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Afferent Influences on Brainstem Auditory Nuclei of the Chicken: N. Laminaris Dendritic Length Following Monaural Conductive Hearing Loss

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ABSTRACT

The influence of monaural acoustic deprivation on the size of dendrites in n. laminaris in the chick was examined. Chicks were raised in a controlled acoustic environment with one ear occluded from 2 days prior to hatching until 25 days after hatching by an earplug which provided a conductive hearing loss of approximately 40 dB across the audible frequency range. Each n. laminaris cell receives spatially segregated binaural excitatory innervation; one dendritic field received input from the plugged ear while the other received input from the normal ear. This arrangement allowed comparison of the size (length) of the "deprived" dendrites and the "nondeprived" dendrites for each cell. The tonotopic organization of n. laminaris allowed these comparisons to be made as a function of the frequency organization of the nucleus.

We observed systematic changes in dendritic size which differed as a function of the tonotopic position of the neurons. In high-frequency regions the dendrites receiving information from the deprived ear were shorter than those receiving input from the normal ear. Unexpectedly, cells responsive to low frequencies showed the opposite result; the dendrites innervated from the deprived ear were longer than those responsive to the nondeprived ear. These results suggests that a relatively flat conductive hearing loss may cause nonuniform changes in activity impinging on high- and low-frequency areas of the auditory system.

Key words: dendrites, plasticity, development, deprivation, sensory systems

One extrinsic factor regulating neuronal ontogeny is the synaptic activity impinging on the cell surface. Following receptor development the organism's environment may influence this parameter. There have now been many demonstrations that changes in the environment can alter the ontogeny of neurons, particularly in the visual system (e.g., Chow et al., '57; Wiesel and Hubel, '63; Guillery, '72; Hickey, '80; Casagrande and Joseph, '80). Similar findings emerge from study of the ontogeny of neurons in the vertebrate auditory system (Webster and Webster, '77, '79; Coleman and O'Connor, '79; Feng and Rogowski, '80; Conlee and Parks, '81). In addition to the obvious importance of evaluating the generality of such phenomena across species, age, sensory systems, and neuronal cell types, important issues remain to be resolved. Two such issues are (1) the precise relation of postsynaptic cellular change to the alteration in presynaptic activity and (2) the regional specificity of the changes within the postsynaptic neurons.

The brainstem auditory system of the chick is a useful preparation for approaching the above issues. The eighth nerve projection onto n. magnocellularis (NM) is the major input to these cells and provides a powerful excitatory influence (Ramón y Cajal, '08; Boord and Rasmussen, '63; Parks and Rubel, '78; Parks, '81a; Whitehead and Morest,

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'81; Hackett et al., '82). NM cells, like the eighth nerve of both birds and mammals, display monotonically increasing activity as the intensity of an appropriate acoustic stimulus is increased (Kiang et al., '65; Sachs et al., '74, '80; Rubel and Parks, '75). N. magnocellularis neurons, in turn, project bilaterally and topographically onto nucleus laminaris (NL), to the dorsal dendrites of the ipsilateral NL, and the ventral dendrites of the contralateral NL. This projection is also excitatory and tonotopically organized (Parks and Rubel, '78; Hackett et al., '82). Thus, any monaural manipulation affects the activity impinging on the dorsal dendrites of the ipsilateral NL cells and the ventral dendrites of the contralateral NL cells. The dorsal and ventral dendrites of NL neurons are normally symmetrical and vary systematically as a function of the tonotopic organization (Smith and Rubel, '79; Smith, '81; Gray et al., '82). Therefore, following any monaural manipulation the most appropriate comparisons are between the opposite dendrites of the same cells. In previous studies we have shown that manipulations which result in direct and transneuronal deafferentation of one set of dendrites cause marked degeneration of the deafferented dendrite, but do not affect the size of the opposite dendrites of the NL cells (Benes et al., '77; Rubel and Smith, '81; Rubel et al., '81).

This report is part of a series in which we have attempted to vary the afferent input to these nuclei and examine postsynaptic alterations in the development or maintenance of NM and NL neurons (Benes et al., '77; Jackson and Rubel, '76; Parks, '79, '81b; Rubel and Smith, '81; Lippe et al., '80; Rubel et al., '81). The eventual goal is to specify the function relating presynaptic activity to postsynaptic structural ontogeny. In this study we have used a method of monaural deprivation (silicone ear plugs) which provides a reversible conductive hearing loss of 40 decibels (dB) that is approximately flat across the auditory frequency range of chickens. This method has previously been shown to retard perceptual development (Kerr et al., '79) and the development of symmetry of the dorsal and ventral dendrites in n. laminaris (Gray et al., '82) and causes hypotrophy of NM cells on the deprived side of the brain (Conlee and Parks, '81).

In the present study chicks have been reared in controlled acoustic environments. Monaural ear plugs were in place from prior to hatching until 25 days posthatch. The lengths of NL dendrites receiving input from the deprived (ear plugged) and the nondeprived ear were then measured throughout the nucleus. The results indicate that a conductive hearing loss that is of similar magnitude across the frequency domain produces opposite effects on the sizes of dendrites in the high- and low-frequency regions of n. laminaris.

MATERIALS AND METHODS

Four chickens (Hubbard × Hubbard) were used in this study. Eggs were obtained from a local supplier and incubated under standard conditions until embryonic day 19. At embryonic day 19, prior to the emergence of the chick into the egg's air space (tenting stage), each egg was opened and the chick's head exposed. The external auditory meatus on one side was gently aspirated and then filled with a silicone plastic following previously published procedures (Kerr et al., '79; Gray et al., '82). One-half of the animals had the ear plug in their right ear while the others had their left ear plugged. The ear plug produces a conductive hearing loss of about 40 dB across the frequency range of the chicken's hearing and is fully reversible when the plug is removed, at least up to 5 days later. The ear plugs were checked daily and replaced when the growth of the ear canal had made the outer end of the ear plug loose, or after every 5 days. The completeness of each ear plug was assessed upon removal by inspection of the impression of the tympanic membrane; most were 100% complete, though occasionally an air bubble was found at the distal portion of the tympanum. All plugs were at least 85% complete.

After the initial ear plugging, the chicks were kept in a quiet incubator until hatching. After hatching, the chicks were transferred to double-walled sound-attenuated chambers, where they were reared until posthatch day 25. The sound-attenuated chambers had an inner dimension of 30.5 × 33.6 × 25.4 cm, and attenuated outside sounds by 45 dB at 100 Hz, 65 dB at 300 Hz, and over 75 dB at higher frequencies. The temperature in each chamber was kept at 34°C for the first 5 days and then gradually reduced to room temperature by 10 days after hatching. Lighting was on a 12-hour–12-hour on-off cycle. The boxes were opened once each day for 3–5 minutes in order to provide fresh food and water, and to check the integrity of the ear plugs. Centrally placed in the ceiling of each chamber was a speaker, which broadcast an ascending series of pulsing pure tones from 200 Hz to 4,600 Hz in 50-Hz intervals. These sounds were corrected for the acoustic properties of the chambers and presented at 65 dB (SPL) during the daylight portion of the light cycle for 12 minutes of each 20 minutes.

At posthatch day 25, the chickens were removed from the chambers, deeply anesthetized with an overdose of sodium pentobarbital (Nembutal), and perfused with Golgi-Kopsch fixative (Stensaas, '67). Brainstems were embedded in nitrocellulose and serially sectioned at 120 μm in the coronal plane. Under high magnification, camera lucida tracings of the dendrites of the impregnated neurons were made by drawing lines down the middle of each dendritic process. A double-blind procedure of tracing and measuring dendrites was used until the final computer analysis. Cells with any dendrites which were not unambiguously directed dorsad or ventrad were eliminated from the quantitative analyses.

A total of 241 cells were sampled from throughout NL, about half from each side of the brains. The total length of each dendritic tree (primary dendrites plus all associated branches) on both the dorsal and the ventral side of every impregnated neuron which was completely included in a section was drawn and then measured using a digital planimeter. From serial reconstructions of the Golgi-stained brains, the cells' positions in NL were determined and assigned to the posterior-anterior and medial-lateral axes as normalized cartesian coordinates (%P-A, %L-M) (cf. Smith and Rubel, '79; or Smith, '81, for a full discussion of the quantification and analyses of NL dendrites). Statistical analyses were performed on a CDC Cyber 172 computer using SPSS programs.

RESULTS

A unilateral ear plug is expected to affect afferent activity to the dorsal dendrites of NL ipsilateral to the plug and to the ventral dendrites of NL on the contralateral side. The afferent input to the ipsilateral ventral dendrites and contralateral dorsal dendrites should be unaffected.

The qualitative appearance of the nucleus was little changed as a result of the deprivation. Closer examination, however, revealed an increased number of neurons which

lacked the typical symmetrical bipolar arrangement of dendrites, and of neurons which had disoriented dendrites. Although the latter neurons were not common, they were much easier to find in the deprived animals than in normal brains (Smith and Rubel, '79; Smith, '81). There was no obvious consistency in the locations of these cells. Figure 1 shows representative line drawings of some abnormal cells (A-C) which are characteristic of what we observed in the deprived animals, and of a normal-appearing neuron (D) also from one of the deprived animals.

To quantitatively analyze changes in dendritic length, each neuron (n = 241) was considered to have a deprived group of dendrites (either dorsal or ventral, depending on the side of the ear plug); and on the opposite side of the cell the dendrites were denoted "nondeprived." A measure of the effect of auditory deprivation can thus be taken as the difference between the deprived dendritic length and the nondeprived dendritic length of each cell (Fig. 2). This measure (deprived length minus nondeprived length) is negative where the deprived dendrites are shorter than the nondeprived, and is positive when the deprived dendrites are longer. Since the lengths of dorsal and ventral dendrites of a cell are normally highly correlated and symmetrical throughout the nucleus, the expected value of this measure in normal animals is zero and is unaffected by the gradient of dendritic length across NL (Smith and Rubel, '79).

The changes in dendritic length could best be analyzed as a function of the frequency organization of the nucleus.[1] Accordingly, the nucleus was divided into four equal tonotopic sectors using the equations from the tonotopic maps of Rubel and Parks ('75). The cells were then assigned to the appropriate frequency sector based on their position in NL (Smith, '81). The mean lengths of the deprived and nondeprived dendrites for each of the sectors, along with the mean difference, the percent difference, and the range of percent differences across the four subjects are shown in Table 1. The expected value of the difference in length between deprived and nondeprived NL dendrites is zero and in normal animals means of dorsal and ventral dendrites with each sector are within 2% (Smith and Rubel, '79; Smith, '81). Additionally, since about half of the dendrites in each sector were from the side of the brain ipsilateral to the earplug and half were from the other side of the brain a dorsal-ventral size bias could not account for the results shown in Table 1. Inspection of the first two columns reveals that on both the deprived side of the cells and the nondeprived side there is a clear gradient of dendrite size as a function of frequency sector. Similar results from normal animals have been reported previously (Smith and Rubel, '79; Smith, '81) as well as from animals which underwent embryonic otocyst removal (Parks, '81b). In all cases,

[1]Regression analyses on the 241 cells from the chickens raised in the sound-attenuated chambers indicated that the effects of deprivation on the dendritic length of NL cells varied systematically as a function of the position of the cell in the nucleus. This variation was from rostromedial to caudolateral, closely paralleling the tonotopic organization of NL described by Rubel and Parks ('75). The correlation between the *change* in dendritic length and the position of a cell in NL was highly significant, though weak ($r = -.28$, $P < .001$), and each animal showed this effect.

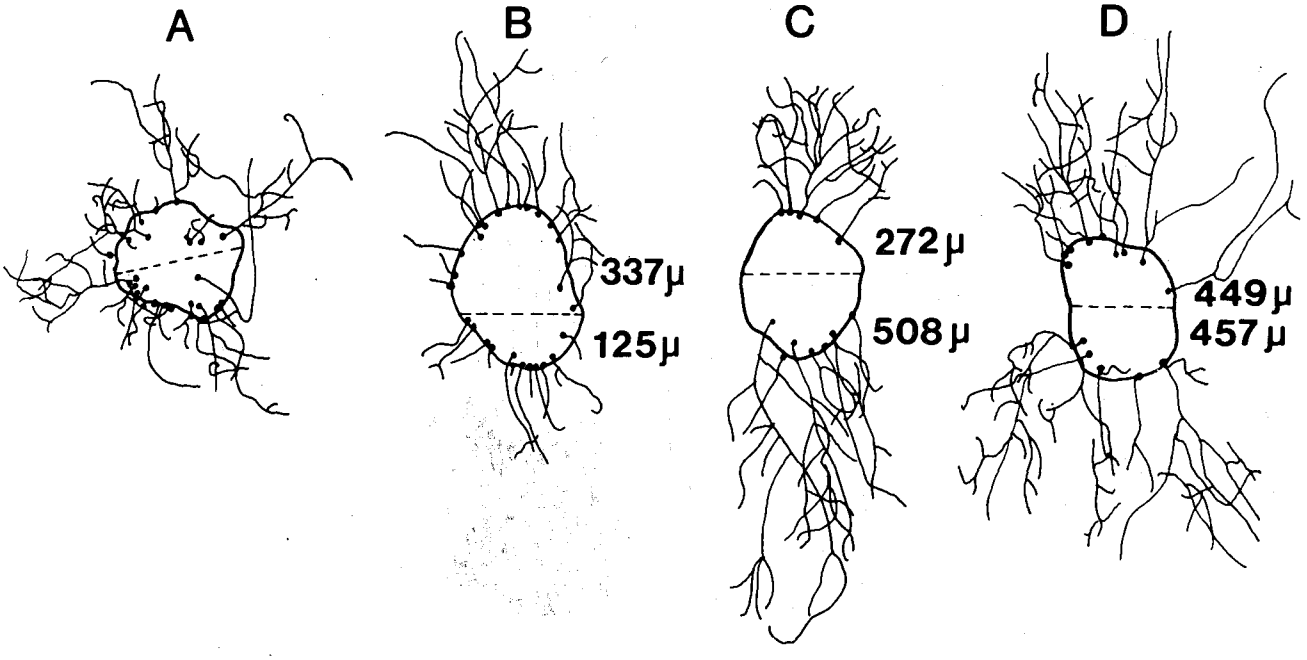

Fig. 1. Camera lucida tracings of characteristic cells from monaurally deprived chickens. All cells are shown as if on the contralateral side of the brain from the deprived ear; therefore the "deprived" dendrites are the ventral dendrites. Numbers next to the cell indicate the total length of the dorsal dendrites and the total length of the ventral dendrites. A. Cell with disoriented dendrites. Since some dendrites are both dorsal and ventral the separate lengths could not be objectively determined. Such cells were not considered in the quantitative analysis. B. Cell from anterior part of NL; deprived (ventral) dendrites are shorter than the nondeprived dendrites. C. Cell from the posterolateral third of NL showing the opposite effect as that shown by cell B; the deprived (ventral) dendrites are longer than the nondeprived (dorsal) dendrites. D. Normal-appearing NL cell from the middle region of the nucleus; note that the lengths of the dorsal and the ventral dendrites are similar.

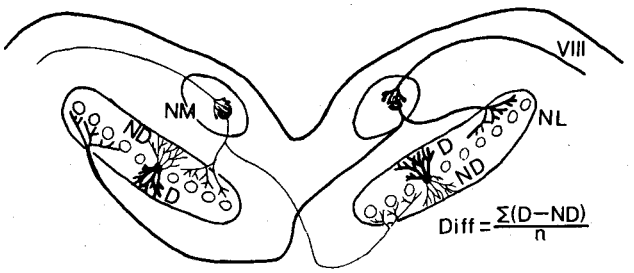

Fig. 2. Schematic drawing of the brainstem of the chick showing an eighth nerve axon (VIII), n. magnocellularis (NM), and n. laminaris (NL). Redrawn from Rubel et al. ('81) to show that each NL cell had dendrites which received input from the deprived ear (D) while the other dendrites received input from the nondeprived ear (ND). The "deprived" side is represented by the heavier processes in the nerve (VIII), NM axon collaterals, and NL dendrites. The principle measure used in this study is indicated: the length of the deprived dendrites minus the length of the nondeprived dendrites averaged across all the cells measured in each brain.

TABLE 1. Mean Dendritic Lengths and Changes Following Ear Plugging as a Function of the Frequency Region of n. laminaris

Frequency Sector	Non-deprived length (μm)	Deprived length (μm)	Mean difference	Mean % difference	Range % difference
1 (low)	404	540	+136	+33.6	+6 to +43
2	354	386	+32	+9.0	−12 to +20
3	240	222	−18	−7.5	−34 to +11
4 (high)	179	167	−12	−6.7	−17 to +0.2

dendritic size is smallest in the rostromedial region (sector 4—high frequencies) and largest in the caudolateral area (sector 1—low frequencies). In columns 4–6, the dendritic lengths on the deprived and nondeprived sides of the NL cells are compared. To derive the values reported in columns 4 and 5 the difference in dendritic length (deprived side—nondeprived side) was determined for each cell, the mean difference was calculated for each sector of the animal, and then the grand mean was determined for each sector (column 4). This value was then converted to a percent change (column 5) by comparison with the mean nondeprived length (column 1). In the final column the range of percent differences across the four animals is shown for each sector. While there was considerable variability, a consistent trend emerged. In the high-frequency sectors (3 and 4) the deprived dendrites tended to be smaller than the nondeprived dendrites while in the low-frequency region of NL (sectors 1 and 2) the opposite relationship was found; surprisingly, the dendritic tree receiving input from the deprived ear tended to be longer than the opposite dendrites which received innervation from the nondeprived ear.

In order to insure that the differences observed in Table 1 were not dominated by a single animal, and to adjust for absolute dendritic size differences between animals, an additional analysis was performed. Differences scores for each neuron (deprived dendrites minus nondeprived dendrites) were converted to Z-scores using the mean difference and standard deviation of differences for that animal. The mean normalized difference scores were then determined for each frequency sector in each animal. These values were then averaged for the four subjects yielding a grand mean difference score (and standard deviation) for each of the four frequency regions of the nucleus. These results are plotted in Figure 3A. The change in dendritic length due to ear plugging is shown in relation to the characteristic frequency of the NL cells. This analysis yielded the same conclusion as derived from Table 1; at the end of the nucleus responding to low frequencies the deprived dendrites were longer than the nondeprived dendrites, while in the half of NL responding to high frequencies the deprived dendrites were shorter. A cross-classified analysis of variance (ANOVA) applied to these date strongly supported this conclusion. There was a highly significant main effect of frequency sector ($F_{3,233} = 4.47$; $P < .004$) which was not dependent on the side of the brain (ipsi- or contralateral to the ear plug).

Changes in total dendritic length may be viewed as resulting from a change in the number of dendritic trees (the primary dendrite plus its connected branches) or from a change in the length of the individual dendritic trees (Smith, '81). Accordingly, the change in the average lengths of the individual dendritic trees resulting from ear plugging was analyzed across the frequency organization of NL. The average dendritic tree length for the dorsal and ventral side of each cell was computed by dividing the total dendritic length by the number of primary dendrites emanating from that side of the cell. Differences in dendritic tree size were then analyzed in the same manner as were the changes in dendrite length. Figure 3B shows the result of this analysis; it nearly replicates the changes in dendritic length seen in Figure 3A. Thus, the change in dendritic length across NL resulted from differences in the size of the individual dendritic trees, not from changes in the number of primary dendrites. This conclusion is also supported by direct examination of the effect on number of primary dendrites, where no consistent change was seen. Again, the dendritic tree length data were statistically analyzed by a cross-classified ANOVA. There was a highly significant main effect of frequency sector ($F_{3,233} = 4.5$, $P < .004$), no main effect of side of the brain relative to the ear plug, and no significant interactions between these variables.

Unilateral auditory deprivation was also found to have an effect on the gradient of dendritic size seen across NL. As described in Smith and Rubel ('79) and Smith ('81), normally there is a marked change in dendritic length along the tonotopic gradient of NL such that at the high-frequency pole the dendrites are much smaller than at the low-frequency pole. Regression analyses of deprived and nondeprived dendritic length vs. position in NL showed that the gradient of dendritic length is steeper on the deprived sides of NL cells (dorsal ipsilateral and ventral contralateral to the ear plug) than on the nondeprived sides of the cells. As suggested by Figure 3, this was chiefly due to longer deprived dendrites at the low-frequency end of NL.

DISCUSSION

Unilateral ear plugging for 4 weeks after entry into the air-mediated sound field differentially affected the length of n. laminaris cell dendrites as a function of the sound frequency to which these neurons normally respond. The deprived dendrites were *longer* than the nondeprived dendrites at the low-frequency end of the nucleus, and the deprived dendrites were *shorter* than the nondeprived den-

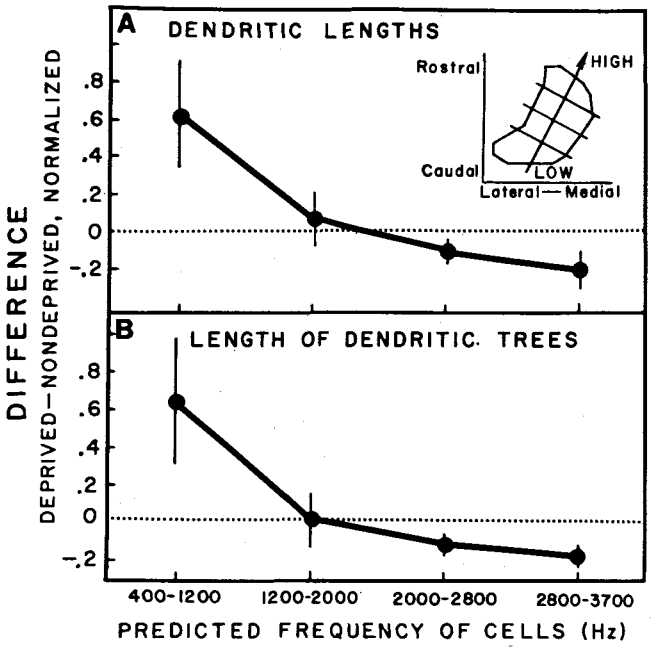

Fig. 3. Mean difference (±1 SE) between deprived and nondeprived dendrites for the four monaurally deprived animals is shown as a function of the frequency region of the nucleus. The length differences for all the cells drawn for each animal were normalized (Z-scores). Averages for each frequency region were then computed and then the mean (and SE) across the four subjects was determined (n = 4 for each frequency region). In both graphs a positive number indicates that the length of the deprived dendrites was greater than the nondeprived dendrites. A negative number indicates the length of the deprived dendrites was *less* than the nondeprived dendrites. A. Mean differences in total dendritic lengths between the deprived and the nondeprived dendrites are indicated. B. Mean differences in the length of the "dendritic trees" (total length/number of primary dendrites) between the deprived and nondeprived sides of the cells are shown.

drites of cells responsive to high frequencies. These changes in dendritic length resulted from changes in the lengths of the individual dendritic trees, and not from the differential loss of primary dendrites. This means of regulating the amount of dendritic surface contrasts sharply with normal development of NL, which is characterized by a marked loss of primary dendrites over the ages studied (Smith, '81). The changes in length were unaffected by whether the nuclei ipsilateral or contralateral to the ear plug were analyzed.

In general previous morphological investigations concerned with auditory deprivation have not subclassified cells or regions on the basis of characteristic frequency, which may be responsible for some of the variability in results (cf. Webster and Webster, '79; Coleman and O'Connor, '79; Feng and Rogowski, '80). In the one previous study of cell body size that did examine regional variations following monaural deprivation (Conlee and Parks, '81) the results are equivocal and the entire nucleus was not sampled (Conlee and Parks, '81; Table 1). Further analysis of cellular changes in NM which directly relate cell size changes to the frequency axis of the nucleus would be of great interest.

Physiological investigations following deprivation have not explicitly explored differential effects on high- and low-frequency areas within the auditory system. However, it may be notable that Silverman and Clopton ('77) and Clopton ('80) report changes predominantly in units with high CFs and little change in the low CF neurons following ear plugging.

Possible mechanisms of differential effect of ear plugs

The underlying hypothesis of this study was that a uniform reduction in the amount of activity impinging on NL dendrites would lead to a uniform change in the dendritic size on that side of the cell. We took a number of steps to insure equivalent deprivation across frequencies and adequate analytical abilities, including the following: The neuronal system is balanced such that each cell had its own control and we analyzed both the dorsal and the ventral dendrites on each side of the brain; there is considerable background data on the normal organization and growth of NL dendrites (Smith and Rubel, '79; Smith, '81); each of the primary synaptic connections from the eighth nerve to NM and from NM to each side of NL is both synaptically and metabolically excitatory (Lippe et al., '80; Hackett et al., '82); calibration of the acute and chronic conductive hearing loss produced by the ear plug showed that it produced a relatively flat 40-dB conductive deficit (Kerr et al., '79); and the subjects were housed in an acoustically controlled enrivonment with a flat frequency spectrum of 65 dB (SPL), which should produce a maximum ear plug-induced change in the evoked firing rate of NM and NL cells (Rubel and Parks, '75; Sachs and Sinnott, '78). While there certainly remained uncontrolled environmental variables (e.g., the animals' own vocalizations), the object of all of these procedures was to provide as close as possible to a uniform difference in the activity impinging on the two dendritic surfaces of NL cells. The expected result of uniform changes across the laminar nucleus would have been interpreted as support for the simple proposition that dendritic size is monotonically related to the impinging activity. Why, then, did the dendritic changes vary as a function of frequency? The three major classes of explanations that seem most likely are discussed below.

Differential cellular response. One possibility is that the difference in activity impinging on the deprived and nondeprived dendrites was uniform throughout the nucleus but the cellular response to the abnormal conditions differed in regions normally responsive to high and low frequencies. Parks ('81b) found that otocyst removal differentially affected the absolute size of NL dendrites such that the dendritic gradient was reduced on the affected side of the cells. In the present study, the differences could result from a number of factors. For example, it is known that the dendrites of NL are not fully developed at embryonic day 19 and the low-frequency (sector 4) cells are the most immature (Smith, '81). Thus, differential effects of deprivation on low- and high-frequency areas could be due to different developmental states at the time of the deprivation. Alternatively, the cells may differ in some other way such as other inputs, transmitters, maintenance of sprouted terminals, etc. In the most general sense this explanation suggests that the dendritic change may not be simply a function of the change in activity impinging upon it. Support for this interpretation can be readily found in the literature on visual system deprivation, where deprivation-induced changes can be separated from changes resulting from "competitive interactions" among cells (e.g., Guillery and

Stelzner, '70; Sherman et al., '74; Casagrande and Joseph, '80; Hickey, '80).

Differential activity. Another class of explanations is derived from the possibility that the *activity change* produced by the ear plug in the low- and high-frequency areas of NL was markedly different. The effect of ear plugging on neural activity impinging on the brainstem auditory system may not be a simple reflection of the conductive hearing loss. Stimulation of the basilar membrane can be accomplished by several means, only one of which is normal air-conducted sound. Basilar membrane movement may also result from bone-conducted external sounds or from internal sounds such as blood passing through the middle ear or the cochlea itself. Normally, these latter sources of stimulation probably play a minor role in the activity of the auditory nerve. With an ear plug, however, the situation may be quite different. For example, occlusion of the meatus and mass loading of the tympanum may enhance low-frequency bone-conducted sounds (Tonndorf, '66) and low-frequency internal noises. Under these conditions the ear plugs would decrease all air-conducted sounds, but bone conduction and internal noises would provide a relative enhancement of low-frequency stimulation of the cochlea. If dendritic size or growth is a reflection of the synaptic activity, then low-frequency NL dendrites which received input from the plugged ear would be expected to be longer than those activated from the nondeprived ear in quiet conditions, exactly the results found here. The most intriguing aspects of this hypothesis are that (1) it suggests that the present study demonstrates both deprivation effects (high-frequency regions) and enrichment effects (low-frequency regions) on individual NL dendrites; and (2) it is testable by studying the effects of ear plugging on spontaneous activity and bone-conducted activity in n. magnocellularis as a function of CF of the neurons, and by examining the effects of other manipulations which would not be expected to produce occlusion effects, such as ossicular removal or disarticulation.

It should be emphasized that the hypothesized effect of mass loading is but one of a class of explanations which posit a differential change in the activity impinging on low- and high-frequency areas of NL due to the ear plug. Other possibilities include conduction of very low frequencies through the earplug; some type of compensation at the level of the middle ear, the efferent system, the hair cells, or NM; or low-frequency transmission involving the interaural pathways known to exist in birds (Hill et al., '80).

Differential damage. A final class of explanations that must be considered in all deprivation studies is the extent to which the deprivation conditions could have caused receptor damage. In the present context one could hypothesize basal cochlea damage resulting in one change and other (deprivation) factors producing the other change. Receptor degeneration has been hypothesized in other cases of auditory deprivation (Clopton, '80). We have analyzed evoked potential thresholds after short survival times (Kerr et al., '79) and hair cell counts after 30 days (Kitch and Rubel, unpublished) and found both to be normal following chronic ear plugging. However, neither this study nor others presuming to produce purely conductive deficits have validated this assumption by showing that bone conduction thresholds are normal at the end of the deprivation period.

Conclusions

The changes in dendritic structure reported here demonstrate that the activity impinging on dendritic processes do play a role in regulating their shape and size. Other examples leading to this conclusion can be derived from a variety of systems in a large number of animals (e.g., Globus, '75; Greenough, '76; Coleman and Riesen, '68; Rutledge, '78; Uylings et al., '78; Ruiz-Marcos and Valverde, '70). On the other hand, relatively little is known about the cellular processes underlying such changes although they may be quite rapid (Desmond and Levy, '82) and involve the local protein-synthesizing machinery of the dendritic process (Steward and Levy, '82). At this juncture it seems that n. laminaris may provide the needed specificity of input and simplicity to approach these problems at a molecular level. No firm conclusions can be made regarding the relationship between afferent activity and dendritic size, per se. However, the fact that the size of dendrites can be differentially regulated on the two sides of the same cell indicates that the cellular mechanisms need involve only local interactions between the presynaptic and postsynaptic membranes.

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