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Study uncovers new molecular signaling mechanism for correcting childhood visual disorders

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Neuroscientists at University of California, Irvine have discovered a molecular signaling mechanism that translates visual impairments into functional changes in brain circuit connections. The discovery may help to develop novel therapeutic drugs to treat the childhood visual disorder amblyopia and other neurodevelopment disorders. Xiangmin Xu, Todd Holmes and Sunil Gandhi conducted the study, which appears online Sept. 15 in *Neuron*.

Amblyopia is the most common cause of permanent visual defects among children and is often a result of improper brain development due to deprivation during the "critical period" of vision development. In a previous study, Xu helped discover that a specific class of inhibitory neurons (parvalbumin-expressing neurons, or PV neurons) control the critical period of vision development. In this study, Xu and colleagues found that neuregulin-1 (NRG1) molecules modulate the activities of these neurons, thus outlining a new path for treatments that can restore normal vision in children who have had early deficits.

As neurodevelopmental disorders such as schizophrenia appear to result from brain developmental defects during defined postnatal windows, the linkage of NRG1 signaling to critical growth periods provides important new insights. Xu said he hopes that therapeutic interventions targeting NRG1 may be exploited to treat cortical neurodevelopmental disorders.

More information: Yanjun Sun et al. Neuregulin-1/ErbB4 Signaling Regulates Visual Cortical Plasticity, *Neuron* (2016). DOI: 10.1016/j.neuron.2016.08.033