Vision Research 50 (2010) 991–994

Contents lists available at ScienceDirect

Vision Research

journal homepage: www.elsevier.com/locate/visres

Letter to the Editor

Reply to Skottun & Skoyles. On interpreting responses to low contrast stimuli in terms of magnocellular activity – A few remarks

Skottun & Skoyles [Skottun and Skoyles (2009). On interpreting responses to low contrast stimuli in terms of magnocellular activity – a few remarks. *Vision Research*] have written a comment on our recent paper (Lalor & Foxe, 2009). Specifically they raise the following three issues: (1) spatial resolution and temporal tuning are similar in magno- and parvocellular neurons. (2) Results from lesion studies make reliance upon low contrast to isolate magnocellular activity problematic. (3) Other neurons exist which have contrast-response functions similar to those of magno- and parvocellular cells and, "[t]hus, it seems difficult to differentiate, on the basis of contrast gain and saturation, contributions that are uniquely magno- and parvocellular from cortical activity". We respond that issues (1) and (2) have not only been called into question several times previously, but that they are irrelevant to the main manipulation carried out in our study. Despite this, we would like to take the opportunity to demonstrate that these arguments are based on a rather selective interpretation of the literature. We feel that we have not contradicted issue (3) in our original article and, furthermore, that the points made are insubstantial.

In our paper (Lalor & Foxe, 2009), we presented Visual Evoked Spread Spectrum Analysis (VESPA) responses to four different stimulus types. In each case, the basic stimulus consisted of a checkerboard pattern subtending visual angles of 5.25° vertically and horizontally, with equal numbers of light and dark checks. The refresh rate of the monitor was set to 60 Hz and on every refresh the contrast of the checkerboard pattern was modulated by a non-binary stochastic signal which had its power distributed uniformly between 0 and 30 Hz. Responses were then obtained by linear least squares estimation on the assumption that the output EEG represented a convolution of the stochastically modulated contrast signal with an unknown impulse response, namely, the VESPA (Lalor, Pearlmutter, & Foxe, 2009; Lalor, Pearlmutter, Reilly, McDarby, & Foxe, 2006). For three of the four stimulus types, each individual check subtended a visual angle of 0.65° and the only manipulation that was carried out was related to the range of contrasts over which the checkerboard was modulated. In the FULL-RANGE case, the checkerboard was modulated between 0% and 100% using a Gaussian random process with a zero-point corresponding to the mid-point of the range (i.e., 50% contrast) and with a scaling that allowed ±3 standard deviations within the range. In the HIGH-CONTRAST case, the checkerboard was again modulated by a Gaussian random process, but the range was restricted to between 32% and 100%. Again the mid-point of this range corresponded to the zero-point of our mapping with ±3 standard deviations allowed within the range. The LOW-CONTRAST stimulus type followed the same trend except that the range was restricted to between 0 and 10%. A fourth stimulus type was undertaken by a minority of subjects that utilized the HIGH-CONTRAST range, but also featured a higher spatial frequency checkerboard with each check subtending a visual angle of 0.27° (HIGH-CONTRAST_HSF).

The main result of the paper plots dramatically different responses to the LOW-CONTRAST stimuli when compared with the FULL-RANGE stimuli. Given the previous evidence that magnocellular (M) and parvocellular (P) cells display marked differences in their luminance contrast gain curves, with that of M cells being much higher at low contrasts than that of P cells, we have interpreted the differences in morphology and timing to be as a result of differing contributions from these cell subgroups. Hence the title of our paper "Visual evoked spread spectrum analysis (VESPA) responses to stimuli *biased towards* magnocellular and parvocellular pathways" (emphasis added). This notion of differing contrast gain curves has been widely reported (Kaplan, 1991, 2003; Kaplan & Shapley, 1986; Levitt, Schumer, Sherman, Spear, & Movshon, 2001; Merigan & Maunsell, 1993) and even appears to have been accepted by Skottun & Skoyles in their comment on our paper (paragraph 5).

Some additional results in the paper compare the VESPA responses to the FULL-RANGE stimulus with those to the HIGH-CONTRAST and HIGH-CONTRAST_HSF stimuli. No difference was evident between the responses to these three stimuli suggesting that they were largely driven by the same subpopulation or populations of cells. Given that the FULL-RANGE stimulus spent a full 98% of it's time above 10% contrast where magnocellular contrast gain is low and that both other stimuli obviously spent 100% of their time above 10% contrast, it seems highly probable that the processing of the smooth contrast manipulations at these higher contrasts have been dominated by the parvocellular pathway.

In light of our experiments and results, we are somewhat surprised by the determination of Skottun & Skoyles that three issues warrant extra clarification. This is particularly true in light of the fact that the first two of their issues have been raised before several times (Skottun, 2000; Skottun & Skoyles, 2007a, 2007b, 2008a, 2008b, 2008c) and have been addressed at length several times (Butler et al., 2007b; Keri, 2008). What is perhaps even more surprising is that just one of their three issues, the third one, actually relates to the primary data presented in our study and in no way invalidates what we have written. Despite this concern about the germaneness of the concerns raised, we hereby address them on an issue-by-issue basis.

Issue 1: This issue is not concerned with our experiment, or with our conclusions, but with a statement made in the introduction of our paper: "M [i.e. magnocellular] cells favor … stimuli with low spatial frequency and high temporal frequency, whereas P cells … respond best to high spatial frequency and somewhat lower temporal frequency stimuli."

0042-6989/$ - see front matter
doi:10.1016/j.visres.2009.12.001

First, Skottun & Skoyles' raise the point that "With regard to spatial frequency, when eccentricity is taken account of, magno- and parvocellular neurons do not actually differ much". *Even* if it were true that they do not "differ much", the fact remains that they *do* differ (Derrington & Lennie, 1984) and thus the sentence with which they choose to take issue is a valid one (for a recent review see Nassi & Callaway, 2009). The accuracy of their statement can also be brought into question. Skottun and Skoyles base their conclusion that spatial frequency does not differ much between the two cell types on a study in Old World monkeys (Blakemore & Vital-Durand, 1986). This study reported that magno- and parvocellular X-type cells had virtually identical spatial resolution, but that magnocellular Y-type cells "...generally had poorer resolution". The issue of the lower resolution magnocellular Y cells is not mentioned by Skottun & Skoyles. Another weakness with their choice of reference is the paper by Dacey and Petersen (1992) that reports that human parasol (M) cells are much bigger than those in monkeys while the midget (P) cells are similar in size. In fact, M cells are reported to be almost twice as large near central retina which strongly suggests a lower spatial resolving ability. Given that our study is carried out in humans with stimuli located at fixation, we feel that our comment that M cells favor stimuli with low spatial frequency is valid.

Furthermore, in terms of spatial frequency differences between M and P at a *system level*, it has been reported that the order-of-magnitude greater sampling density of retinal P ganglion cells mediates superior acuity (Merigan & Katz, 1990), with Dacey and Petersen (1992) suggesting that the ratio of human P to M cells in central retina may be as much as 30:1. This lends considerable weight to the notion put forth by Skottun & Skoyles themselves that there may be differences in average spatial resolution based on the relative distributions of P and M cells in the retina. This is seen to be an especially relevant comment when one considers that center sizes of non-human P and M cell receptive fields are reported to have Gaussian radii of only 2–4 min of arc in macaque parafovea (Lee, Kremers, & Yeh, 1998) and that in our recent study, stimuli subtended 5.25° of visual angle. As illustrated in the table in Kaplan (2003), while the spatial resolution of individual (non-human) neurons is similar between M and P cells, the acuity of the cell groups as a whole differs, with P and M having high and low acuity respectively. This system level consideration would appear to be of even more relevance than the single-cell reports, which nonetheless still argue our case. We would note that this is previously trodden ground and was effectively addressed by Butler et al. (2007b) in response to a similar critique by Drs. Skottun and Skoyles.

The second part of Issue 1 relates to temporal frequency. Skottun & Skoyles say that the "temporal frequency differences between magno- and parvocellular neurons would appear to be rather small". Derrington and Lennie (1984) report that "[p]arvocellular units were most sensitive to stimuli modulated at temporal frequencies close to 10 Hz; magnocellular units to stimuli modulated at frequencies nearer 20 Hz. The loss of sensitivity as temporal frequency fell below optimum was more marked in magnocellular than parvocellular units". This *twofold* difference appears larger than the reports cited by Skottun & Skoyles. For example, Levitt et al. (2001) found that the ratio of temporal frequency cutoff between M and P was only 1.44. For stimuli restricted to the central 5°, this figure fell to 1.29. While this evidence validates our claim that M cells favor high temporal frequency while P cells respond best to somewhat lower temporal frequency stimuli, there is no doubt that there is overlap between M and P cells in terms of preferred temporal frequency. For example, Merigan and Maunsell (1993) report that the P and M pathways differ little in temporal resolution (highest temporal frequency that can be seen) at high-contrasts, however, they also note that they differ *greatly* at lower contrasts. All of this evidence seems to validate the notion that it might be possible to manipulate temporal frequency to bias responses from M and P pathways, particularly at low contrast. That said, in an effort to keep our experiment simple, we did not use manipulations of temporal frequency, and we remain confused as to why Skottun & Skoyles felt it necessary to raise this issue in relation to our paper.

Before leaving Issue 1, Skottun & Skoyles also dedicated a paragraph to discussing the issue of the spatial and temporal frequency of stimuli at or near threshold. It is not quite clear how this paragraph relates to our study, given that our stimuli are above threshold. However, we are nonetheless happy to address those comments when discussing Issue 2.

Issue 2: while Skottun and Skoyles cited single-cell studies in defense of Issue 1, they suggest that single-cell reports are not as suitable for the discussion of Issue 2. The reason for this selectivity is unclear. Instead they choose to focus on a restricted number of lesion and psychophysical studies. With respect to the former, Skottun & Skoyles say "...behavioral studies of contrast sensitivity in monkeys following magno- and parvocellular lesions are at conflict with the single cell research since they reveal that the largest reductions in contrast sensitivity occur following parvocellular lesions (Merigan, Byrne, & Maunsell, 1991; Merigan, Katz, & Maunsell, 1991; Merigan & Maunsell, 1990, 1993; Schiller, Logothetis, & Charles, 1990a, 1990b)". We feel that this is a rather incautious misinterpretation of the results of these studies. Table 1 displays quotes from the abstracts and conclusions of each of these references.

Study	Quote
Schiller et al. (1990a)	Little or no deficits were found in...contrast sensitivity after the disruption of either of the channels
Schiller et al. (1990b)	Parvocellular lesions impaired color vision, high spatial-frequency form vision, and fine stereopsis. Magnocellular lesions impaired high temporal-frequency flicker and motion perception but produced no deficits in stereopsis. Low spatial-frequency form vision, stereopsis, and brightness perception were unaffected by either lesion
Merigan and Maunsell (1990)	Together, these results suggest that the magnocellular pathway makes little contribution to visual sensitivity at low to moderate temporal frequencies. On the other hand, some contribution to detection sensitivity is evident at lower spatial and high temporal frequencies, especially for drifting stimuli. [NB – only magnocellular lesions carried out.]
Merigan, Byrne et al. (1991)	Magnocellular lesions greatly reduced detection contrast sensitivity at high temporal and low spatial frequencies and had a similar effect on contrast sensitivity for opposite direction discrimination under these same stimulus conditions. [NB – only magnocellular lesions carried out.]
Merigan, Katz et al. (1991)	This study demonstrates that the parvocellular pathway dominates chromatic vision, acuity, and contrast detection at low temporal and high spatial frequencies, while the magnocellular pathway may mediate contrast detection at higher temporal and lower spatial frequencies

Merigan and Maunsell (1993) (review paper)	Lesion studies suggest that the most fundamental specialization of these two pathways may be the…range of temporal and spatial frequencies that can be seen

The obvious message that stems from this is that P and M pathways are functionally distinct with basic specializations for low-level properties, such as spatial and temporal frequency, and this message is backed up by other work by some of the same authors (e.g. Merigan & Eskin, 1986). This is in complete agreement with single-cell work (Nassi & Callaway, 2009). Clearly, the statement "that the largest reductions in contrast sensitivity occur following parvocellular lesions" is a very selective interpretation of these results (see Fig. 2 in Merigan & Maunsell, 1993). In fact, later in the same paragraph Skottun & Skoyles appear to admit as much – "The magnocellular system has lower contrast threshold *only* when the stimuli are of low spatial frequency and/or high temporal frequency" (italics added). The reason for this prejudice against stimuli of low spatial frequency and/or high temporal frequency is not clear to us. Furthermore, as stated by Kaplan (2003), "[t]he evidence from lesion experiments is especially difficult to interpret, since the brain is a plastic, adaptable network, and removing, damaging or otherwise inactivating any of its parts tells you not what that part is doing, but rather what the brain can (or cannot) do without that part".

One of the papers that Skottun and Skoyles cite in defense of their argument (that there are reasons for caution with regard to the interpretation that our low contrast stimuli are biased towards the magnocellular pathway) reports that, while lesion studies indicate that the P pathway *can* mediate the detection of contrasts as low as 0.5% at low temporal frequencies, the contrast sensitivity of M cells is typically many times that of P cells, with P cells *rarely* responding well to luminance contrasts below 10%, and M cells *often* responding to stimuli with contrasts as low as 2% (Merigan & Maunsell, 1993). Furthermore, it has been reported that cortical recipients of magnocellular inputs respond to low contrast whereas those receiving parvocellular input do not respond until contrast attains at least ~8% (Tootell, Silverman, Hamilton, Switkes, & De Valois, 1988). This evidence strongly validates the notion that processing of low contrast stimuli is dominated by M cells, especially at high temporal frequencies, and is widely accepted.

Skottun & Skoyles also cite human psychophysics to counter the single-cell results that validate the fact that the magnocellular system responds to lower contrast than the parvocellular system. Their claim that human psychophysics is in agreement with the lesion studies is accurate. However, this is true only in so far as the psychophysical studies suggest a transient "channel" for processing threshold stimuli with low spatial frequency and a sustained "channel" for processing those with high spatial frequency. (Kulikowski & Tolhurst, 1973; Legge, 1978; Tolhurst, 1975a, 1975b). The analogy with results from single-cell studies showing that parvocellular and magnocellular neurons exhibit sustained and transient responses respectively is palpable.

As with Issue 1, it is not entirely clear why Skottun & Skoyles have raised the issue of contrast sensitivity work when the results presented in this paper deal with suprathreshold stimuli and contrast gain. Incidentally, it is worth noting here that more recent psychophysical work involving suprathreshold stimuli has concluded that the magnocellular system dominates close-to-threshold detection, whereas the parvocellular system dominates detection at higher contrasts, when the magnocellular system saturates (Plainis & Murray, 2005).

Issue 3: Skottun & Skoyles take issue here with our claim that the widely accepted and dramatic difference between the contrast gain curves of M and P cells (Kaplan, 1991, 2003; Kaplan & Shapley, 1986; Levitt et al., 2001; Merigan & Maunsell, 1993) allows for us to infer that our low contrast (0–10%) stimuli are biased towards the magnocellular pathway. Their argument is, essentially, that other cells could also play a role. We believe this argument to be insubstantial. Firstly, they cite research that has been performed on Owl Monkeys, the *only* nocturnal member of the Anthropoidea (Old World monkeys, New World monkeys, apes and humans). They refer specifically to the report that, in Owl Monkeys, K cells have been found to have contrast gain curves that are more similar to M cells than P cells (Kilavik, Silveira, & Kremers, 2007; Xu, Bonds, & Casagrande, 2002; Xu et al., 2001). Inferring a similar relationship in humans is highly questionable given the particular nature of the Owl Monkey's customary visual habitat and the unique laminar arrangement of their K cells in LGN (Hendry & Reid, 2000). Perhaps a more appropriate source for drawing comparison with humans might be the report from White, Solomon, and Martin (2001) on K cells in the marmoset, a diurnal monkey with well-defined koniocellular layers. That study did indeed show similarities in the contrast gain curves of K cells and M cells, however it also demonstrated lower maximum response amplitude in K cells compared to both M and P cells, which suggests a reduced global influence for K cells compared to M cells. This issue is amplified by the fact that, in primates, the divergence ratio for M cells (i.e., the number of neurons in layer 4Cα versus thalamocortical neurons in the M layers) is 1:300 (Peters, Payne, & Budd, 1994) whereas that of K cells is roughly 1:50 (Hendry & Reid, 2000). The lower efficacy indicated by the smaller amplitudes and this much reduced divergence ratio is further compounded by the much greater variation in the physiologic properties of K cells than those of either M or P cells (Hendry & Reid, 2000). Together, these reports strongly suggest a more limited impact for K cells at cortex, no matter what the contrast gain characteristics. Also, given the low contrast of our stimuli, which includes transitions from 0% to 2% contrast and vice versa, it is also worth noting that in the central visual field of marmosets, the sensitivity of koniocellular cells lies between that of parvocellular and magnocellular cells, while in the peripheral visual field, koniocellular and parvocellular cells have similar sensitivity (Solomon, White, & Martin, 1999).

With regard to their concerns that other cells contribute to MT (the middle temporal motion processing area), we wish to say two brief things. First, we not only fail to claim that "[a]rea MT will reflect only magnocellular activity", but we actually make no mention whatsoever of MT in our paper and are, thus, again, somewhat perplexed that Skottun and Skoyles chose to raise the issue in their critique. Secondly, while we certainly appreciate that there are other inputs to area MT (e.g. Nassi, Lyon, & Callaway, 2006), recent research on spatial and cell-type specificity in V1 have been shown to be consistent with the relaying of a quick, magnocellular-dominated signal to MT (Nassi & Callaway, 2009). This is widely accepted.

In their final paragraph Skottun & Skoyles remark that contrast sensitivity studies find little support for linking either schizophrenia or dyslexia to magnocellular deficits and that visual masking tasks do not support a magnocellular deficit in schizophrenia. These concerns have been answered several times (Butler et al., 2007b; Oğmen, Purushothaman, & Breitmeyer, 2008; Schulte-Körne, Remschmidt, Scheuerpflug, & Warnke, 2004). Even if there were some validity to the contrast sensitivity claims, the following question would remain: might not a magnocellular deficit in one or other of these disorders be manifest at higher contrasts in the cells operating range than those of threshold stimuli? In other words, is contrast sensitivity the optimal way to assess the full range of the magnocellular pathway's function?

Acknowledgment

We wish to thank Professor Vance Zemon for helpful comments.

References

Blakemore, C., & Vital-Durand, F. (1986). Organization and post-natal development of monkey's lateral geniculate nucleus. *Journal of Physiology (London), 380*, 491–543.

Butler, P. D., Martinez, A., Foxe, J. J., Kim, D., Zemon, V., & Silipo, G. (2007b). Reply: A few remarks on assessing magnocellular sensitivity in Schizophrenia patients. *Brain, 130*, e84.

Dacey, D. M., & Petersen, M. R. (1992). Dendritic field size and morphology of midget and parasol ganglion cells of the human retina. *Proceedings of the National Academy of Sciences, USA, 89*, 9666–9670.

Derrington, A. M., & Lennie, P. (1984). Spatial and temporal contrast sensitivities of neurons in lateral geniculate nucleus of macaque. *Journal of Physiology, 357*, 219–240.

Hendry, S. H. C., & Reid, R. C. (2000). The koniocellular pathway in primate vision. *Annual Review of Neuroscience, 23*, 127–153.

Kaplan, E. (1991). The receptive field structure of retinal ganglion cells in cat and monkey. In A. G. Leventhal (Ed.), *Vision and visual dysfunction*. Boston: CRC Press (pp. 10–40).

Kaplan, E. (2003). The M, P, and K pathways of the primate visual system. In L. H. Chalupa & J. S. Werner (Eds.), *The visual neurosciences* (pp. 481–493). Cambridge: MIT Press.

Kaplan, E., & Shapley, R. M. (1986). The primate retina contains two types of ganglion cells, with high and low contrast sensitivity. *Proceedings of the National Academy of Sciences, USA, 83*, 2755–2757.

Keri, S. (2008). The magnocellular pathway and schizophrenia. *Vision Research, 48*, 1181–1182.

Kilavik, B. E., Silveira, L. C. L., & Kremers, J. (2007). Spatial receptive field properties of lateral geniculate cells in the owl monkey (*Aotus azarae*) at different contrasts: A comparative study. *European Journal of Neuroscience, 26*, 992–1006.

Kulikowski, J. J., & Tolhurst, D. J. (1973). Psychophysical evidence for sustained and transient detectors in human vision. *Journal of Physiology (London), 232*, 149–162.

Lalor, E. C., & Foxe, J. J. (2009). Visual evoked spread spectrum analysis (VESPA) responses to stimuli biased towards magnocellular and parvocellular pathways. *Vision Research, 49*, 127–130.

Lalor, E. C., Pearlmutter, B. A., & Foxe, J. J. (2009). Reverse correlation and the VESPA method. In T. C. Handy (Ed.), *Brain signal analysis: Advances in neuroelectric and neuromagnetic methods* (pp. 1–20). Cambridge: MIT Press.

Lalor, E. C., Pearlmutter, B. A., Reilly, R. B., McDarby, G., & Foxe, J. J. (2006). The VESPA: A method for the rapid estimation of a visual evoked potential. *Neuroimage, 32*, 1549–1561.

Lee, B. B., Kremers, J., & Yeh, T. (1998). Receptive fields of primate retinal ganglion cells studied with a novel technique. *Visual Neuroscience, 15*, 161–175.

Legge, G. E. (1978). Sustained and transient mechanisms in human vision: Temporal and spatial properties. *Vision Research, 18*, 69–81.

Levitt, J. B., Schumer, R. A., Sherman, S. M., Spear, P. D., & Movshon, J. A. (2001). Visual response properties of neurons in the LGN of normally reared and visually deprived macaque monkeys. *Journal of Neurophysiology, 85*, 2111–2129.

Merigan, W. H., Byrne, C. E., & Maunsell, J. H. R. (1991). Does primate motion perception depend on the magnocellular pathway? *Journal of Neuroscience, 11*, 3422–3429.

Merigan, W. H., & Eskin, T. A. (1986). Spatio-temporal vision of macaques with severe loss of P_β retinal ganglion cells. *Vision Research, 26*, 1751–1761.

Merigan, W. H., & Katz, L. M. (1990). Spatial resolution across the macaque retina. *Vision Research, 30*, 985–991.

Merigan, W. H., Katz, L. M., & Maunsell, J. H. R. (1991). The effects of parvocellular lateral geniculate lesions on the acuity and contrast sensitivity of macaque monkeys. *Journal of Neuroscience, 11*, 994–1001.

Merigan, W. H., & Maunsell, J. H. R. (1990). Macaque vision after magnocellular lateral geniculate lesions. *Visual Neuroscience, 9*, 776–783.

Merigan, W. H., & Maunsell, J. H. R. (1993). How parallel are the primate visual pathways? *Annual Review of Neuroscience, 16*, 369–402.

Nassi, J. J., & Callaway, E. M. (2009). Parallel processing strategies of the primate visual system. *Nature Reviews Neuroscience, 10*, 360–372.

Nassi, J. J., Lyon, D. C., & Callaway, E. M. (2006). The parvocellular LGN provides a robust disynaptic input to the visual motion area MT. *Neuron, 50*, 319–327.

Oğmen, H., Purushothaman, G., & Breitmeyer, B. G. (2008). Metacontrast, target recovery, and the magno- and parvocellular systems: A reply to the perspective. *Visual Neuroscience, 25*, 611–616.

Peters, A., Payne, B. R., & Budd, J. (1994). A numerical analysis of the geniculocortical input to striate cortex in the monkey. *Cerebral Cortex, 4*, 215–229.

Plainis, S., & Murray, I. J. (2005). Magnocellular channel subserves the human contrast-sensitivity function. *Perception, 34*, 933–940.

Schiller, P. H., Logothetis, N. K., & Charles, E. R. (1990a). Functions of the colour-opponent and broad-band channels of the visual system. *Nature, 343*, 68–70.

Schiller, P. H., Logothetis, N. K., & Charles, E. R. (1990b). Role of the color-opponent and broad-band channels in vision. *Visual Neuroscience, 5*, 321–346.

Schulte-Körne, G., Remschmidt, H., Scheuerpflug, P., & Warnke, A. (2004). The role of the magnocellular pathway in dyslexia – Reply to Skottun and Skoyles. *Clinical Neurophysiology, 115*, 2836–2838.

Skottun, B. C. (2000). The magnocellular deficit theory of dyslexia: The evidence from contrast sensitivity. *Vision Research, 40*, 111–127.

Skottun, B. C., & Skoyles, J. R. (2007a). Contrast sensitivity and magnocellular functioning in schizophrenia. *Vision Research, 47*, 2923–2933.

Skottun, B. C., & Skoyles, J. R. (2007b). A few remarks on assessing magnocellular sensitivity in Schizophrenia patients. *Brain, 130*, e83.

Skottun, B. C., & Skoyles, J. R. (2008a). Reply to Kéri, S. The magnocellular system and schizophrenia. *Vision Research, 48*, 1183–1185.

Skottun, B. C., & Skoyles, J. R. (2008b). Temporal frequency and the magnocellular and parvocellular systems. *Neuro-Ophthalmology, 32*, 43–48.

Skottun, B. C., & Skoyles, J. R. (2008c). Spatial frequency and the magno-/parvocellular distinction – Some remarks. *Neuro-Ophthalmology, 32*, 179–186.

Skottun, B. C., & Skoyles, J. R. (2009). Are masking abnormalities in schizophrenia limited to backward masking? *International Journal of Neuroscience, 119*, 88–104.

Solomon, S. G., White, A. J. R., & Martin, P. R. (1999). Temporal contrast sensitivity in the lateral geniculate nucleus of a New World monkey, the marmoset *Callithrix jacchus*. *Journal of Physiology, 517*, 907–917.

Tolhurst, D. J. (1975a). Reaction times in the detection of gratings by human observers: A probabilistic mechanism. *Vision Research, 15*, 1143–1149.

Tolhurst, D. J. (1975b). Sustained and transient channels in human vision. *Vision Research, 15*, 1151–1155.

Tootell, R. B., Silverman, M. S., Hamilton, S. L., Switkes, E., & De Valois, R. L. (1988). Functional anatomy of macaque striate cortex V. Spatial frequency. *Journal of Neuroscience, 8*, 1610–1624.

White, A. J. R., Solomon, S. G., & Martin, P. R. (2001). Spatial properties of koniocellular cells in the lateral geniculate nucleus of the marmoset *Callithrix jacchus*. *Journal of Physiology, 533*, 519–535.

Xu, X., Bonds, A. B., & Casagrande, V. A. (2002). Modeling receptive-field structure of koniocellular, magnocellular, and parvocellular LGN cells in the owl monkey (*Aotus trivigatus*). *Visual Neuroscience, 19*, 703–711.

Xu, X., Ichida, J. M., Allison, J. D., Boyd, J. D., Bonds, A. B., & Casagrande, V. A. (2001). A comparison of koniocellular, magnocellular and parvocellular receptive field properties in the lateral geniculate nucleus of the owl monkey (*Aotus trivergatus*). *Journal of Physiology (London), 531*, 203–218.

Edmund C. Lalor [a,b,*]
John J. Foxe [a,c,d,e]

[a] *Trinity College Institute of Neuroscience, Trinity College Dublin, Dublin 2, Ireland*

[b] *Trinity College Centre for Bioengineering, Trinity College Dublin, Dublin 2, Ireland*

[c] *The Cognitive Neurophysiology Laboratory, Nathan S. Kline Institute for Psychiatric Research, Program in Cognitive Neuroscience and Schizophrenia, 140 Old Orangeburg Road, Orangeburg, NY 10962, USA*

[d] *The Cognitive Neurophysiology Laboratory, Children's Evaluation and Rehabilitation Center, Department of Pediatrics, Albert Einstein College of Medicine, Rose F. Kennedy University Center for Excellence in Developmental Disabilities, 1410 Pelham Parkway South, Bronx, NY 10461, USA*

[e] *The Cognitive Neurophysiology Laboratory, Children's Evaluation and Rehabilitation Center, Department of Neuroscience, Albert Einstein College of Medicine, Rose F. Kennedy University Center for Excellence in Developmental Disabilities, 1410 Pelham Parkway South, Bronx, NY 10461, USA*

* Address: Trinity College Institute of Neuroscience, Trinity College Dublin, Dublin 2, Ireland.
Fax: +353 1 6772442.
E-mail address: edlalor@tcd.ie (E.C. Lalor)