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Plasticity, and its Limits, in Adult Human Primary Visual Cortex

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Plasticity, and its Limits, in Adult Human Primary Visual Cortex

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Abstract

There is an ongoing debate about whether adult human primary visual cortex (V1) is capable of large-scale cortical reorganization in response to bilateral retinal lesions. Animal models suggest that the visual neural circuitry maintains some plasticity through adulthood, and there are also a few human imaging studies in support this notion. However, the interpretation of these data has been brought into question, because there are factors besides cortical reorganization that could also explain the results. Still, how reasonable would it be to accept that adult human V1 does not reorganize itself in the face of disease? Here, we discuss new evidence for the hypothesis that adult human V1 is not as capable of reorganization as in animals and juveniles, because in adult humans, cortical reorganization would come with costs that outweigh its benefits. These costs are likely functional and visible in recent experiments on adaptation—a rapid, short-term form of neural plasticity—where they prevent reorganization from being sustained over the long-term.

Keywords

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If the retina is damaged in both eyes, primary visual cortex (V1) no longer receives input. It is widely hypothesized that neurons within the V1 cortical lesion projection zone (LPZ) respond to the absence of incoming information by shifting their receptive fields from the blind toward intact portions of the visual field (as suggested by e.g., Kaas et al., 1990; Gilbert and Wiesel, 1992; Darian-Smith and Gilbert, 1994; Chino et al., 1992; Chino et al., 1995; Calford et al., 1999; Calford et al., 2000; Schmid et al., 1995; Darian-Smith and Gilbert, 1995). This type of visual brain plasticity is often called 'remapping', because the relocation of neuronal receptive fields will change V1's retinotopic map: retinotopic maps in cortex emerge from the fact that nearby visual neurons have receptive fields at nearby locations in the visual field, so when neurons shift the location of their receptive fields, the retinotopic map will change accordingly. Visual brain plasticity in the form of remapping is generally thought to be slow, because it would require that structural changes are made to the underlying neural circuitry. However, recently, the interpretation of the data presented in support of cortical remapping has been seriously questioned (see Wandell and Smirnakis, 2009 for a review).

Another important type of visual brain plasticity is neuronal adaptation. In contrast to cortical remapping, neuronal adaptation is generally very fast (e.g., Clifford et al., 2000; Kohn, 2007; Wandell and Smirnakis, 2009). For instance, if a person looks at a waterfall for a short period of time and then shifts her gaze away, the world will perceptually move upward (Anstis et al., 1998). This motion after-effect is thought to be due to the fact that motion-processing visual neurons adjust their responsiveness, which likely reflects an attempt to optimize visual processing by maintaining a state of equal time-averaged activity and decorrelated neuronal stimulus sensitivity (Attneave, 1954; Andrews, 1964, Ullman and Schechtman,

1982; Barlow and Fo□ldiák, 1989; Anstis et al., 1998; Wainwright, 1999; Benucci et al., 2013; Haak et al., 2014a). Unlike the neuronal receptive field changes underlying cortical remapping, the changes underlying neural adaptation do not appear to rely on structural changes in the visual neural circuitry (Clifford et al., 2000; Kohn, 2007; Wandell and Smirnakis, 2009), but rather on functional interactions, such as mutual inhibition (Movshon and Lennie, 1979; Barlow and Fo□ldiák, 1989).

Whereas neuronal adaptation is a firmly established property of the visual system across species and ages (Clifford et al., 2000; Kohn, 2007), cortical remapping following retinal lesions has yet to receive such status, particularly in adult humans (for a review, see Wandell and Smirnakis, 2009). A handful of functional magnetic resonance imaging (fMRI) studies have characterized abnormal functional magnetic resonance imaging (fMRI) activity in V1 following retinal lesions (e.g., Baker et al., 2005; Schumacher et al., 2008; Baker et al., 2008; Dilks et al., 2009; Dilks et al., 2014), thereby claiming evidence of large-scale reorganization. However, abnormal activity in itself does not warrant the conclusion that cortical remapping has occurred (Masuda et al., 2008; Wandell and Smirnakis, 2009; Baseler et al., 2009; Masuda et al., 2010; Haak et al., 2014c). Cortical remapping can only be concluded on the basis of abnormal activity patterns if it can also be shown that the absence of visual input in itself does not change the measurements in the same way. For instance, in a recent study, Baseler et al. (2011) compared the fMRI activity patterns from the cortical lesion projection zone (LPZ) in macular degeneration patients with real retinal lesions and healthy controls with simulated retinal lesions. They found that although the LPZ in V1 was largely silenced by both real and simulated retinal lesions, for a small fraction of voxels (~5%) the patients with real retinal lesions exhibited the same apparent neuronal receptive

field changes as controls with simulated lesions, indicating that these changes were caused by the absence of visual stimulation alone. Importantly, these voxels were found far into the LPZ, indicating that the receptive field changes could not be easily explained by measurement artifacts at the fringe of the LPZ (Haak et al., 2012; Binda et al., 2013). Rather, they appeared to be a feature of visual cortical processing, unveiled in the absence of visual stimulation.

In the first instance, it seems quite reasonable to expect that the adult human brain would adjust itself in the face of retinal lesions. After all, if children are capable of developing relatively normal vision even when an entire occipital lobe failed to develop (e.g., Werth, 2006, Muckli et al., 2009, but see Haak et al., 2014c), one might also expect the brain to at least ameliorate the consequences of a retinal lesion by dedicating the now-redundant resources of deafferented cortex to processing retinal inputs that are still intact. The net effect of such cortical remapping would be quite similar to the perceptual 'filling-in' of the blind-spot of the healthy retina, as well as the perceptual filling-in that occurs when someone stares steadily at an image with patches of missing 'texture' for a prolonged period of time (Ramachandran and Gregory, 1991; Pettet and Gilbert, 1992; Komatsu, 2006; Weil and Rees, 2011). Here, the patches of missing 'image data' will be perceptually filled-in with the texture from surrounding image regions. Just like the after-effects of neuronal adaptation, perceptual filling-in occurs fairly rapidly, and the neural mechanisms underlying perceptual filling-in could likewise be interpreted as a way of optimizing visual processing in the context of the preceding stimulus history (i.e., in the vein of Horace Barlow's redundancy reduction hypothesis; c.f. Barlow, 1961; Barlow, 2001). Thus, perceptual filling-in, which would be the consequence of cortical remapping in response to retinal lesions if it occurred, seems to share important features with visual neuroplasticity in the form

of neuronal adaptation. Therefore, as the loss of vision endures, it might be expected that the brain would eventually change its neural circuitry via cortical remapping to sustain perceptual filling-in over the long-term. However, this does not appear to be the case—perceptual filling-in does not appear to produce long-term, hard-wired changes in the face of a retinal lesion.

Interestingly, recent work now suggests that neuronal adaptation may also not produce hard-wired changes over the long-term. Using immersive virtual reality, Haak et al. (2014b) exposed a group of young adults to a world with only very little vertical visual contrast energy for four days continuously, in an attempt to mimic classic selective rearing experiments (Hirsch and Spinelli, 1970; Blakemore and Cooper, 1970) in adult humans. Just as staring at a waterfall for a prolonged period of time changes the response gains of motion-sensitive neurons, the prolonged viewing of a world with relatively little vertical contrast will cause adjustments to the responsiveness of orientation-selective cells in primary visual cortex (e.g., Graham, 1989; Maffei et al., 1973; Movshon and Lennie, 1973; Ohzawa et al., 1985; Dragoi et al., 2000). Monitoring for the perceptual consequences of these changes in the responsiveness of orientation-selective neurons, Haak et al. (2014b) found that adaptation increased in magnitude during the first day, but then decreased, despite the sustained presence of the adapting environment. Thus, it appears that there are factors that prevent visual neuroplasticity, in the form of neuronal adaptation, from being sustained over the long-term.

Haak et al. (2014b) concluded that if neuronal adaptation does in fact optimize vision, then the decline in adaptation strength must be due to costs that outweighed its benefits. An obvious candidate cost is the ‘coding catastrophe’, where changes in the firing of neurons responsible for early visual processing are mistaken for

stimulus-changes by neurons that are responsible for subsequent, higher-level stages of visual processing (Schwartz et al., 2007; Series et al., 2009; Druv and Carandini, 2014; Patterson et al., 2014). Indeed, when Patterson et al. (2014) induced adaptive changes in the responsiveness of V1 neurons in the macaque by having them stare at a drifting grating pattern for some time, this impeded the ability of neurons in area MT, which receives most of its inputs from V1, to integrate the V1 signals into a plaid pattern. They concluded that "the effects of adaptation cascade through the visual system, derailing the downstream representation of distinct stimulus attributes".

Importantly, the decline in adaptation strength observed by Haak et al. (2014b) was then followed by an increase in adaptation during subsequent days, indicating that a second, more slowly acting adaptive mechanism was able to overcome the costs of the initial adjustments in neuronal responsiveness. It is likely that this second, slower form of adaptation reflects a process more similar to 'perceptual learning', during which the visual system typically adjusts the neural codes in later rather than in earlier visual areas (see e.g., Hochstein and Ahissar, 2002; Ahissar and Hochstein, 2004). This would make sense, because later stages of visual processing are typically concerned with more abstract sensory representations that require more flexible neural codes. Moreover, compared with the areas responsible for the early stages of visual processing, there are far fewer downstream areas that depend on the information throughput of later visual areas. Thus, by shifting the adaptive neural code adjustments from the early to higher-level visual areas, the brain may be able to optimize vision without the adverse effects of the coding catastrophe. Indeed, Haak et al. (2014b) observed that the tilt-aftereffect, an illusion thought to be due to the coding-catastrophe, began to decline toward the end of the experiment.

Here, we put forward the hypothesis that the same principles may also apply to visual processing in the face of retinal lesions. That is, the retinal lesion would initially cause rapid 'adaptive' changes in the location of the receptive fields of the neurons within the lesion projection zone in V1. These changes, however, will likely cause havoc at later stages of vision, due to the mismatch between the assumed and true stimulus locations that their afferents encode. Thus, early stage changes could soon be undone, as perceptual filling-in is postponed to later stages of visual processing (in line with e.g. De Weerd et al., 1995; Murakami et al., 1997; Cornelissen et al., 2006). A cost-induced shift of cortical reorganization from earlier to later stages of visual processing would straightforwardly explain why reorganization in the form of cortical remapping appears to be absent in adult V1 (Sunness et al., 2004; Smirnakis et al., 2005; Wandell and Smirnakis, 2009; Baseler et al., 2011). The hypothesis is also consistent with the task-dependent differences in V1 activation between patients and controls reported by Masuda et al. (2008, 2010), who reasoned that the task-dependent responses in the LPZ of V1 must reflect unmasked feedback signals from the extra-striate visual areas, rather than cortical reorganization at the level of V1.

As there are many controversies surrounding the claims of cortical remapping in cats, primates and humans (see Wandell and Smirnakis, 2009), perhaps the most convincing evidence of cortical remapping in adult V1 comes from studies in mice (e.g., Keck et al., 2008). Why would the coding catastrophe not limit cortical remapping in adult mice with binocular retinal lesions? One reason could be that mice do not rely as much on vision as cats, primates and humans do (mice are nocturnal and navigate using mainly their nose and whiskers), and so the costs of cortical remapping may be lower for them. Another reason could be that V1 is in fact a relatively high-level area within the mouse visual hierarchy, such that it

serves relatively late-stage rather than early-stage visual processing. If so, fewer visual processing stages would be adversely affected by plastic changes in V1. In a similar vein, the costs of cortical remapping may be lower in mice if mouse V1 were to feed its information straight to the areas with highly flexible processing capabilities (see e.g., Wang and Burkhalter, 2007), thereby bypassing mid-level processing stages (corresponding to e.g. human visual area V2) that are more constrained in the variety of their inputs. Regardless, there is no doubt that visual cortex in mice is very different from visual cortex in (human) primates, with mouse visual cortex containing for instance no orientation columns, and many fewer extrastriate visual areas (Baker, 2013; Huberman and Niell, 2011), leaving open the possibility that it contains different mechanisms of plasticity.

Claims of cortical remapping have not been limited to visual cortex. In auditory cortex, for instance, 'filling-in' like changes have been observed in the topographic cortical representation of sound frequency (i.e., auditory cortex' tonotopic map) starting weeks after localized lesions were applied to the cochlea of the inner ear (e.g., Robertson and Irvine, 1989). Though the criticisms of cortical remapping in the visual domain may also apply to auditory cortex, it is interesting to note that primary auditory cortex (A1) is a relatively later stage within the auditory processing pathways than V1 is within the visual processing hierarchy. That is, there are many more subcortical stops before auditory information reaches cortex than there are stops leading up to V1, and there are many fewer high-level cortical auditory areas than there are high-level visual areas beyond V1. Indeed, it has been proposed that A1 represents stimuli in a highly task-dependent fashion, thereby affording a relatively high amount of learning-induced plasticity (e.g., Ohl and Scheich, 2005; Polley et al., 2006). Thus, the difference in the degree of cortical

reorganization in A1 and V1 appears to fit well with the idea that the brain prefers to make plastic changes at the later stages of sensory processing.

In conclusion, there appears to be converging evidence to suggest that adult human primary visual cortex is not very susceptible to cortical remapping. Here, we have put forward the hypothesis that this could be due to the costs associated with making changes at the very root of the visual processing hierarchy. It would make more sense for the visual system to make long-term structural adjustments at later stages of visual processing, because the later stages have fewer dependencies that may be adversely affected (Hochstein and Ahissar, 2002; Ahissar and Hochstein, 2004). This principle may not only apply to brain plasticity in the form of cortical remapping in response to bilateral retinal lesions, but also when it comes to various multimodal forms of cortical reorganization.

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