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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, such as the presence of sampling bias and/or the unmasking of task-dependent feedback signals from higher level visual areas, that could also explain the results. 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

Neuroplasticity, primary visual cortex, human adults, costs

1. Cortical Remapping and Neuronal Adaptation

If the retina is damaged in both eyes, primary visual cortex (V1) no longer receives patterned visual 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 por-

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tions of the visual field (as suggested by, e.g., Calford *et al*., 1999, 2000; Chino *et al*., 1992, 1995; Darian-Smith and Gilbert, 1994, 1995; Gilbert and Wiesel, 1992; Kaas *et al*., 1990; Schmid *et al*., 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*., 2007; 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 (Andrews, 1964; Anstis *et al*., 1998; Attneave, 1954; Barlow and Földiák, 1989; Benucci *et al*., 2013; Haak *et al*., 2014a; Ullman and Schechtman, 1982; Wainwright, 1999). 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*., 2007; Kohn, 2007; Wandell and Smirnakis, 2009), but rather on functional interactions, such as mutual inhibition (Barlow and Földiák, 1989; Movshon and Lennie, 1979).

Whereas neuronal adaptation is a firmly established property of the visual system across species and ages (Clifford *et al*., 2007; 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 fMRI activity in V1 following retinal lesions (e.g., Baker *et al*., 2005, 2008; Dilks *et al*., 2009, 2014; Schumacher *et al*., 2008), thereby claiming evidence of large-scale reorganization. However, abnormal activity in itself does not warrant the conclusion that cortical remapping has occurred (Baseler *et al*., 2009; Haak *et al*., 2014b; Masuda *et al*., 2008, 2010; Wandell and Smirnakis, 2009). Cortical remapping may 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, and if they are in-

dependent of the experimental task (i.e., to rule out unmasking of pre-existing top-down signals from higher order visual areas). 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 (Binda *et al*., 2013; Haak *et al*., 2012). 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., Muckli *et al*., 2009; Werth, 2006; but see Haak *et al*., 2014b), 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 (Komatsu, 2006; Pettet and Gilbert, 1992; Ramachandran and Gregory, 1991; 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; cf. Barlow, 1961, 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 V1 in the face of a retinal lesion (De Weerd *et al*., 1995; Komatsu, 2006; Murakami *et al*., 1997; Wandell and Smirnakis, 2009; Zur and Ullman, 2003).

Interestingly, recent work now suggests that neuronal adaptation may also not produce hard-wired changes in V1 over the long-term. Using immersive virtual reality, Haak *et al*. (2014c) 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 (Blakemore and Cooper, 1970; Hirsch and Spinelli, 1970) in adult humans. Just as staring at a waterfall for a prolonged period of time changes the responsiveness 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., Dragoi *et al*., 2000; Graham, 1989; Maffei *et al*., 1973; Movshon and Lennie, 1979; Ohzawa *et al*., 1985). Monitoring for the perceptual consequences of these changes in the responsiveness of orientation-selective neurons, Haak *et al*. (2014c) 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.

2. A Cost Theory of Neuroplasticity

Haak *et al*. (2014c) 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 (Dhruv and Carandini, 2014; Patterson *et al*., 2014; Schwartz *et al*., 2007; Series *et al*., 2009). 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*. (2014c) 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., Ahissar and Hochstein, 2004; Hochstein and Ahissar, 2002). 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*. (2014c) 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, the rapid changes, closer to visual adaptation than permanent remapping, in the early stages of cortical visual processing could soon be undone, as perceptual filling-in is postponed to later stages of visual processing (in line with, e.g., Cornelissen *et al*., 2006; De Weerd *et al*., 1995; Murakami *et al*., 1997) (see Note 1). 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 (Baseler *et al*., 2011; Smirnakis *et al*., 2005; Sunness *et al*., 2004; Wandell and Smirnakis, 2009). 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. These unmasked feedback signals may change the neural code just as low-level plasticity does and also cause the coding catastrophe. But because these are pre-existing feedback signals, the brain likely already has found strategies to deal with them, or their costs outweighed their benefits, just as must be the case for receptive field changes caused by visual attention (Anton-Erxleben and Carrasco, 2013).

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, just as the hierarchical position of V1 could explain the difference in the degree of cortical remapping in mice and man, so does the difference in the degree of cortical reorganization in V1 and A1 appear to fit well with the idea that the brain prefers to make plastic changes at later stages of sensory processing.

If the brain avoids making plastic changes at the very basic level of visual processing, how then should we interpret the tactile and auditory responses in V1 that have been reported in individuals who became blind later in life (e.g., Burton, 2003)? The crucial point is that plasticity is not sustained over the long term when its costs outweigh its benefits, so we hypothesize that for the blind, the benefits of cross-modal plasticity outweigh its costs. In addition, it could be that the absence of visually driven activity leads to the unmasking of signals mediated by pre-existing connectivity between V1 and the brain regions serving touch and audition. If signals using these connections have not changed their code, then there will be nothing to misinterpret at later stages of visual processing, and hence no cost to the unmasking.

How likely is it that the principles of plasticity suggested by adaptation also apply to retinal lesions? Theoretically, there is no reason why a similar trade-off between costs and benefits should not exist in both domains. However, ultimately ours is an empirical hypothesis. One piece of evidence in support of it comes from Abe *et al*. (2015). The characteristic feature of prolonged adaptation to lowered levels of visual contrast is its non-monotonicity over time (Haak *et al*., 2014c). Across days, contrast adaptation effects rise then fall and rise again. If the limiting principles of contrast adaptation can be applied to visual processing in the face of retinal lesions, a similar non-monotonicity should also be visible following lesions' onset. Indeed, Abe *et al*. (2015), who examined lesion-induced receptive field changes in the macaque using chronically implanted multi-electrode arrays, reported that "within the first week after the lesion, there is a transient recovery of visually driven activity in the LPZ, followed by a period of decrease in activity lasting a few weeks and then a return to visual responses with clearly shifted RFs" (Abe *et al*., 2015, p. 2786). Thus, it appears that the non-monotonicity in adaptation strength across time also applies to visual deprivation due to localized binocular retinal lesions. Note that the "return to visual responses with clearly shifted RFs" should then be interpreted as a reflection of newly established filled-in signals that are fed-back from higher-level visual cortex, rather than genuine V1 plasticity.

3. Conclusion

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 (Ahissar and Hochstein, 2004; Hochstein and Ahissar, 2002). 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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Note

1. This assumes rapid and reversing plasticity in V1, but our cost theory is also consistent with no real plasticity in V1, just unmasking of feedback.

References

Abe, H., McManus, J. N. J., Ramalingam, N., Li, W., Marik, S. A., Meyer zum Alten Borgloh, S. and Gilbert, C. D. (2015). Adult cortical plasticity studied with chronically implanted electrode arrays, *J. Neurosci.* **35**, 2778–2790.

Ahissar, M. and Hochstein, S. (2004). The reverse hierarchy theory of visual perceptual learning, *Trends Cogn. Sci.* **8**, 457–464.

Andrews, D. P. (1964). Error-correcting perceptual mechanisms, *Q. J. Exp. Psychol.* **16**, 104–115.

Anstis, S., Verstraten, F. A. and Mather, G. (1998). The motion aftereffect, *Trends Cogn. Sci.* **2**, 111–117.

Anton-Erxleben, K. and Carrasco, M. (2013). Attentional enhancement of spatial resolution: linking behavioural and neurophysiological evidence, *Nat. Rev. Neurosci.* **14**, 188–200.

Attneave, F. (1954). Some informational aspects of visual perception, *Psychol. Rev.* **61**, 183–193.

Baker, C. I., Peli, E., Knouf, N. and Kanwisher, N. G. (2005). Reorganization of visual processing in macular degeneration, *J. Neurosci.* **25**, 614–618.

Baker, C. I., Dilks, D. D., Peli, E. and Kanwisher, N. (2008). Reorganization of visual processing in macular degeneration: replication and clues about the role of foveal loss, *Vis. Res.* **48**, 1910–1919.

Baker, M. (2013). Neuroscience: through the eyes of a mouse, *Nature* **502**(7470), 156–158.

Barlow, H. B. (1961). The coding of sensory messages, in: *Current Problems in Animal Behaviour*, W. H. Thorpe and O. L. Zangwill (Eds), pp. 331–360. Cambridge University Press, Cambridge, UK.

Barlow, H. (2001). Redundancy reduction revisited, *Network* **12**, 241–253.

Barlow, H. B. and Földiák, P. (1989). Adaptation and decorrelation in the cortex, in: *The Computing Neuron*, R. Durbin, C. Miall and G. Mitchison (Eds), pp. 54–72. Addison-Wesley, Workingham, UK.

Baseler, H. A., Gouws, A. and Morland, A. B. (2009). The organization of the visual cortex in patients with scotomata resulting from lesions of the central retina, *Neuro-Ophthalmology* **33**, 149–157.

Baseler, H. A., Gouws, A., Haak, K. V., Racey, C., Crossland, M. D., Tufail, A., Rubin, G. S., Cornelissen, F. W. and Morland, A. B. (2011). Large-scale remapping of visual cortex is absent in adult humans with macular degeneration, *Nat. Neurosci.* **14**, 649–655.

Benucci, A., Saleem, A. B. and Carandini, M. (2013). Adaptation maintains population homeostasis in primary visual cortex, *Nat. Neurosci.* **16**, 724–729.

Binda, P., Thomas, J. M., Boynton, G. M. and Fine, I. (2013). Minimizing biases in estimating the reorganization of human visual areas with BOLD retinotopic mapping, *J. Vis.* **13**, 13. DOI:10.1167/13.7.13.

Blakemore, C. and Cooper, G. F. (1970). Development of the brain depends on the visual environment, *Nature* **228**(5270), 477–478.

Burton, H. (2003). Visual cortex activity in early and late blind people, *J. Neurosci.* **23**, 4005–4011.

Calford, M. B., Schmid, L. M. and Rosa, M. G. (1999). Monocular focal retinal lesions induce short-term topographic plasticity in adult cat visual cortex, *Proc. Biol. Sci.* **266**, 499–507.

Calford, M. B., Wang, C., Taglianetti, V., Waleszczyk, W. J., Burke, W. and Dreher, B. (2000). Plasticity in adult cat visual cortex (area 17) following circumscribed monocular lesions of all retinal layers, *J. Physiol.* **524**(2), 587–602.

Chino, Y. M., Kaas, J. H., Smith, E. L. 3rd, Langston, A. L. and Cheng, H. (1992). Rapid reorganization of cortical maps in adult cats following restricted deafferentation in retina, *Vis. Res.* **32**, 789–796.

Chino, Y. M., Smith, E. L. 3rd, Kaas, J. H., Sasaki, Y. and Cheng, H. (1995). Receptive-field properties of deafferentated visual cortical neurons after topographic map reorganization in adult cats, *J. Neurosci.* **15**, 2417–2433.

Clifford, C. W., Webster, M. A., Stanley, G. B., Stocker, A. A., Kohn, A., Sharpee, T. O. and Schwartz, O. (2007). Visual adaptation: neural, psychological and computational aspects, *Vis. Res.* **47**, 3125–3131.

Cornelissen, F. W., Wade, A. R., Vladusich, T., Dougherty, R. F. and Wandell, B. A. (2006). No functional magnetic resonance imaging evidence for brightness and color filling-in in early human visual cortex, *J. Neurosci.* **26**, 3634–3641.

Darian-Smith, C. and Gilbert, C. D. (1994). Axonal sprouting accompanies functional reorganization in adult cat striate cortex, *Nature* **368**(6473), 737–740.

Darian-Smith, C. and Gilbert, C. D. (1995). Topographic reorganization in the striate cortex of the adult cat and monkey is cortically mediated, *J. Neurosci.* **15**, 1631–1647.

De Weerd, P., Gattass, R., Desimone, R. and Ungerleider, L. G. (1995). Responses of cells in monkey visual cortex during perceptual filling-in of an artificial scotoma, *Nature* **377**(6551), 731–734.

Dhruv, N. T. and Carandini, M. (2014). Cascaded effects of spatial adaptation in the early visual system, *Neuron* **81**, 529–535.

Dilks, D. D., Baker, C. I., Peli, E. and Kanwisher, N. (2009). Reorganization of visual processing in macular degeneration is not specific to the "preferred retinal locus", *J. Neurosci.* **29**, 2768–2773.

Dilks, D. D., Julian, J. B., Peli, E. and Kanwisher, N. (2014). Reorganization of visual processing in age-related macular degeneration depends on foveal loss, *Optom. Vis. Sci.* **91**, e199–e206.

Dragoi, V., Sharma, J. and Sur, M. (2000). Adaptation-induced plasticity of orientation tuning in adult visual cortex, *Neuron* **28**, 287–298.

Gilbert, C. D. and Wiesel, T. N. (1992). Receptive field dynamics in adult primary visual cortex, *Nature* **356**(6365), 150–152.

Graham, N. V. S. (1989). *Visual Pattern Analyzers*. Oxford University Press, New York, NY, USA.

Haak, K. V., Cornelissen, F. W. and Morland, A. B. (2012). Population receptive field dynamics in human visual cortex, *PLoS ONE* **7**, e37686. DOI:10.1371/journal.pone.0037686.

Haak, K. V., Fast, E., Baek, Y. and Mesik, J. (2014a). Equalization and decorrelation in primary visual cortex, *J. Neurophysiol.* **112**, 501–503.

Haak, K. V., Langers, D. R., Renken, R., van Dijk, P., Borgstein, J. and Cornelissen, F. W. (2014b). Abnormal visual field maps in human cortex: a mini-review and a case report, *Cortex* **56**, 14–25.

Haak, K. V., Fast, E., Bao, M., Lee, M. and Engel, S. A. (2014c). Four days of visual contrast deprivation reveals limits of neuronal adaptation, *Curr. Biol.* **24**, 2575–2579.

Hirsch, H. V. and Spinelli, D. N. (1970). Visual experience modifies distribution of horizontally and vertically oriented receptive fields in cats, *Science* **168**, 869–871.

Hochstein, S. and Ahissar, M. (2002). View from the top: hierarchies and reverse hierarchies in the visual system, *Neuron* **36**, 791–804.

Huberman, A. D. and Niell, C. M. (2011). What can mice tell us about how vision works? *Trends Neurosci.* **34**, 464–473.

Kaas, J. H., Krubitzer, L. A., Chino, Y. M., Langston, A. L., Polley, E. H. and Blair, N. (1990). Reorganization of retinotopic cortical maps in adult mammals after lesions of the retina, *Science* **248**, 229–231.

Keck, T., Mrsic-Flogel, T. D., Vaz Afonso, M., Eysel, U. T., Bonhoeffer, T. and Hubener, M. (2008). Massive restructuring of neuronal circuits during functional reorganization of adult visual cortex, *Nat. Neurosci.* **11**, 1162–1167.

Kohn, A. (2007). Visual adaptation: physiology, mechanisms, and functional benefits, *J. Neurophysiol.* **97**, 3155–3164.

Komatsu, H. (2006). The neural mechanisms of perceptual filling-in, *Nat. Rev. Neurosci.* **7**, 220–231.

Maffei, L., Fiorentini, A. and Bisti, S. (1973). Neural correlate of perceptual adaptation to gratings, *Science* **182**, 1036–1038.

Masuda, Y., Dumoulin, S. O., Nakadomari, S. and Wandell, B. A. (2008). V1 projection zone signals in human macular degeneration depend on task, not stimulus, *Cereb. Cortex* **18**, 2483–2493.

Masuda, Y., Horiguchi, H., Dumoulin, S. O., Furuta, A., Miyauchi, S., Nakadomari, S. and Wandell, B. A. (2010). Task-dependent V1 responses in human retinitis pigmentosa, *Invest. Ophthalmol. Vis. Sci.* **51**, 5356–5364.

Movshon, J. A. and Lennie, P. (1979). Pattern-selective adaptation in visual cortical neurones, *Nature* **278**(5707), 850–852.

Muckli, L., Naumer, M. J. and Singer, W. (2009). Bilateral visual field maps in a patient with only one hemisphere, *Proc. Natl Acad. Sci. USA* **106**, 13034–13039.

Murakami, I., Komatsu, H. and Kinoshita, M. (1997). Perceptual filling-in at the scotoma following a monocular retinal lesion in the monkey, *Vis. Neurosci.* **14**, 89–101.

Ohl, F. W. and Scheich, H. (2005). Learning-induced plasticity in animal and human auditory cortex, *Curr. Opin. Neurobiol.* **15**, 470–477.

Ohzawa, I., Sclar, G. and Freeman, R. D. (1985). Contrast gain control in the cat's visual system, *J. Neurophysiol.* **54**, 651–667.

Patterson, C. A., Wissig, S. C. and Kohn, A. (2014). Adaptation disrupts motion integration in the primate dorsal stream, *Neuron* **81**, 674–686.

Pettet, M. W. and Gilbert, C. D. (1992). Dynamic changes in receptive-field size in cat primary visual cortex, *Proc. Natl Acad. Sci. USA* **89**, 8366–8370.

Polley, D. B., Steinberg, E. E. and Merzenich, M. M. (2006). Perceptual learning directs auditory cortical map reorganization through top-down influences, *J. Neurosci.* **26**, 4970–4982.

Ramachandran, V. S. and Gregory, R. L. (1991). Perceptual filling in of artificially induced scotomas in human vision, *Nature* **350**(6320), 699–702.

Robertson, D. and Irvine, D. R. (1989). Plasticity of frequency organization in auditory cortex of guinea pigs with partial unilateral deafness, *J. Comp. Neurol.* **282**, 456–471.

Schmid, L. M., Rosa, M. G. and Calford, M. B. (1995). Retinal detachment induces massive immediate reorganization in visual cortex, *NeuroReport* **6**, 1349–1353.

Schumacher, E. H., Jacko, J. A., Primo, S. A., Main, K. L., Moloney, K. P., Kinzel, E. N. and Ginn, J. (2008). Reorganization of visual processing is related to eccentric viewing in patients with macular degeneration, *Restor. Neurol. Neurosci.* **26**, 391–402.

Schwartz, O., Hsu, A. and Dayan, P. (2007). Space and time in visual context, *Nat. Rev. Neurosci.* **8**, 522–535.

Series, P., Stocker, A. A. and Simoncelli, E. P. (2009). Is the homunculus "aware" of sensory adaptation? *Neural Comput.* **21**, 3271–3304.

Smirnakis, S. M., Brewer, A. A., Schmid, M. C., Tolias, A. S., Schuz, A., Augath, M., Inhoffen, W., Wandell, B. A. and Logothetis, N. K. (2005). Lack of long-term cortical reorganization after macaque retinal lesions, *Nature* **435**(7040), 300–307.

Sunness, J. S., Liu, T. and Yantis, S. (2004). Retinotopic mapping of the visual cortex using functional magnetic resonance imaging in a patient with central scotomas from atrophic macular degeneration, *Ophthalmology* **111**, 1595–1598.

Ullman, S. and Schechtman, G. (1982). Adaptation and gain normalization, *Proc. R. Soc. Lond. B Biol. Sci.* **216**, 299–313.

Wainwright, M. J. (1999). Visual adaptation as optimal information transmission, *Vis. Res.* **39**, 3960–3974.

Wandell, B. A. and Smirnakis, S. M. (2009). Plasticity and stability of visual field maps in adult primary visual cortex, *Nat. Rev. Neurosci.* **10**, 873–884.

Wang, Q. and Burkhalter, A. (2007). Area map of mouse visual cortex, *J. Comp. Neurol.* **502**, 339–357.

Weil, R. S. and Rees, G. (2011). A new taxonomy for perceptual filling-in, *Brain Res. Rev.* **67**, 40–55.

Werth, R. (2006). Visual functions without the occipital lobe or after cerebral hemispherectomy in infancy, *Eur. J. Neurosci.* **24**, 2932–2944.

Zur, D. and Ullman, S. (2003). Filling-in of retinal scotomas, *Vis. Res.* **43**, 971–982.