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Multiple Sensitive Periods in the Development of the Primate Visual System

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Early in life, abnormal visual experience may disrupt the developmental processes required for the maturation and maintenance of normal visual function. The effects of retinal image deprivation (monocular form deprivation) on four psychophysical functions were investigated in rhesus monkeys to determine if the sensitive period is of the same duration for all types of visual information processing. The basic spectral sensitivity functions of rods and cones have relatively short sensitive periods of development (3 and 6 months) when compared to more complex functions such as monocular spatial vision or resolution (25 months) and binocular vision (>25 months). Therefore, there are multiple, partially overlapping sensitive periods of development and the sensitive period for each specific visual function is probably different.

A FUNDAMENTAL CONCEPT OF VISUAL development is that there are "critical" or "sensitive" periods during which the infant sensory system requires adequate stimulation for neural information processing mechanisms to progress toward their normal, adult characteristics (*1*). During this sensitive period, there is considerable nervous system plasticity and adverse environmental factors can disrupt the normal developmental process. After the sensitive period is over, abnormal sensory environments can no longer permanently modify the response properties of visual system neurons. However, recent studies have shown that there is not a unitary sensitive period for the whole visual system, but rather, the sensitive periods of development are different for various levels of the visual pathway (*2*). Even at a given level, the different response characteristics of visual neurons have different sensitive periods (*3*). Therefore, psychophysical measures of visual system function would be expected to show different sensitive periods for the processing of different types of visual stimuli. This prediction is confirmed here by the results of behavioral studies of sensitive periods of visual development in monkeys. Specifically, we found that (i) the sensitive period for scotopic spectral sensitivity, an index of the ability of the rod system to respond to various wavelengths of light, ends at about 3 months of age, (ii) the sensitive period for photopic increment threshold spectral sensitivity, an index of cone information processing, is over by 6 months of age, (iii) the sensitive period for spatial vision (spatial modulation sensitivity, a measure of form vision) lasts until about 25 months of age, and (iv) the sensitive period for binocular vision (binocular summation) is longer than 25 months.

The durations of the sensitive periods were determined from an investigation of the alterations of visual function produced by monocular form deprivation (lid suture) initiated at various ages ranging from 1 to 25 months. The duration of deprivation was 18 months for each of the 11 rhesus monkeys (*Macaca mulatta*) that we used (*4*). This relatively long period of monocular deprivation was used so that any remaining plasticity within the visual system would be minimal after the eyelids were parted and the vision defects caused by the deprivation would be stable. At the end of the deprivation period the animals were trained to perform a psychophysical detection task, which has been described (*5*). The task required the monkey to press and hold down a response lever to initiate a trial and then to release the lever within a criterion time (*6*) after a visual test stimulus was presented. If the animal released the lever within the criterion time, we assumed that he had detected the stimulus and we rewarded him with a tone (1.6 kHz) and, in 75 percent of the trials, with liquid (0.5 ml of orange juice). After each rewarded trial we reduced the intensity of the test stimulus by 0.1 or 0.05 log units for the next trial. This trial sequence was continued until the animal failed to release the lever within the criterion time in two consecutive trials. The intensity of the stimulus at this time is defined as the threshold intensity for the particular test wavelength or spatial frequency. The same basic procedure was used to collect data for scotopic spectral sensitivity functions, photopic increment threshold spectral sensitivity functions (3000 Troland achromatic background), and spatial modulation sensitivity functions for monocular and binocular viewing conditions (*7*).

The shortest sensitive period was found for scotopic spectral sensitivity. Although the dark-adapted spectral sensitivity functions for both eyes of all of the monkeys were well fit by the scotopic luminosity function for the standard human observer (*8*), the sensitivities of the deprived eyes of the subjects initially deprived at either 1 or 2 months of age were considerably depressed (3 to 4 log units) when compared to the sensitivities for their nondeprived eyes (Fig. 1A). In contrast, in all of the monkeys which were form deprived at 3 months of age or later, the two eyes had equal sensitivities. The sensitivity ratios in Fig. 1A are for a test wavelength near the peak of the scotopic spectral sensitivity function (500 nm), but since the shapes of the curves were invariant, the ratios are also representative of any other wavelength. Therefore, form deprivation instituted early in life caused substantial sensory deficits for visual functions mediated by the rod photoreceptors. However, the period of sensitivity for these deficits ended by 3 months of age.

The sensitive period for the neurosensory

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interactions involved in the processing of stimulus information during a photopic increment-threshold task (*9*) was somewhat longer than that for the scotopic luminosity task (up to 6 months). The spectral sensitivity defects of the deprived eye of the animal initially treated at 2 months of age were similar to those of the animal treated at 1 month (Fig. 1B). However, as the age of the onset for form deprivation was further delayed, the spectral sensitivity differences between the nondeprived and deprived eyes systematically decreased. The animals initially deprived at 6 months of age or later exhibited identical spectral sensitivity functions for their treated and untreated eyes.

In Fig. 1B data are included for three stimulus wavelengths in order to illustrate that both a decrease in absolute sensitivity and an alteration in the shape of the spectral sensitivity function were produced by early monocular form deprivation. The spectral sensitivity function of monkeys experimentally deprived of form vision at 1 month of age conforms to the scotopic luminosity function rather than the three-peaked function typically obtained for photopic increment-threshold spectral sensitivity tasks for observers with normal trichromatic vision (*10*). This is seen (Fig. 1B) by the larger interocular sensitivity ratios for the long wavelength (600 nm) stimulus in comparison to those for the middle (520 nm) or short (440 nm) wavelength stimuli. Even for the middle wavelength stimulus, the condition that revealed the smallest reduction in absolute sensitivity, there was a difference (2 log units) in the sensitivities for the nondeprived and deprived eyes. Thus, there were two components to the sensitive period for photopic increment-threshold spectral sensitivity. With very early monocular form deprivation (2 months of age or less), in addition to there being a large decrease in sensitivity, the deprived eye's spectral sensitivity appeared to be determined by scotopic mechanisms even at photopic adaptation levels. Form deprivation initiated between 3 and 5 months of age resulted in a relative sensitivity deficit of the deprived eye, but in this case the sensitivity function is definitely determined by photopic mechanisms for both eyes. Finally, the sensitive period for this visual function ended by 6 months of age, since form deprivation at this age or later had no effect on the increment-threshold spectral sensitivity of the monkey eye.

In comparison to the sensitive periods for either of the spectral sensitivity functions, the sensitive period for spatial vision was quite long. The spatial vision defects (Fig. 1C) of the form-deprived animals were characterized by the interocular ratio of the high

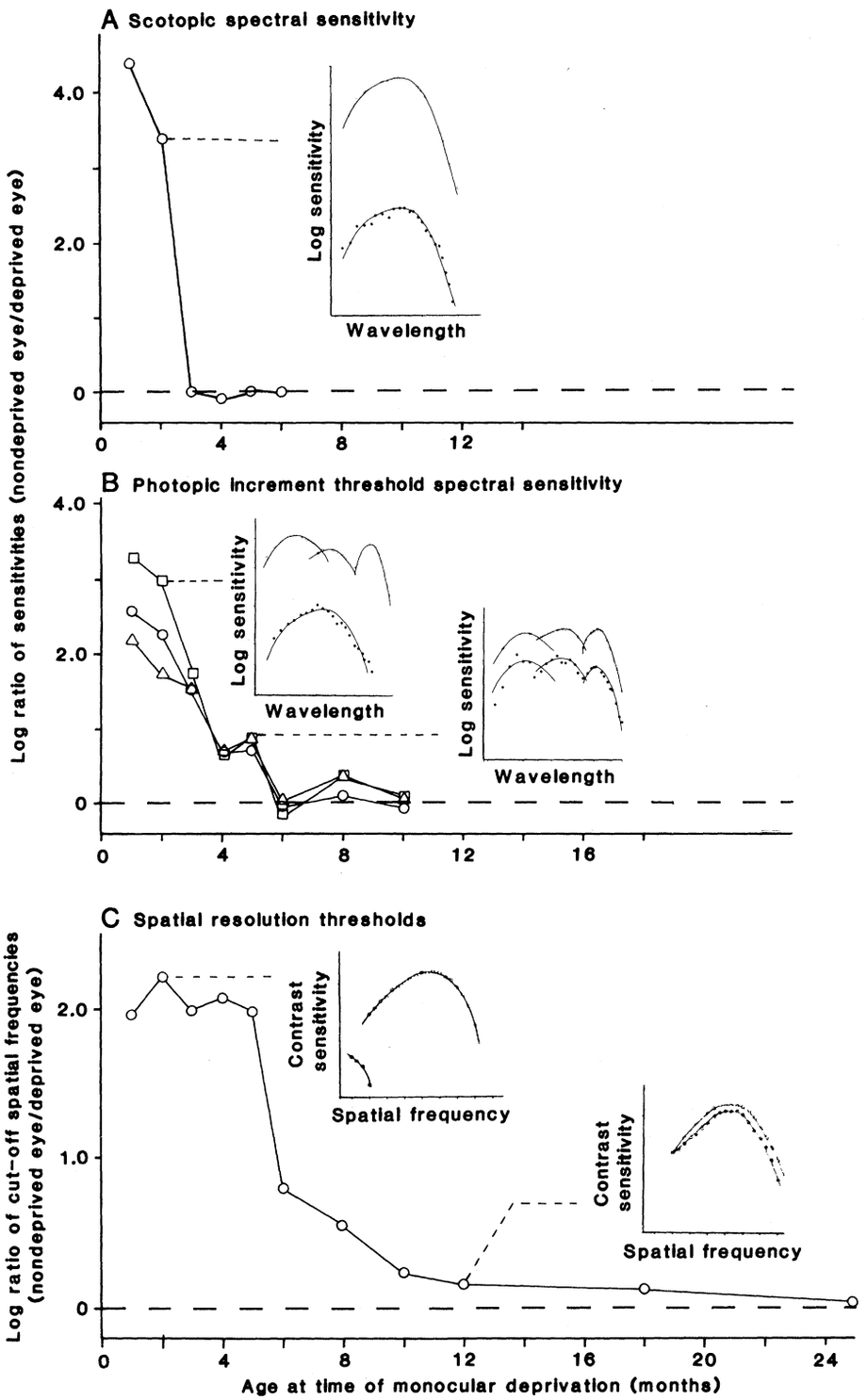

Fig. 1. Psychophysically determined interocular sensitivity ratios as a function of the ages of the monkeys at the time that monocular form deprivation was initiated. (A) Interocular spectral sensitivity ratios obtained under dark-adapted viewing conditions. The wavelength of the test stimulus was 500 nm. The inset shows the complete scotopic spectral sensitivity curves for the monkey deprived at 2 months of age. (B) The interocular sensitivity ratios for the photopic increment-threshold spectral sensitivity task obtained with a 3000 Troland achromatic adaptation field. Sensitivity ratios are shown for stimulus wavelengths of 440 nm (○), 520 nm (△), and 600 nm (□). The two insets represent the complete spectral sensitivity functions for animals deprived at either 2 or 5 months of age. (C) The interocular acuity ratios derived from the extrapolated cutoff spatial frequencies of the spatial modulation sensitivity functions. The two insets represent the spatial modulation sensitivity functions for animals deprived at either 2 or 12 months of age.

spatial frequency cutoff values derived from the animals' spatial modulation sensitivity functions. The cutoff spatial frequency is a measure of the resolution capability of the eye and is defined as the finest detectable grating having 100 percent contrast. The interocular ratios of the cutoff spatial frequencies (Fig. 1C) for all of the monkeys deprived at 5 months of age or earlier are very similar, with about a 2-log-unit difference in the cutoff spatial frequencies between the treated and control eyes (cutoff values of approximately 30 and 0.3 cycles per degree for the nondeprived and deprived eyes, respectively). The animals initially deprived between 6 and 12 months of age each showed successively lower interocular acuity ratios, but only the animal initially treated at 25 months of age had equal visual acuity values for both eyes. Therefore, the investigation of spatial vision anomalies showed that all of the monkeys with reduced photopic spectral sensitivities, that is, those treated at 5 months of age or earlier, had profound spatial vision defects, whereas those treated at later ages until about 25 months of age had progressively smaller deficits when the sensitive period for the development for spatial vision has apparently ended.

The last visual function investigated in these monkeys was binocular summation; that is, the improvement in visual performance with binocular viewing when compared to monocular viewing. In human or monkey subjects with normal binocular vision the expected improvement in sensitivity with binocular viewing is at least 40 percent (0.15 log unit) for spatial modulation sensitivity measurements (*11*). Our previous experiments have also shown that binocular summation is closely correlated with stereopsis (*12*) and the presence of binocularly driven neurons in the striate cortex (*13*). Therefore, the binocular summation experiments allowed us to determine the sensitive period of development for the binocular interactions required for the fusion and stereopsis.

The binocular summation ratios for two experimental animals (deprived at 18 and 25 months) were close to zero, showing a complete lack of benefit from viewing the stimuli with both eyes rather than one (Table 1). On the other hand, the summation ratios for two untreated control animals were within the range of values expected for subjects with normal binocular vision. These data were taken for stimulus frequencies at the peaks of the contrast sensitivity functions (2.51 cycles per degree), but they are representative of every stimulus condition tested. Therefore, the sensitive period for the development of the neural interactions required for normal binocular vision and stereopsis has not ended by 25 months of age.

Table 1. Binocular summation data for the experimental animals monocularly form deprived at 18 months (E-18) or 25 months (E-25) and two normally reared control monkeys (C-1 and C-2). Log contrast sensitivity data (logarithm of the reciprocol of the threshold contrast for the detection of a 2.51 cycle per degree grating) and standard errors are shown for monocular and binocular viewing conditions. The summation ratio is the difference between the log contrast sensitivities for binocular viewing conditions and the left eye monocular viewing conditions. Each number is the mean of 12 threshold determinations.

Subject	Log contrast sensitivity			Summation ratio
	Right eye	Left eye	Both eyes	
E-18	1.64 ± 0.03	1.95 ± 0.02	1.90 ± 0.02	−0.05 ± 0.03
E-25	1.91 ± 0.05	1.97 ± 0.04	1.91 ± 0.03	−0.06 ± 0.05
C-1	2.14 ± 0.04	2.14 ± 0.04	2.32 ± 0.04	0.18 ± 0.06
C-2	1.97 ± 0.02	2.01 ± 0.04	2.19 ± 0.03	0.18 ± 0.05

Our experiments indicate that the sensitive period of development for each visual function is probably different. In addition, the overall organization of sensitive periods appears to be hierarchical. That is, visual functions primarily requiring information processing in the peripheral portions of the visual system have shorter sensitive periods than those requiring more central processing. For example, most of the adaptation and light transduction processes involved in regulating the sensitivity of the scotopic visual system are thought to be located in the retina (*14*) and the sensitive period for scotopic visual defects is quite short. In contrast, the sensitive period for purely binocular functions, which are believed to rely completely on interneuronal interactions in the visual cortex (*15*), are much longer. While our psychophysical data do not suggest a mechanism or site for sensitive period differentiation, the suggested organizational principle is in agreement with the available physiological data for monkeys (*2*). However, more evidence will be required to specify the possible links between anatomy, physiology, and behavior.

One of the important aspects of investigating sensitive periods of development in monkeys is that the data may be directly applicable to certain conditions in humans. In order to use the developmental data from monkeys to estimate the sensitive periods of children, it is important to compensate for the relative rates of development of the two species. A comparison of the development of visual acuity in human and macaque infants indicates an approximately 4:1 ratio for their rates of development (*16*). With this ratio, it would be predicted that factors such as traumatic cataract or ptosis incurred anytime before 8 years of age could cause amblyopia (if the condition was left untreated for a long period of time) and could cause a loss of binocular vision and stereopsis at even later ages. The estimate of 8 years as the end of spatial vision plasticity is in reasonable agreement with estimates of the sensitive period for spatial vision obtained from assessments of visual acuity of children suffering traumatic cataract (*17*). In conclusion, it is evident from the correlation of the available data from monkeys and humans that the concept of multiple sensitive periods of development has important ramifications for both basic studies of development and the clinical management of children with vision disorders.

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New Human T-Lymphotropic Retrovirus Related to Simian T-Lymphotropic Virus Type III ($STLV\text{-}III_{AGM}$)

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This report describes serologic evidence for a virus similar to that known as simian T-lymphotropic virus type III of African Green monkeys ($STLV\text{-}III_{AGM}$) infecting apparently healthy people in Senegal, West Africa, and the isolation of virus from these individuals. Serum samples from selected healthy West African people showed unusual serologic profiles when tested with antigens of HTLV-III/LAV, the etiologic agent of AIDS, and of $STLV\text{-}III_{AGM}$. The samples reacted strongly with all of the major viral antigens of $STLV\text{-}III_{AGM}$ but showed variable or no reactivity with the major viral antigens of HTLV-III/LAV by radioimmunoprecipitation and sodium dodecyl sulfate–polyacrylamide gel electrophoresis. A new human T-lymphotropic virus (HTLV-IV) isolated from these people was grown in vitro and shown to have retroviral type particles, growth characteristics, and major viral proteins similar to those of the STLV-III and HTLV-III/LAV group of retroviruses. The gp120/160, gp32, p64, p55, p53, p24, and p15 proteins precipitated were the same size as and reactive with $STLV\text{-}III_{AGM}$ proteins. The serologic data suggest that this virus shares more common epitopes with $STLV\text{-}III_{AGM}$ than with the prototype HTLV-III/LAV that infects people in the United States and Europe. Further study of this virus and of the origin of the HTLV-III/LAV group of viruses may expand our understanding of the human AIDS virus.

IT HAS RECENTLY BEEN RECOGNIZED that the T-lymphotropic retrovirus family includes not only human retroviruses (HTLV-I, HTLV-II, and HTLV-III/LAV) and bovine leukemia virus but also closely related agents that infect certain nonhuman primate species. The simian T-lymphotropic virus type I (STLV-I) naturally infects most species of Old World monkeys and great apes (*1*). Similar to its human counterpart, STLV-I immortalizes T lymphocytes in vitro and has been linked with spontaneous lymphoid malignancy in the primate host (*1, 2*). STLV-III has been described in both captive ill rhesus macaques (*Macaca mulatta*) and healthy wild-caught African Green monkeys (*Cercopithecus sp.*) (*3–5*). This virus has a cytolytic effect on T4 lymphocytes in culture, Mg^{2+}-dependent reverse transcriptase, and retroviral particles with morphology similar to HTLV-III/LAV, the etiologic agent of AIDS (*3–7*). The major STLV-III proteins have been identified by radioimmunoprecipitation and sodium dodecyl sulfate–polyacrylamide gel electrophoresis (RIP/SDS-PAGE) as 160 kilodaltons (kD), 120 kD, 55 kD, 24 kD, and 15 kD similar to and cross-reactive with the major antigens of HTLV-III/LAV (*3, 5*). Serologic studies on a variety of African primates indicated that approximately 50 percent of African Green monkeys (*Cercopithecus aethiops*) were seropositive for STLV-III ($STLV\text{-}III_{AGM}$), whereas chimpanzees (*Pan troglodytes*), baboons (*Papio sp.*), patas monkeys (*Erythrocebus patas*), and colobus monkeys (*Colobus polykomos*) were seronegative (*5*). Antibodies to $STLV\text{-}III_{AGM}$ have been identified in sera from *Cercopithecus* species sampled as early as 1961 (*8*).

The African Green monkey and other members of the genus *Cercopithecus* are widespread throughout most parts of tropical Africa. The close relation of $STLV\text{-}III_{AGM}$ to HTLV-III/LAV raised the possibility that a family of related viruses may have existed in primates well before the AIDS epidemic began. Therefore, we speculated that STLV-III may have been transmitted to humans at some time during the natural history of these viruses (*5*). AIDS cases may have been present in Central Africa in the mid-1970's (*9*) before the disease was recognized in the United States and Europe. Recent studies indicate that AIDS is endemic in Central Africa with significant transmission occurring in the heterosexual population (*10*). Serologic studies of HTLV-III/LAV indicate that this or a related virus may have been present in Africa as early as 1972 (*11*). We have therefore investigated further the possibility that these viruses may have had a common origin.

By using the differential reactivity of HTLV-III/LAV–positive human sera for $STLV\text{-}III_{AGM}$ viral antigens, we have demonstrated that certain apparently healthy people in Senegal, West Africa, have antibodies that are more strongly reactive with $STLV\text{-}III_{AGM}$ antigens than with the analogous antigens of HTLV-III/LAV by RIP/SDS-PAGE. This reactivity was indistinguishable from that seen in monkeys infected with $STLV\text{-}III_{AGM}$. We subsequently

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