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LETTER

Metacontrast, target recovery, and the magno- and parvocellular systems: A reply to the perspective

Backward masking refers to the reduced visibility of a target stimulus when it is followed in time by a second stimulus called the mask (reviews: Bachmann, 1994; Breitmeyer & Öğmen, 2000, 2006). The visibility of a target (T) masked by a primary mask (M1) can be recovered if an appropriately timed secondary mask, M2, is added to the T–M1 sequence. This phenomenon is known as ''target disinhibition'' or ''target recovery'' (e.g., Dember & Purcell, 1967; Robinson, 1966, 1968; Long & Gribben, 1969; Schiller & Greenfield, 1969; Purcell & Stewart, 1975; Dember et al., 1978; Kristofferson et al., 1979; Byron & Banks, 1980; Tenkink & Werner, 1981; Purcell et al., 1982; Briscoe et al., 1983). Metacontrast is a special type of backward masking where the target and mask stimuli do not spatially overlap. Breitmeyer et al. (1981) showed that maximum target recovery in metacontrast occurs when M2 temporally precedes M1. Furthermore, their results show that for the T–M2 stimulus onset asynchrony (SOA) range where target recovery occurs, there is no concomitant change in the visibility of the primary mask M1, indicating a dissociation between target recovery and the visibility of the primary mask M1. For the T–M2 SOA range where the visibility of M1 is suppressed, the opposite dissociative effect is observed, that is, there is no concomitant change in the visibility of the target. This double dissociation is taken as strong evidence against single-channel/single-process models of metacontrast, which attribute the visibility of a stimulus and its masking effectiveness to the same process. On the other hand, these findings can be explained by the dual-process REtino-COrtical Dynamics (RECOD) model (Öğmen, 1993; Breitmeyer & Öğmen, 2000, 2006). An extensive discussion of this model and its predictive scope can be found in Breitmeyer and Öğmen (2006, Chapter 5). In Öğmen et al. (2006*a*), we presented simulations showing that this model can account quantitatively for the double dissociation reported by Breitmeyer et al. (1981).

In a recent study, published in *Vision Research* (Öğmen et al., 2006*b*), we analyzed further this model and derived the novel prediction that contrast dependence of metacontrast and target recovery should parallel the contrast dependence of afferent magno- and parvocellular pathways, respectively. In a psychophysical experiment, we tested this prediction by systematically varying M2's contrast and the M1–M2 SOA. At the optimal M1–M2 SOA, the target recovery effect increased with M2's contrast without saturating, but at the optimal M1–M2 metacontrast SOA, the reduction of M1's visibility saturated very rapidly as M2's contrast increased. Quantitative comparisons of model simulations with psychophysical results provided additional support for our prediction. We concluded that metacontrast masking is driven by signals originating from the magnocellular pathway and that target recovery in metacontrast is driven by signals originating from the parvocellular pathway.

Recently, Skottun and Skoyles (2007) published in this Journal a commentary on our *Vision Research* paper. Here we are responding point by point to the issues raised by Skottun and Skoyles.

Point 1: ''because pure magno- and parvocellular streams do not exist beyond the primary cortex, any interactions between pure magno- and parvocellular inputs (such as, e.g., inhibition between the systems) should not be sought at a level beyond the primary visual cortex.''

This is not an objection to our model but rather an effort by Skottun and Skoyles to identify the locus of the interactions between the two streams posited in our model. The absence of *pure* parvo- and magnocellular systems beyond V1 does not rule out the possibility that the interactions put forward in our model could occur beyond V1 because we do not postulate interactions between pure magno- and parvocellular inputs. Rather, as we stated in our paper, we postulate interactions between systems that receive their *dominant* input from magno- and parvocellular pathways (see, e.g., Öğmen et al., 2006*b*, p. 4728, line 15). Hence, we consider the suggestion that the interactions between the two streams in our model cannot occur beyond V1 to be inconsequential to our results.

Skottun and Skoyles arrive at the conclusion that the interactions between the two pathways, as described in our model, cannot occur beyond the primary visual cortex based on the findings indicating a certain level of mixing of these inputs in V1. As evidence for the absence of ''pure'' parvo- and magnocellular systems beyond V1, Skottun and Skoyles cite studies that have shown mixing of parvo- and magnocellular signals within some layers and in some neurons of V1. But such mixing within the primary visual cortex is not inconsistent with the well-demonstrated fact that distinct groups of extrastriate neurons show largely independent influences of parvo- and magnocellular subdivisions. For example, Maunsell et al. (1990) studied the effect of selectively blocking parvo- and magnocellular layers of the LGN on MT neural responses in macaque monkeys. They found that blocking magnocellular layers consistently reduced MT responses and that the reduction was pronounced and often complete. In contrast, blocking parvocellular layers of the LGN rarely affected MT responses. Thus, it appears that a majority of MT neurons are influenced far more by magnocellular input than by parvocellular input.

A vast majority of anatomical findings to date are consistent with this physiological finding. Magnocellular layers of the LGN project to layer 4Cα of V1, while parvocellular layers project to layer 4Cβ of V1 (Hubel & Wiesel, 1972; Hendrickson et al., 1978; Blasdel & Lund, 1983; Fitzpatrick et al., 1985). Within V1, spiny stellate neurons in layer 4B receive input only from 4Cα and hence receive only magnocellular input, but some pyramidal cells in layer 4B receive mixed parvo- and magnocellular inputs through both 4Cα and 4Cβ (Lund & Boothe, 1975; Fitzpatrick et al., 1985; Sawatari & Callaway, 1996; Yabuta & Callaway, 1998; Yabuta et al., 2001). In macaques, it was previously known that a majority of 4B neurons projecting to MT were of the spiny stellate type (that receives pure magnocellular input), but the exact connection patterns and types of pyramidal cells projecting to MT were not fully understood. It is now known that in every primate species examined, MT projecting cells are organized in segregated patches that are situated below the cytochrome oxidase (CO) blobs of layers 2/3, with their dendrites confined to magnocellular recipient zones in 4Cα (Lund et al., 1975; Tigges et al., 1981; Diamond et al., 1985; Shipp & Zeki, 1989; Boyd & Casagrande, 1999; Nassi & Callaway, 2007). Finally, a recent tracing study using transneuronal viral vectors has shown that even those pyramidal cells of 4B that project to MT are confined in this manner to the magnocellular recipient zones of 4Cα and are situated below the CO blobs (Nassi & Callaway, 2007). This study also showed that the majority of MT projecting cells in layer 4B of V1 have a distinct morphology with large cell bodies that seem suited for fast transmission, while V2 projecting cells have a different morphology that may mediate slower computations. Therefore, the response of an extrastriate region such as MT can be much more strongly influenced by one pathway than by the other, as demonstrated clearly by selective blocking experiments (Maunsell et al., 1990). There is much to be learned about the precise nature of the anatomical and functional relationship between the different types of neurons and interlaminar circuits in V1 and those of the extrastriate areas. In particular, very little is known about the nature of the synapses between the various circuits identified in the anatomical tracing studies. While it is quite possible that multisynaptic parvo- and magnocellular inputs exist for many extrastriate neurons (Nassi & Callaway, 2006), the impact that input from each pathway has on the responses of the target neuron will depend on a variety of factors including the morphology and neurochemistry of the corresponding synapses. It is not known which striate-to-extrastriate projections are ''drivers'' and which ones are ''modulators'' (Sherman & Guillery, 1998).

In summary, currently known anatomical and physiological facts overwhelmingly support the notion that both at the level of V1 and beyond, parvo- and magno-dominant signals can interact in the manner posited in the RECOD model.

Point 2: The difference in the response properties of parvo- and magnocellular neurons to achromatic stimuli is ''relatively small.''

The function of the RECOD model depends on certain key properties of the two information streams being significantly different. Specifically, the model assumes that temporal responses and contrast sensitivity of the neurons in the two streams are different. Skottun and Skoyles claim that the difference in the response properties of parvo- and magnocellular neurons to achromatic stimuli is relatively small. Somewhat surprisingly, they cite Levitt et al. (2001) as evidence supporting this claim. Contrary to this claim, Levitt et al. (2001) report that in response to achromatic stimuli, ''In agreement with previous studies, we find that on average magnocellular neurons differ from parvocellular neurons by having shorter latencies to optic chiasm stimulation, greater sensitivity to luminance contrast, and better temporal resolution.'' The results of Levitt et al. (2001) only show that there were no ''major differences between magno- and parvocellular neurons on the basis of most *spatial* parameters'' (emphasis added). However, they found that at a given eccentricity, the smallest receptive fields were of the parvocellular type. All these findings are consistent with the parameters chosen to simulate our model in various studies (Purushothaman et al., 2000; Öğmen et al., 2003, 2006*a*,*b*).

Several studies have also shown that magnocellular cells have phasic responses while parvocellular cells have tonic responses in both New and Old World primates (Purpura et al., 1988, 1990; Yeh et al., 1995; review: Kaplan, 2004), and in fact, this seems to be true for all mammals (review: van Hooser et al., 2003). Other major physiological differences reported between parvo- and magnocellular populations include contrast gain (Kaplan & Shapley, 1986; Croner & Kaplan, 1995) and contrast gain control (Benardete et al., 1992; Benardete & Kaplan, 1997, 1999). All these results were obtained for achromatic stimuli. While it is true that statistical distributions of the physiological properties of parvo- and magnocellular neurons show considerable overlap (e.g., White et al., 2001), we know of no studies that have shown that parvo- and magnocellular pathways are indistinguishable based on their responses to achromatic stimuli in any species (for reviews, see Casagrande & Norton, 1991; van Hooser et al., 2003; Casagrande & Xu, 2004; Kaplan, 2004).

Point 3: The differences between response latencies of magno- and parvocellular systems are too small to account for the timing of metacontrast and target recovery.

In our model, we do not assume that the latency difference between afferent magno- and parvocellular signals is the only determinant of the SOA for optimal masking. Rather, these two input signals feed into two cortical systems that mutually interact through inhibitory processes. Therefore, the optimal SOA depends not only on latency differences between afferent magno- and parvocellular signals but also on the dynamics of these two interacting cortical systems. This, in turn, requires a comparison of latencies between cortical networks relevant to the perceptual dimension probed by the metacontrast experiment. For example, we recently showed that the metacontrast SOA for obtaining optimal suppression of contour or edge information was 10 ms, whereas that for obtaining optimal suppression of achromatic surface contrast was 40 ms (Breitmeyer et al., 2006). Moreover, Schwartz and Loop (1982, 1983) showed that chromatic stimulus properties are processed 50–100 ms slower than achromatic properties. These findings suggest that there may be intrinsic latency differences not only between the processing of the contour and the processing of the achromatic surface contrast of a stimulus but also between the processing of surface colors and the processing of its achromatic surface contrast. In view of these psychophysical findings, neurophysiological recordings of response latency differences between M and P neurons need to be subcategorized in the following manner (for a schematic illustration, see fig. 5 in Breitmeyer et al., 2006). One needs to establish separately the latency differences between cortical M- and P-interblob (interstripe) and between cortical M- and P-blob (thin stripe) neurons and, second, differences between the P-thin stripe achromatic and P-thin stripe chromatic neurons (Xiao et al., 2003; Wang et al., 2007). Only after such more careful work can

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generalizations be made regarding how M and P response latency differences relate to optimal metacontrast SOA and thus how they bear on the dual-channel model of metacontrast. Similarly, our model includes feedforward- and feedback-dominant phases of operation corresponding to activities generated by feedforward and feedback signaling, respectively (Öğmen, 1993). Accordingly, when the perceptual dimension probed by the metacontrast experiment relates to feedback signals, the relevant latency comparison is not between the onsets of P and M signals but rather between the timing of the M signal and that of the feedback signal, which can be substantially larger than the M-P onset latency difference (Lamme et al., 1998; Roelfsema et al., 2002).

Point 4: In terms of contrast effects, Skottun and Skoyles do not appear to object directly our interpretation; however, they state that ''although the nature of these effects may allow for differentiation of magno- and parvocellular responses these contrast-response relationships are not unique to these systems.'' They go on to state that neurons in MT exhibit magno-like and some neurons in V1 exhibit parvo-like contrast responses and suggest that cortical models based on these substrates can explain our findings.

The contrast dependence of target recovery and metacontrast was a novel prediction of our model, and the empirical tests provided support for this prediction. Other possibilities do exist, and we would welcome theoretical and empirical explorations of alternative interpretations. However, the suggestions offered by Skottun and Skoyles are neither analyzed nor developed sufficiently to offer viable alternatives.

A typical metacontrast display consists of a target flanked symmetrically by a mask. This display would then activate motion detectors that would signal the target moving in two opposite directions. Kahneman (1967) conceptualized this as ''impossible motion'' and proposed that the perceptual/cognitive system suppresses the target to resolve the conflicting motion information. Thus, this model implicates motion mechanisms in metacontrast. Skottun and Skoyles offered it as an alternative, suggesting that, since MT cells have rapidly saturating contrast responses, this model can explain the contrast dependence of metacontrast. While Skottun and Skoyles consider some aspects of Kahneman's model, they fail to take into account other aspects. Similarly, they consider one aspect of our findings while ignoring the rest. As discussed in Breitmeyer and Öğmen (2006, pp. 102–104), several studies provided direct evidence against Kahneman's model (Weisstein & Growney, 1969; Breitmeyer et al., 1974, 1976; Stoper & Banffy, 1977; Breitmeyer & Horman, 1981). Furthermore, Skottun and Skoyles do not elaborate, and it is not clear how Kahneman's model can predict target recovery, the timing of target recovery, the contrast dependence of target recovery, and the double dissociation between the visibility of the primary mask and target recovery.

Similarly, Skottun and Skoyles mention the Boundary Contour System (BCS) model (Grossberg & Mingolla, 1985) as a candidate. As we have already mentioned in our original manuscript, while this model can generate target recovery, it fails in its current form to explain the double dissociation between target recovery and metacontrast (Francis, 1997). In addition, the timing of target recovery predicted by this model does not match the data well. It is not clear whether this model can predict the specific contrast dependencies associated with metacontrast and target recovery.

Point 5: Skottun and Skoyles stated that ''central to the argument of Öğmen et al. (2006*b*) is the notion that parvocellular activity determines the 'visibility' of stimuli.'' They go on to suggest that ''at least under some stimulus conditions (low contrast, high temporal frequency and low spatial frequency) *detection* is mediated by the magnocellular system, which means that visibility is determined by this system under these conditions'' (emphasis added).

We did not claim that parvocellular activity determines the visibility of stimuli in general. Rather, our paper was very specific in stating, ''And finally, as a linking assumption, the model uses time-integrated activities in the sustained post-retinal areas as a correlate for perceived brightness (. . .).'' That detection can be carried out by systems receiving dominant magnocellular activity in conditions cited by Skottun and Skoyles is completely in agreement with our model (e.g., Breitmeyer & Öğmen, 2006, Section 5.2.5, p. 164). It is well known that in metacontrast, there exists a dissociation between detecting the presence (or location) of a target and detecting the target's perceived brightness (Fehrer & Raab, 1962; Schiller & Smith, 1966). We showed that our model can explain this dissociation by noting that target detection and localization can be carried out by using magnocellular-induced activity (Öğmen et al., 2003). Furthermore, we extended the analysis of the model to paracontrast, a paradigm similar to metacontrast with the exception that the mask precedes in time the target. We made the novel prediction that in paracontrast, reciprocal inhibition between sustained and transient systems should interfere with target detection and localization, thereby causing an increase in reaction times (RTs). This prediction was confirmed by experiments reported in Öğmen et al. (2003). In summary, as the examples above show, the term ''visibility'' needs to be qualified within the context of task requirements of the experiment and the attendant criterion contents that generate the behavioral responses.

Point 6: Regarding the effects of uniform long-wavelength, red backgrounds, relative to medium-wavelength, green or short-wavelength, blue ones, in reducing the magnitude of metacontrast (Breitmeyer & Williams, 1990; Edwards et al., 1996), Skottun and Skoyles argue that such findings cannot be used to support the RECOD model's fundamental assumption that cortical magnocellular activity suppresses parvocellular activity to produce metacontrast. The suppressive effects of red light on the response of magnocellular neurons throughout the retino-geniculo-striate tract of macaques have been reported repeatedly (Wiesel & Hubel, 1966; Dreher et al., 1976; De Monasterio & Schein, 1980; Marrocco et al., 1982; Livingstone & Hubel, 1984). Skottun's (2004) analysis of parvocellular color-opponent cells points out that uniform red, green, and blue backgrounds may also differentially affect responses within, especially, the R-G opponent system. Granted this, it is indeed possible that the effects of red backgrounds, relative to green or blue ones, on metacontrast magnitude may be due to contributions of the parvocellular system. However, unless one can show that *all* the color-dependent modulation of metacontrast strength is due to correlated color-dependent modulation of parvocellular responses, Skottun's (2004) analysis does not rule out the role of the magnocellular system.

Moreover, there are two sources of independent psychophysical evidence showing that the suppression of the magnocellular response by red light ought to contribute to a reduction in metacontrast. First, Breitmeyer and Breier (1994) showed that the effects of a red, as compared to blue or green, background on the detection of luminance increments of the same color as the background interacted with the size (diameter) of the stimulus. For *small-diameter* (8 minarc) increments, red backgrounds *decreased* the detection RT relative to green or blue ones. In contrast, for

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large-diameter (32 and 64 minarc) increments, red backgrounds *increased* the detection RTs. Besides supporting Skottun's (2004) analysis of the effects of red light on the parvocellular R-G opponent system, this would indeed be expected if the high–spatial frequency parvocellular system responds to the small stimuli and the low–spatial frequency magnocellular system responds to the larger stimuli. Additionally, what was particularly telling in the results reported by Breitmeyer and Breier (1994) is that "these interaction effects between background color and stimulus size were eliminated when the stimuli consisted of luminance decrements," again of the same color as the background. These differential effects of luminance increments and decrements were entirely consistent with and thus predictable by the findings of De Monasterio and Schein (1980) and Wiesel and Hubel (1966) that the suppressive effects of uniform red light are particularly strong in on-center magnocellular neurons. This makes a clear prediction vis-à-vis metacontrast: The reduction of metacontrast on a red, as compared to green or blue, background ought to be greater when the stimuli consist of luminance increments than when they consist of luminance decrements.

A second independent source derives from psychophysical studies of the differential effects of red and green backgrounds on categorical and coordinate spatial judgments. According to a neural network model of spatial processing proposed by Kosslyn et al. (1992), categorical spatial judgments are predicted by receptive-field properties of parvocellular neurons, while coordinate spatial judgments are predicted by receptive-field properties of magnocellular neurons. Roth and Hellige (1998) confirmed this prediction by showing that coordinate spatial judgments were slowed down when stimuli were presented on a red, as compared to green, background. Consequently, based on these lines of evidence, we maintain that a reduction in magnocellular response produced by diffuse red light contributes not only to a reduction in metacontrast magnitude but also to a variety of other reductions in psychophysical performances tied to the magnocellular system.

In summary, Skottun and Skoyles' arguments are based on invalid assumptions about our model and therefore do not provide any contradictory evidence against our interpretations and conclusions. Furthermore, their interpretations and generalizations of neurophysiological data do not appear warranted when a broader view of the extant data is taken into account.

References

Bachmann, T. (1994). *Psychophysiology of Visual Masking: The Fine Structure of Conscious Experience*. New York: Nova Science Publishers.

Benardete, E.A. & Kaplan, E. (1997). The receptive field of the primate P retinal ganglion cell, 2. Nonlinear dynamics. *Visual Neuroscience* **14**, 187–205.

Benardete, E.A. & Kaplan, E. (1999). The dynamics of primate M retinal ganglion cells. *Visual Neuroscience* **16**, 355–368.

Benardete, E.A., Kaplan, E. & Knight, B.W. (1992). Contrast gain-control in the primate retina—P-cells are not x-like, some m-cells are. *Visual Neuroscience* **8**, 483–486.

Blasdel, G.G. & Lund, J.S. (1983). Termination of afferent axons in macaque striate cortex. *Journal of Neuroscience* **3**, 1389–1413.

Boyd, J.D. & Casagrande, V.A. (1999). Relationships between cytochrome oxidase (CO) blobs in primate primary visual cortex (V1) and the distribution of neurons projecting to the middle temporal area (MT). *Journal of Comparative Neurology* **409**, 573–591.

Breitmeyer, B.G., Battaglia, F., & Weber, C. (1976). U-shaped backward contour masking during stroboscopic motion. *Journal of Experimental Psychology: Human Perception and Performance* **2**, 167–173.

Breitmeyer, B.G. & Breier, J.I. (1994). Effects of background color on reaction time to stimuli varying in size and contrast: Inferences about human transient channels. *Vision Research* **34**, 1039–1045.

Breitmeyer, B.G. & Horman, K. (1981). On the role of stroboscopic motion in metacontrast. *Bulletin of the Psychonomic Society* **17**, 29–32.

Breitmeyer, B.G., Kafaligonul, H., Öğmen, H., Mardon, L., Todd, S. & Ziegler, R. (2006). Meta- and paracontrast reveal differences between contour- and brightness-processing mechanisms. *Vision Research* **46**, 2645–2658.

Breitmeyer, B.G., Love, R. & Wepman, B. (1974). Contour masking during stroboscopic motion and metacontrast. *Vision Research* **14**, 1451–1456.

Breitmeyer, B.G. & Öğmen, H. (2000). Recent models and findings in visual backward masking: A comparison, review, and update. *Perception & Psychophysics* **62**, 1572–1595.

Breitmeyer, B.G. & Öğmen, H. (2006). *Visual Masking: Time Slices through Conscious and Unconscious Vision*. Oxford: Oxford University Press.

Breitmeyer, B.G., Rudd, M. & Dunn, K. (1981). Metacontrast investigations of sustained-transient channel inhibitory interactions. *Journal of Experimental Psychology: Human Perception and Performance* **7**, 770–779.

Breitmeyer, B.G. & Williams, M.C. (1990). Effects of isoluminant-background color on metacontrast and stroboscopic motion: Interactions between sustained (P) and transient (M) channels. *Vision Research* **30**, 1069–1075.

Briscoe, G., Dember, W.N. & Warm, J.S. (1983). Target recovery in visual backward masking: No clear explanation in sight. *Journal of Experimental Psychology: Human Perception and Performance* **9**, 898–911.

Byron, D. & Banks, W.P. (1980). Patterned stimuli in disinhibition and backward masking. *Bulletin of the Psychonomic Society* **15**, 105–108.

Casagrande, V.A. & Norton, T.T. (1991). Lateral geniculate nucleus: A review of its physiology and function. In *The Neural Basis of Visual Function*, ed. Leventhal, A.G., pp. 41–84. London, UK: MacMillan Press.

Casagrande, V.A. & Xu, X. (2004). Parallel visual pathways: A comparative perspective. In *Visual Neurosciences*, ed. Chalupa, L.M. & Werner, J.S., pp. 494–506. Cambridge, MA: MIT Press.

Croner, L.J. & Kaplan, E. (1995). Receptive-fields of P-ganglion and M-ganglion cells across the primate retina. *Vision Research* **35**, 7–24.

De Monasterio, F.M. & Schein, S.J. (1980). Protan-like spectral sensitivity of foveal Y ganglion cells of the retina of macaque monkeys. *Journal of Physiology* **299**, 385–396.

Dember, W.N. & Purcell, D.G. (1967). Recovery of masked visual targets by inhibition of the masking stimulus. *Science* **157**, 1335–1336.

Dember, W.N., Schwartz, M. & Kocak, M. (1978). Substantial recovery of masked visual target and its theoretical interpretation. *Bulletin of the Psychonomic Society* **11**, 285–287.

Diamond, I.T., Conley, M., Itoh, K. & Fitzpatrick, D. (1985). Laminar organization of geniculocortical projections in *Galago Senegalensis* and *Aotus Trivirgatus*. *Journal of Comparative Neurology* **242**, 584–610.

Dreher, B., Fukuda, Y. & Rodieck, R.W. (1976). Identification, classification and anatomical segregation of cells with X-like and Y-like properties in the lateral geniculate nucleus of old-world primates. *Journal of Physiology* **258**, 433–452.

Edwards, V.T., Hogben, J.H., Clark, C.D. & Pratt, C. (1996). Effects of a red background on magnocellular functioning in average and specifically disabled readers. *Vision Research* **36**, 1037–1045.

Fehrer, E. & Raab, D. (1962). Reaction time to stimuli masked by metacontrast. *Journal of Experimental Psychology* **63**, 143–147.

Fitzpatrick, D., Lund, J.S. & Blasdel, G.G. (1985). Intrinsic connections of macaque striate cortex: Afferent and efferent connections of lamina 4C. *Journal of Neuroscience* **5**, 3329–3349.

Francis, G. (1997). Cortical dynamics of lateral inhibition: Metacontrast masking. *Psychological Review* **104**, 572–594.

Grossberg, S. & Mingolla, E. (1985). Neural dynamics of form perception: Boundary completion, illusory figures, and neon color spreading. *Psychological Review* **92**, 173–211.

Hendrickson, A.E., Wilson, J.R. & Ogren, M.P. (1978). The neuroanatomical organization of pathways between the dorsal lateral geniculate nucleus and visual cortex in old world and new world primates. *Journal of Comparative Neurology* **182**, 123–136.

Hubel, D.H. & Wiesel, T.N. (1972). Laminar and columnar distribution of geniculo-cortical fibres in the macaque monkey. *Journal of Comparative Neurology* **146**, 421–450.

Kahneman, D. (1967). An onset-onset law for one case of apparent motion and metacontrast. *Perception & Psychophysics* **2**, 577–584.

Kaplan, E. (2004). The M, P, and K, pathways of the primate visual system. In *Visual Neurosciences*, ed. Chalupa, L.M. & Werner, J.S., pp. 481–493. Cambridge, MA: 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 Science of the United States of America* **83**, 2755–2757.

Kosslyn, S.M., Chabris, C.E., Marsolek, C.J. & Koenig, O. (1992). Categorical versus coordinate spatial judgments: Computational analysis and computer simulation. *Journal of Experimental Psychology: Human Perception and Performance* **18**, 562–575.

Kristofferson, A.B., Galloway, J. & Hanson, R.G. (1979). Complete recovery of masked visual target. *Bulletin of the Psychonomic Society* **13**, 5–6.

Lamme, V.A.F., Supèr, H. & Spekreijse, H. (1998). Feedforward, horizontal, and feedback processing in the visual cortex. *Current Opinions in Neurobiology* **8**, 529–535.

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.

Livingstone, M.S. & Hubel, D.H. (1984). Anatomy and physiology of a color system in the primate visual cortex. *Journal of Neuroscience* **4**, 309–356.

Long, N.R. & Gribben, J.A. (1969). The recovery of a visually masked target. *Perception & Psychophysics* **10**, 197–200.

Lund, J.S. & Boothe, R.G. (1975). Interlaminar connections and pyramidal neuron organization in the visual cortex, area 17, of the macaque monkey. *Journal of Comparative Neurology* **159**, 305–334.

Lund, J.S., Lund, R.D., Hendrickson, A.E., Bunt, A.M. & Fuchs, A.F. (1975). The origin of efferent pathways from the primary visual cortex (area 17) of the macaque monkey as shown by retrograde transport of horseradish peroxidase. *Journal of Comparative Neurology* **164**, 287–304.

Marrocco, R.T., McClurkin, J.W. & Young, R.A. (1982). Spatial summation and conduction latency classification of cells of the lateral geniculate of macaques. *Journal of Neuroscience* **2**, 1275–1291.

Maunsell, J.H.R. & Gibson, J.R. (1992). Visual response latencies in striate cortex of the macaque monkey. *Journal of Neurophysiology* **68**, 1332–1344.

Nassi, J.J. & Callaway, E.M. (2006). Multiple circuits relaying primate parallel visual pathways to the middle temporal area. *Journal of Neuroscience* **26**, 12789–12798.

Nassi, J.J. & Callaway, E.M. (2007). Specialized circuits from primary visual cortex to V2 and MT. *Neuron* **55**, 799–808.

Öğmen, H. (1993). A neural theory of retino-cortical dynamics. *Neural Networks* **6**, 245–273.

Öğmen, H., Breitmeyer, B.G. & Bedell, H.E. (2006*a*). Dynamics of perceptual epochs probed by dissociation phenomena in masking. In *The First Half Second: The Microgenesis and Temporal Dynamics of Unconscious and Conscious Visual Processes*, ed. Öğmen, H. & Breitmeyer, B.G., pp. 149–169. Cambridge, MA: MIT Press.

Öğmen, H., Breitmeyer, B.G. & Melvin, R. (2003). The what and where in visual masking. *Vision Research* **43**, 1337–1350.

Öğmen, H., Breitmeyer, B.G., Todd, S. & Mardon, L. (2006*b*). Target recovery in metacontrast: The effect of contrast. *Vision Research* **46**, 4726–4734.

Purcell, D.G. & Stewart, A.L. (1975). Visual masking by a patterned stimulus and recovery of observer performance. *Bulletin of the Psychonomic Society* **6**, 457–460.

Purcell, D.G., Stewart, A.L. & Hochberg, E.P. (1982). Recovery and nonmonotone masking effects. *Vision Research* **22**, 1087–1091.

Purpura, K., Kaplan, E. & Shapley, R.M. (1988). Background light and the contrast gain of primate P and M retinal ganglion cells. *Proceedings of the National Academy of Science of the United States of America* **85**, 4534–4537.

Purpura, K., Tranchina, D., Kaplan, E. & Shapley, R.M. (1990). Light adaptation in the primate retina – Analysis of changes in gain and dynamics of monkey retinal ganglion-cells. *Visual Neuroscience* **4**, 75–93.

Purushothaman, G., Öğmen, H. & Bedell, H.E. (2000). Gamma-range oscillations in backward-masking functions and their putative neural correlates. *Psychological Review* **107**, 556–577.

Robinson, D.N. (1966). Disinhibition of visually masked stimuli. *Science* **154**, 157–158.

Robinson, D.N. (1968). Visual disinhibition with binocular and interocular presentations. *Journal of the Optical Society of America* **58**, 254–257.

Roelfsema, P.R., Lamme, V.A.F., Spekreijse, H. & Bosch, H. (2002). Figure-ground segregation in a recurrent network architecture. *Journal of Cognitive Neuroscience* **14**, 525–537.

Roth, E.C. & Hellige, J.B. (1998). Spatial processing and hemispheric asymmetry: Contributions of the transient/magnocellular visual system. *Journal of Cognitive Neuroscience* **10**, 472–484.

Sawatari, A. & Callaway, E.M. (1996). Convergence of magno- and parvocellular pathways in layer 4B of macaque primary visual cortex. *Nature* **380**, 442–446.

Schiller, P.H. & Greenfield, A. (1969). Visual masking and the recovery phenomenon. *Perception & Psychophysics* **6**, 182–184.

Schiller, P.H. & Smith, M.C. (1966). Detection in metacontrast. *Journal of Experimental Psychology* **71**, 32–39.

Schwartz, S.H. & Loop, M.S. (1982). Evidence for transient luminance and quasi-sustained color mechanisms. *Vision Research* **22**, 445–447.

Schwartz, S.H. & Loop, M.S. (1983). Difference in temporal appearance associated with activity in the chromatic and achromatic systems. *Perception & Psychophysics* **33**, 388–390.

Sherman, S.M. & Guillery, R.W. (1998). On the actions that one nerve cell can have on another: Distinguishing ''drivers'' from ''modulators.'' *Proceedings of the National Academy of Sciences of the United States of America* **95**, 7121–7126.

Shipp, S. & Zeki, S. (1989). The organization of connections between areas V5 and V1 in macaque monkey visual-cortex. *European Journal of Neuroscience* **1**, 309–332.

Skottun, B.C. (2004). On the use of red stimuli to isolate magnocellular responses in psychophysical experiments: A perspective. *Visual Neuroscience* **21**, 63–68.

Skottun, B.C. & Skoyles, J.R. (2007). Metacontrast, target recovery, and the magno- and parvocellular systems: A perspective. *Visual Neuroscience* **24**, 177–181.

Stoper, A.E. & Banffy, S. (1977). Relation of split apparent motion to metacontrast. *Journal of Experimental Psychology: Human Perception and Performance* **3**, 258–277.

Tenkink, E. & Werner, J.H. (1981). The intervals at which homogeneous flashes recover masked targets. *Perception & Psychophysics* **30**, 129–132.

Tigges, J., Tigges, M., Anschel, S., Cross, N.A., Letbetter, W.D. & McBride, R.L. (1981). Areal and laminar distribution of neurons interconnecting the central visual cortical area-17, area-18, area-19, and area-MT in squirrel-monkey (*Saimiri*). *Journal of Comparative Neurology* **202**, 539–560.

van Hooser, S.D., Heimel, J.A.F. & Nelson, S.B. (2003). Receptive field properties and laminar organization of lateral geniculate nucleus in the gray squirrel (Sciurus carolinensis). *Journal of Neurophysiology* **90**, 3398–3418.

Wang, Y., Xiao, Y. & Felleman, D.J. (2007). V2 thin stripes contain spatially organized representations of achromatic luminance change. *Cerebral Cortex* **17**, 116–129.

Weisstein, N. & Growney, R. (1969). Apparent movement and metacontrast: A note on Kahneman's formulation. *Perception & Psychophysics* **5**, 321–328.

Wiesel, T.N. & Hubel, D.H. (1966). Spatial and chromatic interactions in the lateral geniculate body of the rhesus monkey. *Journal of Neurophysiology* **29**, 1115–1156.

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-London* **533**, 519–535.

Xiao, Y., Wang, Y. & Felleman, D.J. (2003). A spatially organized representation of colour in macaque cortical area V2. *Nature* **421**, 535–539.

Yabuta, N.H. & Callaway, E.M. (1998). Functional streams and local connections of layer 4C neurons in primary visual cortex of the macaque monkey. *Journal of Neuroscience* **18**, 9489–9499.

Yabuta, N.H., Sawatari, A. & Callaway, E.M. (2001). Two functional channels from primary visual cortex to dorsal visual cortical areas. *Science* **292**, 297–300.

Yeh, T.Y., Lee, B.B., Kremers, J., Cowing, J.A., Hunt, D.M., Martin, P.R. & Troy, J.B. (1995). Visual responses in the lateral geniculate nucleus of dichromatic and trichromatic marmosets (Callithrix jacchus). *Journal of Neuroscience* **15**, 7892–7904.

Haluk Öğmen[1,2]
Gopathy Purushothaman[3,4]
Bruno G. Breitmeyer[2,5]
[1]Department of Electrical & Computer Engineering, University of Houston, Houston, Texas
[2]Center for Neuro-Engineering and Cognitive Science, University of Houston, Houston, Texas
[3]Department of Cell & Developmental Biology, Vanderbilt University, Nashville, Tennessee
[4]Vanderbilt Vision Research Center, Vanderbilt University, Nashville, Tennessee
[5]Department of Psychology, University of Houston, Houston, Texas