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Teaching Literacy to Learners with Dyslexia

A Multisensory Approach

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CHAPTER 1

THE CONTRIBUTION OF DYSLEXIA RESEARCH TO THE DEVELOPMENT OF A MULTISENSORY TEACHING PROGRAMME

Chapter Overview

At the time of writing there is a general acceptance that dyslexia exists (Rose, 2009) and while there is still no universally-accepted definition there is substantial agreement about its characteristics. Current research into the nature of dyslexia shows many areas of commonality as well as some unresolved and contentious issues. This chapter presents a review of relevant research which offers both explanations of the nature of dyslexia and a rationale for a multisensory programme.

Definitions of Dyslexia

A good definition should reflect research evidence, be useful in identifying and assessing learners with dyslexia and provide a basis for intervention. Currently the most widely used definition of dyslexia in England is that proposed in the Rose Report (2009: 30):

- Dyslexia is a learning difficulty that primarily affects the skills involved in accurate and fluent word reading and spelling.
- Characteristic features of dyslexia are difficulties in phonological awareness, verbal memory, and verbal processing speed.
- Dyslexia occurs across a range of intellectual abilities.
- It is best thought of as a continuum and not as a distinct category and there are no clear cut-off points.
- Co-occurring difficulties may be seen in aspects of language, motor co-ordination, mental calculation, concentration and personal organisation, but they are not, by themselves, markers of dyslexia.
- A good indication of the severity and persistence of dyslexic difficulties can be gained by examining how the individual responds or has responded to well-founded intervention.

As a working definition it provides a useful starting point for consideration of the nature of intervention. It emphasizes phonological difficulties, processing speed and verbal memory which have been found to be fundamental impairments associated with dyslexia across languages, as discussed later. However, a weakness is that it does not acknowledge the fact that some learners with dyslexia also have visual processing difficulties, which has led the British Dyslexia Association (BDA, 2011) to add:

> In addition to these characteristics, the BDA acknowledges the visual processing difficulties that some individuals with dyslexia can experience, and points out that dyslexic readers can show a combination of abilities and difficulties that affect the learning process. Some also have strengths in other areas, such as design, problem solving, creative skills, interactive skills and oral skills. (www.bdadyslexia.org.uk)

Rose suggested that Response to Intervention (RTI) could be used to indicate severity and persistence but emphasised intervention should be 'well founded'. The model of RTI, which is widely used in the USA, is recommended in the Special Educational Needs and Disability (SEND) Code of Practice of 2014 (DfE, 2015). Although potentially this could lead to a delay in the assessment of a learners' difficulties the SEND Code specifically notes that where any difficulty appears severe referral should not be delayed. In the case of dyslexia, 'well founded' intervention is normally considered to be structured, sequential, cumulative, multisensory and based on synthetic phonics (as discussed in Chapter 8).

Some earlier definitions attempted to distinguish dyslexia from other forms of reading difficulty by 'ruling out' (excluding) reasons which might otherwise account for those difficulties, such as sensory impairments, additional language learning, the lack of good, conventional and consistent teaching and factors associated with socio-cultural opportunities. It is now accepted that dyslexia can exist alongside these other factors whilst recognising that such factors may contribute to

literacy difficulties. For many years a definition of dyslexia was based on a discrepancy between general 'ability' scores and predicted reading level as this suggests a 'specific' difficulty. An example is given by Selikovitz who defined a specific learning difficulty as:

> an unexpected and unexplained condition, occurring in a child of average or above intelligence, characterised by a significant delay in one or more areas of learning. (1994: 4)

Rose (2009) acknowledges that dyslexia can occur across all ability levels. At the time of writing, screening and assessment for dyslexia still includes verbal and non-verbal reasoning (see Phillips et al., 2013) together with other tests of cognitive processing. However, the overall profile of scores is considered and variations in strengths and difficulties are looked for (a spiky profile) rather than a discrepancy between 'underlying ability' and literacy scores. That type of discrepancy has been heavily criticised for a number of years on the grounds that there is no qualitative difference in the pattern of reading errors between learners with low and high IQs (Stanovich, 1996). Moreover, Badian (1994: 45) concluded in a study of children aged 6–10 that it is possible to distinguish poor readers with dyslexia from other poor readers not on the basis of IQ but by cognitive profiling. Phonological difficulties were found in both types of poor readers (although more severe in those with dyslexia), but those with dyslexia also had 'unique' deficits in both 'automatic visual recognition and phonological recoding of graphic stimuli'. (Nevertheless, some researchers still maintain that the IQ/literacy attainment discrepancy is important in distinguishing the two types of poor readers e.g., O'Brien et al., 2012; Stein, 2015.)

There are many who would not use a discrepancy definition based on IQ, but would still assess learners whose literacy skills are very poor when compared with their oral contributions in lessons to see whether or not they are dyslexic. (Another way of expressing this would be to say that poor literacy is 'unexpected'.)

Research into the nature, causation and prevalence (or incidence) of dyslexia varies according to the definition (criteria) and conceptual framework used by the researchers.

Incidence

Incidence figures can provide valuable information for policy makers, schools, and local authorities when planning teaching and resource allocation. However, as the definitions and criteria from which the figures are derived vary, any statistics about prevalence should be examined carefully. Figures differ according to the definition used, the assessment procedures involved, and the cut-off points in relation to severity. As dyslexia is considered to be a continuum, it is important to note exactly

which methods and criteria have been used in any study of prevalence. One consequence of adopting exclusionary criteria in defining dyslexia, for example, was that researchers often eliminated learners who were of below average ability or from socially disadvantaged backgrounds. A further issue is whether learners who may have more than one co-existing specific difficulty have been included or excluded from a study.

The figures quoted vary from country to country and even within a country. In a seminal work Rutter and Yule (1975) reported 3.9 per cent with specific reading difficulties in the Isle of Wight, but using the same methods and criteria found 9.9 per cent in London. More recently, Chan et al. (2008) described two studies in China, where one suggested that dyslexia affected less than 1 per cent of the population whereas the other suggested about 10 per cent. However, different criteria were employed in these two studies. The BDA (2015) suggests that about 10 per cent of the UK population may be dyslexic with about 4 per cent severely affected. This implies that there could be one learner with severe dyslexia and two with moderate dyslexia in any class of 25. Recent studies to investigate reading difficulties in both England and the USA do not always specify whether the children studied have been identified as having dyslexia. Screening undertaken as part of the 'No to Failure' Project in England (Dyslexia–SpLD Trust, 2009) found that 21 per cent of children of primary school age may have literacy difficulties which could be described as dyslexia. This is similar to the figures reported in the USA (Shaywitz et al., 2008), although these may include other 'poor readers'.

Snowling (2008) points out that dyslexia incidence figures may vary according to the age of learners because they might present different patterns of problems at different ages. She also suggests that while word-decoding problems predominate at primary school age, during adolescence and adulthood many people with dyslexia will have learned to read but will still have problems with spelling and/or writing.

Where literacy difficulties are taken as major indicators of dyslexia, international studies often reflect that the transparency of the orthography of a language can affect learners' ease or difficulty in acquiring literacy (see Chapters 3 and 7).

There also appear to be gender differences, as more boys than girls (about 4:1 according to the BDA) are identified as having dyslexia. This difference may be due to genetic factors but could be related to the process of referral. Shaywitz et al. (2008), reporting on an earlier study, suggest that schools may refer more boys for assessment because of disruptive classroom behaviour, whereas if girls are less disruptive they may not be referred. Their hypothesis may be supported by Singleton (1999) who reported that although more boys than girls were identified as dyslexic when at school, in higher education more female students than males are identified as dyslexic for the first time. Pennington and Olson (2005: 472), summarising genetic research, concluded that genetic influences on dyslexia 'operate similarly in both males and females'.

Implications for Practice

- Given the incidence of dyslexia there should be whole school procedures in place for the identification and assessment of literacy difficulties.
- It is important not to assume that dyslexia is more commonly found in males.
- A 'checklist' for screening is a useful first step and it is worthwhile remembering that characteristics/observable behaviours may be different at different ages.

Definitions of dyslexia reflect both observed behaviours and research into the causes of those behaviours. Research into the causation of dyslexia provides insights into understanding the difficulties of learners with dyslexia and also provides bases for devising appropriate intervention strategies.

Theories of Causation

Research and models of dyslexia underpin the way in which we identify dyslexia and establish a rationale for methods of intervention. In analysing the research into and theories about the nature of dyslexia we find the Morton and Frith (1995) causal modelling framework helpful. This framework was designed to study psychological and learning behaviours such as dyslexia. It involves a consideration of three levels of description:

- biological;
- cognitive;
- behavioural.

All of these are influenced/affected by the environment as discussed later.

A report from the British Psychological Society (BPS, 1999) used the framework to present ten different theoretical explanations of dyslexia so that similarities and differences could be identified. We have used the framework to provide an overview of the three main theories (magnocellular, cerebellar, phonological) producing a model to show how they may be inter-related rather than discrete approaches. We did this because although the three theories offer an explanation of the core deficits, no single theory can explain the full range of difficulties experienced by learners with dyslexia. This integrated model (see Figure 1.2 on p. 26) is therefore compatible with current thinking and research into multiple deficits and dyslexia (see Pennington, 2006; Pennington et al., 2012; van Bergen et al., 2014) and forms the basis for the teaching programme in Part III.

Genetic and neuroscientific research has highlighted the complexity of the nature of dyslexia and moved from single deficit theories to a multiple deficit model (MDM). Two research-based multiple deficit models are also considered within this chapter: Pennington's cognitive MDM (2006) and the intergenerational MDM (van Bergen et al., 2014).

Discussion of Theories of Causation of Dyslexia using Morton and Frith's Framework

In the following discussion we have divided the biological level of Morton and Frith's framework into genetic and neurobiological research because studies often focus on one aspect or the other.

Biological Level

(A) Genetic factors

Long-held theories that dyslexia is largely inherited have been substantiated as a result of medical and technical advances. Pennington and Gilger (1996) have claimed that up to 65 per cent of the children with dyslexic parents and 40 per cent of the siblings of a child with dyslexia will also have the condition. Debate in the last decade has focused on which genes are involved and associated phenotypes. There is general agreement that dyslexia is unlikely to be related to only one gene and also that the genetic loci may not influence dyslexia per se but affect the skills which underpin dyslexic characteristics (Pennington and Olson, 2005).

Research has identified that chromosomes 1, 2, 3, 6, 11, 15, and 18 show a genetic linkage to dyslexia with the possible main site being on chromosome 6 (Grigorenko, 2005; Schumacher et al., 2007) where genes have been linked to particular types of phonological processing difficulties, with the site 6p being linked to phonological decoding (Grigorenko et al., 2000; Francks et al., 2004) and oral reading of non-words (Kaplan et al., 2002) and site 6q to phonological awareness (Petryshen et al., 2001). However, Stein (2008, 2015) has linked the gene KIAA 0319 on chromosome 6 to the control of neuronal migration in utero and proposes that in dyslexia this process is disrupted resulting in ectopias and impaired development of magnocellular neurones (see later discussion). Schumacher et al. (2007) also discuss three studies linking chromosome 1 to phonological aspects of dyslexia. Gilger (2008), in an overview of genetic research and dyslexia, refers to work by Francks and colleagues in 2002 (who found an association between chromosome 2 and phonological awareness and single word reading) and that of Olson in 2006 (linking a gene on chromosome 15 to disrupted auditory processing). Gilger also cites work by Taipale et al. in 2003, suggesting a link between 15q and spelling, and research by Chapman et al. in 2004 showing a link between

chromosome 15 and single word reading, although the findings about chromosome 15 have not always been replicated. A number of other studies have found links to other, less-well-researched chromosomes and a genetic link to dyslexia is no longer disputed.

There is a general consensus that a polygenetic view should be taken and that the genetic loci for dyslexia should be conceptualised as 'susceptibility loci' or placing a child 'at risk'. It is important to recognise that having parents with dyslexia does not necessarily mean that children will have dyslexia. (For discussion, see Pennington and Olson, 2005; Snowling et al., 2007.) It is also acknowledged that environmental factors may affect the foetus (as well as the post-natal environment). Olson and Byrne (2005) suggested that at least 50 per cent of the variance could be explained by genetic factors and the remainder by environmental factors. Snowling (2013) supports the view that dyslexia is the outcome of multiple risk factors both genetic and environmental and also Van Bergen et al. (2014) suggest that we should consider the cognitive profile children inherit from their parents.

Reid (2009) points out that 'dyslexic genes' on chromosome 6 are in the same region as the genes implicated in auto-immune diseases that also show a high level of association with dyslexia. Stein (2008), referring to his work in 2001, suggests that the Major Histocompatibility Complex (MHC) system is responsible for producing antibodies and seems to control the development of magnocells which are particularly vulnerable to environmental factors such as drugs and disease, noting the high incidence of auto-immune problems such as asthma, eczema, and hay fever in 'poor readers'. An impairment in the MHC system offers one explanation for a magnocellular deficit, which is discussed below.

The wide range of difficulties experienced by learners with dyslexia, together with the range of degree of severity, can be explained by the genetic make-up of an individual together with the antenatal influence of environmental factors. The characteristics presented at school will further reflect the effects of environmental factors since birth, both at home and at school.

Implications for Practice

- Children who have a parent/sibling who is dyslexic may be considered 'at risk' and should be observed carefully for early signs of phonological/reading difficulties and other signs of dyslexia.
- Teachers should be aware that a number of compensatory factors (including those in a child's social environment) might mean that a child who has inherited the gene(s) does not necessarily develop literacy difficulties.
- There is also a need to consider the school/learning context in relation to home factors, as the learning context may present unfamiliar challenges to children and young people and therefore be a source of 'barriers to learning'.

(B) Neurobiological basis of dyslexia

(i) Hemispheric differences

Technological developments – in particular positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) – have advanced research into brain-based neurobiological theories of causation. In the last twenty years these developments have provided evidence of the areas of the brain involved in the reading process and have shown that there are differences in the systems used by children with dyslexia.

The cerebral cortex comprises four lobes, as illustrated in Figure 1.1. Three neural systems considered to be critical for accurate, fluent reading are found in the left hemisphere. These are two posterior systems; one in the parieto-temporal lobe (involved in word analysis) and the other in the occipito-temporal lobe – the Visual Word Form Area (VWFA) (involved in rapid word identification) which integrates phonology and print. There is one anterior system in the inferior frontal gyrus, known as Broca's area, which is involved in articulation/speech and word analysis.

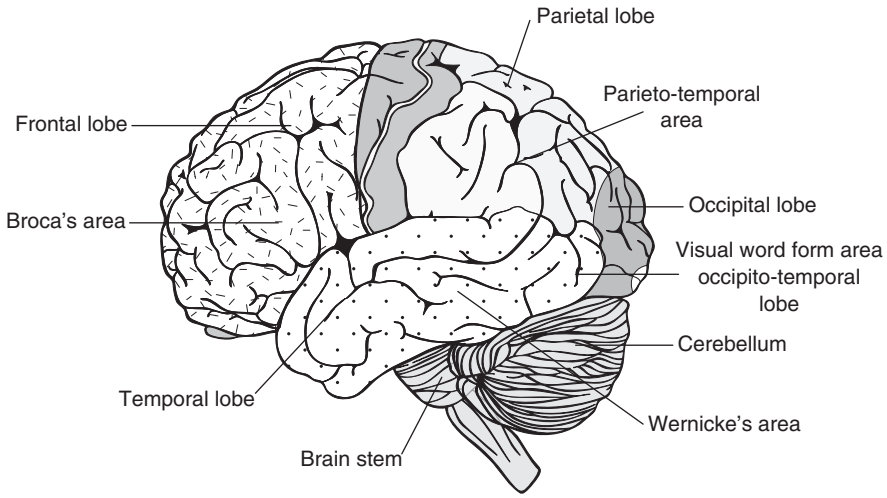

Figure 1.1 *Reading and the Brain*

Shaywitz (2003) and Shaywitz et al. (2007) and other researchers using fMRI have found under-activation of the left occipito-temporal area in readers with dyslexia compared with 'normal' readers. This under-activation of the left posterior system has been described as the neural signature of dyslexia (Shaywitz, 2003). However, children with dyslexia show more activation in Broca's area and in the right frontal lobe and right occipito-temporal regions. She claims this leads to accurate but very slow reading involving sounding out strategies. This view is supported by Stein (2008) who states

that activation in Broca's area becomes inversely related to reading ability and therefore it is more active in dyslexics.

Breznitz's (2008) research into word decoding noted the same hemispheric differences and emphasised speed of processing (SOP) as a highly significant factor in explaining dyslexia. She and her colleagues considered that the visuo-graphic system (right hemisphere) processes information holistically whereas the auditory-phonological system (left hemisphere) processes information sequentially and these differences will affect speed and fluency. Breznitz claims that learners with dyslexia have difficulties transferring information from one hemisphere to the other and proposes the 'Asynchrony Phenomenon' as an explanation of dyslexia. She refers to research which investigated information transfer between the left and right hemispheres among dyslexic compared with normal readers. This showed that information arrived in the right hemisphere first for students with dyslexia and was then transferred to the left, whereas for normal readers information arrived in the left hemisphere first and was then transferred to the right and in about half the time (measured in milliseconds) that the transfer took for readers with dyslexia.

Breznitz's work supports that of others (e.g., Wolf and Bowers, 1999) in claiming that speed of processing is a fundamental factor in dyslexia.

A more recent study by Finn et al. (2014) examined the connections used during reading text. Their study supported earlier findings that in readers with dyslexia there was reduced connectivity within the VWFA and stronger connection made in anterior language areas around Broca's area when compared with non-impaired readers. They confirmed that learners with dyslexia have some difficulty integrating visual information and modulating attention to visual stimuli. They concluded that activity in visual pathways was better synchronised in non-impaired readers and that reduced synchrony might be more important than magnitude of activation. As in earlier studies, they found 'young' learners with dyslexia had increased connectivity in the right hemisphere but also found that older learners do achieve connectivity in the left posterior systems although it develops at a slower rate and to less degree than in normal readers. This study supports an earlier study by Shaywitz (2003) which showed that effective reading intervention (over a one-year period) produced shifts in activation from the right hemisphere to the left posterior system and that these changes in activation were even more marked a year later. This provides some evidence of plasticity in the brain. It would support the claim that structured, sequential, multisensory teaching can achieve its aim to develop new neural pathways and/or strengthen existing neural pathways in the left hemisphere in order to encourage accurate and fluent reading.

(ii) Magnocellular deficit

Stein (2001a, 2008) suggested that dyslexia is largely the result of abnormalities in the neural pathways of the visual system which is divided into two areas – the parvocellular and magnocellular systems. Parvocells are used to distinguish fine detail e.g., distinguish letters and colours. Magnocells form a direct link between the lateral

geniculate nucleus (LGN) of the thalamus and the retina and are very sensitive to rapidly changing visual stimuli. Reading involves the rapid visual identification of letters and words in the correct order and this requires the eyes to flick from one word to the next. Stein (2001a, 2008) argued that the visual system is the most crucial to reading and therefore impairment in the visual magnocellular system is the major cause of dyslexia. He claims (2015) that 75 per cent of problems with reading (including dyslexia) are visual and related to poor linear sequencing. This view has been strongly challenged by those who consider that it is phonological processing that is the major cause (e.g., Vellutino et al., 2004; Goswami, 2014).

The magnocellular deficit hypothesis also proposes that problems in the visual magnocellular system result in binocular instability and visual perceptual instability (Stein, 2001a, 2001b) due to a reduced ability to detect rapidly changing visual stimuli as the eye scans print (Evans, 2001; Stein, 2008). This can result in visual stress and sensory integration problems (see Everatt, 2002; White et al., 2006) where letters appear to blur or move about when trying to read, creating difficulty in determining the order of letters in words and hence a lack of reading fluency. (See also earlier work by Pavlides, 1990; Irlen, 1991.) Signs of visual stress may include headaches, eye strain, poor concentration, tracking difficulties, words or lines omitted when reading or copying text, difficulty remembering what has been read, and poor concentration (Jordan, 2006). Stein (2015) suggests that about 50 per cent of children with dyslexia have a weak magnocellular system causing unstable vision and that the use of coloured filters can alleviate these visual disturbances. His studies suggest that an equal percentage of children with visual stress are helped by yellow and blue filters and use of these improves reading scores. Rose (2009), however, pointed out that many non-dyslexic people also experience visual stress and this should not in itself be seen as a characteristic of dyslexia (a view supported by recent research; see Kranich and Lupfer, 2014). Creavin et al. (2015) claim that the proportion of children with 'minor ophthalmic abnormalities' is not significantly higher in those with dyslexia compared to the normal population, based on a study of 172 seven to nine year olds. Another study by Cicchini et al. (2015) found that people with dyslexia who have an altered copy of gene DCDC2 were unable to detect certain types of visual motion but that not all dyslexics have this mutated gene. These studies confirm the view that dyslexia is complex and multi-factorial.

The magnocellular pathway in the auditory system is not as well researched but has a set of large auditory neurons that detect changes in the frequency and amplitude of sounds. Reading requires fast and accurate processing of both visual and auditory stimuli and the magnocellular deficit theory proposes that readers with dyslexia have lower sensitivity to both visual and auditory stimuli than normal readers due to impaired development of the large neurons (Stein and Talcott, 1999). This claim was supported by post-mortem research (e.g., Livingstone et al., 1991) and by brain imaging studies (Stein et al., 2001) which found magnocells in the deep layers of the visual thalamic nucleus (Lateral Geniculate Nucleus) were disordered and overall smaller

than their normal size. Impairments in magnocellular systems could be due to genetic factors but may also be the result of possible deficiencies in Omega 3, as magnocells need this to maintain flexibility in the membrane surrounding the cells and to function efficiently (Stein, 2008, 2015).

The magnocellular deficit hypothesis suggests that phonological deficits could be a result of poor temporal processing in the magnocellular system. Phonemic awareness seems to depend on the ability to track changes in both sound frequency and amplitude (Stein et al., 2001; Stein, 2008). Stein suggests that subtle auditory processing impairment in learners with dyslexia may reduce sensitivity to changes in sound frequency e.g., for /b/ frequency ascends and /d/ frequency descends (Stein, 2015). Difficulty in processing rapidly changing stimuli in the auditory pathways of the magnocellular system can compromise phonological awareness and memory storage and result in a slower work rate (Tallal, 2007; Valeo, 2008).

At a behavioural level this suggests why many phonemes (speech sounds) (e.g., /b/, /t/, /k/, /d/) may not be distinguished, thereby affecting both reading and spelling. Similarly, poor visual perceptual processing may cause letters to be mis-sequenced, transposed, or blurred.

Stein (2008) argues that the differences in the visual, auditory, phonological, kinaesthetic, sequencing, motor and memory difficulties found in learners with dyslexia are the result of differences in the particular magnocellular systems they have inherited.

Implications for Practice

- This theory points to the need to address *all* behavioural characteristics and both visual and auditory processing should be considered when assessing and teaching learners with dyslexia.
- The use of coloured overlays or lenses may alleviate visual stress in some learners with dyslexia. However, it should not be assumed that they will no longer have any reading difficulties.
- It provides a justification for integrating auditory and visual approaches in teaching.
- It points to the need to make grapheme–phoneme linkages clear, using overlearning to ensure they can be rapidly distinguished (e.g., through the use of structured routines).

(iii) Cerebellar deficit

The cerebellum at the back of the brain (below the cortex) is also divided into two hemispheres (left and right) which receive input from all the senses, the primary motor cortex and many other areas of the cerebral cortex. It is particularly important

for balance, automatisation of motor skills, is involved in language, and acts in conjunction with other brain regions to optimise the functioning of the skill that is controlled by that other region (Fawcett and Nicolson, 2008). They consider that the cerebellar deficit theory may provide a useful hypothesis to explain the literacy difficulties found in dyslexia although they do not claim that *only* the cerebellum is affected in dyslexia. Their research in the early 1990s challenged the phonological deficit theory by identifying a range of non-phonological deficits in dyslexia. Their early experiments suggested that when engaged in balancing tasks, learners with dyslexia could balance as well as those without dyslexia, but when asked to undertake two tasks simultaneously (e.g., count and balance at the same time) they would have difficulty balancing because they could not concentrate sufficiently. Fawcett and Nicolson suggested that poor skill automatisation could result in difficulty in learning to read and problems in multi-tasking and poor motor planning – and might also lead to handwriting difficulties and appearing clumsy and unco-ordinated. (This theory has been challenged by those who would argue that these are indicators of dyspraxia – or developmental co-ordination disorder – and other co-existing difficulties.)

Since the 1990s, their research has concentrated less on the theory related to balance and motor skills and more on automaticity deficit (which can be seen as a cognitive processing deficit). The automaticity deficit hypothesis suggests that a dysfunction of the cerebellum leads to a lack of fluency in skills that should be automatic, such as letter–sound recognition and the motor co-ordination needed for articulation, balance, and handwriting (Nicolson and Fawcett, 2008). They also argue that the cerebellum is central for 'language dexterity' and speech, including verbal memory. Differences in the size and structure of the cerebellum have been found in people with dyslexia compared with non-dyslexics using magnetic resonance spectroscopy (Rae, 2001). Rae suggests that the larger left cerebellum found in people with dyslexia leads to slower information processing (and slower reading). The volume of the left cerebellum is larger but the number of neurones is less and more spread out in people with dyslexia, resulting in greater difficulties in making connections. Differences in both cerebellar asymmetry and gray matter volume are acknowledged as being some of the most consistent structural brain findings in dyslexic compared with good readers (Stoodley and Stein, 2013). They point out, however, that poor performance on cerebellar motor tasks, including eye movements, postural stability and motor tasks, have been found in some but not all children and adults with dyslexia. They argue that impaired cerebella cannot be seen as the primary cause of dyslexia, reminding us that differences in the dyslexic brain are found throughout 'the whole reading network' and not just the cerebellum.

Norton et al. (2014) report that reduced activation in the right cerebellar lobule VI correlates with a deficit in Rapid Automatised Naming (RAN). Children with a

double deficit (rapid naming and phonological awareness) showed even lower activation of the right cerebellum than those with just a single deficit in rapid naming. This suggests that a significant number of learners with dyslexia do have a cerebellar deficit.

Nicolson and Fawcett (2000) proposed a 'square root rule' to indicate the number of repetitions needed for a child with dyslexia to learn a complex task compared with one without dyslexia. This implies that if acquiring a skill normally requires, say, 900 repetitions, a child with dyslexia might take 30 (the square root of 900) times as many (i.e., 27,000). Whilst we would advise that this 'rule' should not be applied rigidly (bearing in mind the range of individual differences in learners with dyslexia), it does remind us of the great difficulties faced by many learners with dyslexia in developing complex skills such as reading. There are clear implications for intervention strategies, in that repetition and overlearning are essential (without 'boring' the learner) and breaking complex tasks down into simpler tasks will aid automaticity. The importance of repetition has also been stressed by Dehaene (2004) who suggested that drills in lessons should be repeated until the optimal level has been achieved. This helps develop and strengthen neural pathways. These tasks can be presented in a cumulative, sequential manner to build more complex skills.

Implications for Practice

- Strategies must be used to foster automaticity in all tasks through 'overlearning' and the use of routines and repetition.
- When accuracy is established, learners should be encouraged to respond 'automatically' and speedily e.g., to letter–sound correspondence.
- Complex skills should be broken down into sub-skills so each is more readily achieved.
- Kinaesthetic strategies should be used to integrate skills and develop motor co-ordination.

Cognitive Processing Level

It is sometimes difficult to determine whether some cognitive processes (such as speed of processing and automaticity) should be considered from a cognitive or neurobiological perspective when examining theories of causation. We have already discussed automaticity, visual/auditory processing and speed of processing above. Two other aspects of cognitive processing are memory (see Chapter 2) and phonological processing discussed below under phonological deficit.

Phonological deficit

There is considerable agreement that most people with dyslexia have a phonological difficulty (Snowling and Stackhouse, 2013; Goswami, 2014). The phonological deficit hypothesis is not the only theory to offer an explanation for this as discussed above. Most researchers recognise difficulties in phonological processing as a core deficit although they may disagree as to its cause (e.g., Nicolson and Fawcett, 2008). Much research has shown that difficulties in phonological processing can distinguish those who have dyslexia from those who do not and that phonological awareness difficulties at an early age can predict later reading difficulties (Snowling, 2000) because of difficulties in learning the alphabetic principle i.e., that letters (graphemes) represent sounds (phonemes). Abilities in phonemic awareness, letter–sound knowledge and rapid automatised naming at the beginning of year 1 predicted children's success in reading at the end of year 1 in a study by Hulme and Snowling (2012). Phonological difficulties have also been found in studies of adults with dyslexia. Ramus et al. (2003), in a case study of 16 dyslexic (and 16 non-dyslexic) university students, found all 16 had a phonological deficit, ten also had an auditory deficit, four a motor difficulty, and two a visual deficit. Five of them had a phonological deficit *only*. (An auditory deficit aggravates a phonological deficit.) A more recent study by Ramus et al. (2013) distinguished between children with Specific Language Impairment (SLI) and those with dyslexia on the basis of a phonological awareness deficit as those with only SLI did not present the same difficulties.

The phonological deficit hypothesis assumes a difference at brain level (possibly in the perisylvian region). Snowling has suggested that the 'causal status' of brain differences in dyslexia is 'debatable because brain development shows considerable plasticity: both its structure and function are shaped by use' (2008: 5). Research into genetics described earlier indicates a direct genetic link, whereas the magnocellular hypothesis suggests temporal processing and disordered auditory systems may give rise to phonological deficit. The cerebellar deficit theory links automaticity and possibly motor control to the phonological deficit and also claims a direct language link to the cerebellum. Snowling (2008) maintains that there is a direct phonological deficit and has suggested that future research should consider whether there might be low-level impairments in the pre-school years that may be 'developmental antecedents'/indicators of the phonological deficit. In earlier studies (e.g., Snowling, 1992) she proposed that difficulty in the retrieval of phonological codes stored in long-term memory was a cause of reading difficulties and suggested that phonological coding deficits were responsible for short-term memory difficulties. Snowling has argued that rather than indicating a limited memory capacity these difficulties may be a result of inefficient verbal rehearsal strategies, resulting in information loss during the transfer from short- to long-term memory. The 2009 Rose Report on dyslexia accepted the view of dyslexia as primarily affecting reading and spelling development due to impairments of phonological processing,

verbal processing speed, and verbal short-term memory. Goswami and colleagues have researched several aspects of phonological processing and in particular identified difficulties in rhythm perception affecting syllabification for learners with dyslexia compared to the normal population (Goswami, 2014; Leong and Goswami, 2014).

A further point to be considered is that English orthography is less consistent in its grapheme–phoneme correspondence than many other alphabetic languages such as Spanish and Italian (i.e., it has a less transparent orthography). This may explain why many learners have particular difficulty with decoding and spelling in English (see Caravolas et al., 2012) and in turn has implications for assessment and teaching. It could also explain some differences in research into dyslexia in different countries. Pavlides (2004), for example, claimed that visual processing difficulties are found in 80 per cent of Greek learners with dyslexia compared with only 20 per cent with phonological difficulties because the grapheme–phoneme correspondence in Greek is more consistent than in English. Nevertheless phonological deficits have been found across many languages world-wide (Goswami et al., 2011). (See also Chapter 7.)

Implications for Practice

- Assessment of phonological awareness will provide a useful baseline for identifying children who may be dyslexic (see Chapter 3).
- Assessing a learner's ability to recognise letters and provide even a single 'sound' correspondence will indicate their knowledge of the alphabetic principle.
- Teaching should involve work based on the alphabetic principle and systematically teach phoneme-grapheme correspondence in order to establish that auditory, visual, and articulation skills are integrated.
- Learners should be encouraged to adopt verbal rehearsal strategies, using routines where they say aloud the grapheme–phoneme correspondence, name letters on presentation, and say each letter name as they write it during spelling.
- Activities to develop a sense of rhythm should be encouraged (e.g., music, singing, clapping, rhymes, poetry) not only in the early years.

Behavioural Level

This level describes the observed literacy difficulties of learners with dyslexia (we acknowledge that while dyslexia affects other areas of school and everyday life, literacy

is the focus of this book). These are the 'signs' or 'indicators' of dyslexia – some learners with dyslexia may present all of them, some may only present a few, and each of these may vary in severity. Such behaviours will be influenced by a range of environmental factors, including the teaching learners have experienced, the emotional support they receive, and a range of social and cultural factors which may exacerbate, alleviate, overcome, or prevent some of the difficulties. These characteristics can provide useful guidance to teachers and parents for identifying those learners who 'may' have dyslexia and therefore further assessment will be required.

The main characteristics *in literacy* may be summarised as:

- difficulty following instructions;
- a slow processing speed;
- a poor standard of written work (compared with oral work);
- confusion of letters/directionality problems (e.g., b/d, p/q, u/n, etc.);
- many reversals of letters;
- transposition of letters e.g. beard (for bread), saw (for was);
- phonetic and/or bizarre spellings;
- sequencing difficulties (letters and numbers);
- loses place in reading;
- a poor grapheme–phoneme correspondence in reading;
- omits/inserts words when reading;
- hesitant in reading aloud;
- no or inappropriate expression in reading;
- first letter guessing.

It is important to relate these to a learner's chronological and developmental age as some of the characteristics may be of a temporary nature (e.g., children of 6 or 7 years will often reverse 'b' and 'd' in their early stages of writing).

Similarly, if the learner's first language is not English this should be taken into account (see Chapter 7 for a consideration of this area).

Implications for Practice

- Clusters of characteristics may indicate that further assessment is required for learners who may have dyslexia (see the BDA website for checklists at pre-school, primary, secondary, and adult levels and Phillips, Kelly and Symes, 2013). Note that checklists should not be seen as a means of identifying dyslexia but they may point to areas which require further investigation.
- Teachers should consider how cognitive processes underlying these behaviours can inform teaching strategies.

Table 1.1 *Characteristics of Dyslexia at Different Ages*

Characteristics of Dyslexia at Different Ages An understanding of theories of causation of dyslexia helps us to appreciate how dyslexia may be manifested at different ages. This is summarised below.	
Age	**Behaviours**
Pre-school 0–5	• Delayed speech • Articulation difficulties • Poor ability to detect rhyme • Poor self-help skills e.g., dressing
Primary school years 5–11	• Poor letter–sound knowledge • Transposition of letters in spelling/poor orientation (beyond the age of 7–8) • Omitting letters or syllables in spelling and reading • Poor decoding skills • Frequently losing place in reading • Poor copying skills • Difficulty following long or complex instructions • Slow recall of facts
Secondary 11–16	• Many of the above may persist • Slow reading speed • Phonetic approximations in spelling • Difficulty organising/structuring written work • Poor/slow handwriting • Difficulty in decoding unfamiliar/polysyllabic words • Poor automatic recall of facts • Poor skimming/scanning skills
Adult Post-16	• Many of those under 'secondary' persist • Poor organisation of study skills • Difficulties in structuring arguments in assignments • Spelling difficulties • Difficulty multi-tasking (e.g., listening and writing at the same time)

Note that not all of these will necessarily be displayed.

Interaction with Environmental Factors

The Morton and Frith (1995) model emphasises not only that there are relationships between the levels but also that all levels are affected by and interact with the environment. In developing a holistic model to integrate the three main theories of causation, we consider the levels should be depicted as encompassed by the environment. This is important because we do not view 'dyslexia' and a learner's manifestations of dyslexia as a 'within child' or 'medical' model. At a biological level, learners may be

affected by birth trauma or pre-natal experiences, including their parents' diet, and at the cognitive and behavioural levels their family and school contexts will affect how they achieve as well as their motivation, learning styles, and compensating strategies.

The Multi-Deficit Model of Dyslexia

It is clear from the discussion of these theories, that there is considerable overlap, and that a single deficit theory is hard to defend. Pennington (2006) proposed a multi-deficit model of dyslexia using a similar framework to Morton and Frith but comprising four levels:

1. A combination of genetic and environmental risk factors.
2. The combination of neural systems affected by each genetic or environmental risk factor.
3. Cognitive processes affected – pathways may overlap leading to co-morbidity.
4. Behavioural or symptom level (including indicators of co-morbidity).

Pennington suggests some causal connections between levels, some of which will feedback to earlier levels, making this a complex model. However, it is particularly interesting because it addresses the fact that dyslexia often co-exists with other specific learning difficulties such as ADHD (Ebejer et al., 2010), Specific Language Impairment (SLI) (Ramus et al., 2013), Dyspraxia (DCD), ASC, and Dyscalculia. Some of these share the same cognitive deficits, e.g., phonemic awareness and naming speed are unique predictors for dyslexia but dyslexia has a shared predictor with ADHD which is processing speed. Stein (2015) suggests a 50 per cent overlap of cognitive behaviours between dyslexia and ADHD. There is also a great overlap between dyslexia and dyspraxia which share many common behavioural characteristics such as poor motor skill development, articulation difficulties, time-management, speech difficulties pre-school, word-finding problems and poor organisational skills.

Recently researchers have again started to question whether there is only one kind of dyslexia and if all learners with dyslexia have a difficulty in phonological processing. Pennington et al. (2012) discuss two large-scale studies, one in the USA and one international (Australia, the USA and Norway), examining the effectiveness of single and multiple deficit models in identifying dyslexia. They found that about 30–36 per cent of learners with dyslexia had multiple deficits. Single deficits were found in phonemic awareness, speed of processing and language skills. They came to the conclusion that a hybrid model is needed to identify dyslexia (i.e., one that looks for both single deficits and multiple deficits). Further support for this approach comes from a small scale study of Portuguese children with dyslexia in the 2nd to 5th grade (Andreia et al., 2014) which found two clusters: one group underperformed on phoneme deletion and rapid automatised naming, the other group underperformed on phoneme deletion and digit span but not rapid naming. In another study

Georgiou et al (2012) found that children with dyslexia learning a consistent orthography (Greek) did not experience auditory processing deficits but about half of them showed visual processing deficits. Both orthographic processing difficulties and rapid automatised naming deficits were also found.

Intergenerational model

Castles and Friedman (2014) argue that the majority of learners with dyslexia will display a deficit on at least one of the wide range of tasks broadly considered to measure phonological skills, but it is the locus of impairment that gives rise to each type of dyslexia. Van Bergen et al. (2014) examined the cognitive profile of parents of children with dyslexia (N=212) and came to the conclusion that we should consider the particular genes and cognitive abilities that children inherit from their parents. (This supports the findings of earlier familial studies by Snowling et al. (2007) that having parents with dyslexia does not necessarily mean that the child will have dyslexia.) Van Bergen et al. developed an 'Intergenerational Model' of dyslexia by adding a level to the beginning of Pennington's model (2006) to show genetic transmission (maternal and paternal genotype and maternal and paternal phenotype). They consider that this gives further explanations of the variation in cognitive profile often observed and believe that multiple cognitive deficits (each due to multiple etiological factors) need to be present to produce the characteristics of dyslexia at the behavioural level.

Implications for Practice

- Teachers need to be aware that most assessment reports will be based on a multiple deficit model of dyslexia reporting on at least two areas of difficulty in cognitive processing.
- Assessment should look for possible co-existing difficulties which may have implications for different approaches to teaching.
- Some co-existing conditions may be noted in the Education Health Care Plan under the SEND Code of Practice (DfE, 2015).

An Integrated Causal Model

In the integrated model (see Figure 1.2) we have included polygenetic influences at the 'top' of the triangle to draw attention to the way that different genetic make-ups can offer explanations for the variety of difficulties experienced by learners with dyslexia, together with other factors. This is in keeping with Pennington's multiple deficit model of dyslexia (MDM).

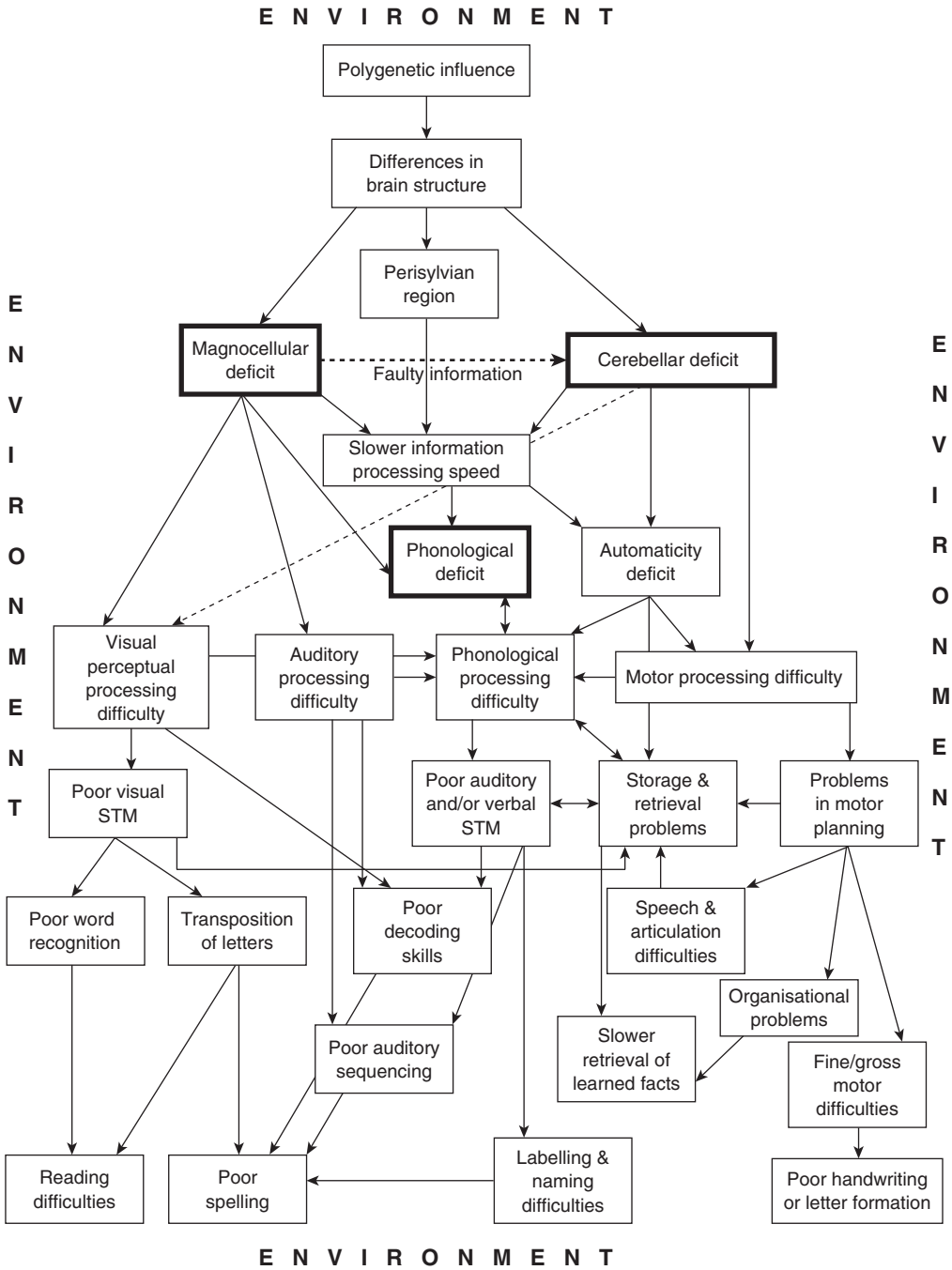

Figure 1.2 *A Model that Integrates the Three Main Theories of Causation*

We consider that the three main theories (outlined in bold) are inter-related. The model demonstrates that because the cerebellum does not normally act alone, but in conjunction with other parts of the brain, it will take in and organise information from the auditory and visual pathways of the magnocellular system. If a magnocellular deficit results in faulty information being received by the cerebellum (indicated by a bold dotted line in the model), then it will impact on organisation, storage, and retrieval.

Nicolson and Fawcett (2008) claim that because the cerebellum is implicated in eye movement, the visual processing difficulties described by Stein (2001a) could be the result of a cerebellar dysfunction rather than a magnocellular deficit. We have represented this as an indirect link (a dotted line) because further evidence is needed in order to establish it as a direct cause. The model gives prominence to the core phonological deficit and shows not only a direct link to the genetic component, but also a relationship to the magnocellular deficit and cerebellar theories.

The inter-relationship between these theories is most evident at the cognitive processing levels, where there are links between speed of processing, automaticity, visual and auditory processing, motor skills, and phonological processing difficulties.

The model also gives significance to interactions with the environment *at all levels*. Earlier we suggested that diet and allergies might affect genetic and neurobiological factors. At the cognitive and behavioural levels diet may also be significant as well as language and other social factors in the home, such as a child's early experiences of literacy which may contribute to (or compensate for) their dyslexia. In addition it is important to consider the school learning context where the amount, nature, and speed of presentation of information may affect the learning of those with dyslexia. In addition, the nature and level of support and social interactions with peers, teachers, and other adults will affect learning and social and emotional development, particularly of self-concept and self-esteem, which in turn can affect cognitive processing and behaviours (Burden, 2008).

Developing the Teaching Programme

Although there remain some controversies about causation, there is agreement that 'dyslexia' is a continuum and also that many aspects of dyslexia – such as poor organisational skills, automaticity and speed of processing, poor memory and sequencing skills – can affect everyday life and not just literacy. However, literacy remains a major concern internationally and improving the literacy skills of learners with dyslexia has become a national priority in England (Rose, 2009).

The research discussed in this chapter suggests that any intervention should be based on an individual's cognitive profile. Learners with moderate to severe dyslexia will need intervention that is phonics-based, is structured to enable overlearning and involves multisensory teaching. Reading and spelling require an ability to make linkages, for example, between sounds and letter names forming a relationship between

auditory (phonological) and visual stimuli and output. If these links are taught in a multisensory way – using visual, auditory, kinaesthetic (speaking/writing) and tactile channels – then neural pathways can be developed (or strengthened) within the brain.

Using a multisensory approach, employing as many senses as possible, *simultaneously*, will aid automaticity and speed of retrieval by enabling each mode of information to be stored in its specific location in the brain, while establishing linkages between them. Multisensory teaching, therefore, aids the transfer from short-term to long-term memory (as discussed in the following chapter).

The Conquering Literacy programme and teaching strategies recommended in this book are based on the implications from the research referred to above. It is interesting to reflect on the fact that research into the nature and causation of dyslexia since the 1980s reinforces a justification for the approach used by Gillingham and Stillman (1956) and Cox (1972) on whose work most current programmes are based. However, whereas those early programmes promoted the view that learners with dyslexia should only use such approaches and not be required 'to read' other books/sources while on the intervention programme, we prefer a more balanced approach. Specialist lessons, therefore, whilst based on a structured programme, should also include some 'real reading' – even if this means the teacher reading to and/or with the learner in order to help them become familiar with 'literary' language and develop comprehension skills. Study skills should also be included from basic to more advanced levels. Many programmes and specialist teachers strongly endorse the view that it is essential for *all* learners to start at the beginning of a structured intervention programme in order to ensure that they do not have 'gaps' in their knowledge. In our experience this practice, even when 'adjusting the pace appropriately', will be disliked by many learners who will either be bored or feel their 'failure' is being reinforced. We have, therefore, provided an 'accelerated' version of the programme, with an associated assessment procedure that may be used with learners who already have basic decoding skills.

Summary

We consider that the needs of learners with dyslexia who present moderate to severe literacy difficulties can best be met through the use of a programme based on the results of research into the behaviours and causes of dyslexia. Such a programme will be structured, sequential, multisensory, and phonics-based, and will build up automaticity. The success of this type of approach has been reported in reviews by Singleton (2009), Torgeson et al. (2006), Torgesen (2005) and Brooks (2013), and is endorsed in the Rose Report (2009). The Conquering Literacy programme contained in this book embraces these principles.

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