
THE FAMILY INCIDENCE OF A VISUAL- PERCEPTUAL SUBTYPE OF DYSLEXIA

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Visual processing problems in people with dyslexia is becoming a more accepted area of research, with most authorities accepting the possibility of at least some degree of visual impairment in large numbers of people with dyslexia (Skottun, 2000; Stein, 2001; Wilkins & Lewis, 1999). One area of investigation has centred upon the proposal by Irlen (1991a) of a specific visual-perceptual dysfunction, which has been called Irlen Syndrome (IS), Meares-Irlen Syndrome (Evans, 2000) or simply visual discomfort (Conlon, Lovegrove, Chekaluk, & Pattison, 1999), and is unrelated to skills normally assessed by an optometric examination (Evans, Busby, Jeanes, & Wilkins, 1995; Scott, McWhinnie, Taylor, Stevenson, Irons, Lewis et al., 2002). Symptoms of IS include a sensitivity to light, blurring and shadowing of letters and words, a doubling, merging or movement of print, eye strain and fatigue when reading, a restricted span of focus and problems focussing when reading or writing for an extended period of time (Irlen, 1991a; Meares, 1980).

It has been hypothesised that the identified symptoms may be related to retinal malfunction. Grosser and Spafford (1990) identified extra peripheral retinal cones in subjects with dyslexia, which they claimed may lead to letter images in peripheral vision competing with letter images in central vision. Irvine and Irvine (1997) suggested a variety of possible retinal problems for people with symptoms of IS, including signal interference between adjacent receptor cells and abnormalities in receptor distribution.

It has also been hypothesised that the identified symptoms could be caused by a deficit in the magnocellular visual neurological pathway (Demb, Boynton, Heeger, 1998), which may cause an overlapping of visual images between consecutive eye fixations when reading (Williams & Lovegrove, 1992). Magnocellular activation may be involved in suppressing the potential overlap of images between consecutive eye fixations, as well as playing an

important part in keeping the two eyes steadily fixed on each word (Stein & Talcott, 1999). Stein (2001) claims that a magnocellular deficit could be related to poor motion sensitivity and unstable binocular control, which could lead to reports of words moving around the page and appearing to merge, as is reported by people with symptoms of IS (Irlen, 1991a). Evans and colleagues (Evans et al., 1995; Evans, Patel, Wilkins, Lightstone, Eperjesi, Speedwell, & Duff, 1999) and Robinson and Foreman (1999a) found a higher incidence of binocular instability and eye movement problems in people with symptoms of IS. A number of studies have identified a diminished or delayed evoked potential for poor readers along the magnocellular pathway in response to moving stimuli (Brannan, Solan, Ficarra, & Ong, 1998; Kubova, Kuba, Peregrin, & Novakova, 1996). Functional imaging studies have found a reduced activation of the V5/MT area of the visual cortex, which is sensitive to visual motion and dominated by magnocellular input (Demb et al., 1998; Eden, Van Meter, Rumsey, Maisog, Woods, & Zeffrio, 1996).

Colour filtering is claimed to influence the functioning ability of the magnocellular pathway (Edwards, Hogben, Clark, & Pratt, 1996; Williams, LeCluyse, & Littell, 1996), and has been reported to reduce symptoms of IS (Harris & MacRow-Hill, 1999; Robinson & Conway, 2000), improve eye movement (Evans et al., 1999; Robinson & Foreman, 1999a) and lead to changes in visual evoked potentials for people with symptoms of IS (Lewine, 1999). Numerous controlled studies have reported improvements in reading with the use of coloured filters, although it should be emphasised that reported improvements in print clarity may assist learning to read, but are unlikely to lead to the development of word recognition skills without additional reading tuition (Kyd, Sutherland, & McGettrick, 1992; Robinson & Foreman, 1999b). These studies have reported improvements in reading when using coloured plastic overlays or coloured computer monitors (Croyle, 1998; Jeanes, Busby, Martin, Lewis, Stevenson, Pointon et al., 1997; Scott et al., 2002; Tyrrell, Holland, Dennis, & Wilkins, 1995; Wilkins & Lewis, 1999; Wilkins, Lewis, Smith, & Rowland, 2001; Williams et al., 1996), as well as improvements in eye strain, headaches and reading when using coloured lenses (Chronicle & Wilkins, 1991; Evans, Patel, & Wilkins, 2002; Irvine & Irvine, 1997; Lightstone, Lightstone, & Wilkins, 1999; Robinson & Conway, 2000; Robinson & Foreman, 1999a, b; Solan, Ficarra, Brannan, & Rucker, 1998; Wilkins, Baker, Amin, Smith, Bradford, Zaiwalla et al., 1999). A number of studies have used placebo controls (Bouldoukian, Wilkins, & Evans, 2002; Jeanes et al., 1997; Robinson & Foreman, 1999a, 1999b; Wilkins, Evans, Brown, Busby, Wingfield, Jeanes, & Bald, 1994; Wilkins & Lewis, 1999).

There has also been evidence of an association between fatty acid metabolism and visual symptoms in dyslexia similar to those described by people with IS (Irlen, 1991a). Richardson, Easton, McDaid, Hall, Montgomery, Clisby et al. (1999) found that signs of fatty acid deficiency were significantly correlated with visual symptoms when reading, and the checklist used to identify visual symptoms included many indicators of IS, such as sensitivity to light, headaches or eye strain while reading and a blurring, movement and doubling of print. Stein (2001) suggests that efficient magnocellular function is dependent upon the metabolism of fatty acids, with docosahexaenoic acid in particular found to improve photoreceptor function and visual acuity (Neuringer, Reisbeck, & Janowski, 1994). The retina contains the highest level of docosahexaenoic acid in the body (Horrocks & Yeo, 1999), and retinal malfunction, as well as magnocellular deficit, have been implicated as

possible causes of the symptoms of IS (Irvine & Irvine, 1997; Lewine, 1999). Robinson and colleagues (Robinson, Roberts, McGregor, Dunstan, & Butt, 1999; Robinson, McGregor, Roberts, Dunstan, & Butt, 2001) have identified a number of biochemical markers associated with the incidence of visual processing problems found in IS. The biochemical markers suggested an alteration in protein and tissue turnover that could be due to infection or stress; both of which can result in a dysregulation of fatty acid distribution (Horrobin, 1999). Significant differences were also found in a dietary derived fatty acid (elaidic acid), which has been linked to macular degeneration of the eye and can induce an alteration in long chain fatty acids.

The investigations cited above suggest that IS may be considered a visuospatial subtype of dyslexia. The validity of such a subtype has been further established by analysis of the familial incidence of symptoms. There is considerable evidence of a high family incidence of reading disability (Castles, Datta, Gayan, & Olson, 1999; De Fries, Alarcon, Olsen, 1997; Elbro, Borstrom, & Petersen, 1998; Scarborough, 1989). Familial analysis helps develop a fuller understanding of possible subtypes of reading disability (La Buda, De Fries, & Pennington, 1990) with the implication that different subtypes may relate to different pathological mechanisms (Galaburda, 1993; Smith, Pennington, Kimberling, & Ing, 1989). Examination of the causes of possible familial links may also help clarify the types of impairment and the likely specificity of their outcome (Smith et al., 1989), which in turn may lead to differential treatment strategies, improved identification of risks, and early recognition (La Buda et al., 1990).

This paper describes two investigations of the familial incidence of symptoms of IS. The preliminary study involved parents only of children referred for screening and identified with symptoms. The second study involved two samples. One sample included parents and siblings of children referred for screening. The other sample involved parents and siblings of children who had been identified with symptoms through mass screening of children at local schools. Prior to these studies, the analysis of familial incidence of symptoms of IS involved only one of the symptoms described by Irlen (1991a), namely, the frequency of headaches and migraine. Wilkins and Neary (1991) found a family history of headaches and migraine in 17 of 20 subjects, and MacLachlan, Yale, and Wilkins (1993) found a similar history in 32 of 40 subjects.

STUDY ONE

This preliminary study included 751 subjects (486 boys, 265 girls) identified as having IS (Robinson, Foreman, & Dear, 1996). These children ranged in age from 7 years, 6 months to 17 years, 11 months and were referred to the Special Education Centre at the University of Newcastle, Australia for screening for symptoms of IS. The initial referral occurred primarily as a result of school concerns about academic achievement, not because of knowledge of specific symptoms of IS or its possible genetic basis. While parents were sometimes aware of their own reading difficulties, they were not conscious of the specific symptoms of the condition until screened. For screening, the Scotopic Sensitivity Syndrome screening manual (Irlen, 1991b) was used. This manual has three sections. The first section involves a 32 item

parent questionnaire concerning visual and eye strain symptoms, as well as reading and writing strategies. The second section involves a series of visual tasks and the reporting of possible visual distortions experienced when undertaking these tasks. The third section involves the identification of plastic coloured overlays which are reported to reduce the identified distortions. This task is followed by a 20 to 30 minute reading session to identify whether improvements in oral reading fluency and accuracy occur when using the coloured overlay claimed to reduce most distortions. Positive validity studies of the Irlen manual have been undertaken by Gray (1999) and Tyrrell et al. (1995). A high reliability of colour choice has also been identified (Jeanes et al., 1997; Robinson & Foreman, 1999b; Wilkins, 1997).

For children with IS, identification of symptoms in other family members occurred concurrently with assessment of the targeted child. At least one parent attended the initial screening session, and attending parents were screened at this session. Parents not present at the screening session were asked to attend for screening at one of the two further sessions required to identify the particular frequency and density of colour claimed to most reduce visual distortion and eye strain symptoms. Those unable or unwilling to attend were contacted and questioned concerning the nature of their symptoms, using items from the questionnaire in the Scotopic Sensitivity Syndrome screening manual (Irlen, 1991b). If positive signs were obtained on more than half of the 32 items, these parents were identified as having symptoms of IS. Identifications obtained by questionnaire were classed as anecdotal, as distinct from those parents for whom identification was based on the full screening assessment.

Table 1 outlines the incidence of parents with symptoms from the Robinson et al. (1996) study, and specifies the proportion of parents fully screened and those where anecdotal interview was used.

Table 1: Parents with Symptoms by Method of Identification*

| | Screened | Anecdotal | Total | % |
|---|----------|-----------|-------|----|
| Mothers with positive symptoms | 370 | 3 | 373 | 50 |
| Fathers with positive symptoms ^a | 290 | 47 | 337 | 45 |
| Children with both parents positive | 70 | 12 | 82 | 11 |
| Children with either one or both parents positive | 578 | 50 | 628 | 84 |
| Children with neither parent positive | 105 | 18 | 123 | 16 |

*From Robinson, Foreman, and Dear (1996). ^aMissing data on two fathers.

Of the 751 children identified as having Scotopic Sensitivity/Irlen Syndrome, 628 (84%) had either one or both parents showing similar symptoms. This high family rate parallels the reported incidence of migraine/headaches in families of children with IS described previously (MacLachlan et al., 1993; Wilkins & Neary, 1991). The results are also similar to investigations of family incidence of reading disabilities (Scarborough, 1989; Smith, 1992, Lewis, 1990).

The question arises of whether the high number of parents found to have IS is a result of referral procedures. If parents with symptoms are more likely to refer their children for

screening, it would be expected that the number of parents screened as positive would be high. However, in most cases, parents were unfamiliar with IS and were surprised when it was suggested that they should also be screened. In a majority of cases, children were referred at the instigation of the school rather than of the parents. It could, therefore, be argued that the children referred were a random sample of the population of children with IS. However, this is not necessarily a safe assumption. Familial incidence may be inflated in a referred sample because some parents may be aware of their own symptoms and actively seek assistance. This initial study also did not screen siblings of subjects with symptoms. In addition, a proportion of parents in this study could not attend for screening of symptoms of the syndrome and had to be questioned by telephone contact.

STUDY TWO

The preliminary data from Study One suggested a possible genetically based underlying deficit in visual processing and that a more detailed study involving extended family members was warranted. Study Two sought to address the limitations of the first study by using a sample in which all siblings and parents were screened and which included both a group of subjects referred for screening and a group identified by mass screening in schools (Robinson, Foreman, & Dear, 2000).

The study involved 158 children, identified as having symptoms of IS, and their natural parents and natural siblings. This sample was divided into two groups, (1) a group of children referred by parents, teachers, and other professionals to the Special Education Centre at the University of Newcastle for concerns about academic achievement (Referred Group) and (2) a group of children identified by mass screening of Grades 3 to 6 in two local schools, one from the public school system and one from the private school system (Screening Group). Two different samples were taken to investigate the possibility of parental referral bias.

The Referred Group of children involved 125 participants and their families. The children ranged in age from 7 years to 14 years 11 months. The Screening Group included 33 children and their families. It should be noted that this screening involved all pupils in Grades 3 to 6 ($N = 251$); it was not restricted to students for whom there were school concerns about academic achievement. The children identified as having symptoms of the syndrome ranged in age from 7 years to 12 years 9 months.

Parents and siblings of children identified to have symptoms of the syndrome were screened in the same way as the identified children, using the same measure as in Study One (Scotopic Sensitivity Syndrome screening manual, Irlen, 1991b). This screening occurred concurrently with assessment of the identified child for any relevant family members attending the screening session. Parents and siblings not present at the screening session for the Referred Group were asked to attend and be screened at one of the two further sessions required for the identified child to determine the specific colour claimed to most reduce symptoms.

For initial screening of the sample identified at two local schools (Screening Group), a modified version of the individual screening battery (Irlen, 1991b) was developed to allow assessment of subjects in groups of five. A validity study of this modified battery by

Robinson, Hopkins, and Davies (1995) identified a significant difference in reading rate for high and low scoring subjects. For those children identified by the group screen as having a significant number of symptoms, an individual screening session for the identified child and all relevant family members (parents and siblings above the age of seven) was conducted at the Special Education Centre, or in the family home for those unable to organise full family attendance at the Centre. Individual screening was conducted using the Scotopic Sensitivity Syndrome screening manual (Irlen, 1991b).

Tables 2 and 3 provide details of parents and siblings with symptoms for both the Referred sample and the Screening sample. Both samples provided similar estimates of parental incidence to those reported in Study One (Robinson et al., 1996), with one or both parents showing similar symptoms in 81% of families in the Referred Group and in 85% of families in the Screening Group.

Table 2: Parents with Symptoms for the Referred Sample (N = 125)*

| | <i>n</i> | % |
|---|----------|----|
| Mothers with positive symptoms ^a | 66 | 54 |
| Fathers with positive symptoms | 65 | 52 |
| Children with both parents positive | 31 | 25 |
| Children with either one or both parents positive | 100 | 81 |
| Children with neither parent positive | 24 | 20 |

*From Robinson, Foreman, and Dear (2000). ^aMissing data on two mothers (one father positive and one not).

Table 3: Parents with Symptoms for Sample Screened in Schools (N = 33)*

| | <i>n</i> | % |
|---|----------|----|
| Mothers with positive symptoms | 21 | 67 |
| Fathers with positive symptoms | 15 | 45 |
| Children with both parents positive | 8 | 27 |
| Children with either one or both parents positive | 27 | 85 |
| Children with neither parent positive | 5 | 15 |

*From Robinson, Foreman, and Dear (2000)

In addition to a high incidence of symptoms in parents, there was a high incidence of symptoms in siblings. For the Referred sample, there were 91 families who had siblings over the age of seven years and thus who were eligible to be screened. Sixty-nine (76%) of these families had siblings with symptoms. For the Screening sample, 26 families had siblings eligible to be screened with 14 (54%) of these families having affected siblings.

DISCUSSION

In Study One and in Study Two, a similarly high familial incidence of symptoms was identified, with 84% of one or both parents showing similar symptoms to their children in

Study One, 81% of one or both parents showing similar symptoms to their children for the Referred sample in Study Two, and 85% for the Screening sample. This high family rate is similar to the previously reported incidence of symptoms of headaches and migraine in families of subjects with IS (MacLachlan et al., 1993; Wilkins & Neary, 1991). The results are also similar to investigations of family incidence of reading disabilities. Scarborough (1989) claimed that familial risk would allow successful prediction in 73% to 81% of subjects, while Smith's (1992) extensive analysis of nine families identified a 79% incidence in cases where people with reading disability had married. Longitudinal data for 34 children of dyslexic families (Scarborough, 1990) identified 22 (65%) who had developed reading problems. Similarly high estimates of incidence come from Lewis (1990), whose examination of the pedigrees of four families found 68% of the nuclear family affected, and Hornsby (1984) who reported that 88% of children attending a dyslexia clinic had a positive family history.

The question as to whether the high incidence rate of IS found in families referred to the Special Education Centre is biased by parents' awareness of their symptoms and tendency to seek assistance, is answered by the similar incidence rates for both the Referred and Screening samples in Study Two, which suggest such a bias does not occur. While parents were often aware of their own reading difficulties in the Referred sample, they were not conscious of the specific symptoms of the Syndrome and, in most cases, children were referred at the instigation of the school and other professionals rather than by the parent.

The high familial incidence of symptoms provides support for the concept of a visual-processing component underlying reading and spelling problems. This concept was indicated in a study by Childs and Finucci (1979) who found that a visual-spatial form of dyslexia (difficulties with word shapes and patterns) was likely to be found in other relatives. Omenn and Weber (1978) also found 10 of 18 relatives of subjects with a visual-spatial disability had similar symptoms. Ho, Gilger, and Decker (1988) found that six of nine monozygotic twin pairs were concordant for a genetic-dyslexic subtype which included spatial skills, while Castles et al. (1999) noted that reading deficits were significantly heritable for visual subtypes. An analysis of twin data by Olson, Forsberg, and Wise (1994) found significant independent genetic influences, with the clearest separation found between phonological decoding and orthographic coding.

The results from these studies suggests that family history would be a useful factor in the identification of children likely to have visual processing problems and reading difficulties (Volger, De Fries, & Decker, 1985). The analysis of family histories would also help professionals to make parents more aware of their own possible symptoms. If parents identify symptoms in themselves, diagnosis of their children would be facilitated and more parental support and advocacy is likely to be generated through a better understanding of the implications of the symptoms for their children.

The visual processing subtype of dyslexia (IS) identified by these studies now needs to be further validated by chromosome analysis (Stein, 2000) or biochemical analysis (Robinson et al., 2001). Linkage analysis could ascertain whether there is a specific set of genes controlling the phenotype (Grigorenko, Wood, Meyer, Hart, Speed, Shuster et al., 1997), while biochemical analysis could ascertain whether there is a distinct pattern of related biochemical anomalies.

Preliminary biochemical analyses have been based on the suggestion that visual processing problems in people with dyslexia might be linked to an abnormality in the metabolism of fatty acids (Richardson, Calvin, Clisby, Schoenheimer, Montgomery, Hall et al., 2000). Robinson et al. (2001) identified biochemical markers related to the incidence of symptoms of IS. These markers included a dietary derived fatty acid (elaidic acid) and indicators of immune system dysfunction, which may influence fatty acid metabolism (Horrobin, Glen, & Hudson, 1995). Immune system problems have also been associated with dyslexia (Galaburda, 1997; Hardman & Morton, 1991; Knivsberg, 1997). Data from the Robinson et al. (2001) study suggests that the biochemical differences may be driven by an infective agent, however, the host's response to this infection would be under the control of the host genetics and/or other environmental influences, with genetic control likely because of the high familial incidence of IS. Anomalies in fatty acid metabolism may also be a factor in a wide range of disorders (Richardson & Ross, 2000), which may partly explain the high degree of co-morbidity between conditions such as Attention Deficit Hyperactivity Disorder (ADHD) and dyslexia (Hardman & Morton, 1991).

The suggestion of immune system dysfunction from the Robinson et al. (2000) study also raises the possibility of the familial transfer of infective agents. While viruses such as Human Herpes-6 (HHV-6) are not transferred across the placenta, children usually contact HHV-6 infections in the first couple of years of life from other family members. This is important as HHV-6 may play some role in alteration of retinal function. Qavi, Green, Lusso, Pearson, and Ablashi (1996) showed that HHV-6 was able to infect corneal epithelial cells, and Arao, Soushi, Sato, Moriishi, Ando, Yamada et al. (1997) found that HHV-6 was able to infect retinal pigment epithelial cells. Thus the alteration in visual processing may be associated with a persistent viral infection by HHV-6.

If specific biochemical anomalies can be identified, early identification would be possible. Robinson, Sparkes, Roberts, Dunstan, McGregor, and Conway (2002) found a high degree of accuracy in identifying subjects with symptoms of IS through biochemical profiling, with control and IS subjects correctly predicted in 85% of cases. Early diagnosis would allow professionals to better inform parents of the likely implications of this disability for literacy and learning difficulties. Early identification is important for the 10% to 20% of the school population with a reading difficulty, as lack of early reading success can lead to discouragement, a passive learning style and further failure (Wong, 1986). If identification of literacy problems is left until significant failure occurs, then the academic gap between failing students and their peers may be too great to overcome (Foorman, Francis, Fletcher, Schatschneider, & Mehta, 1998).

Such an analysis may also help identify symptoms which are the cause of the disorder, rather than the result of the disorder. With current diagnostic categories, the behavioural symptoms of disabilities such as dyslexia and ADHD are predominantly treated as the cause, with students being told to "try hard" or "concentrate more", which is likely to have a minimal effect if they cannot concentrate (ADHD), or have trouble with words moving and merging (IS). Biochemical analysis may also help to highlight the fact that overlapping disabilities may mean multiple treatments are required (Hardman & Morton, 1991). Identification and treatment of ADHD, for example, might mean that the possibility of other disabilities and treatments is not considered. Medication for ADHD may lead to improved

attention, but academic and reading achievement may still be limited if the student also has a distortion of print while reading (IS). The development of more effective diagnostic categories through biochemical analysis could also allow a more rational evaluation of the most effective treatment strategies. The broad diagnostic categories currently used are likely to result in a variety of disabilities, or sub-groups of a disability, being present in any one study population (Farmer & Klein, 1995; Torgesen, 1998). As a consequence, when researchers attempt to compare findings, they are frequently conflicting, due to patient group heterogeneity.

There is growing evidence of a familial basis for a variety of learning and behavioural problems, including a visual processing subtype of dyslexia (IS). There are, however, many questions which remain unanswered and a great deal of further research is clearly needed if we are to determine the place of chromosome analysis or biochemical analysis as a method of early identification. Learning to read involves many cognitive processes, and a breakdown in any of these processes may lead to reading difficulties.

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