Origins of thalamic and cortical projections to the posterior auditory field in congenitally deaf cats

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A R T I C L E I N F O

Article info:
Received 8 April 2016
Received in revised form 27 May 2016
Accepted 6 June 2016
Available online xxx

Keywords:
Anatomical connectivity
Auditory cortex
Crossmodal plasticity

A B S T R A C T

Crossmodal plasticity takes place following sensory loss, such that areas that normally process the missing modality are reorganized to provide compensatory function in the remaining sensory systems. For example, congenitally deaf cats outperform normal hearing animals on localization of visual stimuli presented in the periphery, and this advantage has been shown to be mediated by the posterior auditory field (PAF). In order to determine the nature of the anatomical differences that underlie this phenomenon, we injected a retrograde tracer into PAF of congenitally deaf animals and quantified the thalamic and cortical projections to this field. The pattern of projections from areas throughout the brain was determined to be qualitatively similar to that previously demonstrated in normal hearing animals, but with twice as many projections arising from non-auditory cortical areas. In addition, small ectopic projections were observed from a number of fields in visual cortex, including areas 19, 20a, 20b, and 21b, and area 7 of parietal cortex. These areas did not show projections to PAF in cats deafened ototoxicly near the onset of hearing, and provide a possible mechanism for crossmodal reorganization of PAF. These, along with the possible contributions of other mechanisms, are considered.

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1. Introduction

While much of the way in which different areas of the brain are connected is established genetically, neural plasticity affords the flexibility to adapt in an experience-dependent manner to best perceive those stimuli we encounter most often. In the case of normal development, this process results in auditory, visual, and somatosensory cortices that represent stimuli from each modality with impressive fidelity, and which interact to provide a multisensory representation of the world around us. However, in the deaf brain, areas that would normally process sound are reorganized to respond to visual (Neville et al., 1983; Finney et al., 2001, 2003; Lee et al., 2001; Lambertz et al., 2005; Pekkola et al., 2005; Lomber et al., 2010; Meredith et al., 2011; Karns et al., 2012) or somatosensory stimulation (Levanen et al., 1998; Levanen and Hamdorf, 2001; Allman et al., 2009; Bhattacharjee et al., 2010; Meredith et al., 2011; Karns et al., 2012), offering functional enhancement in the remaining modalities. While there is ample behavioral and electrophysiological evidence for such crossmodal plasticity, the anatomical changes that underlie these effects are poorly understood. To date, a number of detailed anatomical studies have been undertaken to address this issue across auditory cortical areas in the cat (Barone et al., 2013; Kok et al., 2014; Chabot et al., 2015; Wong et al., 2015; Meredith et al., 2016). One area of particular interest is the posterior auditory field (PAF); in the deaf animal, PAF has been shown to be the neural substrate responsible for mediating visual localization of peripherally presented stimuli (Lomber et al., 2010), and moving high-contrast gratings elicit changes in the blood oxygenation level dependent (BOLD) response in PAF of early-deaf animals (Brown and Lomber, 2012). Moreover, the fractional cortical volume occupied by PAF has been shown to be slightly larger in deaf animals than in hearing animals (Wong et al., 2014). Thus, quantifying the connectivity to deaf PAF may

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http://dx.doi.org/10.1016/j.heares.2016.06.003
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help elucidate the structural basis for crossmodal reorganization. In a recent paper we compared the thalamocortical and cortico-cortical projections to PAF in normal hearing, early-deaf, and late-deaf cats (Butler et al., 2016). While some small-scale differences were observed, the results overwhelmingly suggested an absence of substantial change in the pattern of neurons projecting to PAF following hearing loss. This is of considerable interest in light of behavioral evidence that PAF is functionally reorganized following early hearing loss to subserve visual localization in the peripheral field (Lomber et al., 2010). Indeed, this anatomical finding is in accordance with recent examinations of other auditory cortical fields, demonstrating little or no change in the overall pattern of labelled neurons following deafness (Barone et al., 2013; Chabot et al., 2015; Meredith et al., 2016). Taken together, these findings suggest that functional crossmodal reorganization is not the result of substantial increases in the number of transcortically projecting neurons from the remaining sensory cortices. Rather, it suggests projections that exist in the normal hearing brain are designed to become functionally relevant; this may occur via decreased inhibition, increased synaptic efficiency, unmasking of projections that do not normally produce suprathreshold input, changes in top-down influences, or by some other mechanism. Alternatively, it has been proposed that crossmodal plasticity may occur subcortically, such that projections originating in what would normally be considered auditory brainstem, midbrain, and thalamic nuclei are reorganized to relay information related to non-auditory stimuli (e.g. Dehmel et al., 2008; Allman et al., 2009).

Despite this mounting evidence for structural preservation of projections to auditory cortical fields in the deaf, the possibility remains that brief exposure to sound might be sufficient to drive the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in remians that brief exposure to sound might be sufficient to drive the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the development of normal patterns of connectivity, even in models of early-onset deafness. While the duration of hearing in the develop
physiological saline, 4% paraformaldehyde, and 10% sucrose to
cryoprotect the tissue. The head was mounted in a stereotaxic
frame and the brain was exposed and blocked in the coronal plane
at Horsley-Clarke level A27. Brains were frozen and a total of 6
series of 60 μm serial coronal sections were collected. One series
was processed to reveal the presence of BDA using the avidin-
biotin peroxidase method with nickel-cobalt intensification
(Veenman et al., 1992). In order to visualize laminar and areal
borders, three of the remaining series were processed using the
monoclonal antibody SMI-32 (Sternberger and Sternberger, 1983),
cytochrome oxidase (Payne and Lomber, 1996), and cresyl violet to
label Nissl bodies. The remaining two series were spares, and were
processed using the above methods as necessary. All sections were
mounted on gelatin-coated slides, air dried, cleared, and cover-
slipped.

2.3. Data analysis

BDA-labelled neurons were visualized using a Nikon E600 mi-
croscope. Tissue sections and injection sites were outlined and an
unbiased and comprehensive count of labelled neurons was
completed using Neurolucida software (MBF Bioscience, Williston,
VT). Only those neurons in which the entirety of the cell soma was
labelled were included; partial cell bodies or dendritic branches
alone were not quantified, providing a conservative estimate of
labelled cells. The full thickness of each section was examined by
taking focal levels throughout the z-plane. Labels were assigned to
cortical areas based on each animal’s cytoarchitectural, sulcal, and
gyral landmarks defining areal borders. Patterns of SMI-32 labelling
differ by area in auditory and visual cortex, allowing for demarca-
tion of areal borders (van der Gucht et al., 2001; Mellott et al., 2010),
and these patterns are conserved following hearing loss (Wong
et al., 2014). Somatosensory areas can be distinguished from
auditory cortical fields by a marked increase in SMI-32 reactivity
(van der Gucht et al., 2001), while borders between somatosensory
areas are primarily delineated using Nissl labelling profiles (Clasca
et al., 1997). As supported by the cytoarchitecture of the visual
system (van der Gucht et al., 2001), borders between the posterior
lateral suprasylvian areas (PLLS and PMLS), and the dorsal and
ventral lateral suprasylvian areas (DLS and VLS) of visual cortex
were placed on the lateral bank of the middle suprasylvian sulcus
and the dorsal bank of the posterior limb of the suprasylvian sulcus,
respectively (as per Palmer et al., 1978; Updyke, 1986; Rauschecker et al., 1987).

3. Results

3.1. Injection sites & tracer spread

Three cats received injections of the retrograde tracer BDA, ensuring axon terminals in all six cortical layers of the left PAF were exposed. The three injection tracks were placed along the posterior bank of the posterior ectosylvian sulcus (Fig. 1), with no evidence of tracer spread beyond the borders of PAF.

3.2. Projections to PAF in the congenitally deaf cat

A representative profile of labelling throughout the brain is presented in Fig. 3. Following injection of BDA into PAF, labelled neurons throughout the hemisphere ipsilateral to the injection site were assigned to their cortical or thalamic area of origin (Fig. 4), quantified, and converted to a proportion of the total number of labelled cells in that hemisphere on an individual animal basis. Because the current study was interested in the thalamocortical and corticocortical projections to PAF, the total number of labelled cells was taken as the sum of the labelled cells in all thalamic and cortical fields ipsilateral to the tracer injection, excluding those labelled cells within PAF itself; labelled cells in subcortical fields
Counts differed immunohistochemical processing that make interpreting raw cell counts difficult (raw cell counts are provided in Table 1). Within the auditory cortex, projections arose from each of the other 12 fields identified in the cat (Fig. 5), with the largest originating in the primary auditory cortex (A1). Smaller projections arose from the second auditory cortex (A2), the anterior auditory field (AAF), the dorsal, intermediate, and ventral divisions of the posterior ectosylvian auditory cortex (dPE, iPE, vPE), the dorsal zone of auditory cortex (DZ), the auditory field of the anterior ectosylvian sulcus (fAES), the insular and temporal cortices (IN & CVA), the posterior ectosylvian gyrus (EpP), and the posterior suprasylvian area (PS). Small projections also arose from secondary somatosensory areas (S2 & S2m) and the fourth somatosensory cortex (S4) as well as areas 7, 35, and 36, the anterior and posterior cingulate cortices (CGA & CGP), and the retrosplenial area (RS; Fig. 7). Thalamic labelling was restricted to the dorsal, medial, and ventral divisions of the medial geniculate body (MGBd, MGBm, & MGBv) and the lateral posterior nucleus (LP; Fig. 8). In order to compare the relative size of the projection arising from each modality, cells labelled by retrograde injection into PAF were classified as either thalamic in origin, or as arising from auditory, visual, somatosensory, or other cortical areas. The total number of cells of each type was then divided by the total number of labelled cells in each individual animal to determine the proportion of labelled cells from each modality. Fig. 9A shows that in the congenitally deaf animal, neurons projecting to PAF originate overwhelmingly in auditory thalamic and cortical areas, with 12.9% of labelled cells arising from non-auditory cortical areas.

### 4. Discussion

#### 4.1. Summary & comparison to existing data

The current study quantifies the projections to the posterior auditory field in the congenitally deaf cat. A summary of substantial projections from cortical and thalamic fields throughout the brain is provided in Fig. 10A; labelling patterns previously outlined for early-deaf and normal hearing animals are presented in panels B and C (adapted from Butler et al., 2016). While there are some small differences in the magnitude of projections, the overall pattern is qualitatively very similar between groups.

Projections to PAF were also considered at the level of modality of origin. These data are presented along with modality-level differences in the magnitude of projections, the overall pattern is qualitatively very similar between groups.

#### Table 1

<table>
<thead>
<tr>
<th>Cortical area</th>
<th>Mean cell count (mean ± S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>167.0 ± 80.7</td>
</tr>
<tr>
<td>A2</td>
<td>91.0 ± 92.0</td>
</tr>
<tr>
<td>AAF</td>
<td>25.7 ± 15.3</td>
</tr>
<tr>
<td>dPE</td>
<td>83.0 ± 90.8</td>
</tr>
<tr>
<td>DZ</td>
<td>66.0 ± 33.8</td>
</tr>
<tr>
<td>fAES</td>
<td>23.7 ± 11.6</td>
</tr>
<tr>
<td>IN</td>
<td>5.3 ± 6.7</td>
</tr>
<tr>
<td>iPE</td>
<td>46.7 ± 34.5</td>
</tr>
<tr>
<td>T</td>
<td>16.0 ± 10.4</td>
</tr>
<tr>
<td>VAF</td>
<td>44.3 ± 54.4</td>
</tr>
<tr>
<td>VPAF</td>
<td>85.0 ± 66.8</td>
</tr>
<tr>
<td>vPE</td>
<td>23.3 ± 10.1</td>
</tr>
<tr>
<td>17</td>
<td>1.3 ± 1.5</td>
</tr>
<tr>
<td>19</td>
<td>2.7 ± 2.5</td>
</tr>
<tr>
<td>20a</td>
<td>2.7 ± 1.2</td>
</tr>
<tr>
<td>20b</td>
<td>0.7 ± 0.6</td>
</tr>
<tr>
<td>21b</td>
<td>4.7 ± 7.2</td>
</tr>
<tr>
<td>AEV</td>
<td>3.3 ± 2.9</td>
</tr>
<tr>
<td>ALLS</td>
<td>26.0 ± 14.9</td>
</tr>
<tr>
<td>CVA</td>
<td>1.3 ± 1.2</td>
</tr>
<tr>
<td>EPd</td>
<td>35.3 ± 31.2</td>
</tr>
<tr>
<td>PLLS</td>
<td>3.7 ± 2.5</td>
</tr>
<tr>
<td>P5</td>
<td>9.3 ± 7.2</td>
</tr>
<tr>
<td>S2</td>
<td>17.3 ± 20.5</td>
</tr>
<tr>
<td>S2m</td>
<td>23.7 ± 31.9</td>
</tr>
<tr>
<td>S4</td>
<td>17.3 ± 21.0</td>
</tr>
<tr>
<td>7</td>
<td>1.3 ± 0.6</td>
</tr>
<tr>
<td>35</td>
<td>1.0 ± 1.0</td>
</tr>
<tr>
<td>36</td>
<td>8.7 ± 5.7</td>
</tr>
<tr>
<td>CGA</td>
<td>0.3 ± 0.6</td>
</tr>
<tr>
<td>CGP</td>
<td>0.7 ± 1.2</td>
</tr>
<tr>
<td>RS</td>
<td>1.3 ± 0.6</td>
</tr>
<tr>
<td>LP</td>
<td>23.0 ± 15.0</td>
</tr>
<tr>
<td>MGBd</td>
<td>72.0 ± 29.7</td>
</tr>
<tr>
<td>MGBm</td>
<td>56.7 ± 16.0</td>
</tr>
<tr>
<td>MGBv</td>
<td>199.7 ± 112.5</td>
</tr>
</tbody>
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Please cite this article in press as: Butler, B.E., et al., Origins of thalamic and cortical projections to the posterior auditory field in congenitally deaf cats, Hearing Research (2016), http://dx.doi.org/10.1016/j.heares.2016.06.003
quantifications in the hearing animal in Fig. 9 (panel B adapted from Butler et al., 2016). While only 12.9% of labelled cells in the congenitally deaf animal originated in non-auditory cortical areas, this represents more than a doubling of the number of non-auditory projections in hearing cats.

4.2. Ectopic projections from visual and parietal cortex

Following an injection of BDA into PAF in congenitally deaf cats, labelled cells were observed in a number of cortical areas that do not contain projections to PAF in normal hearing animals (Butler et al., 2016). These fields include visual cortical areas 19, 20a, 20b, and 21b, and area 7 of parietal cortex (Fig. 11). While these projections account for a very small proportion of the overall number of labelled cells in these animals (all <0.3%), they were reliably present across the animals tested in the current study.

This begs the question of whether small projections from these visual cortical fields to PAF might play a role in enhanced visual localization, as documented in deafened cats. Lomber et al. (2010) noted that while deaf and hearing animals localize visual stimuli with similar accuracy near the center of the visual field, deaf animals are significantly better than normal hearing cats when these same stimuli are presented in the visual periphery. It is expected then, that projections to PAF underlying such a response arise from visually-responsive areas with representations of the peripheral field. Indeed, the visual field representations of areas 20a, 20b, and 21b are broad, extending into the periphery (Fig. 12; Tusa and Palmer, 1980). Conversely, congenitally deaf PAF does not receive a projection from area 21a, where the visual field representation is confined to an area within 20 degrees of the vertical meridian. Cells in area 21b are binocularly responsive, with large receptive fields and strong sensitivity to the directions of drifting gradients (Tardif et al., 2000), and project primarily to areas 20a and 20b (Segraves and Rosenquist, 1982). Based on position within the cortex and receptive field properties, area 21b (along with 21a) appears to be analogous to V4 of the macaque (Payne, 1993), a field which shows enhanced neural responses to visual targets that are selected for foveation, reflecting a serial component of visual search (Bichot et al., 2005).
In addition to localizing a visual stimulus, the behavioral measure of crossmodal plasticity observed by Lomber et al. (2010) involved an overt approach response. Accordingly, areas 20a and 20b project to the pontine nuclei, and subsequently to the cerebellum of the cat (Bjaalie, 1989), making these areas a likely contributor of visual information related to overt movement. Based on this pattern of projection, as well as cortical position and interhemispheric connectivity, it has been suggested that areas 20a and 20b are homologues of parahippocampal areas TF and TH in the macaque (Payne, 1993), fields which have been shown to play a significant role in spatial memory (Bachevalier and Nemanic, 2008). In addition, area 7 in the cat is a high-level multisensory area that has been described as modulating activity in the visual cortex during the presentation of stimuli that elicit an action (von Stein et al., 2000). Similarly, in the monkey, this area is related to awareness of the location of the body in relation to its spatial environment, and to directed motor behavior toward a target stimulus (Hyvarinen and Poranen, 1974). Thus, while the ectopic projections from areas 20a, 20b, 21b, and area 7 to PAF of the congenitally deaf cat are small, they satisfy two presumptive criteria for areas that may contribute to the enhanced localization behavior observed by Lomber et al. (2010): i) if they have visual field representations that extend into the periphery, and ii) they are involved in linking the perception of visual target stimuli and a behavioral response.

**4.3. Effects of early experience and mechanistic considerations**

While previous studies have failed to provide evidence in support of substantial anatomical change following deafness, their conclusions may be limited by the fact that even early-deaf animals experience a brief exposure to sound as a result of the methodology employed to elicit hearing loss. While there are several approaches to generate animal models of hearing loss (see Butler and Lomber, 2013 for review), ototoxic deafening is the predominant method in the study of feline models. The two most popular methodologies include: 1) the one-time administration of an aminoglycoside in combination with a loop diuretic that is infused until evoked responses are abolished (e.g. Xu et al., 1993; Butler et al., 2016); or 2) daily administration of an aminoglycoside from postnatal day 1 until the desired hearing deficit is obtained (e.g. Leake et al., 1997). The former has been shown to be maximally effective in the cat at or after postnatal day nine (Shepherd and Martin, 1995), with co-administration of aminoglycoside and loop diuretic prior to this age resulting in little or no hearing impairment (in reality, the effect appears to be dependent on body weight rather than postnatal age per se). Moreover, because the end-point of this procedure is typically defined by the absence of the auditory brainstem response (ABR), deafening cannot take place before the ear canals open, at or around postnatal day 11. This latter limitation is avoided by a daily aminoglycoside regimen; however, because ototoxicity is related to...
in the cat, with complete deafening occurring days later. Thus, while both methods of ototoxic deafening provide models of profound, early-onset deafness, neither is capable of addressing changes that occur in the complete absence of stimulus-evoked activity. Fortunately, the deaf white cat provides a model of human congenital deafness, and allows for the quantification of connectivity in the truly naïve auditory cortex.

We have argued that small ectopic projections from visual and multisensory cortical areas to PAF may contribute to enhanced peripheral localization behavior observed in congenitally deaf animals (Lomber et al., 2010). That novel projections from areas 20a, 20b, 21b, and area 7 exist in congenitally deaf cats, but not in animals deafened near the onset of hearing (Butler et al., 2016), suggests that connections between non-auditory cortical areas and PAF are indeed modified by brief periods of early auditory experience. It has been well established that the developing brain undergoes a period of exuberant connectivity that is followed by a refinement period during which transient connections are selectively eliminated (see Innocenti and Price, 2005 for review). Indeed, while a blueprint for connectivity in the auditory cortex is established prior to the onset of hearing, the refinement of this network is experience-dependent (e.g. King and Moore, 2000; Zhang et al., 2001). Moreover, research in the visual system has suggested that even a few hours of patterned input is sufficient to initiate this refinement (e.g. Mower et al., 1983; Rosen et al., 1992). Thus, it is possible that the period of hearing experience that occurs between hearing onset and deafening, even in early-deaf models, may be sufficient to initiate refinement of auditory cortical structure. As a result, transient connections between visual and auditory cortical fields that persist in the congenitally deaf animal may be: i) pruned away entirely, or ii) reduced in number such that the sensitivity of our measurement method is insufficient to quantify these projections.

Despite the absence of ectopic visual projections in early-deaf cats, we have observed visually-evoked BOLD activity in PAF of these animals (Brown and Lomber, 2012). Similarly, the fAES in early-deaf cats has been shown to be behaviorally and electrophysiologically responsive to visual input in the cat (Meredith et al., 2011), despite a lack of novel visual projections (Meredith et al., 2016). These findings suggest that other mechanisms (either in addition to, or in place of ectopic connections between sensory cortices) underlie crossmodal plasticity following sensory loss. Indeed, there is evidence to suggest that crossmodal projections that normally provide subthreshold inputs are unmasked in the

the onset of auditory function (Shepherd and Martin, 1995), the drug is increasingly effective as the cochlea matures throughout the first two weeks of life (e.g. Brugge et al., 1978). That aminoglycosides are maximally effective near the completion of inner ear development is consistent with findings across species, including mice (Chen and Saunders, 1983; Henry et al., 1981) and humans (Bernard, 1981), and typically results in a significant elevation in click-evoked hearing thresholds prior to the opening of ear canals

![Fig. 11. Histogram showing projections to PAF in congenitally deaf animals (white) that were not previously observed in normal hearing animals. An ectopic projection from area 19 was also observed previously in a single early-deaf animal (Butler et al., 2016), and is presented in grey for comparison. Error bars represent the standard error of the mean. For abbreviations, see list.](image1)

![Fig. 12. Visual field representations in visual cortical areas 20a (A), 20b (B), 21a (C), and 21b (D), as adapted from Tusa and Palmer (1980; their Fig. 5). Areas to which neurons are responsive are shaded in grey. The vertical and horizontal meridians are represented by solid and dashed bold lines, respectively.](image2)
absence of sensory stimulation (e.g. Théoret et al., 2004), or that the synaptic density and/or efficacy of these connections are increased in the deaf (e.g. Clemo et al., 2016). It is likely that some combination of these mechanisms gives rise to crossmodal plasticity and resultant compensatory behaviors.

5. Conclusions

Following an injection of BDA into the posterior auditory field of congenitally deaf cats, the pattern of labelled cells throughout the brain is largely similar to that of hearing animals. When considered at the modality level, there are more than twice as many nonauditory projections to PAF than in hearing animals (Fig. 9), driven primarily by an increase in the number of labelled cells in visual cortical areas. While these cells were spread throughout a number of areas, projections were observed in areas 20a, 20b, and 21b areas that do not project to PAF in hearing animals. The presence of these ectopic projections, along with a projection arising from area 7 in parietal cortex, has implications both for the effect of hearing loss on crossmodal connectivity to PAF, and for the consequences of very brief periods of early auditory exposure. These novel projections arise from areas that are typically involved in visual localization and response behaviors, and thus, are strong candidates to underlie enhanced localization in the deaf; however, functional experiments targeting these visual cortical fields and anterograde tracing experiments capable of quantifying synapses between visual cortical areas and PAF are necessary to fully understand the nature of anatomical change underlying functional enhancement.

Acknowledgements

The authors wish to thank Pam Nixon for technical and surgical assistance. We gratefully acknowledge the support of the Canadian Institutes of Health Research, Natural Science and Engineering Research Council of Canada, and Deutsche Forschungsgemeinschaft (GR).