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Minireview

Contrast sensitivity and magnocellular functioning in schizophrenia

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Abstract

It has been suggested that schizophrenia is associated with a magnocellular deficit. This would predict a loss of contrast sensitivity at low spatial and/or at high temporal frequencies. We here review research that tested contrast sensitivity in individuals with schizophrenia. We find that the results of this research tend to show uniform reductions in contrast sensitivity that are generally not consistent with a magnocellular deficit. While much of this data may be consistent with an attentional deficiency on the part of the schizophrenic individuals, it is difficult to link such an attentional deficiency specifically to the magnocellular system. The conclusion of the present review is that contrast sensitivity data do not indicate the existence of an association between magnocellular deficits and schizophrenia.

Keywords: Contrast sensitivity; Magnocellular; Schizophrenia; Temporal frequency; Spatial frequency; Attention

1. Introduction

It has been suggested that schizophrenia is associated with a deficiency in the magnocellular part of the subcortical visual system (Butler et al., 2007; Keri, Antal, Szekeres, Benedek, & Janka, 2002; Laycock, Crewther, & Crewther, 2007; Schechter et al., 2006). The early part of the visual system in primates contains three parallel streams: the magnocellular, the parvocellular and the koniocellular systems (for reviews, see Hendry & Reid, 2000; Merigan & Maunsell, 1993; Shapley & Perry, 1986). The three streams can be differentiated from the retina, through the lateral geniculate nucleus (LGN) to the input layers of the primary visual cortex (V1). Inside the primary visual cortex there is considerable mixing of the inputs which makes it difficult to distinguish magno-, parvo- and koniocellular responses at cortical levels (Lachica, Beck, & Casagrande, 1992; Levitt, Yoshioka, & Lund, 1994; Martin, 1992; Merigan & Maunsell, 1993; Nassi, Lyon, & Callaway, 2006; Nealey & Maunsell, 1994; Sawatari & Callaway, 1996; Sincich & Horton, 2002; Sincich, Park, Wohlengemuth, & Horton, 2004; Vidyasagar, Kulikowski, Lipnicki, & Dreher 2002; see also DeYoe & Van Essen, 1988; Dobkins & Albright, 2003; Kiper, Levitt, & Gegenfurtner, 1999; Skottun & Skoyles, 2006c).

The most effective and reliable way to isolate magnocellular activity in psychophysical experiments is to measure contrast sensitivity (Skottun, 2000a). Studies in which lesions have been placed in various layers of monkey LGN have found that reductions in contrast sensitivity following lesions in the magnocellular layers are confined to cases in which the stimuli are of low spatial frequency and/or high temporal frequency (Merigan, Byrne, & Maunsell, 1991a; Merigan, Katz, & Maunsell, 1991b; Merigan & Maunsell, 1990, 1993; Schiller, Logothetis, & Charles, 1990a, 1990b). Psychophysical studies in humans are consistent with these findings (Legge, 1978; Tolhurst, 1975). Because the link between contrast sensitivity and magnocellular activity has been established by both lesion

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studies in monkeys and human psychophysics, it represents the most reliable and direct psychophysical test of magnocellular sensitivity.

It appears that the magnocellular system mediates sensitivity at spatial frequencies below about 1.5 c/deg and that the parvocellular system (or a combination of the parvocellular and koniocellular systems) carries out detection above this frequency (Skottun, 2000a). One would therefore expect a magnocellular deficit to manifest itself at spatial frequencies below 1.5 c/deg or, alternatively, to be most pronounced at low spatial frequencies. In the case of temporal contrast sensitivity, magnocellular deficits, would be expected to show themselves at predominantly high temporal frequencies.[1] Here, we review the studies that have determined contrast sensitivity in schizophrenic subjects in a sufficiently systematic manner to make it possible to determine how sensitivity varies with spatial and temporal frequencies.

2. Spatial contrast sensitivity

In the case of spatial contrast sensitivity, a magnocellular deficit would manifest itself as a reduction in sensitivity at low spatial frequencies, or, alternatively, as a deficit that is most pronounced at the lowest spatial frequencies. In Figs. 1 and 2, the data from the various spatial contrast sensitivity studies have been re-plotted into a standard Log–Log format to facilitate comparison between the different data sets.

The earliest spatial contrast sensitivity study involving schizophrenic subjects of which we know is the one by Slaghuis from 1998. In this study schizophrenic subjects were divided into two groups: those with positive-symptoms and those with negative- symptoms. (''Positive-symptoms'' are ''hallucinations, delusions, and disturbances in thought disorder'' and ''negative-symptoms... are characterized by absences of normal function such as cognitive impairment, anhedonia, paucity of content of speech, reduced motivation, flattening of affect, and deficits in social function''; Slaghuis, 1998, p. 49). The results of this study are shown in Fig. 1a. As can be seen, both schizophrenic groups have reduced contrast sensitivity relative to the controls. However, the reductions were far larger in the case of the negative-symptom group. The sensitivity reductions of both groups are found across all spatial frequencies suggesting a general loss of sensitivity. This general pattern is not the one that would be expected from a sensitivity loss caused by a deficiency in the magnocellular system.

The second study is that of Keri et al. (2002) which obtained data under two conditions: static (0 Hz) and dynamic (8 Hz). The two data sets are re-plotted in Figs. 1b and c. Under both conditions, the schizophrenic subjects showed reduced sensitivity. In the static condition (Fig. 1b), the deficit is rather more pronounced at frequencies above about 2 c/deg. This is at odds with a magnocellular deficit. In the case of dynamic stimuli (Fig. 1c), the sensitivity loss seems to be somewhat larger at the lowest two frequencies (i.e., 0.5 & 1.2 c/deg). This might be taken to indicate a magnocellular deficit. However, there was also a large deficit present at the highest spatial frequency (i.e., 14.4 c/deg). Since the main trend is roughly that of a general sensitivity reduction, it is difficult to interpret these data as evidence for a magnocellular deficit. [The re-plots of the data from Keri et al. (2002) appear somewhat different from the original plots since we used the frequency values specified by Keri et al. (2002) rather than their plots because it is not clear what kind of X-axes were used in them.]

The next data set is that of Slaghuis and Thompson (2003). The data from this study are re-plotted in Fig. 1d. As was the case in the study of Slaghuis (1998), the schizophrenic subjects were divided into positive- and negative-symptom groups. And again, as was the case in the earlier study (Slaghuis, 1998), the sensitivity reductions were roughly distributed in a uniform manner across all the spatial frequencies. Also, the sensitivity reductions were uniformly larger for the negative-symptom group. As was pointed out above, a general reduction in sensitivity is not what would be predicted from a magnocellular deficit. [The original figures of Slaghuis and Thompson (2003), and Slaghuis (2004), see below, were plotted using sensitivity values given in natural logarithms. For the sake of consistency we have re-plotted these data using logarithms with base 10.]

Slaghuis (2004) studied spatial contrast sensitivity at four different temporal modulation frequencies (0.0, 4, 8 and 12 Hz) with subjects divided into positive- and negative-symptom groups. The results are shown in Figs. 1e–h. At all four temporal frequencies, there were only small deficiencies in the case of the positive-symptom subjects. In the case of negative-symptom subjects there were substantial and uniform deficits afflicting all spatial frequencies. Again, these findings do not point to a magnocellular deficit. Nor is there any evidence when comparing the four panels of any tendency for the deficits to increase with temporal frequency as would be expected for a magnocellular deficiency.

In the data of Butler et al. (2005), which are re-plotted in Fig. 2, there is evidence for deficits at low and medium spatial frequencies, and for them being largest at the lowest frequencies. These findings are roughly consistent with what one might expect for a magnocellular deficit. However, Butler et al. (2005) found significant differences

[1] Dacey and Petersen (1992) have reported that dendritic fields in parasol ganglion cells in humans are larger than those in monkeys. By contrast, the dendritic fields for midget ganglion cells were found to be similar. (The parasol and midget cells are the retinal ganglion cells which provide the input to, respectively, the magno- and parvocellular LGN cells.) This, it has been suggested, might make the spatial frequency tuning of magnocellular neurons different in monkeys and humans. It should therefore be pointed out that the transition point of 1.5 c/deg is based mainly on human psychophysics (e.g., Legge, 1978; Tolhurst, 1975), and is thus unaffected by the observation of Dacey and Petersen (1992).

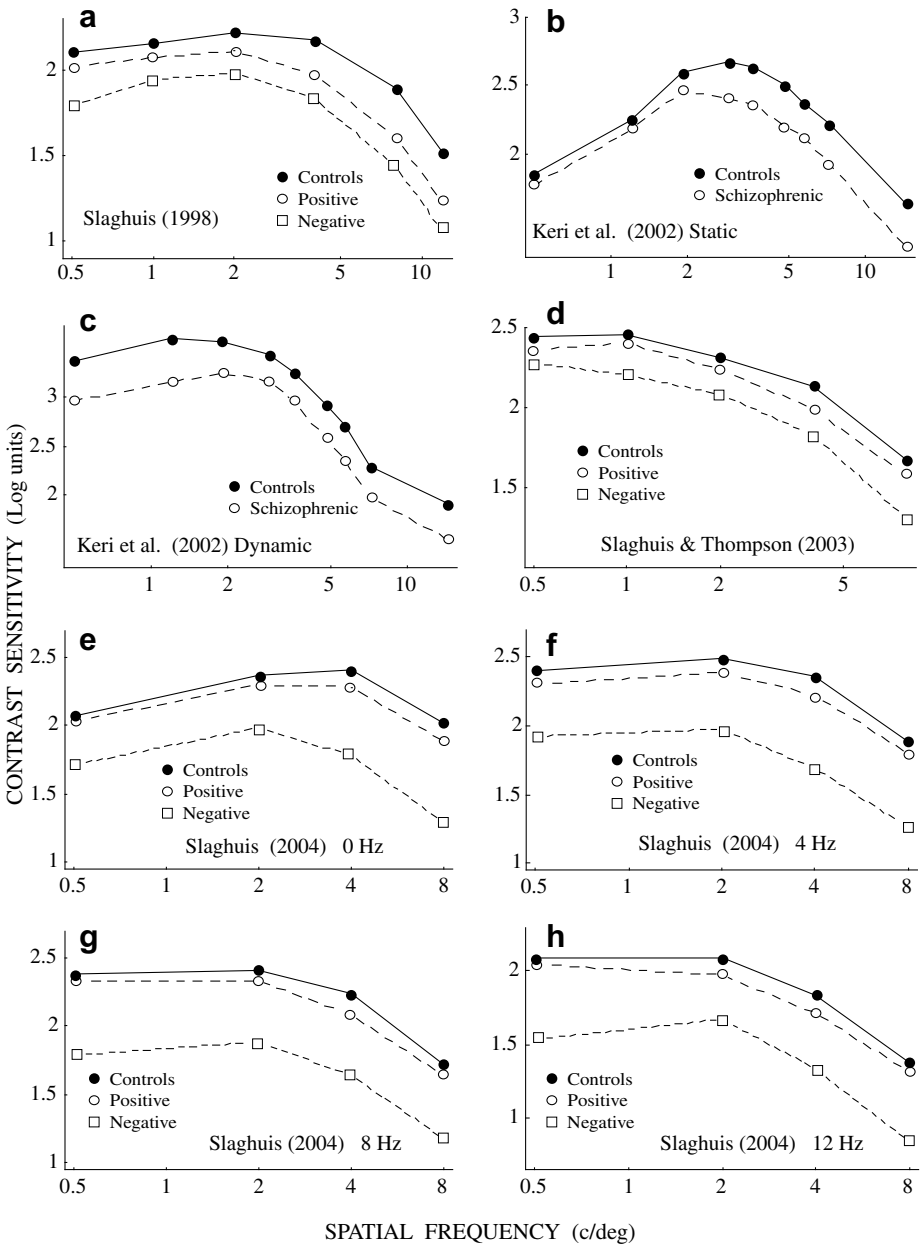

Fig. 1. Contrast sensitivity as a function of spatial frequency. (a) Data from Fig. 2 of Slaghuis (1998) obtained with stimuli subtending 4.03 deg × 3.36 deg at 17.0 cd/m^2. (b,c) Data re-plotted from, respectively, Figs. 3 and 4 of Keri et al. (2002). The stimuli subtended 13 deg × 13 deg and the luminance level was 20 cd/m^2. (d) Data re-plotted from Fig. 1a of Slaghuis and Thompson (2003). The data were obtained with a 5.0 Hz counterphase modulating grating (3.5 deg × 6.7 deg) with a blank surround. (e–h) Data re-plotted from Fig. 2 of Slaghuis (2004). (e–h) show data obtained with stimuli modulating at, respectively, 0, 4, 8, and 12 Hz. Stimulus dimensions were 7.12 deg × 5.71 deg and the luminance was 18.0 cd/m^2.

between the controls and schizophrenic subjects at spatial frequencies all the way up to 7.0 c/deg. The magnocellular system is not generally held to mediate sensitivity at this high a frequency. However, it is possible that the short stimulus presentations used in this study (32 ms), may have caused the magnocellular system to mediate threshold at frequencies as high as 7.0 c/deg. [Tolhurst (1975) showed that the parvocellular system, at the time called the "sustained system", mediates contrast detection by responding to sustained stimulation. It is therefore possible that a very brief stimulus, which has little sustained stimulation, may have favored the magnocellular system relative to the parvocellular system.]

The most recent spatial contrast sensitivity study is that of Revheim et al. (2006). In this study, schizophrenic patients were divided into reading impaired ($N = 9$) and non-reading impaired subjects ($N = 9$). Contrast sensitivity was tested at 0.5 c/deg, 7.0 c/deg and 21 c/deg. Revheim et al. (2006) found deficits on the part of the reading impaired group but not on the part of the non-impaired group at 0.5 c/deg. There were no deficits at 7.0 and 21.0 c/deg for either group. It is not clear that the relevant

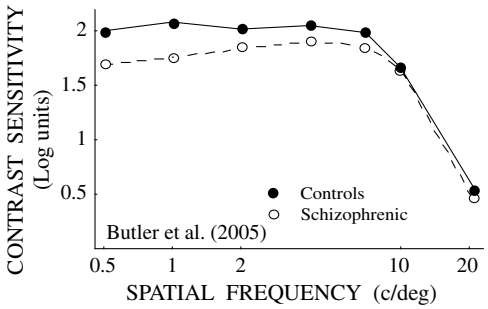

Fig. 2. Spatial contrast sensitivity data re-plotted from Fig. 4 of Butler et al. (2005). The stimuli in this study subtended 5.7 deg × 5.7 deg and the luminance was 100 cd/m^2.

factor is not reading impairment rather than schizophrenia. The findings of Revheim et al. (2006) do not indicate a magnocellular deficit as a general characteristic of schizophrenia.

3. Temporal contrast sensitivity

When testing temporal contrast sensitivity the existence of a magnocellular deficit would be expected to manifest itself at high temporal frequencies, or result in a deficit which is largest at the highest temporal frequencies. Data re-plotted from the various studies are shown in Fig. 3.

The first study is that of Schwartz, McGinn, and Winstead (1987). The results from Fig. 1 of that study are re-plotted in Fig. 3a. As can be seen, the sensitivities for the schizophrenic subjects are lower than those of the controls. The authors reported that the difference between schizophrenic subjects and controls are statistically significant at 3.25 and 6.5 Hz. Furthermore, and most importantly, there is little difference between the two groups at 26 Hz, a temporal frequency at which a magnocellular deficit would be expected to show itself. These results, therefore, do not provide support for a magnocellular deficit. [Schwartz et al. (1987) also studied spatial contrast sensitivity but they did not describe the data in sufficient detail to allow comparisons with predictions based on magnocellular deficits.]

Slaghuis (1998) studied contrast sensitivity as a function of temporal frequency using gratings of two spatial frequencies 1.0 c/deg and 8.0 c/deg. The results are re-plotted in Figs. 3b and c. As in the case of the spatial studies (see above), Slaghuis divided the subjects into positive- and negative-symptom groups. For the 1.0 c/deg stimuli (Fig. 3b), the data for the positive-symptom group were practically identical to those of the control group. The negative-symptom group showed approximately equal reductions in sensitivity at all temporal frequencies. If anything, the deficits seem to have been largest at the lowest frequency. In the case of 8.0 c/deg stimuli (Fig. 3c), both schizophrenic groups demonstrated reductions of a uniform nature at all temporal frequencies. The reductions in sensitivity shown by the negative-symptom group were about twice as large (in the Log–Log plot) as those of the positive-symptom group. In neither of these two sets of data (i.e., Figs. 3b and c) is there any evidence of a magnocellular deficit. On the contrary, if anything, it would seem that the largest deficits in both plots (Figs. 3b and c) are at the lowest temporal frequencies, which is the opposite of what would be expected for a magnocellular deficit.

It should also be pointed out that in the temporal sensitivity data of Slaghuis (1998), the deficits in the data obtained with 8 c/deg stimuli (i.e., Fig. 3c) are larger than in the data obtained with 1.0 c/deg stimuli (i.e., Fig. 3b). This is the opposite of what would be predicted from a magnocellular deficit.

Slaghuis and Bishop (2001) determined temporal contrast sensitivity at three different luminance levels: 3.0, 33.0, and 66 cd/m^2. The data obtained under these three conditions are re-plotted in Figs. 3d–f. The original data of Slaghuis and Bishop were plotted using a linear sensitivity axis. We have re-plotted the data with Log–Log axes. This makes the plots look slightly different. As in the other studies of Slaghuis and Slaghuis and Thompson, the schizophrenic subjects were divided into negative- and positive-symptom groups. In Figs. 3d–f, we see that data for the positive-symptom group is only moderately depressed relative to those of the control group. Slaghuis and Bishop (2001) did not find any statistically significant difference between the control group and the positive-symptom group. The negative-symptom group showed larger reductions in sensitivity. These reductions take the form of a roughly uniformly lowered sensitivity that tends to increase with luminance. Slaghuis and Bishop (2001) found statistically significant differences between the controls and the negative-symptom group at 4.0 and 8.0 Hz but not at 1.0, 16.0 and 32.0 Hz. These findings are not indicative of magnocellular deficits.

Chen et al. (2003) studied temporal contrast sensitivity in two groups of medicated schizophrenic patients: Those who received typical antipsychotic medication and those who received atypical antipsychotic medication. The data are re-plotted in Fig. 3g. As can be seen from the plot, both groups of schizophrenic subjects show reduced sensitivity at low and/or medium temporal frequencies (including the static condition). There are virtually no deficits at the highest frequencies (in fact, the atypical medication group show slightly elevated sensitivity at the highest temporal frequencies). These findings are roughly the opposite of what would be found if there were a magnocellular deficit.

The final study of temporal contrast sensitivity is that of O'Donnell et al. (2006). These authors tested medicated and unmedicated schizophrenic subjects. In this study temporal modulation was generated by drifting the stimuli at various speeds (with the drift speed given in terms of modulation frequency). The study also included a static condition. The results are re-plotted in Fig. 3h. As we can see, there is a general reduction in sensitivity on the part of both schizophrenic subject groups. Again, this does not indicate the existence of a magnocellular deficit. In the case

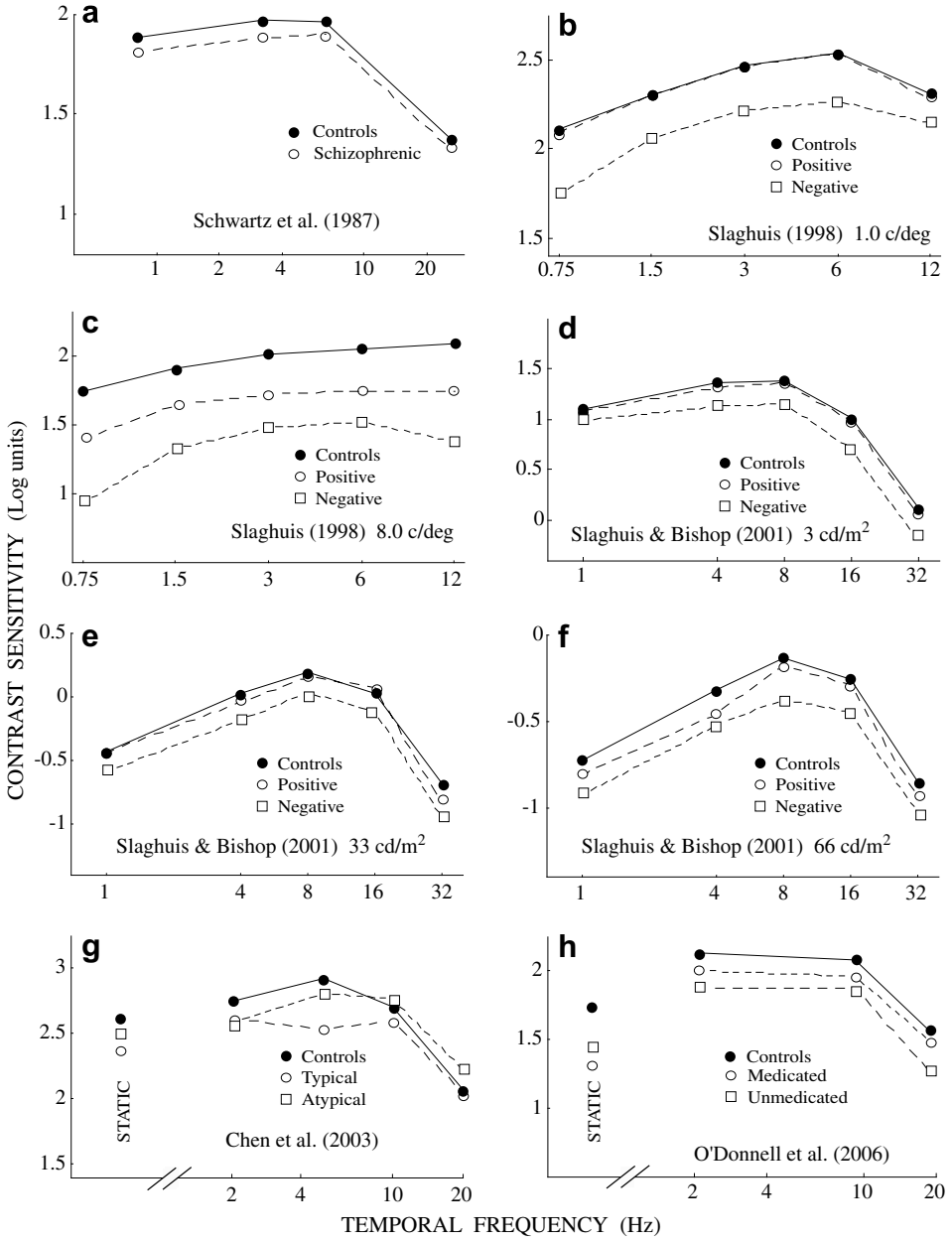

Fig. 3. Contrast sensitivity as a function of temporal frequency. (a) Data re-plotted from Fig. 1 of Schwartz et al. (1987). The stimulus conditions in this study were not specified. (b,c) are re-plotted from respectively panels a and b in Fig. 3 of Slaghuis (1998). (b,c) show data obtained with, respectively, 1.0 c/deg and 8.0 c/deg gratings drifted at 0.75, 1.5, 3.0, 6.0 and 12.0 Hz. Gratings subtended 4.03 deg × 3.36 deg and had a luminance of 17.0 cd/m^2. (d–f) Data re-plotted from Fig. 2 of Slaghuis and Bishop (2001). The three panels give data obtained at 3, 33 and 66 cd/m^2, respectively. The stimuli were Gaussian patches of 4 deg diameter. (g) Data re-plotted from Fig. 3 of Chen et al. (2003). The stimuli were masked down to a 10 deg diameter circular window. These data were obtained with gratings with a spatial frequency of 0.5 c/deg. (h) Data plotted from Table 2 of O'Donnell et al. (2006). Results are shown for unmedicated and medicated schizophrenic subjects as well as controls. The three data points on the left hand side are for static presentations. The spatial frequencies used were 9.9 c/deg for the static test and 1.3 c/deg for the tests using temporally modulated (i.e., moving) stimuli.

of moving stimuli, the sensitivity loss is somewhat larger for the unmedicated schizophrenic subjects than for the medicated subjects.

It should be noted that O'Donnell et al. (2006) used different spatial frequencies in the static conditions (9.9 c/deg) and the drifting conditions (1.3 c/deg). This was done in order to bias the stimuli, respectively, for the parvocellular and magnocellular systems. However, as can seen by comparing the static data to the rest of the data in Fig. 3h, this manipulation did not affect the relative contrast sensitivity of the schizophrenic subjects to a noticeable degree.

4. Other studies

A few studies only determined contrast sensitivity under a single condition. In these cases, the lack of an opportu-

nity to compare performances at different spatial and/or temporal frequencies makes it impossible to know whether or not there is a magnocellular deficiency. For instance, without an opportunity to compare data obtained under different stimulus conditions, a reduction in sensitivity could reflect generally reduced sensitivity, or be the sign of a sensitivity loss which is more or less specific to the particular condition tested. This applies to the work of Chen, Levy, Sheremata, and Holzman (2004) and Cimmer et al. (2006). Chen et al. (2004) used what seems to have been a 0.5 c/deg stimulus modulated at 5 Hz. Cimmer et al. (2006) studied contrast sensitivity to 0.5 c/deg counterphase modulated at a temporal frequency of 4 Hz [Cimmer et al. (2006) give the temporal frequency as 8 Hz but that refers to the reversal rate which is twice that of the temporal modulation frequency since there are two reversals per modulation cycle]. Both of these studies found to various degrees reduced sensitivity on the part of the schizophrenic subjects but since only one condition was tested it is not possible to form any opinion as to the nature of the deficiency.

Another study which involved only one stimulus condition is that of Keri, Antal, Szekeres, Benedek, and Janka (2000). These authors determined contrast sensitivity using 0.5 c/deg gratings modulated at 8.0 Hz and found no statistically significant difference between schizophrenic subjects and controls. Also, Keri, Kelemen, Benedek, and Janka (2004) did not find significantly elevated contrast thresholds to 24 min arc diameter dots. The findings of Keri et al. (2000) are inconsistent with a magnocellular deficit (as well as a general sensitivity reduction) because had there been a magnocellular deficit it would have had to manifest itself at least at 0.5 c/deg and 8.0 Hz. Also the results of Keri et al. (2004) appear to be inconsistent with a magnocellular deficit.

Gutherie, McDowell, and Hammond (2006) measured detection thresholds for three spots presented under scotopic conditions at 10 degrees eccentricity in the visual field. They did not find a deficiency on the part of the schizophrenic subjects. The authors concluded "that magnocellular deficits in schizophrenia may not be due to problems at the level of the rods but are more likely to occur later in the visual pathway" (Gutherie et al., 2006, p. 378). An alternative, and more parsimonious interpretation, would be that there simply is no magnocellular deficit. Irrespective of interpretation, the results of Gutherie et al. (2006) do not provide positive support for a magnocellular deficit.

In a recent study DeLord et al. (2006) used a somewhat different approach. Although this research does not strictly concern contrast sensitivity, it is closely related to it. In this study subjects were presented with four squares one out of which differed in luminance from the others. The task of the subject was to identify the differing square. This was done under two conditions, one supposedly biased for magnocellular detection ("steady paradigm"), and one biased for parvocellular detection ("pulsed paradigm") (the study also included a third condition unrelated to the magno-/parvocellular distinction). The schizophrenic subjects showed elevated thresholds under both conditions. The authors interpreted this finding as arguing against an early magnocellular dysfunction in schizophrenia.

An earlier study which is also related to contrast sensitivity is that of Black, Franklin, de Silva, and Wijewickrama (1975). In this study it was found that critical flicker fusion (CFF) was moderately reduced in schizophrenic individuals. These results have since been interpreted as potential evidence for a magnocellular deficit (Gutherie et al., 2006). In connection with that interpretation, it was pointed out that reduced CFF may reflect a cortical deficiency since CFF is held to reflect cortical processing (Gutherie et al., 2006). We would like to further note that reduced CFF would also be consistent with a general reduction in sensitivity. It should also be kept in mind that the reduction in CFF was quite small.

5. Discussion

The general conclusion of the present review is that the studies of contrast sensitivity in individuals with schizophrenia provide little evidence for a magnocellular deficit. The exact number of different studies is difficult to judge since in the case of the work of Slaghuis (1998, 2004), Slaghuis and Bishop (2001), Slaghuis and Thompson (2003) the degree to which different data sets are independent is unclear (they may or may not represent different groups of subjects). However, the fact that only one study has yielded data consistent with a magnocellular deficit (i.e., Butler et al., 2005) means that even if the four studies of Slaghuis (1998, 2004), Slaghuis and Bishop (2001) and Slaghuis and Thompson (2003) were counted as one, the majority of studies would not support the presence of a magnocellular deficit.

The only study to find deficits consistent with a magnocellular deficit (Butler et al., 2005) was carried out at a relatively high luminance level (100 cd/m^2) compared to many of the other studies (typically 15–20 cd/m^2). This may suggest that magnocellular deficits only manifest themselves at high luminance levels. In regard to this we make two comments: (1) The magnocellular system is more closely associated with low luminance levels rather than with high ones (Purpura, Kaplan, & Shapley, 1988). And, (2) in the temporal contrast sensitivity study of Slaghuis and Bishop (2001), data were obtained at several different luminance levels (3, 33 and 66 cd/m^2). However, in these data (Figs. 3d–f), while there is evidence for the deficits (of the negative-symptom group) to become larger with increasing luminance, there is no evidence that such increases in luminance produce effects resembling those of a magnocellular deficit. Indeed, in the case of the highest luminance level used in this study, 66 cd/m^2, the deficit is that of a uniform reduction in sensitivity. Since, the luminance level in this study is only 0.18 Log units below that of Butler et al. (2005), it seems unlikely that the difference between these

two studies reflects the difference in their stimulus luminance.

The overall trend in the data sets is that schizophrenic subjects show general reduction in sensitivity. As pointed out elsewhere (Skottun & Skoyles, 2007c), such a reduction would be consistent with an attentional deficiency. Moreover, an attentional deficit may be unrelated to vision as similar problems can arise from impaired prefrontal cortex functioning. Alternatively, it has been suggested that visual attentional deficiencies in schizophrenia might be the result of a magnocellular deficit (Laycock et al., 2007). But as we have noted previously, it is difficult to link attention specifically to the magnocellular system (Skottun & Skoyles, 2006a, 2006b, 2007a, 2007c, 2007f). For instance, it has been found that covert visual attention may be directed by stimuli that do not activate the magnocellular system (Cole, Kentridge, & Heywood, 2005; Snowden, 2002; Sumner, Adamjee, & Mollon, 2002). Also, within the context of research upon dyslexia, it has been found that attention deficits can occur without the existence of magnocellular deficits (Roach & Hogben, 2004). Moreover, it would also conflict with the findings of larger sensitivity losses observed at higher spatial frequencies (compare results obtained at 1 c/deg, Fig. 3b, and data obtained at 8 c/deg, Fig. 3c), *and* the finding of Keri et al. (2000) of no contrast sensitivity loss for 0.5 c/deg gratings modulated at 8 Hz, as well as the findings of Schwartz et al. (1987), and those of Chen et al. (2003), of reduced sensitivity at low and medium temporal frequencies. For these and other reasons (see Skottun & Skoyles, 2006a, 2006b, 2007a, 2007c, 2007f) to interpret an attentional deficiency as evidence for a magnocellular deficit on present evidence is speculative.

The possible presence of attentional deficits makes it extremely important to include control conditions in studies that aim to assess magnocellular sensitivity. Moreover, this does not only apply to studies of contrast sensitivity but to all tests of all visual functions in schizophrenia. For instance, Schechter et al. (2006) found reduced stereo acuity in schizophrenic subjects. It is not clear to what extent those reductions are the results of reduced attention. (In the case of attributing stereo acuity to magnocellular activity, there is also the additional problem that larger deficits in stereo vision have been found to occur following parvocellular lesions than after magnocellular lesions. This suggests that stereo vision is more closely linked to the parvocellular system than to the magnocellular system, Schiller et al., 1990a). Another concern is that some studies have made use of staircase methods. There is evidence to indicate that such methods may be particularly vulnerable to lapses of visual attention (Stuart, McAnally, & Castles 2001). If schizophrenia is associated with reduced attention, as has been suggested by e.g., Laycock et al. (2007), this would substantially complicate the study of visual function in schizophrenia with regard to possible magnocellular and other visual deficiencies.

An alternative possibility is that since all studies contained schizophrenic subjects who were medicated to some degree the general reductions in contrast sensitivity could be the results of medication. For instance, Chen et al. (2003) have suggested that reduced contrast sensitivity in schizophrenic individuals may reflect antipsychotic medication. However, [Butler et al. (2005, p. 500) found "[n]o significant correlation... between contrast sensitivity at 0.5 cycles per degree... and chlorpromazine equivalents". [Butler et al. (2003, 2005) reached a similar conclusion with regard to backward masking.] O'Donnell et al. (2006) moreover found larger contrast sensitivity loss (for moving stimuli) for unmedicated schizophrenic subjects than for those who received medication (see Fig. 3h). This does not support the notion that reduced sensitivity is the result of medication. These observations, however, are to some extent inconsistent with the observation of Chen et al. (2003) who reported higher contrast detection thresholds to be associated with typical antipsychotic drugs but not with atypical antipsychotic drugs. However, these observations are themselves inconsistent with the work of Butler et al. (2005, p. 502) who observed visual deficits also in patients receiving atypical antipsychotic medication.

The antipsychotic drugs used in treating schizophrenia affect the dopaminergic system predominantly by acting on the D2 receptors (Seeman, 2002). Bodis-Wollner and Tzelepi (1998) found evidence that blocking D2 receptors in the retina mainly reduces responses to medium and high spatial frequency stimuli (above about 2 c/deg). With the exception of the study of Keri et al. (2002, static stimuli), our review uncovered little evidence for such deficits.

It cannot be ruled out that medication may have masked magnocellular deficits, for instance, by reducing sensitivity at medium and high frequencies (Bodis-Wollner, 1990; Bodis-Wollner & Tzelepi, 1998). If there had been a magnocellular deficit (which would have reduced sensitivity at low frequencies), the combined effect of antipsychotics (reducing sensitivity at medium and high frequencies), and a magnocellular deficit could have been the appearance of a general sensitivity reduction. However, this does not seem to be likely since it would have required that the medication induced sensitivity loss closely matched the loss of magnocellular sensitivity. Further, it would not explain the absence of contrast sensitivity deficits in many studies. However, it is interesting in this connection to note that O'Donnell et al. (2006) found unmedicated patients to have larger sensitivity reductions than medicated patients to 1.3 c/deg stimuli (used in the moving condition) but that the medicated patients had lower sensitivity to 9.9 c/deg (used in the static condition). In this study it would seem that the reduced sensitivity to 9.9 c/deg could have reflected D2 mediated medication. (However, the finding that the unmedicated group showed the largest reductions in sensitivity to 1.3 c/deg, in the case of moving stimuli, indicates that reduced sensitivity under those conditions does not reflect medication.) Also the finding by Keri et al. (2002) of reduced sensitivity above 2 c/deg may be consistent with

a D2 receptor mediated medication effect. However, these are speculations and do not detract from the general conclusion that the studies of contrast sensitivity in schizophrenia provide little support for magnocellular deficits.

Keri et al. (2002) suggested that a magnocellular like deficit might be an artifact resulting from medication. However in this case the medication would have been expected to create sensitivity reductions with the characteristics of magnocellular deficits. The present review finds little support for their existence.

It has been suggested that the issue of medication may be addressed by studying visual function in unaffected siblings or other relatives of schizophrenic patients (Keri et al., 2004). Chen et al. (2003, p. 1797) found that "[t]he visual contrast detection threshold for the group of first-degree relatives of patients with schizophrenia... was not significantly different from the healthy subjects." This might suggest that reduced contrast sensitivity reflects medication. However, unaffiliated relatives are not appropriate controls when it comes to the issue of isolating the effects of medication because the relatives differ from the patients not only with regard to medication but also with regard to diagnosis (that is, of course, the reason the patients receive medication and the relatives do not). (O'Donnell et al., 2006, also studied sensitivity in individuals with schizotypal personality disorder and found these to have sensitivity very close to the controls.)

The overall impression of this overview of the issue of medication is that at present, it is difficult to make a direct link between medication and the reduced contrast sensitivity in schizophrenic subjects. This is obviously a topic that requires further study.

Although contrast sensitivity is the most direct and reliable psychophysical test of magnocellular sensitivity, it is not the only test of magnocellular activity that has been used in connection with schizophrenia. The notion that there may be a magnocellular deficit in schizophrenia, is often brought up in connection with backward masking. A number of studies have investigated backward masking in schizophrenic subjects (e.g., Schechter, Butler, Silipo, Zemon, & Javitt, 2003; Slaghuis, 2004). The results of these studies have frequently been interpreted in terms of magnocellular functioning (e.g., Green, Nuechterlein, & Mintz, 1994; Schechter et al., 2003). In the present context one aspect of these investigations seems particularly relevant: Several studies have found that schizophrenic subjects show enhanced masking (Green et al., 1994; Slaghuis, 2004). This has been interpreted as the result of an "overly active transient system" (Green et al., 1994, p. 950). In the contrast sensitivity data reviewed in the present survey the abnormalities shown by schizophrenic groups are practically all in the direction of reduced sensitivity (with the exception of one data point in Fig. 3e and two in Fig. 3g). It seems that reduced sensitivity would be more consistent with a reduction in activity than with elevated activity. Thus, there is a potential discrepancy between the contrast sensitivity data and the masking studies. [In the case of masking studies there is also the problem that their psychophysical effects span much longer time intervals than the latency difference between the magno- and parvocellular systems, Skottun, 2001; Skottun & Skoyles, 2007d.]

Another approach that has been used in attempts to assess magnocellular sensitivity is Visual Evoked Potentials (VEP). This approach was used by, e.g., Butler et al. (2001). In that study two stimulus manipulations were used to separate magno- and parvocellular activity: spatial frequency and color. To use spatial frequency in suprathreshold stimuli to differentiate magno- and parvocellular responses is problematic since when eccentricity is controlled for, the spatial resolution of magno- and parvocellular neurons is very similar (Blakemore & Vital-Durand, 1986; see their Figs. 6A & 7). Therefore, although spatial frequency may be used to separate magno- and parvocellular activity at contrast threshold (i.e., in contrast sensitivity data), it may be inappropriate to rely on spatial frequency for separating the two systems using stimuli at contrasts above threshold. With regard to color, the problem is that the VEPs are recorded from the scalp above the visual cortex. It is evident that color processing continues at cortical levels (see, e.g., De Valois & De Valois, 1993). Thus, differences between color and luminance in VEP responses may reflect cortical mechanisms rather than the subcortical parvocellular and magnocellular system. There is also the problem that the parvocellular neurons respond to both color and luminance stimuli (Skottun & Skoyles, 2007e). Thus, color and luminance should not be treated as synonymous, respectively, with the parvo- and magnocellular systems.

With regard to VEP responses, in general, it is difficult to link these specifically to the subcortical magno- and parvocellular systems (see, e.g., Skottun & Skoyles, 2004). This is illustrated in the study of Butler et al. (2007) who found abnormal C1, P1 and N1 amplitudes when using low spatial frequency stimuli (see their Fig. 6). According to these authors, the C1 amplitude "is driven more strongly by parvocellular than magnocellular input" (Butler et al., 2007, p. 418); the P1 amplitude "appears to have dual underlying generators, including a dorsal generator within dorsolateral extrastriate cortex (e.g., V3a) and a ventral source within ventrolateral extrastriate cortex (e.g., V4)... The dorsal generator is driven predominantly by magnocellular input and the ventral generator by parvocellular input..." (Butler et al., 2007, p. 418); and the N1 amplitude "appears to reflect primarily ventral stream sources" (Butler et al., 2007, p. 419). Given that Butler et al. (2007) associate the ventral cortical stream with parvocellular activity, it therefore appears that two of the three abnormal amplitudes, according to their reasoning, are associated predominantly with the parvocellular system, and that the third amplitude is associated with a combination of magno- and the parvocellular inputs. It seems difficult to draw conclusions regarding magnocellular deficiencies on the basis of these responses.

Keri et al. (2004), Keri, Kelemen, Janka, and Benedek (2005) used vernier acuity to assess magnocellular sensitivity. This was based on the observation of Lee, Wehrhahn, Westheimer, and Kremers (1995) who found that "[a]t contrast of 20% and below, only the MC-pathway [i.e., magnocellular system] would appear capable of supporting vernier performance with our stimuli" (Lee et al., 1995, p. 2743). The qualification "with our stimuli" is of importance in this context since vernier acuity varies considerably with stimulus parameters (Bradley & Skottun, 1987). There appears to be at least one significant difference between the stimuli of Keri et al. (2004, 2005) and those of Lee et al. (2005). In the study of Lee et al. (1995), the stimuli were presented eccentrically (at 6.5 deg) whereas Keri et al. (2004, 2005) (as far as we can make out) used stimuli that were centrally fixated. Therefore, it is not clear that the conclusion of Lee et al. (1995) applies to the stimulus conditions of Keri et al. (2004, 2005). Furthermore, estimates have indicated that also cortical simple cells have spatial resolution consistent with vernier thresholds (Skottun, 2000b). Thus, vernier acuity deficits could reflect cortical dysfunction. In fact, it would seem that a vernier acuity deficit could indeed be the result of a deficiency at a number of levels in the visual pathway. This illustrates the advantage of using contrast sensitivity to test for magnocellular deficits. Not only does this task differentiate magnocellular from parvocellular deficiencies but it can also differentiate cortical abnormalities from subcortical ones, at least from deficiencies in the dorsal stream: Rudolph and Pasternak (1999, see their Fig. 4) placed lesions in areas MT and MST of the dorsal cortical stream and found that this resulted in substantial loss of motion perception and signal-to-noise detection but only in minor, and largely temporary, deficiencies in contrast sensitivity.

Based on the assumption that dyslexia is associated with magnocellular deficits Revheim et al. (2006) studied reading performance in schizophrenic subjects. In accordance with this assumption, the schizophrenic group showed poorer reading performance than the controls. This might suggest support for a magnocellular deficit in the schizophrenic subjects. The problem in this connection is that the evidence for magnocellular deficits in dyslexia is weak (Skottun, 2000a; Skottun & Skoyles, 2005, 2007b).

Recently Selemon and Begovic (2007) examined cell number and volume in the LGN in post mortem brains of schizophrenic individuals and found that both of these measures were normal in both the magno- and parvocellular layers. These findings undermine the hypothesis of a link between magnocellular deficits and schizophrenia. It should also be pointed out that the functional significance of a magnocellular deficit in schizophrenia has not been made clear in terms of its relevance to the clinical symptomatology of this condition.

In conclusion, the present review has revealed little evidence for a specifically magnocellular deficit in individuals with schizophrenia. The general trend is for schizophrenic individuals to show uniform reductions in contrast sensitivity. This could be consistent with attentional problems or the effects of medication. In the former case (at least), it would mean that particular care needs to be exercised when visual functions are investigated in those with schizophrenia.

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