found that this protein is required for sustained $InsP_3$ production in permeabilized HL60 cells. In *Drosophila*, the *rdgB* gene encodes a PtdIns-TP that is expressed in photoreceptors[9]. Mutations in this gene produce an abnormal photoresponse that further deteriorates upon exposure to light, and *rdgB* mutants, like *cds* mutants, undergo light-dependent retinal degeneration.

Wu *et al.* have shown that there is a unique pathway for synthesis of the CDP-DAG used for phototransduction, and their demonstration of the consequences of overexpressing and underexpressing CDS *in vivo* raises the intriguing possibility that this reaction could have a regulatory role in vision. Many issues now need to be addressed. For example, it is also intriguing that degeneration of photoreceptors deficient in CDS and PtdIns-TP requires light whereas degeneration of photoreceptors deficient in DAG kinase occurs even in darkness. Taken together, these findings highlight the importance of the enzymes of the phosphoinositide cycle that supply substrate on demand for a signal-transduction pathway that is found in nearly all eukaryotic cells. □

James B. Hurley, of the Howard Hughes Medical Institute Research Laboratories, University of Washington, Seattle, is currently in the Biological Laboratories, Harvard Medical School, 16 Divinity Avenue, Cambridge, Massachusetts 02138, USA.

VISUAL PERCEPTION

Vision without awareness

Jon H. Kaas

ON page 247 of this issue[1], Cowey and Stoerig tackle the difficult question of what monkeys see if they don't have a visual cortex. Early studies of monkeys[2] and humans[3] seemed to indicate that there is nothing to investigate. The loss of primary visual cortex (visual area 1 or V1) impaired vision to such an extent that the loss was called 'cortical blindness' — monkeys or people with this condition appeared to have no awareness of objects or attributes of objects such as location, form, size or brightness.

Researchers were therefore surprised by subsequent evidence that, after complete, bilateral lesions of V1, squirrel-like tree shrews[4] and cats[5] could avoid obstacles, follow moving objects and discriminate between simple visual patterns. About the same time, monkeys deprived of V1 were found to be able accurately to reach out for visually presented objects[6], and the absence of vision in humans with such lesions came into question.

Further studies in humans[7] led to the conclusion that considerable vision is possible without V1, so that objects can be detected and visually followed, but not identified. Most remarkably, object detection is not accompanied by awareness. For impaired observers, it seems that there is nothing to detect even though they perform well above chance when forced to make choices. The remaining ability to detect without awareness has been called 'blindsight'[7]. Because V1 provides most but not all of the visual input to higher visual areas of the brain, an indirect visual pathway from the retina through midbrain and thalamus to higher visual cortical areas presumably mediates this condition[8].

The validity of the blindsight concept has been challenged in two ways. First, it can be difficult to determine the extent of lesions in humans. So it is possible that, in at least some cases, preserved but compromised remnants of V1 in humans permit detection without awareness, much as near-threshold stimuli can be detected by normal individuals without them being certain or fully aware of the presence of the stimulus. Indeed, in a study involving brain imaging[9], a patient demonstrating features of blindsight was found to have possibly functional remnants of V1. One current viewpoint is therefore that blindsight in humans reflects suboptimal function in V1.

The second type of challenge has been to question the relevance of animal studies as support for the argument that humans have blindsight. In monkeys and other mammals, it is possible to remove all of V1, test for remaining vision, and then examine the histology of the brain to make certain the lesions were complete. Because monkeys deprived of V1 can locate objects in space and discriminate between simple forms[6,8], the remaining abilities of human subjects after V1 damage do not seem so surprising. Yet, despite many apparent similarities in at least early stages of visual processing, humans are not monkeys, and the possibly more compelling evidence for preserved vision in monkeys after V1 lesions can be dismissed as irrelevant to the issue of human blindsight[10].

Cowey and Stoerig[1] directly address the possibility that monkeys as well as humans have blindsight. They present evidence that monkeys without V1, like humans, detect visual objects without awareness. The trick, of course, is to get monkeys to tell you that they can visually detect something that they don't consciously see. This was cleverly done by demonstrating detection in the 'blind' hemifield of monkeys with unilateral removal of V1, and then having monkeys classify trials in the 'blind' hemifield as a blank (no object present) or a stimulus (object present) trial. Monkeys classified detected objects as unseen (blank trials). Given this apparent demonstration of vision without awareness in monkeys, the case for blindsight in humans becomes more plausible.

The report of Cowey and Stoerig also reminds us of the modularity of processing in our brains, and that we do not have awareness of all modular functions. It seems that V1 is critical for visual awareness, even though some higher visual areas may be activated through the midbrain pathway. Interestingly, monkeys with large lesions of non-visual cortex act as if blind[11], implying that visual cortex alone is not sufficient for visual awareness.

Recognizing a fundamental similarity in the role of V1 in visual perception in humans and monkeys does not mean that there are no species differences in visual processing. Old World monkeys resemble humans in that V1 is profoundly important in visual processing; lesions of V1 alter many parts of the visual system, and in humans and Old World monkeys such lesions result in the loss of 80 per cent of the ganglion cells of the retina[12], making the preserved abilities seem even more remarkable. In other adult mammals that have been examined, including New World monkeys and prosimian primates, no such loss of ganglion cells occurs after V1 lesions. For this reason alone, one might expect greater sparing of visual function after lesions of V1 in many mammals. Unlike humans and Old World monkeys, tree shrews appear to have nearly normal visual behaviour after lesions to V1 (ref. 4), but they seem to be blind[13] after lesions of the visual midbrain, thus apparently suffering from 'midbrain blindness'. □

Jon H. Kaas is in the Department of Psychology, Vanderbilt University, Nashville, Tennessee 37240, USA.

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