Two main pathways lead from the retina to the brain in the human visual system. They are the magnocellular pathway and the parvocellular pathway. Each one is specialised for transmitting different types of visual information. The magnocellular pathway is specialised for transmitting coarse-grain information and information about movement. Magnocellular retinal ganglion cells arise mainly from the non-foveal region of the retina. The parvocellular pathway is specialised for transmitting fine-grain highly detailed information. Parvocellular retinal ganglion cells arise mainly from the foveal region of the retina. Both of these types of information are crucial for visual word recognition. Based on a series of experiments, I am currently developing a PDP model that may help account for the specific roles of each of the visual pathways in the reading of text. A magnocellular deficit has been implicated in certain types of dyslexia (e.g. Stein and Walsh 1997). Dyslexic readers tend to have different patterns of eye movements compared to control participants. They also have difficulty in maintaining stable eye fixations, as well as having difficulty with convergent eye movements (de Luca et al 1999). These problems can be accounted for by the magnocellular theory. If magnocellular retinal ganglion cells arise mainly from the parafoveal region of the retina, a disruption of the magnocellular pathway could be said to disrupt the parafoveal preview. If the brain receives reduced parafoveal preview information, more fixations will have to be made per line to gain the same amount of information from the text. The input to posterior parietal cortex is also magnocellular. PPC is responsible for directing involuntary attention and for integrating information from the two visual fields. A disrupted magnocellular input to PPC would lead to an overlap between the inputs of the two visual fields. Using the distinction of coarse- and fine-grain input to simulate the magnocellular/parvocellular division, the proposed model hypothesises that when the magnocellular component of the model is disrupted, a dyslexia like behaviour will emerge.

References
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