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REVIEW

Effects of Early Pattern Deprivation on Visual Development

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ABSTRACT

Studies of children treated for dense cataract shed light on the extent to which pattern stimulation drives normal visual development and whether there are sensitive periods during which an abnormal visual environment is especially detrimental. Here, we summarize the findings to date into five general principles: (1) At least for low-level vision, aspects of vision that develop the earliest are the least likely to be adversely affected by abnormal visual input whereas those that develop later are affected more severely. (2) Early visual input is necessary to preserve the neural infrastructure for later visual learning, even for visual capabilities that will not appear until later in development. (3) The development of both the dorsal and ventral streams depends on normal visual input. (4) After monocular deprivation has been treated by surgical removal of the cataractous lens, the interactions between the aphakic and phakic eyes are competitive for low-level vision but are complementary for high-level vision. (5) There are multiple sensitive periods during which experience can influence visual development.

The studies described here have important implications for understanding normal development. They indicate that patterned visual input immediately after birth plays a vital role in the construction and preservation of the neural architecture that will later mediate sensitivity to both basic and higher level aspects of vision. The period during which patterned visual input is necessary for normal visual development varies widely across different aspects of vision and can range from only a few months after birth to more than the first 10 years of life. The results point to new research questions on why early visual deprivation can cause later deficits, what limits adult plasticity, and whether effective rehabilitation in other areas can provide new clues for the treatment of amblyopia.
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Key Words: visual deprivation, cataract, infants, children, visual outcome, sensitive periods, normal visual development

Right from birth, babies can see. From the seminal work of Fantz more than 50 years ago, we learned that newborns can discriminate between a plain gray field and black-and-white stripes of 0.75 cpd, between face-like and non–face-like patterns, between moving and stationary patterns, and between high-contrast patterns with rounded vs. straight contours.[1–3] Over the years, the list of newborns' capabilities has grown to include, at least under some circumstances, the ability to distinguish some colors from gray,[4] stripes tilted 45° to the left vs. right,[5,6] biological motion[a] from scrambled motion,[7] and the ability to integrate across rows of black-and-white squares to see the similarity of the pattern they form, based on Gestalt principles, to black-and-white stripes.[8] Vision improves rapidly over the next half year so that by 6 months of age, acuity, for example, has improved fivefold,[9,10] and color vision is essentially adult-like.[11]

Visual capabilities continue to improve after early infancy, but the age at which children's vision is as good as that of adults varies widely with the aspect of vision under study. For example, by 6 to 7 years of age, children are as accurate as adults on measures of acuity, contrast sensitivity, holistic and featural face processing, and sensitivity to global motion (the ability to integrate the overall direction of motion in a pattern of moving dots, only some of which are moving in a coherent direction).[12–16] In contrast, at age 6, children are not as accurate as adults on sensitivity to biological motion[17] and sensitivity to global form (the ability to integrate individual dots forming a swirl among randomly positioned dots).[18] On some measures such as distinguishing faces based on

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[a] To portray biological motion, small lights are attached to the head and major joints of a human or animal performing various actions such as walking or running. When only the dots are visible, adult observers still experience a vivid impression of the organism's activity.

the spacing of internal features and integrating elements with a similar alignment when presented in a background of noise, even 14-year old children are not as accurate as adults.[19,20]

For vision, unlike audition or taste, capabilities present at birth have developed prenatally in the absence of experience from the external environment (although they may have been shaped by spontaneous patterns of neuronal patterning or stimulation of other sensory modalities).[21–23] However, from the moment of birth, the baby experiences visual stimulation. To what extent does such stimulation drive the postnatal changes? Are there sensitive periods during which an abnormal visual environment is especially detrimental?

To answer these questions, we have taken advantage of a natural experiment—children treated for dense cataracts in one or both eyes. The cataracts were so dense that they prevented all patterned vision from reaching the retina until they were treated by removing surgically the natural lens of the eye and replacing it with a contact lens of a suitable refractive power. Studying the visual outcome of such children has shown us that visual deprivation has different effects on different aspects of vision and at different times during development. We summarize the findings below in the form of five general principles, some of which have been discussed briefly elsewhere.[24]

1. At least for low-level vision, aspects of vision that develop the earliest are the least likely to be adversely affected by abnormal visual input whereas those that develop later are affected more severely

In 1993, Levi and Carkeet[25] formulated what they called the "Detroit model"—a biological analogue of the "first hired, last fired" policy common in industry. The model proposes a direct relationship between the rate of normal development and the effects of abnormal visual experience such that the earlier an aspect of vision develops, the less susceptible it will be. We have found that the Detroit model holds for low-level vision, that is, aspects of vision that depend mainly on the primary visual cortex.

Studies of spatial and temporal vision illustrate the point. Critical flicker fusion frequency, the fastest rate of high-contrast flicker that can be perceived, is adult-like at 2 months of age,[26] whereas grating acuity, the narrowest high contrast stripes that can be differentiated from gray, is not adult-like until 4 to 6 years of age.[12,27] As would be predicted by the Detroit model, early monocular or binocular deprivation has little or no effect on critical flicker fusion frequency, but causes marked losses in grating acuity in the surgically treated aphakic eyes.[28,29]

Contrast sensitivity illustrates the same point. Contrast sensitivity for low spatial frequencies—the only spatial frequencies that infants can see[30,31]—is affected little if at all by early monocular or binocular deprivation, unlike the severe losses in the aphakic eyes in sensitivity to medium and high spatial frequencies.[28,29] Comparable patterns of the relation between normal development and the effects of early deprivation hold for peripheral vision and temporal contrast sensitivity. The far temporal visual field is the last to mature and sensitivity there is the most adversely affected after early monocular or binocular deprivation.[32,33] Similarly, temporal contrast sensitivity matures more slowly for middle (5 to 10 Hz) than for high-temporal frequencies during infancy and childhood,[12,34,35] and deficits in the aphakic eyes after early monocular or binocular deprivation are greater for the middle than for the high-temporal frequencies (patient data are not available for low temporal frequencies).[28,29]

Together, the evidence supports the principle that the aspects of basic vision that develop the latest are the most adversely affected by early visual deprivation. However, the Detroit model may be limited to low-level vision—to aspects of vision that involve mainly the primary visual cortex. As shown below in Section 3, the Detroit model did not hold for our studies of higher level vision—aspects of vision that involve networks in the extrastriate cortex and require the integration of basic elements across time or space (e.g., the global integration of form and motion cues). Interestingly, this is only one of several examples of rules that work for low-level vision but seem not to apply to higher level vision. As described in Section 4, we find evidence for competitive interactions between an aphakic and phakic eye for low-level vision but for complementary interactions between an aphakic and phakic eye for higher level vision. As described in Section 5, the sensitive period for acuity, an example of low-level vision, is quite long, much longer than the sensitive period for global motion, an example of higher level vision. Whatever the mechanisms underlying these differences, it is clear that findings from low-level vision cannot be generalized to higher level vision.

2. Early visual input is necessary to preserve the neural infrastructure for later visual learning, even for visual capabilities that will not appear until later in development

As discussed in the previous section, visually normal young infants see only low spatial frequencies.[30,31] Not until at least 2 years of age do they begin to see high-spatial frequencies of 20 cpd, even at maximum contrast.[36] Yet, most children born with cataracts in one or both eyes whose deprivation ended within the first 6 months of life later fail to develop normal sensitivity to those high-spatial frequencies in the aphakic eyes.[28,29] This is true, despite the fact that their deprivation ended long before visually normal children can see such high-spatial frequencies. Studies of visually deprived monkeys suggest that the deficits are likely to arise at the level of the primary visual cortex. After binocular deprivation, V1 cells respond sluggishly, have abnormally large receptive fields, and have reduced acuity; after monocular deprivation, they respond even more abnormally.[37–39] In contrast, cells in the monkey's retina and lateral geniculate nucleus respond normally after monocular or binocular deprivation.[37,38]

A second example of a "sleeper effect" comes from the development of face processing. Adults' expertise in face processing arises, in part, from holistic processing (glueing the features together into a Gestalt) and acute sensitivity to small differences between faces in the spacing of features (e.g., the distance between the eyes).[40] Visually normal infants appear to process faces in a piecemeal fashion with the first evidence of a type of holistic processing at 4 months of age[41,42] and of sensitivity to large differences in the spacing of features at 5 months of age.[43] Yet, children born with cataracts in both eyes whose deprivation ended by 2 to 3 months of age fail to later develop normal holistic face processing or sensitivity to spacing of features,[44,45] despite the fact that their deprivation occurred before the capabilities are normally manifested and during a period when face input to the visually normal infant is degraded by poor acuity and contrast sensitivity.

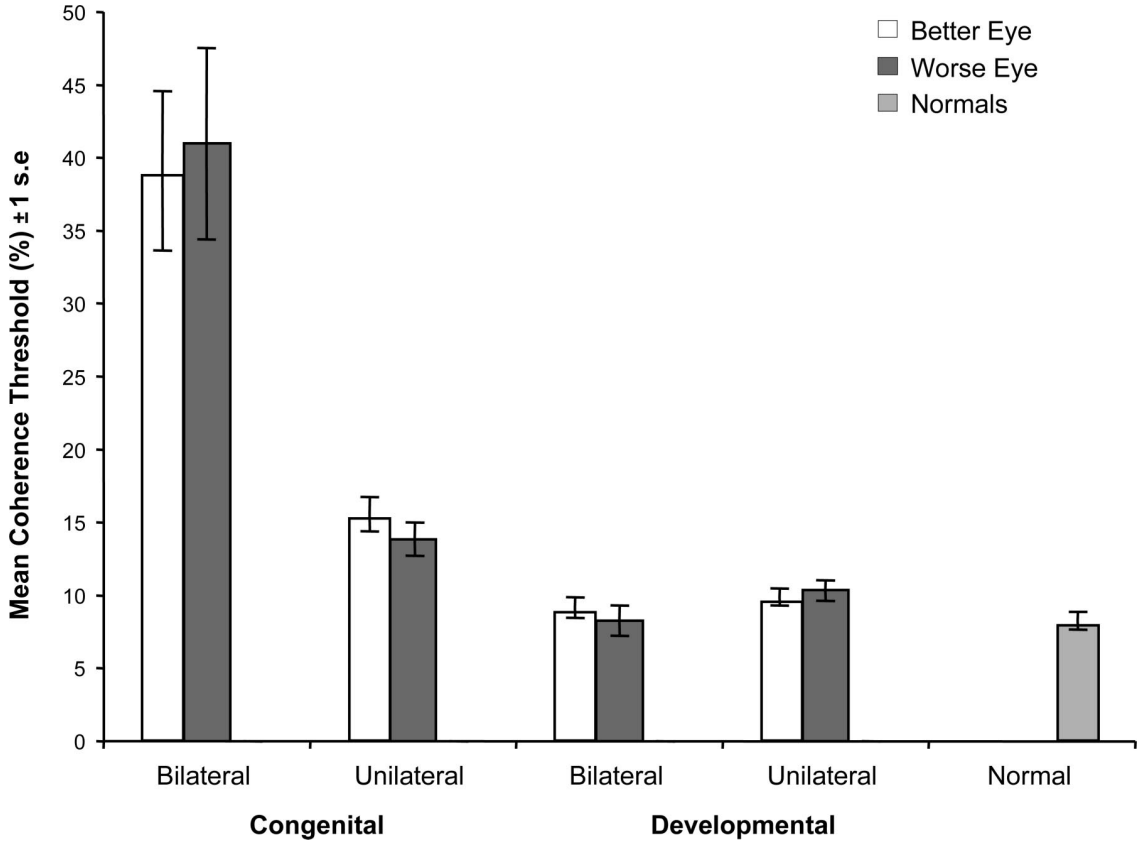

FIGURE 1.

Mean coherence thresholds for the test of sensitivity to global motion. Data are for patients treated for bilateral congenital cataract (n = 8), unilateral congenital cataract (n = 14), bilateral developmental cataract (n = 6), or unilateral developmental cataract (n = 9). White bars represent data for the better eyes of bilateral cases (determined from clinical history of alignment and Snellen acuity) and the phakic eyes of unilateral cases. Black bars represent the data from the worse eyes of bilateral cases and the surgically treated aphakic eyes of unilateral cases. Bilateral cases with equal alignment and acuity histories for the two eyes had one eye assigned randomly to each category. The final gray bar represents the mean of 24 control subjects with normal vision.

In summary, early visual deprivation from congenital cataract prevents the later development of normal sensitivity to high spatial frequencies, holistic face processing, and sensitivity to feature spacing. These deficits are examples of sleeper effects: visual deprivation during a period in normal infancy before the first manifestations of functional ability prevents its later development (see ref. 46 for a detailed discussion). One possible explanation of such sleeper effects is that visual input during early infancy is necessary to set up or preserve the optimal neural architecture for the visual capability. In the absence of visual input, the requisite cells and connections may fail to develop or be lost through competitive interactions involving inputs from other sensory modalities, as suggested by the specialization of the visual cortex, including the primary visual cortex, for touch, hearing, and perhaps even language in the congenitally blind.[46] By this account, the visual capability cannot develop normally at a later point in development because the optimal neural architecture to support it is no longer available.

3. The development of both the dorsal and ventral streams depends on normal visual input

Our studies of sensitivity to global motion and global form in patients treated for congenital cataract indicate that both the dorsal stream, which is involved in sensitivity to global motion,[47] and the ventral stream, which is involved in sensitivity to global form,[47] depend on early patterned input. To test sensitivity to global motion, we showed patients random dot kinematograms in which dots moved in random directions, except for a proportion of signal dots moving coherently in the same direction. Over trials, we varied the percentage of signal dots to determine the minimum percentage of dots needed to move in the same direction, among randomly moving dots, for the subject to accurately perceive that predominant direction of motion. Each of the eight patients treated for bilateral congenital cataract had abnormal coherence thresholds—thresholds that were on average five times worse than normal (see Fig. 1, left side).

To test sensitivity to global form, we used "Glass" patterns[48] like those shown in Fig. 2. The pattern on the left of Fig. 2 has 100% signal: all dots are arranged in pairs that form swirls because the orientation of each pair is tangent to a circle centered on the pattern. The pattern on the right has 50% signal: the global form was degraded by replacing 50% of the signal dot pairs with an equal number of randomly spaced noise dots. Thresholds for detecting global structure were defined as the minimum percent signal necessary to accurately discriminate patterns containing signal dots from noise patterns with no signal. Again, patients showed deficits.

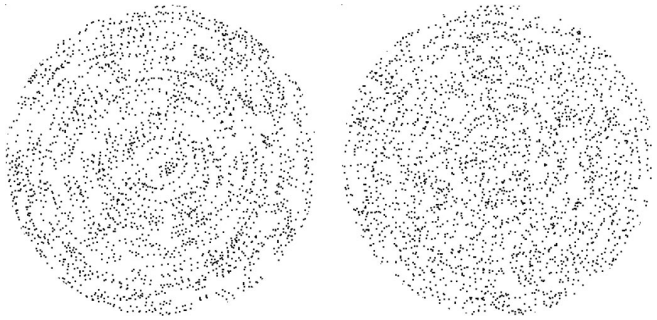

FIGURE 2.

Examples of the patterns used to test for the ability to integrate elements into a global percept of form. In the pattern on the left, 100% of the dots are paired to form a global swirl, whereas in the pattern on the right, there are 50% paired dots and 50% randomly paired noise dots. Threshold was calculated as the maximum number of noise dots that could be tolerated and still allow the subject to accurately discriminate a stimulus containing some paired signal dots from a stimulus comprised entirely of noise dots.

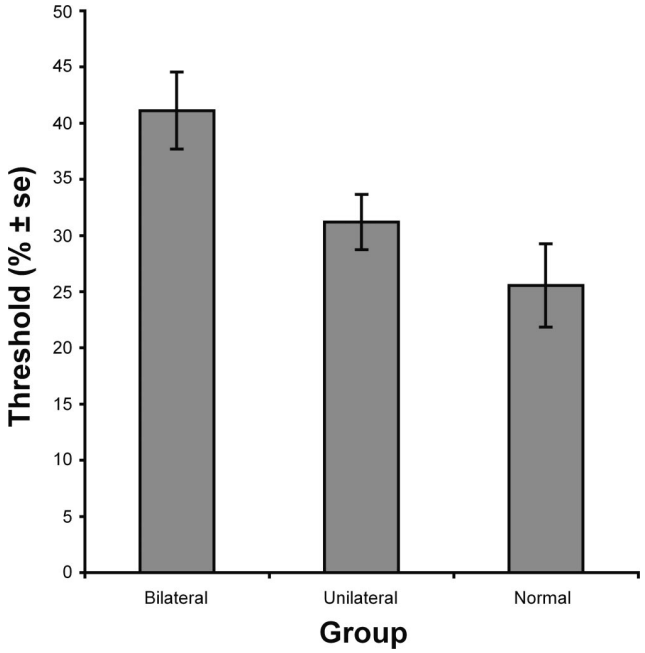

FIGURE 3.

Mean coherence thresholds for the test of sensitivity to global form. Data are for a randomly selected eye of patients treated for binocular congenital cataract, the surgically treated aphakic eye of patients treated for monocular congenital cataract, and age-matched controls. Adapted with permission from Trends Cogn Sci, 9, 144–51, 2005.

Because sensitivity to global form matures much later than sensitivity to global motion,[13,16,18] we had predicted that, according to the Detroit model discussed previously, sensitivity to global form would be more adversely affected by early deprivation than would sensitivity to global motion. However, the deficits for global form were much smaller than those for global motion, especially after binocular deprivation—only 1.6 times worse than normal for global form (Fig. 3) but five times worse for global motion (Fig. 1). Thus, both the dorsal and ventral streams, like primary visual cortex, rely on normal visual input. Although the results for bilateral patients suggest that the effects may be stronger on the dorsal stream, results from unilateral patients presented in Section 4 tell a different story.

4. After monocular deprivation, the interactions between the aphakic and phakic eyes are competitive for low-level vision but are complementary for high-level vision

It is well known to ophthalmologists and vision scientists that vision in an eye treated for cataract is worse after monocular deprivation than after binocular deprivation unless, after monocular deprivation, the phakic eye is patched aggressively. We have demonstrated this general principle many times in our studies of acuity and contrast sensitivity[9,10] and in our studies of peripheral vision.[49] Thus, at least for low-level vision, the phakic eye competes with the surgically treated aphakic eye for cortical connections. The phakic eye must be disadvantaged by occlusion to obtain a reasonable outcome in the aphakic eye.

However, we discovered that the interaction between the eyes is very different for aspects of vision that depend on cortical areas beyond the primary visual cortex. In our studies of sensitivity to global motion described in Section 3, we found that motion coherence thresholds were about five times worse than normal after binocular deprivation. Much to our surprise, motion coherence thresholds after monocular deprivation were only 1.6 times worse than normal and three times better than after binocular deprivation (Fig. 1). The deficits were neither related to the amount of time that the phakic eye had been patched after monocular deprivation nor to age of treatment in monocularly or binocularly deprived cases. Moreover, in unilateral cases, the deficits were as pronounced in the phakic eye as in the surgically treated aphakic eye.

Better motion coherence thresholds after monocular than after binocular deprivation and the comparable motion deficits in both eyes suggest that the deficits originated in higher areas of the visual pathway where there is convergence of inputs from the two eyes across large areas of the visual field that might allow a relative sparing of function after monocular deprivation. The pathway for the perception of global motion likely involves the middle temporal cortex in the dorsal visual stream.[47,50,51] We found a similar pattern—better performance in the aphakic eye after monocular than after binocular deprivation on the tests of global form,[52] which likely involves area V4 in the ventral visual stream.[47] Thus, the effects of early visual input on high-level vision involving networks in the extrastriate cortex are quite different from its effects on low-level vision involving principally the primary visual cortex. In addition, our finding suggest that the size of the deficit after monocular deprivation was similar for global motion and global form cautions against a general conclusion that the dorsal pathway is more plastic than the ventral pathway, as has been reported for other patient populations.[53–55]

5. There are multiple sensitive periods during which experience can influence visual development

Because of the seminal work of Wiesel and Hubel,[56] we have known for nearly half a century that there is a critical period early in life during which normal visual input is necessary for normal

visual development. Because acuity is mature by age 7 in visually normal children, clinicians traditionally have assumed that treatment for amblyopia before that age will improve vision. Conversely, they have assumed that because vision has stabilized by age 7 during normal development, the window of plasticity essentially closes and treatment after age 7 will have little or no effect on vision. However, these traditional notions need to be reevaluated for two reasons. First, we now know that the critical period for visual acuity is not necessarily the same as the critical period for other aspects of vision. Second, the age of maturity does not necessarily correspond to the age at which the system is no longer susceptible to damage nor to the age after which recovery is impossible.[57]

Harwerth et al.[58] were the first to establish that, at least in the monkey, there are different critical periods for the normal development of different aspects of vision. Specifically, they found that deprivation had to begin before 3 months of age to affect scotopic sensitivity, before 6 months of age to affect photopic spectral sensitivity, and before 18 to 24 months of age to affect spatial contrast sensitivity. Surprisingly, binocularity was abnormal even when deprivation began after 2 years of age.

Children who are born with normal vision but then later develop a dense cataract in one or both eyes provide an opportunity to determine if humans, like monkeys, have different critical periods for different aspects of vision. To find out, we measured sensitivity to global motion and grating acuity in 15 such patients who had developed dense cataracts in one or both eyes between the ages of 4 months and 15 years, had suffered an average of 2 to 3 months deprivation in the aphakic eye, and were tested at least 5 years after the cataract was removed.[13] We were surprised to find that the sensitive period for damage to global motion was very short, much shorter than that for grating acuity. Specifically, for every aphakic eye in the unilateral and bilateral developmental groups, motion coherence thresholds were normal, even when the onset of deprivation was as early as 4 months of age (see Fig. 1 for group means). Yet, every one of the 13 patients with onset of deprivation before 5 years of age had abnormal grating acuity in at least one eye. The two patients with later onset of deprivation (>11 years) had normal grating acuity in each eye. Thus, the normal development of grating acuity depends on visual input until at least 5 years of age. These results contradict the traditional belief that sensitive periods are longer at higher than at lower levels of the visual system.[59] Rather, the sensitive period for damage to global motion appears to be very short, much shorter than that for low-level aspects of vision such as grating acuity (which lasts until at least 5 years of age), Snellen acuity (which lasts until about 10 years of age), and peripheral vision (which lasts until at least the early teenage years).[57]

Even within one aspect of vision, such as acuity, we have found evidence for more than one sensitive period.[57] The classic definition of the sensitive period is the time during normal development when normal input is necessary for a normal outcome. Thus, it corresponds to the period during which there are developmental changes in an organism raised with visual input that do not occur if the visual input is missing. We call this the period of visually-driven normal development. However, for some aspects of vision, abnormal visual input can have a permanent deleterious effect even when the abnormal input starts after that aspect of vision is functionally adult like. Thus, a second sensitive period is the time of vulnerability, including any time of vulnerability after normal development is complete. We call this the sensitive period for damage. A third sensitive period is the time during which the visual system has the potential to recover from the deleterious effects of deprivation. We call this period the sensitive period for recovery. With acuity, for example, these three sensitive periods appear to last for about 6, 10, and 5 to 7 years, respectively.[57]

Note, however, that conclusions about the sensitive period for recovery being over by 5 to 7 years of age are based on the common assumption that improvements in acuity after that age are unlikely, if not impossible. Thus, clinicians typically discontinue occlusion therapy by 7 years of age for all types of amblyopia, so long as acuity remains stable.[60] Because occlusion is typically discontinued when acuity stabilizes, it is not surprising that until recently, there has been little evidence of improvement in acuity in amblyopic patients older than age 7. We revisit this topic below when we consider the limits to adult plasticity.

Implications for Normal Development

The studies described here indicate that patterned visual input immediately after birth plays a vital role in the construction and preservation of the neural architecture that will later mediate sensitivity to both basic and higher level aspects of vision. For example, the low spatial frequencies that newborns can see set up a system for later development of fine acuity and sensitivity to mid spatial frequencies of low contrast. Both systems are refined by later visual experience—but only if their basic architecture was set up (or maintained) by stimulating the crude vision of the newborn. When the timing of that crude early visual experience is delayed until cataracts are removed, some visual capabilities will show sleeper effects: they will fail to emerge at a later point in childhood, perhaps, because the requisite neural architecture is no longer available. The period during which patterned visual input is necessary for normal visual development varies widely across different aspects of vision and can range from only a few months after birth to more than the first 10 years of life.

FOOD FOR THOUGHT

Why Does Early Visual Deprivation Cause Deficits in Later Vision?

Do visual deficits after early deprivation occur because the usual pathways have been damaged or because the system has come to use alternative pathways or alternative visual structures that are not designed to do the job as effectively?

What are the Limits to Adult Plasticity?

We have described a sensitive period for recovery, a period during which the visual system has the potential to recover from the deleterious effects of deprivation. However, recent studies demonstrate that, even in adulthood, it is possible to recover at least some spatial vision with extensive training, at least after early strabismus or anisometropia.[60] What is the best training regimen and how permanent are any observed improvements? Can playing action

video games improve basic aspects of vision? Will action games such as Wii improve vision, perhaps by training visual-spatial attention? Can drugs such as dopamine improve vision, perhaps by increasing synaptic efficiency?

Can Looking Beyond Vision Provide New Clues?

Treatment for amblyopia has not changed in over 100 years: remove the peripheral problem (e.g., the cataract) and patch the eye with better acuity until about age 7. Yet, recent evidence from other domains suggests that the adult brain is still plastic. What lessons can we learn about treating poor vision by studying the methods used during adulthood to overcome deficits in hearing, or to learn a second language, or to recover from a stroke? For example, first-time stroke patients suffering severe hand and motor impairment benefit from repetitive transcranial magnetic stimulation (rTMS) over the motor cortex.[61] Similarly, adult strabismic or anisometropic amblyopes given 10 Hz rTMS over the primary visual cortex (but not over the motor cortex) show improvements in contrast sensitivity, at least under some conditions, on retests immediately after stimulation and 30 min later.[62] Although the beneficial effects were no longer evident on 1-week follow-up tests, the technique holds promise as a possible treatment for adult amblyopia.

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