



Developmental Dyslexia

Developmental dyslexia, or specific learning disability for reading, has been defined as attainment of reading skills below normal for age, in the presence of normal intelligence, and adequate learning opportunities. It affects 5-10% of school populations, depending on the threshold adopted, and is probably twice as common in males than females. Ophthalmologists may be asked whether there are any associated ocular problems.

Diagnosing dyslexia

Concerns about a child's reading performance are usually raised by parents or school teachers. The nature of the reading problem should be confirmed by an educational psychologist or experienced staff from a learning support service. A discrepancy between IQ and reading performance, or more specific features of dyslexia such as signs of phonological weakness (difficulty associating written letters with their sounds), will be sought.

What are the origins of developmental dyslexia?

Reading development proceeds against the background of a considerable amount of spoken language already acquired by a child in the first years of life. Research has indicated that conversion of the written word image into its phonological equivalent in the brain, is crucial in the normal process of learning to read fluently¹. Failure to develop the association between letter (grapheme) and sound (phoneme) is likely to be a major cause of reading and spelling impairment in many or most instances of developmental dyslexia. Coexistent auditory information processing deficits have also been shown.

Much effort has been spent in recent years to research the basis of this phonological deficit. Some suspect an even more basic defect in the timing of cerebral events, accounting for other associated features of dyslexia such as clumsiness. The work falls into a number of groups with unclear connections between them.

Defects of the magnocellular (or transient) system.

The magnocellular pathway receives input from both rods and cones across the retina, and extends from ganglion cells in the retina to the two ventral layers of the lateral geniculate nucleus (LGN). The large cell bodies in these two layers give the pathway its name. It projects via the visual cortex and the dorsal or "where" stream to the parietal cortex, and is best adapted to detect position and movement of objects in the visual field. This system is more sensitive to lower contrast and higher temporal frequency, but less sensitive to higher spatial frequency, than the parvocellular system.

The parvocellular (or sustained) system receives input from cones and therefore mainly the central retina, and runs from retinal ganglion cells to the smaller cell bodies of the four dorsal layers of the LGN. It projects via the visual cortex and the ventral or "what" stream to the inferotemporal cortex, and mediates colour vision and detection of fine spatial details. There



is extensive interaction between these parallel pathways.

It has been postulated that dyslexia is due to a defect in the magnocellular system (perhaps resulting in failure, during reading saccades, to suppress the image obtained by the parvocellular system during a previous fixation and causing confusion of printed letters and words). Evidence for a magnocellular defect comes from a postmortem study which showed that cells in the magnocellular layers of the LGN from dyslexic subjects were smaller than cells from brains of normal readers², and also from psychophysical studies. However, the evidence for such defects is conflicting³.

Differences detected on functional neuroimaging.

Several studies have shown differences in spatial and temporal brain activation between normal and dyslexic readers when reading various test materials, using magnetoencephalography, positron emission tomography, or functional magnetic resonance imaging. Although the patterns observed in different studies have varied, most dyslexic responses were late, reduced, or ectopic, which may represent compensatory mechanisms. For example, dyslexic subjects showed increased activation of the inferior frontal area and a tendency to use right hemisphere equivalent areas⁴. Abnormal cerebellar activation during non-visual tasks has also been shown in dyslexic subjects by positron emission tomography.

Differences in neuronal anatomy.

The use of CT, MRI, and postmortem measurements has demonstrated differences between normal readers and dyslexic subjects. Most attention has been paid to the planum temporale, a language related brain area of the posterior temporal cortex. The planum temporale is usually larger on the left side, but it is reported that this asymmetry is reduced, absent, or reversed in dyslexia.

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However, because of differences in anatomical definitions used and lack of control of other variables such as brain weight, sex, or handedness, it is difficult to draw any firm conclusions⁵. Differences in the anatomy of the corpus callosum have also been described.

Genetics.

Linkage studies have identified regions likely to contain candidate genes for dyslexia on chromosome 15q, 6p, and also on chromosomes 1 and 2. It is not known by what mechanisms these genes might influence reading ability.

Other workers have suggested retinal or oculomotor abnormalities rather than central processing problems as responsible for dyslexia.

Scotopic sensitivity syndrome and coloured filters or overlays.

In 1983, Irlen described the oddly named scotopic sensitivity syndrome (SSS) which was said to cause visual discomfort and dyslexia, due to abnormal retinal sensitivity to very specific wave-lengths of light which differed greatly between subjects. In its original form, sufferers from SSS were diagnosed by a set of questions constituting the Irlen Differential Perceptual Schedule (IDPS) test, and treated with coloured lenses specific to each individual. No scientific evidence to support the existence of such a syndrome has been found. The symptoms elicited by the IDPS are vague and medically would have very little diagnostic significance.

Although SSS may not exist, interest in coloured filters or overlays as a treatment for dyslexia has persisted. The colour is selected either by subject preference, or after testing with the Colorimeter developed by Wilkins. Much of the literature is uncontrolled or poorly planned but nevertheless some good studies have supported it. It seems however that there is poor test-retest consistency of colour selected, and that subjects frequently do not persist with their use. There would be some theoretical support for the use of blue filters for stimulating an impaired magnocellular system but in practice this colour does not usually help and is not selected more frequently by reading impaired subjects.

Pattern glare, a hypersensitivity to repetitive visual patterns, including lines of print on a page, has been proposed as a contributory factor in dyslexia, and dark or coloured lenses have also been proposed as a treatment, with similar uncertain results⁶.

Abnormal eye movement patterns.

Reading requires a series of accurate fixations and saccades,

together with convergence and accommodation. A considerable body of research is devoted to the part that deficient eye movements and focussing might play in reading disability, and suggests that convergence or accommodative insufficiency, inaccurate saccades, and binocular instability are commoner in dyslexics than in normal readers. They may be the result rather than the cause of reading problems, or have a common aetiology (for instance, inaccurate saccades or binocular instability could be associated with a magnocellular deficit). There is nothing to indicate that their correction has any effect on the central processing defects that seem to underlie dyslexia⁷.

The Dunlop synoptophore test of vergence stability attempts to identify the lack of a fixed reference eye, which could cause difficulty in integrating information from the two eyes, and therefore dyslexia. If no fixed reference eye is identified, a course of occlusion is recommended to promote its development. Studies have shown that a significant minority of normal readers had abnormal responses, and the value of the test, which may be difficult to interpret, is disputed. The evidence for an improvement in reading performance after occlusion is slim.

Conclusion.

Published work strongly supports a central processing defect causing phonological problems, and perhaps more widespread abnormalities affecting the timing of cerebral events, as the basis for developmental dyslexia.

A successful treatment regime should result in permanent improvement in reading ability to match that of non-impaired readers. Unfortunately, there is no evidence that any regime fulfils this criterion, and dyslexia persists into adolescence⁸ and adult life.

Dyslexic subjects probably develop compensatory strategies, and educational training programmes concentrating on phonological awareness skills may improve reading ability further, although not to normal.

There is no evidence that any other type of visual training works. The value of coloured filters and overlays would bear further investigation in the setting of carefully designed trials.

An optometrist, ophthalmologist, or orthoptist should carry out an orthoptic and refractive assessment in every child in whom a diagnosis of dyslexia is being entertained. Abnormalities in the range that would be significant in a normal reader should be corrected.

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