A literature review examining the impact of genetics on Additional Learning Needs, and implications for learners' academic success

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Abstract

This literature review examines the question of whether children whose parents have specific learning difficulties should be tested regardless of their own personal symptoms. There is significant evidence that specific learning difficulties have a genetic component and that targeted intervention improves the learner's academic results in the short to medium term. It is recommended that a longitudinal study into the genetic causes, and therefore the prevalence of specific learning difficulties be established. The review also recommends a longitudinal study of the efficacy of support mechanisms for specific learning difficulties to establish if they support a learner beyond the academic environment.

Keywords

Specific Learning Difficulties, Additional Learning Needs, genetics

Introduction

This topic has been chosen due to the researcher's personal experience of late diagnosis of specific learning difficulties (SpLD). This led to reflection on the wider implications of undiagnosed or late diagnosed specific learning difficulties on personal development, and the impact this may have on wider society.

As stated in the Additional Learning Needs and Education Tribunal (Wales) Act 2018, due to be enacted in September 2020, key stake holders have a statutory duty to make effective provision for supporting learners with additional needs (Welsh Government, 2018). However, Davies (2016) and Dauncey (2016) suggest that the current legislation surrounding Additional Learning Needs (ALN) is based on outdated

evidence, meaning those effected find it difficult to access provision effectively. In addition to this, the current testing regime is seen as not cost effective, overly bureaucratic, and time consuming for those concerned (Davies, 2016; Dauncey, 2016). Whilst the proposed bill advocates a multi-agency approach to supporting young people with ALN, it still requires teaching practitioners to identify those people who potentially have additional learning needs. It is then their responsibility to ensure that effective individual development plans are put in place to support these learners.

This literature review will address the question as to whether pupils whose parents have ALN should be tested, regardless of any symptoms they may display, and whether this testing regime would be more effective at supporting learners. In order to do this review considers two sub questions: first, is there sufficient evidence to determine whether ALN are heritable? Second, is there sufficient evidence to confirm that a diagnosis of ALN can result in an improved quality of life, and academic results, for the learner?

Definitions of SpLD

The (Welsh Government, 2009) states that;

> ‘The term additional learning needs describes learners with a diverse range of needs who require targeted support to enable them to access educational opportunities and fulfil their potential’

Estyn (2013) adds to this definition by stating that pupils with ALN can be listed in four distinct categories: those with Special Educational Needs, those who have disabilities, those who have medical needs, and those who have emotional, social, behavioural or mental health needs. Whilst an individual pupil’s needs can often fit into multiple categories, this literature review focuses on those pupils with Special Educational Needs. The most recent definition of ALN given in the SEND code of practice (DofE, 2015) draws upon the definitions given in the Education Act 1996, and states that special and/or additional educational needs concerns needs that require specific educational provision to be made for that pupil. This review will further refine that focus by concentrating on pupils with Specific Learning Difficulties. As stated by The Dyslexia-SpLD trust (2019) and The British Dyslexia Association (2019), SpLD

includes: Dyslexia, Dysgraphia, Dyscalculia, Dyspraxia (hereafter referred to as developmental coordination disorder), and Attention Deficit Disorder/ Attention Deficit (Hyperactivity) Disorder (ADD/ADHD). It is also worth noting that SpLD often shows significant comorbidity with autistic spectrum traits, including Asperger's Syndrome. Whilst this review will consider this factor where relevant, it will not be a primary consideration.

The review will first provide definitions of these five SpLD, before going on to examine the evidence regarding the heritability and genetic basis for these specific SpLD.

Reid (2016) notes that there is a distinct lack of a clear definition of dyslexia. However, he also observes that there is a clear grouping of characteristics that are indications of dyslexia, such as a processing difference in literacy acquisition, cognitive processing (particularly regarding memory), and discrepancies in educational performance.

This lack of a common definition of dyslexia is countered by the 2009 Rose review commissioned by the Department for Education, which defines dyslexia as: 'a learning difficulty that primarily affects the skills involved in accurate and fluent word reading and spelling' (Rose, 2009, p.29). This definition is the same as that provided by Reid (2016) and is utilised by the Department for Education when discussing dyslexia.

As discussed by Chu (1997), there are several terms used by a variety of professions to describe a pupil with hand writing difficulties, the most common being developmental dysgraphia. This is a written language disorder which affects the mechanics of writing, presenting as poor hand writing performance in pupils who are not showing either a distinct perceptual motor difficulty or a neurological disability (see Hamstra-Bletz and Blote's definitition as cited in, Engel-Yeger, *et al.*, 2009, p.182).

Landerla, *et al.,* (2004) state that due to the complexities of mathematics, even at a very basic level, there is little agreement on what constitutes the criteria for dyscalculia. However, they do note that a traditional definition would be that a pupil underachieves by at least two age groups on a standardised test and must experience disruption to their academic achievement, or daily, life as a result.

Landerla, *et al.,* (2004) also note that as standardised testing often tests a variety of facets before supplying an overview of mathematical ability, this allows a substantial margin of error. Kucian and Von Aster (2015) agree that there is little agreement on a formal definition of dyscalculia, and add that the condition presents as specific changes to the brains function and structure. As noted by Dalton (2004), research into dyscalculia is still at an early stage when compared with other forms of SpLD. However, it is recognised by the Department of Education as such, and is reported to have a far greater impact on an individuals job prospects than other SpLD.

As with many SpLD there is a lack of consensus regarding the definition of developmental coordination disorder. Dewey (1995) utilises a neuropsychological-based definition to define it as a disorder of gesture, specifically seperating developmental coordination disorder from other developmental disabilities of motor control or motor function, such as Cerebal Palsy. Polatajko and Cantin (2006) expand on this, stating that children with developmental coordination disorder often appear to be developing along normal parameters, but have difficulty with learning every day tasks (such as tying their own shoe laces), indicating potential negative impacts on their daily life.

The final SpLD to be defined is ADD/ADHD. Barkley, *et al.* (1990, p775) define this as 'developmentally inappropriate levels of inattention, impulsivity, and in some cases over activity'. They also argued that rather than being seen as two subsets of the same condition, ADD and ADHD should be seen as two separate conditions. Roberts, *et al.* (2015) supports this definition, noting that in current diagnostic criteria there are degrees of severity to ADD and ADHD. These may range from mild, where few symptoms are present and there is little impact on the individual's social or occupational functions, to severe, where symptoms have a marked impact on an individual's social occupational functioning.

Do Specific Learning Difficulties Have A Genetic Basis?

Due to the diverse nature of the subject area, this review will consider some of the evidence for hereditability for the disorders defined above, individually.

As stated by Dalton (2004) and Carrion-Castillo, *et al.* (2013) genetics are a significant causal factor in the likelihood of an individual demonstrating dyslexic traits. Schulte-

Körne, *et al.* (2006) suggest that for children with one parent who has dyslexia, the risk of them also presenting with dyslexia is between 40 and 60 percent. If other family members also show signs of dyslexia however, this risk increases significantly. Schulte-Körne, *et al.* (2006) also demonstrate that four candidate genes have been identified as being a significant contributor to dyslexic traits, suggesting that dyslexia is a heterogeneous disorder[1]. Nielsen, *et al.* (2016) support this view, and argue that phenotypic expression[2] plays a significant part in how dyslexia presents in an individual. However, as argued by Marino, *et al.* (2003), phenotypic expression also plays a significant part in the prevalence of dyslexia within a population, rather than merely how it presents in an individual. Populations whose language orthography is more transparent, such as Italian, tend to have lower rates of dyslexia than languages that are more opaque, such as English. Elliot (2015) argues that whilst there are clear genetic influences that cause dyslexia to present in a learner, environmental factors are equally as significant. These studies suggest that whilst genetics play a significant part in developmental dyslexia, merely having the genes is not a guarantee that a learner will present dyslexic tendencies or face significant issues in their development. Elliot (2015) argues therefore that many of the studies that have determined the genetic influence on dyslexia have only studied pupils that are already showing poor reading skills, casting some doubt on the reliability and replicability of these studies.

As discussed by Nicolson and Fawcett (2011), until recently developmental dysgraphia was considered to be another facet of dyslexia, therefore research into this SpLD is limited in both scope and quantity. Berninger (2004) states that much of the available research concerning dysgraphia focuses on acquired dysgraphia in adults, which has little to no genetic component or cause. Further to this, Dinehart (2015) argues that this lack of research is due to the perceived decline in the importance of handwriting amongst policy makers and, by extension, teachers. Due to these issues, much of the research available on the causes of developmental dysgraphia suggests

[1] One where the same disease or condition can be caused, or contributed to, by varying different genes or alleles.

[2] The observable physical or biochemical characteristics of an organism, as determined by both genetic makeup and environmental influences.

a significant comorbidity[3] with dyslexia (Betta and Romani, 2006; Zoccolotti and Friedmann, 2010; Berninger and Richards, 2010). However, Berninger and Richards (2010) do suggest that, when completing a spelling test under a functional magnetic imaging test, pupils with dysgraphia use different areas of the brain to those without dysgraphia and also to those with dyslexia. Whilst this literature review has not found a specific large-scale study that purely considers the genetic origins of dysgraphia, given the significant comorbidity with, and heterogeneous cause of, dyslexia, it would be reasonable to conclude that there is likely to be a high degree of hereditability for dysgraphia. However, this does need to be studied in greater depth. This conclusion is supported by Samango-Sprouse and Rogol (2002), who state that between one in 500 and one in a 1000 children a year are born with an extra sex chromosome – a genetic condition that causes known developmental issues, such as dysgraphia.

As discussed by Linden, *et al.* (1996), ADD and ADHD have a significant number of biological underpinnings and incidences of ADD and ADHD are far higher amongst family members than general population trends would suggest. Shelley-Tremblay and Rosen (1996) reported that ADHD in particular has clear inherited characteristics that may have been beneficial in our evolutionary past hence their presence in modern humans. Whilst these studies are almost twenty-four years old, they demonstrate that genetic underpinnings of SpLD have been acknowledged for a significant length of time.

Meulen, *et al.* (2005) suggest rates of heritability in twins of between .75 and .91. This potential high rate of heritability further supports the conclusion of a genetic component to ADD and ADHD. These assertions are supported by Cormier (2008), who states that a large number of familial studies have been consistent in demonstrating the genetic component to ADD and ADHD. These findings are also consistent with evidence of abnormal genes found in children with ADD and ADHD (Meulen, et al. 2005; Cormier, 2008). As with dyslexia, the heterogeneous nature of ADHD is well documented, however, displays of these traits are also reliant on the phenotypic expression of the genes (Nadder, *et al.*, 2001). Faraone, *et al.* (1998)

[3] The presence of one or more additional diseases or disorders co-occurring with a primary disease or disorder.

suggest that ADD and ADHD demonstrate a significant comorbidity with conduct disorder and oppositional defiance disorder. Nadder, *et al.* (2001) support this view and argue that this quartet of disorders should be considered as facets of a single disorder, whereby traits displayed are phenotypic expressions of the heterogeneous makeup of the disorder. Ronald, *et al.* (2008) report that there is also a significant level of comorbidity between ADD/ADHD and autistic spectrum disorder, with findings suggesting a genetic link to similar areas of the human genome responsible for causing these two traits.

There has been very little research into the role genetics play in an individual presenting with developmental dyscalculia. This may be due to the lack of agreement on a definition or potentially due to the complexities involved in diagnosing dyscalculia. However, Shalev, *et al.*'s (2001) study appears to demonstrate a definite familial predisposition to developmental dyscalculia. This predisposition manifests as an almost ten times higher chance of presenting with developmental dyscalculia than in the wider population. In addition to this, Shalev, *et al.* (2001) also suggest a comorbidity for developmental dyscalculia with other SpLD. This view supports Gross-Tsur, *et al.*'s (1993) findings, who additionally demonstrated that children with either fragile X Syndrome, Turner Syndrome, or phenylketonuria are all more likely to present with developmental dyscalculia. These conditions are heterogeneous illnesses that affect either intrauterine brain development or brain function. Gross-Tsur, *et al.* (1993) speculate that the genes responsible for causing fragile X Syndrome are a significant factor in the potential for developmental dyscalculia. Gross-Tsur, *et al.* (1993) and Shalev, *et al.* (2001) each note that developmental dyscalculia has a higher incidence rate amongst females than other SpLD. Shalev, *et al.* (2001) reported that in 66% of cases the maternal family member of the child proband[4] demonstrated dyscalculia tendencies, whereas 40% of paternal relatives showed these signs. However, it must be noted that their study sample was relatively small, and limited to two settings in Israel. Regardless, it is fair to suggest that their conclusions are robust and replicable. Despite the limited research into the genetic origins of developmental dyscalculia, a number of studies have been conducted on potential neuropsychological causes

[4] a person serving as the starting point for the genetic study of a family

(Aster, 2000; Butterworth, 2005; Wilson & Dehaene, 2007). These studies suggest a high comorbidity of developmental dyscalculia and poor visual spatial memory. Additionally, when using functional magnetic resonance imaging, significant differences are observable between those with developmental dyscalculia and those without, in the areas of the brain utilised when undertaking mathematical problems (Landerla, *et al.* 2004; Von Aster and Shalev, 2007; Rotzer, *et al.* 2008; Butterworth, *et al.* 2011; Kuhn, 2015). However, in all these cases there is not yet an underlying explanation for the changes in the proband's brain structure.

As discussed by Richardson and Ross (2000), Visser (2003), and Missiuna, *et al.* (2006) little is understood about the aetiology[5] of developmental coordination disorder, and many researchers believe it may not be a uniform disorder. This lack of understanding of the symptoms of developmental coordination disorder has led to widely differing sample types and thus to the causes of developmental coordination disorder. This view is supported by Lichtenstein, *et al.* (2010), who state that developmental coordination disorder shows a significant comorbidity with autistic spectrum disorder. This comorbidity is in the region of 70% of those with autistic spectrum disorder also demonstrating developmental coordination disorder traits. Zwicker, *et al.* (2012) also noted that developmental coordination disorder shows a significant comorbidity with ADD/ADHD. Both the Lichtenstein, *et al.* (2010) and Zwicker, *et al.* (2012) studies noted a significant level of heriditability for both conditions. Piek, *et al.* (2007) also discussed the significant comorbidity between developmental coordination disorder and ADD/ADHD. They also noted that developmental coordination disorder shows significant comorbidity with depression, which in children is also demonstrated to have a genetic cause. When considering the aetiology of developmental coordination disorder, Landgren, *et al.* (1998) provide some evidence that prenatal factors, such as premature birth or low birth weight, increase a childs risk of demonstrating developmental coordination disorder. However, this increased risk is not significant in percentage terms, and research does not consider the reasons for the childs low birth weight or prematurity. Richardson and Ross (2000) argue that whilst the hereditability of learning disorders demonstrate a

[5] The cause, set of causes, or manner of causation of a disease or condition.

clear genotypic cause, phenotypic expression – particularly through abnormal fatty acid and phospholipid metabolism – provides a more certain incidence of many SpLD and other psychological and neurological conditions. In this case, Richardson and Ross (2000) state that poor fatty acid intake in the diet, and abnormalities in phospholipid metabolism, exacerbate a child's predisposition to developing SpLD including developmental coordination disorder.

In summary, the hereditability of these SpLD is clear: through either mapping of the genome as demonstrated by dyslexia or ADD/ADHD, or through association due to the comorbidity with these SpLD. There are however some issues with the evidence regarding the heritability of these disorders. Firstly, all of the studies considering the heritable nature of these disorders start from a known proband who presents with either one or more disorder, and then works back through the familial line to discover a common vector. Whilst this can help determine the percentage of parents who have passed on this genetic material, it does not consider the number of parents who have not passed this material on or the reasons why it was not passed on. The second issue with the research is the focus on monozygotic and dizygotic twins. Though this type of study does alleviate some of the issues raised in point one, the sample sizes are usually relatively small and, given that the majority of twins are raised in the same environment, does not allow for the phenotypic expression on their particular set of genetic makeup. Whilst these issues are certainly limitations on the available research, they do not diminish the clear hereditable nature of these SpLD or limit the effectiveness of utilising this evidence as the basis for assessing learners for SpLD.

Does Additional and Effective Support Improve a Learner's Academic Prospects and Long-Term Wellbeing?

In order to effectively consider whether additional support can benefit a learner (both academically and in their wider health and wellbeing), it is first necessary to define two areas: some of the impacts of comorbidity issues surrounding SpLD, and the type of support that an individual may receive.

As noted in section three, SpLD has comorbid links with depression and poor episodic memory, which directly impact on a learner's academic abilities. There is also a plethora of research that suggests a number of learners with SpLD have poor

relationships with both their peers and family members, as well as: physical and mental health issues, and increased incidences of substance abuse, and criminal behaviour in both adolescence and adulthood (Humphrey and Mullins, 2002; Klassen, *et al.*, 2004; Karande, 2005; Karande, *et al.*, 2008; Burden, 2008; Rose, 2009; Coughlan, 2011 and Macdonald, 2012).

As discussed by Lindsay (2003), support for learners with ALN is designed to reduce the obstacles that an individual faces, and ultimately reduce discrimination that the individual may face. This definition is broadly in line with UNESCO's Salamanca Statement, which specifies that every child has the right to an inclusive education, to ensure that their uniqueness and individual learning needs are acknowledged and met so that they are allowed to thrive (UNESCO, 1994). It is also in keeping with the United Nations Convention on the Rights of Persons with Disabilities (CRPD), which aims to ensure that individuals with disabilities have adequate legal protection to take an active role in society (UN, 2006). However, Terzi (2014) states that access to support is not necessarily providing all learners equal access to the same education, but rather ensuring that all learners have the opportunity to access meaningful educational opportunities commensurate to their needs. Norwich (2017) states that support is multifaceted including the type of school a pupil attends, academic participation, social participation, and academic achievement. As the majority of pupils with the SpLD being considered in this literature review attend mainstream schools rather than specialised units, this review will focus on the benefits of support in academic participation and achievement for a learner.

Rose (2009) states that there is clear evidence that specific intervention programs increase the pace of learning for a child with dyslexia, to the point where they are at commensurate level with their peers. Brooks (2016) evaluates a number of schemes aimed at supporting learners with dyslexic traits with the specified aim of improving a learner's phonological processing abilities. From this evaluation, he concludes that even the best teaching style cannot help those with dyslexia catch up to their peers without specific interventions being put in place. Any interventions that are implemented must be highly structured in order to be effective, and must be an ongoing process in order to be continually effective as, if the support is removed, a pupil will either fail to make further progress, or potentially regress (Brooks, 2016).

As discussed by Polatajko and Cantin (2006), there are a number of strategies used to support learners with developmental coordination disorder. These interventions are broadly divided into two perspectives: deficit orientated, and task orientated. Deficit orientated intervention strategies focus on repairing a damaged system within a learner in order to allow them to function effectively within their environment. Task orientated intervention strategies focus on showing the learner strategies that help to improve their performance in order to complete a task more efficiently.

Martini and Polatajko (1998) analysed and replicated a study into Verbal Self Guidance. This strategy appears to show that by teaching learners to visualise a task using the '*goal, plan, do, check'* approach, the learner can significantly improve achievement in a given task over an extended period of time. However, it must be noted that the study Martini and Polatajko (1998) analysed, had a sample size of ten children while their comparative case study had four participants, and as such a far larger study would be required to be certain of its effectiveness. This approach is also promoted by Polatajko and Cantin (2006) as part of the Co-op approach to aiding learners with developmental coordination disorder. Missiuna, *et al.* (2006) suggest that parents have a significant role to play in supporting children with developmental coordination disorder, particularly by altering their environment to make everyday tasks smaller and therefore simpler to achieve. Gibbs, *et al.* (2007) also considered the deficit and task orientated strategies as a method of improving the individual's coordination. However, they also noted that this was only successful in the tasks that the learner had been given specific strategies for, and was not replicated across the spectrum of difficulties. Gibbs, *et al.* (2007) argue that rather than focusing on supporting a learner to achieve a task or action, intervention strategies should focus on developing learners' self-confidence to allow them to build confidence in dealing with their coordination issues.

Unfortunately, an extensive literature search has been unable to locate any similar large-scale reviews of intervention strategies for the remaining SpLD being considered in this review. However, there are a number of small scale studies that demonstrate the effectiveness of relevant interventions. As discussed by Linden, *et al.* (1996) EEG biofeedback training does appear to show significant improvements in both concentration levels and IQ tests for those learners diagnosed with ADD/ADHD. Whilst

it must be noted that this now dated study had a sample size of only 18, it is still reasonable to conclude that long term use of specific exercises to aid concentration levels in those with ADD/ADHD will ultimately improve their academic success rates. Cormier (2008) disputes this, stating that the only effective treatment for ADD/ADHD is medicinal. However, the efficacy of the medication is demonstrated for a maximum of 14 months and does have the potential for significant side effects. In contrast to both of these approaches, DuPaul, *et al.* (2011) suggest that rather than focusing on interventions that are done with or to a learner, educators should consider modifications to the classroom environment and assignment setting in order to support learners. This they argue increases the learner's engagement with a task and therefore improves their academic prospects.

As discussed by Gifford and Rockliffe (2012), the discrepancies in defining dyscalculia – and subsequently diagnosing those learners with dyscalculia – make determining the effectiveness of any intervention strategies fraught with difficulties. However, from their study it is clear that intensive instruction in mathematics can improve a learner's ability to a level where they may be on par with their peers. It is suggested though, that these learners will continue to experience difficulties throughout their academic life and will need more instruction than a learner without dyscalculia. An earlier study by Dowker (2005), discusses the initial success shown by two specific intervention strategies in improving learners' mathematical abilities with intensive instruction. In this case, however, it must be noted that at the time of writing, the programs had recently been developed and the number of participants in the programs was limited.

As discussed by Brunsdon, *et al.* (2005), there was a growth in support schemes for adults with acquired dysgraphia, that provided a significant evidence base for the efficacy of these schemes. However, as also discussed by Feder and Majnemer (2007), there are no specific schemes aimed at the support of learners with developmental dysgraphia that have had large scale empirical studies. Dankert, *et al.* (2003) state that developmental dysgraphia is considered a visual-motor skill deficiency, and that occupational therapy does appear to improve a learner's handwriting ability, and by extension their academic ability. They also note that the studies they were reviewing had not taken into account statistical effects of the sample size, or that the groups may have improved due to their natural maturation. Feder and

Majnemer (2007) also support the assertion that occupational therapy can aid a learner in improving their handwriting ability, but argue that the therapy must be a long-term process and appropriate to the learner's particular difficulties. A number of these strategies suggest significant long-term benefits and even potential cures for the individual, particularly for those with mild symptoms. However, Klassen (2001) discussed the lack of research into the long term efficacy of these programs for learners with SpLD. Later, Parker, *et al.* (2013) also highlighted the need for a longitudinal study into the long-term efficacy of specific interventions into treatments for ADD/ADHD.

Dyslexia Action (2013) note that the cost of strategies to support learners with literacy is significant, however, the estimated cost of illiteracy to the UK economy in 2009 was £2.5 billion. Illiteracy also has significant impacts on an individual's ability to take an effective part in society. Butterworth (2005) states that poor mathematical skills have an even more significant impact on an individual's long-term prospects than literacy issues do.

In summary it is clear that SpLD have long-term and potentially life limiting consequences for individuals. Whilst the potential issues noted above do not affect every individual in the same way or to the same degree, short-term interventions do have some success in mitigating the issues faced by individuals in later life, as long as the strategies are maintained. However, and as noted by Dyslexia Action (2013), due to the incredibly diverse nature of SpLD and the individuals affected, not all strategies are guaranteed to work with all individuals in 100% of cases.

Conclusion and Recommendations

In conclusion, whilst there is almost universal acceptance of the existence and prevalence of the SpLD considered in this literature review, it is clear that there is a distinct lack of consensus amongst educators, policy makers, and therapists concerning: how to define the SpLD, and the 'cut off points' for those whom may be diagnosed with the SpLD.

When considering this literature review's first sub question: 'is there sufficient evidence to determine whether additional learning needs are hereditable?', it is confirmed that both dyslexia and ADD/ADHD have genetic abnormalities at the root cause of the

condition. It is also clear that there is a significant amount of evidence that dysgraphia, dyscalculia, and developmental coordination disorder have a genetic component within their causation. However, it is apparent that phenotypic expression plays a significant part in both the individual presenting with the traits of a particular SpLD, and the severity of those traits.

When considering this review's second sub question: 'is there sufficient evidence to confirm that a diagnosis of additional learning needs can result in an improved quality of life and improved academic results for the learner?', it is also clear that current strategies available to educators, therapists, and policy makers have a significant positive impact on the individual's ability to succeed at a commensurate level with their peers. However, it is also clear that this support must be long-term, especially for those with the most severe impairments, and that it requires significant effort on the part of the individual to maintain the beneficial effects.

As discussed in beginning of the paper, the current system of obtaining support for a learner with ALN and SpLD is seen as overly bureaucratic and adversarial. Whilst the Additional Learning Needs and Educational Tribunal (Wales) Act should alleviate this, the process still relies on education professionals to identify learners with additional needs and secure adequate support for them. Mass screening of all learners entering school has often been dismissed as too inefficient to be worthwhile. By targeting those learners who have a known genetic predisposition to SpLD, a clearer picture of the actual rates of SpLD in the population may occur, meaning learners who need support may be able to access it earlier.

This review will now make two recommendations for future consideration. First, as discussed in section three, the case for the genetic cause of SpLD has not been definitively established. This is because most studies commence with a proband that presents with a known impairment, and then works through the familial line to establish a common vector (or relies on twin studies) to establish the genetic links. This review would recommend that a study of pupils currently diagnosed with one or more SpLD is established, with the view to then test those pupil's future offspring for SpLD. A study might then determine the exact rate of heritability for these SpLD, and ultimately determine the actual rates of SpLD in the population. Such a study could also establish the impact phenotypic expression has on the severity of any SpLD.

Second, as discussed in section four, the long-term efficacy of intervention strategies employed to support young people has not been definitively established. This review would recommend a large-scale longitudinal study that monitors learners who receive targeted support for SpLD. This would allow for the efficacy of programs to be fully assessed, and potentially support educators, therapists and policy makers in designing improved strategies to support learners in the future.

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