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Human Visual Plasticity: Lessons from Children Treated for Congenital Cataracts

Daphne Maurer and Terri Lewis

At birth, infants can see only large objects of high contrast located in the central visual field. Over the next half year, basic visual sensitivity improves dramatically. The infant begins to perceive the direction of moving objects and stereoscopic depth, and to integrate the features of objects and faces. Nevertheless, it takes until about 7 years of age for acuity and contrast sensitivity to become as acute as those of adults and into adolescence for some aspects of motion and face processing to reach adult levels of expertise.

An important developmental question is whether, and to what extent, the improvements in vision during normal development depend on normal visual experience. To find out, we have taken advantage of a natural experiment: children born with dense, central cataracts in both eyes that block all patterned visual input to the retina. The children are treated by surgically removing the cataractous lenses and fitting the eyes with compensatory contact lenses that allow the first focused patterned visual input to reach the retina. In the studies summarized in this chapter, the duration of deprivation – from birth until the fitting of contact lenses after surgery – ranged from just a few weeks to most of the first year of life. In other cases, the child began with apparently normal eyes but developed dense bilateral cataracts postnatally that blocked visual input. As in the congenital cases, the cataractous lenses were removed and the eyes fitted with contact lenses. Thus, we can compare the visual development of children with normal eyes to that of children who suffered a period of visual deprivation from bilateral cataracts at different times during childhood. The comparisons allow us to identify the critical periods during which visual input is necessary for normal visual development and to ascertain whether there are developmental changes in the potential for recovery.

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We have measured many aspects of vision in this cohort. In this chapter, we summarize the longitudinal results for acuity and the final outcome for higher-order visual integration involving form, faces, global motion, and biological motion. We end by considering the implications of the findings for understanding the nature of critical periods.

Acuity

To measure acuity, we used preferential looking, the method that takes advantage of babies' preference for patterns such as stripes over plain grey (Fantz, 1963; Fantz et al., 1962). Across trials, the size of the stripes is varied to find the smallest size of stripe eliciting the preference – the presumed limit of the baby's vision. In children with normal eyes, acuity at birth is limited to stripes approximately 40 arc minutes wide, while adults with normal eyes can see stripes less than1 arc minute wide (where 1 arc minute is equal to 1/60 of a degree of visual angle). Over the first 6 months, acuity improves rapidly to 5 to 6 minutes of arc, and then improves more gradually to adult values over the next 6 to 7 years (Mayer et al., 1995; reviewed in Maurer and Lewis, 2001).

On the day that children treated for bilateral congenital cataracts can first see – when they first receive contact lenses to focus visual input – their visual acuity is, on average, around 40 arc minutes, the value for a normal newborn, regardless of when during the first 9 months they were treated (Maurer et al., 1999). The consequence is that children treated later are farther below the normal trajectory, and thus they begin with worse deficits. These results indicate that visual experience is necessary for the rapid developments in acuity observed postnatally in infants with normal eyes. In the absence of visual input – because of bilateral congenital cataracts – the improvements fail to occur.

Nevertheless, the patients' visual systems were not static between birth and the first focused patterned visual input because the patients' acuity began to improve immediately at rates faster than those of age mates. After the first hour of visual input, their acuity had improved on average by a half octave (where an octave is a doubling or halving of a value), and by 1 month after treatment, it had improved on average by an additional half octave; that is, acuity was twice as good as it was initially. The acuity of age-matched controls did not change over such short periods. Thus, the visual system of the patients was experience expectant: it changed postnatally so as to be more easily altered by the first visual stimulation. Likely as a result, by 12 months of age, the acuity of most patients treated for bilateral congenital cataracts is within the normal range, and their mean acuity does not differ from normal (Lewis et al., 1995).

Despite having normal acuity at 12 months of age, patients' final acuity is compromised. Starting at about 2 years of age, the improvement in their acuity ceases, while that of children with normal eyes undergoes an additional fourfold

improvement (Lewis and Maurer, 2009; Maurer and Lewis, 2001). The result is a sleeper effect (Maurer et al., 2007): early visual deprivation prevents the refinement of acuity, but not until after 2 years of age. Presumably, it does so by preventing the setting up or preserving of the neural architecture necessary for that later refinement.

Studies of animals binocularly deprived of visual input from the time of eye opening indicate that the damage to later acuity is likely to have occurred at the level of the primary visual cortex, the earliest level of the visual system where physiological degradation is evident. That degradation includes a nearly four-fold reduction in the number of binocularly driven cells, sluggish responses, reduced spatial resolution, and reduced contrast sensitivity (Blakemore, 1990; Blakemore and Vital-Durand, 1983; Crawford et al., 1975, 1991; reviewed in Movshon and Kiorpes, 1993). Perhaps, as in the congenitally blind (Burton et al., 2004; Collignon et al., 2007; Poirier et al., 2006), some of the putatively visual cortical pathway of the infants with bilateral congenital cataracts began being shaped by other sensory modalities and could not be retrieved for vision after treatment (Maurer et al., 2005). The outcome in humans treated for bilateral congenital cataract is usually better if the treatment occurs very early, with a few bilaterally deprived patients who were treated before 10 days of age achieving normal 20/20 acuity (Birch et al., 2009; Kugelberg, 1992; Lundvall and Kugelberg, 2002; see also Magnusson et al., 2002). However, when the binocular deprivation extends from birth past 3 months of age, the outcome is not worse with longer than with shorter deprivation (Birch et al., 2009). These results suggest that patterned visual input in the first week of life plays an especially important role in setting up the neural architecture for later refinement, with an additional strong influence over the next 3 months.

In children who developed cataracts postnatally, the final acuity deficits are smaller, the later the deprivation started. Surprisingly, there are deficits, albeit small ones, when the deprivation began between 7 and 10 years of age – 3 years longer than the emergence of adultlike acuity in the child with normal eyes (Lewis and Maurer, 2009; Maurer and Lewis, 2001). Thus, visual input is necessary for the improvements in acuity throughout the 7 years of normal visual development and for a number of years thereafter. The results suggest that visual input serves to build the necessary connections over the first 7 years and to consolidate or crystalize them for 3 years thereafter. After 10 years of age, even a long period of deprivation will cause no permanent deficit in acuity – at that point, the system appears to be "hard wired."

Overall, our studies of acuity indicate that during infancy visual input drives the rapid improvement in acuity seen over the first 6 months while also setting up the neural substrate for later improvements. When visual input is absent for as little as the first month of life, a period when the baby with normal eyes is rarely awake and sees very poorly, later refinements fail to occur, leading to a

sleeper effect of a seemingly permanent reduction in acuity. After infancy, visual input still plays a vital role in building the connections underlying adult's fine acuity until 7 years of age and in crystallizing those connections from age 7 to 10. After age 10, those connections are preserved even without visual input.

Higher-Level Vision

Global Form

Adults with normal eyes can not only see fine details, but they can also integrate those details into distinct objects that are separate from the background. To measure the effects of early visual deprivation on the development of that integrative skill, we used Glass patterns (Glass and Hakstian, 1969). Specifically, we contrasted a Glass pattern formed by structured pairs of small dots oriented so as to form a circular pattern of swirls to a pattern with pairs of dots randomly oriented with respect to each other. Over trials, the amount of signal in the structured pattern was reduced by replacing some of the structured pairs of dots with pairs of randomly oriented dots. A threshold was measured by determining the percentage of signal dots needed to discriminate the structured and random pattern. In our version of the task, adults need 20 percent to 25 percent to signal, a threshold reached by children with normal eyes at only about 9 years of age (Lewis et al., 2004). In contrast, patients treated for bilateral congenital cataract needed, on average, about 40 percent signal, regardless of whether we tested them with small dots or with larger dots that they could see more easily (Lewis et al., 2002). There was no effect of the duration of deprivation on the threshold to perceive global form even though the duration of deprivation (defined as the time from birth until the fitting of the first contact lens after surgery) ranged from 3 to 9 months. Thus, as little as 3 months of deprivation after birth is sufficient to prevent the normal development of the ability to integrate details into a global form. Similarly, Jeffrey and colleagues (2004) reported deficits in a different test of global form perception in the one patient they tested who had been treated for bilateral congenital cataracts (patient CH), a patient who had been treated at 4 months of age. These deficits are another example of a sleeper effect: visual input is necessary during a period when the infant with normal eyes has no, or at best rudimentary, ability to integrate features into a global percept (Cashon and Cohen, 2003) in order to later achieve normal refinement of sensitivity to global form. The effect may arise from a failure to form normal connections along the ventral pathway from V1 to V4 (Desimone and Schein, 1987; Gallant et al., 2000; Wilkinson et al., 2000). It suggests that early visual input is necessary to set up not only the neural architecture in the visual cortex for the later refinement of acuity but also the neural connections from the visual cortex to higher extrastriate visual areas. Because there are no published studies of sensitivity to global form in children treated for bilateral cataracts with later onset, we cannot comment on whether visual input continues to be necessary throughout the period of normal development, or even beyond.

Faces

Adults are experts at processing facial identity: they can recognize the faces of thousands of individuals, despite changes in point of view, facial expression, or direction of gaze. Rudimentary forms of these skills emerge during infancy, but it takes into adolescence for the skills to be honed to the adult level of expertise (reviewed in Maurer and Mondloch, 2011). On the day they could first see focused patterned visual input, children treated for bilateral congenital cataracts oriented preferentially toward faces, but their choices resembled those of newborns rather than those of their age mates (Mondloch et al., 1998, 2003). Then, with postnatal experience, the visually deprived children became as adept as normal age-matched controls in detecting that a stimulus is a face rather than a scrambled image (Mondloch et al., 1998, 2003). Those results imply that visual experience is necessary for the postnatal changes in face detection (Mondloch et al., 1999) but that, unlike the case for acuity and global form, delayed visual experience can offset earlier deprivation.

Nevertheless, patients fail to develop the entire panoply of skills that normal adults use to distinguish among faces. They are as good as normal adults at distinguishing differences in the shape of the eyes and mouth (Mondloch et al., 2010) but have a seemingly permanent deficit in detecting differences in the spacing of those features (Le Grand et al., 2001; Robbins et al., 2010), and perhaps as a result, are impaired in recognizing an unfamiliar face in a novel point of view (Geldart et al., 2002) and in recognizing the identity of famous faces or faces learned in the laboratory (de Heering and Maurer, 2012). These deficits may also originate from a failure to process faces holistically during infancy and childhood, instead processing them as the sum of independent features (Le Grand et al., 2004) and/or from a failure to establish the normal tuning of the system for face processing to upright rather than to inverted faces (Robbins et al., 2012). The deficits in sensitivity to feature spacing appear to be specific to upright faces, the category for which adults with normal eyes have developed especial sensitivity. Thus, patients treated for bilateral congenital cataract develop normal sensitivity to feature spacing in monkey faces, inverted faces, and houses (Le Grand et al., 2001; Robbins et al., 2010).

Across the studies documenting deficits, the duration of deprivation ranged from as little as 9 days to almost 2 years, with no correlation between the size of the deficit and duration of deprivation or acuity. Combined, the results indicate that the neural architecture underlying face detection can be tuned equally well by visual input near birth or later during infancy. In contrast,

form a scrambled version of the stimulus (Simion et al., 2008). Thus, unlike the other higher-order visual abilities discussed in this section, the neural substrate may form prenatally before the onset of visual experience.

Summary of Experimental Findings on Human Binocular Deprivation

Our results for patients treated for dense bilateral cataracts indicate that early visual input is necessary for the later normal development of both low-level (acuity) and high level visual processing (global form, face expertise, global motion), but that some aspects of vision are spared, or at least recover from any earlier deficit before adulthood (face detection, featural processing, biological motion). In many cases, we have observed sleeper effects such that after early visual deprivation lasting as little as a few weeks, deficits emerge in visual abilities that are manifest only at a later age in the child with normal eyes. Some visual capabilities (illustrated here by acuity) are dependent on patterned visual input throughout the period of normal development, presumably to build connections, and for some years thereafter, presumably to strengthen or crystalize the connections. Others (illustrated here by global motion) depend only on very early input and thereafter are impervious to even a long period of visual deprivation. Thus, there are multiple sensitive periods during which visual input is necessary to prevent seemingly permanent damage to the visual system, and their timing does not necessarily coincide with the period of normal development.

Implications for Critical Periods

The experimental findings suggest three hypotheses about critical period that are not mutually exclusive.

Hypothesis 1. Early input sets up the neural architecture for later refinement for all visual capabilities that become manifest only after birth and/or for which the requisite neural pathways are remodeled during infancy. The early input may strengthen already existing synaptic connections or cause new connections to form. In its absence, as has been documented in the congenitally blind, connections may be shaped by auditory and tactile input through selective strengthening of the exuberant connections that are present early in development. In adults who never receive visual input because of congenital blindness, the primary visual cortex responds functionally to touch, sound, and even language (e.g., Collignon et al., 2009; Maurer et al., 2005; Pascual-Leone et al., 2005). During visual deprivation from cataract, these nonvisual inputs, which normally would be too weak to compete with visual input, may begin to strengthen connections. Once visual input is restored, it may not be sufficiently strong to reclaim some of those connections, leading to functional deficits. This hypothesis is also supported by the fact that during the period of deprivation, the affected abilities are at best very primitive in the infant with normal eyes, a fact suggesting that the requisite neural architecture is not fully formed. Moreover, behavioral and EEG evidence suggests that in the infant with normal eyes, higher visual areas are not yet as specialized as in adults: there appears to be color input to the dorsal stream that will disappear before adulthood (Dobkins, 2006); there are apparently functional cross-modal and cross-dimensional links like those found in synaesthesia that will be pruned or inhibited (Spector and Maurer, 2009; Wagner and Dobkins, 2011); and the networks involved in the processing of global form and global motion will change their topology between 5 months and adulthood (Wattam-Bell et al., 2010). Under this hypothesis, visual capabilities may be spared despite early visual deprivation when the requisite neural architecture is already well developed, perhaps based on spontaneous retinal input, which is known to affect the development of the visual pathway in animal models (reviewed in Torborg and Feller, 2005). Consistent with this view, newborns with normal eyes differentiate facelike stimuli from distorted images (reviewed in Maurer and Mondloch, 2011), recognize featural changes in faces (reviewed in Maurer and Mondloch, 2011), and distinguish biological and scrambled motion (Simion et al., 2008). In addition, for biological motion, the infant's body movements may help set up the neural architecture for biological motion using a system analogous to mirror neurons that have been identified in monkeys and humans (Ferrari et al., 2003; Iacoboni et al., 1999).

Hypothesis 2. After early visual deprivation, alternative networks are recruited that may be functioning in early infancy, are resilient to early visual deprivation, and are capable of mediating normal sensitivity to some aspects of vision. For example, early visual deprivation damages at least parts of the geniculocortical pathway projecting from the retina through the lateral genuiculate nucleus to the primary visual cortex, but it may spare visual pathways that reach the extrastriate visual cortex despite bypassing the primary visual cortex, perhaps through the superior colliculus, pulvinar, and pretectum. Although such pathways may only play a minor role in the adult with normal vision, anatomical and neuropsychological studies (of blindsight) have verified their existence (Cowey, 2010). By this hypothesis, the preserved abilities are ones that the damaged geniculocortical pathway or the undamaged alternate pathways can mediate; the deficits reflect limits in the alternate pathways, such as having neurons with large receptive fields and poor spatial resolution. Evidence to support this hypothesis comes from studies of kittens who were deprived of visual input by being reared with their heads in a hood (Zablocka and Zernicki, 1996; Zablocka et al., 1976; Zernicki, 1979). Following the visual deprivation, they could learn with difficulty to discriminate between basic shapes and generalize the discrimination to shapes of different sizes and contrast. After learning, unlike the case for normal cats, lesions to the primary visual cortex had no effect on their discrimination, whereas lesions to the pretectum and superior

colliculus, which had no effect on normal cats, abolished the discrimination. That pattern suggests that the visually deprived cats learned to discriminate the shapes using a pathway that reached extrastriate cortex, not via the geniculostriate pathway, but rather through the superior colliculus and pretectum. Such cortical reorganization in response to visual deprivation may be possible only during early development, leading to distinctive patterns of sparing and damage during specific critical periods.

Similarly, in visually normal human adults, although biological motion is mediated primarily by a network involving pSTS, many other areas are responsive to biological motion, including the medial and lateral cerebellum, intraparietal cortex, middle temporal gyrus, posterior inferior frontal gyrus, premotor cortex, kinetic-occipital area (KO), fusiform face area (FFA), amygdala, and the ventral portion of V3 (e.g., Bonda et al., 1996; Grèzes et al., 2001; Saygin, 2007; Servos et al., 2002). Biological motion may be spared because the vast number of structures implicated, some of which are known to receive visual input (intraparietal cortex, KO, V3), compensate for any deficits arising from compromised development within the geniculostriate pathway and its input into pSTS. The possibility of such reorganization after early visual deprivation is strengthened by evidence for considerable reorganization between infancy and adulthood in the pathways mediating at least some aspects of vision (see hypothesis 1).

Hypothesis 3. Despite early visual deprivation, some connections in the visual pathway form normally and are preserved, but an abnormal balance between excitatory and inhibitory inputs prevents them from being functional. Specifically, excessive GABAnergic inhibition prevents the neurons receiving these inputs from firing. Animal models indicate that early visual deprivation alters the excitatory/inhibitory balance and that it can do so only during a critical period shortly after birth. Reduction of GABAnergic inhibition by placing the animal in the dark or by pharmacological manipulations can restore plasticity in adulthood (reviewed in Bavelier et al., 2010, and Maurer and Hensch, 2012). Similarly, in adults with "lazy eye" – that is, reduced acuity secondary to a history of having had crossed eyes during infancy that prevented the visual system from receiving concordant binocular input – reductions in inhibition by transcranial magnetic stimulation (TMS) cause an immediate improvement in contrast sensitivity (Thompson et al., 2008, 2012). Converging evidence comes from studies of blindfolding in adults with a normal visual history: after 5 days of blindfolding, the visual cortex begins to respond to tactile input as some tactile connections remained after infancy but are normally inhibited by the dominant visual input (Pascual-Leone et al., 2005).

Remediation

The effects of early visual deprivation appear to lead to permanent deficits in acuity. Once the patient reaches adolescence, the deficits are stable over years,

and clinicians advise patients that there is nothing more that can be done. Yet, emerging evidence suggests that the brain may be more plastic in adulthood than once thought. For example, in adults with lazy eye, training with feedback to detect basic visual properties leads to improvements on the trained task and in letter acuity (Levi and Li, 2009). Manipulations to decrease GABAnergic inhibition – Levodopa for lazy eye, constraint therapy (Taub, 2012) and TMS (Sharma and Cohen, 2012) for stroke – are also effective. The vision even of adults with normal eyes can improve through playing an action video game (Green and Bavelier, 2007).

Given these promising leads, we are exploring whether playing action video games can improve the vision of adults with seemingly permanent deficits secondary to bilateral congenital cataracts. Video games seem promising not only because of their success in improving the visual sensitivity of adults with normal eyes but also because they capture what we suspect are some of the key components of a successful intervention: they are titrated to the player's level of performance; demand simultaneous monitoring of the central and peripheral visual field; are fast paced; include objects of both higher and lower contrast; and, at least in adults with normal eyes, increase circulating levels of dopamine (Koepp et al., 1998; but see Egerton et al., 2009), which may put the brain in a more plastic state. Like rats placed in complex environments (Sale et al., 2007), they may also reduce the level of GABAnergic inhibition.

To date, we have studied seven adults treated for bilateral congenital cataract whom we had followed since the time of treatment (Jeon et al., in press). Their initial deprivation ranged from birth until 3 to 10 months, and at the beginning of the intervention they were 19 to 31 years old. All had stable acuity deficits that ranged from 20/20 to 20/63 in the eye with the better acuity and from 20/63 to 20/100 in the eye with the worse acuity. They showed no evidence of binocular fusion on the Worth 4 dot test and no evidence of even gross stereopsis on the Titmus or Randot tests. At least with the Worth 4 dot test, they also showed no evidence of suppressing either eye. Because the patients apparently made use of both eyes in everyday vision, we trained them binocularly with one exception. The exception was the only patient who was an active gamer before the intervention. Because he often played action video games and had done so for years, and because acuity differed between his two eyes (20/63 vs. 20/80), we instructed him to play the video game with his better eye patched and with the contrast reduced to make the game more difficult.

After an extensive pretest, patients played Medal of Honor for 40 hours over 4 to 5 weeks, with no more than 2 hours of play on any day and no more than 10 hours per week. The first 10 hours of play were in the lab under the supervision of a researcher; the subsequent play was at home, but with monitoring via webcam and email reports. The patients then returned to the lab for a repeat of the pretest.

The results showed moderate improvements for a wide variety of skills in each case in at least some of the patients. For example, visual acuity improved in the

worse eye and/or with binocular viewing in every patient so that on average they could read one line farther on the eye chart. Spatial contrast sensitivity improved at least for some spatial frequencies in both the worse eye and with binocular viewing. Sensitivity to global motion when viewing binocularly improved in every patient for dots moving at 4 deg/sec and/or at 18 deg/sec. Thus, 40 hours of video game play as an adult is sufficient to effect improvements in both low-level and higher-level aspects of vision in adults with deficits secondary to early binocular deprivation. Despite being past the critical periods during which visual deprivation can damage perception, adults' nervous systems are still sufficiently plastic for remediation. The amount of improvement might be even greater with longer play and/or separate video game input to each eye titrated to balance the level of stimulation and promote binocular coordination. It might also be more effective if combined with other interventions that decrease GABAnergic inhibition or structural brakes on plasticity, such as myelin and perineuronal nets, that have been identified in animal models (Hensch, 2005; Maurer and Hensch, 2012).

Unanswered Questions

A number of questions remain unanswered. First, can complete recovery be effected in adulthood for any or all visual capabilities? The improvements might represent recovery in the normal visual pathway (through the formation and/or release of connections). Instead, they might reflect more efficient monitoring of noisy signals by higher cortical areas and/or refinement of secondary visual pathways bypassing the visual cortex. Studies of perceptual training in adults with lazy eye suggest that a large part of the improvement results from improved efficiency (Levi, 2005; Levi and Li, 2009). Nevertheless, there is evidence that some patients also improve from a reduction in internal noise, which could result from any of the other possibilities.

Second, how specific are the experiential effects revealed by patients treated for bilateral cataracts? Our cohort was deprived of all patterned visual input and ended up with deficits in the processing of form, motion, and faces. Whether normal development in humans requires specific input from, in these examples, different forms, moving objects, and interactive faces is an open question. In animal models, the results conflict for motion versus faces. Selective rearing with patterned visual input that is always static (because it is illuminated by stroboscopic light) leads to severe deficits, at least initially, in cats' motion processing (Cremieux et al., 1987; Pasternak et al., 1985). However, selective rearing for up to 2 years in a rich visual environment without any input from human or monkey faces leaves the monkey, at the end of deprivation, with a normal newborn preference for (monkey or human) faces and the ability to discriminate among individual faces differing only in feature spacing. Over the next month, these face-deprived monkeys show a normal process of perceptual narrowing favoring monkey faces if they were returned to the monkey colony and favoring human faces if that was the only category to which they were exposed (Sugita, 2008).

Third, our longitudinal studies of acuity (Lewis et al., 1995), contrast sensitivity at low spatial frequencies (wide stripes) (Maurer et al., 2006), and face detection (Maurer et al., 2012) indicate that the final deficits result from the combination of a severe initial deficit combined with remarkable but incomplete recovery that can end long before normal development is complete (acuity) or persist past the period of normal development (contrast sensitivity at low spatial frequencies). (Face detection was not studied during the intermediate ages.) An unanswered question is whether the deficits in the perception of form, direction of global motion, and discrimination of facial identity represent a partial recovery from a larger earlier deficit. Similarly, we do not know if the spared abilities such as biological motion represent recovery from an earlier deficit or the manifestation of a neural circuit that does not depend on early visual input for any phase of normal development. Only longitudinal studies can answer these questions.

Fourth, our studies of children who developed bilateral cataracts at different ages have identified different critical periods during which acuity (10 years) versus sensitivity to global motion (8 months) can be damaged by a period of visual deprivation. To date, we have not studied sufficient cases to identify the critical periods for damage to global form or face processing. One possibility is that the critical periods for all higher level aspects of vision will be relatively short because of the convergence of input from multiple neurons in the primary visual cortex onto extrastriate neurons with large receptive fields and generally poor visual resolution. Those extrastriate neurons may be able to function well even when the inputs are reduced or degraded.

Fifth, we do not know the neural underpinnings of the deficits in humans, or what mediates the spared abilities. The answer in both cases could be damaged circuitry in the visual cortex of the type that has been identified in animal models. Thus, after binocular deprivation in monkeys, neurons in the primary visual cortex respond abnormally sluggishly, have large receptive fields with poor spatial resolution, and show a marked reduction in their ability to respond to both eyes at once (Blakemore, 1990; Blakemore and Vital-Durand, 1983; Crawford et al., 1975, 1991; reviewed in Movshon and Kiorpes, 1993). What is not known is the effect on higher visual cortical areas that normally receive inputs from both the primary visual cortex and subcortical inputs that directly innervate extrastriate cortex, such as those from the superior colliculus and pretectum. Our preliminary results from fMRI and ERP measures of the adults in our cohort who were treated for bilateral congenital cataract indicate that the neural underpinnings of their behaviorly normal face detection are abnormal:

when viewing faces (vs. houses or scrambled images), there is less differentiation than normal in the fusiform gyrus (fMRI) and a great deal more activation than normal over occipital leads (ERP) (Grady et al., 2006; Maurer et al., 2012). The greater activation is puzzling and suggests the possibility that binocular deprivation prevents the normal pruning of exuberant connections, possibly because some of those in visual areas continue to respond to auditory and tactile inputs.

Summary

Our studies of children treated for bilateral cataracts that blocked visual input at varying times during development indicate that it is important to distinguish three types of critical period:

(1) the period of normal development that is driven by visual input
(2) the period when the system can be damaged by the absence of visual input
(3) the period when the system can recover from earlier deprivation

The results summarized in this chapter indicate not only that the timing of these critical periods differs across visual capabilities but also that it can differ even within a given aspect of vision. For example, for the development of visual acuity, the first critical period is the entire 7 years of normal development, the second critical period extends further to 10 years of age, and the last critical period may be unlimited. For global motion, the first two critical periods appear to end by 8 months of age, but, again, the critical period for recovery appears unlimited. Combined, our results indicate that in the infant with normal eyes, experience seeing the world is vital for setting up the visual system for the later refinement of both sensory perception and higher-order visual cognition.

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