Amblyopia Reveals the Neuroanatomical Consequences of Prolonged Abnormal Binocular Experience

Bas Rokers
Department of Psychology and McPherson Eye Research Institute, University of Wisconsin-Madison, Madison, Wisconsin, United States; rokers@wisc.edu

Amblyopia is of interest not just as a clinical disorder, but also because it sheds light on the interplay between sensory input and brain development. Duan et al. help identify the neuroanatomical consequences of prolonged abnormal binocular experience. Electrophysiology and neuroimaging studies have linked amblyopia to reduced neural activity, but it has been unclear if this reduction exists purely at the functional level, or has a neuroanatomical basis.

Diffusion weighted imaging (DWI) can measure the diffusion of water molecules, and makes it possible to investigate the architecture of the living human brain at the millimeter level. Because it is easier for water to diffuse along axons rather than through them, diffusion can reveal the location, integrity, and density of white matter tracts.

In the current study, the authors used DWI to investigate the properties of white matter tracts in strabismic amblyopia. When compared with age-matched controls, the authors found elevated diffusion within a number of white matter tracts, including the optic radiation. Thus, these results indicate a reduction in the structural integrity of these tracts following long-term abnormal visual experience.

Clinically, these findings open the way to using neuroanatomical measures to assess the efficacy and impact of novel amblyopia treatments.

In addition, these findings help clarify the complex interaction between amblyopia, neural activity, and neural architecture. While it is clear that the visual deficits cannot simply be linked to a single underlying neural cause, both this paper and a study including strabismic and anisometropic amblyopia now show clear deficits in the optic radiation.

An interesting open question concerns whether these deficits depend on neural plasticity during the critical period, or can arise when input to one of the eyes is systematically disrupted later in life, such as in macular degeneration or glaucoma.

References