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Visual cortex rewiring in Retinitis Pigmentosa: Plasticity is preserved.

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Retinitis Pigmentosa (RP) refers to a group of inherited diseases of retinal function affecting 1 in 4000 people worldwide. RP is characterized by the progressive degeneration and death of photoreceptors, which typically starts with a loss of peripheral visual function and eventually leads to blindness (Verbakel et al., 2018). The anatomy and function of visual pathways from the retina to the visual cortex has been well documented in RP. However, very little is known about the impact of the disease on visual cortical activity and even less on the effect of RP on neural plasticity, the intrinsic property of the brain to be modified by environmental pressures.

A research article by Begenisic et al published in this issue of *Neuroscience*, directly tackles this question by assessing the amount of experience-dependent plasticity in the primary visual cortex (V1) of a murine model of RP (rd10). By using two well-established methods to quantify neural plasticity, Begenisic et al demonstrate that rd10 mice retain a level of plasticity in V1 comparable to wild type animals. In particular, the authors report two main results: (1) rd10 mice show normal-like shifts in ocular dominance following monocular deprivation, indicating preserved structural plasticity in V1 despite the decrease in responsiveness induced by retinal degeneration; (2) the long-term potentiation (LTP) of synaptic strength measured in layer II-III of V1 in response to theta-burst stimulation in layer IV is comparable in rd10 and wild type animals even at late stages of the disease, when the retinal input is severely deteriorated.

These findings are not only novel, but also bear important translational implications. Whereas there is no cure at present for RP, new hope emerges from therapeutic strategies aimed at replacing photoreceptor function using retinal prostheses (see Bloch et al., 2019 for a recent review). Most of the research on retinal prostheses has focused on outstanding technical challenges and on modelling the retinal responses evoked by the prostheses (Beyeler et al., 2019). However, very little is known at present about the ability of the visual cortex to process the restored visual input, a capability that critically depends on experience-dependent plasticity. After many years (sometimes several decades) of blindness, the deafferented visual cortex is recruited for the processing of other sensory modalities (Fine and Park, 2018). Could these visual cortical circuits re-adapt?

A few studies on human patients have explored this issue, showing that short-term ocular dominance plasticity is preserved in RP patients (Lunghi et al., 2019), and that, after prolonged training with retinal prostheses, BOLD responses to visual flashes are enhanced (Castaldi et al., 2016) whilst there is reduced cross-modal activation by touch (Cunningham et al., 2015) in the primary visual cortex of RP patients. The present study confirms and extends these findings, showing for the first time that input-specific structural and synaptic neural plasticity in V1 is not hampered by the disease. This provides a strong indication that the primary visual cortex of RP individuals retains the potential for rewiring in response to new visual inputs and therefore to benefit from visual restoration approaches.

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