Running head: SURFACE-BASED MORPHOMETRIC ANALYSIS

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Effects of Deafness and Sign Language Experience on the Human Brain: Voxel-based and
Surface-based Morphometry

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Abstract

We investigated, using voxel-based morphometry (VBM), how deafness and sign language experience affect the anatomical structures of the human brain by comparing gray matter (GM) and white matter (WM) structures across congenitally deaf native signers, hearing native signers, and hearing sign-naïve controls ($n = 90$). We also compared the same groups on cortical thickness, surface area, and local gyrification using surface-based morphometry (SBM). Both VBM and SBM results revealed deafness-related changes in visual cortices and right frontal lobe. The GM in the auditory cortices did not appear to be affected by deafness; however, there was a significant WM reduction in left Heschl's gyrus for deaf signers only. The SBM comparisons revealed changes associated with lifelong signing experience: expansions in the surface area within left anterior temporal and left occipital lobes, and a reduction in cortical thickness in the right occipital lobe for deaf and hearing signers. Structural changes within these brain regions may be related to adaptations in the neural networks involved in processing signed language (i.e., visual perception of face and body movements). Hearing native signers also had unique neuroanatomical changes (e.g., reduced gyrification in premotor areas), perhaps due to lifelong experience with both a spoken and a signed language.

Keywords: deafness, sign language, voxel-based and surface-based brain morphometry

Effects of Deafness and Sign Language Experience on the Human Brain:

Voxel-based and Surface-based Morphometry

Neurobiological literature has shown that neuroanatomical changes and functional adaptations in the human brain can arise directly in response to sensory deprivation (e.g., compensatory plasticity) or differences in behavior driven by lifelong experience (e.g., expertise). Congenitally deaf individuals who use American Sign Language (ASL) offer cognitive neuroscience researchers a rare opportunity to study how cortical plasticity forms under two different conditions. However, to date, our understanding of how anatomical structures are directly affected by deafness or sign language experience is still limited, and the published results are inconsistent due to differences in study methods, small sample sizes, or differences in the etiologies and characteristics of the deaf population studied.

In the present study, we address the inconsistencies in the published studies by investigating how deafness and sign language experience affect the anatomical structures of the human brain, using two different morphometric analyses with a large number of congenitally deaf native signers, hearing native signers, and hearing control participants (N=90). We first analyzed whole-brain gray and white matter structures across groups using voxel-based morphometry (VBM) and then compared the same groups on the cortical thickness, surface area, and local gyrification using surface-based morphometry (SBM). To our knowledge, there is no study that investigated neuroanatomical changes in deaf brains using both approaches with a large number of deaf and hearing native signer participants. Inclusion of hearing native signers as another control group enables us to tease apart the effects of auditory deprivation from the effects of expertise with signed language. We also examined our data for any evidence of

structural brain changes associated specifically with a lifelong experience of using two languages with distinct sensory demands (i.e., visual and auditory), which is unique to hearing native signers.

Previous studies investigating behavioral performance on perceptual and cognitive tasks have reported that deaf participants responded significantly faster than hearing sign-naïve controls to visual stimuli presented peripherally (e.g., Codina et al. 2017) with higher accuracy in detecting the direction of motion in the right visual field (Neville and Lawson 1987). A large body of evidence now indicates that early deafness results in a redistribution of attentional resources across the visual periphery (Bavaliere et al., 2000; Chen et al., 2006; 2010; Dye, 2015; Dye et al., 2007; 2009; Lore & Song, 1991; Neville & Lawson, 1987; Parasnis & Samar, 1985; Proksch & Bavelier, 2002; Seymour et al., 2017; Sladen et al., 2005; Stevens & Neville, 2006).

Deaf signers' behavioral differences are not limited to the visual processing domain. Deaf signers also exhibit enhanced task performance in other cognitive domains: visual imagery or detecting mirror images (Emmorey et al. 1993), spatial cognition and spatial memory (Bellugi et al. 1990, Wilson et al. 1997, Emmorey et al. 1998), face processing (Bettger et al. 1997, McCullough and Emmorey 1997), and facial expression processing (McCullough et al. 2005; McCullough and Emmorey 2009).

However, some of these same studies also reported superior performance for hearing native signers when they were compared to hearing sign-naïve controls (Emmorey et al. 1993; McCullough and Emmorey 1997; Codina et al. 2017). Thus, one must be careful in interpreting behavioral and neurocognitive differences reported in the literature as the consequence of auditory deprivation when they did not include hearing native signers as "sign language" controls.

The task performance differences observed in deaf signers were often accompanied by significant differences in neural activity. Bavelier and colleagues (2001) reported enhanced MT-MST activity for peripheral attention in deaf signers. Finney and Dobkins (2001) reported increased neural activity in the right auditory cortex during visual motion processing in deaf signers. An MEG study by Finney and colleagues (2003) also found increased activity within the auditory cortex of deaf signers in response to visual stimuli. Similarly, recent fMRI studies revealed that deaf signers showed greater BOLD activity in Heschl's gyrus during peripheral visual processing (Scott et al. 2014; Cardin et al. 2016) or during visual working memory (Ding et al., 2015). These reports of significant neurobehavioral differences strongly suggest that there may also be significant underlying neuroanatomical changes associated with the deafness and/or sign language experience.

In the past two decades, there have been several studies that investigated the changes in the neuroanatomical structures as a result of deafness (Emmorey et al. 2003, Penhune et al. 2003, Shibata 2007, Allen et al. 2008, Kim et al. 2009, Leporé et al. 2010, Allen et al. 2013, Pénicaud et al. 2013, Lyness et al. 2014; Neuschwander, Hänggi, Zekveld, and Meyer, 2019). Studies by Penhune et al. (2003), Emmorey et al. (2003), and Shibata (2007) primarily focused on the auditory cortices and reported that the gray matter (GM) volumes of Heschl's gyrus and the planum temporale region in both hemispheres did not change significantly in congenitally deaf signers. Although Penhune and colleagues (2003) did not find any change in the white matter underneath the auditory cortices, Emmorey et al. (2003), Shibata (2007), Pénicaud et al. (2013), and Olulade et al. (2014) reported a reduction in the white matter (WM) volume within the superior temporal gyrus (STG) in deaf signers. Shiell and Zatorre (2016) investigated structural brain-behavior correlations and reported a negative correlation between the white matter

fractional anisotropy (FA) in the right planum temporale region and visual motion detection thresholds in deaf signers.

Other studies with deaf signers have also investigated the effects of deafness on brain regions other than the auditory cortices. Kim and colleagues (2009) reported decreased diffusion anisotropy in the internal capsule, superior longitudinal fasciculus, and inferior frontal white matter, in addition to reduced WM volume within primary auditory cortices in deaf signers. Li and colleagues (2012) conducted a VBM analysis and found focal reductions in white matter (WM) in the left middle frontal gyrus and right inferior occipital gyrus in deaf adolescent signers. Allen and colleagues (2008) reported increased grey matter in the left posterior insular lobule for deaf and hearing signers when compared with hearing sign-naïve controls. The converging evidence from these findings indicates that deafness does have some effects on the neuroanatomical structures other than the auditory cortices. Allen and colleagues' (2008) findings suggest that sign language experience may also have some effects.

Although the above-mentioned findings found some evidence of morphometric differences arising from auditory deprivation and lifelong sign language exposure, these studies employed different analysis methods--e.g., tensor-based morphometry (TBM), VBM, or volumetric measures of brain regions using manually drawn regions of interest (ROIs) by expert anatomists. Moreover, some of these analyses were limited to a few specific brain regions or a small group of participants. Crucially, the majority of studies did not include hearing native signers as a control for sign language effects, and some studies also did not explicitly state that they had controlled for other variables that could affect the neuroanatomical measurements such as etiologies of deafness, sign language exposure among deaf participants, age, sex, and intracranial volume (ICV).

This study investigates the effects of deafness and sign language experience using two different morphometric analyses (VBM and SBM) with a large group of participants, while controlling for age, sign language exposure, gender, and ICV. Our study disentangles the neuroanatomical effects of deafness and sign language experience by including hearing native signers in addition to congenitally deaf native signers and hearing sign-naïve controls ($N = 30$ in each group). We also examined the results from two different morphometric measurement methods to understand the complex interplay of white matter structure, cortical thickness, surface area, and cortical folding in specific brain regions. Finally, to our knowledge, our study is the first to examine the effects of congenital deafness and lifelong sign language experience on cortical folding in the auditory and visual cortices (or in any other brain regions).

By combining the VBM and SBM anatomical measurements and examining how these anatomical changes are related may help reveal the possible reasons for the variation in the results reported in previous studies (e.g., reduction or no reduction in gray and white matter volumes within the auditory cortices of deaf signers).

Methods

Participants

Thirty congenitally deaf signers (18 female; mean age = 27.4; $SD = 5.17$ years), 30 hearing signers (16 female; mean age = 26.2; $SD = 4.8$ years), and 30 hearing sign-naïve controls (16 female; mean age 25.3; $SD = 3.9$ years) participated in the study. All deaf participants were profoundly deaf (average dB loss > 96 in the better ear) and were exposed to ASL from birth by their signing parents (23 had two deaf parents). The hearing signers were also born into deaf signing families (25 had two deaf parents), and all were exposed to ASL from infancy. All

hearing signers reported using ASL daily (at least 40 percent of the time) with their families or colleagues in the workplace. All hearing sign-naïve control participants had no or very minimal experience with ASL (e.g., limited to knowledge of the fingerspelled alphabet or a few signs). All hearing participants (signers and sign-naïve controls) reported normal hearing. All participants in the study were right-handed and had normal or corrected-to-normal vision. None of the participants reported any current or past neurological or behavioral disorders (e.g., epilepsy or a learning disability). All participants had 12 years or more of formal education. The mean number of years of education was 16.7 ($SD = 2.07$), 15.6 ($SD = 2.39$), and 15.8 ($SD = 2.19$) for the deaf signers, hearing signers, and hearing sign-naïve controls, respectively. There was no significant group difference in the years of education ($F(2, 87) = 1.91, p = .15$). All participants were recruited from the metropolitan areas of San Francisco, San Diego, Los Angeles, and Washington, DC. Informed consent was obtained from all participants according to procedures approved by the UCSD and SDSU Human Research Protection Programs.

Procedure

MR image acquisition. All MRI scans were acquired using a 3-Tesla GE Signa Excite scanner equipped with an eight-element phased-array head coil at the Center for fMRI at the University of California, San Diego. For each participant, two high-resolution MR images were acquired using T1-weighted Fast Spoiled Gradient-Recalled Echo (FSPGR) with the following parameters: FOV 256 mm, 1 mm x 1 mm in-plane, 172 1-mm-thick axial slices, flip angle = 8°, inversion time 600 ms. All structural MRI scans were visually inspected by one of the authors

(SM) for any significant brain abnormalities or head movements (e.g., blurring, ghosting, or stripping).

Voxel-based analysis procedure. For the VBM analyses, the pre-processing workflow, using SPM 12 (Ashburner and Friston 2000, 2001, 2011), starts with the tissue segmentation of the brain into gray matter, white matter, and cerebrospinal fluid. The segmented images were then registered to (MNI152) atlas via DARTEL registration (Ashburner 2007) to ensure voxel-wise correspondence across different brains. All registered images were then spatially smoothed using an 8 mm Gaussian kernel before being used as inputs for second-level voxel-wise statistical analyses. For voxel-based GM and WM group-level statistical analyses, we conducted general linear model (GLM) analyses using SPM 12. The age, sex, and estimated intracranial volume (see below) covariates were included in the analyses as nuisance variables. The contrasts were conducted with the statistical maps thresholded at $p < 0.05$ (corrected for familywise error rate) and $p < .001$ (uncorrected) prior to minimum statistic, conjunction null analyses (Nichols et al. 2005).

Surface-based analysis procedure. Two sets of whole-brain T1-weighted MRI structural scans from each participant were imported and converted into MGH format, a proprietary file format defined by the NMR Center at Massachusetts General Hospital (MGH). The converted structural scans from each participant were processed with FreeSurfer (version 5.3.0), a software package for analysis and visualization, provided by Martinos Center for Biomedical Imaging (<http://surfer.nmr.mgh.harvard.edu>) for cortical surface reconstruction and analysis. All cortical surface reconstructions were processed on an Apple Mac Pro (2009 Nehalem model, two 2.66 GHz Quad-Core Intel 5500 Xeon series processors, 16 GB memory, 8 MB L3 per processor, ATI Radeon HD 4870 with 512 MB GDDR5, Mac OS version 10.6.8).

FreeSurfer's automated surface reconstruction methods and algorithms have been described in more depth elsewhere (Dale et al. 1999; Fischl et al., 1999a; Fischl et al., 1999b; Fischl and Dale 2000, Fischl et al. 2001, Fischl et al. 2002, Fischl 2004). We implemented FreeSurfer's standard processing pipeline identically on all converted MRI structural volumes. The processing steps in the FreeSurfer pipeline were: automated rigid body registering and averaging of two converted MRI scans, affine transformation of averaged MRI volume to the AC-PC line, non-uniform image intensity correction, skull-stripping and brain extraction, second intensity normalization of extracted brain volume, linear registration of non-uniform-intensity-corrected MRI volume to Gaussian Classifier Atlas (GCA), subcortical structure labeling based on GCA probabilistic model, white matter (WM) segmentation of subcortical volumetric structures, tessellation of the boundary between gray and white matter (white surface), and anatomical parcellation.

We visually inspected all data outputs from each stage of the FreeSurfer processing stream for accuracy. All minor processing flaws (e.g., imperfect segmentation of the boundary between gray and white matter) were minimally edited by one of the authors (SM) according to the standard, objective editing rules as described in the MGH's FreeSurfer manual.

In addition to the gray and white matter segmentation and the cortical surface reconstruction, FreeSurfer calculates an estimated total intracranial volume (eTIV) for each participant by first generating an atlas scaling factor (ASF), a metric computed as the reciprocal of the atlas transform determinant required to register each brain (e.g., by expansion or contraction) to the target atlas (MNI 305 template). The eTIV value for each brain is computed by dividing the atlas mask volume with each participant's ASF metric (Bucker et al. 2004). The eTIV values were then used as one of the nuisance variables in all our analyses.

After the cortical surface reconstruction of white matter and pial surfaces for each brain were completed and verified to be free of surface reconstruction imperfections, the measurements for cortical thickness, cortical surface area, and local gyrification index (*lGI*) were computed vertex-wise for each hemisphere of each brain using the *k*th indices of tessellated surface data that were created during the cortical surface reconstruction. The values generated from the cortical surface calculations were registered to the *k*th vertices of each brain's white matter surface geometry. Unlike the pial surface, the white matter surface is minimally affected by changes in gray matter (e.g., cortical thickness), and thus gives more precise local measurements of the cortical folding. The computations for cortical thickness and surface area were processed entirely in the FreeSurfer software suite. The calculations for *lGI* were performed using the Mac R 2014b version of MATLAB (Mathworks). Below are three brief descriptions of the surface-based calculation methods we used for each type of cortical measure.

Cortical thickness. The cortical thickness for each *k*th vertex was measured using an iterative closest point algorithm in FreeSurfer. It computes the shortest distance from a *k*th vertex on the white matter surface to the pial surface and repeats the same process from the same *k*th vertex on the pial surface back to the white matter surface. These two shortest Euclidean distances were then averaged into a cortical thickness measurement for that specific *k*th vertex (Fischl and Dale 2000).

Cortical surface area. At each *k*th vertex in the native space, the cortical surface area value was computed as an average of the areas of the tessellated triangles connected to that vertex. When each cortical surface in the native space was registered and transformed (either stretched or compressed between *k*th vertices) into the standard atlas space, the new values

from transformed tessellated triangles provide quantified measurements of expansion or contraction for each k th vertex on the cortical surface (Fischl et al., 1999).

Local gyrification index (lGI). Schaer and colleagues (2008, 2012) developed a surface-based quantification index for local gyrification, which measures the amount of local cortical folding by computing the ratio of the cortex surface buried within the sulcal folds to the amount of visible cortical surface within a specific radius of circular regions of interest. The higher local gyrification index indicates a more complex cortical folding. For our *lGI* analyses, we set the radius of circular regions of interest to 25 mm as recommended by Schaer et al. (2012). See Schaer et al. (2008) for more details on the algorithms used to calculate the ratio of the buried cortex relative to the outer cortical surface. The MATLAB scripts for *lGI* computation are available at <http://surfer.nmr.mgh.harvard.edu>.

In addition to the cortical surface reconstruction, we registered all surface measurements to a standard atlas space for whole-brain group-level analyses. Cortical surface analyses were conducted using a GLM analysis tool in FreeSurfer (`mri_glmfit`, v5.3.0). We calculated whole-brain vertex-wise group differences in cortical thickness, surface area, and *lGI* for both hemispheres after surface smoothing of each brain surface map with 5 mm full-width and half-maximum (FWHM) kernel. The age, sex, and eTIV covariates were included in the analyses as nuisance variables. Also, to correct for familywise error rate (FWER) across the cortical surface, we implemented Monte Carlo analyses with 10,000 simulations at a cluster-inclusion threshold of $\alpha = 0.05$, and only report the clusters that have survived an FWE-corrected threshold of $p \leq 0.05$ (Hagler et al., 2006).

Results

Voxel-based Analyses

We conducted a GLM analysis on the estimated total intracranial volume (eTIV) across the groups with age and sex as variables of no interest. Although there was no significant difference in the eTIV means across the groups ($F(3,86) = .225, p = .79$), the eTIV was nevertheless included as one of the variables of no interest to increase the power of voxel-based analyses.

Whole-brain voxel-based group contrasts (corrected for familywise error rate) revealed no significant difference in the GM and WM across the groups. However, to compare our results with the previously published studies, we also conducted whole-brain minimum statistic, conjunction null (CS/CN) analyses of contrasts (deaf vs. hearing native signers and deaf vs. hearing controls) at a lower statistical threshold ($p < .001$) and minimum cluster-extent threshold of 10 mm^3 . The results at the lower statistical threshold revealed increased GM in the occipital lobes (left peak MNI coordinates: $x = -22, y = -82, z = +9, t = 3.89$; right peak MNI coordinates: $x = +26, y = -80, z = +2, t = 3.43$; see Figure 1A). The conjunction analysis with WM at the same statistical threshold also revealed clusters of reduction in the WM underneath left Heschl's gyrus (peak MNI coordinates: $x = -36, y = -32, z = +4, t = 3.39$; Figure 1B) and the posterior right middle frontal gyrus (peak MNI coordinates: $x = +28, y = -2, z = +42, t = 3.39$, volume = 100 mm^3 ; not shown in Figure 1) for deaf signers when compared to both hearing groups. Conjunction analyses of signers (deaf signers vs. hearing controls and hearing signers vs. hearing controls) did not reveal any significant regional differences in GM and WM.

A. Clusters of Increased gray matter for deaf signers (minimum statistic, conjunction null) **B. A cluster of reduced white matter for deaf signers (minimum statistic, conjunction null)**

Figure 1. Minimum statistic conjunction null (MSCN) images of voxel-based group comparisons of gray and white matter. First row (A) shows axial, sagittal, coronal, and 3d brain images of the conjunction null of deaf signers vs. hearing controls and deaf signers vs. hearing signers (each contrast thresholded at $p = .001$). White clusters in the images are the regions of increased gray matter (GM) in the occipital lobes for deaf signers. A black cluster in the second row images (B) shows decreased white matter in the left Heschl's gyrus for the deaf signers. All clusters are superimposed on the MNI-152 1mm brain template.

Surface-based Analyses

Cortical thickness. Cortical thickness results are shown in Table 1 and Figure 2. A direct contrast between deaf signers and hearing sign-naïve controls revealed reduced cortical thickness in the left anterior insula and right lingual gyrus (BA 17,18) for the deaf signers (Figure 2A). There were no brain regions where the deaf signers showed increased cortical thickness relative to the hearing sign-naïve controls. A direct contrast between hearing signers and hearing sign-naïve controls revealed reduced cortical thickness in the right precentral gyrus (BA 6) and right lingual gyrus (BA 17,18) for the hearing signers (Figure 2B). There were no brain regions where the hearing signers showed significantly increased cortical thickness relative to the hearing sign-naïve controls. Finally, a contrast between deaf and hearing signers revealed significantly increased cortical thickness in the left precentral gyrus (BA 4,6), the left postcentral cortex (BA 3,1,2), the left superior temporal gyrus (BA 22), and the right pars opercularis (BA 44) for the deaf signers. Right isthmus cingulate (BA 23) was the only brain region where the deaf signers showed significantly reduced cortical thickness compared to the hearing signers (Figure 2C).

Table 1. Brodmann areas (BA), MNI coordinates at the peak *t-values*, cluster sizes, and peak *t-values* for group contrasts in the cortical thickness ($p < .05$, corrected)

Region	BA	Side	X	Y	Z	Vol mm ²	<i>t</i> peak
Deaf > Hearing							
None							
Hearing > Deaf							
Insula		L	-34	-14	-3	112	3.77
Lingual Gyrus	17, 18	R	+4	-80	-2	217	4.65
Hearing > Hearing Signers							
Precentral	6	R	+57	+5	+15	215	4.66
Lingual Gyrus	17, 18	R	+4	-81	-2	189	4.88
Hearing Signers > Hearing							
None							
Deaf > Hearing Signers							
Precentral	4, 6	L	-57	+1	+12	152	3.78
Postcentral	3,1,2	L	-47	-10	+24	220	3.76
Pars opercularis	44	R	+39	+10	+11	176	4.62
Superior temporal gyrus	22	L	-52	-11	-1	150	3.62
Hearing Signers > Deaf							
Isthmus cingulate	23	R	+9	-32	+30	150	3.43

A. Deaf vs. hearing controls: Regions of reduced (black) cortical thickness for deaf signers

B. Hearing signers vs. hearing controls: Regions of reduced (black) cortical thickness for hearing signers

C. Deaf vs. hearing signers. Regions of increased (white) and reduced (black) cortical thickness for deaf signers

Figure 2. Surface-based comparisons of cortical thickness (CT) at FWE-corrected $P < .05$. A) deaf vs. hearing: deaf signers show reduced cortical thickness in the left insula and right lingual gyrus. B) hearing signers vs. hearing controls): hearing signers show reduced CT in the right precentral gyrus and right lingual gyrus. C) deaf signers vs. hearing signers: deaf signers show increased CT in the left precentral gyrus, left postcentral gyrus, and anterior superior temporal gyrus, and the hearing signers show increased CT in the right isthmus cingulate. All cortical surfaces with no significant differences are greyed out.

Surface area. The results of surface area contrasts are shown in Table 2 and Figure 3. A direct contrast between deaf signers and the hearing sign-naïve controls revealed larger surface area in several brain regions for the deaf signers: left anterior middle temporal cortex (BA 21), left pericalcarine cortex (BA 17), left lateral occipital cortex (BA 18), and right precuneus (BA 18) (Figure 2A). The same contrast also revealed a smaller surface area in the right middle frontal gyrus (BA 9) for the deaf signers relative to hearing sign-naïve controls. A direct comparison between hearing signers and hearing sign-naïve controls (Figure 3B) showed a smaller surface area for the hearing signers in the right posterior superior temporal gyrus (BA 22) and larger surface areas for the hearing signers in the following brain regions: right precentral cortex (BA 4, 6), left and right anterior middle temporal gyri (BA 21), left cuneus (BA 18), right inferior parietal cortex (BA 40) and right middle temporal gyrus (BA 37). Finally, a direct contrast between deaf and hearing signers revealed a larger surface area in the right posterior superior temporal gyrus (BA 22) for the deaf signers (Figure 3C). The right precentral gyrus (BA 6) and right inferior parietal cortex (BA 39) were two brain regions where the deaf signers showed smaller surface area relative to the hearing signers.

Table 2. Brodmann areas (BA), MNI coordinates at the peak *t-values*, cluster sizes, and peak *t-values* for group contrasts in the surface area measurement ($p < .05$, corrected)

Region	BA	Side	X	Y	Z	Vol mm ²	<i>t</i> peak
Deaf > Hearing							
Ant. middle temporal	21	L	-58	-11	-25	529	3.33
Pericalcarine	17	L	-12	-89	+6	358	2.80
Lateral occipital	18	L	-20	-89	-9	323	2.19
Precuneus	18	R	+16	-95	+12	336	2.54
Hearing > Deaf							
Middle frontal gyrus	9	R	+36	+44	+23	276.8	-3.56
Hearing > Hearing Signers							
Bank STS	21,22	R	+54	-39	+11	223	3.75
Hearing Signers > Hearing							
Precentral	4,6	R	+56	+1	+9	280	3.35
Ant. middle temporal	21	L	-58	-11	-25	344	3.96
Ant. middle temporal	21	R	+57	-2	-30	285	3.24
Cuneus	18	L	-8	-68	+11	158	3.06
Inferior parietal	39	R	+53	-52	+9	176	3.44
Supramarginal	40	R	+53	-22	+33	169	2.88
Deaf > Hearing Signers							
Supramarginal	40	R	+58	-43	+18	172	3.65
Hearing Signers > Deaf							
Precentral	6	R	+51	+5	+26	177	3.73
Inferior parietal	39	R	+53	-51	+8	212	3.42

A. Deaf vs. hearing controls: Regions of larger (white) and smaller (black) surface area for deaf signers

B. Hearing signers vs. hearing controls: Regions of larger (white) and smaller (black) surface area for hearing signers

C. Deaf vs. hearing signers: Regions of larger (white) and smaller (black) surface area for deaf signers

Figure 3. Surface-based comparisons of surface area (SA) at FWE-corrected $P < .05$: A) deaf vs. hearing controls: deaf signers show larger SA (white) in the left and right occipital lobes, left anterior temporal lobe (ATL), and smaller SA (black) in the right middle frontal gyrus. B) hearing signers vs. hearing controls: hearing signers show larger SA (white) the left and right ATL, left occipital lobe, right middle temporal gyrus, right inferior parietal, and precentral gyrus. Hearing signers also showed smaller SA (black) in the upper bank of the right superior temporal sulcus (STS). C) Deaf vs. hearing signers: deaf signers show larger SA (white) in the upper bank of the right STS, and smaller SA (black) in the right middle temporal gyrus and right precentral gyrus. All cortical surfaces with no significant differences are greyed out.

Local gyrification index (LGI). The results for *LGI* contrasts are shown in Table 3 and Figure 4. A direct contrast between deaf signers and hearing sign-naïve controls revealed increased cortical gyrification in the left occipital and lingual gyri (BA 17, 18) for the deaf signers. The same contrast revealed a reduced cortical gyrification for deaf signers in the right inferior frontal gyrus (BA 44, 45) (Figure 4A). A direct contrast between hearing signers and hearing sign-naïve controls revealed reduced gyrification for the hearing signers in two brain regions: left superior frontal cortex (BA 6) and right inferior temporal cortex (BA 37) (Figure 4B). There were no brain regions where the hearing signers showed significantly greater cortical gyrification than the hearing sign-naïve controls. Finally, a direct contrast between deaf signers and hearing signers revealed increased gyrification for the deaf signers in four brain regions: left superior frontal cortex (BA 6), left middle frontal cortex (BA 6), right superior frontal cortex (BA 6), and the left inferior temporal gyrus (BA 20, 37). The same contrast showed a reduced cortical gyrification in the right inferior frontal gyrus (BA 44,45) (Figure 4C) for the deaf signers.

Table 3. Brodmann areas (BA), MNI coordinates at the peak *t-values*, cluster sizes, and peak *t-values* for group contrasts in *lGI* measurement ($p < .05$, corrected)

Region	BA	Side	X	Y	Z	Vol (mm ²)	<i>t</i> peak
Deaf > Hearing							
Occipital /Lingual gyrus	17,18	L	-12	-84	-13	923	2.34
Hearing > Deaf							
Inferior frontal gyrus	44, 45	R	+44	+28	+14	1071	2.66
Hearing > Hearing Signers							
Superior frontal gyrus	6	L	-37	+10	+55	1996	4.67
Inferior temporal	37	R	+46	-63	-9	920	2.23
Hearing Signers > Hearing							
None							
Deaf > Hearing Signers							
Superior frontal	6	L	-16	+20	+57	2002	3.68
Caudal middle frontal	6	L	-31	+3	+53	1302	2.9
Superior frontal	6	R	+22	+13	+49	986	3.52
Inferior temporal	20,37	L	-48	-18	-33	1981	3.94
Hearing Signers > Deaf							
Inferior frontal gyrus	44,45	R	+47	+14	+21	1050	3.23

A. Deaf vs. hearing controls: Regions of increased (white) and reduced (black) gyrification for deaf signers

B. Hearing signers vs. hearing controls. Regions of reduced (black) gyrification for hearing signers

C. Deaf vs. hearing signers: Regions of increased (white) and reduced (black) gyrification for deaf signers

Figure 4. Surface-based comparisons of local gyrification index (*l*GI) at FWE-corrected $P < .05$. A) deaf vs. hearing controls: deaf signers show increased *l*GI (white) in the left occipital lobe and reduced *l*GI (black) in the right inferior frontal gyrus (IFG). B) hearing signers vs. hearing controls: hearing signers show reduced *l*GI in the left SMA and right inferior temporal lobes. C) deaf vs. hearing signers: deaf signers show increased *l*GI in the left and right supplementary motor areas, and the left inferior temporal lobe and reduced *l*GI in the right IFG. All cortical surfaces with no significant differences are greyed out.

Discussion

In the present study, we examined voxel-based and surface-based measurements across large groups of deaf native signers, hearing native signers, and hearing sign-naïve controls to determine whether there were specific neuroanatomical changes in the human brain that occur as a result of deafness and/or sign language experience. We identified significant neuroanatomical changes as deafness-related effects when they appeared with the same pattern (e.g., increased GM or increased surface area) in the same brain regions only for the deaf signers in all comparisons. Likewise, the same patterns of change observed *only* for both the deaf and hearing signers, when contrasted with the hearing sign-naïve controls, are identified as sign language-related effects. Finally, any structural changes observed *only* for the hearing native signers when compared to the deaf signers or hearing sign-naïve controls are identified as effects associated with hearing signers' unique auditory and visual linguistic experience.

Effects of deafness

Our whole-brain VBM and SBM analyses revealed three specific changes in visual and auditory cortices and a region in the right middle frontal cortex for deaf signers only. The VBM contrasts, corrected for familywise error rate, did not reveal any significant local structural changes in the GM or WM for the deaf group. However, for comparisons with the previous studies, the same contrasts using a less stringent combination of voxel statistical threshold ($p = .001$) and minimum cluster-extent of 10 mm^3 revealed increased GM within the left and right occipital lobes for deaf signers only (Figure 1A). This finding is consistent with the Allen et al. (2013) study, which reported deaf signers to have larger calcarine sulcus cortex volume in both

hemispheres relative to hearing sign-naïve controls. The observed increase in the GM of occipital cortex may be related to the enhanced visual processing observed for deaf signers widely reported in the literature—e.g., enhanced peripheral visual attention (Bavelier et al. 2000, Proksch & Bavelier 2002, Dye et al. 2009) and the enhanced processing of visual motion in the inferior visual field (Bosworth & Dobkins 2002).

Our VBM analysis did not find any differences in the GM in deaf signers' auditory cortices but did reveal a cluster of white matter reduction underneath the left Heschl's gyrus for the deaf signers when compared to both hearing groups (see Figure 1B). Both of our GM and WM results are consistent with the previous findings from the studies investigating neuroanatomical changes within the auditory cortices of deaf brains (Emmorey et al. 2003; Shibata 2007; Olulade et al. 2014). Our successful replication of the earlier studies provides robust evidence that the WM reduction in left Heschl's gyrus is a direct result of deafness alone. We did not find any difference in the WM underlying right Heschl's gyrus for deaf signers. Since Shibata (2007), and Olulade et al. (2014) also did not find any WM difference underneath right Heschl's gyrus, we suggest that WM in the left and right primary auditory cortices may differ in their structural malleability or connections. We did not find any group differences in the surface-based analyses (i.e. cortical thickness, surface area, and gyrification) of auditory cortices, which, again, is consistent with previous studies reporting no difference between deaf signers and hearing sign-naïve controls in the GM structure within auditory cortices (Penhune et al. 2003, Emmorey et al. 2003, Shibata 2007, Shiell et al. 2016).

The preservation of GM in auditory cortices, despite deaf signers' lifelong auditory deprivation, suggests that these regions are likely re-utilized for other cognitive functions through cross-modal plasticity. Previous neuroimaging studies have reported significant neural

activity in the planum temporale regions in deaf signers during visual motion and language processing (Petitto et al. 2000, MacSweeney et al. 2002, Fine et al. 2005). Shiell and colleagues (2016) have reported a positive correlation between the cortical thickness of the right planum temporale (PT) and motion detection threshold in deaf signers. Cardin and colleagues (2016) reported that deaf signers and non-signers did not differ in neural activity within auditory cortices when viewing sign language stimuli; however, both deaf groups showed greater neural activity than hearing sign-naïve controls. Thus, the auditory cortices in deaf individuals appear to be reutilized for processing motion information in the visual domain.

A future study with histological analysis or neuroimaging using layer-dependent fMRI (e.g., Huber et al. 2017) may reveal why GM appears to be intact within the primary auditory cortices in deaf signers. For example, the whole measurement of cortical thickness can remain the same despite significant changes, perhaps due to cross-modal plasticity, in the afferent and efferent connections in the cortical layers within the auditory cortices.

Our analyses also revealed neuroanatomical changes in other brain regions. Our whole-brain VBM analysis of white matter showed a cluster of reduction in the right posterior middle frontal gyrus only for deaf signers, and it was within the area where the frontal eye fields, hand premotor area, and SMA are connected via U-fibres (Catani et al. 2012). Budisavljevic and colleagues (2016) investigated the relationship between human movement kinematics and the short frontal lobe networks and found a strong correlation between the white matter microstructures within U-fibres and smoothness of motor performance in the planning, execution, and feedback control of hand actions. It is plausible that extensive lifelong experience with rapid and precise hand coordination during signing may lead to significant neuroanatomical changes in the WM underneath the right posterior middle frontal gyrus for deaf signers.

However, since we did not collect any diffusion tensor MR images to measure whole-brain fractional anisotropy of white matter, we are limited in our ability to infer what the reduction in white matter signifies in this brain region.

Our whole-brain surface-based analyses (cortical thickness, surface area, and local gyrification) revealed some structural changes in the cerebral cortex that did not show up in the whole-brain VBM analyses. The cortical folding in the right IFG was significantly less convoluted for deaf signers when compared with hearing signers or sign-naïve controls (see Figure 4A and 4C). Since there were no significant group differences in cortical thickness or surface area in this brain region, less gyrification in this region for deaf signers is therefore likely due to changes in the underlying white matter. Specifically, reduced gyrification may be a result of changes associated with the frontal aslant tract (FAT) that connects the right pars opercularis to the border area that lies between pre-SMA and SMA-proper. It has been suggested that the right SMA mediates, through the FAT, the voluntary control of face, tongue, and pharynx movements in the right frontal operculum (Forkel et al., 2014, Fontaine et al. 2002). Thus, the less gyrification in this region may indicate different local cortico-cortico connections between the SMA and the frontal operculum for deaf signers. Given that the deaf signers in our study primarily used signed language rather than speech for their everyday communication, limited experience with speech production may be the reason for less gyrification within the right IFG. Moreover, we found a cluster of reduced cortical thickness in the anterior part of the left insula for deaf signers when compared to hearing sign-naïve controls (Figure 2A). The anterior part of the insula is part of the neural circuitry coordinating vocal tract movements for speech production (Dronkers 1996, Ackermann and Riecker 2010). Reduced cortical thickness observed in this region for the deaf signers, again, may be due to the less frequent use of speech. The lack

of difference between deaf signers and hearing signers in this area also suggests that hearing signers' experience with speech may not be identical to hearing sign-naïve controls, and the hearing signers' cortical thickness measurements may lie in between two other groups.

We also examined the surface-based results to determine if they replicate previously reported anatomical differences outside auditory regions for deaf signers compared to hearing sign-naïve speakers. Previous studies using tensor-based morphometry and volume-based ROI comparisons have reported local white matter volume increases for deaf signers in frontal regions (Leporé et al., 2010), in the right insula (Allen et al., 2008), and increase in cortical volume in the pars triangularis bilaterally (Allen et al. 2013). Our surface-based analyses, however, did not show any differences in cortical thickness (Figure 2A) or surface area (Figure 3A) within the pars triangularis bilaterally for deaf signers, suggesting that the significant differences observed in the previous studies, again, may be due to changes in white matter structure within frontal regions.

In summary, our VBM and SBM findings, along with the previous literature, show that the WM reduction in the left Heschl's gyrus appears to be directly affected by auditory deprivation. The differences in local gyrification within the right IFG, cortical thickness in the left anterior insula, and increased GM in the occipital lobes may arise as a consequence of differences in visual attention, use of speech, and other factors that stem from the life experience of deaf individuals.

Effects of sign language experience

The whole-brain VBM analysis did not reveal any changes in GM and WM that could be attributed uniquely to sign language experience. The surface-based comparisons across the three

groups, however, showed specific neuroanatomical changes related to sign language experience. Both groups of signers showed a reduction in cortical thickness in the right occipital lobe (Figure 2A and 2B). For deaf signers, the reduction in cortical thickness in the right occipital lobe may appear contradictory since the VBM analysis revealed that deaf signers had more GM in that region. However, the increase in the GM for deaf signers may be due to the expansion of cortical surface in the same area, resulting in an increased GM despite the reduction in cortical thickness.

The cortical thickness changes in the occipital lobes observed for both groups of signers point to the possibility of experience-driven plasticity resulting from the high demands of visual processing for signed languages. The hemispheric asymmetry of cortical thickness in the occipital lobes might be due to already inherent asymmetry in the visual perception of signed languages. Most signers are right-handed and thus the bulk of dominant hand movements in ASL, including fingerspellings, appear in observers' left lower visual field because signers normally fixate on the face during sign language conversations (Emmorey, Thompson, & Colvin, 2008; Dye, Seymour, & Hauser, 2015). Therefore the observers' right hemisphere receives and processes more motion information from the dominant hand of another right-handed signer. The inverse relationship between cortical thickness and expertise may be counterintuitive, but it is not unusual. Two previous studies comparing the occipital cortices of congenitally blind individuals and sighted individuals reported reduced cortical thickness for sighted individuals (Jiang et al. 2009, Anurova et al. 2015). Bi, Chen, Zhou, He & Fang (2014) reported an inverse relationship between the cortical thickness in the left face fusiform area (FFA) and the length of perceptual learning of faces. The cortical thickness may be thinner in signers' right hemisphere as a result of the refinement of visual motion processing mechanisms.

Surface areas in the left anterior temporal and left occipital lobe were also found to be larger in deaf (Figure 3A) and hearing signers (Figure 3B) when compared to hearing sign-naïve controls. The expanded cortical surface area observed in these brain regions, again, may be due to the high demands of signed language processing. Previous behavioral and neuroimaging studies have shown that deaf and hearing signers process faces and facial expressions differently from hearing sign-naïve controls (McCullough and Emmorey, 1997; McCullough et al. 2005, Emmorey and McCullough 2009; Letourneau and Mitchell, 2013). Both groups of signers performed better than hearing sign-naïve controls in detecting the local changes of facial features (McCullough and Emmorey, 1997). Moreover, both groups of signers differed from hearing sign-naïve controls in the laterality of neural activity within the superior temporal sulcus and fusiform gyrus when perceiving linguistic facial expressions (McCullough et al., 2005; Emmorey and McCullough, 2009). Letourneau and Mitchell (2013) reported that deaf signers showed typical left visual field (LVF) bias for face identity judgment; however, their visual field bias shifted toward RVF when making judgments about affective facial expressions. Hearing non-signers, on the other hand, consistently showed LVF bias for both types of judgment tasks, which strongly suggests that both groups of signers activate left hemisphere regions more extensively than sign-naïve controls for facial expression perception.

Moreover, it is plausible that the constant, strong linguistic top-down processing involved in the attention to rapid and precise changes in facial expressions and hand movements during sign language conversations may drive the surface expansion to the early visual processing areas in the left hemisphere for signers to improve tuning of low-level visual features. Meng and colleagues (2012) reported that the neural activity in the left FFA is more sensitive to low-level image face semblance (featural-like processing) and can be modulated more easily by contextual

information. The upper layers in primary visual cortex (V1) are also known to be modulated by top-down connections from other extrastriate visual cortices and face-sensitive areas and are involved in higher cognitive functions (Muckli, 2010; Muckli et al., 2013; Petro et al., 2013; Muckli et al., 2015). The structural changes in the early visual processing area may also have downstream cascading effects on the anterior parts of temporal lobes for both groups of signers.

The surface area expansion observed in the signers' left anterior temporal lobe may be an adaptation of the cortical network involved in the processing and integrating of visual information from the faces and hands during signed language comprehension. Harry, Umla-Runge, and Lawrence (2016) proposed that perceptual face and body information from the fusiform face area, extrastriate body area, and STS integrate and terminate in the right ventral ATL in a hierarchical feedforward process. A similar, but language-related, feedforward process may be occurring in the left hemisphere for signers because the perceptual integration of facial expressions and body movements is critical for signed language comprehension. Brentari, Falk, Giannakidou, and colleagues (2018) reported that ASL signers process specific non-manual and temporal prosodic cues more accurately than hearing nonsigners. However, unlike the deaf signers, hearing signers also showed an enlarged surface area in the right ATL, suggesting that there may be some differences in how hearing vs. deaf signers process and integrate perceptual information from face and body movements. Hearing signers may process facial expressions differently because they do not communicate in signed language as extensively as deaf signers (Paludneviciene et al. 2012).

Although all signers were bilinguals, the observed changes in the left occipital lobe and left ATL are not likely due to bilingualism *per se*. Previous morphometry studies comparing the brains of spoken language bilinguals and monolingual speakers did not report any structural

changes in these regions (Mechelli et al., 2004; Ressel et al. 2012; Klein et al., 2014; Stein et al. 2014). Therefore, the neuroanatomical changes found for the deaf and hearing signers in our study are more likely the result of lifelong experience with visuospatial language, and these changes are neural adaptations to allow for more rapid and efficient integration of visual stimuli for signed language comprehension.

Neuroanatomical changes unique to hearing native signers

In addition to the effects of deafness and sign language experience, our analyses also revealed a pattern of neuroanatomical changes that may be due to a unique lifelong experience associated with hearing native signers. Specifically, the local gyrification within the SMA and premotor areas in the left hemisphere were found to be reduced in hearing native signers when compared to both deaf signers (Figure 4C) and hearing sign-naïve controls (Figure 4B). Moreover, hearing signers also showed the same gyrification reduction in the right hemisphere homologs of these regions when compared to deaf signers. (Figure 4C). Both SMA and premotor areas in the left hemisphere are known to be involved in selecting, planning, initiating, coordinating, and monitoring various complex sequences of motor movements--e.g., initiating speech or coordinating complex sequences of bimanual movements (Penfield and Welch, 1951; Lee, Chang, & Roh, 1998; Luppino and Rizzolatti 2000, Hertrich, Dietrich, & Ackermann, 2016). Tourville et al. (2007) suggested that speech production recruits both hemispheres and that each hemisphere subserves different functions. Specifically, the forward control of speech is primarily controlled by language regions in left hemisphere, whereas right hemisphere homologs are involved in the monitoring and correction of speech production through auditory feedback. The reduction in the local gyrification within the left and right SMA and premotor areas suggests

that they may also be connected in some way to hearing signers' reduced cortical thickness in the left and right precentral gyrus (Figure 2C, 2B), *and* enlarged surface area in the right IFG (Figure 3B, 3C) near the motor area known for control of the lips and tongue (Meier et al. 2008).

The cortical thickness, surface area, and local gyrification changes in the frontal regions observed in the hearing native signers may also be connected to the neuroanatomical differences in the right posterior superior temporal sulcus (pSTS) and middle temporal gyrus (MTG). Hearing native signers showed a smaller surface area in the right pSTS and larger surface area in the right MTG when compared to hearing sign-naïve controls (Figure 3B) and deaf signers (Figure 3C). Beauchamp and colleagues (2004) reported that pSTS responds more to meaningful auditory and visual information and produces an even stronger signal when both types of information are integrated. The area pSTS is also known to be sensitive and responsive to changing facial expressions and eye gaze (e.g., Hoffman and Haxby 2000). Harry and colleagues (2016) suggest that the components of facial expressions (e.g., mouth configuration or eye gaze) are processed in the pSTS and integrated into the more anterior part of the temporal lobe and eventually terminated in the right IFG via the amygdala and insula. Davie-Thompson and Andrew (2012) propose that the right IFG processes and monitors facial expressions holistically. Carr et al. 2003 and Engell et al. 2007 reported that the perception and judgment of facial expressions strongly activate the right IFG. The MTG area adjacent to the pSTS is known to be activated during the perception of the human figure or dots representing human body movements (Kanwisher et al., 2001, Saygin et al., 2004). The MTG and pSTS in the right hemisphere showed increased neural activity when hearing native signers process BSL sentences (MacSweeney et al. 2002). Finally, Pyers and Emmorey (2008) reported that native hearing signers often continue to produce and coordinate specific ASL facial grammatical markers even

when they are speaking in English to hearing non-signers, which indicates a strong integration between linguistic facial expressions and spoken language. Thus, the differences in the right hemisphere neuroanatomical structures found for hearing native signers may be due to lifelong experience in processing and integrating linguistic information from auditory signals, facial expressions, and body movements.

Conclusion

Our study with a large number of participants replicated and confirmed previous findings of structural changes in occipital areas and reduction of white matter in left Heschl's gyrus for deaf signers. In addition, our SBM analyses also revealed a deafness-related reduced gyrification in the right middle frontal gyrus, suggesting that there may be significant changes in white matter structures in frontal regions for deaf signers. We speculate that the changes are a result of deafness-related experiences (e.g., less use of speech) rather than directly from auditory deprivation. These observed neuroanatomical changes will require further study to determine exactly what caused these neuroanatomical changes.

Our study also suggests that lifelong experience with processing and integrating visual information from the face and hands during sign language comprehension may lead to significant changes in neuroanatomical structures in the left occipital cortex and left ATL. In addition, our findings indicate that hearing signers have additional structural changes in the SMA and precentral gyrus bilaterally, and the pSTS and MTG in the right hemisphere. We suggest that these differences reflect expertise-related changes in both groups of signers due to a complex interaction among competing neural networks involved in the monitoring, comprehending, and processing of face and body movements for signed and spoken languages.

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