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Researchers identify new neurological deficit behind lazy eye

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Researchers at New York University's Center for Neural Science have identified a new neurological deficit behind amblyopia, or "lazy eye." Their findings, which appear in the most recent issue of the *Journal of Neuroscience*, shed additional light on how amblyopia results from disrupted links between the brain and normal visual processing.

Amblyopia results from developmental problems in the brain. When the parts of the brain concerned with visual processing do not function properly, problems ensue with such visual functions as the perception of movement, depth, and fine detail. It is most prevalent neurological defect of vision in children and adults, affecting 1-3 percent of the population.

Previous research on amblyopia has largely focused on one aspect of visual processing—that in the primary visual cortex, or V1.

However, while abnormalities in V1 explain some amblyopic visual problems, they fail to account for the full range of losses suffered by those with amblyopia—including motion perception. With this in mind, the NYU researchers studied a brain area called MT, which has a well-established role in processing information about moving visual objects.

To do this, the researchers studied the visual processing of macaque monkeys, examining those who had normal vision and those whose vision was impaired by amblyopia. The researchers recorded both the monkeys' ability to detect motion and how MT's neurons functioned in

this process.

Their results showed striking changes in neuron activity in MT. In monkeys with normal vision, the MT neurons responded through both eyes. However, in those with amblyopia, the MT neurons showed stronger response in one eye—usually the one not affected by the disorder. Normal visual motion perception relies on neurons that integrate information about the position of moving objects as they cross the visual image. The NYU researchers found that this ability to integrate motion information was defective in neurons driven through the affected eye, which might explain the animal's deficits in motion perception.

"This study shows that amblyopia results from changes in the brain that extend beyond the primary visual cortex," said J. Anthony Movshon, director of the Center for Neural Science and the paper's senior author, adding that many other affected neurological regions remain undiscovered.

Provided by New York University