

a similar stages approach. Furthermore, in a move to implement IDEA (2004) in the United States, simplification of the identification process is advocated and the use of interventions based on scientific evidence, within a three-tier model of screening, intervention, and diagnosis similar to the UK model. The key concept here is responsiveness to intervention, which is advocated to inform the delivery of more effective intervention targeted to the profile of needs, in order to focus finance on higher need children. This approach is based on a dual discrepancy model, in which children with poor home backgrounds are predicted to respond relatively quickly to intervention, by contrast with children with dyslexia whose problems are more entrenched.

First, we proposed that the entire cohort of children be screened on school entry. Clearly for this to be cost-effective, a screening test needed to be developed that was quick, fun, and predictive and could be administered at low cost. Our Dyslexia Early Screening Test (Nicolson & Fawcett, 1996) was the first such published test.

Second, those picked out as at risk in the screening need to be given immediate and proactive support by the classroom teacher and support staff. This will allow probably the majority of the risk cohort to catch up with their peers. The remainder of the risk cohort will need further support. Clearly these children will suffer diverse difficulties, ranging from extreme deprivation through dyslexia, dyspraxia, specific language impairment, autism, and attention deficit to sensory impairment or physical disability to psychiatric issues. It is unlikely that ''one size will fit all'' for such children, and consequently an individual pedagogic diagnosis would need to be made for each child, to carefully determine the underlying problems and lead to the development of a support structure specially tailored to the specific range of learning abilities and disabilities.

Much of the necessary infrastructure (if not the funding) for such a system is in place in many Western countries, but we considered that there was a critical absence of educational pedagogic theory allowing for a detailed diagnostic approach designed to engage with the subsequent support regime. Consequently, some of our research (Fawcett, Nicolson, Moss, Nicolson, & Reason, 2001; Lynch, Fawcett, & Nicolson, 2000; Nicolson, Fawcett, Moss, & Nicolson, 1999) addressed these issues.

From a theoretical perspective, however, it is clear that in 1990 there were critical gaps in knowledge at every stage in this procedure. If one wishes to identify children at risk of reading failure before they fail, it is necessary to have a screening test capable of picking them out. For this one also needs theoretical knowledge of the precursors of reading difficulty. Furthermore, from the perspective of providing high-quality support, it is necessary to know what is the best support to give to which type of educational need. For this, one needs theory. The major limitation to progress in the UK (and internationally) had been the failure to blend theory and practice in designing pragmatic and effective support systems.[1] This task provided the underpinning of our research program.

1.6 Six Questions for Dyslexia Research

It is understandable, given the diversity of approaches in dyslexia theory and practice, if one begins to lose focus on what one is trying to achieve in dyslexia research. Certainly, if one is unclear about the research objectives, one is unlikely to achieve them! In view of the value of a set of focus-

1. This failure has now been very fully addressed (see McCardle & Chhabra, 2004; NICHD, 2000, for comprehensive reviews).

ing objectives, our research has been focused on the following six questions for dyslexia research:

Question 1 What is dyslexia?
This is surely the fundamental question. It is clear from the preceding definitions that as yet no satisfactory definition or diagnostic method exists. We hope by the end of our research to have made some progress toward an acceptable analysis.
Question 2 What is the underlying cause?
Ideally, an explanation should be grounded not only in the medical levels, but also the life science levels and the developmental levels of explanation (see figure 1.3).
Question 3 Why does it appear specific to reading?
This issue was highlighted by Morrison and Manis (1983). These authors suggested that any viable theory must address four issues: why does the deficit affect primarily the task of reading—later described by Stanovich (1988b) as the *specificity principle*; why do dyslexic children perform adequately on other tasks; what is the mechanism by which the deficit results in the reading problems; and what is the direction of causality?
Question 4 Why are some dyslexic people high achievers?
This issue is perhaps less central, but is nonetheless crucial to an understanding of the fundamental enigma of dyslexia—how can an otherwise high-achieving person be so impaired in learning to read? It also forces one to confront the issue of whether an explanation differentiates between dyslexia and other learning disabilities.
Question 5 How can we identify dyslexia before a child fails to learn to read?
There is now extremely clear evidence that the earlier one intervenes in helping a child learn to read, the more effective (and cost-effective) the intervention is (with many different interventions apparently being effective). Replacement of the ''wait-to-fail'' diagnostic method is arguably the central applied issue.
Question 6 Do we need different methods to teach dyslexic children? If so, what?
Finding a principled linkage between diagnosis and support is, in our view, the second (and currently unresolved) applied issue. Even modest progress toward this goal would transform the opportunities available for the next generation of dyslexic children.

Our attempts to address these questions provide the backbone for the remainder of the book.

1.7 Organization of the Remainder of the Book

Following this lengthy preamble, we are in position to look beyond the apparent diversity of the field, to classify the different theoretical approaches to explaining dyslexia, to present the rationale for our long-term research program, and to move toward the longer-term goals of the discipline.

Our initial, and overriding, priority was to address question 2, the theoretical investigation of the underlying cause(s). We start by giving a succinct but wide-ranging overview of all the major theoretical approaches to dyslexia. We have attempted to undertake this task in an even-handed fashion, outlining the evidence in favor of each approach. Given the centrality of the phonological deficit framework in modern dyslexia theory and practice, we devote considerable analysis to the framework, concluding that it has provided outstanding coherence to theoretical, applied, and political initiatives, but we need to dig deeper in order to understand *why* there are phonological deficits and, indeed, why deficits appear to exist outside the phonological domain.

Following this overview of the extant theories, we provide a brief overview of the literature on reading and learning to read (chapter 3). We then present our own approaches to the issues in order of their developmental progression. While these are directly compatible with the phonological deficit framework, they provide a very different explanation of the range of problems and their causes. We start with a cognitive level analysis, which resulted in our automatization deficit hypothesis (chapter 4). In a range of investigations, automatization deficit provided a remarkable fit to the wide range of data on dyslexia. Nonetheless, it failed to give a principled explanation of some aspects of procedural learning, in particular the fact that problems appeared early as well as late in learning. In particular, we concluded that lack of automaticity was a generic feature (symptom) of dyslexia, but the problems do not arise solely in the process of automatization. This led us to question the ability of a purely cognitive-level analysis to explain the underlying problems in dyslexia.

We then turned to a brain-based explanation in terms of cerebellar abnormality (chapter 5). This hypothesis is supported by a range of further investigations, revealing deficits that had not hitherto been investigated, and providing a coherent brain-based explanation for automatization problems. Third, we developed a novel ''ontogenetic causal chain'' aimed at speculating how a cerebellar abnormality at birth would lead through childhood to the problems known to be associated with dyslexia. The analysis provides one possible route from the basic level of biology, through the

cognitive levels of automaticity and phonology, to an explanation of why dyslexic children have problems in learning to read, our most spectacular cognitive/motor skill (chapter 6). This semi-historical treatment of the development of the cerebellar/automatization deficit framework is followed by a reflective chapter, in which we analyse the strengths, weaknesses, and limitations of the framework, taking into account developments in genetics, neuroscience, and dyslexia in recent years—developments that have confirmed its fundamental tenets (chapter 7).

Despite these successes, we were mindful of the likelihood that not all dyslexic people suffered from cerebellar problems, and that apparent problems in cerebellar function might alternatively be attributable to interactions within the brain circuits that characterize cerebellar involvement rather than the cerebellum itself. Furthermore, independent research on other learning disabilities suggested a surprising overlap in symptoms with those of dyslexia, both at the automaticity and cerebellar levels. This led us to investigate the neural systems level—a level intermediate between brain and cognition—which might provide a perspective from which these various accounts cohere. In an integrative approach, we propose that dyslexia may be seen as a specific deficit in the procedural learning system (as opposed to the declarative memory system). This specific procedural learning difficulties (SPLD) framework is speculative, and not yet supported by the extensive evidence that underpins the automatization and cerebellar deficit hypotheses. Nonetheless, SPLD provides a novel answer to the key question of what is dyslexia and provides a potentially fruitful perspective on the entire range of learning disabilities. In the final chapter, we sketch out how future research can lead to further progress, theoretical and applied, in this and other domains.

For readers with little time, we hope that the summary of the major theories of dyslexia in chapter 2, followed by the summary of current research presented in chapter 7, together with the two subsequent chapters, will prove sufficiently thought-provoking to justify the analysis found in the remainder of the book—the foundation on which our conclusions rest.