

**Spatial-frequency- and contrast-dependent visible
persistence and reading disorder: no evidence
for a basic perceptual deficit**

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Summary. The aetiology of dyslexia is still unclear, the most widely and controversially discussed theory is the magnocellular deficit hypothesis. One of the first and most influential paradigms used to investigate this visual deficit in dyslexia is the visible persistence (VP). However results on VP are decisively influenced by the method measuring VP. Lovegrove et al. (1986) repeatedly found a longer VP in reading disabled children which is significantly influenced by spatial frequency and contrast. However, these results were not investigated with the same method to date. Seventy-six unselected 2nd grade students (41 boys, 35 girls) of a rural primary school were investigated with an identical experimental design comparable to the Lovegrove et al. (1986) studies. Comparing reading disabled ($n = 17$) with controls ($n = 34$) no evidence for a longer VP in the reading disabled group was found. Additionally, correlation analysis revealed no evidence for a significance of VP for spelling, phoneme awareness and speech discrimination. This study does not encourage either a magnocellular nor parvocellular deficit in dyslexia.

Keywords: Dyslexia, magnocellular deficit, visible persistence, aetiology, reading disorder.

Introduction

Dyslexia is a specific learning disorder which typically persists into adulthood (Schulte-Körne et al., 2003). In spite of adequate educational resources, no obvious sensory deficits, adequate sociocultural opportunity, and at least average cognitive abilities (IQ) (Dilling et al., 1991) children are severely impaired in learning to read and spell. About 4–5% of school age children suffer from dyslexia. The aetiology of this impairment is still unclear, molecular genetic findings support that dyslexia is a heritable neurobiological syndrome (Schulte-Körne, 2001a; Fisher et al., 2002). A great amount of research has focused on

basic auditory and visual perceptual deficits, yielding conflicting results. The most widely and controversially discussed theory is the magnocellular deficit hypothesis (Stein and Walsh, 1997). It focuses on basic visual perception of non-verbal visual material and on a malfunctioning interaction of two separate interactive subsystems (the magno- and parvocellular systems) which are characterized by a different spatiotemporal response (Merigan and Maunsell, 1993). The magnocellular system, which arises from cells widely distributed across the retina, projects via the ventral lateral geniculate nucleus (LGN) to the visual cortex. From the primary visual cortex a dorsomedial stream projects forward to MT/V5 and to the posterior parietal cortex. This stream consists of mainly magnocellular neurones. The magnocellular system preferentially mediates movement, fast temporal resolution, low contrast, and low spatial frequencies. The parvocellular system originates in cells concentrated in the fovea and projects via the dorsal LGN to the visual cortex and then mainly to the temporal cortex. It is responsible for colour resolution, high contrast, and high spatial frequencies characteristics (Merigan and Maunsell, 1993).

A number of studies report visual pathway deficits linked to the magnocellular system (Stein, 2003). Depending on experimental designs and methods, research on the sensitivity of the magnocellular system has produced conflicting results (e.g. Schulte-Körne et al., 1999a; Skottun, 2000; Amitay et al., 2002). One of the first and most influential paradigms used to investigate this visual deficit in dyslexia is the visible persistence (VP; Lovegrove et al., 1980a). Visible persistence refers to the extended visibility of a stimulus for a short time after its termination. It is phenomenally indistinguishable from the visibility during the actual presence of the stimulus (Coltheart, 1980). Further it was argued that visible persistence for longer stimulus duration is cortical and not retinal in origin (Badcock and Lovegrove, 1981). An impairment of VP is a result of insufficient time to process the stimulus before the masking stimulus arrives (DiLollo et al., 1983). This is assumed to reflect ongoing neural activity initiated by the stimulus presentation.

Because the magnocellular system is sensitive for low contrast and low spatial frequency the influence of spatial frequency and contrast on visible persistence in reading disabled has been examined repeatedly (Lovegrove and Brown, 1978; O'Neill and Stanley, 1976; DiLollo et al., 1983; Slaghuis and Lovegrove, 1985; Slaghuis et al., 1993; Lovegrove et al., 1980a) and across a large age range (from 8 to 13 years). Visible persistence was measured with a subjective rating task where subjects had to report whether or not they had seen a luminance-matched blank stimulus between repeatedly presented grating stimuli (continuation-of-form technique, Lovegrove et al., 1986). The interstimulus interval which is taken as a measure of visible persistence is varied until the subject can see a clear blank. Using sine wave gratings ranging between low (2 cycles per degree) and high (12 cycles per degree (cpd)) spatial frequencies, they found visible persistence to depend on spatial frequency, i.e. the duration of visible persistence increases with increasing spatial frequency (Lovegrove et al., 1980a; Bowling et al., 1979; Meyer and Maguire, 1977). In dyslexic children a significantly longer visible persistence than in controls at low spatial frequencies and a smaller increase in visible persistence with increasing spatial

frequency (the mean regression slope is steeper in controls than dyslexics) were found (Lovegrove et al., 1980a; Badcock and Lovegrove, 1981; Slaghuis and Lovegrove, 1985; Slaghuis et al., 1993). Higher visible persistence means in this task that the interstimulus interval at which a blank field becomes detectable was extended in dyslexic children. This indicates that the visual system processes low spatial frequencies slower in dyslexics than in controls.

Visible persistence is also influenced by contrast, i.e. increasing contrast produces a decrease in duration of visible persistence (Bowling et al., 1979). Comparing visible persistence at low and high contrasts in dyslexic children and controls Badcock and Lovegrove (1981) found a higher visible persistence in dyslexic children.

Also an interaction of spatial frequency and contrast on visible persistence was found. At low contrast (2 cpd) the difference in slopes (regression slope: duration of visible persistence increases with spatial frequency) between the groups was greatest (Badcock and Lovegrove, 1981).

These findings indicate that dyslexic children differ in terms of temporal processing across spatial frequency and across contrast specific channels, and these results are consistent with the theory of a magnocellular functioning deficit.

However, Arnett and DiLollo (1979), DiLollo et al. (1983), Walther-Müller (1995), and Hogben et al. (1995) could not replicate these findings. They investigated visual persistence via a temporal integration of form task (where two different parts of an array of elements are shown in successive frames, only a successful integration of the two parts above time makes it possible to recognise the position of a missing element) and did not find evidence for a longer duration of visible persistence in dyslexics.

Because no evidence was found for differences in visible persistence when visible persistence was examined by tasks different from the ones used by Slaghuis and Lovegrove (1985), we chose the paradigm of the latter group. In an unselected sample of 76 German 2nd grade children we assessed visible persistence with a continuation-of-form task.

We expected to find VP depend on spatial frequency (longer VP for higher spatial frequency, hypothesis 1) and contrast (longer VP for lower contrast, hypothesis 2).

Finally, we expected to find a group influence on VP in the form of an interaction of group with spatial frequency (longer VP at lower spatial frequency in dyslexic children) (hypothesis 3) and contrast (longer VP at low contrast in dyslexic children) (hypothesis 4).

Materials and methods

Seventy-six unselected 2nd grade students (41 boys, 35 girls) of a rural primary school in Germany participated in the study. Inclusion criteria were to be native monolingual German speakers, to have normal or corrected normal visual acuity and no hearing problems. Intelligence was assessed with a nonverbal IQ test (Culture Fair Intelligence Test-1, Weiß and Osterland, 1997), spelling was assessed with a writing-to-dictation test (Weingartner Rechtschreibtest 2+, Birkel, 1994), and reading was assessed with a word reading speed test (Salzburger Lese- und Rechtschreibtest, Landerl et al., 1997). Table 1 shows the sample characteristics.

Studies that showed the significance of VP for dyslexia have utilized a group comparison design. Therefore we selected affected and unaffected subjects by means of a regression-based discrepancy criterion (Schulte-Körne et al., 2001): a child is considered affected if the reading

Table 1. Sample characteristics (age, IQ, spelling, and reading). T values are given for spelling and reading

n = 76	Mean	s.d.
Age	8.5	0.5
IQ	104.6	12.0
Spelling	45.0	8.6
Reading	49.1	8.4

ability is more than one standard deviation below the expected reading ability based on IQ. Inclusion criterion for the controls was to have an actual reading that is better than the expected reading value (based on IQ).

Group comparison was conducted with these two groups, reading disabled ($n = 17$; age = 8.8; IQ = 106.8; reading T value = 37.3) and controls ($n = 37$; age = 8.5; IQ = 102.6; reading T value = 54.9).

Borsting et al. (1996) and Slaghuis and Ryan (1999) found that basic visual processing has an impact on auditory processing. Therefore we calculated correlations of VP with speech discrimination and phonological processing in the unselected 2nd grade sample.

Speech stimuli (standard /da/- deviant /ba/) were synthesized and were presented binaural and randomly via headphones (see Schulte-Körne et al., 1999b). Subjects had to identify a /ba/ by pressing the left button of a computer mouse, /da/ stimulus by pressing the right button. Depending variable was the number of correctly identified speech stimuli.

Phonological decoding was measured with a standardized nonword reading speed test (Salzburger Lese- und Rechtschreibtest, Landerl et al., 1997). Additionally a phoneme deletion test (Schulte-Körne, 2001) was conducted. Subjects had to first repeat an item (to ensure they had heard it correctly) and then were instructed to repeat the word sans the first phoneme. 44 items (22 pseudowords and 22 real words) were used for this test. For the statistical analysis the number of correct answers was counted.

The experimental procedure for the assessment of VP was chosen quite similar to that by Slaghuis and Lovegrove (1985) and Slaghuis et al. (1993). During the assessment of visual persistence, the children sat in a darkened room (average luminance of 1.2 cd/m^2) at a 60 cm viewing distance to an EIZO 21' computer monitor. The visual stimuli presented consisted of sine wave vertical gratings in a circle on a dark background at a 3° visual angle. Background luminance was 2 cd/m^2 and grating luminance was 12 cd/m^2 . Items of four separate conditions comprising the combination of two spatial frequencies (2 and 11.33 cpd) and two contrast levels (0.2 and 0.6) were presented in random order.

Each trial consisted of a grating-blank-grating sequence repeated 10 times. Thus 11 occurrences of a grating circle were followed (except for the last one) by a blank field each, matched in space-average luminance. The duration of the grating circles was fixed to 300 ms which was chosen because the interaction of reading disability and spatial frequency was strongest at stimulus durations about 300 ms (Lovegrove et al., 1986). The duration of the blank started with 400 ms and was changed depending on the child's answer, using a staircase method. After a trial has been completed, subjects were instructed to press the left mouse button if they had seen a blank and to press the right mouse button if they had not seen a blank between the gratings. At brief durations of the blank the cycled grating appears to be continuously present, its visible persistence phenomenally filling the gap between the cycled gratings and subjects typically report the absence of the blank field. However, the blank field becomes detectable when its duration exceeds the duration of visible persistence of the grating. Thus the duration of the interstimulus interval at which the blank field becomes detectable represents a measure of the duration of visible persistence. As long as the subject could detect the blank field, the duration of the blank field was shortened from one trial to the next by 10%. When the subject could not see the blank field any longer (lower reversal point), the duration was lengthened by 10% until the subject was able to detect it again (upper reversal point).

Each run of the experiment ended after 8 reversal points. Using the last 3 upper and 3 lower reversal points (the first two reversal points were excluded from the analysis) respectively, the medians for these upper and lower points were calculated. The duration of visible persistence was estimated by measuring the duration at which the blank field was just visible (in ms). The visible persistence was calculated as mean of the two medians.

Results

Figure 1 shows the VP means for low contrast, at high and low spatial frequencies, and Fig. 2 shows the VP means for high contrast at high and low spatial frequencies for both groups (reading disabled and controls).

The main assumption based on the sensitivity of the magnocellular system is that dyslexics are mainly impaired at low spatial frequencies and low contrast. Inspection of the mean values for reading disabled and controls (Figs. 1 and 2) show a mean difference for VP at low contrast and low spatial frequency, but the direction is contrary to our hypothesis: in the actual data, the duration of VP at low spatial frequency and low contrast is longer for the controls than for the reading disabled. Therefore, our hypotheses 3 and 4 are to be rejected without further statistical analysis. At high spatial frequency, there is virtually no VP difference between the groups.

To further illustrate the correlation between reading and visible persistence both variables were plotted (x-axis: reading speed of words, T-value; y-axis: mean visible persistence at 2 cpd spatial frequency and 0.2 contrast). The plot shows that independently of the reading ability no correlation was found with visible persistence.

Regarding hypothesis 1 and 2, a Repeated Measures ANOVA with within-subjects factor spatial frequency (2 and 11.33 cpd) and contrast levels (0.2, and 0.6) was carried out with the entire sample ($n = 76$). Concurring with our hypotheses, significant main effects contrast and spatial frequency were found (s. Table 3): visible persistence increases with increasing spatial frequencies and with decreasing contrast. Table 2 illustrates the corresponding mean values.

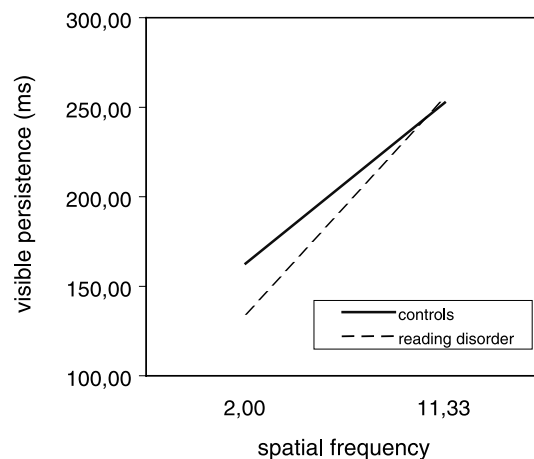


Fig. 1. Mean visible persistence at contrast 0.2 for reading disabled and controls

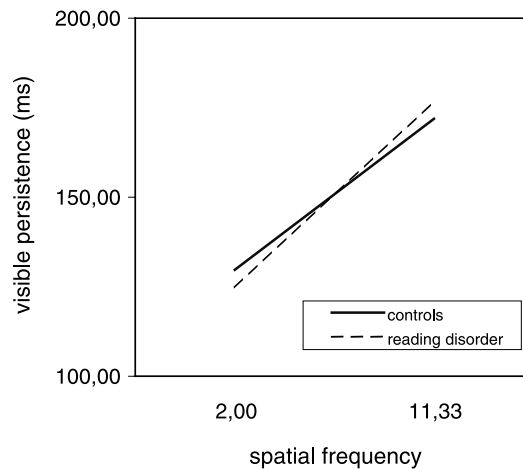


Fig. 2. Mean visible persistence at contrast 0.6 for reading disabled and controls

It has been hypothesised that the slope of visible persistence for the two spatial frequencies is steeper for controls than for dyslexics (Slaghuis and Lovegrove, 1985; Slaghuis et al., 1993). As can be seen from Fig. 1, we cannot confirm this finding because in our data the slopes are on the contrary smaller for controls than for dyslexics.

Regarding the possible relation of VP with spelling, phonological decoding, phonological processing, and speech discrimination, the following slopes were calculated (the denominator is omitted because it is a constant value):

$$sl2 = VP(\text{contr. } 0.2, \text{ spat. freq } 11.33) - VP(\text{contr. } 0.2, \text{ spat. freq } 2) \text{ and} \\ sl6 = VP(\text{contr. } 0.6, \text{ spat. freq } 11.33) - VP(\text{contr. } 0.6, \text{ spat. freq } 2)$$

Correlation coefficients were then calculated, s. Table 4. For this calculation, the entire sample ($n = 76$) was used. All of the correlations are small and not significant.

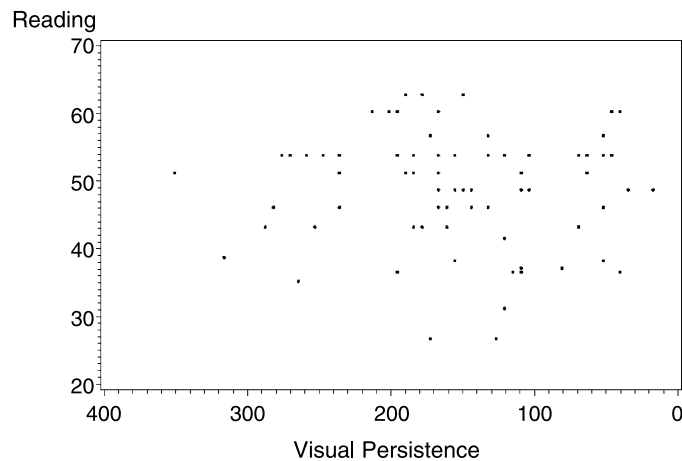


Fig. 3. Plot of reading ability (T-value) and visible persistence (ms) at 2 cpd spatial frequency and 0.2 contrast

Table 2. Means and standard deviations of visible persistence for all subjects

n = 76	Spat. freq. 2	Spat. freq. 11.33
Contrast 0.2	153.9 \pm 72.9	247.3 \pm 167.5
Contrast 0.6	131.2 \pm 79.0	170.1 \pm 117.6

Table 3. Results of the variance analysis (p values)

Factor	p value
Contrast	<0.0001
Spatial frequency	<0.0001
Contrast * spat. freq.	0.011

Table 4. Correlation between spelling, speech discrimination, phonological decoding, phoneme deletion and the slopes

	sl2	sl6
Reading	-0.18	-0.05
Spelling	-0.13	-0.07
Speech discrimination	-0.08	-0.07
Phonological decoding	-0.13	-0.17
Phoneme deletion	0.13	0.01

Discussion

This is the first study that investigated visible persistence in an experiment comparable to the experiment used by the research group at the University of Tasmania (Lovegrove et al., 1980a; Slaghuis and Lovegrove, 1985; Slaghuis et al., 1993) who first and repeatedly found the significance of VP for reading disability. The application of an identical experimental design is of high relevance because other researchers (Arnett and DiLollo, 1979; DiLollo et al., 1983; Walther-Müller, 1995; Hogben et al., 1995) who used a different procedure (i.e. a temporal integration of form technique) to measure visible persistence could not replicate the findings.

First an unselected population of second grade school children was examined. We replicated the finding that the duration of visible persistence is significantly influenced by contrast and spatial frequency (visible persistence increases with increasing spatial frequencies and increases with decreasing contrast) as described by Slaghuis and Lovegrove (1985). Thus we assume that our experimental design and the implementation of this in our laboratory is appropriate to measure visible persistence.

Second the significance of VP for reading was analysed. In order to follow the approach of Lovegrove et al. (1980b), Slaghuis and Lovegrove (1985) and Slaghuis et al. (1993), the sample was divided into two subsamples, a group of

reading disabled children and a group of control children. As opposed to the findings of the University of Tasmania group, in our sample controls had longer durations of visible persistence and smaller slopes than the reading disabled (at low contrast).

Borsting et al. (1996) and Slaghuis and Ryan (1999) suggested that a magnocellular deficit might only be found in subgroups of dyslexic children. However their findings are not as clear as they stated because they found lower contrast sensitivity at low spatial frequencies in the not specified (in phonological and visual deficient) dyslexic group. We calculated correlations of VP with several tasks typically used to measure phonological processing to further investigate whether magnocellular functions are related to basic auditory perception in dyslexic children. However, no evidence for a significance of VP on phonological processing was found.

Our experiment is very similar to Slaghuis and Lovegrove (1985), but one difference is that the children in our sample are native German speakers. As Walther-Müller (1995) pointed out, there is no obvious reason why a low level visual function like visible persistence should have a different influence on reading in different languages as it affects visual perception of the stimuli but not language processing.

There is a relevant difference between the studies finding the group differences described above (Slaghuis and Lovegrove, 1986; Slaghuis et al., 1993) and our study. Whereas in our study both groups have very similar IQ values, the significantly higher IQ in the control group than in the reading disabled group could have influenced the findings in the studies of Slaghuis and Lovegrove (1986) and Slaghuis et al. (1993). In our study we found a correlation of IQ with VP (range for two contrasts and two spatial frequencies) from -0.22 to -0.33 . This means that the more intelligent subjects tend to have lower values of VP. These correlations are higher than the correlations of VP with reading, spelling and auditory processing (speech perception, phonological decoding, phoneme deletion). Thus the higher IQ of the control group in the Slaghuis et al. (1993) study could have contributed to the shorter duration of VP in the control group.

Another relevant aspect might be the influence of attention and vigilance as suggested by Haduck et al. (1996). Because a high comorbidity of dyslexia with ADHD has repeatedly been found (e.g. August and Garfinkel, 1980), longer visible persistence might be more attributed to an attentional deficit. Because a detailed sample description with regard to comorbidity is missing in the studies of the Tasmania research group, one can only speculate whether or not the reading disabled in their studies are affected by ADHD.

Our study does not support the hypothesis of an altered visible persistence in reading disabled children. The literature on basic visual perception in dyslexia is not consistent. Using another technique to measure VP, Arnett and DiLollo (1979), DiLollo et al. (1983), Walther-Müller (1995) and Hogben et al. (1995) did not find evidence for different VP values in reading disabled and controls. Also, some results found on VP are contradicting the theory of a magnocellular deficit (Martin and Lovegrove, 1987).

This study does not add evidence to either a magnocellular nor parvocellular deficit in dyslexia. These findings do not necessarily imply that magnocellular

functions are not relevant for reading and spelling. Recent studies show that motion perception (mainly coherent motion) correlates with reading (Eden et al., 1996; Demb et al., 1998; Cornelissen et al., 1995; Schulte-Körne et al., 2004). However all the studies finding a correlation between magnocellular functions and reading (summarised in Stein, 2003) did not allow to conclude that deficient magnocellular functioning is causally related to reading and spelling disorder.

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