Editorial

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Blue Cone Signals in the Extra Striate Cortex: Explanation for Blind Sight?

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Our perception of vision is largely as a result of the signals conveyed from the eye to the brain *via* the retino-thalamo-cortical pathway. Visual signals within this pathway originate from three cone photoreceptors (responsible for day vision) and one scotopic receptor, the rods. The cones are classified according to their spectral sensitivity peaks as Long(L), Medium(M) and Short(S) wavelength sensitive cones. Our chromatic perception is dependent upon how the brain processes variations in the activity among these photoreceptors. Chromatic signals are generally thought to be processed by two parallel streams, the red-green system by the parvocellular system[1,2] and the blue-yellow system by the koniocellular system.[3,4] A large body of work has already been documented to identify these streams within the retina and the main thalamic visual nucleus, the Lateral Geniculate Nucleus (LGN). However, our knowledge about the processing of the chromatic signal within the visual brain, particularly the pathways taken by the blue-yellow colour signals within the brain is at its infancy.

Majority of relay cells within the LGN project to the primary Visual cortex (V1) and hence is considered vital for our visual perception. However, in some clinical cases, even after extensive damage to V1, patients have some residual vision. This phenomenon is called "blind sight".[5,6] The patients usually exhibit little or no awareness of visual stimuli, however can perform tasks that seemingly needs vision such as navigation. Most common hypothesis that has been postulated explaining this phenomenon is that blindsight is mediated by inputs to middle temporal area or area MT that bypass V1.[7,8] Area MT remains active even after complete ablation[9] or reversible inactivation[10] of V1, suggesting that there are alternative pathways that bypass V1 to reach MT. Experiments with inactivation of the superior colliculus showed that one such pathway exists *via* the colliculus.[11] Whether there is any evidence that blindsight is made up of chromatic signals is still unresolved. The perception of colour is usually associated with the "what" pathway as evident from area V4[12] often considered to be the area that processes color information and for being the gateway to the ventral pathway. Area MT, an extrastriate visual area is considered to be part of the " where" pathway and until recently was not considered to receive any colour signals but see Conway[13] for a review of color signals in the dorsal and ventral pathways. However, more recent studies[14,15] have shown that this area receives robust chromatic signal not only *via* V1 but also bypassing V1.[15] Other studies[16] have also shown that in subjects with blindsight found that within the hemianopic field, S-cone modulating stimuli were very effective in eliciting visual performance, and in fact in one patient presentation of narrow band blue stimuli (427 nm) led to excellent performance but not red stimuli (peaking at 630 nm). Thus, there is increasing evidence that S-cone signals reach area MT *via* a more direct route that bypasses V1 and providing an alternate explanation of blindsight in humans.

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