Contribution of melanopsin-expressing retinal ganglion cells to pupillary control pathway studied with a receptor-silent substitution technique

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It is widely accepted that the constriction and dilation of the pupils is evoked by changes in the ambient luminance, suggesting that retinal cone pathways contribute to the pupillary control mechanism. Recently, several studies have shown that retinal ganglion cells containing the photopigment melanopsin, which are intrinsically photosensitive in primates, project to the pupillary control centre in the pretectum. The aim of this study was to investigate how signals driven by melanopsin-expressing retinal ganglion cells (mRGCs) contribute to the pupillary control mechanism. We designed and built a novel multi-primary stimulation system to independently stimulate mRGCs from the other photoreceptors using a receptor-silent substitution technique. The stimulation system consists of an optical diffuser and an integrating sphere. We calculated excitations of the mRGCs and the other photoreceptors on a test field. In the mRGC condition we changed an excitation of mRGCs alone without changing the colour and the luminance on a test field. In the luminance condition we changed luminance of the test field alone without changing the colour and the excitation of mRGCs. The mRGC excitation and the luminance for test field varied 3.3 times in each condition. The test field was presented for 10 minutes and the pupillary diameter was recorded for 2 seconds and repeated with an interval of 30 seconds during test field presentations. In mRGC condition, when pupil diameter was compared across the five excitation levels each observer showed highly significant differences in pupil diameter across mRGC excitation. The pupil diameter decreases as mRGC excitation increases. Although the colour and luminance of test field were constant, the remarkable decrease of pupil diameter was found. In luminance condition the pupil diameter decreases as luminance of test field increases. These results indicate that the mRGC signals contribute greatly to the pupillary control mechanism as well as luminance signals.