Vision Research 207 (2023) 108212

Contents lists available at ScienceDirect

Vision Research

journal homepage: www.elsevier.com/locate/visres

Is ocular dominance plasticity a special case of contrast adaptation?

Robert F. Hess [a,*], Seung Hyun Min [a,*]

[a] McGill Vision Research, Department of Vision Sciences and Ophthalmology, McGill University, Montreal, Quebec, Canada

ABSTRACT

The visual system can regulate its sensitivity depending on the prevailing contrast conditions. This is known as contrast adaptation and reflects contrast gain changes at different stages along the visual pathway. Recently, it has been shown that depriving an eye of visual stimulation for a short period of time can lead to neuroplastic changes in ocular dominance as the result of homeostatic changes in contrast gain. Here we examine, on the basis of previously published results, whether the neuroplastic ocular dominance changes are just manifestation of the mechanism responsible for contrast adaptation. The evidence suggests that these two visual processes are separate and do not have a common neural substrate.

1. Introduction

Ocular dominance plasticity (termed OD plasticity) is a phenomenon that results from the short-term deprivation of one eye's input in adults (Lunghi et al., 2011). It results in sensitivity changes related to contrast in both eyes (Zhou et al., 2013, 2014; Zhou et al., 2017) as well as timing alternations (Novozhilova et al., 2021) to the input from the previously deprived eye. These changes are short-lived, lasting for only 30–60 min after the cessation of deprivation (Zhou et al., 2013; Novozhilova et al., 2021; Lunghi et al., 2013). They result in a temporary change in the interocular balance (or ocular dominance) that are thought to have a locus in early visual cortex (Zhou et al., 2014; Binda, 2018; Lunghi et al., 2015; Zhou, 2015). This phenomenon is thought of as a unique example of residual brain plasticity limited to ocular dominance in cortical area V1 (Binda, 2018). However, whether this neuroplastic change could just be a special case of contrast adaptation, which is a homeostatic regulation of contrast gain that occurs in V1 (Movshon & Lennie, 1979) and enables the visual system to be optimally sensitive to changes in contrast in an ever-changing environment, remains elusive (Andrews, 1976).

It has been widely known that neurons in both the retina (Shapley & Victor, 1978) and visual cortex (Albrecht et al., 1984) can adapt to the prevailing levels of mean contrast in an image. For instance, after an exposure to a high-contrast environment, the sensitivity of the adapted eye can be diminished (Blakemore & Campbell, 1969; Pantle & Sekular, 1968). Conversely, exposure to low-contrast stimuli can increase the sensitivity of the adapted eye (Zhang et al., 2009). This mechanism ensures that the perceptual contrast of our visual world remains relatively constant even though the mean contrast changes over space and time. The effect of short-term monocular deprivation could be thought of as a special form of contrast adaptation where the prevailing mean contrast falls to zero (opaque patch) or close to zero (translucent patch) in the affected eye. After the contrast conditions of one eye changes, its contribution will be automatically adjusted and, hence its contribution to binocular vision may change. There is certainly a case to be made that the so-called ocular dominance plasticity changes that have been described (Lunghi et al., 2011; Zhou et al., 2013, 2014, 2017; Lunghi et al., 2013; Binda, 2018; Chadnova et al., 2017) may follow as a simple consequence of what we already know about the visual system's contrast adaptation capacity. In this review, we explore whether this is in fact likely to be the case. We examine the properties of OD plasticity (summarized in Fig. 1) against a number of predictions based on our understanding of contrast adaptation. A closer look at the literature suggests that the differences between contrast adaptation and OD plasticity are too numerous to expect a common explanation/site of action for these two phenomena.

1.1. Measurement conditions

A typical experiment from an adaptation study ensures that either both eyes are adapted or one eye is adapted while the other is occluded. After adaptation, the visual effect is tested either under binocular viewing (if adaptation was binocular) or monocular viewing (if adaptation was monocular). However, in studies of changes in ocular dominance plasticity resulting from short-term monocular deprivation, the eye that is not deprived (equivalent to nonadapted eye in contrast adaptation) remains open and the measurement pertains to the balance between the eyes, which is an interocular measurement. Therefore, there is a fundamental difference in the way contrast adaptation and

* Corresponding authors.
E-mail addresses: robert.hess@mcgill.ca (R.F. Hess), seung.min@mail.mcgill.ca (S. Hyun Min).

https://doi.org/10.1016/j.visres.2023.108212
Received 1 September 2022; Received in revised form 15 November 2022; Accepted 15 December 2022

ocular dominance plasticity effects are induced and measured. Contrast adaptation can be measured both monocularly (one eye adapted) and binocularly (both eyes adapted), whereas ocular dominance plasticity has to be measured in an interocular fashion that compares the measurement value of the deprived eye to that of the non-deprived eye.

2. Other methodological issues

Induction. Short-term monocular deprivation has been achieved by either occlusion (Lunghi et al., 2011; Zhou et al., 2013) or spatial filtering (Zhou et al., 2014, 2017). In the occlusion case, this could be with an opaque or a translucent patch, the former involving a deprivation of both pattern and mean luminance while the latter attenuates the

Parameter	Contrast adaptation	OD plasticity	Agreement
Measurement protocol	Binocular or one eye adapted, the other occluded	One eye deprived, the other eye open	✗
Spatial frequency	Bandpass (Blakemore & Campbell, 1969)	Lowpass (Zhou, Reynaud & Hess, 2014)	✗
Orientation	Bandpass (Blakemore & Campbell, 1969)	Untuned (Zhou, Reynaud & Hess, 2014)	✗
Phase	Independent (Webster & Miyahara, 1997)	Independent (Zhou, Reynaud & Hess, 2014) Dependent (Bai et al., 2017)	✓✗ Task dependent
Interocular transfer	Same polarity (Bjorklund & Magnussen, 1981)	Opposite polarity (Zhou, Clavagnier & Hess, 2013)	✗
Duration of adaptation	Strong dependence (Greelee, Georgeson, Magnussen & Harris, 1991)	Weak dependence (Min, Baldwin, Reynaud & Hess, 2018; Min & Chen et al., 2022)	✗
Storage	Some (Thompson & Movshon, 1978)	None (Min et al., unpublished)	✗
Spacing effect	Enhancement (Magnussen & Greenlee 1986; Magnussen & Johnsen, 1986)	None (Min, Baldwin & Hess, unpublished)	✗
Temporal	Processing delay (Magnussen & Greenlee 1986) for contrast adaptation predicts speeding up for contrast adaptation	Processing delay (Novozhilova et al., 2020)	✗
Colour	Selective (Goddard et al., 2019)	Unselective (Zhou et al., 2017)	✗
Incremental sensitivity	Elevated at low contrasts for adaptation (Greenlee & Heitger, 1988) or raised at low contrast for deprivation (Kwon et al., 2009)	Unaffected (Wang et al., 2020; Min et al., unpublished)	✗

Fig. 1. Contrast adaptation and ocular dominance (OD) plasticity. A cross indicates no agreement between contrast adaptation and OD plasticity. A combination of a cross and a check indicates that there are both agreement and disagreement between contrast adaptation and OD plasticity (ex. Phase).

mean luminance by about 20% and only allows through only very low spatial frequencies at much reduced contrasts. Contrast adaptation involves viewing either high or low contrast spatially narrowband adapting stimuli or involves the spatial filtering of contrast in broadband images (Bao & Engel, 2012; Bao, 2013). In the general sense these two procedures (short term monocular deprivation and contrast adaptation) are similar in that they both involve viewing stimuli of reduced contrast. It could be argued that unlike short term monocular deprivation, contrast adaptation never involves the case where the contrast is zero (as it does with opaque occlusion). The importance of this mitigated by the fact that short-term monocular deprivation studies have found quite similar deprivation effects for opaque occluders, translucent occluders (Zhou et al., 2013) and spatial filtering (Zhou et al., 2014). This issue of zero contrast is discussed later.

Quantification of induced effects. A number of different methodologies have been used to quantify the effects of short-term monocular deprivation. The main ones being binocular rivalry (Lunghi et al., 2011), binocular phase combination (Zhou et al., 2013), contrast thresholds (Zhou et al., 2013) and contrast discrimination (Wang et al., 2020). Notwithstanding the fact that binocular rivalry was used in the first report of short-term monocular deprivation (Lunghi et al., 2011), binocular phase combination is the method of choice (Min et al., 2021). This involves measurement of the relative contribution that each eye's input makes to the binocularly fused percept. Stimuli of equal but opposite phase are seen by each eye and the input weights are determined by the extent to which one eye's input has to be reduced to equalize the contribution from each eye. Interocular contrast is varied to achieve this. A more detailed account can be found elsewhere (Min et al., 2021). Contrast adaptation effects have been quantified in terms of contrast thresholds (Blakemore & Campbell, 1969) and contrast discrimination measurements (Greenlee & Heitger, 1988).

3. Spatial frequency

Fig. 1 summarizes the main parameters that have been investigated for both phenomena. The tuning for stimulus spatial frequency was one of the initial most striking features of contrast adaptation (Movshon & Lennie, 1979; Blakemore & Campbell, 1969) and provided a strong link to the known spatial tuning of cells in the visual cortex, particularly simple cells in striate cortex (Movshon & Lennie, 1979). A bandwidth of approximately one octave was found and it was shown to be independent of spatial frequency in as far as similar tuning in octaves was found at a wide range of stimulus spatial frequencies (Movshon & Lennie, 1979; Blakemore & Campbell, 1969), suggesting a self-similar array of underlying spatial detectors exhibiting contrast adaptation. *The contrast adaptation prediction would be that monocular deprivation within a restricted spatial passband should only alter ocular dominance for stimuli within the deprived passband.* The spatial dependence of OD plasticity does not follow this prediction. It is not spatially-tuned in the same way. For instance, deprivation of high spatial frequencies (lowpass filtering) appears to be particularly important, whereas the deprivation of low spatial frequencies is relatively unimportant (i.e. high-pass filtering) in inducing a shift in changes in OD plasticity (Zhou et al., 2014). No shift in OD plasticity was observed after the removal of low spatial frequency components even when it was measured with visual stimuli at a low spatial frequency. This represents an important difference between the two phenomena. Contrast adaptation is spatial frequency bandpass in its tuning, whereas OD plasticity from monocular deprivation is not.

4. Orientation

Another equally important property of contrast adaptation that was initially outlined by psychophysicists (Pantle & Sekuler, 1968; Blakemore & Campbell, 1969) was the dependence of contrast adaptation on the stimulus orientation; contrast adaptation exhibited orientational tuning which was independent of stimulus spatial frequency. The half-width, half height bandwidth was approximately 10° (Movshon & Blakemore, 1973). This too provided a strong link with the underlying cortical physiology where it had been shown that neurons in the lower reaches of the cortical pathway were tuned for orientation (Campbell & Maffei, 1970). The inverse of contrast adaptation is contrast deprivation (binocularly applied) which is effective in altering the gain over a wide range of deprivation durations (Bao & Engel, 2012). This type of deprivation also exhibits orientational selectivity, changes in contrast gain occur that are orientationally tuned (i.e. corresponding to the orientationally specific deprivation) (Zhang et al., 2009). *The prediction on the basis of what we know about contrast adaptation, whether it be to an exposure of abnormally high contrast (*i.e.*, adaptation) or abnormally low contrast (*i.e.*, deprivation) is that OD plasticity changes from monocular deprivation should exhibit comparable orientational tuning if they are governed by common underlying processes. However,* OD plasticity does not appear to exhibit a dependence on orientation. An orientationally-dependent monocular deprivation (e.g., of just vertical contours) results in an OD change for stimuli of orthogonal orientation (i.e., horizontally oriented stimuli) (Zhou et al., 2014; Wang et al., 2017). The implication is that OD plasticity, unlike contrast adaptation/binocular deprivation, is not tuned for orientation, suggesting possibly an early cortical site (e.g., layer $4C_B$ in V1) where there are untuned striate cells (Snodderly & Gur, 1995; Blakemore et al., 1978).

5. Phase

The relative spatial phase of the adapting and test stimuli is not important for contrast adaptation. In fact, subjects are usually directed to move their fixation about on the stationary adapting stimuli so that retinal afterimages are not produced (Blakemore & Campbell, 1969). Also, comparable adaptation can be demonstrated for adapting and test stimuli that are in motion so long as this motion is not too fast (Zaidi & Sachtler, 1991). For images composed on multiple spatial frequency components such as natural images or synthetic textures, it's the amplitude not the phase structure that is important for contrast adaptation (Webster & Miyahara, 1997). *The prediction from the contrast adaptation literature would be that OD post-deprivation changes should also depend on the amplitude and not the phase changes that occur during deprivation.* In terms of OD changes to phase-based deprivation (i.e., phase scrambling of natural images*)*, the results depend on what methodology is used to quantify the change in interocular balance. For binocular combination (i.e., phase combination) (Zhou et al., 2014), OD plasticity is not altered after spatial phase information (i.e., phase scrambling) is monocularly deprived. However, for binocular rivalry (Bai et al., 2017), phase scrambling of one eye's image for a period of time does produce a change in the rivalry balance. Thus, the effect of spatial phase disruptions in producing ocular dominance changes is task-dependent, which might suggest that combination tasks reflect mainly bottom-up influences and binocular rivalry, top-down influences (Tong et al., 2006).

6. Interocular transfer

Suppose that one eye is monocularly adapted with a high contrast stimulus during visual adaptation while the other eye remains occluded as is typically done in adaptation experiments (see section *Measurement protocols)*. The adapted eye will experience an increase in its threshold. Surprisingly, the nonadapted eye will also experience a change in its sensitivity in the same direction (Bjorklund & Magnussen, 1981). This phenomenon is known as interocular transfer. The transferred change in the nonadapted eye is known to be less than the direct change experienced by the adapted eye. Also, interocular transfer is also observed after one eye has been deprived of contrast for a period of time while the non-adapted eye remains occluded (Kwon et al., 2009). In this setting, the adapted eye gains sensitivity from the direct effect of adaptation, and the non-adapted eye also gains albeit reduced in magnitude from the transfer) (Kwon et al., 2009). This is thought to reflect the fact that at the site of the adaptation/deprivation in the striate cortex the vast majority of cells are binocular (Dougherty et al., 2019). *The prediction from these monocular contrast adaptation/deprivation effects is that if the same underlying mechanism is also responsible for OD plasticity, then there should be binocular transfer resulting in a similar but reduced effect for vision through the previously nonadapted/nondeprived eye.* However, monocular deprivation of one eye produces a change in the ocular dominance for a short time afterwards that involves not only strengthening the effectiveness of the previously deprived eye but also reducing the effectiveness of the previously nondeprived eye. Both psychophysical (Zhou et al., 2013; Reynaud et al., 2020) and electrophysiological studies (Chadnova et al., 2017) that monitor monocular sensitivity changes, show that OD plasticity involves a *reciprocal change* of sensitivity to the inputs of each eye in the cortex. The deprived eye's inputs are strengthened and the non-deprived eye's inputs are weakened. For instance, as one eye's input is deprived over the course of one hour, the non-deprived eye's contrast sensitivity deteriorates over the same time period (Reynaud et al., 2020). This is a striking difference between OD plasticity changes and contrast adaptation; the sign of the interocular transfer is different for the former.

7. Duration

The effect of the duration of adaptation seems to be uniquely different between contrast adaptation and OD plasticity. Studies have shown that the duration of contrast adaptation linearly increases the magnitude of contrast adaptation (Zhang et al., 2009; Bao & Engel, 2012; Magnussen et al., 1991; Greenlee et al., 1991). It is interesting that both short (seconds) and long (hours) durations of contrast adaptation affect the degree of adaptation. This also seems to be the case for both high- (Greenlee et al., 1991) and low-contrast (i.e., contrast deprivation) adaptations (Zhang, 2009; Bao, 2013). On the other hand, Min et al. (2018) has shown that there is at best only a very weak dependence of OD plasticity on the duration of monocular deprivation; a 20-fold increase in the duration of the deprivation results in less than a 2-fold change in OD plasticity (Min et al., 2018). The duration dependencies of these two effects are strikingly different. In a more recent study (Min et al., 2022) using a more sensitive psychophysical task we found that a 10-fold increase in the deprivation period result in only a 3-fold difference in the OD change. In sum, the duration dependence of contrast adaptation is much stronger than the duration dependence of OD plasticity.

8. Storage

Contrast adaptation exhibits a degree of "storage" of the after-effect. For instance, Thompson and Movshon (Thompson & Movshon, 1978) measured the contrast threshold for the detection of orientation before and after contrast adaptation. They observed an increase in the threshold immediately after the adaptation which decays over time. However, they noted that the adaptation effect maintained itself after a period in the dark following adaptation. Their study shows that the increase in contrast threshold after adaptation exhibits a degree of "storage" in the dark and the expected decay can be revealed later upon testing in the light. *The prediction, on the basis of there being a common basis for contrast adaptation and OD plasticity, would be that OD plasticity should also exhibit a "storage" effect if a period of darkness follows the initial deprivation.* Our data (Min et al., unpublished; data plotted in Fig. 2A) shows that changes in eye dominance after short term monocular deprivation are not maintained in the dark and so do not exhibit "storage" in the way the contrast adaptation after-effect does.

In our preliminary study, six subjects were tested in two conditions. In the first condition, the dominant eye of the subjects was patched for 2 h and tested when the patch was removed. In the second condition, the subjects were also patched for 2 hrs and then put in a dark room for 1 hr after which they were tested. We used a binocular phase combination task and measured eye balance before patching (i.e., baseline) and at 0, 3, 6, 12, 24, 48 and 96 min after patching. We determined changes in sensory eye balance by computing the difference in eye balance between before and after patching. The data shown in Fig. 2A is the area under curve (changes in eye balance over 96 min); the higher the value, the larger the change in eye balance over time. We used a paired *t*-test to compare the areal measure between the two conditions and found a significant difference: $t(5) = -3.74$, $p = 0.014$, suggesting that the after-effect had significantly dissipated in the dark. Therefore, our results show that the patching effect does not demonstrate storage in darkness in the way that contrast adaptation does (Thompson & Movshon, 1978). Instead, it decays in the darkness over time as it does in the light. Therefore, it seems that the sensitivity changes from contrast adaptation and monocular deprivation also differ in how they interact with subsequent testing.

9. The spacing effect

Previous studies (Magnussen & Johnsen, 1986; Magnussen &

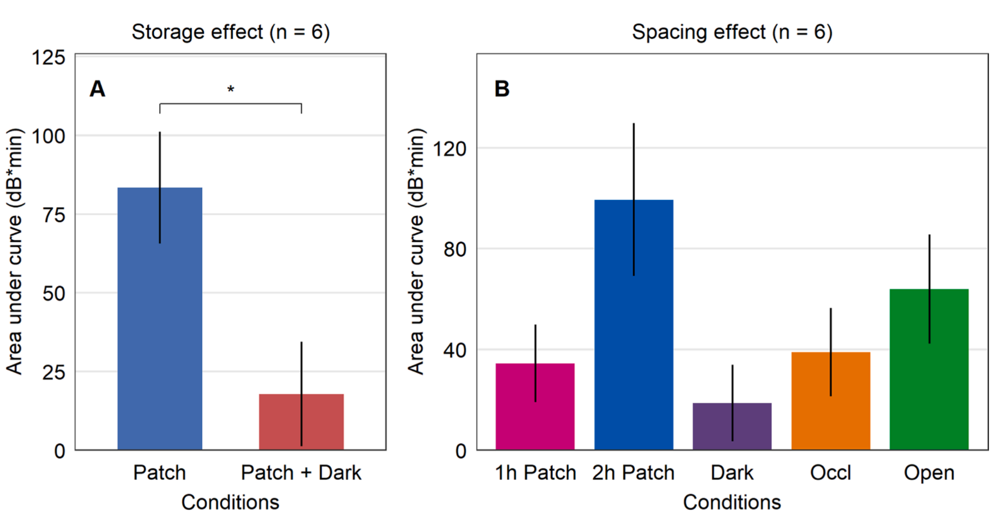

Fig. 2. Unpublished data on OD plasticity. (A) Storage effect for OD plasticity. Comparison between 2 h Patching and 2 h Patching followed by 1 h in the dark. There is a significant difference ($t(5) = -3.74$, $p = 0.014$) between the two groups, suggesting that the changes in eye dominance over time significantly dissipated in the dark (i.e., no storage effect in the dark). (B) The spacing effect for OD plasticity. Crimson block represents 1 h patching. Blue represents 2 h patching. Purple represents 1 h patching + 30 min binocular deprivation in the dark (i.e., space) + 1 h patching. Orange represents 1 h patching + 30 min binocular occlusion from contrast (i.e., space) + 1 h patching. Green represents 1 h patching + 30 min in normal viewing (i.e., space) + 1 h patching. According to a Kruskal-Wallis test, there is no statistical significance between groups ($\chi^2(4) = 5.65$, $p = 0.23$). If there was a spacing effect, orange, green and purple bars (conditions with 'space') would have induced a larger patching effect than 2 h patching. Thus, there is no evidence for a spacing effect.

Greenlee, 1986) have shown that short-term contrast adaptation exhibit what is called, "a spacing effect". For example, five sessions of adaptation (each 2 min), separated by 1-min recovery (i.e., interrupted adaptation), induce a larger and more long-lasting aftereffect than 10-minute of continuous adaptation. This finding suggests that the 'space' between the adaptation periods potentiated the magnitude of the after-effect for contrast adaptation. *The prediction is that OD plasticity would also exhibit a "spacing effect" if contrast adaptation and OD plasticity have a common basis*. However, our data (Min, Baldwin, Hess, unpublished- data plotted in Fig. 2B) shows no evidence for a spacing effect for OD plasticity.

In our preliminary study, the experimental procedure was similar to that in previous spacing effect studies (Magnussen & Greenlee, 1986; Magnussen & Johnsen, 1986). Subjects were monocularly deprived (i.e., patched) for 1 h, then spent 30 min without a patch (i.e., space), then they were patched for 1 hr again. There were three different conditions for the 'space.' In the first condition, subjects spent the 30 min 'space' in the dark (both eyes were deprived of light and contrast; this is labelled as 'Dark' in Fig. 2B). In the second condition, both eyes of the subjects were occluded and deprived of contrast but not luminance with translucent patches (labelled as 'Occl' in Fig. 2B). In the third condition, the subjects were allowed to view normally without a patch during the 30-min 'space' (labelled as 'Open' in Fig. 2B). As for controls, we patched the same subjects (matched subject design) for 1 and 2 hrs (crimson and blue bars in Fig. 2B). For our measurement, we used a binocular phase combination task. Eye balance was measured before patching (i.e., baseline) and at 0, 3, 6, 12, 24, 48 and 96 min after patching. We determined changes in sensory eye balance by computing the difference in eye balance after patching with respect to baseline. The data shown in Fig. 2B is the area under curve (integrated changes in eye balance over 96 min). We observed that the patching effect from 2 h of deprivation was slightly larger than that for conditions with 30-min 'space' (Fig. 2B) although there was no significant difference between the groups (Kruskal-Wallis test, $\chi^2(4) = 5.65$, $p = 0.23$). If there was a spacing effect, we would have observed a larger shift in eye dominance relative to the 2 hrs of continuous patching for the three conditions with the 'space.' Therefore, it seems that the 'space' between the patching sessions does not magnify the changes in eye balance, as it does for contrast deprivation (Magnussen & Greenlee, 1986; Magnussen & Johnsen, 1986).

10. Temporal frequency

Contrast adaptation is associated with not only a reduction in contrast gain but also a response delay which is reflected in increased reaction times (Menees, 1998). This response delay, similar to the contrast gain change, is tuned for spatial frequency. The finding that when contrast gain is reduced (as in the case of adapting to a high contrast), there is an associated temporal delay, is not unexpected, as processing dynamics are known to reduce at low stimulus contrast (Albrecht, 1995). *If OD plasticity was simply due to contrast adaptation (albeit, involving an increase in contrast gain as the result of a lack of stimulation), then one would expect OD plasticity to be associated with a speeding up of the response of the patched eye (*i.e.*, the opposite of the high contrast adaptation condition). Novozhilova et al.* (Novozhilova et al., 2021) *showed that the deprived eye's response is delayed which is the opposite of what would been expected on the basis of the gain changes associated with adapting to zero contrast. Thus, the temporal consequences that result from one eye being patched (OD plasticity) are the opposite of that expected on the basis of the gain changes associated with contrast adaptation (in this case adapting both eyes to zero contrast).*

11. Colour

The contrast adaptation effects from achromatic and chromatic stimuli are complex in that they show asymmetries and depend on whether threshold or suprathreshold measures are used (Magnussen & Johnsen, 1986; Menees, 1998). In general, both the effects of contrast adaptation (Heinrich & Bach, 2001) and OD plasticity (Zhou et al., 2017) are reduced for chromatic compared with achromatic stimuli. More specifically, after contrast adaptation to achromatic stimuli, detection thresholds for chromatic test stimuli are unaffected (Heinrich & Bach, 2001). The results for OD plasticity are quite different; after monocular deprivation of achromatic image content, there are comparable OD changes for achromatic as well as chromatic stimuli (Zhou et al., 2017). Thus, the selectivity of the effects for chromatic and achromatic inducing stimuli are very different for OD plasticity compared with contrast adaptation. Contrast adaptation exhibits chromatic selectivity, whereas OD plasticity does not.

12. Incremental sensitivity

A short-term (milliseconds to 10 min) contrast adaptation after viewing a high contrast stimulus increases contrast detection thresholds (Blakemore & Campbell, 1969) and contrast incremental thresholds in the low contrast range (Greenlee & Heitger, 1988; Ross & Speed, 1991; Gold et al., 2000; Heinrich & Bach, 2001; Gardner et al., 2005; Pestilli et al., 2007). A long-term (i.e., 4 hrs) contrast deprivation using a translucent patch, which is the opposite of high-contrast adaptation, reduces contrast incremental thresholds in the low contrast range. Therefore, exposure to both abnormally high and low contrasts affects contrast gain in different directions with expected alternations to incremental sensitivity in the low contrast range, suggesting possibly a similar underlying regulatory mechanism. Changes in contrast incremental sensitivity have been examined in the context of OD plasticity. For example, Wang et al. (Wang et al., 2020) assessed contrast incremental sensitivity after depriving one eye with an opaque occluder for 2.5 hrs and showed that there was no change in incremental sensitivity of the deprived eye after the patch was removed. However, Wang et al. (Wang et al., 2020) did show a change in the degree of interocular suppression as assessed with continuous flash suppression using an opaque patch. In addition, Kwon et al. described an almost complete interocular transfer for the contrast deprivation effect in terms of incremental sensitivity, whereas the results of Wang et al. for OD plasticity show no direct or transferred effect for incremental sensitivity. Recently, we have extended measurements to a range of different timepoints after patch removal for both deprived and nondeprived eyes using a translucent patch (Fig. 3) and replicated the Wang et al conclusion for an opaque patch that the incremental sensitivity of the deprived eye (OD plasticity) does not significantly change from baseline as the result of short-term patching (Min et al., unpublished). What this suggests is that the change in eye dominance as a result of patching, which defines OD plasticity, may be fundamentally different from contrast adaptation; it cannot be simply explained by a change in the response gain of the type that Kwon et al. (Kwon et al., 2009) showed after contrast deprivation. This seemingly contradictory result may be reconciled in the following way. The contrast deprivation effects of Kwon et al. were binocular in nature, as there was no imbalance between the eyes. To illustrate, when the adapted eye was deprived of contrast for four hours as part of the contrast adaptation procedure, the nonadapted eye was entirely occluded throughout the experiment (Kwon et al., 2009). In this set-up, both eyes were deprived of contrast. Conversely, OD plasticity, by definition, involves a monocular deprivation that sets up a binocular imbalance (one eye is nondeprived); this causes an interocular shift, during which the deprived eye experiences a gain in its sensitivity and an enhanced contribution to binocular vision, whereas the non-deprived eye experiences a sensory change in the opposite direction (Zhou et al., 2013; Chadnova et al., 2017; Reynaud et al., 2020). Contrast adaptation and OD plasticity seem to be very different, possibly involving potentially different sites in the brain and underlying mechanisms.

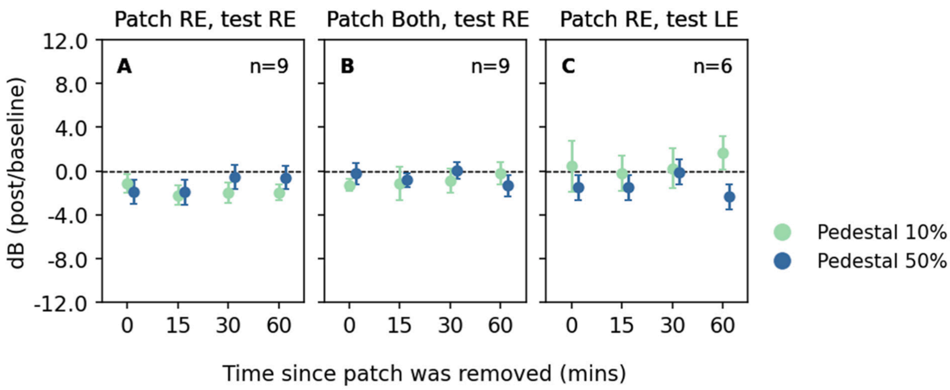

Fig. 3. Unpublished data on incremental contrast threshold. Observers performed a 2-interval forced choice task, during which the subject was asked to identify the interval with the grating at a higher contrast. Two pedestal contrasts (10% and 50%) were used as separate conditions. Subjects performed baseline measurements, underwent translucent patching for 30 min, and performed the same test at 0, 15, 30 and 60 min after the deprivation. According to a three-way ANOVA, there was no significant effect of pedestal contrast, the patched eye(s), and time after patching (p's $>$ 0.05). Horizontal dashed line represents no change in the incremental contrast threshold. Most points reside on or about this line, suggesting no significant shift in the threshold after patching (one sample t-tests, p's $>$ 0.05). (A) Right eye was patched for 30 min, and the right eye was tested before and after patching. (B) Both eyes were patched for 30 min, and the right eye was tested. (C) Right eye was patched for 30 min, and the left eye was tested.

12.1. The zero-contrast argument

As alluded to previously, it could be argued that there is fundamental difference between depriving an eye with a patch, as is often done in the case of short-term monocular deprivation, and reducing the contrast, as is done in contrast adaptation; zero contrast and reduced contrast are not equivalent. While this may be true, the same monocular deprivation effects have been shown using translucent patches that do let through low spatial frequency information, albeit at much reduced contrasts (Zhou et al., 2013). The binocular phase combination task that is used to quantify the resultant imbalances involves the use of a low spatial frequency target (0.3c/d) and the zero contrast argument for a translucent occluder would not strickly apply in this case. Furthermore, the effects of short-term monocular deprivation are of the same form when the deprivation is produced by opaque patches (Zhou et al., 2013), translucent patches (Zhou et al., 2013) or spatial filtering without the use of any patch (Zhou et al., 2014).

12.2. Relationship to the animal deprivation studies

Time scale. Animal studies involving monocular deprivation invariably result in reduced vision in the deprived eye (Hubel & Wiesel, 1970), the opposite of the short-term deprivation discussed here. Although it has previously been uncertain to what extent this is a childhood vs adulthood difference, it has recently been shown that it is more likely due to the period of the monocular deprivation; short deprivation (up to 5 hrs) results in strengthening of the deprived eye's input whereas longer periods of deprivation (e.g. 10 hrs) leads to a weakening of the deprived eye's input (Ramamurthy & Blaser, 2021).

Meta plasticity. Neural plasticity can be modulated by other factors including prior darkness (He et al., 2007; Duffy & Mitchell, 2012). This is an example of meta plasticity and has provided a means of rapidly improving the visual deficit resulting from a prolonged period of prior monocular deprivation in young animals. Unpublished results from the Zhou lab (Min et al., 2022) have recently demonstrated that a period of prior darkness can also modulate the effects of short-term monocular deprivation in human adults.

13. Conclusions

In this paper, we argue that OD plasticity, which results from short-term monocular deprivation, is not simply a consequence of the homeostatic changes that underlie contrast adaptation. Due to the inherent binocularity of the primate visual system, the consequences of depriving one eye of its visual input also depend on whether the other eye has a normal input or not. There are both excitatory and inhibitory circuits that reciprocally regulate the gain of monocular inputs before binocular combination (Meese et al., 2006). OD plasticity is a consequence of a disruption to this binocular control circuit whereas contrast adaptation does not have the same interocular competitive basis. The former is fundamentally a between-eye effect, whereas the latter is fundamentally a within-eye effect. Their neural bases are likely to be different.

CRediT authorship contribution statement

Robert F. Hess: Conceptualization, Methodology, Writing – original draft, Writing – review & editing. **Seung Hyun Min:** Writing – original draft, Methodology, Visualization, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Acknowledgements

We would like to thank Victor Tran for the data collected for Fig. 3. RFH was supported by a Canadian Institutes of Health Research (CIHR) grant #148579. SM was supported by a CIHR doctoral award.

References

Albrecht, D. G. (1995). Visual cortex neurons in monkey and cat: Effect of contrast on the spatial and temporal phase transfer functions. *Visual Neuroscience, 12*(6), 1191–1210.

Albrecht, D. G., Farrar, S. B., & Hamilton, D. B. (1984). Spatial contrast adaptation characteristics of neurones recorded in the cat's visual cortex. *The Journal of Physiology, 347*, 713–739.

Andrews, D. P. (1976). Error-correcting perceptual systems. *Quart. J Exper. Psychology, 16*, 104–115.

Bai, J., et al. (2017). Monocular deprivation of Fourier phase information boosts the deprived eye's dominance during interocular competition but not interocular phase combination. *Neuroscience, 352*, 122–130.

Bao, M., & Engel, S. A. (2012). Distinct mechanism for long-term contrast adaptation. *Proceedings of the National Academy of Sciences of the United States of America, 109*(15), 5898–5903.

Bao, M., et al. (2013). Distinct mechanisms control contrast adaptation over different timescales. *Journal of Vision, 13*(10).

Binda, P., et al. (2018). Response to short-term deprivation of the human adult visual cortex measured with 7T BOLD. *eLife, 7*.

Bjorklund, R. A., & Magnussen, S. (1981). A study of interocular transfer of spatial adaptation. *Perception, 10*(5), 511–518.

Blakemore, C., & Campbell, F. W. (1969). On the existence of neurones in the human visual system selectively sensitive to the orientation and size of retinal images. *Journal of Physiology (London), 203*, 237–260.

Blakemore, C., Garey, L. J., & Vital-Durant, F. (1978). The physiological effects of monocular deprivation and their reversal in the monkey's visual cortex. *Journal of Physiology (London), 283*, 223–262.

Campbell, F. W., & Maffei, L. (1970). Electrophysiological evidence for the existence of orientation and size detectors in the human visual system. *The Journal of Physiology, 207*(3), 635–652.

Chadnova, E., et al. (2017). Short-term monocular occlusion produces changes in ocular dominance by a reciprocal modulation of interocular inhibition. *Science Reports, 7*, 41747.

Dougherty, K., et al. (2019). Binocular Modulation of Monocular V1 Neurons. *Current Biology, 29*(3), 381–391.

Duffy, D. R., & Mitchell, D. E. (2012). Complete darkness reverses maturation of visual cortex and promotes fast recovery from amblyopia. *Nature*. in submission.

Gardner, J. L., et al. (2005). Contrast adaptation and representation in human early visual cortex. *Neuron, 47*(4), 607–620.

Gold, J. M., et al. (2000). Deriving behavioural receptive fields for visually completed contours. *Current Biology, 10*(11), 663–666.

Greenlee, M. W., & Heitger, F. (1988). The functional role of contrast adaptation. *Vision Research, 28*(7), 791–797.

Greenlee, M. W., et al. (1991). The time course of adaptation to spatial contrast. *Vision Research, 31*(2), 223–236.

He, H. Y., et al. (2007). Experience-dependent recovery of vision following chronic deprivation amblyopia. *Nature Neuroscience, 10*(9), 1134–1136.

Heinrich, T. S., & Bach, M. (2001). Contrast adaptation in human retina and cortex. *Investigative Ophthalmology & Visual Science, 42*(11), 2721–2727.

Hubel, D. H., & Wiesel, T. N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *The Journal of Physiology, 206*(2), 419–436.

Kwon, M., et al. (2009). Adaptive changes in visual cortex following prolonged contrast reduction. *Journal of Vision, 9*(2).

Lunghi, C., Burr, D. C., & Morrone, C. (2011). Brief periods of monocular deprivation disrupt ocular balance in human adult visual cortex. *Current Biology, 21*(14), R538–R539.

Lunghi, C., Burr, D. C., & Morrone, M. C. (2013). Long-term effects of monocular deprivation revealed with binocular rivalry gratings modulated in luminance and in color. *Journal of Vision, 13*(6).

Lunghi, C., et al. (2015). Short-term monocular deprivation alters early components of visual evoked potentials. *The Journal of Physiology, 593*(19), 4361–4372.

Magnussen, S., & Greenlee, M. W. (1986). Contrast threshold elevation following continuous and interrupted adaptation. *Vision Research, 26*(4), 673–675.

Magnussen, S., & Johnsen, T. (1986). Temporal aspects of spatial adaptation. A study of the tilt aftereffect. *Vision Research, 26*(4), 661–672.

Magnussen, S., et al. (1991). Stimulus-specific mechanisms of visual short-term memory. *Vision Research, 31*(7–8), 1213–1219.

Menees, S. M. (1998). The effect of spatial frequency adaptation on the latency of spatial contrast detection. *Vision Research, 38*(24), 3933–3942.

Meese, T. S., Georgeson, M. A., & Baker, D. H. (2006). Binocular contrast vision at and above threshold. *Journal of Vision, 6*(11), 1224–1243.

Min, S. H., et al. (2018). The shift in ocular dominance from short-term monocular deprivation exhibits no dependence on duration of deprivation. *Scientific Reports, 8*(1), 17083.

Min, S. H., et al. (2021). Some psychophysical tasks measure ocular dominance plasticity more reliably than others. *Journal of Vision, 21*(8), 20.

Min, S. H., Wang, Z., Chen, M., Hu, R., Gong, L., He, Z., Wang, X., Hess, R. F., & Zhou, J. (2022 Sep 6). Metaplasticity: Dark exposure boosts excitability and visual plasticity in adult human cortex. *bioRxiv*, 2022-09.

Min, S. H., et al. (2022). Issues revisited: Shifts in binocular balance depend on the deprivation duration in normal and amblyopic adults. *Ophthalmology and Therapy*.

Movshon, J. A., & Blakemore, C. (1973). Orientation specificity and spatial selectivity in human vision. *Perception, 2*(1), 53–60.

Movshon, J. A., & Lennie, P. (1979). pattern-selective adaptation in visual cortical neurones. *Nature (Lond), 278*, 850–852.

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

Novozhilova, S., Reynaud, A., & Hess, R. F. (2021). Short-term monocular deprivation induces an interocular delay. *Vision Research, 187*, 6–13.

Pantle, A., & Sekuler, R. (1968). Size-detecting mechanisms in human vision. *Science, 162*(3858), 1146–1148.

Pantle, A., & Sekular, R. (1968). Size detecting mechanisms in human vision. *Science, 62*, 1146–1148.

Pestilli, F., Viera, G., & Carrasco, M. (2007). How do attention and adaptation affect contrast sensitivity? *Journal of Vision, 7*(7).

Ramamurthy, M., & Blaser, E. (2021). The ups and downs of sensory eye balance: Monocular deprivation has a biphasic effect on interocular dominance. *Vision Research, 183*, 53–60.

Reynaud, A., et al., *Monocular vision is intrinsically unstable: a side-effect of binocular homeostasis.* bioRxiv, 2020.03.17.987362.

Ross, J., & Speed, H. D. (1991). Contrast adaptation and contrast masking in human vision. *Proceedings of the Royal Society of London - Series B: Biological Sciences, 246*(1315), 61–69.

Shapley, R. M., & Victor, J. D. (1978). The effect of contrast on the transfer properties of cat retinal ganglion cells. *The Journal of Physiology, 285*, 275–298.

Snodderly, D. M., & Gur, M. (1995). Organization of striate cortex of alert, trained monkeys (*Macaca fascicularis*): Ongoing activity, stimulus selectivity, and widths of receptive field activating regions. *Journal of Neurophysiology, 74*, 2100–2125.

Thompson, P. G., & Movshon, J. A. (1978). Storage of spatially specific threshold elevation. *Perception, 7*(1), 65–73.

Tong, F., Meng, M., & Blake, R. (2006). Neural bases of binocular rivalry. *Trends in Cognitive Sciences, 10*(11), 502–511.

Wang, Y., et al. (2017). The cortical mechanisms underlying ocular dominance plasticity in adults are not orientationally selective. *Neuroscience, 367*, 121–126.

Wang, M., McGraw, P., & Ledgeway, T. (2020). Short-term monocular deprivation reduces inter-ocular suppression of the deprived eye. *Vision Research, 173*, 29–40.

Webster, M. A., & Miyahara, E. (1997). Contrast adaptation and the spatial structure of natural images. *Journal of the Optical Society of America. A, Optics, Image Science, and Vision, 14*(9), 2355–2366.

Zaidi, Q., & Sachtler, W. L. (1991). Motion adaptation from surrounding stimuli. *Perception, 20*(6), 703–714.

Zhang, P., et al. (2009). Effects of orientation-specific visual deprivation induced with altered reality. *Current Biology, 19*(22), 1956–1960.

Zhou, J., Clavagnier, S., & Hess, R. F. (2013). Short-term monocular deprivation strengthens the patched eye's contribution to binocular combination. *Journal of Vision, 13*(5).

Zhou, J., Reynaud, A., & Hess, R. F. (2014). Real-time modulation of perceptual eye dominance in humans. *Proceedings of the Biological Sciences, 281*(1795).

Zhou, J., et al. (2015). Short-term monocular patching boosts the patched eye's response in visual cortex. *Restorative Neurology and Neuroscience, 33*(3), 381–387.

Zhou, J., et al. (2017). Chromatic and achromatic monocular deprivation produce separable changes of eye dominance in adults. *Proceedings of the Biological Sciences, 284*(1867).