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# Cross-modal plasticity in specific auditory cortices underlies visual compensations in the deaf

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**When the brain is deprived of input from one sensory modality, it often compensates with supranormal performance in one or more of the intact sensory systems. In the absence of acoustic input, it has been proposed that cross-modal reorganization of deaf auditory cortex may provide the neural substrate mediating compensatory visual function. We tested this hypothesis using a battery of visual psychophysical tasks and found that congenitally deaf cats, compared with hearing cats, have superior localization in the peripheral field and lower visual movement detection thresholds. In the deaf cats, reversible deactivation of posterior auditory cortex selectively eliminated superior visual localization abilities, whereas deactivation of the dorsal auditory cortex eliminated superior visual motion detection. Our results indicate that enhanced visual performance in the deaf is caused by cross-modal reorganization of deaf auditory cortex and it is possible to localize individual visual functions in discrete portions of reorganized auditory cortex.**



Studies of deaf or blind subjects often report enhanced perceptual abilities in the remaining senses. Compared with hearing subjects, psychophysical studies have revealed specific superior visual abilities in the early deaf[1] as well as enhanced auditory functions in the early blind[2–4]. The neural substrate for these superior sensory abilities is thought to reside in the deprived cerebral cortices that have been reorganized by the remaining sensory modalities through cross-modal plasticity. Thus, in early blind animals, acoustic stimuli evoke responses in what are normally visual regions of the cerebral cortex[2], and heteromodal activity after early deprivation has been repeatedly demonstrated in the visual and somatosensory systems[4–8], consistent with the results of imaging studies performed in deaf subjects[9–11]. In this context, it has been proposed that auditory cortex of the deaf may be recruited to perform visual functions[12]. However, a causal link between supranormal visual performance and the visual activity in the reorganized auditory cortex has never been demonstrated. Furthermore, if auditory cortex does mediate the enhanced visual abilities of the deaf, it is unknown whether these functions are distributed uniformly across deaf auditory cortex or whether specific functions can be differentially localized to distinct portions of the affected cortices. It is also unknown whether reorganized cortex retains any relationship to functions performed in these regions in hearing subjects. These fundamental questions are clinically important now that restoration of hearing in prelingually deaf children is possible through cochlear prosthetics.

To address these issues, we first examined the visual abilities of congenitally deaf and hearing cats to identify those visual functions that are enhanced in the early deaf. We then examined the role of deaf auditory cortex in mediating the superior visual abilities by reversibly deactivating specific cortical loci with cooling. This combination of experimental approaches revealed a causal link between the cross-modal reorganization of auditory cortex and enhanced visual abilities of the deaf and identified the neural regions responsible for those improvements in visual performance.

## RESULTS

### Enhanced visual abilities of congenitally deaf cats

We compared the performance of adult hearing ($n$ = 3) and congenitally deaf ($n$ = 3) cats[13] on seven visual psychophysical tasks. Prior to training on the visual tasks, deafness was confirmed with a standard screening method using auditory brainstem responses (**Supplementary Fig. 1**). In the hearing cats, the same method was used before the experiment to confirm that their hearing thresholds were within normal limits (**Supplementary Fig. 1**).

In the first task, we tested visual localization by placing the cats in an arena and examining their ability to accurately localize, by orienting and approaching, the illumination of red LEDs that were placed at 15° intervals across 180° of azimuth (**Supplementary Fig. 2**). In hearing controls, performance was excellent throughout the central 90° of the visual field (45° to the left and right), but accurate localization declined across the most peripheral targets tested (60–90°; **Fig. 1a**). In contrast, visual localization performance of deaf cats was maintained at higher levels throughout the most peripheral visual field (**Fig. 1a**). Performance of the deaf cats was significantly better for the 60°, 75° and 90° positions ($P < 0.01$), whereas there was no difference across the central 90° of the visual field (left 45° to right 45°; **Fig. 1a**,**b**). This result was consistent for both binocular and monocular testing. Overall, the superior visual localization abilities of deaf cats correspond well with findings from prelingually deaf human subjects[8].

We conducted an additional six visual tests in a two-alternative forced-choice apparatus using standard staircase procedures to

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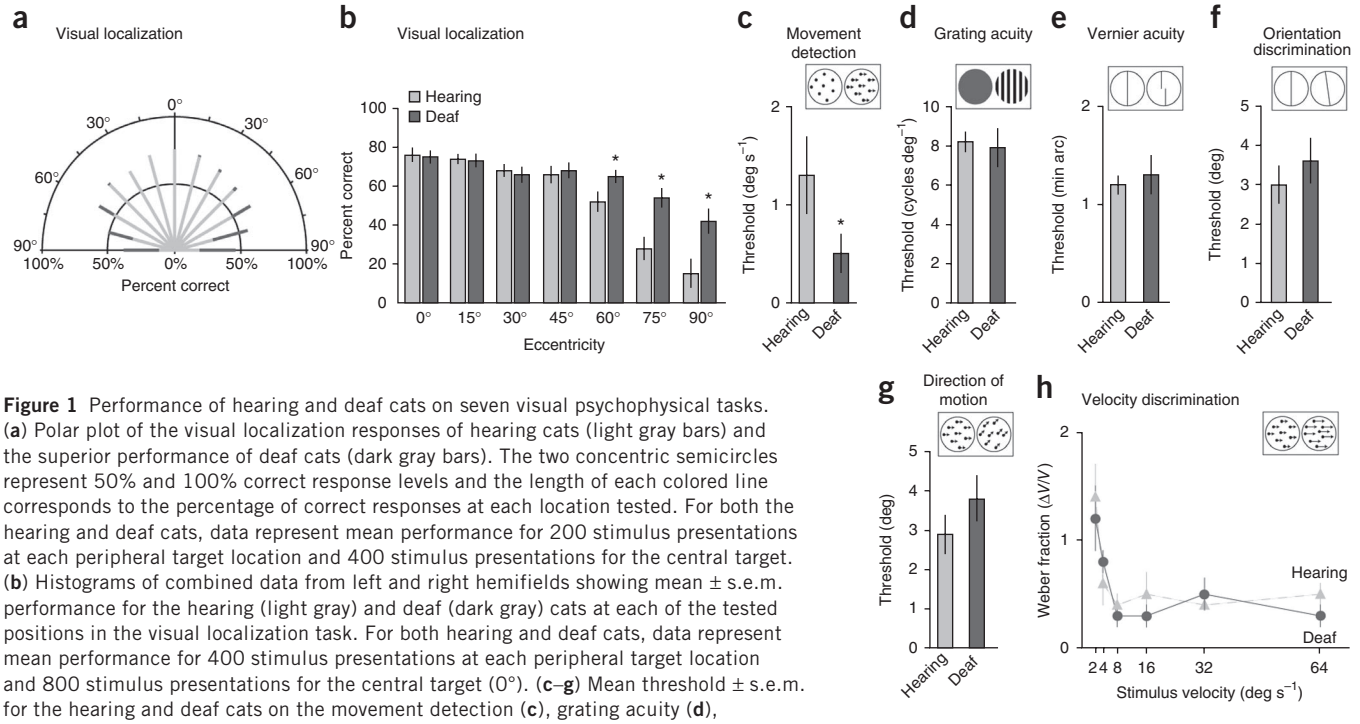


**Figure 1** Performance of hearing and deaf cats on seven visual psychophysical tasks. (**a**) Polar plot of the visual localization responses of hearing cats (light gray bars) and the superior performance of deaf cats (dark gray bars). The two concentric semicircles represent 50% and 100% correct response levels and the length of each colored line corresponds to the percentage of correct responses at each location tested. For both the hearing and deaf cats, data represent mean performance for 200 stimulus presentations at each peripheral target location and 400 stimulus presentations for the central target. (**b**) Histograms of combined data from left and right hemifields showing mean ± s.e.m. performance for the hearing (light gray) and deaf (dark gray) cats at each of the tested positions in the visual localization task. For both hearing and deaf cats, data represent mean performance for 400 stimulus presentations at each peripheral target location and 800 stimulus presentations for the central target (0°). (**c**–**g**) Mean threshold ± s.e.m. for the hearing and deaf cats on the movement detection (**c**), grating acuity (**d**), Vernier acuity (**e**), orientation (**f**) and direction of motion (**g**) discrimination tasks. (**h**) Performance of the hearing and deaf cats on the velocity discrimination task. Data are presented as Weber fractions for six different stimulus velocities. *$P < 0.01$ between the hearing and deaf conditions. Sample stimuli are shown for each task.



determine psychophysical thresholds (**Supplementary Fig. 2**). In hearing cats, the movement detection threshold was $1.3 \pm 0.4°\ s^{-1}$ (**Fig. 1c**), consistent with earlier reports[14]. In contrast, the movement detection threshold for the deaf cats was significantly lower ($P < 0.01$; $0.5 \pm 0.2°\ s^{-1}$; **Fig. 1c**). For the remaining five tests of visual function (grating acuity, Vernier acuity, orientation discrimination, direction of motion discrimination and velocity discrimination), the performance of the deaf cats was not significantly different from that of hearing controls (**Fig. 1d**–**h**). Overall, visual psychophysical performance of the three deaf cats examined was highly stereotyped.

## Deaf PAF mediates enhanced visual peripheral localization

At the conclusion of psychophysical testing, we collectively and individually deactivated portions of auditory cortex (**Fig. 2a**) to determine whether specific cortical areas mediated the enhanced visual functions. In both the deaf and hearing cats, individual cooling loops[15] were bilaterally placed over the posterior auditory field (PAF), the dorsal zone of auditory cortex (area DZ) and primary auditory field (A1), as they are involved in auditory localization in hearing cats[16,17] (**Fig. 2b**). An additional control cooling loop was placed over the anterior auditory field (AAF), as it is involved in pattern, but not spatial, processing[18].

For the visual localization task, we first determined whether auditory cortex could be mediating the enhanced performance of the deaf cats (**Fig. 3**). We simultaneously deactivated PAF, DZ, A1 and AAF bilaterally, which resulted in a significant reduction in visual localization performance restricted to the most peripheral targets (60°, 75° and 90° positions, $P < 0.01$; **Fig. 3a**,**b**). Although the cats often failed to accurately or precisely localize the stimulus in the far periphery, the illumination of any LED always triggered a response. Therefore, the nature of the deficit was one of localization and not detection. Errors made during bilateral deactivation of all four areas were almost

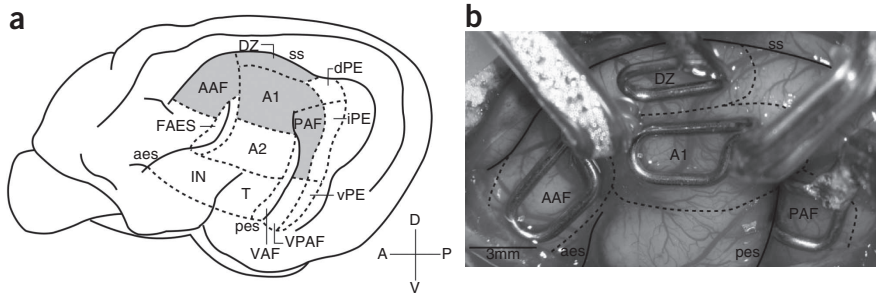


**Figure 2** Cortical areas deactivated in deaf auditory cortex. (**a**) Schematic illustration of the left hemisphere of the cat cerebrum showing all of the auditory areas (lateral view). The areas that we examined are highlighted in gray. A, anterior; A2, second auditory cortex; aes, anterior ectosylvian; D, dorsal; dPE, dorsal posterior ectosylvian area; FAES, auditory field of the anterior ectosylvian sulcus; IN, insular region; iPE, intermediate posterior ectosylvian area; P, posterior; pes, posterior ectosylvian; ss, suprasylvian; T, temporal region; V, ventral; VAF, ventral auditory field; VPAF, ventral posterior auditory field; vPE, ventral posterior ectosylvian area. The areal borders shown in this figure are based on a compilation of electrophysiological mapping and cytoarchitectonic studies. (**b**) Cooling loops in contact with areas AAF, DZ, A1 and PAF of the left hemisphere of a congenitally deaf cat at the time of implantation. Left is anterior. The areal borders presented in this figure are based on the post-mortem analysis of SMI-32 processed tissue from the brain shown here.

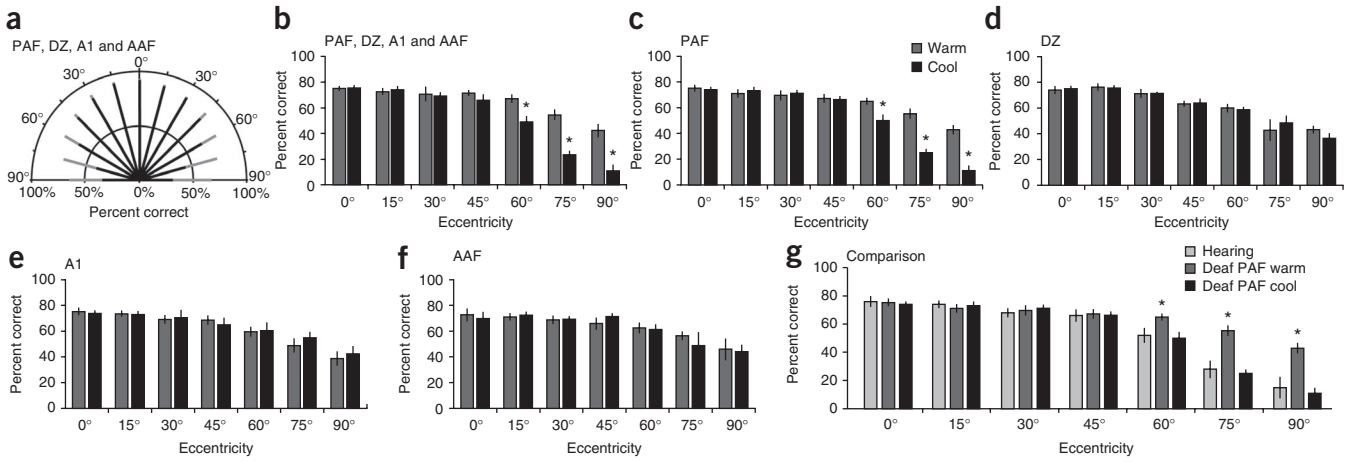


**Figure 3** Visual localization task data from deaf cats during bilateral reversible deactivation of PAF, DZ, A1 and AAF. (**a**) Polar plot of the visual localization responses of deaf cats while cortex was warm (dark gray) and active and during simultaneous cooling deactivation of PAF, DZ, A1, and AAF (black). (**b**–**f**) Histogram of combined data from the left and right hemifields showing mean ± s.e.m. performance for deaf cats while cortex was warm (dark gray) and active and while it was cooled (black) and deactivated. Asterisks indicate a significant difference ($P < 0.01$) between the warm and cool conditions. (**b**) Data from the simultaneous deactivation of PAF, DZ, A1 and AAF. (**c**–**f**) Data from individual area deactivations. (**g**) Visual localization data comparing performance at each position for hearing cats (light gray), deaf cats while PAF was warm (dark gray), and deaf cats while PAF was cooled (black). *$P < 0.01$ from the hearing and deaf PAF cool conditions.

always undershoots of 30–60° (97.8% of all errors). Rarely (4.3% of all errors) were errors made to the incorrect hemifield. These results clearly indicate that auditory cortex does have a role in mediating the enhanced visual localization performance of the deaf cats.

To ascertain whether the enhanced localization skills could be further localized to specific loci, we individually bilaterally deactivated each of the four auditory areas. In the deaf cats, bilateral deactivation of PAF significantly reduced localization performance to the most peripheral targets (60°, 75° and 90° positions, $P < 0.01$) while leaving localization performance for the 0°, 15°, 30° and 45° targets unchanged (**Fig. 3c**). The reduction in visual localization at the most peripheral locations resulted in performance that was not different from deactivating all four areas simultaneously (**Fig. 3b**). Moreover, the localization performance of the deaf cats during cooling of PAF was not different from that of hearing cats (**Fig. 3g**). Unilateral deactivation of PAF resulted in reduced visual localization to the same peripheral positions; however, the deficit was specific to the contralateral hemifield (**Supplementary Fig. 3**). Neither bilateral nor unilateral deactivation of DZ, A1 or AAF modified visual localization performance (**Fig. 3d–f** and **Supplementary Fig. 3**). Consequently, the neural basis for the enhanced visual localization skills of the deaf cats can be ascribed to PAF. This is notable, as PAF is normally involved in the accurate localization of acoustic stimuli in hearing cats[18]. These results suggest that, in deafness, PAF maintains a role in localization, albeit visual rather than acoustic.

## Deaf DZ mediates enhanced movement detection

To determine whether auditory cortex could be mediating the enhanced motion detection performance of deaf cats, we simultaneously deactivated PAF, DZ, A1 and AAF. Bilateral deactivation of these four areas significantly increased motion discrimination thresholds from 0.44 ± 0.19° $s^{-1}$ to 1.39 ± 0.35° $s^{-1}$ ($P < 0.01$; **Fig. 4a**). This finding establishes that auditory cortex does have a role in mediating the enhanced motion detection performance of the deaf cats.

To determine whether a specific auditory region could be mediating the enhanced visual motion detection skills of deaf cats, we bilaterally cooled areas PAF, DZ, A1 and AAF individually (**Fig. 4b–e**). Bilateral deactivation of DZ significantly increased the motion detection thresholds from 0.40 ± 0.15° $s^{-1}$ to 1.46 ± 0.4° $s^{-1}$ ($P < 0.01$; **Fig. 4c**). This increase resulted in performance that was not different from that seen when all four areas are simultaneously deactivated (**Fig. 4a**). Moreover, the increase in threshold resulted in performance that was not different from that in hearing cats (**Fig. 4f**). There was no evidence of any functional lateralization, as unilateral deactivation of either left or right DZ did not alter performance (**Supplementary Fig. 4**). Neither bilateral (**Fig. 4b**,**d**,**e**) nor unilateral (**Supplementary Fig. 4**) deactivation of PAF, A1 or AAF resulted in any change in motion detection thresholds. These results indicate that DZ cortex mediates the superior visual motion detection thresholds of deaf cats. DZ has neuronal properties that are distinct from those of A1 (refs. 19,20) and is involved in sound source localization[17] and duration coding[20]. We found that DZ is involved in

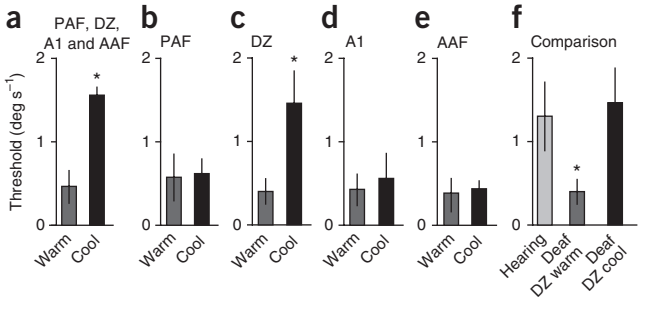


**Figure 4** Motion detection thresholds for the deaf cats before and after cooling deactivation and during bilateral reversible deactivation. (**a**–**e**) Histograms showing mean ± s.e.m. motion detection thresholds for deaf cats while cortex was warm (dark gray) and active and while it was cooled (black) and deactivated. *$P < 0.01$ between the warm and cool conditions. Motion detection thresholds from deaf cats during bilateral reversible deactivation of PAF, DZ, A1 and AAF are shown in **a**. Data from individual area deactivations are shown in **b**–**e**. (**f**) Motion detection thresholds to compare performance of hearing cats (light gray), deaf cats while DZ was warm (dark gray) and deaf cats while DZ was cooled (black). *$P < 0.01$ from the hearing and deaf DZ cool conditions.

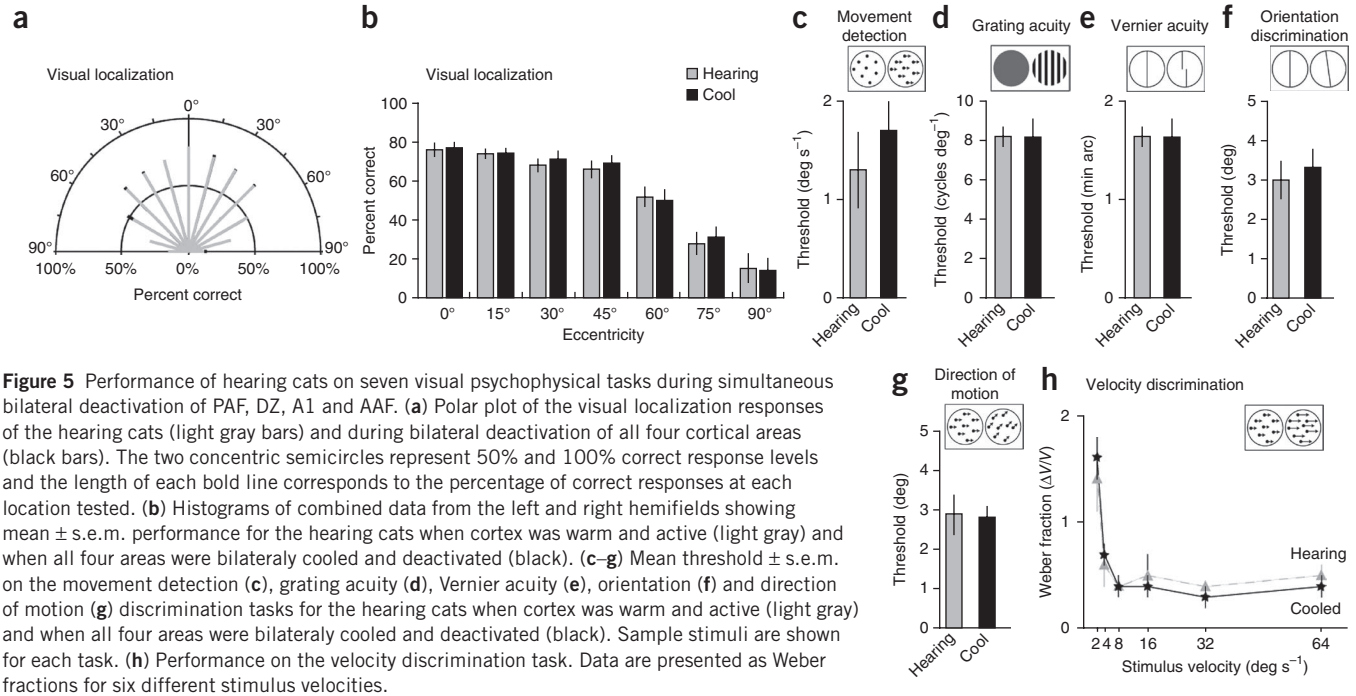


**Figure 5** Performance of hearing cats on seven visual psychophysical tasks during simultaneous bilateral deactivation of PAF, DZ, A1 and AAF. (**a**) Polar plot of the visual localization responses of the hearing cats (light gray bars) and during bilateral deactivation of all four cortical areas (black bars). The two concentric semicircles represent 50% and 100% correct response levels and the length of each bold line corresponds to the percentage of correct responses at each location tested. (**b**) Histograms of combined data from the left and right hemifields showing mean ± s.e.m. performance for the hearing cats when cortex was warm and active (light gray) and when all four areas were bilateraly cooled and deactivated (black). (**c**–**g**) Mean threshold ± s.e.m. on the movement detection (**c**), grating acuity (**d**), Vernier acuity (**e**), orientation (**f**) and direction of motion (**g**) discrimination tasks for the hearing cats when cortex was warm and active (light gray) and when all four areas were bilateraly cooled and deactivated (black). Sample stimuli are shown for each task. (**h**) Performance on the velocity discrimination task. Data are presented as Weber fractions for six different stimulus velocities.



visual motion detection in deaf cats. Assessing the contribution of DZ to acoustic motion perception in hearing cats remains to be determined. In summary, we were able to ascribe superior visual localization functions to PAF (**Fig. 3g**) and the superior motion detection abilities to DZ (**Fig. 4f**) in the same cats.

### Deaf auditory cortex does not mediate unenhanced vision

In addition to deaf auditory cortex serving as the neural substrate for enhanced visual functions, it is also possible that there was an overall redistribution of visual functions in the deaf brain. It might be hypothesized that visual functions that are normally localized in visual cortex may become distributed into deaf auditory cortex. To investigate the possibility that the visual functions that are not enhanced in deaf cats are redistributed over both visual and auditory cortex, we simultaneously deactivated all four of the auditory areas that we examined. For the five visual tasks that were devoid of enhancement in the deaf cats (grating acuity, Vernier acuity, orientation discrimination, direction of motion discrimination and velocity discrimination), neither bilateral nor unilateral collective deactivation of PAF, DZ, A1 or AAF altered performance (**Supplementary Fig. 5**). This evidence suggests that the unenhanced visual functions of deaf cats are not redistributed into auditory cortex.

Given our findings that deaf auditory cortex is the neural substrate for the enhanced visual abilities of the deaf, we sought to determine whether the auditory cortex of hearing cats contributes to visual function. For the group of hearing cats, we both simultaneously and individually deactivated the four auditory areas on each of the seven visual tasks. Overall, neither simultaneous nor individual deactivation of the four auditory regions altered the ability of the hearing cats to perform any of the seven visual tasks (**Fig. 5**). These results indicate that, in the presence of functional hearing, the auditory cortex does not contribute to any of the visual tasks examined. Thus, deficits in visual function identified during deactivation of PAF or DZ in the deaf cats must be caused by underlying cross-modal plasticity in each area.

### Extent of cortical deactivations

At the conclusion of the behavioral testing, we determined the extent of cortical cooling deactivation provided from each cryoloop by cooling each loop individually to the same temperature used during behavioral testing and recording the temperature from cortex in and surrounding the cooling loops. In the left hemisphere of a congenitally deaf cat (shown in **Fig. 2b**), we collected temperature measurements from 335 recording sites across dorsal auditory cortex (**Fig. 6**). As we have done previously, we constructed thermal cortical maps from cooling each individual cryoloop by generating Voronoi tessellations[21] (**Fig. 6c**–**f**). Consistent with previous findings[15–18,21], three observations can be made concerning the thermal maps. First, the cooling of each loop is highly circumscribed, with cooling seldom spreading more than 1 mm from the lateral border of a cooling loop. Second, even though the four areas were in close proximity, there was little overlap of the deactivated regions. Third, a heat-shielding compound applied to the anterior surface of the PAF loop was highly effective at directing the cooling posteriorly, with little or no cooling being evident on the anterior bank of the posterior ectosylvian sulcus. Overall, the cooling loops were highly effective at producing localized and reversible deactivation of discrete regions of auditory cortex.

Prior to the surgical placement of the cooling loops, it was not possible for us to determine the exact locations of the four deaf auditory areas, as they are classically defined by the characteristic frequency maps that establish the borders between these four areas. Using previously established procedures[18], we confirmed the locations of the four areas post-mortem with SMI-32 staining and aligned these borders with the cooling deactivation extents determined from the thermocline mapping (**Fig. 6** and **Supplementary Fig. 6**).

For all deactivation extents of the PAF cryoloops, the regions included the anterior-dorsal posterior ectosylvian gyrus, just posterior to the posterior ectosylvian sulcus (**Supplementary Fig. 6**). For the largest extents, the deactivation spread slightly more dorsally and posteriorly away from the posterior ectosylvian sulcus. All deactivations extended down the posterior bank of the posterior ectosylvian sulcus to the

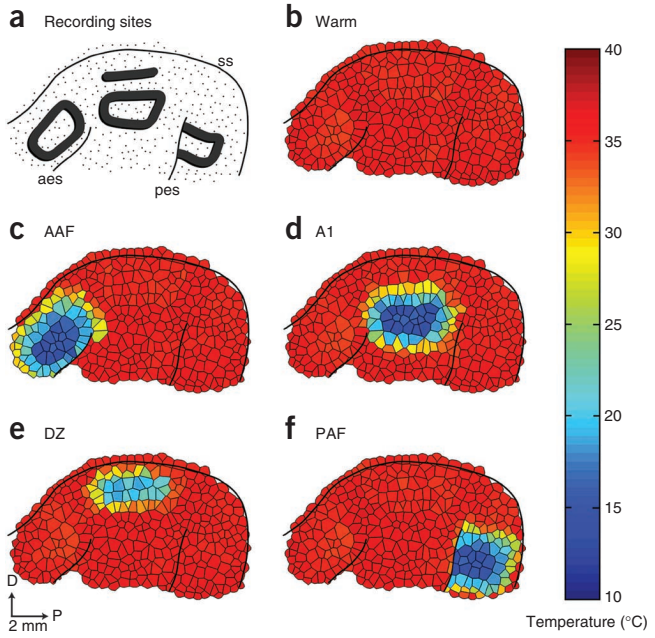


**Figure 6** Thermal cortical maps constructed by generating Voronoi tessellations[21] from 335 temperature recording sites during deactivation of each individual cooling loop. Each image is a dorsolateral view of dorsal auditory cortex from the same brain pictured in **Figure 2b**. A color-coded temperature scale is provided on the right. (**a**) Line drawing showing the locations of the four cooling loops (wide black lines) on the cortical surface and the positions of the 335 temperature recording sites. At each site temperature was recorded 500 μm below the pial surface. (**b**) Cortical temperatures before cooling. (**c**–**f**) Thermal profiles during cooling of each individual cryoloop to 3 °C. Sulci are indicated by thick black lines.

fundus. The deactivations did not include the anterior bank of the sulcus. Thus, the deactivated regions included all of area PAF[22].

For the DZ cryoloops, the dorsal edge of the middle ectosylvian gyrus along the lip of the middle suprasylvian sulcus was deactivated (**Supplementary Fig. 6**). However, the cooling did not appear to directly affect either the anterolateral or posterolateral lateral suprasylvian visual areas[23]. For each loop, the deactivated region included the region previously described as the dorsal zone[24].

For all A1 cryoloop coolings, the central region of the middle ectosylvian gyrus between the dorsal tips of the anterior and posterior ectosylvian sulci was deactivated (**Fig. 6**). The deactivation extended from stereotaxic coronal levels A1 to A12. The deactivated region did not include the dorsal-most aspect of the middle ectosylvian gyrus, along the lateral lip of the middle suprasylvian sulcus (**Supplementary Fig. 6**). In general, compared with the hearing cats, the medial border of A1 tended to be more lateral in the deaf cats, which caused DZ to also be laterally displaced (**Supplementary Fig. 6**). This resulted in A1 deactivations to be more dorsally situated in A1. For each loop, the deactivated region included the dorsal two-thirds of the classically defined area A1 (ref. 22).

Cooling of any of the AAF cryoloops deactivated a large region of the anterior ectosylvian gyrus (**Supplementary Fig. 6**). All deactivations included the dorsal half of the lateral bank of the anterior suprasylvian sulcus and the dorsal half of the medial bank of the anterior ectosylvian sulcus. The largest deactivations extended along the gyrus from A9 to A19, whereas the smaller deactivations extended from A10 to A18. The larger extents deactivated all of area AAF or area A[25]. Although the position of AAF can be variable between animals, its posterior border is seldom caudal to A10 (ref. 25). Thus, the smaller extents also deactivated all of area AAF.

For each region of the auditory cortex that was cooled, the cytoarchitecture of Nissl-stained sections was characteristic of healthy cortex. We were unable to find any evidence of physical damage, gliosis or necrosis. Both myelin and cytochrome oxidase staining were dark, indicative of healthy cortical tissue. Consistent previous findings[26], neither the presence of the cryoloops nor their repeated deactivation over 4 years changed the structure or long-term function of the four cortical sites that we assayed.

## DISCUSSION

Our data suggest a causal link between the cross-modal reorganization of auditory cortex and specific visual functional improvements in the congenitally deaf. Notably, cortical deactivation revealed that different perceptual improvements were dependent on specific and different subregions of auditory cortex. The improved localization of visual stimuli in deaf cats was eliminated by deactivating area PAF, whereas the enhanced sensitivity to visual motion was blocked by disabling area DZ. Because neither cortical area influenced visual processing in hearing cats, these data indicate both that cross-modal reorganization occurred in the PAF and DZ and that the reorganization was functional and highly specific. This close relationship between cross-modal plasticity, specific cortical loci and discrete perceptual enhancements has not, to the best of our knowledge, been previously shown.

The superior visual functions of the congenitally deaf cats are in close agreement with the enhanced visual abilities described in congenitally deaf or early deaf human subjects. Early deaf humans exhibit adaptive or compensatory improvements in detection tasks involving the visual periphery[27,28]. In deaf, but not hearing, subjects, visual stimuli and sign language have been reported to activate auditory cortex[9–11]. However, it remained unknown if the compensatory effects observed in behavior were determined by enhanced cortical processing in visual cortical areas (for example, see ref. 27) or by visual processing in reorganized auditory cortex. Our results suggest that the auditory cortices are involved in this adaptive phenomenon.

Rather than being uniformly distributed across deaf auditory cortex, our results indicate that the neural bases for enhanced visual functions in the deaf are localized to specific auditory cortical subregions. In addition, we went a step further and found that a particular enhanced function could be localized in deaf auditory cortex and that the two different compensatory visual effects could be localized to two distinct regions of deaf auditory cortex. These results reveal a double-dissociation of visual functions in reorganized auditory cortex of the deaf cat (**Fig. 7**). A double dissociation is considered to be the 'gold standard' of behavioral neuroscience, as the results indicate that two cortical regions mediate independent functions/behaviors. Classically, double dissociations are sought by testing two independent groups of subjects, each with a different locus of brain damage (for example, ref. 29). We did not examine two different populations of cats, but, using reversible cooling deactivation, found the dissociations in the same experimental cats.

It has been argued that the visual functions that are most likely to reorganize following early deafness are those that are attention demanding and would have benefited from convergence with the now missing auditory input, such as peripheral (non-foveal) processing[8]. This proposal seems consistent with our results. Both enhanced visual abilities of the deaf cats required the active attention of the animal. However, although it is necessary that the superior peripheral stimulus localization requires non-foveal vision, it is unclear whether the cats

| Task | PAF deactivation | DZ deactivation |
|---|---|---|
| Visual localization in the peripheral field | Deficit | No deficit |
| Movement detection | No deficit | Deficit |

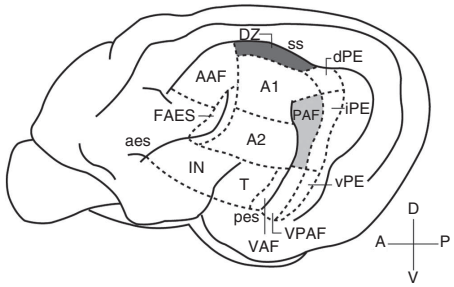


**Figure 7** Summary diagram illustrating the double-dissociation of visual functions in auditory cortex of the deaf cat. Bilateral deactivation of PAF, but not DZ, resulted in the loss of enhanced visual localization in the far periphery. On the other hand, bilateral deactivation of DZ, but not PAF, resulted in higher movement detection thresholds. The lower panel shows a lateral view of the cat cerebrum highlighting the locations of PAF and DZ.

were performing the motion detection task with non-foveal vision. Although minor head movement was observed in the apparatus, it is impossible to determine whether the superior movement detection abilities were accomplished with non-foveal vision without monitoring of both head and eye movements. However, as the stimulus itself (14°) was substantially larger than the fovea, it was presented to both the fovea and to the retinal periphery. It is likely that non-foveal vision was used in both tasks in which we found enhanced visual function.

### Specificity of compensatory functions in cats and humans

What we did not observe is also important. In cross-modal studies of the early blind, the primary area of visual cortex has been repeatedly identified as a major participant (for reviews, see refs. 30,31). Thus, it seems logical to expect a cross-modal involvement of A1 in early-deafened subjects. However, although examined in each of the current behavioral tasks, cooling deactivation of A1 had no effect on the examined visual functions in the deaf cats. This is consistent with numerous studies that have reported a lack of cross-modal effects in A1 (refs. 6,8–10,32,33), although some studies have suggested otherwise[11,34]. It might be argued that A1 may still have been cross-modally reorganized, either in a non-adaptive (for example, disorganized) manner, or may receive new inputs from modalities that were not tested (for example, somatosensory). However, the lack of visual reorganization of A1 is underscored by the fact that, in hearing animals, PAF function in localization behaviors[16] is highly dependent on A1 inputs[21], which is clearly not the case in the deaf (**Fig. 5**).

We did not find enhancements in visual acuity and discrimination of orientation, motion direction and velocity in the deaf subjects. Deactivation of any auditory cortical location had no effect on their performance in these tasks. A similar lack of adaptive or compensatory effects has been observed in human psychophysical studies of brightness discrimination[35], visual contrast sensitivity[36], visual shape identification[37] and visual motion sensitivity[38,39]. Collectively, these observations indicate that, although some features of vision are improved in deaf individuals, others are not. This is important because it indicates that cross-modal plasticity does not uniformly affect the entire cortical machinery for vision, a notion that is consistent with the location specificity of the reorganization itself.

### Compensatory functions and auditory cortical loci

The fact that compensatory effects occur for some perceptual tasks, but not others, may be a result of the lack of correlation between vision and audition in those particular tasks. For example, it is unlikely that spatial processing features underlying visual orientation discrimination exist in the auditory system. Similarly, color perception (which was not tested) would not be expected to be affected by cross-modal plasticity in deaf subjects.

On the other hand, stimulus location and stimulus movement are sensory features that both vision and audition have in common (they are of a 'supramodal nature'). It seems more than coincidental that area PAF, which is involved in auditory localization processes in the hearing[16,18], aids visual localization in the deaf. Similarly, area DZ, which is adjacent to the visual motion processing regions of the middle suprasylvian sulcus[40], underlies improvements in visual motion sensitivity in the deaf. Given these relationships, it seems possible that cortical modules subserving supramodal functions change their input modality in response to deafness while maintaining established output functions (as suggested by surgically engineered cortical rewiring experiments[41]). Conversely, features that are modality specific (such as color, tone, orientation, etc.) might have less potential for cross-modal reorganization.

In summary, using a spatially discrete technique of reversible neural deactivation, we found that superior visual perceptual abilities in the congenitally deaf are based on the cross-modal reorganization of specific regions of auditory cortex, demonstrating a causal relationship. These observations indicate, for the first time, to the best of our knowledge, that cross-modal effects do not occur uniformly across regions of deaf cortex, but principally occur in an adaptive fashion in those regions whose functions are also represented in the replacement modality. Similarly, cross-modal compensatory effects are specific and appear to enhance those functions that the deprived and replacement modalities hold in common. Ultimately, these considerations are important when evaluating the potential for compensatory forms of cross-modal plasticity resulting from any form of sensory loss.

## METHODS

Methods and any associated references are available in the online version of the paper at http://www.nature.com/natureneuroscience/.

*Note: Supplementary information is available on the Nature Neuroscience website.*


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### AUTHOR CONTRIBUTIONS

S.G.L. and A.K. conceived and designed the project. A.K. bred and provided the cats. All psychophysical work was performed or supervised by S.G.L. M.A.M. provided assistance with data analysis and interpretation. The manuscript was written and edited by all of the authors.

### COMPETING FINANCIAL INTERESTS

The authors declare no competing financial interests.


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## ONLINE METHODS

**Overview.** Mature (>1 year) congenitally deaf cats and age-matched hearing cats were trained on seven visual psychophysical tests. Deafness was confirmed by an absence of auditory brainstem responses (**Supplementary Fig. 1**). The cats' ability to detect and localize flashed visual stimuli was assessed in a visual orienting arena (**Supplementary Fig. 2**). The six discrimination tasks were conducted in a two-alternative forced-choice apparatus (**Supplementary Fig. 2**). A standard staircase procedure was used to determine psychophysical thresholds. Individual cryoloops were bilaterally implanted over A1, DZ, AAF and PAF (**Fig. 2b**). Each cat was re-tested on each task while all four loci, or each individual cortical locus, were bilaterally and unilaterally deactivated. Temperature monitoring electrodes were used to determine the extent of cooling deactivation for each of the cooling loops. At the conclusion of thermocline mapping, the cats were killed by perfusion with aldehyde fixatives. Brains were sectioned and processed for Nissl, myelin, SMI-32 and cytochrome oxidase. Deactivation reconstructions were compared with areal boundaries determined by SMI-32 to confirm location of the cooling loops.

**Subjects.** Three congenitally deaf cats were selected from a colony of white cats (University of Hannover) using a standard screening method[42] based on the absence of an acoustically evoked brainstem response to a condensation click (50-μs duration) up to an intensity of >125 dB SPL (**Supplementary Fig. 1**). In three adult hearing cats, similar screening methods were used to ensure normal acoustic detection thresholds (**Supplementary Fig. 1**). All animals were housed in an 'enriched' colony environment with water provided *ad libitum*. Caloric intake was restricted to the training/testing sessions and to 1 h at the conclusion of each day, when the cats had free access to dry cat food. We used a moist food (purée of beef liver and ground pork) as a reward. All procedures were conducted in accordance with the Canadian Council on Animal Care's *Guide to the Care and Use of Experimental Animals*, the US National Research Council's *Guidelines for the Care and Use of Mammals in Neuroscience and Behavioral Research* and the European Communities Council Directive (November 24, 1986; 86/609/EEC) and were approved by the University of Western Ontario Animal Use Subcommittee of the University Council on Animal Care.

Adult congenitally deaf cats have a Scheibe type of dysplasia in the organ of Corti with no hair cells being present, although the spiral ganglion and cochlear bony structure are preserved[42]. The central auditory system of the congenitally deaf cat shows expected deprivation-induced changes[42,43], although the central visual system appears normal in structure and function[44]. The visual pathways of the white cats are normal, provided they were not mated with Siamese cats[45].

**Apparatus.** For the visual localization task, training was conducted in an orienting arena[16–18], which allowed for presentation of visual or acoustic stimuli (**Supplementary Fig. 2**). Training was conducted in a dimly lit room. For the visual discrimination tasks, training was conducted in a two-alternative forced-choice apparatus[46] (**Supplementary Fig. 2**). The stimuli could be viewed through the response keys. Two monitors (17″ ViewSonic G70f graphics series CRT monitor, 1,024 × 768 pixels, 75-Hz refresh rate) capable of presenting precisely calibrated (Syder with OptiCAL, Pantone) visual stimuli were used for the presentation of two stimuli. A center divider prevented the cat from viewing both stimuli simultaneously. A Macintosh G4 computer running a modified version of Vista software (Cerebral Mechanics) controlled stimulus presentation, staircase protocols, data collection and reward dispensing.

**Training procedures.** The visual localization task was performed in a manner described previously[47]. A block consisted of 35 trials: two trials to each of the 12 peripheral positions, four trials to the central position and seven catch trials (no secondary stimulus). Five blocks of data were collected per session. Catch trials, where no target stimulus was presented, were randomly conducted. In a catch trial the cats were trained to approach the 0° position and receive the low incentive food. Training took ~3 months and was complete when a criterion performance level of ≥50% correct (average across all positions) was reached on three consecutive days.

The behavioral protocol was the same for all discrimination tasks. The cats viewed the stimuli through two plexiglass response panels and were rewarded for a nose-press response toward the correct stimulus. The cat viewed the stimulus for 1 s after stimulus onset, and then pressed a response key. Responses in the first 1 s were ignored to extinguish random responses immediately after stimulus onset and to enhance attention. The intertrial interval was 4 s. An incorrect response resulted in no food reward and an 8-s intertrial interval. To eliminate potential position habits, we used a correction procedure whereby three consecutive incorrect responses to the same side resulted in a trial being repeated until the cat made a correct response.

Thresholds were measured using a staircase procedure with three consecutive correct responses resulting in a decrease in the difference between the two stimuli and each incorrect response resulted in an increase in the difference. Discrimination thresholds were determined using the 79.4% correct staircase procedure[48]. The cats were trained twice a day for 5–7 d per week. A staircase procedure for determining thresholds was used because other methods, such as constant stimuli procedures, have been reported to be distressing to many cats[49]. In each training/testing session, the staircase was terminated at 200 trials. This point was set to prevent motivational issues, as the animals often became sated beyond 250 trials. Sample stimuli are provided (**Fig. 1c–h**).

Stimuli were generated using the CRS Toolbox for MATLAB (Mathworks). The video monitors were located 28 cm from the cats' eyes (thus, 1 cm on the screen corresponded to a visual angle of 2°). Stimuli were viewed through circular apertures (14° diameter). All stimuli presented in the discrimination apparatus, with the exception of the gray grating acuity stimulus, were high-contrast black-on-white stimuli. For the movement detection task, the cats had to discriminate a field of coherently moving dots from a field of stationary dots. Each stimulus had a dot density of 1.5 dots per deg$^2$ and the mean luminance of the display was 0.1 cd m$^{-2}$. Each dot was 0.03° in diameter and its luminance was set to 3.5 log units above detection threshold for human observers. Both fields had the same mean luminance. The positions of the dots in the stationary field were different on each trial. The cats were rewarded for choosing the field containing the moving dots. For the grating acuity task, the cats had to discriminate vertical square-wave gratings from a uniform gray stimulus of the same mean luminance (100 cd m$^{-2}$). The cats were rewarded for choosing the square-wave grating. For the Vernier acuity task, the cats were rewarded for selecting the two vertical lines that were offset from the two vertical lines with no offset. The lines were 12° × 0.2°. The cats were rewarded for choosing the offset line stimulus. For the orientation discrimination task, the cats had to discriminate between a vertically oriented line and a line with a clockwise deviation. The cats were rewarded for choosing the non–vertically oriented line. For the direction of motion task, the cats had to discriminate two fields of coherently moving dots at identical velocities (13.6 deg per s). One field was moving horizontally rightward and the other field was moving rightward at an elevation above horizontal. The moving dots were identical to those described for the detection of motion task. The cats were rewarded for choosing the field containing the non–horizontally moving dots. Finally, for the velocity discrimination task, the animals had to discriminate which of two fields of coherently rightward moving dots was moving faster. The moving dots were identical to those described for the detection of motion task. The cats were rewarded for choosing the faster moving stimulus. Speed thresholds were converted into Weber fractions. Six base (reference) speeds were examined: 2, 4, 8, 16, 32 and 64 deg per s.

**Surgical procedures.** Cooling loops were implanted after training was complete. Surgical procedures to bilaterally place cooling loops over PAF, DZ, A1 and AAF have been described previously[15–18]. Cryoloops were fabricated by shaping loops of 23-gauge stainless steel hypodermic tubing to conform to one of the four areas examined[15].

**Testing procedures and cooling deactivation.** Following cooling loop implantation and before any deactivations, baseline performance levels were re-established. For the visual localization task, we used a three-step testing procedure. First, we collected baseline data with all sites active. Second, testing began with the cooling of a cryoloop to 3 °C. Finally, after completion of all cooling, baseline levels were re-established. For the visual localization task, two blocks of 35 trials were conducted for each of the three conditions. Each testing session consisted of 210 trials. We conducted 25 testing sessions. Therefore, for each deactivation condition and for each cat, data presented is based on 100 trials at each of the 12 peripheral target positions. Deactivation data was collected while all four cortical loci were deactivated both unilaterally and bilaterally. Finally, deactivation data was collected while each locus was cooled unilaterally (left, right) and bilaterally.




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For the visual localization task, we calculated percent correct responses. Performance was assessed with a mixed ANOVA with one within hemisphere variable (warm versus cold; locus of cooling loop). Orienting responses were assessed with multi-factor mixed ANOVA variables (warm versus cold, azimuth, locus of cooling loop). The order of sessions was counter-balanced between areas (loops), functional states (active versus deactivated) and hemispheres.

For the discriminations, daily testing occurred 6–7 d per week. The deactivated locus was randomized for each testing session. Thus, the cat could not predict which, or if, a cortical locus was going to be deactivated. Thresholds for each task were determined at least 25 times for each deactivation condition. For each of the four cortical loci, thresholds were determined for left, bilateral, and right deactivation at least 25 times in each deactivation state. Thresholds were similarly determined during the simultaneous deactivation of all four cortical loci. Prior to the initiation of a daily testing session, a cryoloop, or cryoloops, was cooled to 3 °C. Testing then commenced and a session threshold was determined. Noncooling sessions were randomly introduced in numbers equal to that for each individual cortical locus. Statistical significance between active and deactivated performance was assessed using an analysis of variance and follow-up *t*-tests ($P < 0.01$). In total, testing all cats on all tasks and deactivation configurations took ~4.5 years.

**Thermocline mapping.** All of the cats were anesthetized (sodium pentobarbital, 25–30 mg per kg of body weight, intravenous), a craniotomy was performed and the cortical temperatures surrounding the cooling loops were measured using multiple microthermocouples (150 µm in diameter; Omega Engineering) to determine the region of deactivation[21]. Across the cortical surface, 300–400 thermal measurements were taken from positions 500 µm below the pial surface. From these measurements, thermal cortical maps from cooling each individual cryoloop were constructed by generating Voronoi tessellations (**Fig. 6**)[21]. The depth of the cooling deactivation was also measured at four different coronal levels to provide a cooling assessment in the *z* dimension. All temperature measurements were taken with each loop cooled to 3 ± 1 °C. After mapping, the craniotomies were closed.

**Tissue processing.** After thermocline mapping, anesthesia was deepened with sodium pentobarbital (40 mg per kg, intravenous) and the cats were killed by perfusion[18]. Brains were frozen and 50-µm coronal sections were cut and collected serially for the entire cerebrum. The first series of sections, at 250-µm intervals, was stained with cresyl violet. Series 2 was processed histochemically to demonstrate the presence of cytochrome oxidase. Series 3 was processed with monoclonal antibody SMI-32 (Sternberger Monoclonal) using established protocols[50]. Series 4 was processed for myelin. Selected sections from series 5, as needed, were processed using any of the previously described methods. All histochemically reacted sections were then mounted onto gelatinized glass slides, dehydrated and coverslipped.

**Cooling deactivation assessment.** Alignment of the deactivation loci with areas PAF, DZ, A1 and AAF was confirmed by comparing the thermocline mapping results with histology from the Nissl and SMI-32 processed tissue[18]. SMI-32 histochemistry localized areas PAF, DZ, A1 and AAF and confirmed that the deactivation loci included each area with minor spread into flanking cortices (**Supplementary Fig. 6**).


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