Levitt JB, Schumer RA, Sherman SM, Spear PD, Movshon JA. Visual response properties of neurons in the LGN of normally reared and visually deprived macaque monkeys. J Neurophysiol 2001;85: 2111–29.
Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proc Natl Acad Sci USA 1991;88:7943–7.
Maunsell JHR, Nealey TA, DePriest DD. Magnocellular and parvocellular contributions to responses in the middle temporal visual area MT of the macaque monkey. J Neurosci 1990;10:3323–34.
Merigan WH, Maunsell JHR. How parallel are the primate visual pathways? Annu Rev Neurosci 1993;16:369–402.
Ramus F, Rosen S, Dakin SC, Day BL, Castellote JM, White S, Frith U. Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults. Brain 2003;126:841–65.
Raymond JE, Sorensen RE. Visual motion perception in children with dyslexia: normal detection but abnormal integration. Vis Cogn 1998;5: 389–404.
Sawatari A, Callaway EM. Convergence of magno- and parvocellular pathways in layer 4B of macaque primary visual cortex. Nature 1996; 380:442–6.
Scheuerpflug P, Plume E, Vetter V, Schulte-Koerne G, Deimel W, Bartling J, Remschmidt H, Warnke A. Visual information processing in dyslexic children. Clin Neurophysiol 2004;15:90–6.
Schulte-Korne G, Bartling J, Deimel W, Remschmidt H. Visual evoked potentials elicited by coherently moving dots in dyslexic children. Neurosci Lett 2004a;357:207–10.
Schulte-Korne G, Bartling J, Deimel W, Remschmidt H. Motion-onset VEPs in dyslexia. Evidence for visual perceptual deficit. NeuroReport 2004b;15:1075–8.
Shapley R, Perry VH. Cat and monkey retinal ganglion cells and their visual functional roles. Trends Neurosci 1986;9:229–35.
Sincich LC, Horton JC. Divided by cytochrome oxidase: a map of the projections from V1 to V2 in Macaques. Science 2002;295:1734–7.
Skottun BC. The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. Vis Res 2000;40:111–27.
Skottun BC. On the use of metacontrast to assess magnocellular function in dyslexic readers. Percept Psychophys 2001a;63:1271–4.
Skottun BC. On the use of the Ternus test to assess magnocellular function. Perception 2001b;30:1449–57.
Skottun BC. On the use of red stimuli to isolate magnocellular responses in psychophysical experiments: A perspective. Vis Neurosci 2004;21: 63–8.
Skottun BC, Parke LA. The possible relationship between visual deficits and dyslexia: examination of a critical assumption. J Learn Disabil 1999;32:2–5.
Skoyles J, Skottun BC. On the prevalence of magnocellular deficits in the visual system of non-dyslexic individuals. Brain Lang 2004;88: 79–82.
Spinelli D, Angelelli P, De Luca M, Di Pace E, Judica A, Zoccolotti P. Developmental surface dyslexia is not associated with deficits in the transient visual system. NeuroReport 1997;8:1807–12.
Stein J, Walsh V. To see but not to read; the magnocellular theory of dyslexia. Trends Neurosci 1997;20:147–52.
Stuart GW, McAnally KI, Castles A. Can contrast sensitivity functions in dyslexia be explained by inattention rather than a magnocellular deficit? Vis Res 2001;41:3205–11.
Victor JD, Conte MM, Burton L, Nass RD. Visual evoked potentials in dyslexics and normals: failure to find a difference in transient or steady-state responses. Vis Neurosci 1993;10:939–46.
Walther-Muller PU. Is there a deficit of early vision in dyslexia? Perception 1995;24:919–36.
Yabuta NH, Sawatari A, Callaway EM. Two functional channels from primary visual cortex to dorsal visual cortical areas. Science 2001;292: 297–300.

Bernt Christian Skottun
Skottun Research, 273 Mather Street, Piedmont, CA 94611-5154, USA

John R. Skoyles
Centre for Philosophy of Natural and Social Science, London School of Economics, Houghton Street WC2A 2AE, UK
E-mail address: j.r.skoyles@lse.ac.uk

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The role of the magnocellular pathway in dyslexia—reply to Skottun and Skoyles

The controversy about the significance of the magnocellular system for the aetiology of dyslexia (Stein, 2003) has been ongoing for a long time. To investigate the sensitivity of the magnocellular pathway we applied visual evoked potentials elicited by motion stimuli (Scheuerpflug et al., 2004, Schulte-Körne et al., 2004a,b).

Skoyles and Skottun (this issue) now question whether VEPs elicited by motion stimuli are suited to investigate magnocellular sensitivity. They argue that the magnocellular system is essentially a subcortical system. Therefore, the registration of cortical neuronal activity does not justify to conclude that this activity is mainly due to magnocellular neurons.

However, the analysis of motion in nonhuman primate as well as human visual systems suggest that cortical neurons of the M pathway are sensitive to the motion perception.

The visual magnocellular pathway projects directly through layers of the lateral geniculate to layer 4Cα of the nonhuman primary visual cortex (V1) which in turn projects (via layer 4B) directly or indirectly via thick stripes of area V2 or area V3 to middle temporal (MT) and medial superior temporal (MST) cortical areas (Zeki and Shipp, 1988). The motion pathway extends beyond MST to ventral intraparietal (VIP) area in the parietal lobe and the frontal eye fields (Ungerleider and Desimone, 1986). A large majority of neurons in the middle temporal visual area (MT) respond selectively to the direction and speed of stimulus motion (Maunsell and Van Essen, 1983).These neurons are organized into cortical columns on the basis of their preferred direction of motion (Albright et al., 1984). MST, an area lying ventral and anterior to MT in the depths of the superior temporal sulcus has also been found to contain a large majority of directionally selective neurons (Celebrini and Newsome, 1994). The hypothetical human homologue of MT and MST are areas V5 and V5a (Zeki and Shipp, 1988) and the superior parietal-occipital area (SPO) (Tootell et al.,

1996). Different numbers of cortical areas in the occipital and temporal lobe have been identified when subjects were viewing moving stimuli. Coherently moving stimuli activated the cortical areas V1, V5 and the parietal cortex (Zeki et al., 1991). The findings from a lesion study of a single subject with bilateral posterior damage, who exhibited dramatic deficits in motion discrimination while performing normally on tasks involving colour vision, confirms the importance of these cortical areas to motion perception (Zihl et al., 1983).

Skoyles and Skottun mentioned that the magnocellular system is not specifically sensitive for motion perception. The finding that the motion of isoluminat colour stimuli could be perceived, although the magnocellular system is not sensitive for isoluminat colour stimuli, suggests that the motion perception cannot be exclusively attributed to the magnocellular system. Although this did not refer directly to our published studies, since, we did not use colour stimuli. This remark refers to the neurophysiological and neuroanatomical finding that motion and colour are processed by different visual pathways, whereas motion is primarily processed by the magnocellular pathway and colour by the parvocellular pathway (Livingstone and Hubel, 1984; Zeki, 1978). However, the neurophysiological relationship between motion and colour processing is still under debate (Takeuchi et al., 2003). It is beyond the scope of this manuscript to review the discussion on the neurophysiological basis of the relationship between motion and colour perception. However, findings from neurophysiological studies provide evidence that motion selective cortical areas, MT and MST, are also sensitive to colour processing (Wandell et al., 1999).

Another aspect mentioned by Skoyles and Skottun is the long latency of the VEP elicited by the moving stimuli. Skoyles and Skottun suggest that the latency of magnocellular neurons in the lateral geniculate nucleus is very short. Consistently, we and other researchers investigating VEPs in human subjects (Kubova et al., 1996; Lehmkuhle et al., 1993; Livingstone et al., 1991) found group differences between dyslexics and controls of at least 100 ms. In order to analyse subcortical magnocellular neurons we measured VEPs at the scalp and found peak latency at about 200 ms suggesting that human P200 reflects excitatory depolarizing potentials in apical dendrites of pyramidal cells. Perception of visual motion can be intensively studied by applying VEPs, in particular motion-onset VEPs (Bach and Ullrich, 1994; Hoffmann et al., 1999). In concordance with fMRI studies in human subjects, electrophysiological correlates of motion perception were found at motion sensitive cortical areas (V1, V5/MT) (Probst et al., 1993). Thus, VEPs are well suited to investigate neurophysiological correlates of motion perception. These correlates are mainly attributed to cortical areas, which receive direct input from the M pathway.

Skoyles and Skottun remark that magnocellular neurons have little or no direction selectivity. Thus, our finding of the VEP amplitude differences between dyslexic and controls elicited by coherently moving dots cannot be caused by magnocellular dysfunction. However, Schiller et al. (1980) demonstrated that lesions of the M pathway in monkeys eliminated the ability to detect motion of a group of dots in a field of random dots. Furthermore, single-unit physiological experiments have shown that directionally selective neurons in area V5 are effectively driven by stimuli, which contains a number of elements, all moving coherently in one direction (Snowden et al., 1991). More recently, microstimulation of single sites in MT showed that the coherent motion perception is mainly influenced by MT (Nichols and Newsome, 2002).

Finally, electrophysiological correlates of motion perception mainly generated in cortical areas do not exclude that other motion sensitive areas, i.e. subcortical areas (LGN) are impaired in dyslexics. The MT receives input from the M pathway. This means that the nature of MT response depends on the afferent input to MT. Starting from the ganglion cells of the retina via neuronal layers of the lateral geniculate to V1 in the primary visual cortex, the significance of the M pathway on perceptual behaviour can only be expressed through the M pathway as a whole. Thus, the reduced amplitude of the VEP over parietal-temporal cortical areas in our study might also be influenced by subcortical M pathway disruptions.

Since, the M pathway transmitted motion signals through MT to higher cortical areas, the perceptual decision individuals made in our experiments, do not result from an impairment of the motion sensitive cortical areas only, but also from the visual attention (Vidyasagar, 1999). This means that the feedback projections from several cortical areas selectively enhance or suppress responses of striate neurons (Hupe et al., 1998). According to Vidyasagar (1999), this feedback serves like attentional focussing of a target within the receptive field. This could mean that the receptive field of the neuron could shrink around an attended location. Thus, attentional spotlight is a neural mechanism that allows only selected outputs of a location to higher visual cortical areas. One prediction from this theory is that M-mediated attentional spotlighting via feedback on the V1 and V2 acts as gate for the parvocellular inputs. For reading words, this model predicts that attentional spotlight brings the focus of attention to a set of letters. The rapid and sequential spotlighting function of M pathway during the fixation periods is necessary for the ventral M pathway to order the letters appropriately (Vidyasagar, 1999). One further assumption is that some functions of the parvocellular pathway are influenced by the magnocellular pathway. Evidence for this was found by Vidyasagar and Pammer (1999). This model may also serve to explain some contradictory findings that behavioural deficits in discrimination tasks could better be explained by a parvocellular deficit than by a magnocellular deficit. In summary, Skottun is correct that the subcortical retinogeniculate pathway is difficult to investigate by visually evoked potentials elicited by moving stimuli. However, we like others (Demb et al., 1997; Eden et al., 1996) are interested in investigating

the cortical part of magnocellular system. Since, several cortical areas, i.e. MT/V5, can be regarded as part of the magnocellular pathway and motion sensitive neurons have been found in these areas; it seems justified to argue that the neurophysiological correlates of motion perception in dyslexia are related to magnocellular function.

References

Albright TD, Desimone R, Gross CG. Columnar organization of directionally selective cells in visual area MT of the macaque. J Neurophysiol 1984;51:16–31.

Bach M, Ullrich D. Motion adaptation governs the shape of motion-evoked cortical potentials. Vision Res 1994;34:1541–7.

Celebrini S, Newsome WT. Neuronal and psychophysical sensitivity to motion signals in extrastriate area MST of the macaque monkey. J Neurosci 1994;14:4109–24.

Demb JB, Boynton GM, Heeger DJ. Brain activity in visual cortex predicts individual differences in reading performance. Proc Natl Acad Sci USA 1997;94:13363–6.

Eden GF, VanMeter JW, Rumsey JM, Maisong JM, Woods RP, Zeffiro TA. Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 1996;382:66–9.

Hoffmann M, Dorn TJ, Bach M. Time course of motion adaptation: motion-onset visual evoked potentials and subjective estimates. Vision Res 1999;39:437–44.

Hupe JM, James AC, Payne BR, Lomber SG, Girard P, Bullier J. Cortical feedback improves discrimination between figure and background by V1, V2 and V3 neurons. Nature 1998;394:784–7.

Kubova Z, Kuba M, Peregrin J, Novakova V. Visual evoked potential evidence for magnocellular system deficit in dyslexia. Physiol Res 1996;45:87–9.

Lehmkuhle S, Garzia RP, Turner L, Hash T, Baro JA. A defective visual pathway in children with reading disability. N Engl J Med 1993;328: 989–96.

Livingstone MS, Hubel DH. Anatomy and physiology of a color system in the primate visual cortex. J Neurosci 1984;4:309–56.

Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proc Natl Acad Sci USA 1991;88:7943–7.

Maunsell JH, Van Essen DC. Functional properties of neurons in middle temporal visual area of the macaque monkey. I. Selectivity for stimulus direction, speed, and orientation. J Neurophysiol 1983;49:1127–47.

Nichols MJ, Newsome WT. Middle temporal visual area microstimulation influences veridical judgments of motion direction. J Neurosci 2002;22: 9530–40.

Probst T, Plendl H, Paulus W, Wist ER, Scherg M. Identification of the visual motion area (area V5) in the human brain by dipole source analysis. Exp Brain Res 1993;93:345–51.

Scheuerpflug P, Plume E, Vetter V, Schulte-Körne G, Deimel W, Bartling J, Remschmidt H, Warnke A. Visual information processing in dyslexic children. Clin Neurophysiol 2004;15:90–6.

Schiller PH, True SD, Conway JL. Deficits in eye movements following frontal eye-field and superior colliculus ablations. J Neurophysiol 1980; 44:1175–89.

Schulte-Körne G, Bartling J, Deimel W, Remschmidt H. Visual evoked potential elicited by coherenty moving dots in dyslexic children. Neurosci Lett 2004a;357:207–10.

Schulte-Körne G, Bartling J, Deimel W, Remschmidt H. Motion-onset VEPs in dyslexia. Evidence for visual perceptual deficit. Neuroreport 2004b;15:1075–8.

Snowden RJ, Treue S, Erickson RG, Andersen RA. The response of area MT and V1 neurons to transparent motion. J Neurosci 1991;11: 2768–85.

Stein J. Visual motion sensitivity and reading (Review). Neuropsychologia 2003;41:1785–93.

Takeuchi T, De Valois KK, Hardy JL. The influence of color on the perception of luminance motion. Vision Res 2003;43:1159–75.

Tootell RB, Dale AM, Sereno MI, Malach R. New images from human visual cortex. Trends Neurosci 1996;19:481–9.

Ungerleider LG, Desimone R. Projections to the superior temporal sulcus from the central and peripheral field representations of V1 and V2. J Comp Neurol 1986;248:147–63.

Vidyasagar TR. A neuronal model of attentional spotlight: parietal guiding the temporal. Brain Res Brain Res Rev 1999;30:66–76.

Vidyasagar TR, Pammer K. Impaired visual search in dyslexia relates to the role of the magnocellular pathway in attention. Neuroreport 1999;10: 1283–7.

Wandell BA, Poirson AB, Newsome WT, Baseler HA, Boynton GM, Huk A, Gandhi S, Sharpe LT. Color signals in human motion-selective cortex. Neuron 1999;24:901–9.

Zeki SM. Functional specialisation in the visual cortex of the rhesus monkey. Nature 1978;274:423–8.

Zeki S, Shipp S. The functional logic of cortical connections. Nature 1988; 335:311–7.

Zeki S, Watson JD, Lueck CJ, Friston KJ, Kennard C, Frackowiak RS. A direct demonstration of functional specialization in human visual cortex. J Neurosci 1991;11:641–9.

Zihl J, von Cramon D, Mai N. Selective disturbance of movement vision after bilateral brain damage. Brain 1983;106:313–40.

Gerd Schulte-Körne, Helmut Remschmidt
Department of Child and Adolescent Psychiatry and Psychotherapy, Philipps-University, Hans-Sachs-Str. 6, 35033 Marburg, Germany

Peter Scheuerpflug, Andreas Warnke
Department of Child and Adolescent Psychiatry and Psychotherapy, Julius-Maximilians-University Würzburg, Würzburg, Germany
E-mail address: schulte1@med.uni-marburg.de

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