Normal eye dominance is not necessary for restoring visual acuity in amblyopia

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Amblyopia, commonly known as "lazy eye," is a visual disorder common in children. The symptoms often are low acuity in the affected or "lazy" eye and impaired depth perception. Researchers have long believed that the impaired vision by one eye is a consequence of exaggerated eye dominance that favors the fellow or "good" eye.

Amblyopia typically is treated by patching the fellow eye to strengthen the affected eye with the goal of restoring normal eye dominance. If correction is not achieved prior to the closing of a "critical period" that ends in early adolescence, visual impairments are more difficult to treat, if not permanent.

Research published today, led by Aaron W. McGee, Ph.D., assistant professor in the University of Louisville Department of Anatomical Sciences and Neurobiology, may lead to changes in how amblyopia is treated, particularly in adults. The research shows that eye dominance and visual acuity are controlled by different areas of the brain, and that one can be corrected without correcting the other.

"We unexpectedly discovered that they aren't related. They're independent," McGee said. "It may not be necessary to instill normal eye dominance to correct visual acuity."

Previously, McGee and fellow researchers identified a gene called ngr1 as essential in closing the critical period. He found that deleting ngr1 in animal models permits the critical period to remain open or to re-open, facilitating recovery of normal eye dominance and visual acuity. However, the relationship between the improved visual acuity and eye dominance was not clear.

Today's research reports that recovery of eye dominance alone is not sufficient to promote recovery of acuity, and recovery of acuity can occur even if eye dominance remains impaired.

McGee and his colleagues found that eye dominance is regulated by the brain's primary visual cortex, while visual acuity is governed by another area of the brain, the thalamus.

McGee is the senior author on the article, published in *Current Biology*, (Distinct Circuits for Recovery of Eye Dominance and Acuity in Murine Amblyopia). Co-authors include Céleste-Élise Stephany Ph.D., a graduate student at the University of Southern California at the time of the research and now a postdoctoral fellow at Harvard Medical School, Shenfeng Qiu, Ph.D., assistant professor of the University of Arizona, and others.

The researchers applied tools to selectively delete the ngr1 gene in different areas of the brain. When ngr1 was deleted from the primary visual cortex, normal eye dominance was recovered but acuity remained impaired. When ngr1 was deleted from the thalamus, eye dominance was impaired, but visual acuity recovered to normal.

"Genes that are limiting recovery from amblyopia are working in parts of brain circuitry that previously were not recognized to have a role in improving visual acuity," McGee said. "This could allow researchers to address acuity directly, without having to restore normal eye dominance."

More information: Céleste-Élise Stephany et al, Distinct Circuits for Recovery of Eye Dominance and Acuity in Murine Amblyopia, *Current Biology* (2018). DOI: 10.1016/j.cub.2018.04.055

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