Plasticity and Adaptation in Adult Binocular Vision

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Understanding the relationship between changes in sensory perception and functional/structural changes in the brain is a major endeavor in the field of systems neuroscience. Progress in this area holds the potential to reveal how the brain adapts to the demands of a complex and changing environment, as well as to assist with the development of therapeutic interventions to reverse the negative effects of abnormal experience. The cells and circuits that make up the mammalian visual system provide a unique scientific test-bed for studying brain plasticity, thanks to the rich literature on their basic organization and similarity across a range of species. In this minireview, we highlight recent advances in the study of plasticity in adult binocular vision, emphasizing the importance of considering changes that occur over different timescales. We discuss key new insights, significant open questions, and how this research is leading to a broader understanding of the ways that the adult brain maintains a robust ability for adaptation and change.

Introduction

Binocular vision is common to humans and many other mammals. The integration of binocular signals is thus an important focus of visual neuroscience, a major aim of which is to connect work on model animal species with studies of perceptual processes in humans. Binocular processing primarily emerges when neurons receiving input from the two eyes converge onto common cells in the primary visual cortex (Figure 1). Starting over 50 years ago, researchers began examining experience-dependent plasticity in these binocular circuits in cats and primates by selectively occluding one eye for days or weeks during early life, and examining the impact of this manipulation on neuronal anatomy, physiology, and visually-guided behavior [1]. Extensive evidence from this work showed that monocular deprivation caused dramatic alterations to the organization of binocular circuitry. These alterations included anatomical changes in thalamo-cortical projections, as well as an enduring reduction in cortical responsiveness and visual acuity associated with the deprived eye. Such shifts in ocular dominance were not observed following the same manipulation in more mature animals, and were often not reversible by the restoration of normal binocular input. This seminal work thus established that plasticity in binocular integration is part of normal visual development, but becomes restricted after a critical period early in life.

From this historical perspective, current trends in research showing robust experience-dependent changes to binocular perception in adult humans are unexpected [2–13]. But despite the dogma of the ‘critical period’ for plasticity in early life, both long-standing and recent research have revealed that the visual system has a range of different forms of plasticity, many of which are maintained throughout the lifespan. Obtaining a complete picture of the nature of adult brain plasticity, and the implications of that plasticity for perception, is an on-going and demanding goal. Here, we aim to bring together several differing lines of evidence for plasticity in adult binocular vision. We emphasize what can be learned by examining perceptual changes over different timescales, beginning with rapid phenomena, extending to intermediate durations, and ending with work on long-term effects. Within each timescale, we focus on a particular type of experience-dependent perceptual change that has been characterized in adult humans, examine the relevant literature that informs our understanding of the potential underlying plasticity mechanisms, and highlight key unresolved questions.

A Brief Note on Adult Neural Plasticity

Plasticity is a general term often used for any structural or functional change observed in the brain. By this definition, substrates for plasticity include temporary alterations in the cellular resting membrane potential, short-term and long-term modifications of synaptic strength, and structural modifications of neurons — affecting synapses, axons or dendrites — as well as glial cells. Importantly, any change in perception that occurs as a result of prior experience — and that cannot be attributed to changes preceding sensory transduction, such as tissue damage or pupillary responses — must be a result either of experience-dependent plasticity or, relatedly, of shifts in on-going neuronal dynamics [14]. Key questions in examining the neural substrates of perceptual changes are where the changes occurred, how they happened, and what is the timescale of the changes.

Adult Binocular Vision Undergoes Rapid Sensory Adaptation

Perceptual Effects

Adaptation is a pervasive example of experience-dependent perceptual change, and refers to an often-rapid reduction or modification in sensitivity that follows exposure to a particular stimulus. For example, after viewing a high contrast visual pattern, such as a sine-wave or square-wave grating, for even a few seconds, the detection sensitivity for that pattern (and similar patterns) decreases [15]. While much of the work on adaptation focuses on the perceptual effects of viewing high-contrast patterns, complementary perceptual changes (increases in sensitivity) have been reported following reductions in contrast elicited by wearing contrast-reducing or blurring goggles [16]. Perceptual adaptation effects are typically reported...
to persist for approximately as long as the adaptation stimulus was viewed [17].

The extent to which perceptual adaptation occurs in binocular vision can be assessed with dichoptic adaptation paradigms. For instance, one eye may be adapted to a high contrast pattern, while the other eye views a uniform field (Figure 2A). These paradigms often reveal interocular transfer: pattern detection thresholds become elevated in both eyes, even though one eye saw no pattern at all [15]. When interocular transfer is present, it provides good evidence that some of the neurons contributing to the ultimate percept are binocularly responsive cells in primary or higher-order visual cortex that are capable of changing their response properties over the timescale of exposure [18]. It is important to note, however, that interocular transfer of adaptation is variable, ranging from near 0% (purely monocular adaptation) to almost 100%, depending on the task and properties of the adapting stimulus [19].

**Potential Mechanisms**

Changes in sensitivity, along with other perceptual shifts observed with adaptation, may be supported by a combination of neuronal changes anywhere along the visual processing pathway. Past work on the general neural mechanisms underlying perceptual adaptation, not specific to binocular vision, revealed a diverse set of potential processes operating from milliseconds to minutes. These processes include resting membrane hyperpolarization in the retina and cortex, short-term depression at excitatory thalamo-cortical and cortico-cortical synapses, and strengthening of inhibition [20]. Recent modeling research also suggests that rapid adaptation can occur in recurrently-connected neural networks in the absence of any structural changes, though only on very short timescales (hundreds of milliseconds) [14].

What mechanisms may underlie adaptation of binocular neurons? Hyperpolarization of binocularly-driven neurons (Figure 1A) and depression at excitatory cortico-cortical synapses in primary visual cortex (Figure 1B) are both appealing potential substrates for dichoptic adaptation effects (those involving full or partial transfer of sensitivity changes from one eye to the other). Research further suggests that primary visual cortex, V1, contains competitive binocular circuits for interocular gain control, which

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**Figure 1. Illustration of binocular integration in mammalian primary visual cortex and potential sites of neuroplasticity.**

Visual input to the left (blue) and right (red) eyes is initially processed in the retinas and undergoes partial decussation such that each visual thalamus (LGN, lateral geniculate nucleus) receives input from one hemifield. Thalamo-cortical afferents carrying information from the left and right eyes synapse in a sub-lamina of layer 4 of the primary visual cortex. Their axons remain largely (but not completely) segregated, such that these input layers contain primarily monocularly-driven neurons (LM/RM, left/right monocular). Thalamo-recipient neurons may be pyramidal or spiny stellate, depending on the species. Neurons in the input layers typically project to more superficial layers of cortex, at which point the monocular pathways converge onto common cells, leading to populations of binocularly responsive neurons. Convergence can occur via diagonal ascending connections or via horizontal connections within the same layer [49]. The balance with which the left and right eyes drive binocular neurons is variable: cells may be primarily driven by the left eye, primarily driven by the right eye, or balanced (LB/ RB/BB, left-dominant/right-dominant/balanced binocular). In some mammals (for example primates and cats), these neurons are organized together into ocular dominance columns, and in others (such as mice) there is no clear macro-scale organization. Inhibitory interneurons (inh), illustrated here between LB and RB cells, are thought to mediate interocular gain control [21]. This inhibition may also operate between monocular cells (not shown). In the cortical diagram, axons are illustrated as thin lines and dendrites as thick lines. Beyond layers 1 and 4, specific layers are not labeled, due to the interspecies variability of cortico-cortical connections among mammals [49]. Feedback from higher-order cortex and modulatory signals to V1 are distributed across the cortical laminae (acetylcholine/serotonin; ACh/5HT) [50]. A few key aspects are highlighted as follows: (A) cellular membrane; (B) cortico-cortical synapses; (C) inhibitory interneurons; (D) peri-neuronal nets; (E) neuromodulatory input. Park image by Ohiolove, modified from Vector4Free under Creative Commons Attribution 3.0 License https://creativecommons.org/licenses/by/3.0/.
are essential for stable perception of the world (for example, so that perceived light intensity is similar when viewing with one or both eyes at the same time). Unequal stimulation of the two eyes, as occurs during dichoptic adaptation, may thus also modulate inhibitory interactions. Physiological measurements in cats suggest that interocular gain control is likely mediated by GABAergic interneurons in V1 that provide mutual inhibition between neurons dominated by monocular inputs (Figure 1C) [21]. Recent work in humans also supports the role of V1 in modulating perceptual suppression between the eyes [22].

Current perceptual work suggests that the neural circuits sensitive to interocular differences are themselves capable of rapid adaptation, which results in decreased sensitivity to global interocular differences following exposure to unmatched stimuli in the two eyes [23]. It has been suggested that this type of adaptation may reflect an independently-modulated gain control mechanism for processing differences between the left and right eye images, which has previously been proposed as a useful operation for stereoscopic depth perception [24]. In support of this idea, a recent study in humans [25] found that decreases in resting GABA concentration in V1 correlated with induced changes in interocular dynamics; however, the precise role of inhibitory plasticity in modulating interocular gain at this point is not known. While inhibitory neurons and synapses are capable of experience-dependence plasticity, the mechanisms that govern changes in GABAergic interneurons and synapses are relatively less well understood, in part because of the great diversity of inhibitory cell types [26].

Taken together, the evidence suggests that a combination of rapid intrinsic and synaptic changes across populations of excitatory and inhibitory cells in V1 likely contributes to altered binocular sensitivity at the perceptual level, but exactly how is still an open question. At the network level, it was recently suggested that neuronal adaptation effects in V1 are well-modeled via interactions amongst excitatory neurons that homeostatically maintain their pooled response magnitude via ‘divisive normalization’, in which an individual neuron’s initial input-driven activity is divided by some function of the activity of other neurons in the network [27]. Such population-wide changes could help the visual system adjust to stabilize the statistical structure of neuronal activity amidst on-going environmental variations using a simple Hebbian learning rule. This model accounted for existing data better than more conventional models that assume adaptation only reflects neuron-specific fatigue or gain-modulation. The model did not specifically include binocular adaptation, however, so it remains to be seen whether a similar model would accurately capture the effect of adaptation on binocular interactions, particularly the potential role of inhibition.
On a final note, it is important to observe that the basic perceptual phenomenon of adaptation has been reported to increase for up to an hour and decay for a similar duration [17]. Although such longer-term adaptation has similar effects to short-term adaptation, the neural mechanisms of adaptation over hours may be fundamentally different from adaptation that occurs over seconds and minutes, because the decay times of membrane modifications and short-term synaptic depression may be too brief (though the effect of continuously extending the adaptation period on these neuronal properties is not known). Psychophysical work suggests that the perceptual effects of short-term and longer-term adaptation are dissociable, indicating neuronal mechanisms that are at least partially distinct [28]. One clue comes from a recent physiological study [29] in rabbits, suggesting that spontaneous activity levels in the thalamus can change for over an hour following prolonged adaptation. However, the underlying neuroplastic mechanisms supporting these effects, whether they differ from shorter adaptation effects, and their relationship to V1 are yet to be determined. In the next section, we will examine a specific case of binocular perceptual change recently reported at intermediate timescales (over several hours).

**Adult Binocular Vision Is Altered by a Few Hours of Monocular Deprivation**

**Perceptual Effects**

Eye dominance, also sometimes termed ocular dominance, refers to the extent to which one eye is favored over the other during a binocular task. A spate of recent work has reported robust, temporary modifications of eye dominance in adult humans with typical binocular vision following a few hours of altered experience [4,7–9,11,13]. To examine eye-dominance plasticity, adult participants undergo monocular deprivation for periods ranging from 2–3 hours, while the undeprived eye receives natural visual input (Figure 2B). The primary reported outcome of these studies is a temporary strengthening of eye dominance in favor of the eye that was deprived. This strengthening is notably contrary to the observed effect of long-term monocular deprivation in young animals, in which the deprived or blurred eye becomes weaker.

Unlike measures of acuity or contrast sensitivity, eye dominance is a fundamentally binocular measure, often assessed by showing conflicting images to the two eyes in a paradigm called binocular rivalry. For example, when gratings with equal contrast but orthogonal orientations are presented to each eye, people tend to perceive one pattern at a time, with the perceived pattern alternating every few seconds. The sensory-dominant eye is the eye for which the stimulus tends to perceptually dominate for longer. Importantly, there is no clearly established link between eye dominance and the strength of neuronal ocular dominance; in particular, sensory eye dominance is not correlated to monocular measures of contrast sensitivity or brightness perception, and thus may selectively reflect suppressive interocular interactions [30].

Given the qualitative similarity in time course of eye dominance shifts with longer-term adaptation [11,17], it is worth considering what is currently known about possible overlap in their perceptual effects, specifically, experience-dependent changes in contrast sensitivity in the two eyes. Early work directly examining the influence of brief dichoptic adaptation on binocular rivalry established that adaptation can influence the dynamics of eye dominance [31]. Researchers interrupted a period of binocular rivalry with 60 seconds of dichoptic adaptation to one of the test gratings, and observed that eye dominance shifted away from the adapted eye in the following 30 seconds — similar to the longer eye dominance shifts reported with monocular deprivation. It was proposed that this shift could result from neural adaptation to the grating prior to the locus of interocular suppression, which would disproportionately weaken that eye’s contribution to the rivalrous percept. Whether this reflects a form of monocular adaptation, or just incomplete interocular transfer, is unknown.

Can monocular deprivation be viewed as a form of dichoptic adaptation, in which the adapter is natural visual input, rather than a single pattern (Figure 2A,B)? While natural images are highly variable, they do have characteristic frequency spectra and spatio-temporal correlations. Indeed, a recent meta-analysis [19] indicates that the strength of interocular transfer is strongly affected by the spatial frequency of the adapting stimulus, suggesting a complex pattern for how monocular viewing of natural imagery may affect binocular contrast sensitivity. Despite these similarities, several lines of research suggest that known contrast adaptation phenomena are insufficient to account for eye dominance shifts. For example, one study [7] reported no monocular sensitivity changes following short-term deprivation, whereas others [4,9] found that contrast sensitivity in the deprived eye increases and sensitivity in the undeprived eye decreases. Importantly, the sensitivity reduction in the undeprived eye cannot be explained by monocular contrast adaptation, because the visual input is unchanged, or by transfer from the deprived eye, because the deprived eye’s sensitivity has increased.

Recent work that explored more exotic forms of interocular manipulation, such as monocular phase scrambling (without contrast reduction) [13], and studies that isolated sensory eye dominance changes associated with isoluminant patterns [4,8], suggest that first-order contrast adaptation is not sufficient to account for temporary eye dominance changes. Indeed, the binocular rivalry paradigm itself is known to be affected by many properties of visual stimuli beyond their contrast and is often used to study visual phenomena thought to be mediated by later-stage visual processing. Thus, the eye dominance changes observed following short-term monocular deprivation likely include modified contrast sensitivity as well as changes driven by higher-order patterns present in natural images [8].

**Potential Mechanisms**

To examine potential neural mechanisms of eye dominance changes, one research group measured the neural effects of short-term monocular deprivation using optical imaging and single-unit recordings in macaque V1 [32]. The single-unit recordings revealed a heterogeneous set of changes in response to this paradigm, including both strengthened and weakened responses to the stimulation of the undeprived eye. Yet optical imaging of functional ocular dominance columns suggested that the population-level balance of activity elicited by stimulating the left and right eye returned to baseline soon after short-term monocular deprivation was ended.

Several groups have explored human population-level neural responses in V1 following the same manipulation. Two studies
In amblyopic vision, these deficits persist even after better than 20/20 in one eye (the ‘weak eye’) and deficits in binocular vision vary, individuals have a best-corrected visual acuity of less than 20/20 in both eyes. Amblyopia is a common condition that results from atypical binocular experience early in life, and can persist into adulthood. Amblyopia is not simply a deficit in vision that can be corrected with traditional spectacle lenses. It is a neurodevelopmental disorder that can induce perceptual changes that are substantially more enduring. Much of this research has focused on amblyopia, a condition that can induce perceptual changes that are substantially more enduring.

Another factor to consider is how neurons may change their responses during exposure to natural versus unnatural visual patterns. Measurements from V1 in cats indicate that neuronal tuning properties change when viewing noise patterns versus natural images. If these changes do not transfer completely between the neurons responsive to input from each eye, they may also contribute to shifting measures of eye dominance.

Whether observed population-level neural changes in humans reflect unique forms of cellular plasticity that require a couple of hours to trigger is unknown, because measurements were not conducted after shorter deprivation intervals, 10 minutes for example. Nonetheless, it is worth considering that adaptation of interocular gain control may accumulate relatively slowly and be primarily mediated by GABAergic activity. In addition, the broadband nature of the interocular conflict introduced by monocular deprivation — not just one spatial frequency and orientation, but the full range found in natural images — may be more likely to robustly elicit adaptive re-weighting within interocular suppression circuits, which has not been reported before in dichoptic adaptation studies that use isolated, and typically high, spatial frequencies. Indeed, it has been suggested that the changes characterized in human brain imaging may reflect maintenance of excitatory–inhibitory balance in response to the reduced input from the deprived eye.

Excitatory–inhibitory balance provides an attractive principle; however, more research is needed to gain a mechanistic understanding of these effects.

Adult Binocular Vision Can Be Improved through Long-term Training

Perceptual Effects

Perceptual changes and neuroplasticity have particular relevance for rehabilitation following acquired perceptual deficits. When it comes to rehabilitation, it is essential that perceptual changes are long lasting. Thus, a separate thrust of research on adult binocular plasticity is focused on finding paradigms that can induce perceptual changes that are substantially more enduring.

Much of this research has focused on amblyopia, a common condition that results from atypical binocular experience early in life, and can persist into adulthood. Amblyopia is typically caused by either a large uncorrected refractive error (blurry vision) in one eye or a binocular misalignment (strabismus). While the specific characteristics of amblyopic vision vary, individuals have a best-corrected visual acuity of less than 20/20 in one eye (the ‘weak eye’) and deficits in binocular integration. In fact, with both eyes open, the visual input from the weak eye may be fully or largely suppressed from conscious vision.

In amblyopic vision, these deficits persist even after normal visual input is restored via refractive correction or eye alignment.

Clinical treatments for amblyopia have historically employed monocular deprivation as a therapeutic measure. Specifically, the patient wears an eye patch over the stronger eye, requiring complete reliance on the weaker eye for several hours during daily life, over the course of multiple weeks. Outcome assessments focus on improvement in monocular acuity in the weaker eye and/or recovery of binocular function. However, eye-patch training has been shown to be less effective after early childhood.

In pursuit of treatments that can better address amblyopia in adult patients, a large body of work has employed strategies designed to elicit perceptual learning — improvements in detection and discrimination that occur with repeated practice of a task. While adaptation may temporarily decrease detection sensitivity to the adapting pattern following repeated exposure, the goal of perceptual learning is typically to increase sensitivity. Successful learning paradigms in adults involve intensive, multi-day practice protocols with thousands of trials, and skills acquired can be maintained for at least a year following the end of training.

Adult amblyopia training strategies have employed both monocular and binocular perceptual learning paradigms. In monocular training, the stronger eye is occluded while participants perform intensive, multi-week perceptual training of the weaker eye, using tasks such as contrast detection or orientation discrimination. These paradigms result in consistent improvements in acuity of the weaker eye (1.5–2 eye chart lines), with long-term retention similar to perceptual learning in non-amblyopes. Other studies have examined perceptual learning approaches using binocular tasks that encourage the eyes to work together, rather than train the weak eye alone.

To prevent complete interocular suppression in these ‘dichoptic training’ paradigms, the stimulus contrast for the stronger eye is reduced. For example, participants might play a video game in which success depends on combining high-contrast elements shown to the weaker eye with low-contrast elements shown to the stronger eye. To prevent complete interocular suppression in these ‘dichoptic training’ paradigms, the stimulus contrast for the stronger eye is reduced.

Importantly, several studies show that improvements with training can transfer to novel stimuli and tasks; this perceptual transfer is essential if training gains are expected to provide useful visual improvements in day-to-day life.

Potential Mechanisms

Long-term recovery from amblyopia in adulthood must rely on neuroplastic mechanisms that either alter or compensate for abnormal neural circuits that developed in early life. Notably, while the development of amblyopia in human children somewhat mirrors the effects of monocular deprivation in animal studies (described in the Introduction), current research suggests that the neural effects of blur and strabismus in early life differ from the effects of complete deprivation. Specifically, measurements from the primary visual cortex of non-human primates reared to model amblyopia suggest only a weak shift in neuronal ocular dominance preferences in primary visual cortex — more notable effects include an overall reduction in binocularly responsive neurons, as well as abnormal monocular receptive fields for the weaker eye.

Consistent with this observation, physiological measurements in primates suggest that the visual cortex of adult amblyopes is characterized not only by reduced excitatory drive from the weaker eye, but also...
by active, ongoing interocular suppression of the weaker eye, perhaps reflecting asymmetric gain control [39]. Primate amblyopia models have also revealed changes in tuning and interocular suppression beyond V1 [40].

How might perceptual learning paradigms alter amblyopic neural circuits to create enduring perceptual improvements? Animal research suggests that large-scale structural reorganization is limited in the adult brain due to the maturation of extracellular structures, such as perineuronal nets (Figure 1D) [41]. Nonetheless, long-lasting functional and anatomical changes in synaptic strength — as well as formation of new, enduring synapses — have been observed in the adult mouse neocortex [42]. It is an appealing idea that the new therapies for adult amblyopia function by moderating existing excitatory and suppressive neuronal connections in V1, which might allow the remaining excitatory drive from the weak eye to be expressed [2]. However, correlations between perceptual improvements and direct measures of synaptic changes in V1 for inputs from the weaker eye have not yet been reported. Synaptic plasticity could also lead to population-level shifts in cell tunings that increase stimulus discriminability and perceptual acuity. Interestingly, such tuning changes have been reported to be stronger in higher-order, as opposed to early-stage, visual areas following perceptual learning of visual orientation discrimination in primates [43]. Indeed, human research supports the idea that some improvements in amblyopic vision with training are not well-matched with the properties of neuronal populations in V1, indicating that these improvements may be better accounted for by changes in cognitive strategy likely mediated by higher-order association cortices [3].

Perceptual learning is also thought to incorporate neuromodulatory signals, such as synaptic acetylcholine, which have been shown to facilitate experience-dependent synaptic plasticity in V1 (Figure 1E) [44]. However, a recent pilot study [45] in amblyopes reported that pharmacologically-induced increases in synaptic acetylcholine did not improve perceptual learning outcomes, even though the same intervention has been shown to improve perceptual learning in non-amblyopes. This result highlights the importance of pursuing basic neuroscientific questions, such as those of neuromodulators on plasticity, both in typical and atypically-developing visual systems to inform future clinical practice.

Before concluding, it is worth considering whether the shorter-lived plasticity phenomena described in the previous sections can play a role in perceptual learning. Certainly, the binocular imbalances produced in both monocular and dichoptic training paradigms likely elicit adaptation and gain control responses, elaborated in the previous sections. A recent interesting study [5] employed a modified version of monocular deprivation, and made the compelling suggestion that repeated bouts of adaptation can ‘accumulate’ over several days and lead to long-term perceptual changes in amblyopia akin to perceptual learning. Other recent work [6] revisiting the efficacy of passive eye patching in adulthood also suggests that long-term patching of the stronger eye in some amblyopic adults can have similar effects to perceptual learning. But two research groups have very recently posted preprints [46,47] revisiting the idea that the opposite strategy, patching the amblyopic eye, can be similarly effective. These results highlight the robust and varied forms of long-term binocular plasticity, and the potential role of physiological context (such as exercise) in facilitating perceptual changes [47]. The diversity of similarly-effective interventions also highlights how far we still have to go in understanding the ways that prolonged changes to perceptual experience influence the neurons and networks subserving binocular vision.

Conclusions
The visual system has to be flexible throughout life in order to accommodate the demands of a dynamic and variable environment. The sheer diversity of neuroplastic mechanisms and the scope of the neural networks supporting perception necessitate careful interpretation when linking perceptual changes to neural mechanisms. Here, we briefly summarize key preliminary insights and open questions derived from current work in binocular vision.

First, a distinction can be drawn between adaptation-type plasticity that occurs on timescales of milliseconds to minutes, and learning-type plasticity that occurs on timescales of weeks [3,15,17,36]. Adaptation-type plasticity likely reflects the ability of the visual system to optimize performance within the typical variations of natural environments in an on-going manner. Learning-type plasticity reflects the fact that the adult visual system maintains the ability to acquire new perceptual expertise based on extensive experience with specific visual tasks. A survey of the current literature highlights a notable gap in our understanding of the underlying mechanisms of perceptual changes that occur at intermediate timescales. Are these changes adaptation-like, learning-like, or do they have unique functional and perceptual properties? At what point do repeated perceptual experiences transition to long-term, learning-like retention? Experiments in humans and animals that perform the same manipulation and test the same outcomes across multiple timescales can provide unique insight into the transition and trade-offs between adaptation and learning.

Second, current research is revealing how the exact pattern of visual stimulation may impact the neuroplastic response of the visual system [2,19]. Natural images are highly variable, but also contain a number of statistical regularities, both in terms of their monocular and binocular patterns [24,33]. It is appealing to consider that the perceptual changes elicited by controlled lab stimuli may be limited relative to the changes elicited by more natural variations in visual input used in recent work. In addition to timing differences, the patterns of results obtained in dichoptic adaptation and short-term monocular deprivation paradigms may reflect differences in intrinsic properties or cortico-cortical interactions amongst neurons that favor either the pooling or re-balancing of binocular inputs. Emerging virtual and augmented reality-based techniques [5,13] may soon enrich our understanding of how the adult visual system, in concert with other brain systems, maintains an ability to change under natural viewing situations.

Finally, network-level modeling analyses are currently allowing researchers to examine the necessity and sufficiency of certain neuronal changes in accounting for perceptual phenomena [14,27]. This work highlights the fact that counter-intuitive phenomena can emerge from neural networks, such as the observation of adaptation without any plasticity. These models also highlight the challenges associated with specifying hypothetical links...
between brain plasticity and observed perceptual changes. In the case of binocular vision, detailed network-level models that emphasize the importance of explaining multiple binocular phenomena at once — such as the extensive model recently proposed by Ding and Levi [48] — may soon help elucidate the dynamics of monocular adaptation, binocular adaptation, and interocular gain control. Such models can also open avenues to explore how dynamics differ in conditions such as amblyopia, and account for recent unexpected results of newly proposed therapies [45]. At this point in the history of binocular vision research, it is no longer sufficient to observe the existence of adult plasticity, but rather we must gain a network-level understanding of where, how, and for how long changes occur.

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