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An Instrumental Single Case Study: The development of a Multi-Dimensional Interactive Model that Illustrates Barriers faced by a man with Developmental Dyslexia

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Abstract

The variety of difficulties that individuals with developmental dyslexia face makes it challenging to diagnose, to develop appropriate intervention strategies, and teach coping and learning skills. An instrumental single case study was used to explore the experiences of a young man who had been formally diagnosed with severe developmental dyslexia. The various barriers he faced because of having developmental dyslexia were examined. A multi-dimensional interactive model was developed from the results of the study, as well as from cases of people with developmental dyslexia diagnosed before and after this case study was conducted. This multidimensional interactive model illustrates the five primary barriers or factors, which a person with developmental dyslexia may have to deal with on a daily basis, and thus have to compensate for, in order to pass secondary school. All five factors form a continual flow or interplay between one another. This means that there is a constant influence of one or more factors on another. The model assists to illustrate the complexity of developmental dyslexia and the difficulty in diagnosing and treating the disorder, as each individual presents with a different set of difficulties or factors. This multi-dimensional model includes: the neurological factors, the intrapersonal factors, the interpersonal factors, the behavioral factors, as well as the emotional factors.

Keywords: Developmental dyslexia, brain-based disorder, barriers to learning, compensatory skills, multi-dimensional interactive model, instrumental case study.

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INTRODUCTION

Developmental dyslexia has been described as a life-long, neurologically based condition that is often inherited (Dyslexia International, 2018; Reid, 2011; Rutter, 2008; Snowling, 2000; Thomson, 2009). In addition, developmental dyslexia also affects reading, spelling, writing, motor skills, development of automaticity, emotions, and many other areas of functioning; and its nature changes with maturation and development (Alexander-Passe, 2010, 2015; Dyslexia International, 2018; Fletcher, 2009, Nicolson and Fawcett, 2010; Reid, 2011; Rutter, 2008; Scott, 2004; Snowling, 2001; Thomson, 2009). In his recent review of literature, Stein (2018) wrote that defining and diagnosis has generally stuck closely to the early 20th Century view that developmental dyslexia is the inability to learn to read despite normal oral and non-verbal abilities, with a strong hereditary (genetic) background. Hence, to diagnose dyslexia, we needed to show a big difference between a child's non-verbal and oral intelligence, and his reading abilitiesin short, a significant discrepancy. Fortunately, Stein (2018) postulates that, in the near future, recent advances in neuroscience should mean that much greater understanding of the causes of reading failure will emerge. So diagnosis should no longer depend on the demonstration of discrepancy between general oral and nonverbal reasoning abilities compared with very poor reading, which is at best only an indirect indicator of its underlying causes. Instead, we should be able to directly measure the deficient visual and auditory processing that causes the discrepancy in the first place, in order to make a diagnosis of dyslexia (Stein, 2018).

Snowling, Hulme & Nation (2020) wrote a fascinating article that developmental dyslexia is far more than just a reading, spelling and writing disorder. Snowling, Hulme & Nation (2020) query the validity of the discrepancy definition of dyslexia, which was also questioned by Stanovich as far back as 1992. Dropping the IQ-discrepancy definition of dyslexia has proven controversial for many, particularly for those who see 'dyslexia' as a special category of disorder and reject the view that it is only a reflection of poor reading. Snowling, Hulme & Nation (2020) argue that it is important is to have a better understanding of the dimensionality of reading disorders and how they frequently occur with other (co-morbid) difficulties. Therefore, individuals with developmental dyslexia are at a distinct disadvantage, especially when they have a more severe form of this disability, as they struggle to learn to read, and therefore perform poorly at school, despite intensive remedial intervention. Given the aforesaid, this may lead to several barriers and other co-morbid difficulties that they must compensate for, if they are to succeed, pass secondary school, and go on to tertiary studies.

In this article, an attempt to produce an appropriate model incorporating all of these aspects into one will be presented. This builds on previous work presented in an earlier article published in this journal (Holmes, Fourie, Van Der Merwe, Burke and Fritz, 2021).

LITERATURE

Owing to the complex nature of developmental dyslexia and unique combination of traits everyone may exhibit, it has taken many years for consensus to be reached about a universally recognised definition for this disorder (Holmes et al., 2021). Although most definitions cover similar basics, all agree that dyslexia is a brain-based disorder, characterised by a difference in the way the individual with dyslexia processes information.

Early diagnosis and treatment is vital, if individuals with developmental dyslexia are to reach their full potential. As a result of this early intervention, and the formation of new brain pathways due to brain plasticity, many go on to complete secondary school and eventually pass tertiary studies (Holmes et al., 2021; Reece, Booth & Jones, 2016). In his recent research, Stein (2018) concludes that:

"Developmental Dyslexia is a hereditary temporal processing defect, associated with impaired magnocellular neuronal development that impacts selectively on the ability to learn to read, leaving oral and non-verbal reasoning powers intact".

Armed with this definition, we should soon be able to test children specifically for these visual and auditory temporal processing deficits. This will not only enable us to diagnose dyslexia earlier, but also to set in motion remediation programmes tailored to each child's particular, individual, pattern of needs (Stein, 2018).

Snowling, Hulme and Nation (2020) propose that

"First, the term dyslexia should not be used as a shorthand for 'reading disorder' but should be used to refer to a difficulty with decoding and spelling fluency which is evident from the early school years and persistent over time. Second, it should affect academic functioning, such that progress is less good in literacy-based areas of the curriculum than that of peers in a similar setting. Third, if there are co-occurring features, these should be labelled as such but should not be considered core to the 'diagnosis'. Finally, the diagnosis should be qualified as mild (fully compensated when appropriate arrangements are in place), moderate or severe; we hypothesise that those with 'severe' difficulties are often those with a range of comorbid conditions".

The Information Processing Model (IPM) (Ashcraft, 2006; Anderson, 2005; Ehri, 1995; Frith, 2002; Hunt & Ellis, 1999; Morton & Frith, 1995; Seymour, 1997) and Causal Modelling Framework (CMF) (Frith, 1997, 1999, 2002; Morton & Frith, 1995) link suitably with the primary focus of this study, which is to identify what unique barriers and difficulties individuals with developmental dyslexia experience at all levels – biological, cognitive, behavioural and environmental; what compensatory skills they develop to cope with

these barriers; and thus go on to pass secondary school in spite of having this life-long condition. Additionally, the CMF model addresses a further focus of the study which was to determine how and by whom people with developmental dyslexia are supported to assist them to successfully complete their schooling and go on to tertiary studies.

Much research into the causes and effects of developmental dyslexia has been conducted worldwide over the past few decades. Moreover, a fair number of studies have discussed the use that people with dyslexia make of both positive and negative compensatory skills to cope better and to assist in this task. Furthermore, the earlier the intervention the more likely that people with developmental dyslexia will learn to read to some extent and succeed better at school, because of brain plasticity and the development of new neural pathways (Reece, Booth, & Jones, 2016; Reid, 2011; Spitzer, 2012).

The published literature and research findings that I accessed have been conducted in other countries, primarily in the UK, USA, various European countries, Canada, China, and Australia. I could find no studies conducted within the Sub-Saharan context on barriers caused by developmental dyslexia, as well as for those in the South African context, within which the study was conducted.

AIMS OF RESEARCH

An instrumental single case study was used to explore the experiences of a young man who had been formally diagnosed with severe developmental dyslexia. Firstly, the various barriers he faced as a result of having developmental dyslexia were examined. Secondly, the compensatory techniques and skills he used in order to learn to read sufficiently in order to pass secondary school, go on to tertiary studies and to realise his lifelong dream of becoming a pilot were also explored. These first two aims were discussed in detail in a previous article entitled "Developmental Dyslexia and Compensatory Skills: The man who could not read but learned to fly" (Holmes et al., 2021). Additionally, the study described how and by whom the male participant with developmental dyslexia was assisted to pass secondary school and go on to tertiary studies.

The final aim of the study was to create a new multi-dimensional, interactive model from the results of the study, as well as additional information from the researcher's exposure to other people with developmental dyslexia over the course of the research period, as well as prior to, and after this. This article will discuss the development of the model and how it can be used to assist people with developmental dyslexia, as well as the people who teach, help, and work with them.

The model assists to illustrate the complexity of developmental dyslexia and the difficulty in diagnosing and treating the disorder, as each individual presents with a different set of difficulties or factors. This multi-dimensional interactive model includes: the neurological factors, the intrapersonal factors, the interpersonal factors, the behavioural factors, as well as the emotional factors.

DEVELOPMENT OF A MULTI-DIMENSIONAL MODEL

Step 1: Integrating the Causal Modelling Framework (CMF) and the Information Processing Model (IPM)

In an attempt to construct a model to explain the barriers faced by people with developmental dyslexia, and how they compensated for these barriers, the CMF (see Figure 1) and the IPM (see Figure 2) were first integrated or combined. These were used in this study as part of the theoretical framework to better describe and explain the barriers and other difficulties the participant, Paul, faced because of having developmental dyslexia, and had to compensate for in order to learn to read sufficiently well to pass secondary school. This worked well and fitted with the literature study and theory, as illustrated in Figure 3.

The CMF (see Figure 1) was developed and expanded over a number of years, based on the premise that "dyslexia is a neuro-developmental disorder with a biological origin and behavioural signs which extend far beyond problems with written language" (Frith (2002, p. 45). When a person has developmental dyslexia; this can be traced back to a genetic or a brain-based abnormality, which is linked to neurology or brain function at the biological level (Frith, 2002).

The genetic or biological level is shown in yellow in Figure 1. This in turn, gives rise to cognitive deficits, which include specific deficits such as poor learning of the reading and writing system and many others (Frith, 2002). The cognitive level is illustrated in blue in Figure 1. These cognitive deficits lead to behavioural symptoms. These behaviours can be observed as poor literacy skills and specific impairments such as poor reading, spelling, writing difficulties and phonological difficulties (Frith, 2002). The behavioural level is illustrated in pink in Figure 1.

Interacting with all three of these levels – biological, cognitive and behavioural – is the environment illustrated in green in Figure 1, which would include input from school, teachers, experiences from home, parents, psychosocial problems, emotional difficulties and so on (Frith, 2002). All of these might affect and have an influence on the person's initial biological input or genetic/brain input (Frith, 2002), especially if one of the parents carried a genetic disposition for developmental dyslexia; if the mother smoked, drank or took drugs; if the mother did not have sufficient nutrients during pregnancy; or had some sort of illness that may have predisposed the foetus to some type of abnormality.

Therefore, environmental factors may affect the cognitive level if there is inadequate

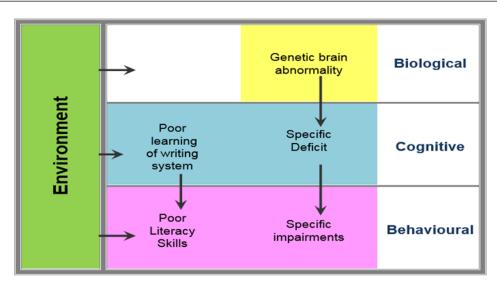


Figure 1: Causal modelling framework for developmental dyslexia. (Adapted from Frith, 1999, 2002)

early input, poor teaching, and insufficient follow up at home; or an inflexible curriculum (Frith, 2002). Environmental factors all affect learning either positively or negatively, and hence the behaviours, that is reading, spelling, writing, phonemic awareness and other difficulties that one might observe (Frith, 2002).

This framework should be seen as being fluid and flexible and incorporates many overlapping dimensions (Frith. 2002). This implies that some aspects such as phonological processing may have an impact on three levels – biological (neurological), cognitive and behavioural (educational) dimensions. It is also important to note that a cause of neurological origin does not mean that nothing can be done to assist a person with developmental dyslexia (Thomson, 2009, p. 106). The earlier the diagnosis is made, the earlier intervention may commence, as each person has unique deficits and individual differences (Nicolson & Fawcett, 2010).

The CMF provides an overall or common framework for developmental dyslexia. However, it does not give sufficient detail when exploring the difficulties people with developmental dyslexia experience with processing of information, as well as various specific cognitive deficits that are commonly found in people with developmental dyslexia. These may include poor short-term and working memory skills; as well as poor attention and visual and auditory processing difficulties. Thus, the IPM was included as part of the theoretical framework, to address the various cognitive processing difficulties that people with developmental dyslexia experience which the CMF does not include.

The IPM therefore adds to the CMF, as it addresses the cognitive processing difficulties experienced by people with developmental dyslexia at the cognitive level or domain of

the CMF in more detail. Furthermore, the IPM explains the complexity of the processing that must take place at the cognitive level; and how much can "go wrong", leading to the large variety of difficulties people with developmental dyslexia experience. Additionally, the IPM is one of the theories that explain how the brain processes and remembers incoming information. There are several theories of memory, but the most well-known, and most commonly used are the information processing explanations (Ashcraft, 2006; Anderson, 2005; Ehri, 1995; Frith, 2002; Hunt & Ellis, 1999; Morton & Frith, 1995; Seymour, 1997). A simplistic model of the IPM, which was adapted for explanation purposes, is shown in Figure 2.

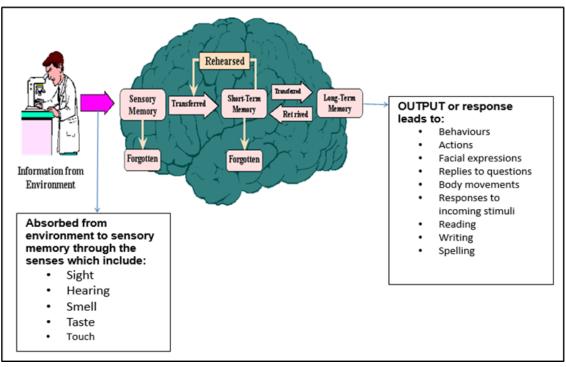


Figure 2: Information processing model (Adapted from APA, 2016; Baddeley, 2001; Woolfolk, 2007)

Stimuli or information from the external environment is received via the senses, which include sight, hearing, smell, taste, and touch, regarding our surroundings, which equates to the environment level or domain of the CMF (Woolfolk, 2007). This information is received into the first part of the information processing system called the sensory memory (APA, 2016). This first step is concerned with analysis of the stimuli which takes place in the sensory memory (Woolfolk, 2007). Thus, the sensory memory is the initial processing that transforms the incoming stimuli into information that makes sense to the person. Even though sights and sounds may last only fractions of a second, the sensory register or memory can briefly hold information for a duration of between 0,5 to 3 seconds for this initial processing to take place, before this information is forgotten (APA,

2016; Baddeley, 2001). The sensory memory can cope with 3 to 7 units of information at a time (Woolfolk, 2007).

The process of detecting a stimulus and assigning meaning to it is called perception (Woolfolk, 2007, p. 251). The person has to take more notice of the stimulus, pay attention to it, or perceive it, and assign meaning to it. Attention takes effort, as it means ignoring other stimuli, is a limited resource and most people can only pay attention to one cognitively demanding task at a time (Driscoll, 2005). If sufficient attention is paid to the incoming information this moves to the working memory (WM), also known as the short-term memory (STM), which has a memory capacity duration of between 5 to 30 seconds without rehearsal, practice or repetition, before it is forgotten (APA, 2016; Baddeley, 2001).

The WM has a capacity of 7 to 9 units of information (Woolfolk, 2007). It is the WM or STM which assists and directs the process of attention in the sensory memory. People with developmental dyslexia struggle with STM and WM, thus remembering oral instructions or more than one instruction at a time can be problematic for them (Reid, 2011, p. 5). Additionally, many struggle to maintain attention on the correct stimuli, and this is even further exacerbated if they have co-morbid attention deficit hyperactivity disorder (ADHD) (Reid, 2011, p. 6).

If the information is maintained, elaborated upon, and rehearsed, practiced, or repeated sufficiently, the information is able to be encoded, learnt or saved and then can move to the long-term memory (LTM) store, which has an infinite storage capacity (APA, 2016). In the LTM storage, coding and manipulation of data and stimuli occur, which can also be transferred back to or retrieved by the WM. The WM can thus activate and retrieve memories stored in the LTM (Woolfolk, 2007).

However, new information stored in the LTM needs to be organised and categorised, to understand and remember sequences of events. People with dyslexia often struggle with understanding, organising and categorising information which may negatively affect storage and retrieval of information from the LTM (Reid 2011). Information stored in the LTM is responsible for preparing an output process which leads to appropriate responses, behaviours, actions, facial expressions, replies to questions, or body movements, as a response to the original incoming stimuli. This equates to the behavioural level or domain of the CMF, and will also include reading, writing, and spelling (See Figure 1).

Therefore, to summarise, the information is encoded in the sensory memory where perception and attention determine what will be held in the WM or STM for further use. In the WM, new information connects with knowledge from the LTM, and can be activated to return to the WM.

Thus, there is significant synergy between the IPM and CMF, and if viewed together, they provide a relatively robust explanation of the underlying processes in developmental dyslexia. The two models explain the majority of the difficulties experienced by people with dyslexia as all sensory information comes from the environment, the cognitive processing takes place at a cognitive level, and the output occurs at a behavioural level. Additionally, the IPM clarifies dyslexia as a difference in how people process information. That is, how they take information in (input), how they understand it, memorise it and organise it in their mind (cognitive processing), and how they demonstrate they know this information (output) (Reid 2011, p. 3). People with dyslexia can therefore experience different difficulties at all or any of these stages above – input, cognitive processing, or output.

Many people with developmental dyslexia have sensory difficulties, especially in the visual and auditory area; short-term and/or working memory difficulties; as well as other processing difficulties (Reid, 2011, p. 4). This is part of the complexity involved when attempting to diagnose developmental dyslexia, as it may present very differently for each person. Generally, these differences can be seen in relation to print, but other areas of learning may also be affected. Therefore, dyslexia can persist even when reading skills improve.

In Figure 3 it can be seen that the IPM integrates into and further clarifies the cognitive domain of the CMF (Frith, 2002). The incoming stimuli that the senses receive come from the environmental domain, shown in green in Figure 1. The brain then processes the information cognitively which involves the short-term and working memory. This occurs in the blue section in Figure 1. The outputs can be seen as various behaviours, shown in pink in Figure 1. These behaviours could manifest as reading, spelling or writing deficits or even compensatory skills and strategies used.

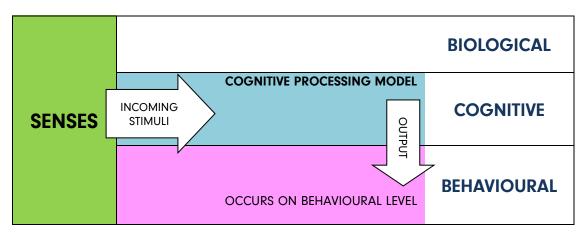


Figure 3: Integrated causal modelling framework and information processing model (as proposed by the researcher)

This integrated model of the CMF and IPM as shown in Figure 3, could explain in a limited manner, some of the compensatory strategies and skills used by Paul, on both the cognitive and behavioural levels, but did not show that they also occur at other levels simultaneously. It was not clear where to place emotions that emerged as a major theme from the data which posed an added dimension to the picture.

Step 2: Adding Emotions to the model

The second attempt at devising a model from the findings, comprises three levels of barriers linked to developmental dyslexia, each represented as three concentric circles placed one inside the other. These will be explained one level at a time and then the final combined model will be shown. It should be noted that emotions affect neurological or brain functioning as well as cognitive functioning, physical functioning, interpersonal and intrapersonal functioning; and are thus included in each of the levels, as was found from the data in this study as well as from research studies linked to trauma (Alexander-Passé, 2010, 2015; Levin, 1997; Perry, Pollard, Blaicley, Baker & Vigilante, 1995; van der Kolk, 2015).

Level 1: Compensating for biological or neurological, emotional and behavioural barriers linked to developmental dyslexia

The first circle or level 1 (illustrated in pink) represents the biological, neurological, hereditary, brain-based deficits or barriers that are linked to developmental dyslexia. This is shown in Figure 4. Additionally, these can be noted through observable behaviours. Emotions may have both positive and negative effects on brain function.

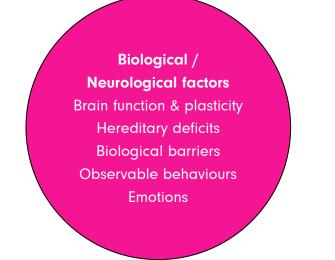


Figure 4. Biological or neurological, behavioural and emotional barriers linked to developmental dyslexia

Compensation can occur in various ways and can be seen through observable behaviours.

It should be noted that in this study, neurological changes could not be observed, as expensive scanning machines were not available, so interviews regarding hereditary factors/deficits and observable behaviours/characteristics had to be relied upon. The model for this area was based mainly on current research available, as well as the small amount of data captured during the interviews, and interaction with Paul and the other participants in the study.

The literature suggests that developmental dyslexia has a strong biological, hereditary origin, with various "defective" genes identified such as those found on chromosome 6 (Berninger, Raskind, Richards, Abbott & Stock, 2008; Cardon, Smith, Fulker, Kimberling, Pennington & De Fries, 1997; Fisher, Marlow, Lamb, Maestrini, Williams, Richardson et al., 1999; Gayan, Smith, Chemy, Cardon, Fulker, Brower, Olsen et al., 1999; Gilger, Pennington & De Fries, 1991; Grigorinko, Gombart, Fay, Bouazzaoui, Isingrini et al., 1997), on chromosome 15 (Smith et al., 1982; Grigorinko, et al., 1997), the DCDC2 gene and a variant on the gene regulator the READ1 within it (Gruen & Eicher, 2013; Meng, Powers, Tang, Cope, Zhang, Fuleihan et al., 2011), as well as the variant gene KIAA0319 (Cope, Harold, Hill, Moskvina, Stevenson, Holmans et al., 2005; Gruen & Eicher, 2013), which all lead to various deficits and behaviours. Paul likely inherited the dyslexia from his father and grandfather who both had significant signs of this disorder, although neither had been formally diagnosed. Which gene was responsible, is unknown as genetic testing was not conducted, and a number of genes have been implicated.

Neural pathways connected to memory may have degraded and volumes of grey and white matter fluctuate (Gogtay, Giedd, Lusk, Hayashi, Greenstein, Vaituzis et al., 2004; Lenroot & Gied, 2006; Schmithorst & Yuan, 2010). The cognitive assessment conducted, showed that Paul has deficits in short term and working memory, as the working memory scale on the Wechsler Adult Intelligence Scale (Wechsler, 1997) was in the average range compared to Paul's superior verbal skills on this test. Whether this discrepancy is due to degraded volumes of grey or white matter is unknown, as no scanning was done.

Decreased connectivity between the superior temporal regions and left inferior frontal regions, leads to difficulty processing speech sounds (Boets, Op de Beek, Van der Mosten, Scott, Gillebert, Mantini et al., 2013). Neurological, biological and brain-based deficits that may have occurred during development, such as irregular neuronal migration (Scerri & SchulteKőrn, 2009), visual stress (Evans, 2001; Everatt, 2002; Irlen; 1983; Meares, 1980; Reid, 2009; Wilken, 2003); cerebellar abnormalities, causing magnocellular deficits (Fawcett & Nicolson, 2004; Frith, 2002; Stein, 2008); structural differences in brain hemispheres (Galaburda, Rosen & Sherman, 1990; Hynd, Semrud-Clikeman, Lorys, Novey & Eliopulos, 1990); as well as the presence of misplaced cells in the outer layer of the cortex (Galaburda & Rosen, 2002; Knight & Hynd, 2002).

Many of these brain-based difficulties may well be present in Paul, but without brain scans, these are difficult to prove. However, the likelihood of Paul having these difficulties can be shown by many of the observable behaviours that Paul exhibited and link to Frith's Causal Modeling Framework (1999, 2002). The fact that Paul reported seeing words "float or move" across the page at times when reading is possible evidence of magnocellular deficits (Stein, 2001). Furthermore, Paul found phoneme-grapheme mapping challenging, which is also linked with possible magnocellular difficulties (Stein, 2001). Magnocellular abnormalities lead to deficits within the auditory and visual systems which then result in observable behaviours such as poor reading; poor tone discrimination; which leads to poor speech discrimination; as well as poor motion detection. Paul found the visual glare of the white paper made reading more difficult and was issued with green tinted glasses.

Fawcett, Nicolson and Dean (1996) noted that there are magnocells in the cerebellum and in the motor output systems, which make it difficult to distinguish the Magnocellular Deficit Hypothesis from the Cerebellar Deficit Hypothesis. Fawcett and Nicolson (1999,2001, 2004, 2008), Nicolson and Fawcett (2010), and Frith (1999, 2002) found that people with developmental dyslexia who had cerebellar impairments often had serious consequences and one could see these as observable behaviours such as poor rapid naming skills, balance difficulties, motor skills difficulties, and poor phonological awareness, all of which should have become automatic, but had not developed in many cases. These affect reading, writing, and spelling. Paul struggled his whole life with poor balance, poor fine motor skills, he still cannot catch a ball, and was teased as a child because he could not take part in ball sports – his hands would close before the ball reached him due to poor co-ordination; he took many years to tie his shoelaces and his tie; and riding a bicycle was extremely difficult for him. He had years of occupational therapy which helped a little, but he never fully gained complete control or automaticity of many of these skills.

Brain plasticity, is the brain's ability to form new and alternative pathways through intervention (Caleo, 2015; Keller & Roberts, 2009; Meyler, Keller, Cherkassky, Gabrieli & Just, 2008; Reece, Booth & Jones, 2016). The earlier the intervention, the more successful this is likely to be (Aylward, Richards, Berninger, Nagy, Field, Grimme et al., 2003; Simos, Fletcher, Bergman, Breier, Foorman, Castillo et al., 2002). Although Paul had a severe form of developmental dyslexia, through hard work, constant drilling, practice, the strategic use of colours, his senses and various study strategies, he managed to form new brain pathways, due to brain plasticity, and learnt to read sufficiently well (an observable behaviour) to pass high school and tertiary studies and become a pilot (Holmes et al., 2021).

Finally, in the absence of early support, emotional factors such as depression, anxiety, low self-esteem, isolation, loneliness, frustration, anger, poor reaction to criticism, amongst others; have an additive impact on dyslexia. These co-morbid conditions require compensatory skills to develop to cope with them and survive in the world (Alexander-Passe, 2009 a, b; Nelson & Gregg, 2012; Nicolson & Fawcett, 2010; Sundheim & Voeller, 2004; Thomson, 1995; Yoshimoto, 2005). Paul faced bullying, and almost all the emotions described above, which did affect his functioning negatively (Holmes et al., 2021).

Level 2: Compensating for intrapersonal barriers

The intrapersonal barriers experienced by the participant, shown in the purple circle in Figure 5 include all the cognitive barriers Paul faced, his personality, his disposition, his worldview, his self-esteem, his emotions, and other personal issues. These can be observed through various types of behaviours. Emotions are also included in this level. These intrapersonal barriers are illustrated in Figure 5.



Figure 5: Intrapersonal barriers linked to developmental dyslexia

Compensation had to occur because of the following intrapersonal barriers:

Executive control links Level 2 closely to Level 1. This link is shown in the combined diagram in Figure 7 below. The brain or neurological functioning from the frontal brain may lose many of its functions because of the developmental dyslexia (Gombart et al., 2016; Smith et al., 2016). However, certain executive functioning can be adapted, such as planning, shifting and inhibition skills and used to compensate for various deficits caused by developmental dyslexia (Cohen et al., 2007; Locascio, et al., 2010). Developmental dyslexia is primarily a difference in the way information is processed and so much of the compensation takes place at this level in some form. Moving information to the LTM through rehearsal and repetition was often more effective for Paul because of poor STM and WM.

Cognitive, learning, and behavioural compensation skills occur at this level. Learning how to study and learn using various techniques such as rehearsal and multi-sensory approaches are some examples of the compensatory skills Paul used to succeed (Banai & Ahissari, 2010; Fry, 2012; Reid, 2011). Personality and dispositional factors such as tenacity, courage, perseverance, determination, stubbornness, diligence, his competitive nature, as well as an internal drive to succeed and achieve best possible results contributed to Paul overcoming barriers to learning. Emotional difficulties such as dealing with bullying, teasing and name-calling, as well as being introverted and shy, hampered Paul's social interaction, and he had to learn to compensate for these.

Paul had to deal with and compensate for numerous co-morbid emotional conditions such as anxiety, depression, trauma, as well as dyspraxia, ADHD and loneliness. These resulted in low self-esteem and the need to prove his worth and value. He had to manage guilt and shame. Paul compensated and sought assistance by going for therapy with a psychologist, seeing a psychiatrist, taking medication, and allowing others to assist. Other coping skills that had to be learnt included: organisational skills, time management, difficulties with procrastination, as well as improving poor short-term and working memory. Compensatory strategies and other skills that assisted Paul included escaping from reality by going into his own imaginary world, using his imagination, listening to soothing music, his spirituality, and other relaxing pursuits such as going to gym.

Level 3: Compensating for Interpersonal Barriers:

Interpersonal barriers faced by Paul, shown in the circle in Figure 6, represented in blue, include the external environmental, cultural, language, social, home, family and school factors, which are all observable through various behaviours, actions or outputs, as well as emotions.

Compensation had to occur because of the following interpersonal barriers experienced by Paul because of having developmental dyslexia:

There were no official government/school policies in place for most of Paul's schooling career; the teachers were poorly trained; no concessions/accommodations were available to him until Grade 12; he had to endure hostile teachers and peers; no or very little assistance was offered to him at school; and there was a severe lack of knowledge and education about developmental dyslexia on the part of his teachers and the education system in general at that time in South Africa.

Paul's mother was his greatest asset but may have overcompensated. He had to learn to work alone without his mother's assistance when he left home and develop his own strategies and compensation skills.



Figure 6. Interpersonal barriers linked to developmental dyslexia

Other interpersonal barriers included environmental, cultural, language, social, school, family, emotional, and behavioural issues. Paul experienced language and cultural difficulties because, in the Afrikaans culture in which he grew up, boys were expected to play rugby. However, he was unable to play rugby because of poor co-ordination. Paul's home language is Afrikaans, which has a shallow orthography. Studying in English, which has a more complex orthography, at a tertiary level, proved to be a challenge for Paul. English is an international language used in flying, and it is compulsory for pilot training. There were high cultural expectations from family, peers, and self. His maternal family are highly educated, thus, there was emphasis on education and academic achievement within the family ethos. Paul was able to emulate this culture of learning and excellence, set for him by his maternal side of the family.

Paul experienced poor interpersonal and social skills outside of his home environment because he had limited time between studies to develop these life skills. Due to long hours studying, as well as poor co-ordination; he could not take part in sports with the other boys; so he did not develop appropriate peer relationships. He thus identified more with girls, his grandmother, his mother, his aunt, and sisters.

This led to social isolation, loneliness, guilt, shame, as well as humiliation and bullying by peers, especially boys. He tended to "use" and manipulate people and make friends with them to get what he needed. He became shy, introverted, and unsure of how to approach people. This resulted in him compensating and developing these skills late in his life. Many people assisted Paul during his school years as well as during his tertiary studies to compensate for the barriers he experienced because of the developmental dyslexia.

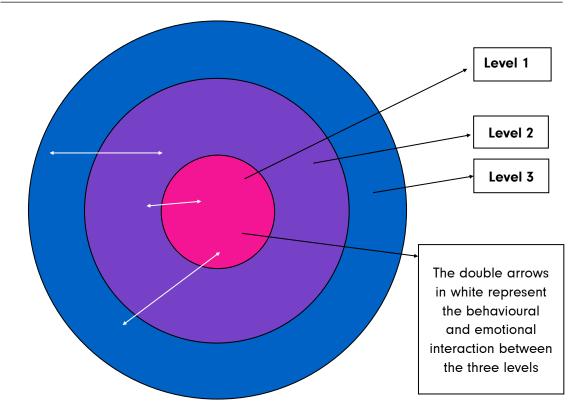


Figure 7: Combined model: Compensating for barriers linked to developmental dyslexia at three levels

Paul compensated by wearing a mask of competence. Participating in this research has given him the confidence to speak out and no longer be ashamed of having developmental dyslexia.

Behavioural and emotional factors have an influence on all three of the abovementioned levels and are illustrated with the red arrows in the combined model diagram shown in Figure 7.

BEHAVIOURAL AND EMOTIONAL FACTORS

Behavioural and emotional factors have an influence on all three of the abovementioned levels and are illustrated with the red arrows in the combined model diagram shown in Figure 7. Behavioural factors originate in each of the three levels and result in a myriad of different outward symptoms, which is why developmental dyslexia is so difficult to diagnose, as each individual person, may present with a set of unique combinations of symptoms and behaviours. These include poor automaticity; slow rapid naming speed; difficulties with direct lexical access; delayed and poor reading fluency and speed; poor spelling (spelling may be phonetically correct or bizarre/may spell the same word in various different ways); poor reading comprehension; confusion of left and right (laterality)/mixed handedness/ambidexterity; poor sequencing ability and sequential memory; poor organisational skills; poor short term and working memory skills; problems acquiring mathematical tables & mathematical word sums cause difficulties; auditory processing difficulties; poor phonemic awareness; poor fluency and reading speed; visual difficulties linked to magnocellular deficits; poor motor skills; poor balance; difficulties expressing ideas in written form although verbally strong; poor graphic (handwriting) skills; poor time management skills; slow/poor decoding ability even of familiar words; as well as poor ability to read whole words or remember sight words.

In addition to behavioural difficulties that occur on all three levels, individuals with developmental dyslexia can also be affected emotionally by being unable to learn as well as their peers, commonly resulting in low self-image, low self-concept, frustration, poor social skills, bullying, exclusion, loneliness, isolation, anxiety, and even depression (Alexander-Passe 2008, 2010; Edwards, 1994; Riddick, 1996). Nicolson and Fawcett (2000) note that a skill that would normally take a non-dyslexic child 400 hours to develop, learn and master, may take a dyslexic child up to 20 times longer to achieve. This could lead to frustration, loneliness, exclusion, and poor social skills as hours of extra work are required to master basic skills. Paul, the participant in this study experienced all these emotions which affected him on all three levels, as he, too had to work for hours longer than his peers to pass. Whilst they played sport and enjoyed themselves socialising, he had to go over everything that he had done at school every day and repeat things over and over to move the work to his better developed long term memory store. The quotation below illustrates just how difficult studying and learning was for the participant Paul.

"It comes with hard work and I had to put in far more effort than anybody else. At school I had to work ten times as hard as everyone else... it's impossible ... you, you're doomed. Now people can argue with me ... I'm telling you only a dyslexic will know you are doomed before you even started ... but then later I learnt to work smarter not harder, this is a difference, only four times more ... but it was far more productive" (Interview 2 lines 635–646).

Finalisation and limitations of a model for compensating for barriers linked to developmental dyslexia

Although the barriers Paul experienced have been broken down for simplicity and description purposes into the three different levels, there is a constant recursive pattern or feedback loop occurring between the levels which is difficult to fully explain in a twodimensional model. There is an ongoing interplay between the different barriers at all three levels which leads to different compensatory skills, because of the developmental dyslexia, which makes it is difficult to determine cause and effect. The barriers are described at different levels, and it became more difficult to separate one from the other. Recurring themes and barriers appeared as well as much overlapping, especially between levels 2 and 3. There was constant interplay between behavioural and emotional factors which occur at all three levels. This is shown in the combined model. (See Figure 7).

Two-dimensional combined model

The two- dimensional combined model shows the three levels combined into one. One can determine some causes on the genetic or biological level, but the resulting interplay between the social and environmental influences on the third level is crucial for development of personality. Additionally, this interplay reveals Paul's sense of self, cognition, and the way he learns to process information differently as a means of compensating for his barriers due to having developmental dyslexia. In the same way, developmental dyslexia itself and all the associated barriers, will also play a crucial role as to the type of personality and character a person develops, which will determine how he interacts socially and with his environment. This whole process is far more complex than it initially appeared to be.

Additionally, Paul's behavioural and emotional compensation skills, both positive and negative, thread their way throughout all three levels, and one sees different elements of these from both an internal and external context. The double arrows in red in Figure 7 represent the behavioural and emotional interaction between the three levels.

Thus, it was necessary to go "back to the drawing board" and to look at some other options. A more complex, multi-dimensional, interactive model began to develop, and this is how the final model evolved.

Step 3: A Multi-Dimensional, Interactive Combined Model

Multi-dimensional combined model: Process 1

The first attempt at forming a more complex multi-dimensional, interactive model is illustrated in Figure 8. This shows the five factors or barriers that Paul had to compensate for. However, this multi-dimensional model, takes the process shown in Figure 7 shown as three concentric circles; one step further, as it illustrates that there are multiple dimensions within which a person compensates for their barriers to learning. There are five factors or barriers that are at play here, which interact in a dynamic and continual flow of energy, which include neurological factors (shown in yellow); intrapersonal factors (illustrated in blue); interpersonal factors (in green); behavioural factors (in red) and emotional factors (in pink). Each of the first three factors interacts with the other in a continual flow of energy, as shown by the black arrows. Thus level 1, the neurological factors interact with level 2, the intrapersonal factors as well as level 3 the interpersonal

factors, as shown by the black lines. Similarly, there is a reciprocal interaction between each of these factors back with the others, thus the double arrow on the black line. In the same way, the behavioural and emotional factors interact with all three of the other levels 1, 2 and 3, simultaneously. This is illustrated in Figure 8. As one can see, the behavioural factors (shown in red) and emotional factors (shown in pink), interact with and penetrate through each of levels 1, 2, and 3; the neurological, intrapersonal and interpersonal factors respectively.

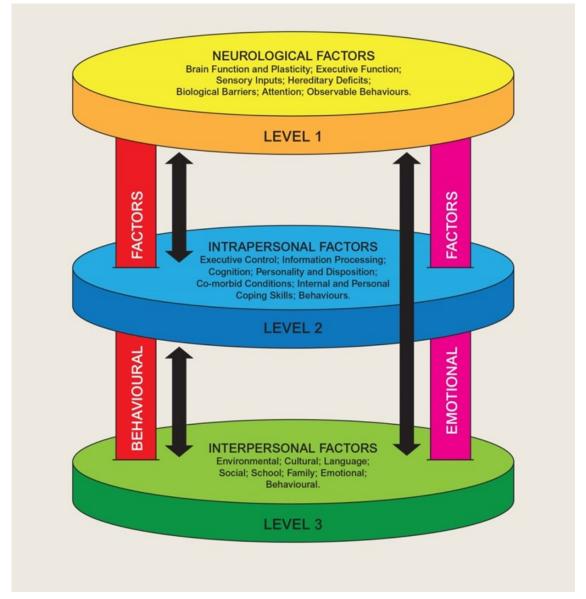


Figure 8: A multi-dimensional model explaining the barriers linked to developmental dyslexia: Process 1

Multi-dimensional combined model: Process 2

The black arrows did not seem entirely satisfactory, as they did not give the exact "feel" of a multi-dimensional interaction or flow of energy between the first three levels and the behavioural and emotional factors. The model was therefore refined and process 2 was developed, as illustrated in Figure 9. The red arrows, make the interaction between the five different factors or barriers, seem to be more three dimensional and makes the interaction appear to "flow" better, as illustrated in Figure 9

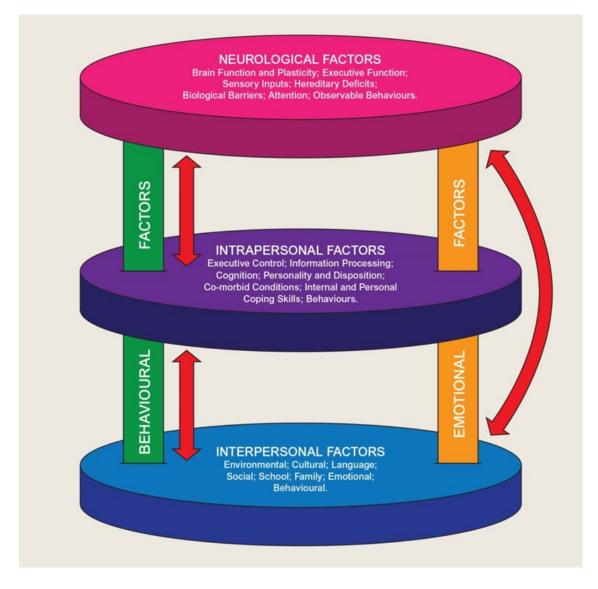


Figure 9: Multi-dimensional combined model explaining the barriers linked to developmental dyslexia: Process 2

The straight red arrows show the flow or interception between the neurological and intrapersonal factors and then the flow or interaction between the intrapersonal and interpersonal factors, respectively. The slightly rounded red arrow shows that continual flow or interaction of the interpersonal and neurological factors, as the model although multi-dimensional, makes it difficult to illustrate this interplay between these two factors, without "bending" the model over into an arch.

Three-dimensional combined model: Process 3

Although the multi-dimensional model shown in Figure 9 illustrates the "flow" of energy (red arrows) between the neurological (shown in pink), inter-personal (shown in blue), and intra-personal (shown in purple) factors or barriers, this model was still not completely satisfactory. A colleague showed the researcher a model she had bought at a toy shop which demonstrated the mixing of pink and blue coloured oil, which combined to form the colour purple. This triggered the idea of an hour-glass instead of the circles in the initial combined model, illustrated in Figure 10.



Figure 10: Image of an "hour-glass" toy

In the toy shop an "hour-glass" toy which contained blue oil was found, as shown in Figure 10. This single hour-glass toy was used to represent one of the factors or barriers to learning. Three of the hour glasses in different colours, were then placed one on top of the other to represent the neurological (orange), interpersonal (blue), and intrapersonal (green) dimensions of the model, as shown in Figure 11. In this manner, a three-dimensional, flowing form of the interaction of the five factors or barriers that Paul faced living with developmental dyslexia began to be conceptualised.



Figure 11: Image of three "hour-glasses"

However, because the three hour-glasses were sealed, it was impossible for the three colours to mix or "flow" between one another, or for the "energy" between the three factors or dimensions to be shared, as it was envisaged by the researcher. The three oils or liquids would have to be three different densities, so that they could mix and then separate again with the least dense at the top (orange) and the densest at the bottom (green), so that each colour could maintain its identity as a specific factor or dimension.

However, the emotions and behaviours that were included across all three of these dimensions could not be explained using the hour-glass model, so this explanation was too limiting.



Figure 12: Toy showing mixing of blue and pink oil to form purple

A more unusual toy was found, which showed the type of flow and mixing that was attempting to be illustrated by the model far better than the hour-glass models. This is shown in Figure 12.

The pink and blue colours interacted together and formed the colour purple, which better explained the flow of energy and integration between the different factors of the model in Figure 9. This "mixing of colours" also assisted to include the emotions and behaviours that the hour-glass model could not illustrate. However, even this model was too limiting, and further thought had to go into developing a more complex model.

This then led to the final model as illustrated in Figure 13 which shows the continual flow of energy and interaction between the five factors or barriers that Paul faced because of having developmental dyslexia.

Figure 13 Illustrates the final multi-dimensional interactive model that shows the dynamic interaction of the biological or neurological factors (in pink), the intra-personal factors (in purple), and inter-personal factors (in blue); while there is also a continual flow of energy and interaction between all three of these factors from the behavioural and emotional factors; which vary in colour, depending on with which of the three of these former factors they interact. This is a similar colour interaction illustrated by the toy in Figure 12, where the blue and pink combine to form purple. Compensation occurs continually and

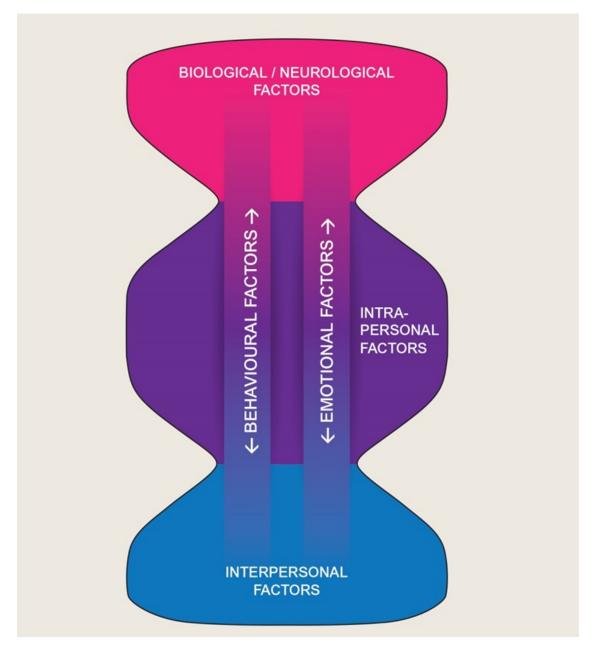


Figure 13: A multi-dimensional interactive model explaining the barriers linked to developmental dyslexia: Process 3

simultaneously as required, at some or all of these levels in a positive or negative manner in order to assist the person with developmental dyslexia to cope more effectively in life.

This explanation illustrates the complexities of the human brain. Furthermore, this model illustrates the interplay between the intrapersonal and interpersonal factors or barriers that a person with developmental dyslexia faces, and these cannot be reduced to a simple model. However, this model was developed for explanation purposes and to simplify and illustrate the level of complexity of the barriers Paul faced because of having developmental dyslexia, and thus the complexity of the compensatory skills and techniques he had to learn and use to cope with these barriers.

A three-dimensional (3D) computer-generated model may have illustrated this in a more meaningful manner. However, when an attempt was made to construct this 3D model using a computer programme, besides the fact that this was well beyond the researcher's computer expertise, it become too complex, and the explanation lost its meaning. Thus, this final third attempt at a model given in Figure 13 best suited this purpose.

Implementation of the model

It is evident from the aforementioned model (Figure 13) that the person compensating for their barriers linked to developmental dyslexia, must do so at five different levels, sometimes simultaneously at two or more levels. Firstly, the neurological or biological barriers, which include hereditary factors, brain-based hereditary deficiencies such as magnocellular and cerebellar deficits, executive function, attention, as well as visual stress, brain function and brain plasticity. Sensory input occurs through the neurological system and links closely to trauma as well as to sensory integration difficulties experienced by individuals with developmental dyslexia. Thus, one can see how complex and multi-dimensional the links are even within one of the levels. Secondly, intrapersonal factors, which include executive control functions, which links levels 1 and 2, information processing difficulties, cognition, personality and disposition, co-morbid conditions such as ADHD and motor difficulties, as well as internal and personal coping skills. Thirdly, the intrapersonal factors that include the environment, cultural and language influences, social and psycho-social interactions including those with family, peers, colleagues, and others such as teachers and lecturers. This factor also includes the educational system, policies, and other external factors. Fourthly and fifthly are behavioural and emotional factors, respectively.

It is recommended that the above barriers be addressed in the following manner because of the researcher's experience with Paul, and other people with developmental dyslexia of a wide variety of ages. Paul used a multi-sensory approach; which has been proven to work effectively for people with developmental dyslexia (Banai & Ahissari, 2010; Fry, 2012; Reid, 2011). His compensatory techniques in these areas when studying/ learning included drawing, making diagrams, talking to himself, teaching himself, listening to tapes, using different coloured paper, and using coloured pens and highlighters. He used his body (kinaesthetic approach) by walking around, moving his arms to follow sequences using the charts. He used authentic illustrations to assist him to remember what the instruments looked like in the cockpit as he rehearsed and memorised the sequences. Both children and adults learn far better using as many senses as possible, rather than using just one or two by reading (visual) or listening (auditory)(Banai & Ahissari, 2010; Fry, 2012; Reid, 2011).

Currently in South Africa, children and students diagnosed after a full assessment by an educational psychologist or other suitably qualified professional are entitled to various accommodations from Grade 4, provided they meet the criteria. These accommodations were first introduced in Education White Paper 6 (DoE, 2001) and then revised and updated when the Policy of Screening, Identification, Assessment and Support (SIAS) document (DoE, 2014) was introduced. Accommodations could include a reader, a separate venue, extra time, spelling concessions, the use of oral assessment, use of a scribe, or a computer, voice recognition software, and various other accommodations depending on the severity of the developmental dyslexia and other co-morbid conditions that occur. Paul and his teachers were unaware of these accommodations until he reached Grade 12, although they were already available from when he was in Grade 10 in 2001. He was only granted extra time, whereas he was entitled to a spelling concession, a reader, and a separate venue from Grade 10 (DoE, 2001). One will never know what Paul might have achieved if he had been given this opportunity. In the researcher's personal dealings with parents and teachers, many are unaware of the fact that children with developmental dyslexia and other difficulties are entitled to accommodations if they have been accurately assessed and diagnosed and qualify for these. This means many children are struggling unnecessarily.

Furthermore, dealing with emotional issues was essential for Paul to function better. Most children and adults with developmental dyslexia tend to have co-morbid emotional difficulties such as anxiety, depression, frustration, or anger, and may have been bullied or exposed to long-term trauma (Alexander-Passe, 2010, 2015; Nelson & Gregg, 2012; Scott, 2004). Seeing a psychologist, psychiatrist or neurologist and taking suitable medication if they require this is important for optimal functioning.

Similarly, it is essential that children with suspected dyslexia are screened and diagnosed as early as possible, as the sooner intervention begins, the more likely they are to learn to read more efficiently (Elbro, Nielson & Pietersen, 1994; Hulme & Snowling, 2009; Reid, 2009, 2011). At a younger age, the brain is more plastic and early intervention has been shown to be more effective than later intervention. Assistance and assessments by speech therapists, occupational therapists, and other professionals to address speech and motor difficulties will also minimise long-term effects in these areas.

Unfortunately, in the South African context, resources are severely limited, and many children and schools have no or very limited access to any of the above-mentioned specialists. This means that many children, students, and adults, will remain undiagnosed in South Africa and thus may never reach their full potential. This will probably result in their dropping out of school because of frustration and failure that may not be any fault of their own.

CONCLUSION

This study aimed to explore the experiences of a male with developmental dyslexia, and the barriers he faced because of having developmental dyslexia. Given the results of the study an attempt was made to develop a multi-dimensional, interactive model that showed the barriers faced by people with developmental dyslexia, and for which they have to compensate.

The current research models did not explain or incorporate the rapid advances being made and knowledge being generated by the brain-scanning techniques used in the last decade. Therefore, based on the assumptions about developmental dyslexia, and the barriers faced, and using the IPM and CMF as a sound theoretical basis to explain developmental dyslexia, I set out to develop a model to show the barriers faced by people with developmental dyslexia.

LIMITATIONS AND DIRECTIONS FOR FURTHER RESEARCH

One of the potential criticisms levelled against this model in Figure 13 is that the model could not possibly be valid if the data from only one person with developmental dyslexia had been used. This was, however, never the case from the beginning, as a large amount of data from other participants had already been gathered along with Paul's data; as the initial study was to be a multiple case study. However, the data from the participant "Paul" that was used to present the single case study was so rich and had so much depth, that the thesis was eventually based on his information. However, the model itself was developed with a far larger number of participants' information at hand.

A total of 8 participants' initial information was used, and as more data became available over the past seven years, the model could be recognised as more credible. In the future, it would be useful to follow up this article with additional information gleaned over the 7 year period from up to 30 people between the ages of 9 and 52 with developmental dyslexia, and how they compensated for their challenges. However, the written permission to use the information from these assessments, and interviews conducted (mostly for accommodation purposes), would have to be obtained from the people involved to make a further article ethical. This permission is in the process of being sought and the data is being processed along with other data from another South African colleague, Sandra Stark, who is the director of The Stark Griffin Dyslexia Academy of South Africa (SGDA); who will assist with additional data for a more comprehensive article to support the model and validate it further in the future.

It is hoped that through this study, that there will be increased awareness of the large numbers of children, students and adults in South Africa who could have developmental dyslexia. In a country where the mid-year population in 2020 was reported at 59.62 million (Statistics South Africa, 2020), and where approximately 10% of the population may have developmental dyslexia, this could be as many as 5.962 million people; the majority of whom may never be diagnosed, due to a severe lack of resources and access to necessary assistance. By educating teachers, parents and other professionals through workshops, courses and through internet platforms, it is possible to make them more vigilant and to teach them how to identify children or students who may have developmental dyslexia so that they can be sent for testing as early as possible. In this way, they can receive maximum early support and appropriate intervention through effective, tailor-made treatment plans, to achieve their optimum potential.

This process has already begun through training programmes for teachers throughout South Africa, specific lectures targeted at students at UJ, in the teaching and educational psychology programmes, as well as talks at conferences.

This may be a dream, to reach so many with so few resources, but to make a difference, one must start somewhere. One person cannot change the world, but we can start in the small area of influence where we live and work. It is hoped that the few thousand teachers and parents that have heard the dyslexia talks based on the research and literature summarised from this study; or attended workshops about developmental dyslexia: how to identify it and how to assist their children at home and in the classroom; may be a small drop in the ocean on a worldwide scale, but to each of those children, it has made a significant difference to how they function in the world and to their future prospects. Reaching a wider population with the publication of this article would move this dream one step closer to being achieved.

REFERENCES

- Alexander-Passe, N. (2006). How dyslexic teenagers cope: An investigation of self-esteem, coping and depression. *Dyslexia, 12*(4), 256–275. http://dx.doi.org/10.1002/dys.318
- Alexander-Passe, N. (2008). The sources and manifestations of stress amongst school-aged dyslexics, compared with sibling controls. *Dyslexia, 14*(4), 291–313. http://dx.doi.org/10.1002/dys.351
- Alexander-Passe, N. (2009a). Dyslexia, gender and depression: Research studies. In Hernandez, P.
 & Alonso, S. (Eds.) Women and Depression. (pp. 15–74). NY: Nova Science Publishers.
- Alexander-Passe, N. (2009b). Dyslexia, gender and depression: Dyslexia defense mechanisms (DDMs). In Hernandez, P. & Alonso, S. (Eds.) *Women and Depression*. (pp. 75–140). NY: Nova Science Publishers.

Alexander-Passe, N. (2010). *Dyslexia and depression* (1st ed.). NY: Nova Science Publishers.

- Alexander-Passe, N. (2015). Investigating Post-Traumatic Stress Disorder (PTSD) Triggered by the Experience of Dyslexia in Mainstream School Education. *Journal of Psychology & Psychotherapy, 5*(6), 215–225. doi:10.4172/2161-0487.1000215
- American Psychiatric Association. (2016) http://www.apa.org/Images/Forgetting_tcm7-93332.gif Accessed 5th July 2016.
- Anderson, J. (2005). *Cognitive psychology and its implications* (6th ed.). NY: Worth.
- Ashcraft, M. H. (2006). Cognition. (4th ed.). New Jersey: Prentice-Hall.
- Aylward, E., Richards, T., Berninger, V., Nagy, W., Field, K., Grimme, A., Richards, A., Thomson, J., & Cramer, S. (2003). Instructional treatment associated with changes in brain activation in children with dyslexia. *Neurology, 61*(2), 212–219. http:// dx.doi.org/10.1212/01.wnl.0000068363.05974.64
- Baddeley, A. (2001). Is working memory still working? *American Psychologist, 56*(11), 851–864. http://dx.doi.org/10.1037/0003-066x.56.11.851
- Banai, K., & Ahissar, M. (2010). On the importance of anchoring and the consequences of its impairment in dyslexia. *Dyslexia*, *16*, 240–257.
- Berninger, V., Raskind, W., Richards, T., Abbott, R., & Stock, P. (2008). A Multidisciplinary Approach to Understanding Developmental Dyslexia Within Working-Memory Architecture: Genotypes, Phenotypes, Brain, and Instruction. *Developmental Neuropsychology, 33*(6), 707–744.
- Boets, B., Op de Beeck, H., Vandermosten, M., Scott, S., Gillebert, C., Mantini, D., Bulthe, J., Sunaert, S., Wouters, J. & Ghesquiere, P. (2013). Intact but Less Accessible Phonetic Representations in Adults with Dyslexia. *Science*, *342*(6163), 1251–1254. http:// dx.doi.org/10.1126/science.1244333
- Caleo, M. (2015). Rehabilitation and plasticity following stroke: Insights from rodent models. *Neuroscience, 311*, 180–194. http://dx.doi.org/10.1016/j.neuroscience.2015.10.029
- Cardon, L., Smith, S., Fulker, D., Kimberling, W., Pennington, B., & DeFries, J. (1994). Quantitative trait locus for reading disability on chromosome 6. *Science, 266*(5183), 276–279. http://dx.doi.org/10.1126/science.7939663
- Cohen, L., Manion, L., & Morrison, K. (2007). *Research methods in education* (6th ed.). New York: Routledge.
- Cope, N., Harold, D., Hill, G., Moskvina, V., Stevenson, J., Holmans, P., Owen, M., O'Donovan, M., & Williams, J. (2005). Strong evidence that KIAA0319 on chromosome 6p is a susceptibility gene for developmental dyslexia. *American Journal of Human Genetics, 76*, 581–591.
- Department of Education. (2001). *Education White Paper 6: Special Needs Education: Building an inclusive education and training system*. Pretoria: Department of Education.
- Department of Education. (2014). *Policy on screening, identification, assessment and support*. Pretoria: Department of Education. http://www.education.gov.za
- Driscoll, M. P. (2005). Psychology of learning for instruction. (3rd ed.). Boston MA: Allyn and Bacon.
- Dyslexia International. (2018). Dyslexia International Basics for teachers. Dyslexia. *Dyslexia International.* Retrieved 2 January 2018, from http://www.dyslexia-international.org/ eCampus/ONL/EN/Course/Intro.htm
- Edwards, J. (1994). *The scars of dyslexia: Eight case studies in emotional reactions*. London: Cassell.
- Ehri, L. C. (1995). Phases of development in learning to read words by sight. *Journal of Research in Reading, 18*, 116–125.
- Eicher, J. & Gruen, J. (2013). Imaging-genetics in dyslexia: Connecting risk genetic variants to brain

neuro-imaging and ultimately to reading impairments. *Molecular Genetics and Metabolism, 110*(3), 201–212. http://dx.doi.org/10.1016/j.ymgme.2013.07.001

Elbro, C., Nielson, I., & Pietersen, D. K. (1994). Dyslexia in adults: Evidence for deficits in non-word reading and in the phonological representation of lexical items. *Annals of Dyslexia, 44*, 205–226.

Evans, B. J. W. (2001). *Dyslexia and Vision*. London: Whurr.

- Everatt, J. (2002). Visual processes. In G. Reid & J. Wearmouth (Eds.), *Dyslexia and Literacy: Theory and Practice*. Chichester: Wiley.
- Fawcett, A., & Nicolson, R. (1994). Naming Speed in Children with Dyslexia. *Journal of Learning Disabilities, 27*(10), 641–646. http://dx.doi.org/10.1177/002221949402701004
- Fawcett, A. J., & Nicolson, R. I. (1998). *The Dyslexia Adult Screening T*est. London: Pearson.
- Fawcett, A., & Nicolson, R. (1999). Performance of Dyslexic Children on Cerebellar and Cognitive Tests. *Journal of Motor Behaviour, 31*(1), 68–78. http:// dx.doi.org/10.1080/00222899909601892
- Fawcett, A. J., & Nicolson, R. I. (2001). Dyslexia: The role of the cerebellum. In A. J. Fawcett (Eds.), *Dyslexia: Theory and Good Practice*. London: Whurr.
- Fawcett, A. J., & Nicolson, R. I. (2004). Dyslexia: The Role of the Cerebellum. *Electronic Journal of Research in Educational Psychology*, 2 (2), 35–58.
- Fawcett, A. J., & Nicolson, R. I. (2008). Dyslexia and the cerebellum. In G. Reid, A. Fawcett, F. Manis, & L. Siegel (Eds.), *The Sage Dyslexia Handbook*. London: Sage.
- Fawcett, A. J., Nicolson, R. I., & Dean, P. (1996). Impaired performance of children with dyslexia on a range of cerebellar tasks. *Annals of Dyslexia*, 46, 259–283. https://doi.org/10.1007/ BF02648179
- Fisher, S., Marlow, A., Lamb, J., Maestrini, E., Williams, D., Richardson, A., Weeks, D., Stein, J., & Monaco, A. (1999). A Quantitative-Trait Locus on Chromosome 6p Influences Different Aspects of Developmental Dyslexia. *The American Journal of Human Genetics, 64*(1), 146–156. http://dx.doi.org/10.1086/302190
- Fletcher, J. M. (2009). Dyslexia: the evolution of a scientific concept short review. *Journal of the International Neuropsychological Society, 15*(5), 501–508.
- Frith, U. (1997). Brain, Mind and Behaviour in Dyslexia. In C. Hulme and M. J. Snowling (Eds.), *Dyslexia, Biology, Cognition and Intervention*. London: Whurr.
- Frith, U. (1999). Paradoxes in the definition of dyslexia. *Dyslexia*, 5(4), 192–215.
- Frith, U. (2002). Resolving the paradoxes of dyslexia. In G. Reid and J. Wearmouth (Eds.), *Dyslexia and Literacy, Theory and Practice.* Chichester: John Wiley & Sons.
- Fry, R. (2012). Improve your memory. New York: Career Press Inc.
- Galaburda, A. M. & Rosen, G. D. (2001). Neural plasticity in dyslexia: A window to mechanisms of learning disabilities. In J. L. McClelland & R. S. Siegler (Eds.), *Mechanisms of Cognitive Development: Behavioural and Neural Perspectives.* (pp. 307–323). Mahwah, NJ: Lawrence Erlebaum.
- Galaburda, A. M., Rosen, G. D., & Sherman, G. F. (1990). Individual variability in cortical organization: Its relationship to brain laterality and implication to function. *Neurophychologia, 28*, 529–546.
- Gayan, J., Smith, S. D., Cherny, S. S., Cardon, L. R., Fulker, D. W., Brower, A. M., Olson, R. K., Pennington, B. F., & DeFries, J. C. (1999). Quantitative trait locus for specific language and reading deficits on chromosome 6p. *American Journal of Human Genetics*, 64 (1), 157–164.
- Gilger, J. W., Pennington, B. F., & DeFries, J. C. (1991). Risk for reading disability as a function of family history in three family studies. *Reading and Writing: An Interdisciplinary Journal 3,*

205-217.

- Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., & Toga, A. W. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. Proceedings of the National Academy of Sciences of the United States of *America*, *101*(21), 8174–8179.
- Gombart, S., Fay, S., Bouazzaoui, B., & Isingrini, M. (2016). Age differences in reliance on executive control in fluid reasoning. *Perceptual and Motor Skills, 123*(3), 569–588.
- Grigorenko, E. L., Wood, F. B., Meyer, M. S., Hart, L. A., Speed, W. C., Shuster, A., & Paus, D. L. (1997).
 Susceptibility loci for distinct components of developmental dyslexia in chromosome 6 and 15. *The American Journal of Human Genetics, 60*, 27–39.
- Gruen, J. R., & Eicher, J. D. (2013). Imaging-genetics in dyslexia: Connecting risk genetic variants to brain neuroimaging and ultimately to reading impairments. *Molecular Genetics and Metabolism, 110* (3), 201–212. http://dx.doi:10.1016/j.ymgme.2013.07.00
- Holmes, L.C., Fourie, J. V., Van Der Merwe, M., Burke, A., & Fritz, E. (2020). Developmental Dyslexia and Compensatory Skills: The man who could not read but learnt to fly. *Asia Pacific Journal* of Developmental Differences, 8(1), 143-171. DOI: 10.3850/S2345734121000061
- Hulme, C. &, Snowling, M. J. (2009). *Developmental disorders of language, learning and cognition*. Oxford: Blackwell/Wiley.
- Hunt, R. R., & Ellis, H. C. (1999). *Fundamentals of cognitive psychology*. (6th ed.). New York: McGraw-Hill.
- Hynd, G., Semrud-Clikeman, M., Lorys, A., Novey, E., & Eliopulos, D. (1990). Brain Morphology in Developmental Dyslexia and Attention Deficit Disorder/Hyperactivity. *Archives of Neurology*, 47(8), 919–926. http://dx.doi.org/10.1001/archneur.1990.00530080107018
- Irlen, H. (1983). Successful treatment of learning disabilities. In: *91st Annual Convention of the American Psychological Association* August 1983. CA: Anaheim.
- Keller, S. S., & Roberts, N. (2010). Measurement of brain volume using MRI: software, techniques, choices and prerequisites. *Journal of Anthropological Sciences, 87*, 251–259.
- Knight, D. K., & Hynd, G. W. (2002). The Neurobiology of Dyslexia. In G. Reid & J. Wearmouth (Eds.), *Dyslexia and Literacy*. Chichester: Wiley & Sons.
- Lenroot, R., & Giedd, J. (2006). Brain development in children and adolescents: Insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioural Reviews, 30*(6), 718–729. http://dx.doi.org/10.1016/j.neubiorev.2006.06.001
- Levin, P. (1997). *Waking the tiger: Healing trauma: The innate capacity to transform overwhelming experiences.* Berkeley, CA: North Atlantic Press.
- Locascio, G., Mahone, E. M., Eason, S. H., & Cutting, L. E. (2010). Executive functioning amongst children with reading comprehension deficits. *Journal of Learning Disabilities, 43*(5), 441–454. https://doi.org/10.1177/0022219409355476
- Mears, O. (1980). Figure/ground, brightness contrast and reading disabilities. *Visible Language 14* (1), 13-29.
- Meng, H., Powers, N., Tang, L., Cope, N., Zhang, P., Fuleihan, R., Gibson, C., Page, G. & Gruen, J. (2010). A Dyslexia-Associated Variant in DCDC2 Changes Gene Expression. *Behaviour Genetics*, *41*(1), 58–66. http://dx.doi.org/10.1007/s10519-010-9408-3
- Meyler, A., Keller, T., Cherkassky, V., Gabrieli, J., & Just, M. (2008). Modifying the brain activation of poor readers during sentence comprehension with extended remedial instruction: A longitudinal study of neuroplasticity. *Neuropsychologia*, *46*(10), 2580–2592. http:// dx.doi.org/10.1016/j.neuropsychologia.2008.03.012
- Morton, J., & Frith, U. (1995). Causal modelling: A structural approach to developmental

psychopathology. In D. Cicchetti, & D. J. Cohen (Eds.). Manual of Developmental Psychopathology. (pp. 357-390). NY: John Wiley & Sons.

- Nelson, J. M. & Gregg, N. (2012). Depression and anxiety amongst transitioning adolescent and college students with ADHD, dyslexia, or co-morbid ADHD/dyslexia. Journal of Attention Disorders, 16(244). http://dxdoi.org/10.1177/1087054710385783
- Nicolson, R. I., & Fawcett, A. J. (2000). Long-term learning in dyslexic children. European Journal of Cognitive Psychology, 12, 357-393.
- Nicolson, R., & Fawcett, A. (2010). Dyslexia, learning, and the brain. (1st ed.). Cambridge, MA: MIT Press.
- Perry, B. D., Pollard, R. A., Blaicley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaption, and "use dependent" development of the brain: How "states" become "traits". Infant Mental Health Journal, 16 (4), 271-291.
- Rees, P., Booth, R., & Jones, A. (2016). The emergence of neuroscientific evidence on brain plasticity: Implications for educational practice. Educational and Child Psychology, 33(1), 8-19.
- Reid, G. (2009). Dyslexia: A Practitioner's Handbook (4th ed.). Chichester UK: John Wiley & Sons.
- Reid, G. (2011). Dyslexia: A Complete Guide for Parents and Those Who Help Them. (1st ed.). Chichester UK: John Wiley & Sons.
- Riddick, B. (1996). Living with Dyslexia: The Social and Emotional Consequences of Specific Learning Difficulties (1st ed.). London: Routledge.
- Rutter, M. (2008). Developing concepts in developmental psychopathology. In J.J. Hudziak (Ed.), Developmental psychopathology and wellness: Genetic and environmental influences (pp. 3-22.) Washington, DC: American Psychiatric
- Scerri, T., & Schulte-Körne, G. (2009). Genetics of developmental dyslexia. European Child & Adolescent Psychiatry, 19(3), 179-197. http://dx.doi.org/10.1007/s00787-009-0081-0
- Schmithorst, V., & Yuan, W. (2010). White matter development during adolescence as shown by diffusion MRI. Brain and Cognition, 72(1), 16-25. http://dx.doi.org/10.1016/ j.bandc.2009.06.005
- Scott, R. (2004). Dyslexia and counselling (1st ed.). London: Whurr.
- Seymour, P. H. K. (1997). Foundations of orthographic development. In C. A. Perfetti, L. Rieben, & M. Fayol (Eds.), Learning to read and spell: Research, theory, and practice across languages (pp. 319-337). Mawah, NJ: Lawrence Erlbaum Associates Inc.
- Simos, P., Fletcher, J., Bergman, E., Breier, J., Foorman, B., Castillo, E., Davis, R., Fitzgerald, M., & Papanicolaou, A. (2002). Dyslexia-specific brain activation profile becomes normal following successful remedial training. Neurology, 58(8), 1203-1213. http:// dx.doi.org/10.1212/wnl.58.8.1203
- Smith, S., Kimberling, W., Pennington, B., & Lubs, H. (1983). Specific reading disability: Identification of an inherited form through linkage analysis. Science, 219(4590), 1345–1347. http://dx.doi.org/10.1126/science.6828864
- Smith-Spark, J. H., Henry, L. A., Messer, D. J., Edvardsdottir, E., & Ziecik, A. (2016). Executive functions in adults with developmental dyslexia. Research in Developmental Disabilities, 56 (3), 197-213. https://doi.org/10.1016/j.ridd.2016.03.001
- Snowling, M. J. (2000). *Dyslexia* (2nd ed). Oxford: Blackwell.
- Snowling, M. J., Hulme, C., & Nation, K. (2020). Defining and understanding dyslexia: past, present and future, Oxford Review of Education, 46:4, 501-513, DOI: 10.1080/03054985.2020.1765756
- Spitzer, M. (2012). Education and neuroscience. Trends in Neuroscience and Education, 1(1), 1–2.

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http://dx.doi.org/10.1016/j.tine.2012.09.002

- Statistics South Africa (2020). Downloaded on 14th September 2020 from http:// www.statssa.gov.za/
- Stein, J. (2001). The magnocellular theory of developmental dyslexia. *Dyslexia, 7,* 12–36. http:// dx.doi.org/10.1002/dys.186
- Stein, J. (2008). The neurobiological basis of dyslexia. In G. Reid, A. Fawcett, F. Manis & F. Siegel (Eds.), *The Sage Dyslexia Handbook*. London: Sage.
- Stein, J. F. (2018). What is Developmental Dyslexia? *Brain Sciences, 8*(2), 26 DOI: 10.3390/ brain8020026
- Sundheim, S., & Voeller, K. (2004). Psychiatric Implications of Language Disorders and Learning Disabilities: Risks and Management. *Journal of Child Neurology*, 19(10), 814–826. http:// dx.doi.org/10.1177/08830738040190101001
- Thomson, M. (2009). *The Psychology of dyslexia. A handbook for teachers.* (2nd ed.). London: Whurr.
- Thomson, P. (1995). Stress factors in early education (pp. 5–32). In T. R. Miles, & V. Varma, (Eds.). *Dyslexia and Stress*. London: Whurr.
- Van der Kolk, B. (2015). *The body keeps the score: Brain, mind, and body in the healing of trauma*. New York: Penguin Books.
- Wechsler, D. (1997). WAIS-III (1st ed.). San Antonio, TX: Psychological Corporation
- Wilkins, A. J. (2003). *Reading through Colour: How coloured filters can reduce reading difficulty, eye strain, and headaches.* Chichester: Wiley-Blackwell.
- Wise, B., Ring, J., & Olson, R. (1999). Training Phonological Awareness with and without Explicit Attention to Articulation. *Journal of Experimental Child Psychology*, 72(4), 271–304. http:// dx.doi.org/10.1006/jecp.1999.2490
- Woolfolk, A. (2007). Educational psychology. (10th ed.). New York: Sage.
- Yoshimoto, R. (2005). *Gifted dyslexic children: Characteristics and curriculum implications.* Presentation at the 56th Annual Conference, IDA, Denver, CO, USA, November 9–12.

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