12 Development of the Tectofugal Visual System of Normal and Deprived Zebra Finches

Kathrin Herrmann and
Hans-Joachim Bischof

Extensive studies in the mammalian visual system have demonstrated that the specificity of connections that characterizes the adult nervous system results from a complicated interplay between genetic and environmental influences early in an animal's life. Important insights into the problem of visual system development came from the studies of Hubel and Wiesel, who demonstrated that monocular deprivation and other manipulations of the rearing conditions during early infancy profoundly influence the morphological and physiological development of the geniculocortical pathway of mammals (review in Wiesel, 1982). To determine whether similar processes played a role in the development of the avian visual system, we examined the effects of monocular and binocular deprivation on the normal development of the visual system of zebra finches.

We chose the zebra finch (*Taeniopygia guttata castanotis*) because it is an altricial species, born very immature with closed eyes, after an in ovo period of only 13 days, so that most of its visual system development takes place posthatching. An ethological study of the ontogeny of visual function suggested that young birds do not react to visual stimuli before day 10, which is 4 or 5 days after eye opening (Bischof and Lassek, 1985).

In zebra finches, as in many other avian species (e.g., chickens, pigeons), the eyes are situated laterally, so that the binocular visual field is rather narrow (± 15°, Bischof, 1988), relative to the monocular field (about 150°). These size differences of the binocular and monocular visual fields are correlated with differences in the relative size of the tectofugal and thalamofugal visual systems, which have been thought to process information about the unilateral and bilateral visual fields, respectively (but see chapter 8). The thalamofugal system has been homologized with the geniculostriate system of mammals; the tectofugal system has been assumed to correspond to the mammalian extrageniculostriate system (Nauta and Karten, 1970). Our studies focused on the development of the tectofugal system because of its extensive size in this lateral-eyed species.

Previous anatomical studies have shown that the tectofugal system conveys information from the retina via the tectum opticum to the nucleus rotundus of the thalamus and thence to a telencephalic nucleus, the ectostriatum (Benowitz and Karten, 1976; Nixdorf and Bischof, 1982). Because the optic nerve crosses completely in the optic chiasm, it was originally assumed that the tectofugal pathway processes information exclusively from the contralateral eye (figure 12.1). However, there is evidence suggesting that tectofugal nuclei (rotundus, ectostriatum) may also process information from the ipsilateral retina. Physiological data demonstrate that the ectostriatum can be driven by stimulation of either eye (Engelage and Bischof, 1988, 1990; this volume), although up to now there are no reports of truly binocular neurons in any tectofugal nucleus. There are at least three sources for such input: the projection from the visual wulst back to the tectum opticum (Bagnoli et al., 1980), the tectotectal projection (Robert and Cuenod,

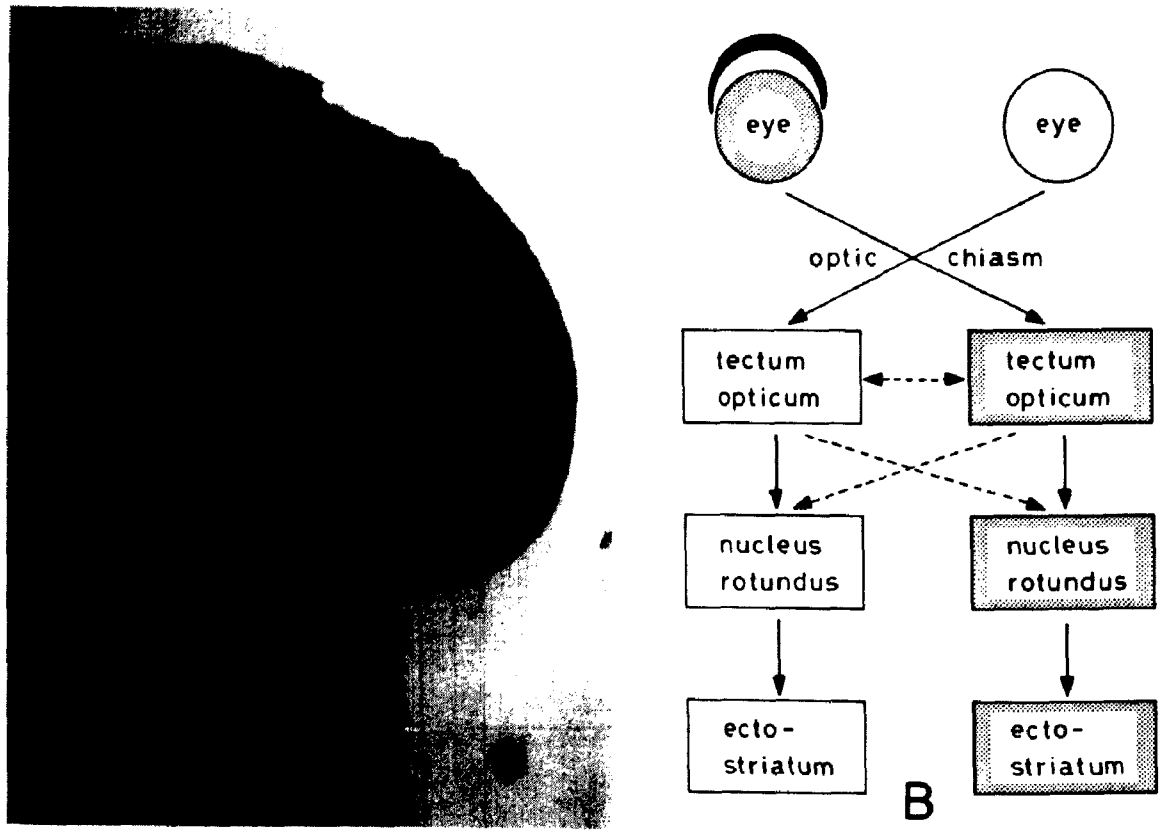


Figure 12.1 (A) Nissl-stained cross section through the right hemisphere of a zebra finch brain, showing the main stations of the tectofugal visual pathway: retinal axons (Ret) enter superficial layers of the contralateral tectum opticum (TO). Visual information is then processed to deeper tectal layers. From there, visual information in transferred via the nucleus rotundus in the thalamus (Rt) to the telencephalic way-station of this pathway, the ectostriatum (E). (B) Due to the complete crossing of the optic nerve in the optic chiasm, monocular deprivation in birds creates a "deprived" hemisphere contralateral to the closed eye (graphically depicted by shading). The stippled lines, however, show interhemispheric projections that seem to be of much greater importance than previously expected. From Herrmann and Bischof (1986b).

1969; Bischof and Niemann, 1990), and the projection from the tectum opticum to the contralateral nucleus rotundus (Benowitz and Karten, 1976; Bischof and Niemann, 1990, figure 12.1B). A transient ipsilateral retinotectal connection, which has been demonstrated in chicks (O'Leary et al., 1983) and in pigeons (Bagnoli et al., 1983) is not detectable in adult birds and thus cannot be a source for the input from the ipsilateral eye.

It is, therefore, not improbable that binocular interactions are possible in all three relays of the tectofugal pathway. This is an important possibility, since, in mammals, deprivation-induced changes in visual system development have been attributed almost exclusively to unbalanced binocular competition (e.g., Wiesel and Hubel, 1965; Hubel et al., 1988; Guillery, 1972). The data presented in this chapter demonstrate that, as in mammals, monocular deprivation does effect the anatomical development of tectofugal nuclei, and that most of these effects are best interpreted as effects of a crosstalk of the tectofugal systems of both sides of the brain.

NORMAL DEVELOPMENT OF THE NUCLEUS ROTUNDUS AND THE ECTOSTRIATUM

Nucleus Rotundus

The nucleus rotundus is the largest thalamic area in adult zebra finches, with a volume of about 0.4 mm^3. At birth, however, its volume comprises only one-fifth of the adult volume. Nucleus rotundus quadruples in size between birth and day 10, and by day 20 it is significantly (ca. 20%) larger than in adults.

The development of individual rotundal neurons shows a similar pattern, i.e., an increase in size between birth and day 20 followed by a reduction until adulthood (figure 12.2a). Myelination of axons within the nucleus starts between days 5 and 10, and the adult pattern is achieved between days 20 and 40 (Herrmann and Bischof, 1986a). Electron microscopic data indicate that the density of synapses, as well as the size of the presynaptic terminals, increase steadily between hatching and day 20, the largest increase in synapse density occurring between days 5 and 10, around the time of eye opening (Nixdorf and Bischof, 1986).

Ectostriatum

The development of zebra finch ectostriatum (figure 12.2a) follows a time course similar to that of rotundus. Cell size increases between birth and day 20 and decreases thereafter. The myelination process of ectostriatal axons starts slightly later than in rotundus, between days 10

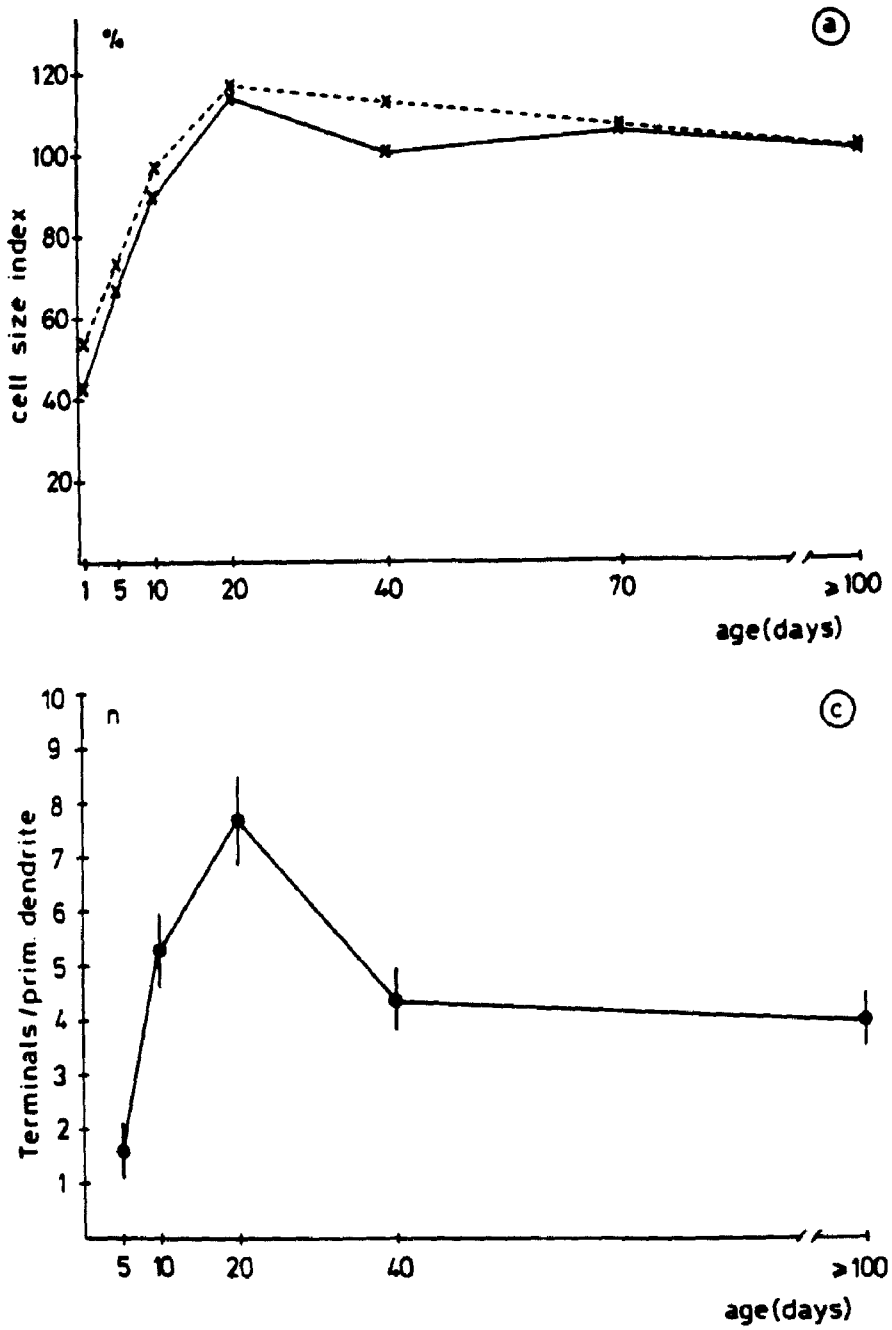
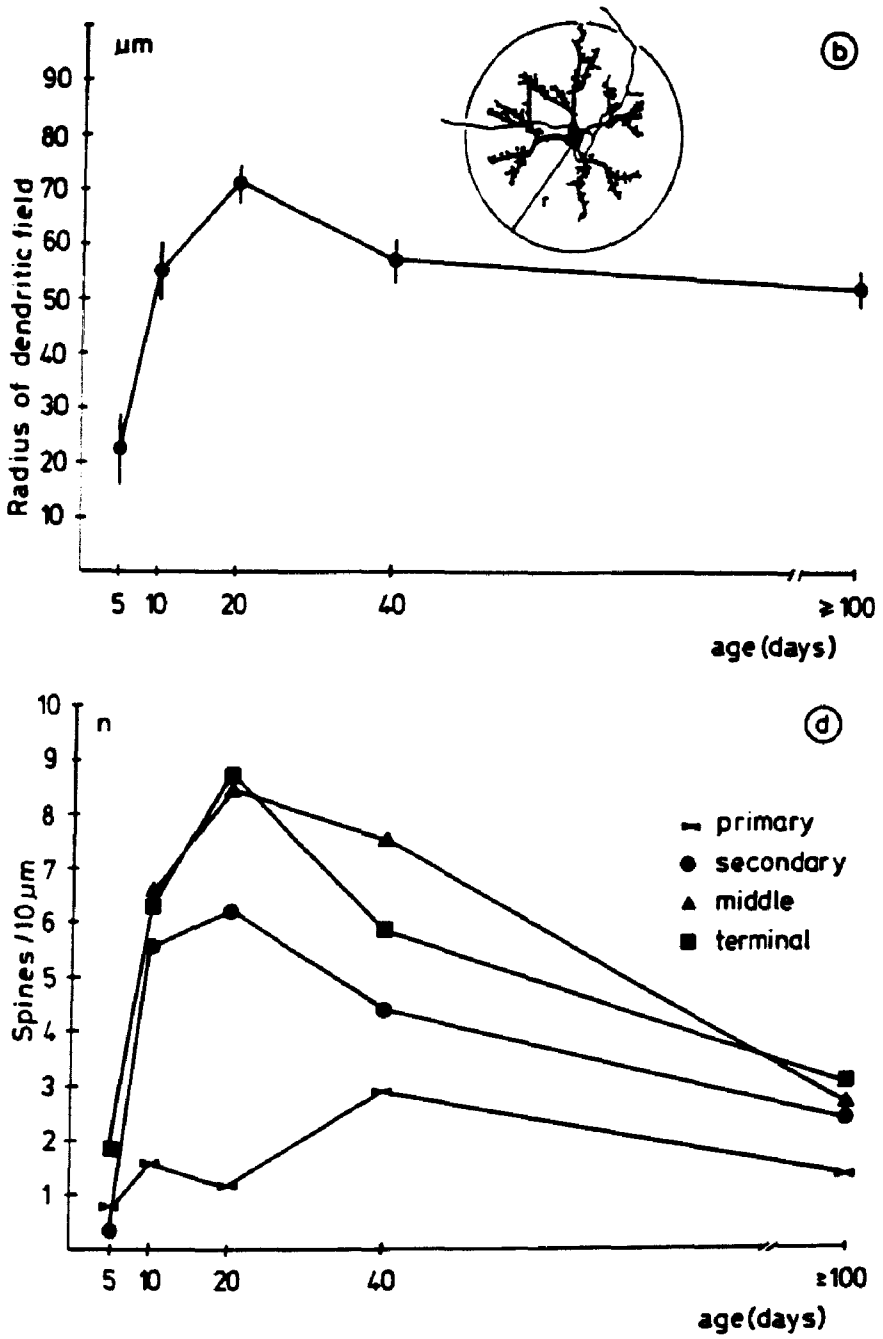


Figure 12.2 Time course of development of different neuronal elements. (a) Neuron size (% of adult values). Stippled line, ectostriatum; full line, n. rotundus. After Herrmann and Bischof (1986a). (b–d) Ectostriatal measurements. (b) Average radius of dendritic field. (c) Branching index: number of terminal dendritic segments per primary dendrite.



(d) Number of spines per 10 µm for different segments of the dendrite. Primary, segments directly adjacent to the cell body; secondary, segments following the primary ones; terminal, end segments of each dendrite; medial, segments that were not primary or terminal. After Herrmann and Bischof (1988a).

and 20, adult myelin density being achieved by 40 days (Herrmann and Bischof, 1986a).

Using the Golgi-impregnation technique, which stains not only cell bodies but also their axons and dendrites, we have followed the morphological development of ectostriatal neurons. The major neuron type in this structure resembles the spiny stellate cell of mammalian neocortex, with a soma diameter of about 15 μm and three to five radially oriented primary dendrites. Each dendrite branches three or four times and bears a significant number of dendritic spines of different morphology. The dendrites extend about 55 μm from the soma but never leave the ectostriatum. Axons could be traced in some preparations as far as 100 μm. They sometimes arborize, and single axon collaterals could be traced into the paleostriatum or the neostriatum (Herrmann and Bischof, 1988b). The same neuron type has been reported for the ectostriatum of chicken (Tömböl et al., 1988) and quail (Watanabe et al., 1988).

Figure 12.3 illustrates selected examples of the development of the main ectostriatal neuron type. The most rapid development occurs between days 5 and 10, at the time of eye opening. Neurons of 5-day-old finches are typically undifferentiated cells, with irregularly thickened, short dendrites bearing growth cones and filopodia. Between days 5 and 10 there is a tremendous growth spurt, reflected in increasing soma diameter, the growth and bifurcation of dendrites, and the occurrence of numerous thin dendritic spines. Between days 10 and 20 there is a further increase in dendritic length and branching frequency. After day 20, a substantial reduction in dendritic length, branching frequency, and the number of spines can be detected.

A quantitative analysis of these parameters was carried out and the results are plotted in figure 12.2b–d. Taken together there seems to be a good correlation between cell size, nucleus volume, dendritic field size, number of branch points, and number of dendritic spines. All these parameters show an increase between birth and day 20, followed by a decrease to adulthood. Thus early neuronal development in the zebra finch, as in other birds and mammals, is characterized by both proliferative and regressive phenomena (see, e.g., Changeux and Danchin, 1976; Herrmann and Bischof, 1986a).

The transient overproduction of neuronal elements is often interpreted as providing conditions under which selection processes can eliminate nonfunctional synapses while stabilizing functional ones. The model, however, implies the existence of competitive processes that have not been thus far demonstrated directly for the tectofugal system. Indirect evidence, however, indicates that binocular competition may be possible in the ectostriatum. Engelage and Bischof (1988) have recently shown that ipsilaterally evoked visual potentials could be recorded as acutely enucleated zebra finches, suggesting that there may

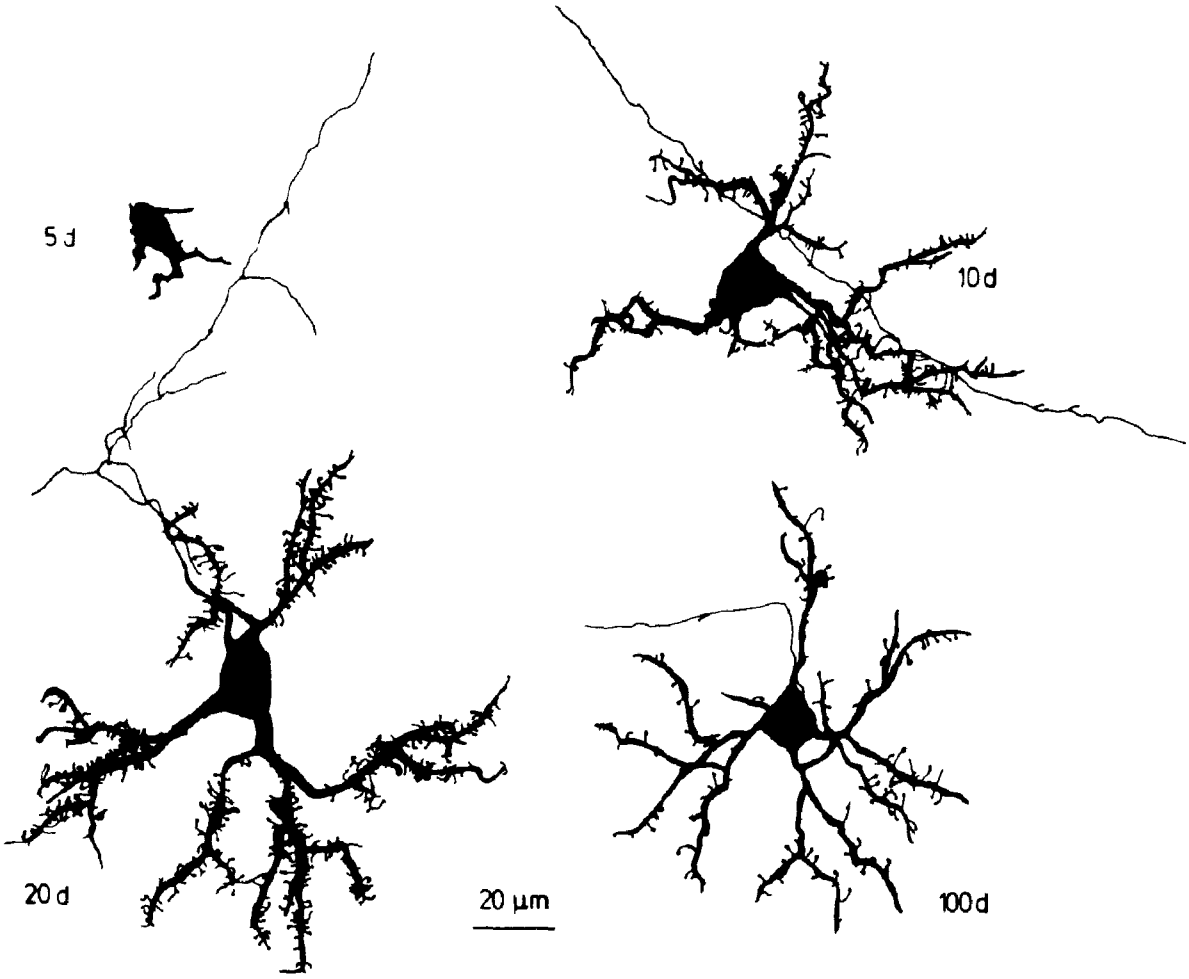


Figure 12.3 Typical examples of ectostriatal neurons at postnatal days 5, 10, 20, and 100. The most pronounced growth spurt occurs between days 5 and 10, right around the time of eye opening. From Herrmann and Bischof (1988b).

indeed be binocular interaction, which is difficult to detect under normal conditions (see chapter 8).

Against this background, then, the remainder of the chapter examines the effects of monocular deprivation on the retina and the tectofugal pathway.

EFFECTS OF MONOCULAR DEPRIVATION

Retina

It is now well established that monocular deprivation in birds, as in mammals, causes a substantial elongation in the anterior-posterior length of the eyeball accompanied by a major myopia (Bagnoli et al., 1985; Wiesel and Raviola, 1977; Yinon et al., 1982, 1983). Surprisingly, however, in pigeons, the only avian species studies thus far, electroretinograms (ERGs) evoked by presentation of alternating gratings to the

deprived and nondeprived eyes were not different in amplitude (Bagnoli et al., 1985).

The ERG is generated by sources other than retinal ganglion cells and probably reflects the activity of cells in the inner nuclear layer of the retina (Bagnoli et al., 1984). To see whether and to what extent retinal ganglion cells were affected by monocular deprivation, we deprived zebra finches from birth to 20, 40, or 100 days. We examined retinal ganglion cells in two locations: in the fovea, where they are known to be small and densely packed, and in the far periphery, where they are larger and loosely packed (figure 12.4). The results show that retinal ganglion cells in both the fovea and the periphery are only marginally affected by monocular deprivation. If changes were seen, they were in the direction opposite from what might be predicted, i.e., retinal ganglion cells were lightly larger in the *deprived* eye. While more detailed morphological data and physiological studies are needed, the evidence

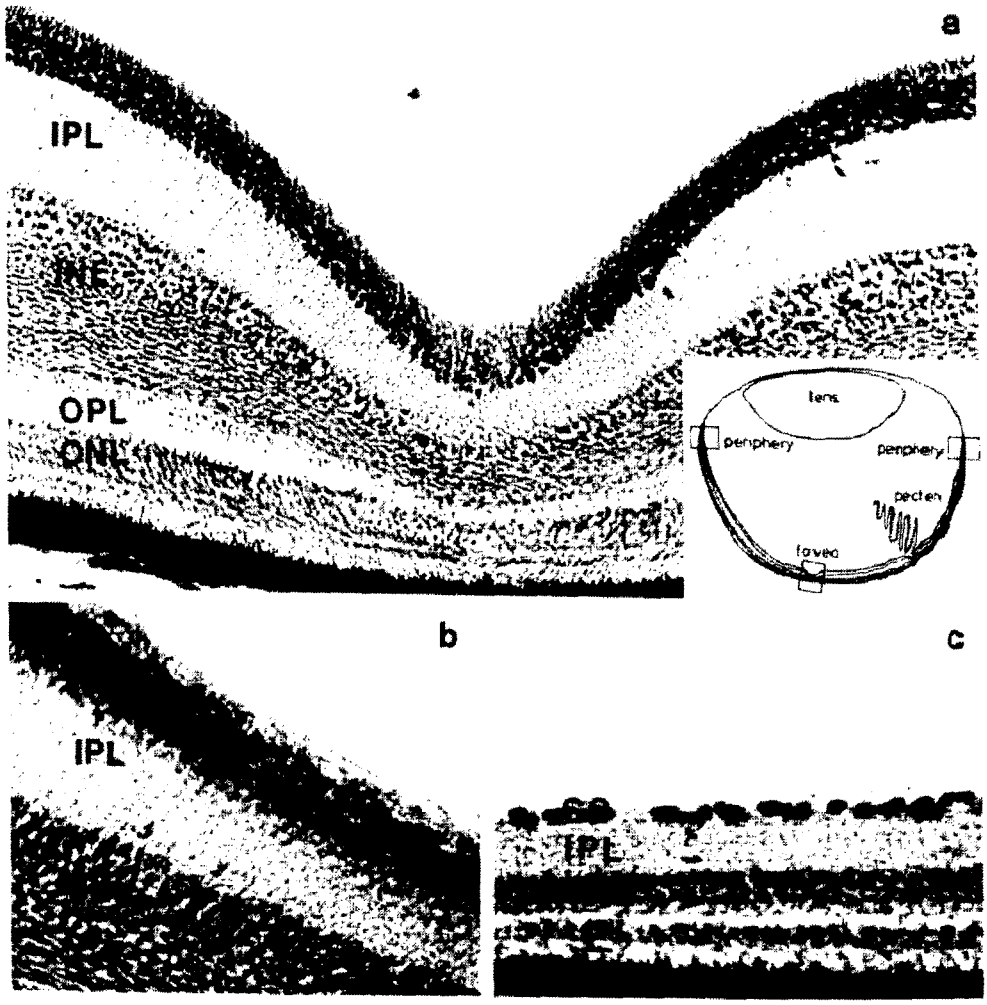


Figure 12.4 Nissl-stained cross section of the retina of a zebra finch. (a, b) Sections right through the fovea centralis; (c) a section through the periphery, as shown in the inset of (a). Note the difference in the size and density of retinal ganglion cells in (b, fovea) and (c, periphery).

to date suggests that retinal ganglion cells are more or less unaffected by deprivation. Thus the more central changes seen after monocular deprivation are not due to the effects of peripheral atrophy but reflect effects on central processes.

Thalamofugal System

While much known about the mechanisms mediating monocular deprivation effects on the mammalian geniculocortical pathway, surprisingly little is known about deprivation effects on the visual Wulst, the avian homologue of area 17. Pettigrew and Konishi (1976) were the first to demonstrate that neurons in the owl Wulst respond to monocular deprivation in a manner similar to that of neurons in mammalian visual cortex. Almost all neurons recorded from had lost their binocularity and could be driven only monocularly by the nondeprived eye. Correlated with this loss of binocularity was a morphological change. Whereas ocular dominance columns are not seen in normal owls, they seem to appear after monocular deprivation, forming stripes running orthogonal to the vertical meridian as in mammals (Pettigrew and Gynther, 1990).

Bagnoli and co-workers demonstrated that monocular visual deprivation produced changes in the neurotransmitter phenotypes of neurons in visual Wulst. These included a loss of GAD activity and an increase in ChAT activity in the dorsolateral Wulst contralateral to the deprived eye, as well as a decrease in the endogenous level of norepinephrine in the ipsilateral hemisphere (Bagnoli et al., 1982, 1983).

Nucleus Rotundus and Ectostriatum: Anatomical Effects

In these experiments deprivation began on the first or second day of life (when the eyes were still closed) by covering one eye (left or right) with an eye cap, just like for the measurements of retinal ganglion cells. The deprivation was maintained until the day of sacrifice on day 20, 40, or 100 (adulthood), so that the birds actually never saw with the occluded eye. Cell size and volume of the nucleus rotundus and ectostriatum were measured in Nissl-stained sections and compared to normally reared birds of the same age (Herrmann and Bischof 1986b,c).

Deprivation during the first 20 days of life did not result in morphological differences between the deprived and the nondeprived rotundus or ectostriatum. In contrast, following eye closure for a longer period (40 or >100 days), neurons in the deprived hemisphere were about 15% smaller than in the contralateral, nondeprived hemisphere. However, when the data of the deprived birds were compared to age-matched normally reared zebra finches, there were several unexpected findings.

As figure 12.5 shows, both rotundal and ectostriatal cell sizes of 20-day-old deprived birds are about 10% larger than those of normal birds.

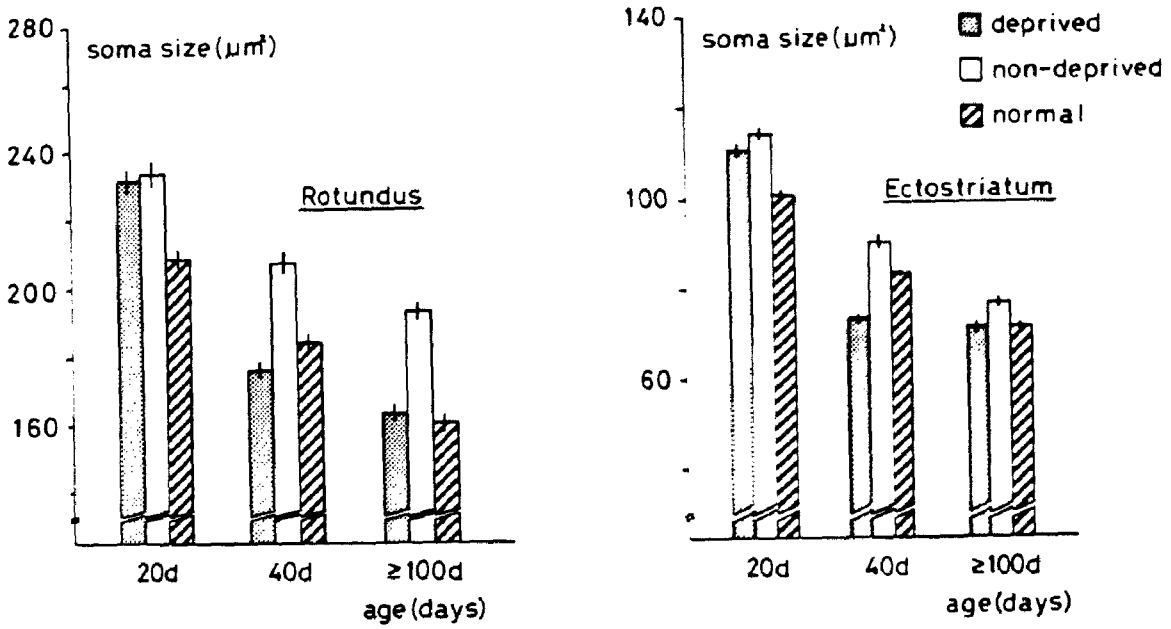


Figure 12.5 Comparison of the mean cross-sectional areas of neurons in the nucleus rotundus (a) and ectostriatum (b) of normal and monocularly deprived zebra finches of different ages. Gray bars, cell size in the deprived hemisphere (i.e., contralateral to the deprived eye); open bars, cell size in the nondeprived hemisphere (contralateral to the open eye); hatched bars, cell size of normal control birds (from Herrmann and Bischof, 1986b,c).

Also, the observed left-right asymmetry after 40 or 100 days of monocular deprivation must be attributed to a hypertrophy of neurons in the nondeprived rotundus, not to a shrinkage in the deprived hemisphere: neurons in n. rotundus and ectostriatum contralateral to the deprived eye are not statistically different from normal controls, while the neurons of n. rotundus and ectostriatum driven by the nondeprived eye are larger than those in controls. Thus, the effects of monocular deprivation are biphasic. Short-term deprivation leads to an "unselective" growth of neurons in both hemispheres. A longer deprivation period, however, causes the neurons driven by the deprived eye to shrink to a size that is also observed in normal animals. In contrast, the neurons driven by the nondeprived eye remain hypertrophied as they were with 20 days of deprivation. These effects of monocular deprivation can be shown not only for the neuron size within n. rotundus and ectostriatum, but also for the total volume of nucleus rotundus.

Our data indicate that there must be some crosstalk between the tectofugal system of both sides of the brain: the hemisphere driven by the nondeprived eye obviously also responds to deprivation and must therefore gain the information that the other hemisphere is deprived. Likely sources of this interhemispheric transfer are the tectotectal, the tectocontralateral rotundal, or the Wulst-tectum projections (see Engelage and Bischof, this volume). By these connections information from both eyes could meet in both sides of the brain.

Guillery and Stelzner (1970) and Guillery (1972) found in the cat that cell size of neurons in the monocular segment of the LGN is less affected by monocular deprivation compared to that of neurons of the binocular lamina. This can be interpreted as to show that cell size changes occur only if there is competitive interaction between the inputs from both eyes. Our results can be interpreted accordingly: Cell size changes due to monucular deprivation can be observed in areas where, by connections between the two brain sides, binocular interaction (and competition) is possible. The retina and the outer layers of the tectum, which do not have input from the nondeprived ipsilateral hemisphere, do not show effects.

Monocular deprivation also affects ultrastructural parameters in nucleus rotundus. The size of the presynaptic terminals is significantly reduced in both hemispheres after deprivation from birth to day 20 and stays low in the deprived hemisphere, if deprivation is maintained into adulthood. In addition, synapse density was much higher in the deprived hemisphere (Nixdorf and Bischof, 1987). These changes in ultrastructural parameters should be reflected in dendritic parameters like dendritic length or branching frequency. To our surprise, this was not the case for the ectostriatum. However, monocular deprivation did not have an effect on either dendritic length or branching frequency of the main ectostriatal neuron type. Both parameters paralleled the normal development, were at a maximum at 20 days, and declined until adulthood. We found, however, that monocular deprivation interfered with the development of dendritic spines. In zebra finches deprived for at least 40 days, neurons in the deprived hemisphere bear significantly fewer spines than those in the nondeprived hemisphere. This interhemispheric difference is mainly due to a lack of the normally occurring spine reduction in the nondeprived hemisphere, rather than to spine loss in the deprived hemisphere. We can conclude this because in comparison with normally reared birds, spine density is not lower in the deprived hemisphere, but rather is higher in the nondeprived brain side (figure 12.6). The excess of spines can be interpreted as a retention of a juvenile status, and might reflect a longer susceptibility to environmental changes. Another speculation might be that the amount of processed stimuli positively correlates with the number of spines. It could be argued that the nondeprived hemisphere has to compensate the lack of information in the hemisphere driven by the deprived eye and therefore retains more spines. These two hypotheses must not be mutually exclusive but rather the cause and consequence of the same process.

In any case, these results again point directly to interhemispheric interactions. As mentioned earlier, in contrast to the thalamofugal system, neither binocular neurons nor competitive interactions have been described before. Tectotectal as well as tectorotundal interhemispheric projections, however, indicate that such interactions between the two brain sides are possible. It can also not be excluded that then normally

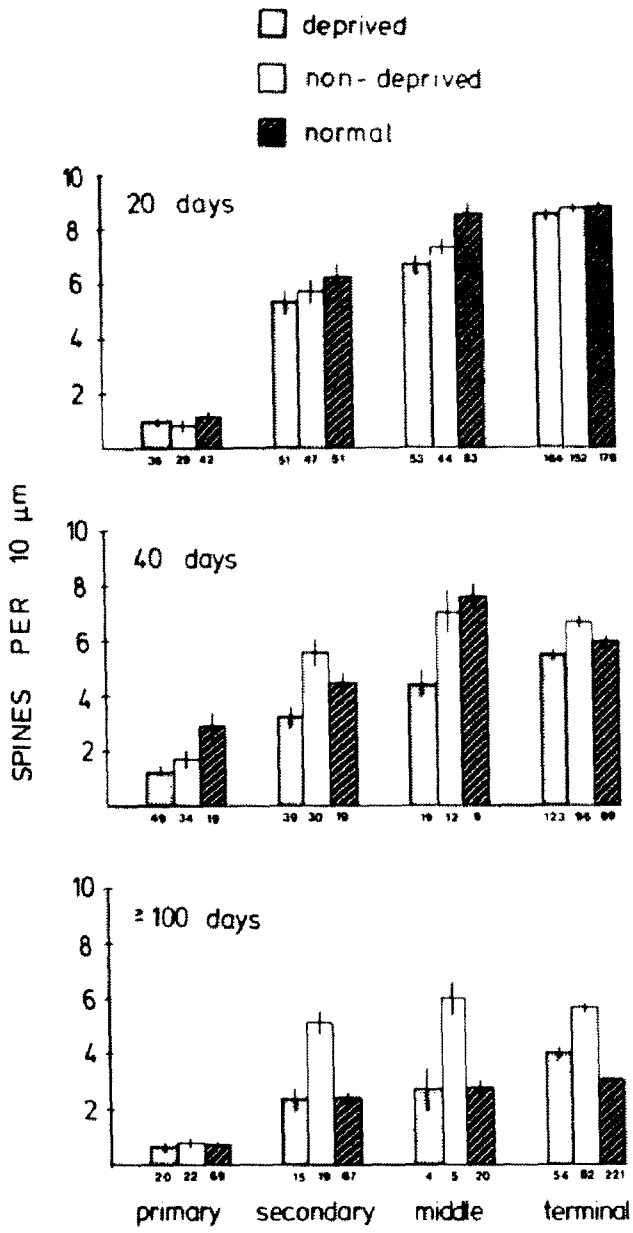


Figure 12.6 Spine density of neurons in the ectostriatum of zebra finches deprived from birth to day 20 (upper), 40 (middle), and >100 days of age (bottom). Bars represent median values + SEM for various dendritic segments (primary, secondary, middle, terminal). Gray bars, spine density in the deprived hemisphere (i.e., contralateral to the deprived eye); open bars, spine density in the nondeprived hemisphere (contralateral to the open eye); hatched bars, spine density of normally reared birds. Differences in spine density between deprived and nondeprived hemisphere occur only after 40 days of deprivation and manifest themselves basically in an excess of spines in the nondeprived hemisphere. Spine density in the deprived hemisphere appears to be normal (see data in bottom row; from Herrmann and Bischof, 1988b).

transient ipsilateral retinotectal projection might be maintained as a result of monocular deprivation, as it was demonstrated in enucleation studies in chickens (O'Leary et al., 1983). However, our preliminary findings that cell size is not altered by monocular deprivation in the outer tectal layers indicate that this is not a likely explanation.

Nucleus Rotundus and Ectostriatum: Functional Effects

To determine whether the deprivation-induced morphological changes discussed above might have functional correlates, we used the 2-deoxyglucose (2DG) method. This technique identifies differential activity in various brain areas by measuring the accumulation of a radioactively tagged glucose marker as an index of the energy utilization, and hence the functional involvement, of these areas.

For this study, zebra finches were monocularly deprived from birth to adulthood. The day before the 2DG experiment the eyecaps were taken off, and the birds were allowed to see with both eyes while being exposed to the 2DG. The results were not surprising: despite the fact that the birds were now seeing binocularly, both the nucleus rotundus and the ectostriatum showed a tremendous asymmetry in the optical density: in the hemisphere contralateral to the deprived eye, the glucose consumption and therefore the activity was drastically lower than in the nondeprived hemisphere (Herrmann and Bischof, 1986b, figure 12.7). Nonvisual areas like the telencephalic auditory field were symmetrically labeled. It is likely that the asymmetry in the nucleus rotundus and the ectostriatum is due to a decrease of activity in the deprived

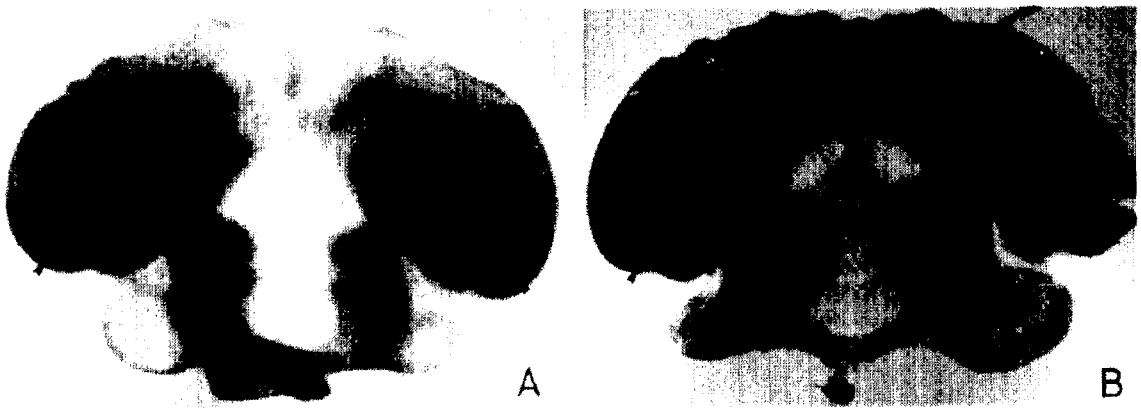


Figure 12.7 Computer-generated densitometric plot of 2-deoxyglucose autoradiography. (A) Cross section of the brain of a zebra finch that was monocularly deprived from birth to day 100, and stimulated binocularly during the 2DG experiment. Nucleus rotundus (arrow) and ectostriatum (arrowhead) are asymmetrically labeled. The deprived (right) hemisphere shows weaker labeling in both areas. In contrast, (B) shows a cross section of the brain of a bird that was deprived for the same length of time but as adult. 2DG labeling of both nucleus rotundus and ectostriatum is symmetrical in both hemispheres, indicating that monocular deprivation in adult birds does not lead to metabolic changes in these tectofugal areas.

hemisphere, because we know from experiments with intact birds that nucleus rotundus and ectostriatum are usually heavily labeled and stand out in 2DG experiments. This was not he case in the deprived hemisphere of our experimental animals.

In contrast to these results, however, Burkhalter et al., (1982) reported no asymmetries in 2DG activity in the tectofugal pathway of pigeons after monocular deprivation. While there are a number of methodological differences between the studies that might explain the discrepancy, it may also reflect species differences between pigeons and zebra finches. Güntürkün and Böhringer (1987) found that the tectum opticum of normally reared pigeons shows hemispheric differences (see also chapter 13), a phenomenon that is absent in zebra finches (Herrmann, unpublished observations). This asymmetry in normal pigeons might of course interfere with structural changes arising after monocular deprivation. At present we do not know whether the pigeons studied by Burkhalter et al., (1982) were consistently deprived on either the left or the right eye, but it is possible that the morphological asymmetry in pigeons might explain the absence of effects in the 2DG study of these animals.

THE SENSITIVE PERIOD FOR THE DEPRIVATION EFFECTS

In recent years a large body of literature has accumulated showing that the nervous system is susceptible to manipulations only during a brief time early in an animal's life, the so-called sensitive or critical period (Blakemore and VanSluyters, 1974; Hubel and Wiesel, 1970; Knudsen and Knudsen, 1986; Olsen and Freeman, 1980). We therefore wanted to establish whether such a sensitive period also existed for the effects of monocular deprivation in zebra finches. Using the 2DG method we deprived adult zebra finches for the same length of time as the neonates in the experiment mentioned above (for 100 days) and found that this late deprivation did not result in any asymmetric labeling of nucleus rotundus and ectostriatum as seen in developing animals (Herrmann and Bischof, 1986b, figure 7b). This suggests that during a critical period of development, birds were susceptible to deprivation manipulations to which adults, with fully differentiated nervous systems, are immune.

To explore the time course of this sensitive period for the effects of monocular deprivation, we measured cell size and volume changes in zebra finches subjected to a period of 40 days of unilateral eye closure (the time, when all deprivation effects seem to be stable) starting at ages spaced regularly throughout the first 70 days of life, i.e., we deprived birds from days 1 to 40, 10 to 50, etc. until days 70 to 110.

The result of this study (Herrmann and Bischof, 1988a) shows that monocular deprivation markedly affects cell size of nucleus rotundus and ectostriatum if the treatment starts at 1 or 10 days post hatching. The differences between deprived and nondeprived hemisphere de-

creased with increasing visual experience prior to deprivation. Surprisingly to us, however, was that deprivation onset at day 40 caused again as severe effects as early monocular closure. Deprivation starting at day 50 or later, on the other hand, did no longer lead to abnormalities.

The measurements of the rotundus volume parallel the cell size results, with the exception that the second increase in sensitivity occurred at day 50 instead of day 40 (figure 12.8). Taken together, these results indicate that the sensitive period for the effects of monocular deprivation may be double-peaked: the sensitivity to external stimuli declines from hatch until day 30, but has another peak at 40–50 days of life. Therefore we can conclude that a period of normal vision prior to deprivation reduces the sensitivity to anatomical and functional changes caused by deprivation. It is worth noticing that the first peak of sensitivity occurs at a time early in ontogeny, when both ectostriatum and nucleus rotundus are not fully developed: between hatching and day

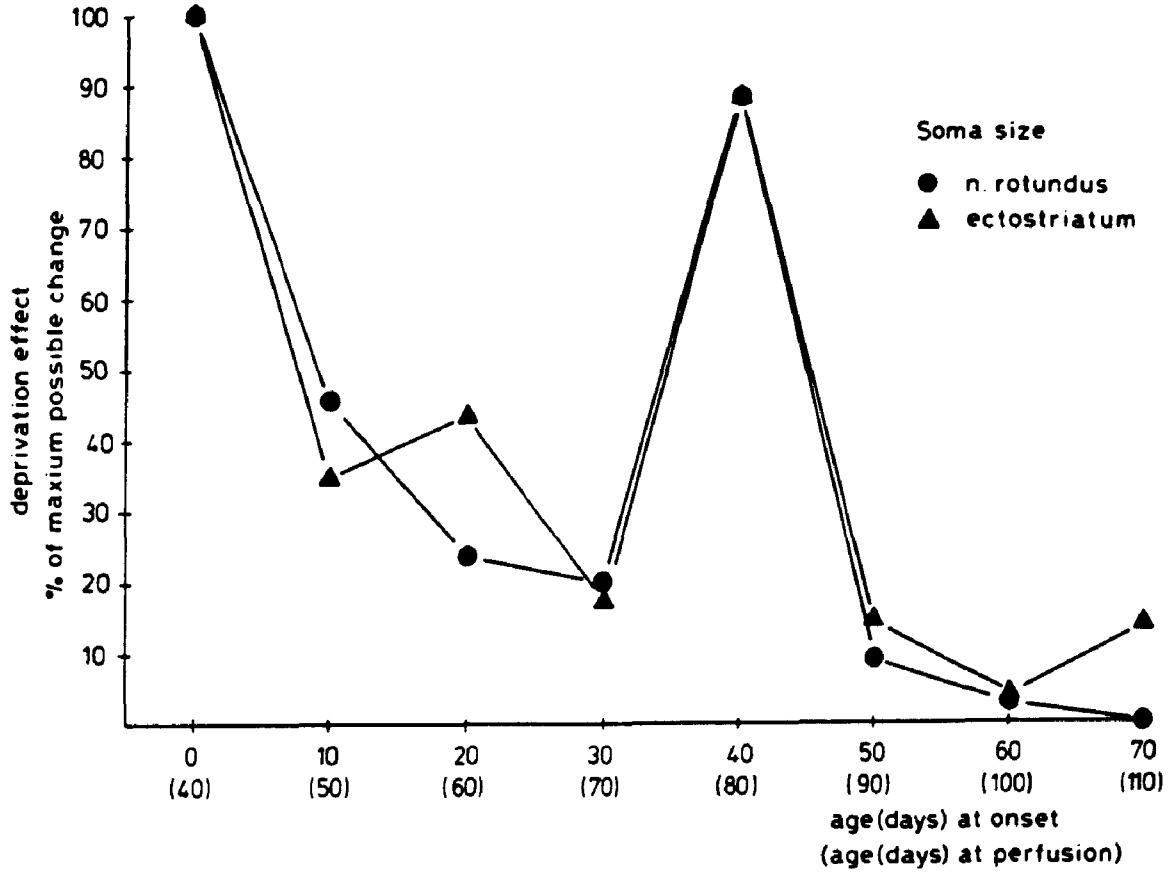


Figure 12.8 The time course of the sensitive period for the effects of monocular deprivation as derived from cell size measurements of rotundal and ectostriatal neurons. Birds were monocularly deprived for a constant period of 40 days starting at days 0, 10, 20, 30, 40, 50, 60, and 70, and sacrificed immediately after the deprivation period. The absolute difference between the soma size in the deprived and nondeprived hemisphere following neonatal eye closure for 40 days was set 100% (maximal possible change). The left–right differences in cell size following later deprivation onset were calculated with respect to this value.

20 neurons grow, dendritic arbors increase in length and complexity, dendritic spines are being added, and synaptic contacts are still weak and probably modifiable. The peak of sensitivity seems to occur *before* the transient overshoot in all these parameters can be observed, and it would be interesting to find out whether the presence of redundant and superfluous neuronal elements allows plastic changes to occur.

At present we have no good explanation for the second rise in susceptibility at day 40, a time when the anatomical development of both nucleus rotundus and ectostriatum was thought to be completed. A study by Engelage and Bischof (1990) clearly demonstrated, however, that the physiological response pattern of neurons in the ectostriatum is not adultlike by day 30. Probably the second rise in susceptibility may be explained by late occurring projections, which weaken the previously established connections by competitive interactions. Interestingly, such a second rise in sensitivity after the end of the originally presumed sensitive period was also demonstrated in the LGN of primates (Headon et al., 1985). These authors, however, also did not provide an explanation. Thus further experiments will be needed to clarify the nature of this second peak.

BINOCULAR DEPRIVATION

Most effects of monocular deprivation in the tectofugal system arise in the hemisphere driven by the nondeprived eye, and these effects manifest themselves in a hypertrophy of cell size and volume, and an increased spine density. How would these parameters be affected if we deprived both eyes of vision? There are two arguments to suppose that binocular deprivation would not have any effect at all: first, if deprivation effects are due to an imbalance of the inputs from the two eyes to the competitive sites, one should not see an effect because both eyes are reduced in activity. Second, if the absence of effects in the deprived hemisphere, as demonstrated above, could be interpreted as to show that patterned visual input is not necessary for the establishment of these anatomical features, one could predict that binocular deprivation should have no effect at all. To test this hypothesis, we deprived zebra finches binocularly from birth until day 20 or 40, the age when deprivation effects were stable in monocularly deprived birds. As a first attempt, we measured cell size and volume of the nucleus rotundus and ectostriatum and compared those to normally reared birds of the same age.

As we predicted, the data of this study clearly demonstrated that binocular deprivation does not arrest neuron growth in the nucleus rotundus and the ectostriatum of zebra finches (figure 12.9). These results suggest that at least the anatomical development of tectofugal system can proceed in the total absence of patterned vision, although the possible physiological changes induced by binocular deprivation

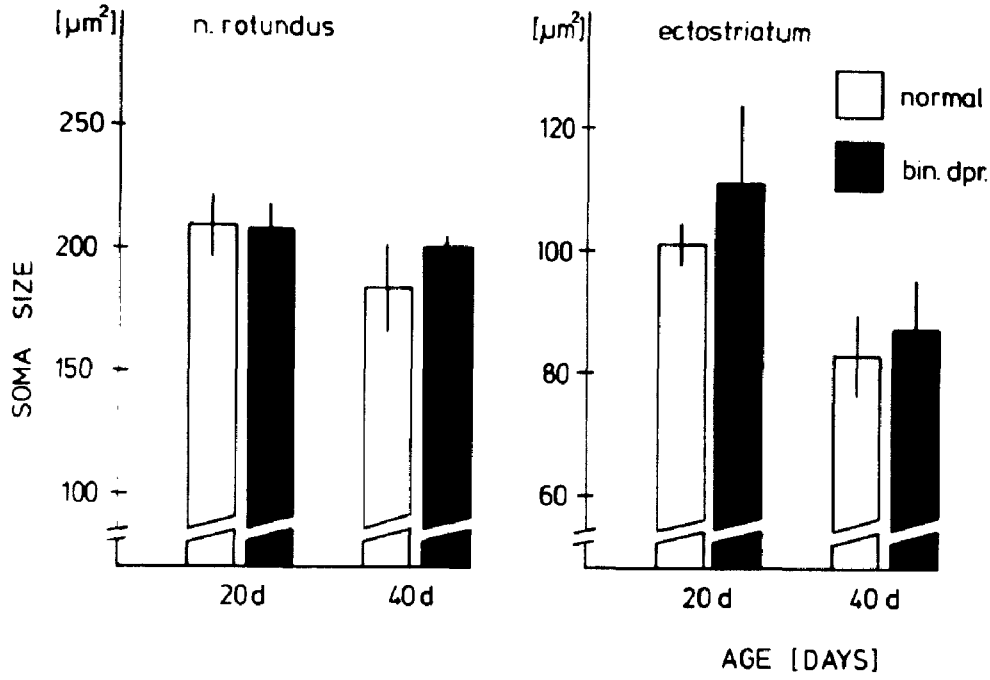


Figure 12.9 Soma size (+ SE) in the nucleus rotundus (left) and ectostriatum (right) of zebra finches that were binocularly deprived from birth until day 20 or 40, compared to age-matched normally reared control birds. Binocular deprivation does not lead to significant changes in neuron size of either area.

remain to be determined. It is also possible that in birds the exposure to light in ovo is sufficient to induce the normal course of development. A study in dark-reared primates has shown that even short periods of light are sufficient to trigger the normal cell differentiation process (Chow, 1955). The importance of light in ovo was established by Rogers and co-workers and will be reviewed in another chapter of this volume.

CONCLUSIONS

In this chapter we provided extensive evidence that both the tectofugal and the thalamofugal system of birds are susceptible to visual deprivation. We have shown that monocular deprivation affects cell size, volume, and spine density in the nucleus rotundus and the ectostriatum, and these effects are mainly due to a hypertrophy (or failure to decline) of these neuronal elements on the nondeprived hemisphere, whereas the hemisphere driven by the deprived eye seems more or less unaffected. We are far away from understanding the underlying cellular mechanisms that lead to these changes. We believe, however, that the morphological and physiological abnormalities in the tectofugal pathway, traditionally thought to be strictly monocular, have to be interpreted as a result of an imbalance of binocular interactions, and we speculate on the anatomical substrate for these interhemispheric interactions.

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