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Dyslexia
A problem with sounds

Adinda Robberechts

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Promotor: Prof. Dr. D. Sandra
Tweede lezer: Dr. H. Taelman

Universiteit Antwerpen
Faculteit Letteren en Wijsbegeerte

Authenticiteitsverklaring

Ondergetekende, Adinda Robberechts, studente Taal- en Letterkunde Engels-Spaans, verklaart dat deze scriptie volledig oorspronkelijk is en uitsluitend door haarzelf geschreven is. Bij alle informatie en ideeën ontleend aan andere bronnen, heeft ondergetekende expliciet en in detail verwezen naar de vindplaatsen.

Datum Handtekening

Preface

Ever since I began my studies in philology, linguistics has particularly appealed to me, more specifically, grammar and psycholinguistics. When the list of potential topics for the bachelor paper was made available, the subject of dyslexia drew my attention because my cousin suffers from this disorder. So, working on this bachelor paper gave me the opportunity to gain insight into the cause of her reading difficulties.

I would like to mention a few people who made writing this bachelor paper less difficult for me. First of all, I am grateful towards Prof. Dr. Dominiek Sandra, who supervised this work, for helping me in the right direction with constructive comments and for spending time on correcting the drafts. I would also like to thank Joris Heirstrate and Els Gansemans, who read through the final version.

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1 Introduction

When learning to read, a considerable percentage of children fail to develop the normal level of literacy skills. Furthermore, this delayed or impaired development cannot be attributed to a low level of intelligence, a lack of adequate instruction or socio-economical problems. The failure to learn to read at the normal level is a great burden for otherwise bright children and can hamper their education substantially. This particular reading problem is often referred to as 'dyslexia' or 'specific reading disability', as opposed to a general learning disability. In the last century, many researchers have investigated this fascinating problem and have, especially, been concerned with the cause of it. This search has lead to many different hypotheses and, eventually, to a consensus on the underlying cause of dyslexia, being a phonological processing deficit. Some researchers have even gone further and have tried to map the biological basis of that deficit.

In this bachelor paper, I will be concerned with the research on dyslexia. After the delineation of what exactly is dyslexia, I will try to give an overview of the different pathways concerning the underlying cause of dyslexia which researchers on dyslexia have followed and often also abandoned, including a recent development in the research. Naturally, most attention will be paid to the current view on dyslexia; I will explain how a phonological coding deficit may lead to a specific reading impairment. Furthermore, in the light of the importance of phonological skills and phonemic awareness, I will discuss the effect of orthography types on the profile of dyslexia. Finally, I will touch upon the research on the biological basis of dyslexia.

2 What is dyslexia?

In this section, we will look at the development of the definition of dyslexia, as a good definition of dyslexia is a useful starting point for a thorough discussion of this reading problem. In addition, a few paragraphs will be spent on the discussion of the dichotomy between 'all-or-none' theories and 'continuous' theories on the nature of dyslexia.

2.1 Definition

In order to make sure that it is clear what exactly is meant when talking about dyslexia, a well-defined definition of dyslexia is necessary. A definition of dyslexia also provides a basis for scientific research as it is clearly falsifiable and offers objective criteria that make diagnosis of dyslexia possible.

It is necessary to point out that definitions of dyslexia may have different aspirations. Some definitions try to account for the underlying cause of the problem in order to come to an insight into the true nature of the problem, whereas other ones mostly focus on a description of the reading problem to make a successful diagnosis possible. Recently, endeavours have been made to combine several layers of description, thereby accounting for both the cause and the developmental pattern of dyslexia.

2.1.1 The medical model

The first attempts to define dyslexia came from the medical world. Pringle-Morgan and Hinshelwood tried to define this particular form of reading difficulties as a disorder of 'congenital word blindness' already a century ago (Hinshelwood, 1896; Morgan 1896). Hinshelwood, being an ophthalmologist, considered the failure to read to be a visual problem specifically related to words. The term 'word blindness' has proved to very persistent, even after the underlying hypothesis was refuted.

Later on, when research on dyslexia advanced, it was necessary to develop a clear definition of this concept. The definition developed in the 1960s by the World Federation of Neurology reflected the attempt to provide a systematic approach in making the distinction between dyslexic and normal readers (Critchley, 1970).

> [Dyslexia is] a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and socio-cultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin.

The problem with this definition is that it is based on the 'exclusion' of causes – it enumerates the factors that cannot be the causes but says nothing about what actually is the cause - and that it provides virtually no basis for a positive diagnosis of dyslexia. Moreover, several unspecified concepts are used, as, for instance, the concept of 'adequate' intelligence is not further explained (Snowling, 2004). Finally, it leaves the underlying nature of the reading problem unspecified.

2.1.2 The regression approach

In order to arrive at more objective criteria, researchers turned to epidemiological studies. By using large representative samples of children, it was possible to avoid arbitrariness of symptoms. By means of these large studies, many signs formerly considered as typically dyslexic, such as reversing *b* and *d*, were proven to be normal in the early stages of reading development. Most influential among epidemiological studies are the Isle of Wight studies. These studies led to the distinction between a 'general reading backwardness', an expected reading disadvantage considering the IQ of the child, and a 'specific reading retardation', a reading problem that is disproportionate to the expectations based on the child's IQ score (Rutter & Yule, 1975). Hence, in order to determine whether children are affected by specific reading retardation, i.e. dyslexia, it is necessary to find out whether there exists a discrepancy between their expected reading age and their actual reading age. This method of classification of readers is often called a regression approach[1] and implicates the use of a cut-off point, which is to a certain extent arbitrary, as diagnostic of dyslexia. The conclusions of the Isle of Wight studies also included a 'bimodal' hypothesis[2], suggesting that the specific reading disability is of a different nature, i.e. originates from different cognitive problems, compared to the reading problems of generally low-achieving readers, but other recent studies have failed to find evidence for such a bimodal distribution of reading scores (Fletcher, Foorman, Shaywitz & Shaywitz, 1999).

Because of the arbitrariness of the cut-off point, diagnosis of dyslexia according to the discrepancy-based approach was often rather ambiguous. Many children diagnosed as dyslexics no longer fitted the criteria several years after their diagnosis. Furthermore, because the discrepancy-based definition did not specify the cause of the difficulty in acquiring literacy skills, children who suffered from reading difficulties that originated in e.g. inadequate reading instruction were included as well, as the definition only made it possible to observe retardation (Snowling, 2004). Another problem with IQ-based definitions is that studies have shown the correlation between IQ and reading skill in normal readers to be imperfect (Stanovich, 1986). In other words, it is difficult to predict an expected reading capacity on the basis of a child's IQ. Consequently, the distinction between dyslexics and normal readers based on this correlation is questionable. Moreover, poor literacy skills can influence the verbal IQ, so that the discrepancy may be masked (Snowling, 2004).

2.1.3 The dimensional approach

Studies showed that the approaches mentioned may in fact be obstacles in the development of a proper definition, as these studies consider dyslexia to be more than a disorder of reading. Contemporary

[1] This method tries to predict reading performance on the basis of a linear equation of the proportion of IQ (on the x-axis) and reading skills (y-axis; Reading level = a . IQ + b). As an equation is described as the regression of the response (y-factor) on the carrier (x-factor), this method is referred to as a regression approach.

[2] The mode is the value that has the most observations in a series of experimental data. Two modes suggest the existence of two distinct groups.

views on dyslexia, however, no longer try to account for all of the problems that may accompany dyslexia, such as problems in other academic areas and attention deficit hyperactivity disorder (ADHD). Instead, they focus on the core deficit. There seems to be a consensus among researchers that this core deficit is situated at the level of phonological processing and that the main problem is single word decoding. These definitions often view dyslexics as being at the lower end of a continuum of reading ability rather than undergoing an entirely different reading development (Shaywitz, 1996) and therefore exhibit a dimensional approach. Again, these definitions assume the application of a cut-off point in the continuum of reading ability in order to establish whether a child is dyslexic. A definition that can be considered to be a dimensional definition is the one offered by the Orton Dyslexia Society of the USA (Fletcher, Foorman, Shaywitz & Shaywitz, 1999, 275):

> Dyslexia is one of several distinct learning disabilities. It is a specific language-based disorder of constitutional origin characterised by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities. These difficulties in single-word decoding are often unexpected in relation to age or other cognitive abilities; they are not the result of generalised developmental disability or sensory impairment. Dyslexia is manifested by a variable difficulty with different forms of language, including, in addition to a problem with reading, a conspicuous problem with acquiring proficiency in writing and spelling.

According to Margeret Snowling, a possible way to arrive at a satisfactory definition of dyslexia is to consider three levels of description (2004). She states that a definition should include the three levels of the 'causal chain' of dyslexia: the biological, the cognitive and the behavioural level, and mention the influence of environmental factors so as to come to a 'cognitive-developmental approach to dyslexia' (Snowling, 2004, 27). This means that, on developmental level, this definition offers a description of the behavioural pattern of dyslexia and how this changes with age. Next, from the perspective of the cognitive approach, it proposes a hypothesis on the underlying cause at the cognitive level. This is an 'invisible' level at which mental activities take place. Ultimately, the cognitive-developmental approach seeks to understand the biological basis of dyslexia.

2.2 Types of theories: all-or-none vs. continuum

As stated earlier, the Isle of Wight studies suggested that the distribution of reading skills was bimodal, i.e. it showed a high concentration of children near the average of the distribution and another rather high concentration in the lower end of the distribution. This is what led the researchers to believe that two types of reading disability existed: a general reading backwardness, consistent with the expectations based on the IQ of the child, and a specific reading retardation, in other words, dyslexia (Rutter & Yule, 1975). Hence, the Isle of Wight studies supported the idea of a qualitative difference between children affected by dyslexia and normal readers, of an 'all-or-none' theory.

All-or-none theories of dyslexia support the notion that dyslexia originates from a specialized deficit interfering with the normal workings of one of the many skills involved in reading, caused by structural or functional anomalies in the brain. In short, they claim that something in the brains of

dyslexic children works differently. Continuous theories of dyslexia, on the other hand, are based on the assumption that the nature of the difference between dyslexics and normal readers is quantitative. They believe that dyslexics are on the lower end of a 'continuum' ranging from children who find it very difficult to acquire the skill that is crucial to the development of reading proficiency (which is commonly agreed to be phonological awareness) to children who have no difficulties at all with acquiring this skill (Vellutino, Fletcher, Snowling & Scanlon, 2004).

The difference between the all-or-none nature and the continuous nature has important consequences for the treatment of dyslexia. If dyslexic children develop their reading skills in a way entirely different from normal readers, the usual teaching strategies for reading will not be of use to them. However, a quantitative difference would mean that whatever helps children normal will also help dyslexic readers. Hence, prevention of dyslexia would become a lot easier, as a 'universal' strategy could be applied. Bryant and Bradley (1990) are firm supporters of this continuous theory of dyslexia. They suggest to expose children in nursery schools to all sorts of word-games, poems and rhymes so as to encourage their awareness of sounds. This, they claim, will not only benefit the children that will become dyslexics – and, hence, be a protective factor – but the normal readers as well.

3 Normal development of literacy skills

The search for the causes of dyslexia will lead to different hypotheses concerning the 'abnormal' development of the reading skills. Hence, in order to come to a profound understanding of what goes wrong in the development of dyslexic children's reading skills and why it does, it is necessary to understand the pathway that normal readers go through.

3.1 Learning to read

When learning to read in an alphabetic script, children have to become aware of the letter-sound consistency, or rather, the grapheme-phoneme consistency. Before they come to the task of learning to read, many children are already familiar with words, for example their own name. They are able to recognize some written words and their meanings, but this is only because they have memorized the global characteristics of these words. This stage, in which children recognize words as a whole and therefore interpreting the entire word as a single symbol, is called the logographic stage. However, in the alphabetic script that is used in most Western languages, the written representation is not of the logographic type (in contrast to Chinese, for example). The basic representational units, i.e. the letters, or rather graphemes, do not represent concepts but phonemes. Importantly, phonemes are not sounds but an abstraction of such sounds, i.e. they are the smallest linguistic units that can bring about a change of meaning in a word (e.g. /b/ and /p/ are phonemes because they distinguish ***b****at* vs. ***p****at*). Often they are represented by a single letter, but as they are sometimes represented by a sequence of letters (e.g. *ph* in *phone*), it is better to talk about graphemes. So, the sequence of phonemes that graphemes represent makes up the phonological form of a word, which in turn discloses the meaning of the word.

When learning to read, it is exactly this connection between the phonemes and graphemes that children need to pick up. However, this is not as easy as it seems since, in practice, phonemes do not appear as separate entities in the physical speech signal. In speech, sounds seem to 'blend into one another', which is the result of the continuous motor movements of the articulators during speech, a phenomenon which is referred to as coarticulation. Obviously, children are able to hear the differences between phonemes, considering the fact that they have no difficulty in distinguishing e.g. *be****d*** vs. *be****t***. However, their ability to distinguish between phonemes does not mean that they are consciously aware of them. Consequently, learning to read in an alphabetic script also involves the development of explicit phonological awareness, more particularly, phoneme awareness. Moreover, research has shown that children that are more sensitive to phonological differences have an advantage over other children when their literacy development is concerned (Caravolas, Hulme & Snowling, 2001).

Nevertheless, acquiring this alphabetic principle is not the last step in the development of learning to read. Children also have to get acquainted with orthographic relationships that go beyond simple phoneme-grapheme correspondences. An example of this is the ending *-tion* in e.g. *nation*, whose

pronunciation cannot be determined solely by using a phonetic strategy. According to Share (1995), children acquire word-specific orthographic knowledge, thus developing a sight vocabulary, on the basis of every successful decoding of an unfamiliar word, using phonological decoding as a kind of self-teaching device (as it allows the child to identify a word and then store the spelling pattern of the successfully decoded word). This device has to be supplemented by the linguistic context to disambiguate partial decoding attempts, in order to develop a skilled word recognition system. Thus one could talk about a 'lexicalization' of the phonological recoding system, as the latter no longer shelters only the basic phoneme-grapheme consistency but also larger spelling patterns.

In other words, according to Share's theory, children start off by using the phonetic strategy to determine the pronunciation when they encounter printed words. However, when this pronunciation is too difficult to determine by means of a phonetic strategy only, as is the case for a word like *nation*, they will only be able to decode it partially, i.e. know how to pronounce the first syllable *na-*. Consequently, they will have to turn to the context. The surrounding words in a sentence like 'The United Nations (UN) is an international organization' may give 'clues' that are useful to determine the possible meaning and the corresponding phonological representation of *nation*. The orthographic information derived from this successful decoding is then internalized, thus developing a sight vocabulary.

According to a model proposed by Ehri (1992), children develop their reading skills as a result of an interaction between their phonological representation of words and the printed words they encounter. Children supposedly use 'phonetic cues' to give access to the phonological representations of the words in their memories. Put differently, children rely on only a few letters of words to lead them to the pronunciations of those words. Thus, connections are built between spelling and sounds and orthographic representations are developed.

3.2 Learning to spell

The majority of children find it a lot harder to spell than to read. The reason for this is that production is always harder than recognition, both in the spoken and written language domains. In the case of written words, it is possible to read a word on the basis of partial phonetic cues and the context, but if one has to spell a word one has to know its precise orthographic pattern. For dyslexics, this is even more difficult, as it is easier to use compensatory strategies in reading than in spelling. For a long time, it was assumed that when children learned to spell, they did this in the same way as they learned to read, because the two activities make use of the same alphabetic code. However, this is not the case, although phonological awareness plays an important role in spelling, too.

Typically, children learning to write in English start of by using letter-names to construct words. An example of this is *genius* spelled as *gnys*, using the letter-name 'g' as the entire first syllable (Bissex, 1980). In the beginning, they may just write one letter representing a marked sound, then expand this to boundary sounds. After a phase of semiphonetic spelling errors in which consonant

clusters are simplified and the representations of voiced and voiceless phonemes are confused, non-dyslexic children reach the end of the alphabetic phase and can spell with phonetic accuracy. Nevertheless, they still have to learn a lot in order to master the awkward spelling of English (Snowling, 2004).

Treiman (1993) performed an extensive study on the spelling development of children and found that they soon replace the use of letter-names by phoneme-grapheme correspondences, which shows they exploit phonology, although they still leave out difficult parts. Treiman also demonstrated that children are sensitive to spelling regularities or orthographic conventions from early on in their spelling development. For instance, they will misspell *cake* as *kack*, but never as *ckak*. The increasing mastery of these conventions, more specifically conventions on the spelling of morphemes, was studied by Nunes, Bryant and Bindman (1997). They focused on the *ed* ending of the regular past tense and showed that children first spell phonetically, then detect that the orthographic sequence *ed* is a possible spelling of word-final /t/ in English but generalize the *ed* ending to irregular verbs and go on to overgeneralize it to non-verbal elements, e.g. *sofed* instead of *soft*. Finally, they restrict the use of the suffix to the regular past tenses, understanding that it is attached to a stem of regular verbs only. However, the command of these grammatical distinctions was shown to be related to the child's awareness of these morphological distinctions in spoken language, as it was predicted by performance on a morpho-syntactic task half a year earlier. In this task children had to make word analogies (e.g. *anger* -> *angry* / *strength* -> x) and sentence analogies (e.g. Tom helps Mary. Tom helped Mary. Tom sees Mary. Tom x Mary). Good performance on this task showed an awareness of morphological relations. Hence, in order to develop proficiency in spelling not only phonological skills are important, but also morphological skills, i.e. an understanding of the meaning and syntactic functions of different types of linguistic units (Snowling, 2004).

4 What is the problem?

In the search for the underlying cognitive deficit of the basic problem of dyslexia, which manifests itself in the form of single word decoding, several hypotheses have been proposed in the last century. In the following section, an overview will be given of the different ideas that have been disproved and of the current ideas about dyslexia.

4.1 Theories on dyslexia

4.1.1 Methodological problems in studying the cause of dyslexia

a) Mental-age match vs. reading-age match

In the last decades a lot of hypotheses about dyslexia have been proposed, often backed up by evidence derived from experiments. But many of these hypotheses have been proven wrong. Hence, the evidence must have been misleading, or the strategy used in the experiments must have been flawed. Many of these 'flawed experiments' are those that use the so-called 'mental-age match' in order to show a difference between dyslexic children and normal readers. In these experiments, impaired readers are compared to control children of the same age and intelligence. Leaving aside the difficulty of matching the IQ's of controls to the variation that exists between the verbal and the performance IQ's of dyslexic children, the problem here is that, obviously, dyslexic children will perform worse on reading-related tasks, since they are characterised by exactly this impairment. Moreover, reading difficulties will prevent dyslexic children from being exposed to printed words to the same extent as normal readers. In addition, the disproportionate amount of attention children need in order to decode a word will hamper them in actually understanding what they are reading. This, evidently, will have many effects, for instance, on language comprehension. So, it is unclear whether the problems of dyslexic children at tasks are either a consequence of their lower reading level or a cause of it (Bryant & Bradley, 1990; Snowling, 2004). Any theory of dyslexia should attempt to identify the cause of this problem.

Bryant and Bradley (1990) insist that it is necessary to use the 'reading-age match' in order to avoid this kind of ambiguity. When dyslexic children are compared to children of the same reading level (who are obviously younger), differences can no longer be characterized as a possible consequence of dyslexic children's bad reading. Indeed, in such a comparison the two groups of readers read equally well. Hence, if the dyslexic children perform worse at a certain task, e.g. a task in which children are asked what a word would sound like after omitting the first sound, it can be inferred that this is related to the cause of their reading problem. So, whereas experiments using the mental-age match can only be used to find the areas affected by dyslexia, only reading-age matching can reveal the underlying deficit (Snowling, 2004).

However, the reading-age match does not come without problems either. Indeed, when no difference is found when using a reading-age match, this does not automatically mean that there isn't one. This null result may be caused by the difference in maturation between the dyslexics and the younger controls. Dyslexic children may already have gained skills to cope with tests the controls haven't, so that some tasks may prove to be very easy for the older, impaired children but not so for the younger control children. Moreover, if the control children are too young, some skills may just have begun developing, and are unlikely to be picked up in an experiment (Snowling, 2004).

b) Establishing causes

Another way to study the cause of a problem is to use a longitudinal design. This makes advantage of the fact that a cause precedes its effects. Adapted to research on dyslexia, a longitudinal study should begin before children come to the task of learning to read and last at least until several years after children have started to go to school. Differences that exist between pre-reading abilities of children that become dyslexics on the one hand and normal readers on the other hand may then be the cause of dyslexia. At least, they will certainly not be a consequence of the failure to learn to read. However, such differences may also originate from the same cause that is responsible for dyslexia, without being the cause itself. In this case, both the differences found in the pre-reading and reading abilities would be consequences of an unknown third factor (Bryant & Bradley, 1990).

To make sure that the difference found in the longitudinal study is a cause and not another consequence of another, hidden, underlying deficit, it is useful to combine it with a training study. The rationale behind this type of study is that if a certain skill, or rather the lack of it, is the cause of a problem, improving that skill will reduce the problem. So, after training one group of dyslexic children in the skill that was found to be different in e.g. a longitudinal study, it is possible to determine whether the improvement of the skill in question has also had a positive effect on reading. In order to avoid any ambiguity of the results derived from the training study, it is necessary to provide a control group of dyslexic children with the same amount of attention in the form of an another type of training, which is however not crucial to the skill at stake. If there is a positive effect of training the skill, this is proof of a causal relationship between the particular skill and reading (Bryant & Bradley, 1990).

4.1.2 A visual deficit

As mentioned earlier, dyslexia was initially considered to be a visual deficit. This is not very surprising, as the first one to study dyslexia, Hinshelwood, was an ophthalmologist. Together with Morgan, he believed the phenomenon they were facing to be a result of not seeing written words the way they were and introduced the term 'word blindness' (Hinshelwood, 1896; Morgan 1896). However, Hinshelwood and Morgan were not able to offer the necessary empirical support.

In the 1920's, Samuel Orton, a neurologist, put forward a theory about a more specific visual problem. He observed that dyslexics made many mirror image confusions of letters and words, e.g. confusing *b* with *d* or *was* with *saw*. On the basis of these mistakes, Orton proposed his optical reversibility theory of dyslexia (1925), suggesting that dyslexics suffer from a perceptual deficit. This was supposed to be the result of the two, symmetrical, hemispheres of the brain, each of which would create an image that mirrors the images generated in the other half, thus creating confusion between two images. However, Orton's theory has been found to be wrong. Mirror image confusions turn out to be neither specific to nor characteristic of dyslexic children, as this type of mistakes is made by all children who learn to read and only amounts to a small percentage of the errors made by dyslexics (Bryant & Bradley, 1990). Furthermore, they are not the result of a visual problem, but a naming problem that is resolved when becoming more familiar with letter-sound correspondences (Vellutino & Scanlon, 1982).

Theories suggesting a visual deficit to be at the core of dyslexia, either at the perceptual level or at the level of visual memory, are undermined by experiments performed by Frank Vellutino. One of these experiments asked children to copy words. It was found that the dyslexic children had no more problems with this than did other children of the same age, which proves that they are not at a disadvantage as far as their visual memory is concerned. When asked to pronounce the letters, though, dyslexics performed remarkably worse than non-dyslexic children (Vellutino, Steger & Kandel, 1972). Another experiment made use of an orthography that was unknown to the tested children: Hebrew. Dyslexic children turned out to perform as well as normal readers when they had to recall short Hebrew words, i.e. they were asked down the words immediately after they were presented with them (Vellutino, Pruzek, Steger & Meshoulam, 1973). These results suggest that dyslexic children do not experience any considerable difficulties at the level of visual perception and memory. It was only when linguistic codes could be used to support the memory of visual information that dyslexic children were at a disadvantage, i.e. when the information can be retained by means of words. Mind that the mental-age match used here does not undermine the results of the experiments, as it is used to provide negative evidence, i.e. evidence for the lack of a difference: the difference in reading performance is not matched by a difference in visual performance.

Several researchers have claimed low-level visual deficits to be at the core of dyslexia, more specifically visual tracking problems, a deficit in the 'transient visual system' or abnormalities in the perception of visual motion. The former implies that dyslexic children have occulomotor deficiencies, i.e. defects with reference to the eyeball movements. However, studies investigating eye movements have not been able to find differences on visual tracking of non-verbal information (Vellutino, Fletcher, Snowling & Scanlon, 2004). The transient system theory and the visual motion perception theory, on the other hand, are supported by some evidence derived from experiments.

The transient visual system is part of the human visual system. Put simply, the visual system consists of two types of pathway, the magnocellular and the parvocellular pathway. The magnocellular

system is made up of M cells, which are specialized in detecting motion and rapidly changing stimuli, whereas the P cells of the parvocellullar layers are specialized for form and colour vision. This distinction corresponds to the distinction between the transient and the sustained visual system. This is relevant for reading as the parvocellular, sustained, system is believed to be operative during eye fixations and the transient, magnocellular, visual system during saccadic eye moments. The latter supposedly suppresses the visual trace that the former activates when reading. Defenders of the transient system theory of dyslexia claim that this suppression does not work properly in dyslexics, causing 'visual trace persistence', i.e. an abnormally long-sustained visual trace which 'confuses' them when reading text (Snowling, 2004; Vellutino, Fletcher, Snowling & Scanlon, 2004). Several studies have tried to show this to be the case, using spatial frequency grids. They have shown that dyslexic children and normal process high and low frequency grids differently. Furthermore, they differ in contrast sensitivity function, which implies that dyslexic children need greater luminosity for distinguishing low frequency grids (Vellutino, Fletcher, Snowling & Scanlon, 2004).

However, as stated by Hulme (1988), visual trace persistence can only account for reading difficulties when reading connected text, whereas dyslexic children suffer problems when reading single words, too. Note that in the case of isolated words there is no visual trace or a preceding word that could interfere with the visual processing of the fixated word. Further critique on the transient visual system theory concerns the fact that no evidence exists that poor readers experience the hypothesized visual masking problems (trace persistence) under normal reading circumstances and the fact that some normal readers were also found to have transient system deficiencies without showing any symptoms of dyslexia. In addition, the dyslexic children who were found to have transient visual system deficits also showed the phonological deficits that are generally acknowledged to be at the core of dyslexia (Vellutino, Fletcher, Snowling & Scanlon, 2004). When putting all this together, it is doubtful that transient visual deficits are causally related to dyslexia. However, this does not rule out the possibility that is a correlate of specific reading disability. But even this is questioned by recent studies suggesting that it is not the parvocellular system that is suppressed during saccadic eye movement, but the magnocellular system (Skottun & Parke, 1999). Obviously, if this turns out to be true, the entire transient visual system theory of dyslexia, as a causal explanation of the phenomenon, is undermined.

In the visual motion theory, too, the magnocellular system is involved, as this theory suggests that the perception of visual motion in dyslexics is deficient. In order to prove that a difference exists between dyslexic and non-dyslexic readers, Cornelissen, Richardson, Mason, Fowler and Stein (1995) turned to random dot kinetograms. In this method, the test subject has to look at two adjacent panels filled with thousands of white dots on a darker background and say in which panel they see a stream of movement. The movement is created by shifting the positions of the dots every 20 milliseconds and perception of this motion is dependent on the percentage of dots that move coherently. The experimenters increase the proportion of dots moving until the participant can detect movement

among the dots. Thus, thresholds for motion detection are measured. In the study by Cornelissen et al., it was found that, initially, thresholds were higher for dyslexics than for control children. But the measures showed a significant overlap between the two groups and, after a first block of trials, both groups improved, especially the dyslexics. Raymond and Sorensen (1998) used random dot kinetograms, too, to assess visual motion in impaired and fluent readers. They found that the detection thresholds for short duration stimuli were almost double those of the age-matched control group. And, though nothing distinguished these children from the others, six dyslexics even showed thresholds that fell outside of normal limits. In a second experiment of the study, Raymond and Sorensen changed the conditions of the test, more specifically the duration and number of frames in the RDK. In the normal 2-frame condition with a brief duration of 32 ms, no group differences were found. But when seven frames were involved and the duration of the stimuli was increased to 112 ms, the dyslexic group showed a mean threshold that was two times higher than the mean threshold of the controls. On the basis of these findings, Raymond and Sorensen concluded that dyslexics suffer an abnormality in the perceptual integration of motion information.

The empirical evidence found in the studies on both the visual transient system and visual motion perception indeed suggests deficits in the magnocellular system. But it is still unclear how such a deficit could have an influence on reading acquisition, as the deficiency is rather subtle. Moreover, the magnocellular deficiencies seem to co-occur with phonological processing deficits (Eden, Van Meter, Rumsey, Maisog, Woods & Zeffiro, 1996). A possibility that many investigators consider is that magnocellular deficits are biological markers for dyslexia, i.e. they accompany the reading impairment, but are not causally related to it (Snowling, 2004; Vellutino, Fletcher, Snowling & Scanlon, 2004).

4.1.3 Deficits in general learning abilities

As opposed to theories that attribute dyslexia to a very specific deficit, some theories suggest that the cause of dyslexia is to be found in deficiencies of general learning abilities. These are abilities that are not only used when learning to read, but are involved in learning in general. For instance, theories of this type have suggested that dyslexia is caused by deficiencies in attention or serial-order processing; cross-modal transfer; and problems with rule learning and association learning. In this section, we will deal with the cross-modal transfer theory.

Herb Birch, an American child psychologist, introduced the idea of a cross-modal deficit. This means that people suffering from this deficiency have difficulties with the integration of information deriving from cross-modal perception, i.e. from different senses. Put differently, the theory claims that affected children find it hard to recognize the connections or equivalences in the perceptions of different senses. This deficit would prove to be very cumbersome when learning to read, as it would prevent certain children from understanding the association between a spoken word and its written equivalent (Birch & Lefford, 1963).

The evidence used to support this hypothesis came from experiments in which children were asked to match an auditorily presented pattern to the visual representation of the pattern (Birch & Belmont, 1964). With a pencil, the experimenter tapped a rhythmic pattern which consisted of three, four or five taps, separated by either short or long pauses. The child then was presented with several visual patterns that were made out of dots separated by long or short spaces, e.g. ●●● ● and ● ●●●, and had to say which of the visual patterns was equivalent to the auditory pattern. According to Birch's findings, eleven year-old dyslexics performed significantly worse on these tasks than did age-matched control children.

However, there are several problems with Birch's hypothesis and the experiments from which he derives the evidence to support it (Bryant & Bradley, 1990; Vellutino & Scanlon, 1982). The most important objection can be raised on methodological grounds. Not only was the experiment based on a comparison between age-matched groups, which makes it impossible to determine whether the cross-modal deficit is either cause or effect of the dyslexia, but also did the experimenter fail to provide controls covering possible group differences concerning, for instance, intrasensory functioning and the use of encoding (Vellutino & Scanlon, 1982). It was very well possible that the difference between the normal readers and the dyslexic readers was not due to a problem with the cross-modal transfer, but to just one of the many abilities used in the process of comparing the two types of information. The children had to perceive the auditory pattern, store the stimuli in their short term memories, distinguish between visual patterns, understand the meaning of the dots and spaces as temporal order patterns, know that they had to 'read' the dots from left to right, etc. A problem in one of these areas would have disrupted the interpretation of the entire experiment. Indeed, further research showed dyslexic children to be at a disadvantage in tasks that require matching temporal order patterns, even within the a single sensory modality, which undermines Birch's hypothesis, as it suggests a problem with short-term memory rather than with cross-modal transfer (Blank & Bridger, 1966; Blank, Weider & Bridger, 1968; Vande Voort, Senf & Benton).

In addition, Vellutino and his colleagues provided evidence against the cross-modal theory through experiments on intra- and intersensory association learning (Steger, Vellutino & Meshoulam, 1972; Vellutino, Harding, Phillips & Steger, 1975; Vellutino, Steger & Pruzek, 1973). These studies controlled for possible group differences in rapid encoding, as they tested paired-associate learning (between stimuli from different modalities) instead of perceptual matching. The results showed that poor readers did not perform at a lower level than did normal readers under any of the presentation modes, except when information that the children had to learn was made up of verbal stimuli. When learning did not involve any verbal information, no differences were found. This is an important finding, suggesting that the weakness of dyslexics stems from a linguistic deficit, rather than from a problem with cross-modal matching.

Other theories suggesting that a deficit in general learning abilities is at the core of dyslexia were also disproved. A major objection raised to this type of theories is based on logical grounds. By

definition dyslexia is a 'specific' disability which only manifests itself in reading development. Furthermore, a less than average intelligence and general learning difficulties are ruled out when diagnosing dyslexia. This is contradictory to a vision that ascribes the cause of dyslexia to general learning deficiencies.

4.1.4 A linguistic deficit

Several experiments mentioned earlier made it apparent that the problems with which dyslexic children are confronted can almost always be shown to be related to the verbal information they have to process. These findings led researchers to contemplate the idea of a linguistic deficit to be at the core of dyslexia rather than perceptual deficits. As language is a probably the most important aspect of reading and writing, a problem with linguistic coding will obviously hamper the development of reading and writing skills. Visual and motor skills, on the other hand, play a mostly supplementary part in the reading and writing processes. However, there is a considerable difference between the assumption that linguistic coding difficulties are responsible for dyslexia and finding the exact nature of these difficulties. Indeed, the linguistic skills that are used in reading and writing are themselves a combination of several skills that have become automatic. These skills can be divided into three groups: the semantic, the syntactic and the phonological aspects of language. The semantic aspect of a word is the concept or entity to which it refers. Its particular use in sentences, on the other hand, is determined or limited by its syntactic properties. Together, the semantic and syntactic properties of a word give it its particular meaning in concrete linguistic usage. The phonological properties of a word determine its specific structure of sounds. The task of dyslexia researchers is to find which of these aspects are responsible for the problems dyslexic children encounter.

Semantic coding, as defined by Vellutino and Scanlon (1982, 218) is 'the use of words, phrases, and sentences to code meaningful information'. Semantic coding deficiencies can lead to difficulties with naming and pronouncing printed words and may originate from impairments in storage and retrieval of lexical information. Note that the reverse is not true: difficulties with naming and pronunciation are not necessarily a result of semantic coding deficiencies, as, for instance, phonological coding is involved in these processes, too. These malfunctions may be related to a deficient development of vocabulary; name retrieval problems; difficulties with word and sentence comprehension; and syntactic and phonological coding deficiencies or inefficiencies (Vellutino & Scanlon, 1982). Some researchers have looked into the possibility that vocabulary deficits were the cause of dyslexia, as they assumed that it is easier to learn to read words that are part of one's active vocabulary than it is to read entirely new words. They were right, as research has shown the existence of a certain correlation between lexical development and reading achievement. Several studies found that vocabulary knowledge in first grade predicts early and later reading level (Dickinson & Tabors, 2001; Scarborough, 1990, 1991; Snow, Barnes, Chandler, Goodman, & Hemphill, 1991; Snowling, Gallagher & Frith, 2003). Furthermore, Tabors and Snow showed that deficient vocabulary knowledge

can cause significant reading difficulties in second language readers with limited proficiency in spoken English (2001). Hence, a child having limited lexical knowledge is likely to experience difficulties in acquiring fluency in reading. More evidence for a link between limited vocabulary and reading skills comes from investigations carried out by Vellutino and colleagues, demonstrating that it is more difficult for poor as well as for normal readers to establish associations between low-meaning words and their written representations, than it is between high-meaning words and their written forms. The difference between high-meaning and low-meaning words in the experiment, both words that the children had heard before, was determined on the basis of the number of semantic associations they evoked on an association task (Vellutino, Scanlon & Spearing, 1995). Together, these studies demonstrate an intrinsic relationship between vocabulary knowledge and reading. They do not, however, constitute proof of a causal relationship as there are no studies showing that an improvement of lexical knowledge would improve reading skills. Furthermore, as the effect of meaning on the establishment of connections with written representations has been shown to apply for non-dyslexic readers, too, this cannot be the cause of the impairment. Hence, on the basis of these studies can only be inferred that they are probably both the effects of a yet unknown third factor[3].

Syntactic coding is the understanding and application of meanings and functions in the verbal discourse. To this end, it is necessary to be able to apply the grammatical rules of a one's language correctly and to understand the process of transformation which words and sentences undergo in order to make them fit a particular usage (Vellutino & Scanlon, 1982). The idea that syntactic deficits may cause the problems that characterise dyslexia can be dismissed easily. Although it seems logical that difficulties with the exact use of linguistic context to support word identification could hamper beginning readers, investigation has shown that, typically, no distinction exists between dyslexic children and normal readers when it comes to syntactic knowledge except when the dyslexic children have suffered from long-lasting reading difficulties. This suggests that syntactic deficits in dyslexic children are a consequence, not a cause, of their impairments.

Phonological coding refers to the understanding that the graphemes of written words represent phonemes which, together, make up the phonological form of words, and the ability to divide spoken words into their phonemic segments. Contrary to the semantic and syntactic deficit hypotheses of dyslexia, the theory that considers weak phonological coding to be the underlying cause of dyslexia is supported by strong evidence. It is now the consensus that a phonological coding deficit is responsible for dyslexia. This will be further explained in the next section.

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[3] This third factor might be the 'clarity' of phonological representations in the mental lexicon. This might (i) cause dyslexics – who are not able to establish specified phonological representations, as a result of the phonological deficit that is presumed to underlie dyslexia (cf. infra) – to build up an extensive vocabulary and (ii) cause high-meaning words, which probably occur more often that low-meaning words, to have stronger phonological representations. This may account for the connection between dyslexia and vocabulary knowledge.

4.2 A phonological deficit

As was explained earlier, in order to achieve fluency in reading, children need to understand the principle of the alphabetic script, i.e. that graphemes are the written representations of phonemes. This means that children first need to become consciously aware of phonemes, before they can start making connections between these and the corresponding graphemes, and vice versa. Hence, a problem with phoneme awareness is likely to lead to significant difficulties in the reading development. Research has proved that it is exactly this type of deficit that leads to the problem pattern that dyslexics show, as it explains not only the slow development of reading skills, but also other problems like handling verbal data in, for instance, lists of disconnected facts.

The phonological deficit theory of dyslexia is supported by evidence derived from multiple experiments. These experiments have tried to narrow down the problem of dyslexia to its most basic deficit. As 'a phonological problem' still means nothing more than 'a problem with speech sounds', such a definition is in itself not specific enough. The idea that there exists a significant difference between dyslexic children and normal children with respect to their awareness of the sounds of language has been proved by a seminal study carried out by Bryant and Bradley (1990). In this study, they investigated the sensitivity to rhyme in children who had on average a high intelligence but experienced considerable reading difficulties that could not be explained as the result of emotional, physical or social handicaps. The performance of these children was compared to that of reading-age matched normal readers. In the first of two tests, the experimenters said four words, three of which shared a common sound, and the children had to say which word was the 'odd man out'. The position of the common sounds in these words varied. Sometimes it was the middle vowel (in e.g. *nod, red, fed, bed*) but at other times it was the initial (*sun, sea, sock, rag*) or final consonant (*weed, peel, need, deed*). Thus both alliteration and rhyme were tested. In the second task the children were asked to produce a word that rhymed with a given word. These tasks proved to be very difficult for the dyslexic children, especially the task focussing on alliteration. Indeed, the scores of the older impaired readers were three to six times worse than those of the control children. The finding that dyslexic children are rather insensitive to rhyme and alliteration shows that they suffer from a phonological deficit, i.e. have a basic problem with their sensitivity to speech sounds. This lack of sensitivity might be the cause of their reading difficulties.

4.2.1 The nature of the phonological deficit

The above tests, however, do not pinpoint the exact deficit. They are merely proof of a problem in the area of speech sounds. In order to find a more specific deficit attached to a particular skill, researchers turned to an investigation of the structure of the phonological abilities. Among researchers there are two opposite ideas concerning the structure of phonological abilities. Some authors believe that all phonological skills derive from a single underlying ability, whereas others argue that separable,

though related, factors account for different phonological skills (Snowling, 2004). The crucial issue, then, is which phonological ability is the main determinant of dyslexia.

Muter, Hulme, Snowling and Taylor (1998) identified two so-called separable phonological factors, which they named 'segmentation' and 'rhyme'. Both of these phonological abilities accounted for performance on phonological awareness tasks in 4- to 6-year-old children. 'Segmentation' was tested by means of syllable and phoneme segmentation tasks, whereas 'rhyme' stood for performance in rhyme detection and production. The investigators found that reading levels were predicted more accurately by segmentation than by rhyme in this group of children. A follow-up study showed that early segmentation skills predicted not only the reading level of these children at the age of 6, but also their spelling development at the age of 9 (Muter & Snowling, 1998a & b).

Studies carried out by Wagner, Torgesen, Laughan, Simmons and Rashotte (1993) assigned kindergarten and second-grade children with an extensive variety of phonological tasks. A first list of tasks measured explicit phonological awareness. Phoneme elision and segmentation tasks, rime oddity tasks and first sound categorization tasks tested phonological analysis; tasks in which children had to blend (i) the onset and rime components of words, (ii) phonemes into words and (iii) phonemes into non-words evaluated the children's phonological synthesis abilities. A second set of tasks measured *implicit* phonological awareness. These tasks focused on working memory, discrete naming of isolated digits and letters and serial naming tasks. The researchers found four correlated phonological ability factors: 'analysis/working memory', 'synthesis', 'isolated naming' and 'serial naming' (Wagner, Torgesen, Laughan, Simmons & Rashotte, 1993). Furthermore, each of these factors was a predictor of the reading level later on. However, phonological analysis and phonological synthesis were redundant with respect to each other: only phonological analysis predicted first-grade reading, whereas only phonological synthesis affected second-grade reading levels (Wagner, Torgesen & Rashotte, 1994).

Still, one has to be very cautious when interpreting the results derived from these tests. Indeed, the assumption behind these tasks is that reading development depends on performance on metaphonological tasks which ask children to reflect consciously on the phonological form of words, whereas it is possible that these tasks are only indirect test, i.e. they only pick up the indirect consequences of how the brain represents phonology. In short, such metaphonological tasks would, for instance, reflect impairments with respect to phonological representations, without, however, being suited to identify this particular deficit (Snowling, 2004).

This brings us to the other view on the structure of phonological abilities, which claims that phonological skills derive from a single underlying ability. This is relevant for our discussion as the prevailing ideas on dyslexia claim that the impairment is due to a single deficit, more particularly, a phonological coding deficit, which is believed to be the underlying cause of many, if not all, problems that are related with dyslexia. This hypothesis, which is commonly accepted nowadays, states that the brains of dyslexics show a deficit when it comes to coding phonological information, which creates a

causal chain of problems eventually leading to poor reading development. In short, the phonological coding deficit prohibits the brain from establishing clear phonological representations and from achieving normal phonemic awareness, which, in turn, affects alphabetic mapping, i.e. the establishment of phoneme-grapheme connections. Hence, dyslexic children find it hard to identify words and have great difficulty in achieving fluency and accuracy in reading.

This hypothesis is supported by the results of intervention studies in which the experimenters helped children to improve their phonological awareness and letter-sound mapping skills. This type of direct instruction has proved to have a considerable positive effect on their word identification, spelling and reading abilities. One such intervention study was carried out in Denmark. Lundberg, Frost and Peterson (1988) designed a programme in which Danish pre-school children - hence, not diagnosed with dyslexia - played metalinguistic games using rhymes and phonemes. One year later, when the children started to go to school, the trained children performed higher on phonemic awareness tasks than did children of a control group. This phonological training proved to have a long-lasting effect on reading development, as the trained children showed a better reading performance than the control children at each moment they were tested in the first three grades in school. Furthermore, trained children that were considered to be 'at risk' for reading failure before arriving at school obtained a normal reading level three years later (Lundberg, 1994). As training on phonological skills creates better opportunities for becoming a good reader, the conclusion seems warranted that reading performance relies on the quality of phonological sensitivity. If one knows, as was demonstrated earlier, that dyslexic children consistently perform worse at phonological awareness and grapheme-phoneme decoding tasks than do normal children (Snowling, 2004), it is only a small step towards the idea that their reading impairment is caused by their phonological problems.

In an attempt to find more direct evidence for the hypothesis that weak phonological coding is at the core of dyslexia, a number of researchers have turned to studies examining speech perception and production in both impaired and normal readers. Typically, studies investigating speech perception made use of paradigms in which words that varied in a single phoneme had to be categorized. Investigators exploited the fact that, for instance, the initial consonants of *bath* and *path* only slightly differ in their Voice Onset Time (VOT), i.e. the time between lip closure and the onset of the vibration of the vocal chords. By means of 'synthetic speech', it is possible to manipulate the VOT to the extent that /ba/ and /pa/ are almost undistinguishable. However, speech perception tests have shown that, though the /ba/ and /pa/ variants make up an acoustic continuum, in speech perception, listeners typically perceive a clear (artificial) distinction between the two sounds. They either perceive the sound as /ba/ or /pa/, never as a mixture of the two or as a sound 'in-between'. This is what is called the categorical perception in phonological processing. If it turned out that dyslexic children had more problems in categorizing this type of closely-related sounds and words, this would be proof that they perceive phoneme boundaries less sharply than do normal readers (Snowling, 2004; Vellutino, Fletcher, Snowling & Scanlon, 2004). Indeed, some of these studies suggest that this is the case, but

the evidence is inconsistent. Brandt and Rosen (1980), investigating the perception of stop consonants, found that children suffering specific reading disability perceived and decoded phonemic information much in the same way like normal readers did at an earlier developmental age, suggesting a problem with speech perception. However, Hurford and Sanders (1990) found differences between impaired and good readers in the discrimination of syllable pairs such as /gi/ - /bi/ for second-graders, but not for fourth-graders. Other studies have tried to account for these inconsistencies by considering the possibility that a phoneme discrimination deficit was present in some or many impaired readers, but not in all. If so, such a deficit is not likely to be the major cause of dyslexia. Still, interpreting the results of these studies remains problematical, as the tasks used to evaluate speech perception require a high amount of attention, which is difficult for many dyslexics (Snowling, 2004). Furthermore, the encoding of spoken words usually happens in the context of other words in the speech stream. This context is important for the establishment of phonological representations of words that are added to the spoken vocabulary. As such, phoneme discrimination tasks are an imperfect tool for evaluating spoken word encoding skills (Vihmann, 1996).

Another tool for investigating whether dyslexics have unclear phonological representations caused by speech perception deficits is the so-called 'gating' paradigm. In this paradigm listeners hear small parts of words, on the basis of which they have to try to identify the word. The segments get progressively larger until the listener is able to say the word. Typically, listeners are also asked to rate their confidence in their answers. As is to be expected, when it comes to words with many 'phonological neighbours' a relatively large part of the word is necessary in order to identify it (phonological neighbours of *birth* would be *bird*, *burn*, and *worth*). Furthermore, high-frequency words are identified much sooner that low-frequency words (Snowling, 2004). However, the auditory gating method failed to demonstrate a difference between dyslexics and normal readers; although dyslexic children seem to require more information in order to identify words that do not have many phonological neighbours (Elliot, Scholl, Grant & Hammer, 1990; Metsala, 1997).

Speech production has often been evaluated on the basis of verbal repetition tests. For instance, Snowling (1981) asked both dyslexic and non-dyslexic children to repeat polysyllabic words, e.g. *pedestrian* and *magnificent* and non-words that were matched to the polysyllabic words regarding their phonological structure, such as *kebestrian* and *bagmivishent*. The dyslexic children showed greater difficulty when repeating non-words. In contrast, an auditory discrimination test in which children had to decide whether two non-words, e.g. *fizidor-fizitor* were identical or not, found no group differences. This contrast between the findings in production and perception tasks suggest that the problem is probably to be found in speech production rather than in speech perception. In order to test the role of speech perception, some tests made use of noise-masking in the verbal repetition task. This made it more difficult to perceive the auditory signal. Snowling, Goulandris, Bowlby and Howell (1986) used three levels of noise-masking, in order to manipulate the perceptual stage of processing. The first level was at the same sound intensity as the words to be repeated, the second was 3 decibels below the

speech signal, the third group of words was not masked at all. The speech stimuli consisted of three groups, too: high-frequency words, low-frequency words and non-words. Overall, the performance of both impaired and normal readers declined equally according to an increase in the sound mask, which rules out a perceptual deficit. However, when looking at low-frequency words and non-words alone, significant differences became apparent. Dyslexic children obtained lower scores on both conditions and were the only group who found it more difficult to repeat the non-words than the low-frequency words. Snowling (2004) claims that the difference may be due to the fact that the articulatory motor programmes of unfamiliar words, as opposed to those of high-frequency words, are not immediately accessible, as their lexical representations are not as easy to retrieve. Non-words even lack this lexical entry. This inefficiency or impossibility of memory retrieval for unfamiliar and non-existing words implies that participants will only be able to repeat these items when they can identify their phonological structure on the basis of processes of segmentation and analysis. The finding that dyslexic children experience significant difficulties with exactly these processes once more endorses the phonological coding deficit hypothesis of dyslexia.

Before we go on to explain how the phonological deficit exactly hampers dyslexics in their reading development, we will look at several problems which dyslexics encounter even when they are not confronted with written text. As we will see, some of these problems will 'work together' with the direct consequences of the deficit underlying reading and thus aggravate the reading problem. Therefore, it is better to understand these difficulties before dealing with the effects of the phonological deficit on reading development.

4.2.2 Problems outside the reading context, caused by the phonological deficit

The phonological deficit presumed to underlie dyslexia explains several problems that dyslexics encounter outside a reading context, such as problems with verbal short-term memory. As the experiments by Vellutino mentioned above show, no difference exists between impaired and normal readers regarding the memory span for visual information. Dyslexics, however, do show an impairment when it comes to verbal short-term memory, as they cannot make efficient use of the sounds in words. Moreover, this memory deficit cannot be considered a 'developmental delay', as it seems unlikely that it is a result of the reading impairment itself and as adult dyslexics who have overcome their reading problems still encounter verbal short-term memory difficulties (Snowling, 2004). The fact that verbal working memory is not as efficient in dyslexics as in normal readers explains why many impaired readers find it hard to follow a list of instructions. In addition, many other tasks that can be facilitated by using verbal short-term memory, such as memorizing photographs, prove to be more difficult for dyslexics.

But not only short-term memory is affected by dyslexia. Dyslexics also show difficulties in word retrieval from long-term memory (i.e. their mental lexicon), which manifests itself in naming tasks, in which one must rapidly name letters, numbers or pictures. This is not the consequence of problems in

concept identification. Indeed, the fact that dyslexic children perform just as well as normal readers when asked to decide whether two pictures are the same proves that it is the process of name retrieval that causes the difficulties (Braams, 1997). Furthermore, the vocabulary knowledge of dyslexics usually surpasses their naming performance, i.e. they regularly fail to retrieve words that they know. Even this failure to overcome the 'tip of the tongue' state can be explained in terms of ill-specified phonological representations of words.

Obviously, all these problems with the processing of verbal information (temporary storage, fast word retrieval) can cause significant problems for dyslexics. In school context, for example, it is necessary to memorize disconnected facts and names or follow lists of instructions, something which is more difficult for dyslexics. Even mental arithmetic requires the storage of verbal information in working memory. This means that dyslexic children that are good at mathematics can, even in this domain, experience the consequences of their condition.

4.2.3 The effect of a phonological coding deficit on reading

First and foremost, a phonological coding deficit has direct consequences for a skill that is crucial to reading, namely alphabetic mapping. Since dyslexic children have deficits in the way their brains process and represent phonological information, it is very difficult for them to 'grasp' the phonemes in spoken words. In other words, they have weak phonemic awareness. As alphabetic orthographies lean on correspondences between graphemes and phonemes – exactly the units that dyslexics find hard to recognize – impaired readers will find it hard to access the phonological and lexical forms connected to a particular written word. Thus, word identification becomes a difficult task for dyslexic children. As a consequence, their reading comprehension is also hindered, as the task of word identification alone already absorbs a lot of energy. Indeed, the many working-memory resources that are needed for phonological decoding leaves only little capacity for semantic processing. This type of hindrance is sometimes referred to as a 'bottleneck' process.

Dyslexic children are also confronted with other problems deriving from the phonological deficit which in turn contribute to their reading difficulties. For instance, some researchers suggest that the difficulties in name storage and name retrieval discussed in the previous section also have a negative effect on the establishment of connections between the spoken and written counterparts of words. They believe that it is difficult to make a strong association when one of the equivalents, the phonological form, is badly accessible and when, in addition, storage of this form is hindered. This problem in the establishment of connections between the written and the spoken forms of words can prove to be an obstacle for the reader's ability to store representations of word spelling as these representations may not be retrieved as unitized orthographic representations (Stanovich and Siegel, 1994). Hence, dyslexics cannot exploit this 'aid' for word identification and are, again, hampered in reading comprehension (Vellutino, Fletcher, Snowling & Scanlon, 2004).

Another interesting domain in which phonological deficits indirectly hamper the reading development of dyslexics is the acquisition of vocabulary. Indeed, some researchers suggest that the phonological deficit of dyslexic children may inconvenience them in acquiring new vocabulary (Snowling, 2004), as they often have difficulties to 'grasp' the phonological forms of words and, because of that, have qualitatively inferior phonological representations of words. Later on in development, though, vocabulary knowledge contributes to phonemic awareness as the phonemic contrasts and resemblances of 'phonological neighbours' such as *mail-sail* and *bin-bit* help children to become aware of the phonemic units that are crucial for reading (Snowling, 2004; Vellutino, Fletcher, Snowling & Scanlon, 2004). It can be inferred that the phonological deficit is such a fundamental problem that it interferes with normal reading development at several levels.

The merit of the phonological deficit hypothesis of dyslexia is that a single deficit accounts for the large variety of problems that confront dyslexics and that, moreover, it is compatible with what is known about normal reading development, i.e. the importance of phonological awareness (Snowling, 2004). The evidence is so compelling that it has lead to a definition of dyslexia as a 'core phonological deficit'. An empirical study by Stanovich and Siegel (1994) has shown that all impaired readers differ from normal readers on skills that were closely related to the core-deficit such as phonemic awareness, a fact which the 'phonological core-variable difference model' accounts for. In contrast, dyslexics that had been diagnosed on the basis of the traditional discrepancy-based definition differed from other impaired readers in skills further removed from the core, e.g. listening comprehension and working memory.

4.2.4 Connectionist models

The phonological core deficit is also supported by connectionist models of reading development that are used to conceptualize reading development. Connectionist models make use of parallel distributed processing (PDP) and are implemented as computer simulations. They infer abstract statistical regularities from the input that they receive and develop rule-like behaviour without explicitly representing any rule in their memory architecture. The information that a connectionist model receives is represented by simple processing elements in input and output systems. When the input represents graphemes and the output phonemes, this resembles the process of reading development. Children who learn to read set up connections between phonemes and graphemes on the basis of the information which they are presented with. Just as children make associations between the spoken and written counterparts of words, syllables, and so on, connectionist models learn that there exists a correlation between certain processing elements (e.g. English *ea* is pronounced as /ɪə/, as in *dear*, or as /ɛə/ in *there*, the former link being stronger than the latter). Connectionist models are able to do this because they can 'strengthen' certain associative paths by increasing the weight of the connection; if an association is irrelevant, the weight can also be decreased. In the initial state, all connections are set to random weights. Then, during a learning phase, the model is presented with a large amount of

information. When it encounters a certain association it has seen before, it adds weight to the already existing connection. Thus, a learning algorithm helps the model represent the connectivity that is implicit in the training material so that the model is capable of integrating a system that comes close to, in this particular case, the quasi-regularity of English spelling (Snowling, 2004).

Researchers have succeeded in creating computer models that approach the performance of adults on reading tasks (Plaut, McClelland, Seidenberg & Patterson, 1996). More particularly, they showed the traditional effect of word frequency and grapheme-phoneme consistency on reading accuracy. High-frequency words were read more accurately than low-frequency words, regular words were read better than irregular words and the system fell back on regular patterns more often with low-frequency words than with high-frequency words. Important for research on dyslexia is that connectionist models with limited powers of generalization, making them unable to generalize to the level of phoneme-grapheme consistency (such that they came to the task of reading with poorly specified phonological representations), show an error pattern on reading non-words that is comparable to the errors that are made by dyslexics. This way, the results from connectionist modelling support the idea that the lack of clear phonological representations impedes the dyslexic reader to create optimal associations. Impaired readers need to resort to establishing rough mappings that are only adequate for the establishment of relationships between entire words and their phonological forms, but which are not effective enough when it comes to grapheme-phoneme correspondences. Accordingly, the self-teaching device of which normal readers make use does not develop in dyslexics (Snowling, 2004).

4.3 Low-level auditory deficits

A theory proposed by Tallal (1980) relates problems in phonological decoding to low-level auditory deficits, more specifically, difficulties with temporal ordering (Snowling, 2004; Vellutino, Fletcher, Snowling & Scanlon, 2004). Tallal's hypothesis stems from earlier research by herself and her colleagues, in which they found that children with specific language impairment – this is an impairment of oral language skills – had difficulty making temporal order judgments with high and low tones when the interstimulus intervals were short (50 milliseconds). On the basis of these results, the investigators predicted that language impaired children should have difficulties with the discrimination of stop consonants, as this demands the analysis of fast changes and transitions (Tallal and Percy, 1973, 1975). Later on, Tallal expanded these findings to a theory of dyslexia, stating that the phonological decoding problems typically observed in dyslexics were caused by a deficit in the temporal resolution of rapidly changing auditory stimuli, which hampers speech perception. Put differently, dyslexics, according to Tallal, suffer from a deficit which prevents them from processing the large amount of rapidly alternating phonemes in the speech signal. This means that the cause of dyslexia would not be a phonological deficit but a basic problem in auditory perception.

However, even in Tallal's study (1980), only a minority of the dyslexic children was found to have difficulties with auditory temporal processing. Later research showed that only children with dyslexia

that were also affected by oral language impairments showed this type of deficits (Heath, Hogben & Clark, 1999). Furthermore, Tallal did not use speech stimuli in her experiments. Hence, it is speculative to infer a relationship between auditory temporal processing and phonological decoding abilities, as it is not known whether both skills derive from the same underlying perceptual mechanism (Vellutino, Fletcher, Snowling & Scanlon, 2004). Indeed, later studies have shown that the difficulties of dyslexics at temporal order judgment tasks that do use speech stimuli were due to speech discrimination deficits, i.e. were phonological in nature, rather than temporal order judgment deficits (Mody, Studdert-Kennedy & Brady, 1997).

4.4 A recent development in the research on dyslexia: the visual attention span deficit theory

The phonological deficit hypothesis of dyslexia is now widely accepted. However, some investigators doubt whether a phonological deficit is the only cause of dyslexia, since there are reported cases of good phoneme awareness skill in some dyslexics. An article by Bosse, Tainturier and Valdois (in press) suggests that an alternative cause of dyslexia, one that is independent of a phonological deficit, is to be found in a visual attention span disorder. In order to understand the theory which Bosse and her colleagues propose, it is necessary to discuss the difference between surface and phonological dyslexia first.

4.4.1 Surface dyslexia vs. phonological dyslexia

Apart from the readers who suffer from the congenital type of dyslexia discussed in this paper, sometimes referred to as 'developmental dyslexia', there are also impaired readers whose reading problems are due to brain damage. The reading problems that derive from this damage are often referred to as 'acquired dyslexia'. This type of dyslexia shows different profiles, depending on the type of damage to the brain. A number of researchers have compared 'developmental' with 'acquired dyslexia' and claim that some parallels exist. This has led to a sub-categorization of developmental dyslexia.

Two typical patterns of acquired dyslexia exist, due to an impairment of either the semantic or lexical pathway or the phonological pathway. The former offers direct orthographic access to the mental lexicon. Consequently, damage to this system predicts difficulties with gaining access to the mental lexicon on the basis of orthographic representations of words. On the other hand, damage to the phonological pathway, which is responsible for phonological recoding, will prevent access to the lexicon through the phonological analysis of words. This causes a profile that is called 'phonological dyslexia'. The readers suffering this kind of acquired dyslexia experience difficulties when analysis of the sounds of a word is needed, for example, when reading new words or non-words. In spelling this is reflected in dysphonetic errors such as *rember* instead of *remember* and *refets* for *rough*; derivational errors, e.g. *weight* instead of *weigh*; and visual errors, e.g. *cape* instead of *camp*, as phonological dyslexics seem to rely mostly on the visual appearance of words. Patients with damage to the semantic

or lexical pathway, on the other hand, are said to suffer from 'surface dyslexia'. Roughly, their error pattern can be described as the reverse of the pattern that phonological dyslexics show. Surface dyslexics have problems memorizing the written form of words, which means that they find it hard to read words that cannot be read using the normal phoneme-grapheme correspondences, such as irregular words. For instance, surface dyslexics may read *debt* (/det/) as /debt/. Their error pattern is characterized by 'regularisation' errors when reading and spelling (irregular) English words. Moreover, surface dyslexics tend to confuse the meaning of homophones, as they can only turn to the phonological form of words - and not to the memory of their written representation - in order to access the meaning. A third type of acquired dyslexia is called 'deep dyslexia'. This type of acquired dyslexia is not related to the two contrasting types mentioned above in that it is caused by damage that goes beyond just one of the pathways. Indeed, the reading skills of patients affected by this type of dyslexia are severely damaged. Deep dyslexics are not able to exert the kind of phonological analysis that is necessary for reading at all, so non-word reading is out of the question. The reading errors which they make when reading existing words are mostly semantic. They may read *boat* as *captain* or *rose* for *daffodil*, which shows that they read words without analysing their sounds. Furthermore, abstract words prove to be significantly more difficult to read for deep dyslexics than concrete words (Bryant & Bradley, 1990; Snowling, 2004).

A number of researchers have claimed to have found analogies between these types of acquired reading disorder and developmental dyslexia. Temple and Marshall (1983), for instance, claimed to have found a case of developmental phonological dyslexia in a 17-year old girl whose reading and writing skills were at the nine-year level. Her reading errors showed a pattern that was to be expected from patients with acquired phonological dyslexia. She made visual and derivational errors, reading for instance *appeared* as *appearance* and was not able to read long but regular words such as *herpetology*. However, the researchers did not compare the mistakes of this girl to those of normal readers of her reading-level. Also, the girl was able to spell certain words that she could not read. This is a discrepancy that more beginning readers show, suggesting that a reading-age based comparison had been in order. Hence, it cannot be concluded that her errors are symptoms of a specific type of dyslexia (Bryant & Bradley, 1990). Another attempt to link developmental with acquired dyslexia was made by Colthaert, Masterson, Byng, Prior and Riddoch (1983), this time with respect to the profile of surface dyslexia. They described the case of a 16-year old girl whose reading and writing skills were around a ten-year level. According to the investigators, this girl made mistakes one would expect of surface dyslexics. She found it easier to read regular words than irregular words and her reading mistakes were mostly regularisation errors, e.g. she read *quay* as /kway/. However, this girl's phonological reading skills, as evaluated by means of non-word reading tests, were poor, as opposed to what is normally the case for acquired surface dyslexics (Snowling, 2004). Furthermore, a number of other critical comments can be made. Again, no comparison was made between the mistakes of the girl and those of non-dyslexic readers at her level. For instance, beginning readers have more difficulty

with irregular words than with regular words and so would a dyslexic with the same reading level (Bryant & Bradley, 1990). In other words, the problem with irregular words was possibly only a symptom of the girl's low reading level rather than a defining feature of her dyslexia. This failure to compare the performance of the dyslexics that are studied to the performance of adequately matched control groups (i.e. a reading age match) is a recurrent problem among the first case studies that are used to classify dyslexia and undermines many of the conclusions drawn on the basis of these studies.

Nevertheless, the terms 'phonological' and 'surface dyslexia' are still used in the context of developmental dyslexia. Indeed, several scholars went on to propose sub-categorisations of developmental dyslexia, no longer on the basis of a comparison with acquired dyslexia, but from a developmental perspective (Frith, 1985) or by means of a regression approach which compares the performance of dyslexic children to the expected performance for their age (Castles & Colthaert, 1993). According to the latter view, phonological dyslexia is used to refer to a specific deficit in non-word reading, whereas surface dyslexia denotes a specific deficit in irregular word reading.

4.4.2 The visual attention span deficit hypothesis

Bosse, Tainturier and Valdois (in press) start from a connectionist model of reading: the multi-trace memory model of polysyllabic word reading proposed by Ans, Carbonnel and Valdois (1998). This model takes visual attentional processes into account as part of the reading system, as opposed to most reading theories, which consider these to be peripheral to the reading process. Moreover, the model states how damage to these processes can cause specific reading disorders. According to the computer model, reading relies on two types of reading procedures which vary in terms of the kind of visual attention (VA) they involve and their reliance on phonological processing. These procedures are the global reading mode, typically used when reading familiar words, and the analytical mode, normally used for new words and non-words. When applying the former, the VA window is supposed to cover the entire sequence of the input letter-string and the whole phonological output is said to be generated at once. In the analytic reading mode, however, the VA window narrows down to focus the attention on the successive parts of the written word. Similarly, the phonological output is generated in segments corresponding to the focal sequences of VA and are then temporarily stored in short-term memory.

The computer model has been tested, not only for normal reading, but also for acquired dyslexia. When the VA window size was reduced moderately, the model showed a surface dyslexia pattern. Reading in global mode was impossible and irregular words were subject to regularisation errors. A severe reduction of the VA window still had the largest effect on irregular word reading, but also led to an increase in the number of errors on regular words and non-words, thus causing the model to exhibit a mixed profile. An independent phonological impairment yielded an error profile similar to acquired phonological dyslexia. By analogy to the performance of the 'damaged' computer model, Bosse and her colleagues predicted that a selective visual attention span deficit in children might

influence reading development and lead to a pattern of developmental surface dyslexia, whereas a phonological deficit might cause developmental phonological dyslexia.

The purpose of their article is to provide evidence to support the hypothesis that both phonological and a visual attention span deficit can contribute independently to developmental dyslexia. This means that the VA span deficit accounts for unique variance in the performance of dyslexics besides the variance explained by a phonological deficit. The investigation consists out of two studies: one carried out in France, the other in Great-Britain. In the first study, 68 French-speaking dyslexic children with a mean chronological age of 11 years and six months were compared to chronological-age matched normal readers. The average reading age of the dyslexics was seven years and 11 months, so that the reading age of the control group was significantly higher. First, all children were given three reading tasks (testing regular word, exception word and non-word reading) and three metaphonological tasks (a phonemic segmentation, a phoneme deletion and an acronym task). Then, the children's visual attention span was evaluated in two tests. At each trial, the children were presented with a central fixation point for 1000 milliseconds, which was followed by a blank screen for 500 ms. Next, a random five-letter string (e.g. RHSDM) appeared at the centre of the screen for 200 ms. In the full report test the children were asked to orally report all the letters immediately. After five training trials, in which the experimenter provided the children with feedback, this was repeated for 20 times. Two scores were obtained: the number of letter strings repeated correctly and the total number of letters accurately reported. In the partial report test a vertical bar appeared one cm below one of the letters. The task of the participants was to report the cued letter only. This time, there were ten training trials and 50 actual test trials. The score was the number of cued letters correctly reported.

In general, the dyslexic children performed worse than the control children on the reading tasks (which is obviously predicted by their dyslexia), two of the phonological tasks and on all of the VA measures. The experimenters found a correlation between the reading scores and both the children's phonological performance and their VA scores. Moreover, some phonological skills correlated slightly with some of the VA measures. After controlling for chronological age (as both the VA measures and reading skills showed significant correlations with age) it appeared that non-word reading correlated with the three phoneme awareness tasks, whereas both regular and irregular word reading only correlated with the segmentation task.

The following findings addressed the question concerning the relative importance of VA and phonological skills in dyslexia. Although strong correlations of VA processing skills and reading subskills were found, none of the correlations between VA processing skills and phonological skills appeared to be significant. This is important for the VA span theory, as it is in line with the idea that VA span and phonological skills derive from independent abilities. Crucially, however, it was also found that, independent of phonological skills, VA span was a predictor of reading abilities. More specifically, it accounted for 29,4 % of unique variance in exception word reading and 36,4 % in non-word reading, which means that these differences in the children's reading scores could not be

explained by differences in their phonological skills but could be captured by differences in their VA scores. On the basis of these findings, the experimenters concluded that their group of dyslexics could be divided into four subgroups. The first group accounted for 19 % of the tested children and consisted of readers exhibiting a selective phonological deficit. The second group (44 %) showed a disorder of the VA span without phonological impairments. The third group showed both deficits (8 %) and the fourth group of 22 % did not seem to show either of the deficits.

The second experiment had two main goals. In the first place, it was meant to confirm the results of the first experiment in the 'deeper', English, orthography, i.e. an orthography in which the grapheme-phoneme correspondences are less clear and that, consequently, demands more effort in terms of phonemic awareness. Secondly, the British experiment was to exclude the possibility that uncontrolled factors had affected the results of the first experiments. Hence, nonverbal IQ, spoken vocabulary knowledge and single letter identification were controlled, too. The test subjects were British children, 29 dyslexics and 23 chronological age controls of an average age of ten years and six months. The groups differed slightly on non-verbal IQ. The VA tasks that were given to the children were the same as in the French study and the reading tasks were comparable to the ones in that first study. The phonological tasks of the British study, however, were a spoonerism task and an alliteration fluency and a rhyme fluency task. Furthermore, three control tasks were added: a letter identification task, a semantic fluency test, in which children had to name as many members of a given semantic category as possible, and a picture vocabulary test, in which the participants had to match a spoken word to one of four pictures in order to evaluate their receptive vocabulary knowledge.

The results of the second experiment confirmed the main findings of the first: 'Both phonological and VA processing skills were independent and significant predictors of reading performance' (Bosse, Tainturier, Valdois, in press, 20), even when age, IQ, vocabulary, semantic fluency and letter identification skills were controlled. The subgroups found in the first experiment were found in the second experiment, too. All these findings taken together persuaded the experimenters that a number of dyslexics suffer from a reading impairment caused by a visual attention span deficit, and not by a phonological deficit.

4.4.3 How does a visual attention span deficit affect reading?

In order to understand what the effect of a possible VA span deficit is on reading acquisition, we need to go back to the multi-trace connectionist model we discussed in the previous section (Ans, Carbonnel & Valdois, 1998). According to this model, irregular word reading depends on global processing which activates word-traces. These traces are created each time both the entire input letter string and the entire output phonological representations of the input item are available at the same time. This makes accurate processing of the input letter sequence, i.e. identification of all letters in their respective positions, necessary. Hence, a VA span deficit limiting the number of letters that can be processed simultaneously may interfere with this creation of memory word-traces and,

consequently, hamper normal global reading development, which, in turn, interferes with normal irregular word reading.

The correlation found between VA span abilities and non-word reading can, according to Bosse et al., also be interpreted in terms of the multi-trace memory model. Whereas, in accordance with the model, irregular word reading is supposed to depend on global processing, non-word reading is supported by analytic processing which relies on the activation of segment-traces, i.e. the connections between orthographic and phonological sub-lexical segments. These segment-traces are created every time children succeed in dividing the phonological representation of a word into relevant phonological units (e.g. divide *chapeau* (hat) into /ʃ/-/a/-/p/-/o/), when they are confronted with both the spoken and written representations of words, and simultaneously process all of the letters of the sub-lexical orthographic units that respond to those phonological units (e.g. ch-a-p-eau)[4]. This creation of segment-traces demands that the VA span is large enough to process in parallel a sufficient number of letters. Hence, a severe VA span impairment will prevent the processing of large orthographic units, such as the (French) graphemes *eau* (/o/) and *oux* (/u/), and thereby impede the analytic processing that is necessary in order to decode non-words.

Summing up, according the multi-trace memory model, a VA span deficit is a possible cause of reading problems in both real word and non-word reading. More specifically, such a deficit is supposed to account for the different profiles that are found among dyslexics. A slight impairment of the VA span that makes it impossible to process in parallel all letters of a word but still allows the simultaneous processing of most graphemes will result in a developmental surface dyslexia profile. A more severe VA span impairment will, according to the researchers, lead to a mixed pattern of dyslexia, which is the most prevalent dyslexia profile (Bosse, Tainturier & Valdois, in press).

4.4.4 A discussion on the methodology of the experiment

The use of the chronological-age match of the control children in the experiments mentioned above goes against what was stated on methodology in the research on dyslexia earlier on in this paper, namely that experiments using this type of control match are unreliable, as the differences that are found by means of the experiment may very likely be a consequence of the impairment rather than a cause. In this particular case this means that the possibility that the visual attention span is trained by reading is not controlled.

Bosse, Tainturier and Valdois defend their choice for this particular match by referring to McDougall, Borowsky, MacKinnon and Hymel (2005). These authors demonstrated that the nature of the tasks used for the match have a significant influence on the study itself. Bosse et al. state that, in

[4] At this stage, the multi-trace memory model also accounts for a possible phonological coding deficit causing reading problems, as such a deficit would prevent this parsing process necessary for analytic processing acquisition, thereby impeding with non-word reading (Bosse, Tainturier & Valdois, in press).

this case, a reading-age match would interfere with the set-up of the experiments. In their view, a reading-age match that is based on speed and accuracy in real word reading (and includes irregular words) will reduce the possibility of finding the difference that was predicted by the multi-trace model (i.e. an impairment which would mostly affects irregular word reading) between the dyslexic children affected by a single VA span deficit and the control children. A match based on non-word reading, on the other hand, might increase the possibility to find significant differences between the impaired and normal readers with respect to VA span. On the other hand, this would make it impossible to compare these groups on the basis of their phonological abilities. The authors do admit that more research is necessary in order to identify the type of match that is necessary in order to obtain reliable results.

Another objection that could be raised is also countered by Bosse and her colleagues. This objection is that the information that needs to be processed in tasks testing the VA span is of a verbal nature, as the children were asked to report letter-names. Hence, one could argue, these tasks draw on phonological abilities and verbal short-term memory, which makes them unreliable for evaluating a VA span deficit. Bosse, Tainturier and Valdois, however, take the view that the effects of a possible phonological coding deficit on the whole and partial report tasks are negligible. In the first place, they refer to a study by Pelli, Burns, Farell and Moore (in press) which proves that performance in the whole report task is barely affected by a concurrent verbal short-term memory task. The second argument of Bosse et al. is that the errors produced in the whole report tasks seem to be visual, rather than phonological, confusions and, thirdly, the investigators claim that, as in the partial report task only a single letter has to be reported, it is unlikely that phonological short-term memory is a major factor. On the basis of these arguments, the authors draw the conclusion that the tasks which were used to assess whole and partial report in essence reflect visual attention. Still, the report task requires the retrieval of letter-names from long-term memory, another task on which a possible phonological deficit would have a negative influence. However, according to Bosse and her colleagues, difficulties with the retrieval of the phonological representations of the letter-names would specifically become apparent in the global report tasks, but this was not the case. Furthermore, since a number of the tested dyslexic children showed a VA span deficit but no phonological deficit and vice versa, they conclude that the report tasks are not susceptible to phonological difference and are, hence, adequate tools for measuring VA span. They nevertheless state that more research is needed to confirm this point.

The methodological issues addressed in this section make it clear that more research on a visual attention span deficit as a possible cause of dyslexia is needed. The criteria that are used to interpret the findings of the experiments still have to be refined and, furthermore, longitudinal and experimental studies are still needed to confirm the possible causal relation ship between visual attention span and reading acquisition. All this is necessary before a VA span deficit can be considered a potential second core deficit of developmental dyslexia, next to the phonological coding deficit.

5 Dyslexia in different languages

Within the domain of alphabetic orthographies, the English orthography poses particular difficulties to beginning readers, since it is what is called a 'deep' or 'opaque' orthography. This is an orthography in which the correspondences between the graphemes and phonemes are difficult to discover; they are often even inconsistent and allow for many exceptions. German and Spanish, on the other hand, are considered to be among the more 'transparent' orthographies, in which the links between letters and speech sounds are far more obvious. Consequently, children who are taught to read in these transparent orthographies are at an advantage in their reading development because it is relatively easy for them to detect the phoneme-grapheme consistencies and to develop phoneme awareness and will be able to reach a high accuracy level relatively early on (Harris & Hatano, 1999; Cossu, 1999).

This difference in the development of reading acquisition has several consequences. For a start, the predictability of reading skills on the basis of phonological abilities will differ according to the language. For instance, rhyming skill is a better predictor for reading ability in English than it is, for instance, in German (Wimmer, Landerl & Schneider, 1994). This has its effect on the type of reading problems that are associated with dyslexia, too. Many German dyslexic children, for instance, are shown to experience no significant difficulties when reading long unfamiliar words and non-words. Indeed, the influence of dyslexia on the reading performances of the German-speaking impaired children is found mostly in the fluency of their reading and their reading comprehension, as a result of the 'bottleneck' in the reading process (Wimmer, Mayringer & Landerl, 1998). It is obvious that the typically high accuracy levels in regular orthographies make it more difficult to recognize the core phonological deficits of dyslexia than it is in opaque orthographies. Hence, in order to identify impairments in these transparent orthographies one must, rather than turn to explicit phonemic awareness tasks, turn to tasks that demand implicit phonological processing, such as tasks evaluating verbal working memory (Wimmer, Mayringer & Landerl, 1998). On the other hand, this also means that some dyslexic children can 'overcome' their impairment in transparent orthographies such as German and may not even be considered to be dyslexics, whereas the same children would suffer considerable difficulties in an opaque orthography such as English.

However, not all languages make use of alphabetic scripts. In Chinese, for example, visual characters represent morphemes, not phonemes. As is to be expected, seeing that this type of orthography requires memorizing hundreds of complex visual symbols, it has been found that visual skills are more useful to predict reading ability in Chinese than in alphabetic orthographies. Phonological abilities, however, are predictors of reading performance in Chinese, too, but to a lower degree than in alphabetic scripts (Ho & Bryant, 1997). A small scale study on dyslexia in Chinese, carried out by Ho, Chang, Tsang and Lee (2002), showed that over half of the impaired readers in their investigation exhibited deficits in three or more cognitive domains, such as visual processing, phonological processing, rapid naming, etc. An association also existed between the number of deficits

and the degree of the reading and spelling impairment. These findings contribute to the idea that dyslexia in Chinese is not so much a result of a core phonological deficit, as it is in alphabetic orthographies, but that it is related to multiple deficits.

6 The biological basis of dyslexia

In the previous sections, we have mostly been concerned with the cause of dyslexia at the cognitive level. Investigators, however, have also made attempts to understand the biological basis that underlies dyslexia. We will now briefly look at some of the results that were derived from studies of the brain and genetic studies.

6.1 Studies of the brain

The first studies of the brain of dyslexics focused on the structure of the brain. Several differences were found in post mortem studies, but the results were tentative because, for obvious reasons, the sample sizes were small and there was no possibility for comparison with the brains of controls. Furthermore, using this method, it is difficult to control for individual differences (Vellutino, Fletcher, Snowling & Scanlon, 2004). The development of anatomical magnetic resonance imagining (aMRI) made it possible to 'map' the structure of the brain in an non-invasive manner. Using this method, attempts were made to replicate the main set of findings derived from post-mortem studies. These findings implicated that the temporal lobe (or planum temporale) of the left hemisphere, the area which is considered to support language functions, showed a symmetry with the left temporal lobe in the brains of dyslexic, whereas, in the brains of normal adults, the left temporal lobe is larger than the right one. Indeed, aMRI studies carried out by Schultz and his colleagues (Schultz et al., 1994) revealed a small reduction in the size of the left temporal lobes of dyslexics. In these studies age, gender and handedness were controlled in order to rule out possible causes of differences in the results of earlier experiments.

Currently, most neurological studies of dyslexia investigate the function, rather than the structure, of the brain. Technology has made it possible to examine the activity of the brain as a response to cognitive stimuli. This way, it is possible to determine which areas of the brain are engaged in certain processes and to assess possible abnormalities. Findings from positron emission tomography (PET), functional magnetic resonance imaging (fMRI) and magnetic source imaging (MSI) have made it possible to locate the areas that are crucial for reading and phonological processing. Moreover, differences in these areas of the brain between normal and dyslexic readers were found. Normal readers typically show activity in and an increase in the blood flow of the left temporal lobe during reading and phonological tasks. Dyslexic readers, however, do not show the same level of activity in these regions; they even exhibit a decrease in the blood flow (Rumsey et al., 1992; Paulesu et al., 2001). Thus, many investigations suggest that the phonological deficit affecting dyslexia may be originated by a different left hemisphere brain function in dyslexics.

6.2 Genetic studies

Not only have biological peculiarities been found in dyslexics, there are also reasons to assume that there is a genetic origin to dyslexia. Several studies have been concerned with the risk at dyslexia and have succeeded in establishing risk estimations of dyslexia on the basis of knowledge of affected relatives. According to Gilger, Pennington and DeFries (1991), a boy is at a 40 per cent risk of developing dyslexia if he has a dyslexic father and a 36 per cent chance if the mother is affected. A girl has a 20 per cent risk when one of her parents suffers from dyslexia. Since families also share environments, supplementary research is needed to determine whether genetic factors, and not all environmental factors, account for some of this 'heritability'. This research takes shapes in studies that compare concordance rates in the two types of twins: monozygotic (identical) and dizygotic (fraternal) pairs of twins. Monozygotic twins share 100 per cent of their genes; dizygotic twins share on average half of their genes. A concordance rate of dyslexia of 100 per cent in monozygotic twins and round 50 per cent in dizygotic twins would then mean that dyslexia is entirely hereditary. This does not seem to be the case but there is a higher possibility that both twins are dyslexic in identical than in fraternal pairs of twins. The twin studies demonstrated that reading abilities were influenced by both hereditary and environmental factors, with the former accounting for a substantially larger part of the variance than the latter (Olson & Gayan, 2001).

Some investigators have even tried to locate the 'faulty' genes that are responsible for dyslexia on the basis of linkage studies of families with many affected members. A first gene marker for dyslexia has been detected on the short arm of chromosome 6 (Grigorenko, 2001). On the long arm of chromosome 15, another area was detected. The evidence suggests that these genes are involved in approximately 30 per cent of families (Grigorenko et al., 1997). Finally, a potential marker on chromosome 1 has been suggested, but not yet replicated (Grigorenko, 2001).

7 Conclusion

The search for the cause of dyslexia, discussed in the third chapter of this bachelor paper, has led investigators to a consensus concerning the underlying cause of dyslexia at the cognitive level: weak phonological coding skills. This deficit is assumed to impede with the literacy acquisition of dyslexic children as it prevents them from developing the level of phonemic awareness that allows them to discover the consistencies between phonemes and graphemes. Hence, it is more difficult for dyslexics to set up grapheme-phoneme connections, which, in turn, makes both word identification and spelling more difficult for them. Even successful word decoding endeavours may thus demand so much from working memory that reading comprehension suffers a 'bottleneck' effect. Children learning to read in English are at a particular disadvantage since the English 'deep' orthography is rather inconsistent and demands 'more' phonemic awareness. A phonological deficit will thus be an even greater stumbling block in the reading development of children learning to read in such an orthography.

Recently, another possible explanation of dyslexia is contemplated. Investigators supporting the visual attention span deficit claim that a reduction in the number of distinct visual elements which can be processed in parallel will lead to a dyslexia profile. However, more research is needed before a VA span deficit can be considered a second core deficit of dyslexia, since the criteria that were used in the experiments providing evidence for this theory still need to be refined. In addition, the potential causal link between a VA span deficit and a reading impairment has not yet been proven. Hence, at least for the time being, the prevalent view on dyslexia still considers this specific reading impairment to be the result of an isolated phonological coding deficit.

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