



Review

Critical periods re-examined: Evidence from children treated for dense cataracts



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ABSTRACT

Studies of children treated for dense cataracts afford an opportunity to examine the role of visual experience in driving visual perceptual development. Collectively, the data indicate that there are multiple periods during which deprivation can damage visual development, but their timing and duration cannot be predicted from the normal developmental trajectory. For lower level vision, the deficits are worse in the previously deprived eye if the deprivation had been monocular rather than binocular, but for higher level perception, that pattern reverses, perhaps because of cross-modal neural completion during the deprivation. Emerging neuroimaging evidence suggests that the neural underpinnings of vision after early visual deprivation may be abnormal, even when the deprivation ended shortly after birth and normal behavioural performance has been achieved. The implication is that in the baby with normal eyes, despite poor acuity and contrast sensitivity, visual experience at birth sets up the neural architecture for later refinement.

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Infants can see as soon as they are born but there are serious limitations on their vision. Not only do they have limited acuity (Brown & Yamamoto, 1986) but they are not able to integrate the details of objects into a whole percept (Cohen & Younger, 1984) or discern the direction in which they move (Braddick, Birtles, Wattam-Bell, & Atkinson, 2005). There are rapid improvements over the first few months with the onset of sensitivity to direction of movement and configural cues at 2 months, and a fourfold improvement in acuity by 6 months (Braddick, Atkinson, & Wattam-Bell, 2003; Cohen & Younger, 1984; Mayer et al., 1995). Nevertheless, it takes into adolescence (Golarai, Liberman, Yoon, & Grill-Spector, 2010)—and perhaps even longer (Germine, Duchaine, & Nakayama, 2011)—for all aspects of visual perception to become adult-like.

We have studied the role of visual input in driving these postnatal changes. We have done so by taking advantage of a natural experiment: children born with dense central cataracts that blocked all patterned input to the retina until the

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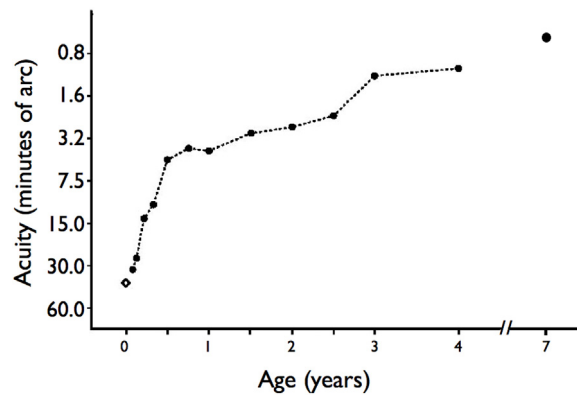


Fig. 1. The development of visual acuity in children with normal eyes. Shown are the size of the smallest stripes for which children show a looking preference at ages from birth (0 years) to 7 years, when acuity reaches adult levels. Acuity is shown in minutes of arc, the size on the retina of the threshold stripes, such that smaller values represent better acuity. Reprinted from [Lewis and Maurer \(2005\)](#).

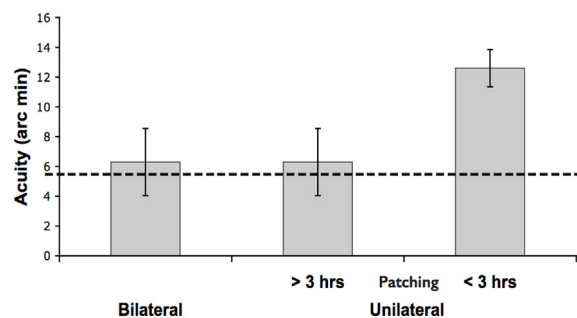


Fig. 2. Mean acuity \pm 1 s.e. at 12 months of age for infants treated for congenital cataracts that were bilateral or unilateral. The results for unilateral patients are divided into those from patients whose fellow eye was patched for at least 3 waking hours from the time of treatment until the first birthday and those for whom there was less patching. The dotted line shows the normal value at 12 months of age. The abscissa shows acuity as the size in minute of arc of the smallest stripes for which there was a looking preference, with better acuity represented by smaller values. Bilateral patients had normalized by the first birthday, as had unilateral patients with extensive patching. Reprinted from [Lewis and Maurer \(2005\)](#).

cataracts were removed and the eyes fitted with compensatory contact lenses. In the patients we studied, this occurred as early as the first month of life to as late as 9 months postnatally. Comparisons of these patients to children with normal eyes allowed us to deduce the role of normal visual experience in driving normal postnatal changes in visual perception. Correlation of the outcome for parents with the duration of the initial deprivation allowed us to draw inferences about the role of patterned input at different points during infancy, as did parallel studies with children born with normal eyes who developed cataracts postnatally. We have studied many aspects of vision in these patients; here I will illustrate our findings with the results for acuity, sensitivity to global motion, and face perception.

1. Acuity

Newborns' acuity has been measured using their tendency to look at something patterned like stripes in preference to a plain grey. Newborns show a robust preference as long as the stripes are large and contrasty: 60 times larger than the limit for adults with normal vision ([Brown & Yamamoto, 1986](#)) and 100–200 times more contrasty ([Brown, Lindsey, Cammenga, Giannone, & Stenger, 2015](#)). Over the first 6 months there is rapid improvement, followed by slower increments until adult levels are reached around 7 years of age ([Ellemberg, Lewis, Liu, & Maurer, 1999](#); [Mayer et al., 1995](#)) (see [Fig. 1](#)). Our studies of infants treated for bilateral congenital cataract indicated that visual experience is necessary for the initial rapid change: when it was missing, no improvement occurred. These infants had surgery to remove the cataracts and 1–2 weeks later, after the eye had healed, received contact lenses to focus visual input. Within 10 min of that time—the first moment of receiving focused patterned visual input, their acuity was like that of a normal newborn ([Maurer, Lewis, Brent, & Levin, 1999](#)). The rapid changes seen in babies with normal eyes had not occurred. But the system had not been dormant during the visual deprivation: after the first hour of visual input, there was a significant improvement in acuity, not seen in control infants. Patients continued to improve faster than control age mates, so that by the first birthday, almost all had acuity within normal limits (see [Fig. 2](#)). This pattern indicates that the system is experience-expectant: during the period of visual deprivation, the patients' nervous system was becoming increasingly ready to respond to visual input, once it was received. As a result,

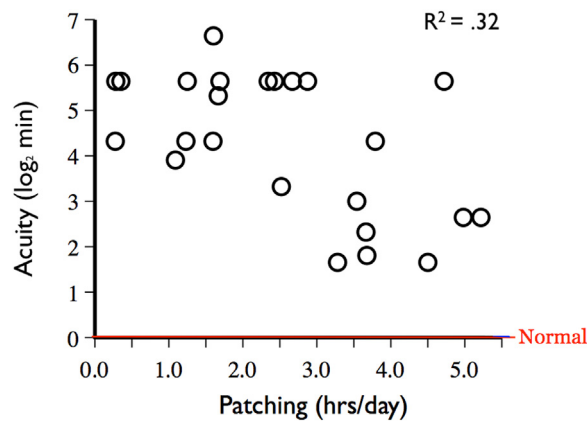


Fig. 3. Final letter acuity of patients treated for unilateral congenital cataract as a function of the amount of patching from the time of treatment until 5 years of age. Each dot represents the final acuity of a single patient. Acuity is shown as the size of the threshold letters in minutes of arc. Values for an adult with normal vision are 0. No patient achieved normal acuity and the final letter acuity was worse when there had been less patching.

patients treated later during the first 9 months of life—who were further behind on the day of treatment—were able to improve at especially accelerated rates in the few months between treatment and their first birthday.

As the evidence for normal acuity at 1 year of age accumulated, it looked like treatment of bilateral congenital cataracts by 9 months of age was early enough to allow complete recovery of vision. However, our longitudinal study indicated that conclusion was premature. The patients treated for bilateral congenital cataract continued to improve until about the second birthday, at which point their acuity reached an asymptote, while that of children with normal eyes improved about 4-fold amount from age 2–7 years. The result is a seemingly permanent acuity deficit in the patients, even when deprivation was for as little as the first 2 months of life (Lewis, Maurer, & Brent, 1995; Maurer & Lewis, 2001), although treatment even earlier can reduce but not eliminate the deficit (Birch, Cheng, Stager, Weakley, & Stager, 2009). We term this a sleeper effect (Maurer, Mondloch, & Lewis, 2007): visual input during the first few months of life, at a time when the infant with normal eyes cannot see fine detail, is necessary for that ability to develop later, after 2 years of age. Studies of children who were born with normal eyes but developed cataracts postnatally indicate that visual input is necessary throughout the 7 years of normal development and even beyond: visual deprivation from dense cataract any time during the first 10 years of life results in an acuity deficit (Lewis & Maurer, 2009). Collectively, these findings indicate that patterned visual input near birth is necessary to set up the neural substrate that can be refined by later input to mediate fine visual acuity. Even when the network is fully formed—when the 7-year-old demonstrates adult-like acuity—it requires input for the next 3 years to be consolidated or crystallized. Only at age 10 has the child reached the end of the critical period during which visual deprivation can damage visual acuity.

Children born with a dense cataract in one eye, paired with a seemingly normal eye, afford an opportunity to study the effects of visual deprivation compounded by unfair competition between the eyes for cortical connections. The treatment is the same—surgical removal of the cataractous lens and fitting of a compensatory contact lens—except that the parents are instructed to patch the “good” eye during much of the babies’ waking time in order to encourage usage of the previously deprived eye. Such patching is prescribed from the time of treatment until about 5 years of age.

Unilateral patients’ initial results for the affected eye mirror those from bilateral patients: their initial acuity, on the day they can first see focused patterned visual input, is like that of newborns, but, as in bilateral cases, it begins to improve at faster-than-normal rates, even during the first hour after treatment (Maurer et al., 1999). But by 1 year of age, the results diverge: the acuity in the previously deprived eye of unilateral patients has normalized, like that of bilateral patients, only if the parents patched the good eye for more than 3 h per day (Lewis et al., 1995) (see Fig. 2). When there was less patching, acuity is roughly two times worse, that is, like the acuity of a 4-month-old with normal eyes (see Fig. 2, cf Fig. 1). These results suggest that unfair competition between the eyes is the major influence on the development of acuity in unilateral cases. The same pattern is evident in later development: the good patchers, like the bilateral patients, continue to improve until about 2 years of age, at which point a deficit begins to be evident (Birch, Stager, & Wright, 1986; Lewis et al., 1995). Late onset deprivation—from traumatic cataract—leads to an acuity deficit in the affected eye until about age 10, as is the case for late onset bilateral cataracts (Lewis & Maurer, 2009). Children treated for unilateral congenital cataract with less patching of the good eye during early childhood end up with even larger deficits, with the size of the deficit strongly correlated with the amount of patching that occurred in early childhood (see Fig. 3). Collectively, these data indicate that the tuning of the neural circuits mediating fine visual acuity depend on early and continued patterned visual input that is balanced between the eyes. When visual experience is blocked to both eyes anytime during the first 10 years of life, acuity will be abnormal. When one eye sees more poorly than the other, as in unilateral cases, it will be even less effective in tuning those neural circuits, as has been demonstrated in animal models (Wiesel & Hubel, 1965). In fact, it is only when treatment is very early—in the

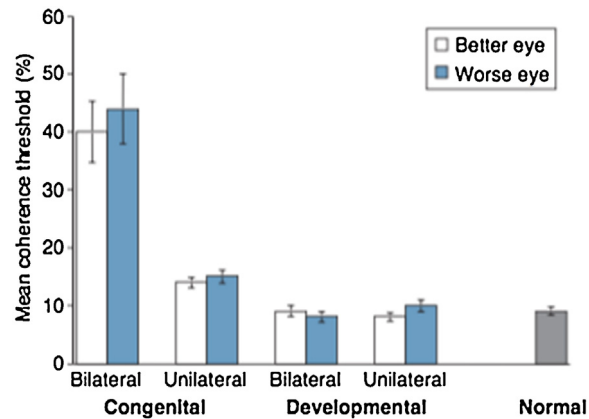


Fig. 4. Mean sensitivity (± 1 s.e) to global motion in controls and adult patients treated for cataract, that were present from birth (leftmost columns) or developed postnatally (middle columns) compared to normal controls tested monocularly (rightmost column). Shown is the threshold level of coherence for the better and worse eyes, defined by acuity and history of eye misalignment and for the previously deprived (worse) and fellow eyes (better) eyes of unilateral patients. Congenital patients had elevated thresholds, unlike developmental patients who were normal, even when the onset of deprivation was later in the first year of life. Unilateral congenital patients had deficits, but they were significantly smaller than those in bilateral congenital patients. Reprinted from Maurer et al. (2005).

first month—and there is aggressive patching of the good eye that the final acuity outcome is favorable in infants born with unilateral congenital cataract (Birch & Stager, 1996).

2. Global motion

Human adults can detect the direction in which small elements of a figure are moving (e.g., the fingers of a chimp or the wings of a bird) but also integrate all the elements of a figure to perceive the overall direction of drift (e.g., the chimp is jumping up into the tree; the bird is flying south). In the lab, sensitivity to the direction of drift, termed *global motion*, is measured with the random dot kinematograms that were introduced by Newsome and Pare (1988). The kinematogram is formed from moving elements, usually dots, all of which can move coherently in one direction, all of which can move in random directions, or some combination of the two. The measure of sensitivity is the percentage of dots that can move in random directions without preventing the observer from perceiving the direction in which the coherently moving dots are traveling, that is, the global direction of motion. To prevent solution from following individual dots, each dot has a limited lifetime: it moves in its specified direction (either the coherent or a random direction) for a short time (e.g., 100 s) and then is reborn on another part of the display. The threshold depends on parameters like the size of the dot, their number, their size and velocity, etc. (Hadad, Schwartz, Maurer, & Lewis, 2015), but in adults with normal eyes, it is typically less than 10%, that is, 90% of the dots can move randomly yet the adult observer can pick out the direction in which the remaining 10% are moving (Hadad, Maurer, & Lewis, 2011). For the first 2 months after birth, infants show no sensitivity to changes in the direction of motion, either when tested behaviorally with habituation/dishabituation (Wattam-Bell, 1996), or with visually evoked potentials (Braddick et al., 2005). Sensitivity is evident from about 2–3 months and then it takes about 12 years for children with normal eyes to reach that level of sensitivity (Hadad et al., 2011).

Despite its postnatal onset in children with normal eyes, sensitivity to global motion does not develop normally in children deprived of visual input by bilateral congenital cataracts. Even when the deprivation was short and testing occurred in adulthood, patients can see the direction of coherent motion in random dot kinematograms only when the level of coherence is at least 40%, compared to a threshold in controls of 8% (Elleberg, Lewis, Maurer, Brar, & Brent, 2002; Hadad, Maurer, & Lewis, 2012). (see Fig. 4) In other words, the global motion signal must be 5 times higher for them to see its direction accurately. These results, like those for acuity, represent a sleeper effect (Maurer et al., 2007): visual deprivation in the first few months of life, when the infant with normal eyes is not yet sensitive to direction of motion, is necessary for the later normal development of sensitivity to global motion. However, the results from patients treated for cataracts that developed postnatally were entirely different: even when the deprivation had started before the first birthday, sensitivity to global motion was entirely normal when tested in adulthood (see Fig. 4). (Since this study was not longitudinal, we do not know if the normal adult threshold for global motion arises from recovery from an earlier deficit or is the endpoint of an entirely normal developmental trajectory). This is the same cohort in whom we observed abnormal acuity results. Thus, early visual input is necessary for the later normal development of both visual acuity and sensitivity to global motion, but the critical periods during which these visual functions can be damaged are very different: 10 years for visual acuity and less than 1 year for global motion.

The results for children treated for unilateral congenital cataract are also surprising: their threshold sensitivity to global motion in the previously deprived eye is not normal, but it is significantly *better* than the thresholds for bilateral patients (Elleberg et al., 2002; Hadad et al., 2012) (see Fig. 4), despite their having much poorer acuity (see above). Thus, the general

principle that monocular deprivation is worse than binocular deprivation unless offset by aggressive patching of the non-deprived eye does not hold for global motion, nor we found in another study for global form (Lewis et al., 2002). What global motion and global form have in common is the integration of small details—likely processed in V1—into a global percept, which is known to require processing in extrastriate cortex, namely V5 in the dorsal stream for global motion and V4 in the ventral stream for global form. Neurons in those areas receive input from both eyes and large swaths of the visual field. The implication is that normal input through one eye from birth is sufficient to set up, or preserve, the requisite neural architecture that can later be driven by either eye to mediate nearly normal development of global motion and global form. Thus, despite the hierarchical nature of the visual system, damage to lower cortical areas does not necessarily lead to the expected level of damage in the higher cortical areas they feed.

In summary, early visual input is necessary for the development of both acuity and global motion, both of which have long developmental trajectories, but the critical periods for damage and the added effects of unfair competition between the eyes are entirely different. They differ in the length of the critical period—10 versus less than 1 year—and the detriment versus benefit of having the deprivation restricted to one eye. This leads to a cautionary note: one cannot predict the critical period during which experience is necessary by knowing the normal developmental trajectory for a sensory capability.

3. Face processing

Newborns are attracted to face-like patterns (Mondloch et al., 1999) and rapidly learn to recognize the mother's face (Pascalis, de Schonen, Morton, Deruelle, & Fabre-Gremet, 1995), but their initial processing of faces appears to be based on isolated features on the external counter and not the spacing among internal features, a configural cue that underlies adults' expert face processing (Maurer, Grand, & Mondloch, 2002). Sensitivity to spacing cues emerges only several months after birth, around 5 months of age, at which point it is already better for upright than inverted faces, as is true in adults (Bhatt, Bertin, Hayden, & Reed, 2005; Hayden, Bhatt, Reed, Corbly, & Joseph, 2007). Sensitivity to the spacing of faces' internal features continues to improve during early childhood, reaching adult levels after 10 years of age (Mondloch, Le Grand, & Maurer, 2002).

Patients treated for bilateral congenital cataracts do not develop this expertise: even when tested as adults, they are less accurate than controls in using small differences in the location of internal features to distinguish among upright faces (de Heering & Maurer, 2014; Le Grand, Mondloch, Maurer, & Brent, 2001; Robbins, Nishimura, Mondloch, Lewis, & Maurer, 2010) (see Fig. 5). (Unilateral patients show a similar deficit when early visual deprivation was restricted mainly to the right hemisphere (Le Grand, Mondloch, Maurer, & Brent, 2003). As would be expected, this deficit affects their memory for faces: they are poorer than controls at remembering famous faces or faces they learned earlier in the session (de Heering & Maurer, 2014). No such deficit occurs when patients are tested with stimuli for which normal adults do not show the same expertise: inverted human faces, monkey faces (upright or inverted), or houses (de Heering & Maurer, 2014; Robbins et al., 2010).

Despite their many deficits, some aspects of perception are normal in patients treated for bilateral congenital cataract. They are normal at face detection (Mondloch et al., 2013), at discriminating among individual faces based on the shape of individual internal features (the eyes and the mouth) or the external contour (Mondloch, Robbins, & Maurer, 2010), at detecting the direction of biological motion, that is, the direction in which key points on an animate being are moving (Hadad et al., 2012), and at discriminating most facial expressions (Gao, Maurer, & Nishimura, 2013; Geldart, Mondloch, Maurer, de Schonen, & Brent, 2002). (Unilateral patients have not been tested on most of these tasks). What these spared abilities have in common is that they are observable in rudimentary form at birth; in other words, they were able to develop during pregnancy in the absence of visual input. Yet our subsequent probing of one of these spared abilities—face detection—indicated that behaviorally normal does not necessarily mean that the underlying neural substrate is normal.

Adults treated for bilateral congenital cataract and controls viewed faces in which each pixel had been altered to be black or white to create what are called a Mooney faces or scrambled versions of these images (see Fig. 6). Their task was to indicate whether each stimulus was a face or not. The advantage of using such Mooney stimuli is that the configuration of the face must be recognized based on the relationships among features, rather than the edges of an individual feature like the nose. As predicted from our earlier results (Mondloch et al., 2013), patients were as accurate as controls in performing the task, and almost as fast. It took them slightly longer than controls to reject a scrambled image as a face but they were just as fast at identifying the face stimuli. However, their event-related potentials on correct trials were highly abnormal. Controls showed the expected pattern of a negative potential around 170 s after the onset of the stimulus that was larger for faces than scrambled images and most prevalent over occipital and parietal electrodes. The same was the case for patients treated for bilateral congenital cataracts, except that all of their potentials were *much* larger (see Fig. 7). The same pattern emerged in a second task in which patients and controls saw pictures of faces, objects, and houses, pushing a button on the rare trials when a butterfly occurred: both the positive deflection to all classes of visual stimuli around 100 s and the negative deflection to faces around 170 s were much larger in patients than in controls, with the size of the amplification highly correlated with the duration of the original deprivation. The implication is that early visual input is necessary to set up, or preserve, the normal neural substrate for face detection but that patients can develop normal detection by an alternative route either involving more neurons and/or a different mapping from retina to behavior. fMRI studies of this same patient cohort while passively viewing faces suggests that it is the connectivity within both the core and the extended face processing network that is altered (Grady, Mondloch, Lewis, & Maurer, 2014).

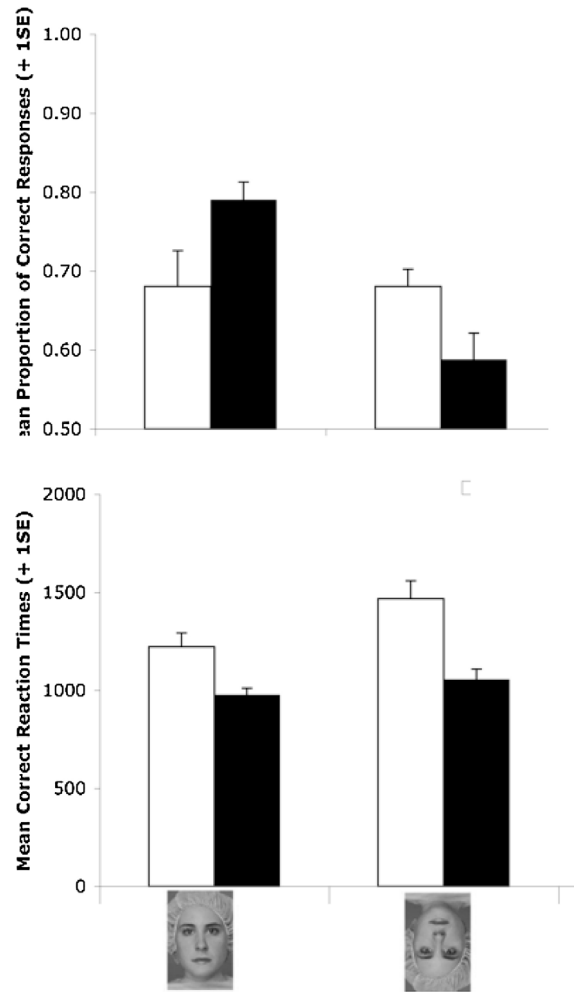


Fig. 5. Mean sensitivity ($\pm 1s.e$) to the spacing of facial features (i.e., second-order relations) in adults treated for bilateral congenital cataracts. Shown are accuracy (proportion correct) and reaction time for upright and inverted faces in patients (open bars) and matched controls (black bars). Patients were less accurate than controls only for upright faces. Reprinted from [de Heering and Maurer \(2014\)](#).



Fig. 6. An example of a Mooney face created by changing each lighter pixel to white and each darker pixel to black (right side) and of a scrambled image created by rearranging the patches in the face. Reprinted from [Mondloch et al. \(2013\)](#).

4. Cross modal integration

In patients with lifelong blindness—the congenitally blind—cortical areas that normally would be specialized for vision respond instead to auditory, tactile, and even linguistic input ([Maurer, Lewis, & Mondloch, 2005](#)). These rewired areas are functional: temporary disruption of neurons in “occipital” cortex by transcranial magnetic stimulation degrades auditory perception, leading to more errors in auditory localization ([Collignon, Lassonde, Lepore, Bastien, & Veraart, 2007](#)). This

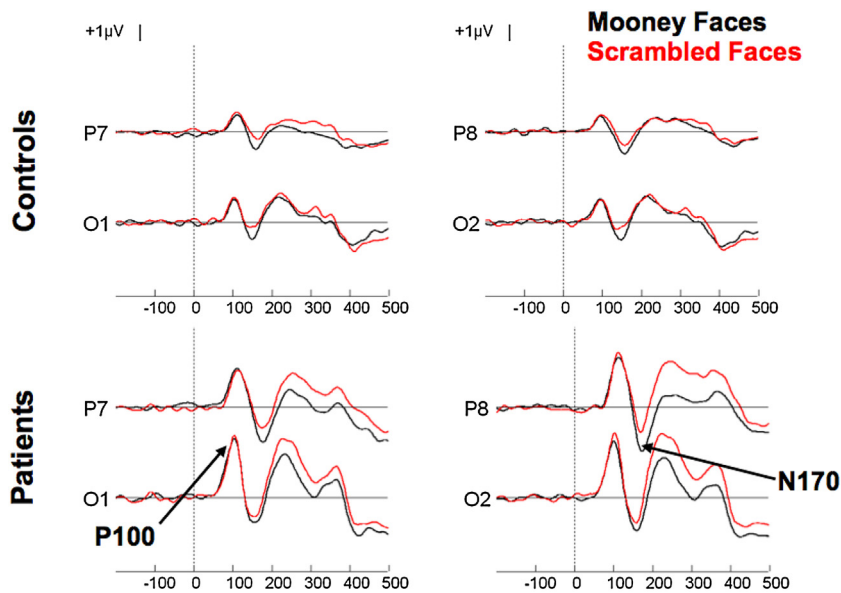


Fig. 7. Grand average potentials from controls (upper panel) and adults treated for bilateral congenital cataract (lower panel) in response to Mooney faces (black) versus scrambled images (red). Highlighted are the P100, a positivity about 100 s after stimulus onset that does not distinguish the two classes of stimuli, and the N170, a negativity about 170 s after stimulus onset that is larger for faces than scrambled images. The patterns are similar except that both the P100 and N170 have a much larger amplitude in patients than in controls. Reprinted from [Mondloch et al. \(2013\)](#). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

rewiring may be related to the reported hyperconnectivity among sensory cortical areas in normal development: in the infant, as in other mammals, there are connections among visual, motor, auditory, and motor cortices that will be shaped by experience to prune those less often stimulated and reinforce those that are fired more often and at the same time as neighboring neurons, which for vision, means neurons with nearby receptive fields ([Maurer, Gibson, & Spector, 2013](#)). When all visual input is missing, as in the congenitally blind, that modeling by visual experience cannot occur and many of the original hyper connected neurons may remain.

The findings on congenital blindness made us rethink our surprising findings that, for higher level visual functions, patients treated for unilateral congenital cataract have smaller deficits than patients treated for bilateral congenital cataracts. Perhaps the process of takeover of visual areas by other sensory modalities begins during the period of deprivation in bilateral cases, but is prevented in unilateral cases because many neurons receive input from the non-deprived eye, which may be strong enough to compete effectively with the non-visual inputs. To evaluate this hypothesis, we have begun to measure cross-modal perception and its underlying neural correlates in patients treated for congenital cataract. The behavioral results indicate that, as predicted, the unilateral patients have normal audiovisual interactions as indexed by the sound-induced flash illusion and the shape of the function relating their judgments of whether a flash and beep were simultaneous to the temporal gap between them. Bilateral patients, in contrast, have a much smaller sound-induced flash illusion and a distorted function for judging audiovisual simultaneity ([Chen, Lewis, Shore, & Maurer, 2014](#); [Chen, Shore, Lewis, & Maurer, 2015](#)). Intriguingly, fMRI results for bilateral patients listening to auditory motion or spoken vowels shows the same atypical pattern as found in the congenitally blind: responses to these auditory stimuli in the visual cuneus bilaterally and the left superior occipital gyrus ([Collignon et al., 2015](#)). (Unilateral patients have not yet been tested). Collectively, the results to date suggest that some of the deficits in bilateral patients may arise because putatively visual areas are not as specialized as they normally would become, responding more to stimuli from other sensory modalities than in visually normal adults.

5. Plasticity in adulthood

Because the critical period to damage the visual system by deprivation ends before adulthood, it is usually assumed that the adult visual system is no longer plastic and hence that it is too late for rehabilitation. But recent evidence from animal models and stroke recovery suggest there is unexpected residual plasticity ([Bavelier, Levi, Li, Dan, & Hensch, 2010](#); [Maurer & Hensch, 2012](#)). To explore this possibility in adults with residual visual deficits after treatment for bilateral congenital cataract, we turned to action video games. We chose that intervention because first person shooter games contain many elements that should be beneficial: fast paced, attention-holding and arousing, with difficulty adjusted to the level of performance, requiring integration of vision with action on a complex virtual world ([Goodale, 1988](#)), and known to stimulate dopamine production that may make the brain more plastic. Moreover, playing them enhances many aspects of vision in adults with normal eyes ([Green & Bavelier, 2007](#); [Li, Polat, Makous, & Bavelier, 2009](#)).

In our study, 7 adults treated for bilateral congenital cataract performed an extensive set of pretests, played Medal of Honor for 10 h a week for 4 weeks, then returned to the lab to repeat the pretests (Jeon, Lewis, & Maurer, 2012). The first 10 h of play were in the lab and subsequent play was monitored (and coached) by videocam. There were improvements after the video game play in acuity, spatial contrast sensitivity, temporal contrast sensitivity, sensitivity to global motion, and the processing of upright human faces. There were no measurable changes in stereopsis or peripheral vision after this short intervention. For none of the measures, did any patient improve to normal levels, but there is no reason to assume that a 40-h intervention exhausts the potential for recovery in adulthood. In any event, these results, like those with other forms of amblyopia (Levi & Li, 2009a, 2009b; Li et al., 2013; Li, Ngo, Nguyen, & Levi, 2011), suggest that the adult brain, although specialized for efficient processing of discrete sensory modalities, retains enough plasticity for some rehabilitation. In fact, it may be that the brain is always plastic but that with development we impose more and more brakes to keep it stable (Bavelier et al., 2010).

6. Summary and conclusion

In summary, early visual input sculpts the brain to be responsive to later input. When it is missing—as in congenital cataract—not only do initial functional improvements fail to occur but there can be sleeper effects, deficits in capabilities that are only manifest much later. When the input is missing to only one eye, the deleterious effects of competition between a deprived eye and a normal eye are manifest in poorer acuity and other low level visual functions but not higher level visual integration, for which deficits are *smaller* following unilateral than bilateral congenital cataract. The implication is that the initially normal input through one eye to higher level visual cortical areas that integrate inputs across eyes and across the visual field was sufficient to set up, or preserve, a neural architecture that is nearly normal. A few aspects of vision are not impaired by a period of visual deprivation near birth but, even in these cases, the neural substrate may not be normal. One reason for these deficits may be crossmodal rewiring of putative visual areas during the initial visual deprivation.

When visual deprivation begins postnatally, there can also be deficits, but they are smaller, the later the deprivation began and eventually cease to occur at all. The end of the critical period for damage varies across visual capabilities and cannot be predicted from the duration of normal development. Here I gave two examples: (1) visual acuity takes about 7 years to reach adult levels of sensitivity and visual input is necessary throughout those 7 years and even a few years beyond, presumably to consolidate connections; (2) sensitivity to global motion, which emerges postnatally and takes about 12 years to become adult-like, has a very short critical period for damage, which ends before the first birthday. Yet despite the ending of all documented critical periods for damage by adolescence, the visual system of the adult remains plastic enough to allow some rehabilitation.

Future research is needed to study the neural basis of the recovery seen after video game play. Did video games cause changes in the primary visual cortex, in higher visual areas and/or in attentional modulation of the visual system? The answer to that question has implications for designing the most effective therapy and for understanding developmental changes in plasticity. For example, it might be the case that the improvements are from attentional modulation and that the visual cortex itself is no longer plastic in adulthood. Finally, what is the role of altered cross-modal functional interactions and neural rewiring in causing the deficits, the timing of critical periods for damage, and the potential for recovery?

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