

# Injuries to primary visual cortex cause long-term dysfunction of neural circuits

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Injuries to primary visual cortex cause long-term dysfunction of neural circuits.  
Credit: ICTER / Grzegorz Krzyzewski

Even mild head injuries can mean serious consequences for brain function at its most basic level. Research published in *Communications*

*Biology* shows that neuroplasticity, too, has its limits.

Injuries to the posterior occipital cortex are common in humans. Traumatic brain injury (TBI) can lead to long-term visual impairment (like loss of visual acuity). For example, estimates suggest that as many as 75% of current or former soldiers live with permanent visual dysfunction or cortical blindness. TBI is associated with mechanical brain damage and a wide range of neuronal abnormalities.

The human brain is characterized by surprising plasticity. Even when one part is injured, the functions of the damaged [neurons](#) can be taken over by other cells. This is because [neural tissue](#) has a remarkable ability to form new connections to reorganize, adapt, change, and self-repair the entire organ.

Such neuroplasticity is also characteristic of the sensory areas of the visual cortex. This region of the brain is the final component of the visual pathway, responsible for receiving and processing visual impressions. The [primary visual cortex](#) (V1) is reached by the nerve fibers of the optic radiation, which carry nerve impulses from the retinas of both eyes.

Until now, scientists knew little about the effects of TBI on long-term visual circuit function. A team of researchers led by John C. Frankowski from the Department of Anatomy & Neurobiology, University of California, Irvine, and Dr. Andrzej Foik from the International Centre for Translational Eye Research (ICTER) at the Institute of Physical Chemistry, Polish Academy of Sciences, examined in vivo (in adult mice) how neurons respond to [visual stimuli](#) two weeks and three months after mild injury to the primary visual cortex (V1). V1 neurons normally show sensitivity to different features of a visual stimulus, such as color or direction of movement. The preprocessed data is transmitted to subsequent areas of the visual cortex. This study showed that although

the primary visual cortex remained largely intact after the brain injury, there was a 35% reduction in the number of neurons. This loss largely affected inhibitory neurons rather than excitatory neurons, which, as their names indicate, inhibit or stimulate action in the target cells, respectively.

After TBI, less than half of the isolated neurons were sensitive to visual stimuli (32% at two weeks after injury; 49% at three months after the event), compared with 90% of V1 cells in the control group. There was as much as a threefold decrease in neuronal activity after the brain [injury](#), and the cells themselves had worse spatial orientation. The overall results mean that even minor, superficial [brain](#) injuries cause long-term impairment in the way visual stimuli are perceived, persisting several months after the event.

A deeper understanding of the functional impairments in damaged visual cortex is important because it can provide a basis for developing circuit-level therapies for visual [cortex](#) damage.

**More information:** Jan C. Frankowski et al, Traumatic brain injury to primary visual cortex produces long-lasting circuit dysfunction, *Communications Biology* (2021). [DOI: 10.1038/s42003-021-02808-5](https://doi.org/10.1038/s42003-021-02808-5)

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