Acta Neuropsychiatrica

Acta Neuropsychiatrica 2012: 24: 50–55

DOI: 10.1111/j.1601-5215.2011.00611.x

Visually evoked potentials, NMDA receptors and the magnocellular system in schizophrenia

Skottun BC, Skoyles JR. Visually evoked potentials, NMDA receptors and the magnocellular system in schizophrenia.

Background: It has been claimed that schizophrenia can be linked to the magnocellular system by way of *N*-methyl-D-aspartate (NMDA) receptors. The present report examines this claim.
Methods: A review is made of relevant research literature.
Results: The NMDA studies that have been referenced to connect visual deficits in schizophrenia to the magnocellular system are based on the cat, a species whose visual system is fundamentally different from that of primates. The cat visual system cannot easily be divided into magno- and a parvocellular portions.
Conclusions: Owing to the substantial differences between the visual systems of cats and primates, it is difficult to link sensory abnormalities in schizophrenia specifically to the magnocellular system based on data from the cat.

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Keywords: cat; lateral geniculate nucleus; monkey; motion; parvocellular; vision

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Accepted for publication July 30, 2011

Significant outcomes

- It has been proposed that NMDA receptor abnormalities in schizophrenia are linked specifically to the magnocellular system but all the data cited in support of this are all from the cat.
- The cat visual system is fundamentally different from that of primates making it difficult to use the cat's visual system as a model for humans.
- It is also difficult to draw conclusions about the magnocellular system based on motion perception.

Limitations

- The cat visual system could still be similar to the human magnocellular system in spite of its differences from the monkey magnocellular system, although no data exists to support this.
- It is possible that the magnocellular system in humans has a closer link to motion perception than that found in the monkey.

Introduction

The primate visual system consists initially of three parallel systems: the magnocellular, the parvocellular and the koniocellular (1,2). It has been proposed that schizophrenic individuals have an impairment in their magnocellular system (3–5). This suggestion is not yet established because of lack of support from tests of magnocellular functioning. The most direct psychophysical test of magnocellular integrity – contrast sensitivity (6) – has provided little evidence for magnocellular deficits in connection with schizophrenia (7,8). Studies of backward masking – a task

which by many is taken to depend on magnocellular activity (3) – have also failed to offer support for magnocellular deficiencies (9,10). In addition, a magnocellular deficit would be expected to produce anatomical abnormalities in the lateral geniculate nucleus (LGN). However, two studies (11,12) have not observed such abnormalities in post-mortem brains of schizophrenic subjects.

Steady-state VEPs

It has been claimed or implied that *N*-methyl-D-aspartate (NMDA) receptors provide a link between perceptual deficiencies in schizophrenia and the magnocellular system. For instance, Butler et al. (13) (see figure legend to their Fig. 3) discussed reduced steady-state visually evoked potential (ssVEP) responses in schizophrenic subjects in terms of magnocellular responses and blocking of NMDA receptors and wrote that '... NMDA dysfunction seems to be linked to gain control in the M-pathway ...' (13, p. 44). More recently, Javitt (14) suggested that the deficits in ssVEPs of schizophrenic individuals are linked with the magnocellular system through involvement of NMDA receptors: 'Deficits in magnocellular ssVEP generation similar to those observed in schizophrenia are seen in animal models following local infusion of NMDA antagonists into LGN or V1' (14, p. 261). To support this claim, reference was made to Fig. 5 of that article (i.e. to Fig. 5 in the work of Javitt (14)). The data are shown in two panels in this figure, one of which gives responses from cat LGN neurons, with and without infusion of the NMDA antagonist, whilst the other panel displays ssVEP data (adapted from the study of Butler et al. (4)) from schizophrenic subjects along with data from controls. (According to Javitt (14), Fig. 5b of this article is taken from Kwon et al. (15) However, the article of Kwon et al. (15) does not contain a figure of this form. It seems that the source of this figure may have been from Kwon et al. (16). Irrespective of this issue, both studies of Kwon et al. (15,16) deal with data from the cat brain). The argument is that the VEP data from the schizophrenic subjects, relative to those of controls, are similar to the LGN responses in the cat following infusion of an NMDA antagonist, relative to the data obtained without such infusion.

A similar attempt at linking NMDA receptors to the magnocellular system was made more recently by Kantrowitz and Javitt (17), who sought to set up this link by way of nonlinear responses: 'The magnocellular system, in particular, functions in a nonlinear gain mode that is dependent upon NMDAR-mediated (i.e. NMDA-receptor-mediated) neurotransmission' (17, p. 117). Attempts to identify magnocellular responses based on nonlinearity faces two issues: (a) in terms of nonlinearities, in general, there is considerable overlap between magno- and parvocellular neurons (as determined by the responses to the second harmonic; see Table 3 of the study of Levitt et al. (18)) and (b) with regard to nonlinear contrast response functions (i.e. the degree to which the functions saturate) there exist neurons, other than the magno- and parvocellular cells, with contrast response functions similar to those of these two cell types (19). Moreover, as with Butler et al. (4,13) and Javitt (14), the argument of Kantrowitz and Javitt (17) is made based on data from the cat: 'Administration of NMDAR antagonists to cat LGN produces a characteristic reduction in gain that is also observed in schizophrenia' (17, p. 117).

Cat vision and the magnocellular system

All the reports that have been invoked in attempts to link the magnocellular system in schizophrenic subjects to NMDA receptors have done so based on recordings made in the cat. But, this linkage would only be valid if cats had a magno–parvocellular division, or if the cat visual system were in other respects an appropriate model for the primate magnocellular system.

The early cat visual system, however, is profoundly different from that of primates (20,21). Anatomically, the magno–parvocellular distinction is most evident in the LGN. In the primate, this nucleus can be divided into four (or two depending on how the structure is sectioned) parvocellular layers and two magnocellular layers. In contrast, the cat's LGN consists of three layers. This raises the question how these three layers in the cat can be compared to the layers in the primate LGN. The issues will be briefly reviewed.

Kaplan and Shapley (22) hypothesised that the magnocellular layers in the monkey LGN may be homologous to the A and A1 layers in the cat. This, however, leaves the cat C layer unaccounted for. The C layer is unlike the parvocellular system as the parvocellular system receives inputs from both the contra- and ipsilateral eyes, whereas the C layer in the cat receives only contralateral (i.e. crossed) inputs (20,23).

Also, in terms of functional properties there are difficulties in linking neurons in the subcortical visual system in the cat to the magno- and parvocellular systems in primates. Cells in the early visual system of the cat can be divided into X- and Y-cells on the basis of physiological response properties (24). Dreher et al. (25) and Sherman et al. (26) have suggested that Y-cells in the cat are homologous with

https://doi.org/10.1111/j.1601-5215.2011.00611.x Published online by Cambridge University Press

magnocellular neurons in the primate. More recently, Crook et al. (27) have provided evidence to support this notion in the finding that parasol ganglion cells *in vitro* have the characteristics of Y-cells. As parasol cells are closely linked to the magnocellular system, this raises the possibility that Y-cells might serve as a model for magnocellular neurons. However, to consider Y-cells as homologous to magnocellular cells faces the problem that in the primate LGN most magnocellular neurons are X-cells and only a minority of the cells in the magnocellular layers are Y-cells (22,28–30).

In connection with X- and Y-cells it should also be noted that NMDA receptors in the cat are mainly linked to lagged X-cells. Thus, even if it had been the case that X- and Y-cells corresponded to, respectively, magno- and parvocellular neurons, there would not have been support from the cat for linking NMDA receptors to the magnocellular system. That lagged cells in general are linked to NMDA receptors whereas non-lagged cells are not is well established (31). Thus, the fact that, in the monkey, lagged cells are found in the parvocellular layers (as well as in the magnocellular layers) of the LGN is difficult to reconcile with the suggestions of Butler et al. (4), Javitt (14) and Kantrowitz and Javitt (17) that NMDA receptors are specifically linked to the magnocellular system.

It has been suggested that the whole of the early visual system in the cat is, moreover, homologous with the magnocellular system in the monkey, i.e. that the cat has a magnocellular system but no parvocellular system (20). This has been interpreted to mean that the cat visual system in its entirety can be used as a model for the magnocellular system.

In regard to this suggestion, it is important to consider the retinal input to the LGN. In primates the magno- and parvocellular neurons of the LGN are linked, respectively, to parasol and midget retinal ganglion cells. If the cat lacks the parvocellular system (at the level of the LGN) one would then expect it also to lack retinal midget cells. It has been proposed that the alpha cells in the cat retina are similar to the parasol cells in the monkey retina (32). However, to what extent the beta cells in the cat retina correspond to primate midget cells is less clear. Some similarities have been noted to exist (32) with, for instance, Leventhal et al. (33) suggesting that alpha and beta cells in the cat retina are similar to magno- and parvocellular projecting cells in the monkey. But, this raises difficulties as it leaves only two possibilities neither of which would entail a correspondence between cats and primates. (a) The beta cells are homologous with the midget cells. However, in this case the cat visual system would differ from the primate magnocellular system in that the supposed homologue to the magnocellular system would receive input from the midget cells. (b) Cat beta cells are not homologous with monkey midget cells. However, this would suggest that there are cells in the cat visual system, which do not have a homologue in the monkey. Again, this would indicate the existence of an important difference between the magnocellular system in the monkey and the cat visual system. Thus, in either case, important differences would exist between the cat visual system and the primate magnocellular system.

Shapley and Perry (20) provide a model in which both the cat and monkey LGN receive input from two classes of retinal ganglion cells (see their Figs 4 and 6). In their model, in both the cat and primate, one of the cell classes have large dendritic fields, and the other class has small fields. In the case of the monkey, the large and small cells project to, respectively, the magno- and parvocellular layers of the LGN. In the cat, according to the model, the two cells types, however, correspond to, respectively, Y- and X-cells. This in consequence makes these cells different from the cells in the monkey as the cells with large dendritic trees in the monkey (i.e. magnocellular neurons) comprise both X- and Y-cells, whereas in the cat they consist only of Y-cells. In addition, the cells with the small dendrites would also be different in the two animals. This is because in the monkey these cells project to the parvocellular system, whereas in the cat – if it is assumed that the whole visual system of this species were homologous to the magnocellular system – these cells would project to the homologue of the magnocellular system. This is not a criticism of the proposal of Shapley and Perry (20) but a demonstration of how it is difficult to use their model to establish a homology between the cat visual system and the primate magnocellular system.

These above observations, therefore, indicate that, at the level of the LGN, major differences exist between the cat visual system and the monkey magnocellular system with regard to the inputs they receive from the retina. In the case of LGN cells, the difference in the nature of these inputs is of particular importance. This is because LGN cells are considered to be ‘relay cells’ that mainly pass on the response properties of their retinal inputs. In consequence, if the inputs to the magnocellular layers of the primate LGN differ from those of the cat LGN, it is difficult to consider them as homologous systems.

Another difference is that the magnocellular system in the monkey receives considerable inputs from cones (mainly L- and M-cones (34)), whereas the cat’s visuals system receives mainly input from rods as the cat has a rod-dominated retina (35).

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Even if the cat visual system had been an appropriate model for the primate magnocellular system, testing the cat would not by itself have been a sufficient basis to establish a specifically magnocellular effect. This is because in order to attribute visual abnormalities specifically to the magnocellular system, it is also necessary to exclude the possibility that such abnormalities do not arise from other causes. For instance, it needs to be ascertained that they had not arisen from a general visual impairment. However, to be able to exclude such possibilities would require studies of species (unlike the cat) with visual systems containing both magno- and parvocellular streams.

Functional aspects of NMDA receptors

To attempt to link results from the cat to the magnocellular systems of schizophrenic subjects by the way of functional properties also faces difficulties. In regard to NMDA receptors, Javitt (14, p. 264), for example, wrote: '... magnocellular neurons give rise to the percept of motion, largely through the involvement of NMDA receptor-dependent mechanisms' (14, p. 264). Three studies are cited in support of this claim: Kwon et al. (15), Heggelund and Hartveit (36) and Rivadulla et al. (37). Of these three studies, the first two do not deal with motion perception and all three were performed not in primates but in cats. However, as shown above, the use of the cat as a model for the primate is highly problematic. Another issue is that there is evidence to indicate that movement perception is not linked specifically to the magnocellular system (38,39). For instance, Merigan et al. (40) placed lesions in the magnocellular layers of the LGN in monkeys and studied the effect upon motion perception. The conclusion of that study was that the magnocellular system does not provide a 'motion-specific contribution' (40, p. 3426), and that the magnocellular system is not necessary for the discrimination of direction or speed.

The study of Rivadulla et al. (37), cited by Javitt (14), investigated the role of NMDA receptors in creating cortical direction selectivity in the cat. The roles of the magno- and parvocellular systems (in monkeys) in cortical direction selectivity has been investigated by Malpeli et al. (41). These researchers found that while inactivation of either the magno- or the parvocellular layers of the LGN reduced cortical responsivity, it did not abolish direction selectivity. This again indicates that the magnocellular system does not play a special role in regard to direction selectivity in the visual cortex. Further difficulties arise also in regard to linking the magnocellular system to cortical functioning in general as there is considerable mixing of the magno- and parvocellular inputs inside the visual cortex (42–49).

In connection with schizophrenia, attempts have been made at linking NMDA receptors to the magnocellular system by the way of contrast gain control (13,14). This faces the problem in regard to using the cat as a model for the magnocellular system in that there is very little contrast gain control in the early visual system (i.e. the LGN) of the cat (50). The concepts of contrast gain control and contrast saturation appear to be confused in the schizophrenia literature. For instance, Javitt (14 p. 260) wrote that '... magnocellular neurons function in a nonlinear gain mode, in which they show rapid increase in firing at low contrast levels but saturating response at higher contrast levels. This response profile is also frequently described as gain control, in that the degree of gain decreases with increasing contrast'. 'Contrast gain control,' as this term is commonly used, refers to the ability of neurons to adjust their contrast–response functions in accordance with the ambient contrast. This can occur irrespective of whether or not the curve shows saturation. For a discussion of this issue, see Ohzawa et al. (50). Also, the cat LGN differs from the primate magnocellular system in regard to adaptation in general since Solomon et al. (51) found that magnocellular neurons, in contrast to parvocellular cells in the macaque LGN are highly susceptible to adaptation. This susceptibility stands in marked contrast to the LGN of the cat where only modest adaptation effects have been found (50,52–54).

Conclusion

The subcortical visual systems of cats and primates show important differences. These differences make it difficult to draw conclusions about the magnocellular system in schizophrenic subjects based on experiments upon the LGN of cats. This does not mean that important similarities also may not exist between the visual systems of cats and primates. But, rather that the marked differences noted above suggest the need for considerable caution.

In the particular case of NMDA receptors, there is also the complicating factor that the lagged cells, which in the cat have been found to be specifically linked to NMDA receptors, have also been found in the parvocellular layers of the monkey LGN. Therefore, to make conclusions about the magnocellular system in humans, e.g. schizophrenic individuals, on the basis of studies in the cat is unlikely to be fruitful. Also, results from contrast sensitivity studies and anatomical investigations have found little evidence for a magnocellular deficit in schizophrenia. This suggests that the reduced

https://doi.org/10.1111/j.1601-5215.2011.00611.x Published online by Cambridge University Press

ssVEP amplitudes (noted in the Introduction section) associated with schizophrenia are unlikely to be specifically linked to the magnocellular system.

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