Age of Onset and Duration of Deafness Drive Brain Organization for Biological Motion Perception in Non-Signers

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Abstract

We used fMRI to characterize the neural responses to biological motion in adults who became deaf at a wide range of ages, none of whom were fluent sign language users. Although hearing people showed stronger activation for communicative than non-communicative gestures throughout the occipito-temporal biological motion and frontal-parietal action perception networks, deaf people showed equivalent levels of activation to both types of stimuli, suggesting an enhanced sensitivity to biological motion. Deaf people exclusively showed responses to communicative gestures in the left superior temporal gyrus (associated with speech processing) and right inferior parietal lobe. Further, earlier onset and longer duration of deafness led to stronger cortical responses in the biological motion and action perception networks, and extended into left superior temporal lobe areas associated with speech and other auditory processing. Together these results demonstrate that auditory deprivation, in the absence of sign language experience, can profoundly change the sensitivity of cortical networks for communicative biological motion processing. Furthermore, we demonstrate for the first time that neuroplastic reorganization of the visual system can occur even in people who became deaf after childhood.

Keywords: fMRI, neuroimaging, gesture, communication, vision, cross-modal plasticity

Age of Onset and Duration of Deafness Drive Brain Organization for Biological Motion Perception

Unimodal sensory deprivation, such as deafness and blindness, provide opportunities for us to understand the neuroplastic capabilities of the brain to adapt to experience. Extensive animal and human evidence suggests that when the cerebral cortex is deprived of input from one sensory modality from birth, the brain regions that would normally process information show increased sensitivity to input from other modalities, and these changes are often associated with functional changes as well. For example, congenitally deaf cats outperform hearing cats on a number of visual tasks, including motion detection and detection of targets in the visual periphery (Lomber et al. 2010). Although primary auditory cortex (A1) is not activated by visual stimuli in deaf cats, evidence suggests that secondary auditory processing areas not only show increased sensitivity to specific visual stimuli (specifically, moving stimuli and those presented in the visual periphery), but that this increased sensitivity underlies the observed changes in behaviour (Lomber et al. 2010).

Similar changes have been observed in deaf humans. Congenitally deaf adults show a very specific profile of greater sensitivity in tasks requiring selective attention to the visual periphery (Bosworth and Dobkins 2002a; Dye et al. 2007; 2009; Bottari et al. 2011; Bosworth et al. 2013), and to visual motion (Neville and Lawson 1987; Bosworth and Dobkins 1999; Brozinsky and Bavelier 2004; Mohammed et al. 2005; Stevens and Neville 2006; Hauthal et al. 2013), but not other visual tasks such as contrast sensitivity (Bavelier et al. 2001; Finney and Dobkins 2001; Bosworth and Dobkins 2002b; Bavelier et al. 2006). Visual attentional abilities are also modulated, with deaf people showing more interference when distracting stimuli are presented in the visual periphery, but less interference from stimuli presented centrally, than

normally hearing individuals (Proksch and Bavelier 2002; Dye et al. 2007; 2009). These behavioural changes associated with deafness are paralleled by specific neuroplastic changes. Early cortical visual responses are altered in deaf people (Neville and Lawson 1987; Bottari et al. 2011), and the N1 event-related potential (ERP) response to attended, peripheral visual motion is enhanced in deaf native signers and has an altered scalp distribution consistent with greater involvement of temporal cortex (Neville and Lawson 1987). Consistent with behavioural findings, functional MRI (fMRI) studies have demonstrated greater activation of human motion-sensitive cortex (labelled hMT or V5) in deaf than hearing people during attention to visual motion in the periphery, but greater activation in these regions for hearing people during attention to the central visual field (Bavelier et al. 2000; 2001). As well, attended peripheral visual motion has been shown to activate right superior temporal gyrus (STG) in the general region of A1 in congenitally deaf native sign language users (Finney et al. 2001; Fine et al. 2005), as well as both greater right intraparietal sulcus (IPS) and prefrontal activation, and increased functional connectivity between V5 and the IPS (Bavelier et al. 2001; 2006). Laterality differences have been found as well: while normally hearing non-signers tend to show right-lateralized activation to visual motion, both deaf and hearing signers show left-lateralized activity in V5 (Bavelier et al. 2001), suggesting that lifelong use of a visual-manual language induces a leftward shift in the cortical resources used in motion processing, perhaps because of the left hemisphere's dominance for language (Hickok et al. 1996; Neville et al. 1998; MacSweeney et al. 2002; 2008; Newman, Supalla, Hauser, Newport, and Bavelier 2010a; 2010b; Newman et al. 2015).

Neural responses to human movements (biological motion) are also altered in congenitally deaf native signers. In normally hearing non-signers, biological motion perception

activates a characteristic set of occipito-temporal regions (hereafter referred to as the "biological motion perception network") that includes V5, the posterior part of the superior temporal sulcus (STSp), the extrastriate body area (EBA), and the fusiform body area (FBA) — particularly in the right hemisphere (Grossman et al. 2000; Grossman and Blake 2002; Peelen et al. 2006; Rizzolatti and Sinigaglia 2010). A second, frontal-parietal network is typically activated during both perception and execution of object- or self-oriented actions. This putative human "mirror neuron system" includes dorsal premotor regions (PMd), the inferior frontal gyrus (IFG), the pre-supplementary motor area (pre-SMA), and intraparietal sulcus (IPS). Because only a small subset of the neurons comprising the system have true "mirror" properties (i.e., firing similarly when an action is observed or performed), it can more generally be defined as a frontal-parietal network involved in human action observation and execution (Rizzolatti and Sinigaglia 2010), and we refer to it hereafter as the "action perception network". The patterns of activation elicited by biological motion also depend on the content; several studies have contrasted symbolic gestures with established meanings (emblems, e.g., "thumbs-up" or "OK") with object-oriented actions (either involving real objects or pantomimed actions). Normally hearing non-signers show greater activation in the action perception network for meaningful than non-meaningful gestures, including bilateral frontal (Lotze et al. 2006; Villarreal et al. 2008; Andric et al. 2013), STS (Lotze et al. 2006; Montgomery et al. 2007; Andric et al. 2013), inferior (supramarginal gyrus — SMG) and superior (IPS) parietal (Nakamura et al. 2004; Andric et al. 2013); greater activation has also been reported in occipito-temporal biological motion perception regions (Nakamura et al. 2004).

Deaf signers show distinct patterns of activation during biological motion perception. Several studies have provided evidence of cross-modal plasticity, in the form of activation in

superior temporal gyrus (STG). This includes the auditory cortex and the planum temporale, which integrates auditory and visual information in hearing people. STG activation has been seen in deaf people relative to hearing people in tasks requiring them to derive meaning from biological motion, including sign language, emblems, meaningless gestures, and speechreading (Petitto et al. 2000; Campbell et al. 2001; MacSweeney et al. 2001; Calvert and Campbell 2003; MacSweeney et al. 2004; Capek et al. 2010; Husain et al. 2012; Cardin et al. 2013). Husain and colleagues (2012) found posterior STG activation unique to deaf signers during gesture perception; hearing non-signers instead activated the action perception and occipito-temporal biological motion networks. Although all of the above studies involved prelingually deaf native signers, one study provides insight into the relative influence of auditory deprivation versus sign language in driving STG recruitment. Cardin and colleagues (2013; 2016) compared prelingually deaf signers and non-signers, and found significant right STG activation for both groups when viewing sign (which was only meaningful to signers), along with left STG activation for both groups, but stronger and more extensive in the signers. This suggests that auditory deprivation leads to greater sensitivity of auditory processing regions to biological motion, and additionally that some tuning towards meaningful biological motion occurs in the left STG. Cardin and colleagues' data also suggest that within the STG some areas are tuned specifically to linguistically structured manual gestures (signs).

Both the task and the meaningfulness of the stimuli can influence the networks engaged in deaf signers during biological motion perception. Two other studies have found overlapping activation for sign language and gestures within classical language areas — but not the action perception network — when the task involved trying to derive meaning from the gestures (MacSweeney et al. 2004; Newman et al. 2015). This was true for both meaningful (Newman et

al. 2015) and meaningless (MacSweeney et al. 2004) gestures; hearing non-signers showed activation only within the frontal-parietal action perception network and STSp (Newman et al. 2015). This suggests that when deaf signers attempt to understand sequences of gestures, they may apply their knowledge of sign language — such as abilities to segment fluid sequences into individual units of meaning, and attempts to derive meanings based on the similarity of gestures to signs — rather than using the action perception network used by hearing non-signers. This suggests, however, that deaf non-signers would not show activation within language centers as they have no experience with visual-manual language.

Two other studies have also suggested that deaf signers may not engage the frontal-parietal action perception network during either communicative or non-communicative biological motion perception (Corina et al. 2007; Emmorey et al. 2010). Both studies contrasted brain activation for pantomimed actions and American Sign Language (ASL) in deaf signers and hearing non-signers, and found similar results. Both studies found that while hearing non-signers engaged the occipito-temporal biological motion and frontal-parietal action perception networks, deaf signers did not activate the action perception network for either gesture or sign. Rather, deaf signers showed activation in the occipito-temporal biological motion network for pantomime, while for sign language they activated classical left-lateralized language areas (including the IFG and middle/anterior STS/STG) consistent with numerous other studies of sign language processing (Neville et al. 1998; MacSweeney et al. 2002; 2008; Husain et al. 2009; Newman, Supalla, Hauser, Newport, and Bavelier 2010a; 2010b; Newman et al. 2015). Similar absence of action perception network activation has been reported in subsequent studies of deaf native signers (Husain et al. 2012; Newman et al. 2015). It should also be noted that although numerous studies have reported occipito-temporal biological motion perception network activation in both

signers and non-signers for sign language and gesture (MacSweeney et al. 2004; Corina et al. 2007; Emmorey et al. 2010; Husain et al. 2012), Newman and colleagues showed that this is an artifact of using a fixation or other baseline that does not control for biological motion; this activation is eliminated when well-matched control stimuli are used.

Taken together, these results suggest that congenitally deaf native signers show an altered pattern of neural responses to meaningful non-linguistic gestures. Specifically, while hearing non-signers engage a human action perception network for perceiving gestures, deaf signers do not activate these premotor or parietal regions. Rather, when viewing meaningful gestures deaf signers engage a left-lateralized network of regions largely overlapping with the classical left-lateralized language networks engaged by sign language itself. A likely interpretation for this is that lifelong use of a visual-manual language leads to increased sensitivity of language cortex for manual communication, even when it is not linguistically structured. As well, some evidence points to greater activation of peri-auditory regions of the STG in deaf signers, both for low-level visual motion and when making sense of biological motion. Following from the animal research mentioned above, an intriguing hypothesis is that in the absence of auditory input to this multimodal integration area, the projections of other modalities (notably vision) predominate — leading to higher responsivity to visual input than in people whose brains also provide auditory input to these areas.

While these studies collectively paint a fascinating picture of experience-induced neuroplasticity, they have restricted themselves to a relatively narrow population: people who were pre-lingually deaf and who were exposed to fluent sign language from birth (though Cardin:2013ck and Cardin et al. 2016 included congenitally deaf non-signers). This reduces uncertainties in interpretation that could arise in a more heterogeneous group, because later onset

of deafness would would introduce variance in both auditory experience and spoken language exposure. As well, people not exposed to fluent signing from birth are unlikely to develop native-like sign language proficiency (Newport 1990) and, if pre-lingually deaf, will not have any natural native language and may show lifelong language deficits (Mayberry et al. 2002). At the same time, studies of deaf native signers are limiting in three important ways. Firstly, they do not allow us to distinguish the effects of lifelong auditory deprivation from sign language exposure. Secondly, they do not provide insight into whether there are sensitive periods early in development during which auditory deprivation has a magnified effect on later brain organization — or whether later-onset deafness could also lead to altered functional brain organization. Finally, studies of deaf native signers are not generally representative of the deaf population, as less than 5% of deaf people meet these criteria. Understanding how the age of onset of deafness affects brain organization, and whether sign language exposure is critical for inducing changes in the sensitivity of different neural networks, have important implications both for our understanding of the neuroplastic capacities of the human brain, as well as practical applications for people receiving, or considering, cochlear implants to restore hearing after an often prolonged period of deafness.

In the present study we began the exploration of these questions by comparing brain activation in deaf people who were not fluent signers (most of whom, in fact, knew no sign language at all) and whose deafness began at ages varying from birth to late adulthood. We compared brain activation, measured using fMRI, between deaf and hearing individuals while they viewed communicative gestures (emblems), non-communicative but meaningful gestures (pantomimed actions), and scrambled control stimuli. The stimuli we developed and used for this study point-light movies (Johansson 1973), showing only white dots positioned on major joints

of the body as well as all the joints in the hands (Zaini et al. 2012). This allowed very strict control of low-level stimulus features, both in eliminating many such features altogether (including the face and human form), and in allowing us to create control stimuli by scrambling the starting positions of each dot, preserving exactly the motion trajectories and speeds of the individual points while eliminating the percept of interpretable human movement.

We predicted that normally hearing non-signers would show a pattern of activation similar to previous studies. That is, activation of the occipito-temporal biological motion perception network (likely with a right hemisphere dominance) and the frontal-parietal action perception network bilaterally, with stronger activation for communicative emblems than pantomimes in the IFG and MFG of the frontal lobe, the STS (particularly anterior regions), the IPS, and the occipito-temporal junction including V5. For deaf non-signers, we had alternative predictions depending on the nature of neuroplastic changes that occur with deafness, in the absence of any significant sign language experience. If ASL fluency drives an increased sensitivity of classical language areas to meaningful non-linguistic gesture, then we predicted that deaf non-signers would not differ from hearing non-signers in IFG or STS activation. Alternatively, if auditory deprivation alone is sufficient to sensitize classical language areas to nonlinguistic, manual symbolic communication, then we predicted greater activation in the IFG and STS (including STG and temporal-parietal junction) for meaningful gesture (emblems) in deaf people, both relative to hearing people and relative to pantomime.

On the assumption that sign language experience, and not auditory deprivation, drives a leftward shift in laterality of V5 activation (as indicated by Bavelier et al. 2001), we predicted that deaf non-signers would show similar right-lateralization of this area to hearing non-signers. In a similar vein, if previous studies showing that deaf signers do not activate the action

perception network (Corina et al. 2007; Emmorey et al. 2010; Husain et al. 2012) are due to sign language fluency, then we expected that these regions would be activated in deaf non-signers. Alternatively, greater activation of the action perception network and/or temporal-occipital biological motion perception networks in hearing than deaf non-signers would suggest that auditory deprivation, rather than sign language exposure, is the cause of reduced activation previously reported for deaf native signers.

We further reasoned that deaf non-signers might differ from both deaf signers and hearing non-signers in their neural responses to human actions, specifically because they might have to develop greater sensitivity to human movements in the absence of access to either spoken or sign language. Specifically, we predicted reduced differences in activation between emblems and pantomimes in deaf than hearing non-signers. Finally, we considered the question of whether age of onset of deafness affected the predicted neuroplastic changes — in other words, whether such changes would be attenuated with later onset and/or shorter duration of deafness. Thus in addition to comparing conditions within groups and between groups, we conducted regression analyses with age of deafness onset and duration of deafness as predictors. We predicted that both earlier age of onset, and longer duration of deafness, would be associated with greater differences between deaf and hearing people's activation patterns.

Materials and Methods

Participants

Normally hearing participants (n = 17; 10 males; mean age 45 years, 16 right-handed; hereafter simply referred to as "hearing") were recruited through posters, on-line advertisements, and word of mouth; normal hearing was confirmed with a brief audiogram to ensure a threshold of 30 dB or better in each ear for a 1000 Hz pure tone. Deaf participants (n = 14; 7 males; mean

age 52 years, 12 right-handed) were all candidates for cochlear implantation and had pure tone thresholds of 90 dB or greater in both ears as determined by an audiologist. Deaf participants were all recruited through the Nova Scotia Cochlear Implant Program (Nova Scotia Hearing and Speech Centres, and Queen Elizabeth II Health Sciences Centre/Capital District Health Authority). Mean age of onset of deafness was 15.4 years, with a mean duration of 33.4 years. None of the participants in this study reported being fluent in any sign language, though some reported some knowledge of American Sign Language. Detailed information for each deaf participant appears in Table 1. Two-tailed t-tests were performed to determine whether there were significant differences between deaf and normally hearing participants for age or years of education. There was no significant difference in age between the groups, $t(29) = 1.43$, $p = 0.164$, although normally hearing participants had more years of education (16 years, SE = 0.8) than deaf participants (14 years, SE = 0.8), $t(29) = -2.05$, $p = 0.05$. Participants received nominal financial compensation for their participation. All study procedures were reviewed by the Capital District Health Authority Research Ethics Board. All subjects gave written informed consent in accordance with the Declaration of Helsinki.

TABLE 1 ABOUT HERE

Stimuli

Stimuli included point-light animated videos of communicative gestures (emblems), non-communicative gestures (pantomimes), and scrambled versions of these gestures. The details of the development, recording, and standardization of these stimuli were reported in detail by Zaini and colleagues (Zaini et al. 2012); here we provide a summary. All stimuli were performed by a human actor and recorded using a motion capture system (Measurand Inc., Fredericton, NB). Motion capture recordings were then mapped onto a 3D skeleton of a human body and edited in

Motion Builder 2008 (Autodesk, San Rafael, CA) to correct for motion capture errors (such as collisions of body parts) and to ensure that all movements appeared natural and were clear from a frontal viewing angle. Point-lights (white 3D spheres) were then placed at key joints on the skeleton, including the fingertips and knuckles to provide detailed information about hand shape and orientation. The point-light animations were then exported with a black background and no other body parts visible, as Quicktime movie files. Scrambled stimuli were created by randomizing the starting position of each point-light in the first frame of the animation, while preserving each point's motion vector from that first frame. The full set of stimuli is available as supplementary material published with Zaini and colleagues.

Procedure

Stimuli were presented using the Psychophysics Toolbox (Brainard 1997; Pelli 1997) implemented in MATLAB (The Mathworks, Inc., Natick, MA) running on an Apple computer (Apple Inc., Cupertino, CA). Stimuli were back-projected onto a bore-mounted screen by a DLP projector (Texas Instruments Inc., Dallas, TX). Participants viewed the stimuli projection through a mirror attached to the MRI head coil.

A block design was used to present the stimuli; block length varied due to differences in the duration of the individual stimulus movies. Each block lasted 15–20 sec, and included a minimum of six movies from one of the three stimulus categories (communicative, non-communicative, and scrambled). Rest blocks, during which a white fixation cross was presented against a black background, were also included. Blocks were presented in pseudorandom order, with the restriction that two blocks of the same category did not immediately follow one another. Including rest blocks, there were 21 blocks in each run, which lasted for 6.1 min. Each participant completed two fMRI runs.

Prior to entering the MRI, participants received verbal and, if desired, written instructions (deaf participants varied in whether their aided hearing, combined with speechreading, was sufficient to understand verbal instructions). All participants practiced the task prior to entering the MRI scanner and had the opportunity to ask questions. These instructions were also repeated in printed form on the screen immediately prior to each fMRI run. For each stimulus movie, participants were asked to make a decision about whether the movie showed a person performing an action (biological motion), or scrambled (non-biological) motion. Participants used two buttons on a hand-held button-press response pad (Current Designs, Inc., Philadelphia, PA) to record their decision, and were told to respond as quickly as possible upon seeing the start of each movie and making a decision; no prompts to respond were made during stimulus presentation. The response pad was pseudo-randomly assigned to either the left or right hand, counter-balanced across participants. Due to technical errors, for two deaf participants behavioral responses were only recorded for one of the two fMRI runs and for two control participants no behavioral responses were recorded; however in all cases participants made responses, and all fMRI data were included in the analyses presented here.

MRI Acquisition

We used a 1.5 T General Electric MRI scanner. For fMRI, 27 contiguous 4 mm thick slices were acquired using a spiral in/out sequence (TR: 2.2 s, TE: 40 ms, flip angle: 90, FOV: 24 cm) at a resolution of 3.75 x 3.75 mm in-plane. T1-weighted structural images were obtained to facilitate image registration, using a SPGR pulse sequence with a 256 x 256 matrix, 162 slices, 1 mm isotropic resolution, TR = 11.3 s; TE = 4.2 ms; flip angle = 20 deg.

Data Analysis

Behavioral Data

For behavioral analysis only, any trials that did not contain a response were removed from the data set. Linear mixed effects modeling (Baayen et al. 2008) using the *lme4* package (Bates 2005) in the R language was used to analyze reaction time, as a function of fixed effects including trial, hearing status (deaf or normally hearing), and stimulus type (communicative, non-communicative, or scrambled), as well as random effects of subject, item, and trial. Outliers were defined as data points that fell more than 2.5 standard deviations outside of the mean and were removed from analysis. This represented 0.79% of the data (34 trials removed out of 4265). Afterwards, the model was re-fitted to the trimmed data. Multiple comparisons were controlled for using Bonferroni corrections across the three stimulus types. Generalized linear mixed effects modeling using a binomial family was used to analyze accuracy as a function of fixed effects including trial, hearing status, and stimulus type, as well as random effects of subjects and items.

fMRI Data

Functional MRI (fMRI) data analysis was performed using FEAT (FMRI Expert Analysis Tool), version 6.0, in FSL (FMRIB's Software Library, www.fmrib.ox.ac.uk/fsl), version 5.06. Pre-statistical analysis involved skull removal using FSL's Brain Extraction Tool (Smith 2002), motion correction via MCFLIRT (Jenkinson et al. 2002), spatial smoothing with a Gaussian kernel FWHM of 7 mm, and 0.01 Hz highpass temporal filtering. The estimates of head motion generated by MCFLIRT were examined and any runs containing 4 mm or greater of motion within three time points were eliminated from further analysis; this resulted in rejection of only one run in this dataset. As well, based on the motion correction output three runs (one from each of three deaf participants) were identified as having scanner artifacts and were excluded from

further analysis. Using FMRIB's Linear Image Registration Tool (FLIRT; Jenkinson et al. 2002), fMRI data were registered to a T1-weighted anatomical image of the participant's brain at 6 degrees of freedom (DOF), and the anatomical data were registered to standard MNI space at 12 DOF; these two transforms were combined to map the functional data to standard space. Functional data were resampled into standard space at a resolution of 2 mm isotropic after the first-level analyses were performed.

The time-series of individual fMRI runs were analyzed using the general linear model as implemented in FEAT, with local autocorrelation correction (Woolrich et al. 2001). Separate regressors for each stimulus type (communicative, non-communicative, and scrambled) were included, with head motion estimates included as model parameters of no interest. The planned contrasts performed at this stage, and passed to subsequent analysis stages, included the following: each stimulus type versus baseline; communicative versus scrambled; non-communicative versus scrambled; and communicative versus non-communicative. Parameter estimates and their variance for each coefficient from the first-level analysis were subsequently combined within subjects, across runs, using fixed-effects modelling in a second level analysis.

At the group level, each of the above-described contrasts were computed separately for each group (hearing and deaf). As well, direct comparisons were made between groups for each of these contrasts. Group-level analysis was performed using nonparametric statistics ("Nonparametric permutation tests for functional neuroimaging: a primer with examples" 2002) implemented in FSL's *randomise* (v. 2.9) function (Freedman and Lane 2012) with 7 mm variance smoothing. The nonparametric analysis approach was chosen because it makes fewer assumptions than typical general linear model (GLM)-based parametric analysis, particularly with respect to the distributions of the data and homogeneity of variance between groups (or

even conditions; "Nonparametric permutation tests for functional neuroimaging: a primer with examples" 2002). It also allowed us to estimate statistics within each ROI that appropriately corrected for multiple comparisons and yielded a robust estimate of cluster-level statistics within each cluster. In this method, the design matrix is partitioned into tested and nuisance effects. The data are fit to the nuisance effects alone to form nuisance-only residuals, which are then permuted to create an approximation of the data under the null hypothesis. The full model is then fit using this dataset, and this process is repeated to estimate the data-defined distribution of the test statistic under the null hypothesis. For the simple models above, this method is equivalent to the standard exact tests; otherwise, it accounts for nuisance variation present under the null. In the present case, 5,000 permutations were computed using a Conditional Monte Carlo (CMC) permutation test, which is estimated to yield an estimation of $p = .05$ with 95% confidence intervals of ± 0.0062. Following this nonparametric test to estimate the p values at each voxel, clustering was performed using FSL's threshold-free cluster enhancement procedure, which uses a nonlinear image processing technique suitable for detecting both focal and diffuse signals by amplifying the height of spatially continuous clusters without affecting the location of their maxima (Smith and Nichols 2009). In reporting the results in the tables and test, clusters were ignored if they were less than 50 voxels in size, or 10% of the volume of the ROI (whichever was less; to accommodate small ROIs) as a further control against type I error and to minimize the effects of "spillover" of activation that was predominantly in an adjacent ROI. This nonparametric analysis was performed at the whole-brain level for the contrasts of each stimulus condition versus fixation baseline. For all other contrasts, this analysis was performed only within a set of pre-defined regions of interest (ROIs) as discussed in the next paragraph.

Planned region of interest (ROI) analyses were also performed for a set of regions predicted a priori to show task modulation and group differences (see Introduction for a review of the supporting literature). These ROIs were defined from the Jülich probabilistic cytoarchitectonic atlas provided with the FSL software package (version 5.0.8; Eickhoff et al. 2005). Areas were defined based on the Jülich atlas if they were defined there (this atlas does not encompass the entire brain), and the Harvard-Oxford atlas otherwise. These included: primary auditory cortex (A1; defined cytoarchitectonically as area TE and likely including secondary and tertiary auditory areas as well); anterior and posterior superior temporal gyrus (STGa and STGp); anterior, posterior, and temporal-occipital portions of the middle temporal gyrus (MTGa, MTGp, MTGto); the supramarginal gyrus (SMG), angular gyrus (AG), and inferior parietal sulcus (IPS) defined cytoarchitectonically (Caspers et al. 2006); the IFG (defined cytoarchitectonically as BA44 and 45; Amunts et al. 1999), premotor cortex (including the SMA; defined cytoarchitectonically), and V5 (defined cytoarchitectonically; (Malikovic et al. 2007). Both left and right hemisphere regions were defined and analyzed, separately, for each ROI.

In addition to these anatomically-defined ROIs, we also investigated two functionally-defined ROIs based on prior literature: the STSp and STSa. STSp was defined as centered on MNI152 coordinates -51, -56, 11 (left hemisphere) and 51, -50, 10 (right hemisphere), based on a meta-analysis of STSp activation during the processing of biological motion, audio-visual integration, and faces (Hein and Knight 2008); converted from their reported Talairach coordinates to MNI using http://sprout022.sprout.yale.edu/mni2tal/mni2tal.html}. STSa was defined based on the coordinates obtained in Newman and colleagues (Newman, Supalla, Hauser, Newport, and Bavelier 2010a) for the contrast of ASL with well-controlled nonlinguistic, biological motion stimuli, as -64, -4, -10 (left) and 64, -8, -16 (right); these coordinates are very

similar to those obtained for both ASL and gesture contrasted with well-matched control stimuli in both signers and non-signers (Newman et al. 2015). Both the STSa and STSp ROIs were defined as 10 mm diameter spheres centered on the coordinates specified. It should be noted that the STSa and STSp ROIs are not entirely orthogonal with the anatomically-defined ROIs. The STSa overlaps with STGa and MTGa, while the STSp overlaps with STGp, MTGp, and MTGto. We chose to present results from both since the fact that these functionally-defined regions overlap multiple anatomically defined ones, means that in a purely anatomically-driven ROI analysis real clusters of activation might be missed by virtue of being divided amongst ROIs. On the other hand, the functionally-defined ROIs are relatively small, and activation outside of the functionally-defined ROI would only be captured by the anatomically-defined ROIs.

Individual Differences Analysis. We also investigated how duration and age of onset of hearing loss might modulate brain activation within the group of deaf participants. The variables we investigated were: age of first diagnosis of hearing loss; age of onset of profound, bilateral deafness; and duration of profound deafness.[1] For these analyses, we employed whole-brain parametric analysis using FLAME 1 in FSL. The procedures and parameters were as described above, with the addition of an explanatory variable in each analysis that coded the mean-centered individual difference parameter of interest. For consistency with the other analyses reported, in the paper we report results within the same set of ROIs described above.

Results

Behavioral Data

Reaction times were analyzed by fitting a linear mixed effects model where reaction times were regressed on a three-way interaction including trial, stimulus type (communicative, non-communicative, scrambled), and group (normally hearing, deaf). The model also included

by-subject and by-item random intercepts. There was a main effect of stimulus type, $F(1, 3806) = 7.6$, $p < 0.001$, and trial, $F(1, 3806) = 145.0$, $p < 0.001$, as well as a significant interaction between trial and group, $F(1, 3806) = 5.9$, $p = 0.015$. No other main effects or interactions were significant.

To understand the main effect of stimulus type, we performed a post-hoc analysis that compared every pair of stimulus types. Probability values were two-tailed and Bonferroni corrected for three comparisons. The post-hoc comparisons revealed that reaction times for communicative gestures ($M = 861$ ms, 95% CI [737, 1006]) were significantly faster than for non-communicative gestures ($M = 920$ ms, 95% CI [787, 1076]), with a difference of 59 ms, $t(3815) = -3.0$, corrected $p = 0.003$. Reaction times for scrambled controls ($M = 847$ ms, 95% CI [727, 988]) were also faster than for non-communicative gestures, with a difference of 73 ms, $t(3815) = -4.2$, corrected $p < 0.001$. Reaction times for communicative gestures and scrambled controls did not differ, difference = 13 ms, $t(3815) = 0.83$, $p = 0.406$. Because trial was included as a covariate of non-interest (to account for, e.g., possible effects of practice or fatigue over the course of the experiment), effects involving this variable were not investigated further.

In terms of accuracy, participants made only 29 errors out of 4265 trials. Due to this very low number of errors, which were not evenly distributed across cells, it was not possible to perform a statistical analysis of this data. However, given the low proportion of errors (0.67%) it is clear that all participants were able to perform the task very well across all conditions.

Each Stimulus Type versus Fixation

The results of the contrast between each stimulus type and fixation are shown in Figure 1. Both hearing and deaf groups showed extensive bilateral activation that included occipital, posterior temporal, inferior parietal, and prefrontal areas. Details of the activation patterns

identified within a priori ROIs are provided in Supplementary Table 1. Overall, most of the ROIs that we predicted to be involved in this task showed significant clusters of activation bilaterally across all three stimulus types. These included, in the frontal lobes, the IFG and precentral sulcus; in the parietal lobes, the SMG, AG, and IPS; the posterior temporal-lateral occipital region, including posterior temporal biological motion area STSp and the fusiform gyri; and visual motion area V5. The middle panel of Figure 1 shows the areas of conjunction between deaf and hearing groups, demonstrating the high degree of overlap in the overall network engaged by moving point-light displays across conditions and populations.

FIGURE 1 ABOUT HERE

Although some differences in the spatial extent of activation were apparent in a visual comparison between the hearing and deaf groups for these contrasts, direct statistical comparisons between the two groups, for each condition compared to fixation, revealed relatively focal differences. Deaf people showed no regions of greater activation than hearing. Hearing people, however, showed significantly stronger BOLD signal in area V5 of the right hemisphere than deaf people, for all three contrasts. Additionally, for biological motion (communicative and non-communicative) hearing people showed stronger activation than deaf in the right precentral gyrus. For scrambled stimuli only, hearing people showed stronger activation in the left precentral gyrus and the anterior STG in the region of the primary auditory cortex. These group differences are shown in Supplementary Figure 1; details are provided in Supplementary Table 2.

Biological versus Scrambled Motion

As expected, comparing each biological motion condition with the scrambled point-light motion stimuli yielded more restricted areas of activation focused largely on posterior temporal-occipital and parietal cortex, as shown in Figure 2. These included area V5 bilaterally as well as right SMG, AG, IPS, and precentral gyrus in both groups, for both types of biological motion. As well, consistent activation was obtained in the posterior temporal/lateral occipital region, including STSp, for both hearing and deaf people in the communicative–scrambled contrast, and for hearing people in the non-communicative–scrambled contrast, but not in the non-communicative–scrambled contrast for deaf people. Deaf people additionally showed left IFG and anterior, superior temporal activation for communicative gestures. Details of these contrasts are provided in Supplementary Table 3.

FIGURE 2 ABOUT HERE

Comparing the biological motion versus scrambled contrasts between groups yielded few differences. The only significant differences between groups were for deaf relative to hearing people, in the communicative – scrambled contrast, as shown in Figure 2. Here, deaf people showed stronger activation in a cluster in the right inferior parietal lobe (comprising the AG and IPS), and in the left STG area anterior to primary auditory cortex that was seen in the communicative–scrambled contrast. However, the IFG activation in the communicative–scrambled contrast seen only for deaf people did not emerge as a significant between-group difference. Details are provided in Supplementary Table 3.

Communicative versus Non-Communicative Gestures

The final contrast we examined was the direct comparison between communicative and non-communicative biological motion (emblems vs. pantomimes). In this contrast, shown in Supplementary Figure 2, deaf people showed no significant differences; the only differences obtained were in hearing people, with greater activation for communicative gestures. These areas included regions of the right superior and middle temporal gyri, extending from the A1 ROI ventrally and anteriorly, as well as a more restricted portion of the anterior left superior and middle temporal gyri, and the right precentral gyrus. Details are provided in Supplementary Table 4. The right precentral and left temporal lobe clusters overlapped with that seen in the communicative–fixation contrast for this group. However, interestingly the right temporal cluster seen in this contrast was not present in the contrasts of communicative biological motion with either fixation or scrambled control stimuli. No significant between-group differences were obtained for the non-communicative–communicative contrast.

Individual Differences: Age of Onset and Duration of Deafness

In contrast to previous research — which has focused on congenitally deaf, native signers to ensure a homogeneity of experience — in this study we recruited a more diverse group of deaf people. Our participants represented the larger population of deaf people who have had varying histories of hearing loss; as well none were native signers and few reported knowing any sign language at all (although two people did report higher fluency). We thus were able to investigate how their history of hearing loss may have affected the neural networks recruited for biological motion perception, in the absence of fluency in a visual-manual language. We performed additional linear mixed effects analyses on the communicative–scrambled and non-communicative–scrambled contrasts, with each of the following variables as an additional fixed

effect regressor: age of first clinically noted hearing loss; age of diagnosis of profound, bilateral loss; and duration of profound bilateral loss.

The results of these analyses are shown in Figure 3. There were no significant correlations with any variables for the non-communicative–scrambled contrast, nor between age of first reported hearing loss and the communicative–scrambled contrast. However, both age of profound bilateral loss and duration of profound loss showed correlations with strength of activation in the communicative–scrambled contrast, with largely overlapping patterns of regional involvement; details of these are reported in Table 2. The overlap between these two measures is not entirely surprising, given that they were correlated in our sample (-.78). However, we investigated both measures because they are not identical, as seen in the scatterplots for each a prior ROI shown in Figure 4. For example, one participant who became deaf at age 45 had been deaf for 17 years at the time of the study, while another who became deaf at age 46 had been deaf for only 1 year.

FIGURE 3 ABOUT HERE

FIGURE 4 ABOUT HERE

TABLE 2 ABOUT HERE

For age of profound bilateral loss, the correlation was negative, meaning that stronger activation in the communicative–scrambled contrast was associated with earlier onset of deafness. As shown in the top panel of Figure 3, the correlations involved most of the occipital-temporal biological motion network (including STSp, V5, and the fusiform gyrus; the strongest

correlation was found in the right temporal-occipital part of the fusiform gyrus) as well as bilateral inferior parietal lobe (including AG and SMG) and right precentral gyrus and IFG. In the temporal lobe, correlations were found in the left STSp, bilateral posterior and temporal-occipital parts of the MTG, and right anterior MTG. Because age of profound loss was correlated with chronological age in our sample (r = .60), we wished to ensure that the apparent effects of age of loss were not merely attributable to chronological age. Thus we also examined whether chronological age correlated with the communicative–scrambled contrast. The only area of significant correlation was in the right occipital lobe, however when we examined the relationship between the effects of age of deafness onset and chronological age, the overlap was restricted to a small region of the occipital fusiform gyrus (comprising less than 10% of the total number of voxels significantly correlated with age of profound loss), and did not overlap with any of our a priori regions of interest. Thus the effects of age of profound loss that we focus on here (including all areas shown in Figure 3) are independent of chronological age.

For duration of profound loss, all of the significant correlations were positive, meaning that larger communicative–scrambled differences were associated with longer durations of auditory deprivation. This is consistent with the correlation with age of profound loss, since earlier age of loss generally translates into longer duration, and indeed the two correlation maps showed largely overlapping patterns, as seen in Figure 3, again with the strongest correlations in the fusiform gyri. As can be seen in Table 2, the only differences within our a priori ROIs were correlations in the right IFG and anterior MTG for duration of loss but not age of loss; however examination of Figure 3, combined with the relatively small size of these clusters (< 100 voxels) suggests that these differences represent slightly different spatial extents of relatively large areas

of activation rather than distinct foci of activation. Another notable difference is greater spatial extent of correlations in the fusiform gyri for age of loss than for duration of deafness.

Discussion

The goal of this study was to characterize the patterns of brain activation elicited by communicative and non-communicative biological motion perception in deaf people who were not fluent sign language users, in order to examine neuroplastic changes stemming from auditory deprivation in the absence of visual-manual language experience. Further, we recruited a natural sample of deaf people whose age of hearing loss — rather than being pre-lingual as in most prior studies — was highly variable as was their duration of loss. This allowed us to investigate whether these variables influenced any neuroplastic changes that we observed.

We predicted task-related activation in both the occipito-temporal biological motion perception network (likely with a right hemisphere dominance) and the frontal-parietal action perception network (bilaterally), in both normally hearing and deaf people. Consistent with prior studies (Grossman et al. 2000; Grossman and Blake 2002; Lotze et al. 2006; Peelen et al. 2006; Villarreal et al. 2008; Rizzolatti and Sinigaglia 2010; Andric et al. 2013), these predictions were confirmed — with robust activation of these networks for both groups across communicative, non-communicative, and scrambled point-light stimuli, and stronger activation for both types of biological motion than for scrambled motion. Consistent with Bavelier et al.'s (2001) (2000) conclusions that sign language experience, and not auditory deprivation, drives a leftward shift in the laterality of V5 activation, we did not see such a shift here: hearing and deaf non-signers both showed consistently greater spatial extent and maximum *z* values in right than left V5 (or equivalent in both hemispheres),[2] as well another regions of the biological motion and action perception networks, including the fusiform gyrus and IPS.[3] Interestingly, however, we did not

find evidence for right lateralization of STSp activation in either group, in spite of the fact that we defined this area based on a meta-analysis of previous fMRI studies , which have generally suggested right-lateralization (Hein and Knight 2008).

Our next hypothesis concerned previous reports of left-lateralized activation of classical language areas by both communicative and non-communicative biological motion in deaf native signers, in contrast to bilateral action perception network activation typically observed for hearing non-signers (MacSweeney et al. 2004; Corina et al. 2007; Emmorey et al. 2010; Husain et al. 2012; Newman et al. 2015). Although these findings have generally been interpreted as an effect of sign language knowledge and experience, this hypothesis had not been tested in deaf non-signers viewing meaningful gestures (Cardin et al. 2013; 2016). Thus our study was the first to investigate whether auditory deprivation (and the associated need to pay closer attention to non-verbal information in communicative settings) was sufficient to drive a change from action perception to language processing network engagement for meaningful biological motion. Our results suggest separable influences of auditory deprivation and sign language experience. In the contrasts of each condition with fixation, both deaf and hearing people showed highly similar patterns of activation. This included the occipito-temporal biological motion network and — unlike deaf signers in previous studies — the action perception network. Furthermore, deaf non-signers did not show significantly greater activation than hearing people in any brain region. However, in the better-controlled contrast of communicative gestures with scrambled motion, deaf people did show stronger activation than hearing in the left posterior STG/STS, as well as in a right parietal lobe region that included the angular gyrus and IPS.[4]

Interestingly, the MNI coordinates of the peak of this activation place it within regions of auditory cortex identified as important for speech perception in a meta-analysis (Scott and

Johnsrude 2003). In that analysis, the closest previously-reported peaks of activation were associated (in normally hearing people) with acoustic complexity, including spectrally or harmonically modulated sounds relative to simpler ones, and speech relative to acoustically-matched controls. As such, our findings provide evidence of neuroplastic reorganization of auditory cortex regions typically used for speech perception, to understand communicative human movements in deaf people. This left STG/STS activation overlaps with activation we previously found for both sign language and communicative gesture in native signers, but not hearing non-signers (Newman et al. 2015). Likewise, Cardin and colleagues reported stronger activation in this region in response to sign language in congenitally deaf signers then non-signers (Cardin et al. 2013). In the present study activation in this area did not correlate with either age of onset or duration of profound hearing loss. This suggests that the neuroplastic reorganization we observed can occur even with adult onset — and relatively short durations — of deafness, rather than being a phenomenon related to sensitive periods in cortical development, as previously assumed. This may suggest that this change is functionally-driven, i.e., a neuroplastic adaptation due to the greater need for deaf people living in a hearing world to rely on nonverbal communication.

Beyond the left STG/STS, we did not find other classical language areas (such as the left IFG or more extensive regions of the left STS or inferior parietal lobe) to be more strongly activated in deaf than hearing non-signers. Although deaf, but not hearing, people showed significant activation in the left IFG in the communicative-scrambled contrast, this did not emerge as a significant between-group difference. This lack of language network engagement contrasts with prior studies of deaf native signers, and suggests that stronger activation of language areas for non-linguistic gesture seen in those previous studies were largely driven by

sign language experience. Related to this observation were our predictions stemming from prior findings that deaf signers do not show activation of the frontal-parietal action perception network when viewing pantomime (Corina et al. 2007; Emmorey et al. 2010; Husain et al. 2012). Corina and colleagues had proposed that deaf signers possess a gating mechanism whereby early stages of visual processing identify and filter communicative (or potentially communicative) biological motion from other types of motion for processing by the language network. Since our deaf participants were not signers, we expected to see the action perception network engaged by our biological motion stimuli, and this was confirmed: both deaf and hearing people showed robust activation of the inferior parietal lobe, IPS, premotor cortex, and IFG. This finding confirms that auditory deprivation alone does not induce such a gating system, even though deafness may sensitize certain cortical areas to communicative gestures (i.e., the left STS and right parietal lobe).

A further prediction was that deaf non-signers might show patterns of sensitivity that were different both from those previously reported for either deaf signers or hearing non-signers. Specifically, we predicted reduced differences in activation between communicative (emblems) and non-communicative (pantomimed) gestures in deaf non-signers. This was based on the reasoning that, living in a world where most of their interactions were with hearing people using a combination of speech and gesture to communicate, deaf non-signers would develop increased sensitivity within biological motion and action perception networks due to the relatively greater importance of nonverbal communication. This hypothesis was confirmed, in that while hearing people showed stronger activation for communicative than non-communicative gestures in temporal, parietal, and frontal ROIs, deaf people showed no significant differences in activation between these two stimulus types. This was true in spite of the fact that relative to scrambled

control stimuli, both deaf and hearing people showed similar and extensive activation for both communicative and non-communicative stimuli. In other words, the absence of communicative–non-communicative differences in deaf people was not because they didn't show any activation to either stimulus — rather, they showed equivalent (and significant) activation levels to both.

Before turning to individual differences, we note that besides the left STS, the only other brain area in which deaf people showed stronger activation than hearing people (in the communicative–scrambled contrast) was in the right inferior parietal lobe, including the AG and IPS. This is interesting because this region has previously been implicated in numerous neuroimaging studies of deaf people. In a previous study (Newman et al. 2002) we found that the right AG was the only area in the brain that showed "critical period" effects for sign language acquisition: this area was activated by sign language perception (relative to meaningless gesture control stimuli) in native signers but not in fluent signers who learned the language as adults. Other studies have also indicated a greater role for the parietal cortex in signed than spoken language (Emmorey et al. 2014), and in particular for aspects of sign language (motion and location classifier constructions) that involve encoding space in literal or metaphoric ways (Emmorey et al. 2013). Further, Bavelier and colleagues have shown neuroplastic changes in the IPS associated with deafness: the IPS is activated more strongly by attended, peripheral visual motion in deaf than hearing people (Bavelier et al. 2001), and also shows stronger functional connectivity (correlated activity) with V5 in deaf than hearing people (Bavelier et al. 2000). The present results add to these data, suggesting that both sign language and auditory deprivation may contribute to neuroplastic changes in the right parietal lobe. On the one hand, hearing people who learn sign language early, but not later, in life show activation here(Newman et al. 2002), but on the other hand the present study showed activation for deaf people who were not

fluent signers. Further, not all of our participants became deaf early in life, and the region of the right parietal lobe that showed stronger activation for deaf people did not show a significant correlation with age of onset (nor duration) of deafness. Theories behind the functional role of the parietal cortex in action perception generally, and sign language in particular, have included motor preparation and, relatedly, a mechanism for understanding the meaning of others' movements (or their goals) in terms of one's own movement patterns (Emmorey et al. 2013; 2014). Based on the present evidence we suggest that activation in the right AG/IPS region must reflect a type of sensitivity to communicative biological motion that may be malleable in different ways at different ages. Although our previous work suggested a sensitive period for sign language exposure only early in life, the present data suggest that the right inferior parietal lobe may become sensitized to manual, non-linguistic communication even later in life, when deafness forces a greater reliance on nonverbal communication.

Our final hypotheses concerned individual differences among deaf people based on the age at which they became profoundly deaf, and the duration of their deafness. Both of these variables have been shown to influence the extent of neuroplastic changes in both humans and other animals (Kral and Sharma 2012), with a general consensus that across species, sensitive periods exist early in life — if auditory stimulation is not provided during these periods, normal development does not occur and later auditory restoration (i.e., with cochlear implants) is much less effective. However, to our knowledge no previous studies have investigated the influence of age or onset or duration of deafness on biological motion perception. Our results show that both of these factors do influence the sensitivity of the biological motion and action perception networks to human movements — independently of chronological age — and further that this is specific to meaningful, communicative gestures, as no such effects were seen for non-

communicative (pantomimed) actions. In all regions that showed significant correlations, the pattern of the relationship was the same: earlier onset of deafness, and longer duration, were associated with stronger activation of these brain regions in response to communicative gestures.

It is interesting to compare the set of regions showing these correlations with the network activated across our group of deaf participants as a whole in the same communicative–scrambled contrast. Some areas that showed significant correlations were also activated overall in the group contrast — including bilateral fusiform gyri, right precentral gyrus, bilateral AG, left STSp, bilateral temporal-occipital parts of the MTG, and bilateral V5. These areas were all activated in hearing people as well, and — with the exception of the right AG — showed no overall differences in activation between the groups. Therefore we interpret this set of results as indicating that earlier onset and longer periods of deafness lead to enhanced sensitivity to communicative biological motion within a subset of the regions normally associated with biological motion and action perception.

Additionally, there were other areas that were not present in the communicative-scrambled contrast across all deaf (or hearing) people, but which showed significant individual differences associated with onset and duration of deafness. These included the right IFG, left SMG, and the right anterior and left posterior MTG. This suggests that these regions are largely recruited for communicative biological motion perception only with prolonged and/or relatively early onset deafness. In examining the relationships with age of onset of deafness shown in Figure 4, it can be seen that the participants cluster into a subgroup comprising those who became deaf at age 10 or earlier; the remaining participants became deaf after age 30, with none in between those ages. Notably, a general pattern among these regions is that the early-deafened people tend to show positive communicative–scrambled differences, whereas the older-deafened

subgroup tend to show only weakly positive, or even negative contrast values. Thus although the data were well-described by a linear function of age (or duration), it is possible that there is a more qualitative, nonlinear sensitive period effect at play. Although prior studies have suggested that sensitive periods for auditory processing occur in early childhood (Sharma et al. 2005), the present data suggest that sensitive periods for biological motion perception pathways may be more prolonged, stretching into adolescence and even early adulthood.

The high correlation in our subject group between age of profound loss and duration of deafness makes it difficult to distinguish these two effects. Although most of the people with long durations of deafness also had profound loss beginning in childhood, three people with long duration of deafness (> 15 years) had only become deaf after age 30. However, careful examination of the scatterplots in Figure 4 suggest that the intercepts for duration of loss occur around 20 years, and so the effect of duration of loss may well be largely driven by shared variance with age of profound loss. Obtaining a sample of deaf people with uniform and uncorrelated distributions of age of loss and duration of deafness would help clarify this issue, however in practice our experience suggests that this may be challenging; increasing proportions of children are receiving cochlear implants soon after diagnosis of profound deafness, and also relatively few people become deaf in early adulthood compared to in childhood or in later adulthood.

It is notable that the right IFG showed a correlation with age of onset and duration of deafness, as we have previously found this area active for sign language (Newman et al. 2002), especially for sentences using complex sign language morphology including syntactic constructions that make use of space (Newman, Supalla, Hauser, Newport, and Bavelier 2010a). Its activation here suggests that it may assume a role in symbolic manual communication

independent of language, at least with early onset/long duration of deafness. The right anterior MTG is an area of interest because not only have we found it consistently active for sign language sentence processing in both native and late learners (Newman et al., 2002; 2015; Newman, Supalla, Hauser, Newport, & Bavelier, 2010a; 2010b), but it is the one area in which we previously found overlapping activation in both deaf signers and hearing non-signers, when they tried to interpret both sign language and communicative gesture (Newman et al., 2015). Similarly, Andric and colleagues (Andric et al. 2013) found activation in this area in hearing non-signers for communicative gestures (emblems) but not grasping actions, consistent with a specialization for meaningful symbolic manual communication. The present results are consistent with this interpretation, and extend it to suggest that this region may become increasingly sensitized to such information by early onset and/or prolonged periods of deafness.

The other two areas that showed sensitivity to age of onset and duration of deafness were in the left hemisphere. One cluster of correlation included the left SMG, STSp, and temporal-occipital MTG. This corresponds with areas typically associated with biological motion perception. However, included in this cluster was part of the left SMG, which was not active for the communicative-scrambled contrast in either deaf or hearing people. Notably, this area is typically associated with phonemic perception in both hearing and deaf people (Corina et al. 1999; Hickok and Poeppel 2000); we speculate that in the absence of either signed or spoken language phonemic input, the left SMG may develop a sensitivity to the segmental properties of manual gestures (subcomponents such as hand position, orientation, and movement). The left posterior STG also showed sensitivity to age of onset/duration of deafness in spite of not being activated by communicative gestures in either group alone. As with the stronger activation in a more anterior portion of the left STG that was obtained for all deaf people relative to hearing for

communicative stimuli, we note that these posterior parts of the STG are also typically involved in speech perception and so this result may represent greater neuroplastic reorganization for nonverbal, symbolic communication in people who become deaf earlier in life (or have been deaf for prolonged periods of time).

Conclusion

We investigated how long-term deafness, in the absence of sign language use, affects the neural networks engaged by communicative and non-communicative biological motion. We found that, in contrast to previous studies using deaf native signers, deaf non-signers engage the occipito-temporal biological motion perception and frontal-parietal action perception networks in ways largely similar to hearing non-signers. However, there were some exceptions to this similarity. While hearing people showed stronger activation within these networks for communicative than non-communicative gestures, deaf people showed equivalent levels of activation, suggesting that auditory deprivation increases the sensitivity of these networks to any biological motion, irrespective of its communicative content. As well, we found that two brain areas — the left STG, typically associated with speech processing, and the right inferior parietal lobe (AG and IPS), typically associated with motion, spatial, and gesture perception — showed enhanced activation in deaf people relative to hearing people, specifically for communicative stimuli. This suggests that, although there are reduced differences between responses to communicative and non-communicative stimuli in deaf people, deafness creates an increased sensitivity of specific language- and gesture-related areas to gestures with established meanings. Finally, because we recruited deaf people with a range of histories of hearing loss, we were able to examine the relationships between age of profound deafness, and duration of deafness, with brain activation for biological motion. We found that earlier onset and longer duration of loss

were associated with increasing sensitivity specifically to communicative gestures, in a widespread network that included much of both the biological motion and action perception networks, and also left hemisphere areas typically associated with speech processing. The effects in these latter areas suggest that with auditory deprivation, areas typically specialized for language processing can become recruited for visual-manual symbolic communication even when a formal sign language is not known. This in turn suggests that the specialization of these regions may not be specific to structured natural human languages (i.e., with segmental, rule-governed syntactic and phonetic systems), but to symbolic communication and learned form-meaning associations more generally (e.g., “thumbs-up” means “good”). Finally, the fact that these effects of deafness were not restricted to people who were pre-lingually deafened, but rather showed linear effects with age of deafness, suggests that these neuroplastic changes may be able to occur at least throughout childhood and adolescence, rather than being restricted to a relatively narrow sensitive period in early childhood. This finding in particular has important implications for our understanding of neuroplasticity across the lifespan.

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Footnotes

[1] All but two participants had the same age of diagnosed profound loss in both ears. Those two people with differing age of loss in the two ears had 1 and 3 years of unilateral loss, respectively, and both had < 1 year bilateral loss. We did not deems these differences sufficient to warrant investigating the effects of unilateral loss separately from those of bilateral loss.

[2] Although for all three stimulus types (communicative, non-communicative, and scrambled) relative to fixation, hearing people showed stronger activation in right V5 than deaf people, this was also seen in left V5 (albeit with more restricted spatial extent) and did not emerge as a group difference when comparing biological motion to scrambled control stimuli.

[3] Note that these are qualitative comparisons; we did not statistically test laterality but there is no evidence for a trend towards left-lateralization in either group.

[4] We note that there is an apparent contradiction in the data, whereby deaf people did not show significant differences in the direct contrast of communicative versus non-communicative gestures, but did show stronger activation than hearing people for communicative versus scrambled stimuli in the left STG and right AG/IPS. This likely reflects the nature of the different contrasts: as can be seen in Figure 2, neither the left STG nor right AG/IPS were activated in the non-communicative-scrambled contrast. However, these areas may have had sub-threshold levels of activation such that the difference in the direct comparison between communicative and non-communicative stimuli did not reveal significant differences in these areas.

Tables

Table 1: Characteristics of individual deaf participants.

Subject ID	Subject ID	Sex	Handedness	Age	Age of Profound Loss	Duration of Deafness	Cause of Hearing Loss	Sign Language Knowledge (0-4)
CI09-08	1	F	R	45	0	45	Rubella, progressive	0
CI09-22	2	F	R	33	0	33	Congenital hyperbilirubinemia	0
CI09-24	3	F	R	50	10	40	Unknown from childhood	0
CI09-25	4	M	R	67	59	8	Blow to head/noise	0
CI09-34	5	M	R	62	45	17	Unknown progressive	0
CI09-36	6	M	R	61	48	13	NF2	1
CI09-37	7	M	L	49	7	42	Mumps	0
CI09-40	8	M	R	66	9	57	Meningitis	1
CI09-41	9	M	R	59	34	25	Meniere's disease	0
MLD-08	10	F	R	49	46	3	Mumps	0
MLD-09	11	F	R	47	46	1	Hereditary progressive	2
MLD-101	12	F	L	19	0	19	Hereditary	3
MLD-102	13	F	R	51	49	2	German measles	3
MLD-103	14	M	R	70	40	30	Meniere's disease	0

F, female; M, male; R, right; L, left; for sign language knowledge participants rated their ability to produce and understand sign language on a scale from 0–4, defined as: 0 = not at all; 1 = hardly; 2 = sufficiently; 3 = well; 4 = perfectly.

Table 2: Summary of significant clusters of voxels that were correlated with either age of onset of profound hearing loss, or duration of profound loss, within the group of deaf participants. Cluster size (in terms of number of 2 x 2 x 2 mm voxels), maximum z value within the cluster, and MNI coordinates of the voxel with the maximum z value, for each of the a priori-defined regions of interest (ROIs) in which a significant cluster of activation was detected.

		Age of Profound Loss					Duration of Profound Deafness				
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
IFG	R	12	2.60	50	10	50	72	3.45	52	8	50
Precentral	L						40	2.82	-14	-20	42
	R	434	3.45	32	-8	40	502	3.71	60	10	42
SMG	L	170	2.88	-56	-48	22	287	3.25	-60	-42	12
	R	35	2.82	48	-40	60					
Angular Gyrus	L	254	3.38	-50	-60	32	211	3.12	-50	-56	30
	R	17	2.85	62	-64	6					
STSp	L	54	3.10	-52	-50	20	165	3.02	-58	-52	8
MTGa	R	33	2.82	54	2	-32	90	3.24	50	2	-34
MTGp	L	147	3.44	-64	-36	-18	188	3.04	-54	-42	-12
	R	45	2.73	56	-38	-12					
MTGto	L						93	3.02	-58	-52	8
	R	89	3.17	68	-56	4					
Fusiform	L	779	4.09	-36	-60	-18	653	3.95	-36	-56	-20
	R	708	4.12	34	-60	-12	412	3.27	38	-44	-20
V5	L	191	3.84	-38	-68	-10	146	3.35	-48	-76	-12
	R	33	3.00	58	-68	6					

Abbreviations: hemi, hemisphere; A1, primary auditory cortex; IFG, inferior frontal gyrus; precentral, precentral gyrus; SMG, supra marginal gyrus; IPS, intra-parietal sulcus; STGa, superior temporal gyrus – anterior part; STGp, superior temporal gyrus – posterior part; MTGa, middle temporal gyrus – anterior part; MTGp, middle temporal gyrus – posterior part; MTGto, middle temporal gyrus – temporal-occipital part; STSa, superior temporal sulcus – anterior; STSp, superior temporal sulcus – posterior; fusiform, fusiform gyrus; V5, human motion-sensitive cortex (hMT).

Figure Legends

Figure 1: Significant areas of activation for each stimulus condition relative to the fixation baseline, for each group. The conjunction map represents voxels that were significantly active in both groups (i.e., the overlap between the hearing and deaf groups). See Methods for details of how these maps were generated.

Figure 2: Top, activation maps for the contrasts of each biological motion condition versus scrambled point-light control stimuli, within each group. Bottom, areas that showed significant between-group differences in the contrasts between biological motion and control stimuli. No significant differences were seen for the non-communicative versus scrambled contrast, nor for hearing people relative to deaf people.

Figure 3: Areas showing significant correlations between BOLD fMRI signal in the communicative–scrambled contrast, and age of profound deafness (top) and duration of deafness (bottom) in deaf participants.

Figure 4: Scatterplots with regression lines of the correlations between BOLD fMRI signal for the communicative–scrambled contrast from each participant, and age of profound hearing loss (left panels) and duration of deafness (right panels). Within each pair of panels, data from the ROI in each hemisphere are plotted separately (L, left, and R, right). Shaded areas are 95% confidence intervals of the linear model fit. For the purposes of illustration in these plots, BOLD fMRI activation was derived as the average parameter estimates from the communicative-scrambled contrast for each participant, across all voxels within each ROI that showed a significant correlation at the group level.

Figure 1: Significant areas of activation for each stimulus condition relative to the fixation baseline, for each group. The conjunction map represents voxels that were significantly active in both groups (i.e., the overlap between the hearing and deaf groups). See Methods for details of how these maps were generated.

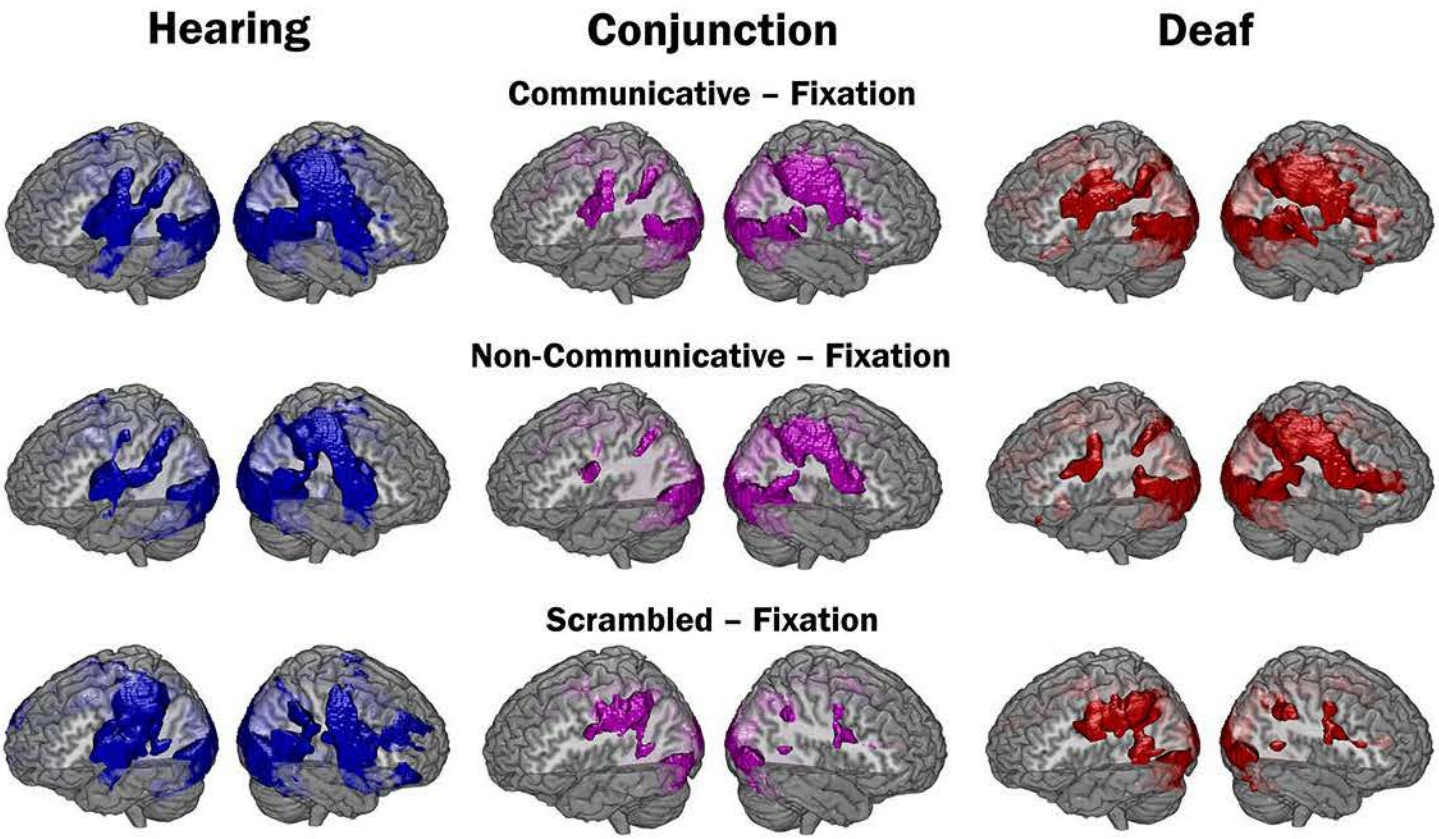


Figure 2: Top, activation maps for the contrasts of each biological motion condition versus scrambled point-light control stimuli, within each group. Bottom, areas that showed significant between-group differences in the contrasts between biological motion and control stimuli. No significant differences were seen for the non-communicative versus scrambled contrast, nor for hearing people relative to deaf people.

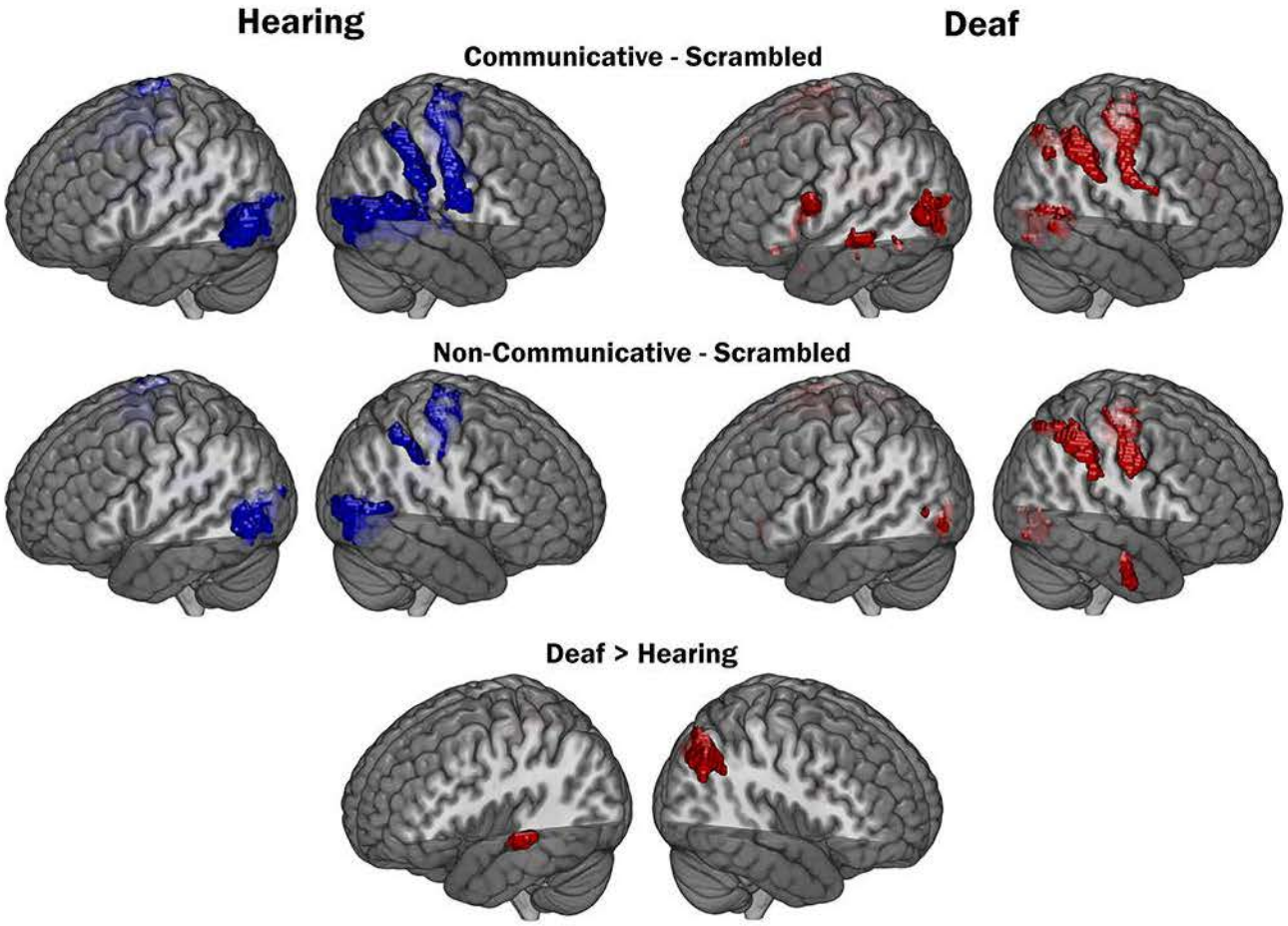


Figure 3: Areas showing significant correlations between BOLD fMRI signal in the communicative–scrambled contrast, and age of profound deafness (top) and duration of deafness (bottom) in deaf participants.

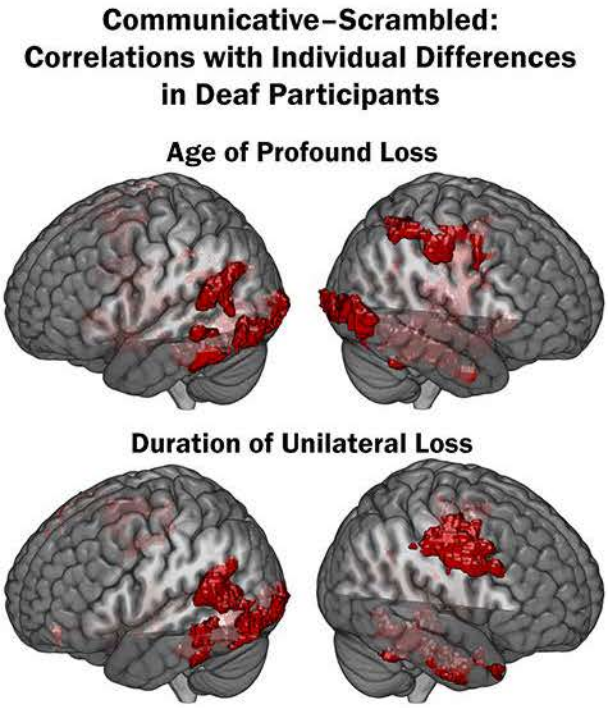
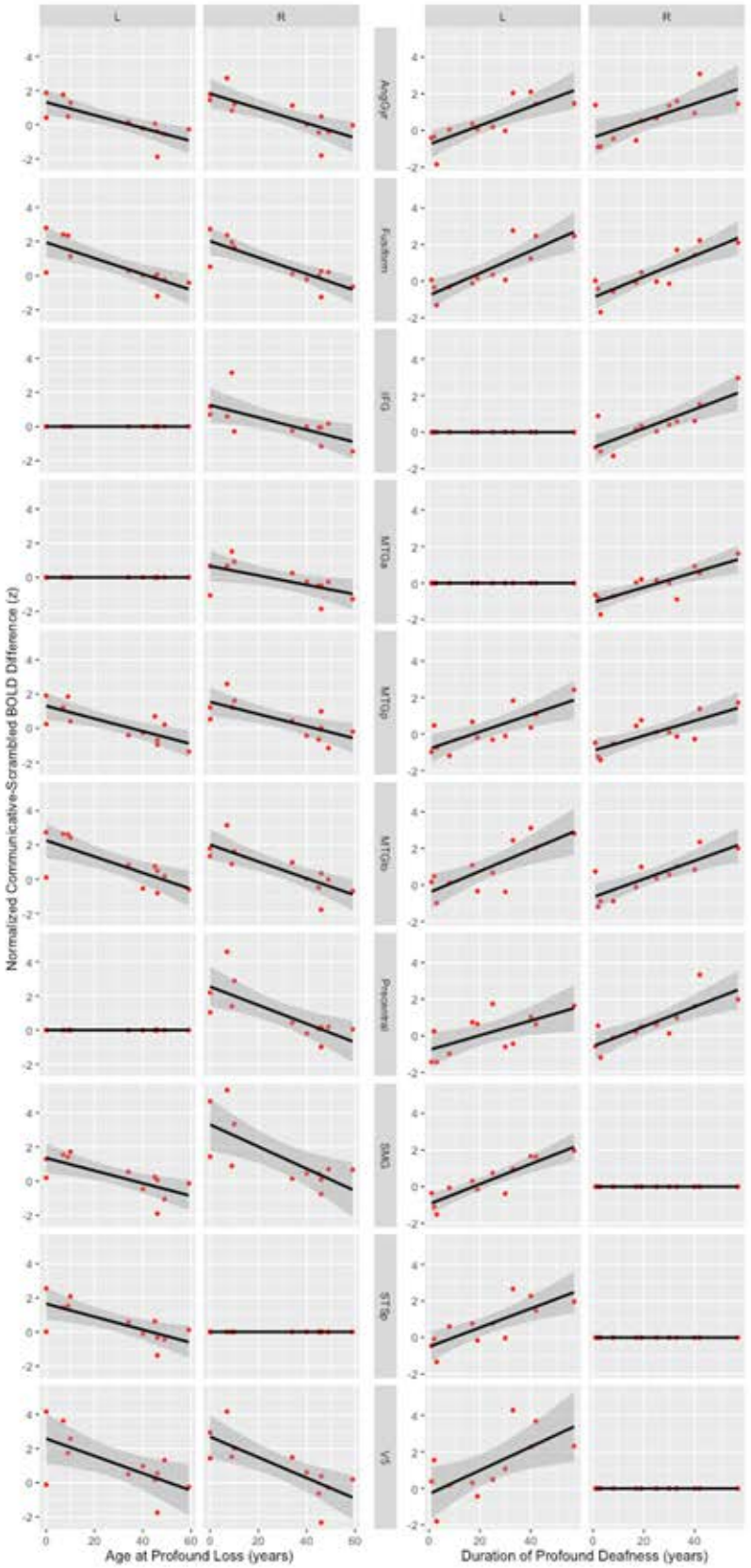
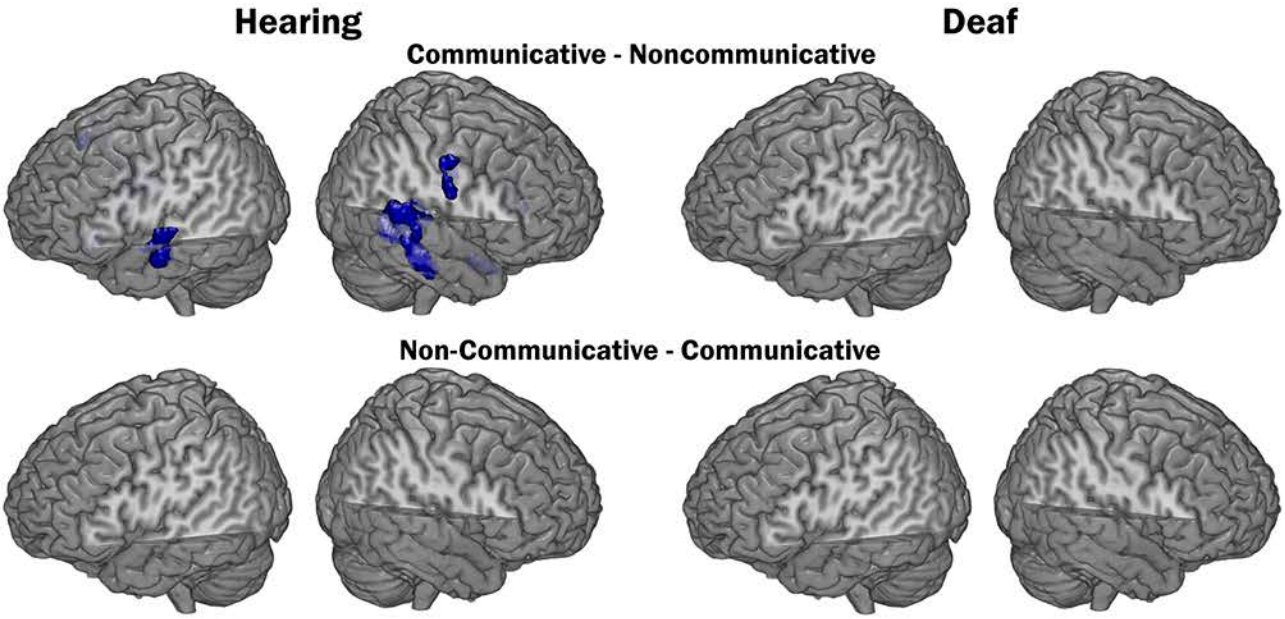


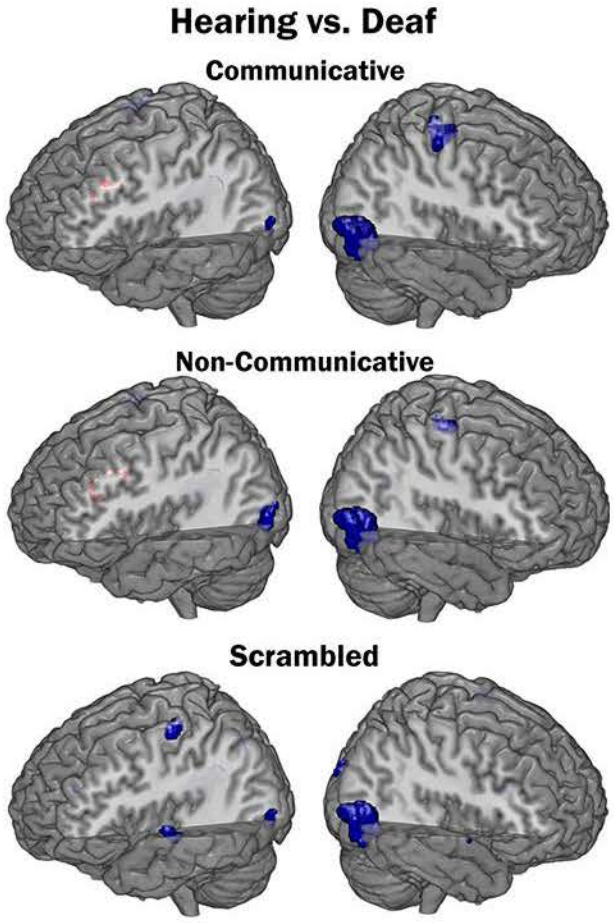
Figure 4: Scatterplots with regression lines of the correlations between BOLD fMRI signal for the communicative vs. scrambled contrast from each participant, and age of profound hearing loss (left panels) and duration of deafness (right panels). Within each pair of panels, data from the ROI in each hemisphere are plotted separately (L, left, and R, right). Shaded areas are 95% confidence intervals of the linear model fit. For the purposes of illustration in these plots, BOLD fMRI activation was derived as the average parameter estimates from the communicative-scrambled contrast for each participant, across all voxels within each ROI that showed a significant correlation at the group level.



Supplementary Figure S2: Statistical maps for the direct comparisons between communicative and non-communicative point-light biological motion, within each group.



Supplementary Figure S1: Areas of significant difference in activation between hearing and deaf people within the set of pre-defined regions of interest (ROIs), for the contrasts of each stimulus condition versus fixation baseline. Greater activation for hearing people are shown in blue; no areas showed significantly greater activation for deaf than hearing people in these contrasts.



Supplementary Table 1: Summary of results from the contrasts of each stimulus condition versus the fixation baseline, for each group. Cluster size (in terms of number of 2 x 2 x 2 mm voxels), maximum z value within the cluster, and MNI coordinates of the voxel with the maximum z value, for each of the a priori-defined regions of interest (ROIs) in which a significant cluster of activation was detected. Abbreviations: hemi, hemisphere; A1, primary auditory cortex; IFG, inferior frontal gyrus; precentral, precentral gyrus; SMG, supra marginal gyrus; IPS, intra-parietal sulcus; STGa, superior temporal gyrus – anterior part; STGp, superior temporal gyrus – posterior part; MTGa, middle temporal gyrus – anterior part; MTGp, middle temporal gyrus – posterior part; MTGto, middle temporal gyrus – temporal-occipital part; STSa, superior temporal sulcus – anterior; STSp, superior temporal sulcus – posterior; fusiform, fusiform gyrus; V5, human motion-sensitive cortex (hMT).

Communicative vs. Fixation

		Hearing					Deaf				
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
A1	L	81	3.43	-56	2	-8					
	R	117	4.08	50	-30	6					
IFG	L	1589	5.38	-38	4	22	839	5.07	-54	6	32
							414	4.74	-40	26	-8
	R	1931	6.66	38	8	26	1160	4.27	44	6	50
Precentral	L	2367	5.84	-38	4	22	1184	5.01	-58	6	34
	R	4949	8.73	52	-4	48	2399	6.13	42	-6	58
SMG	L	459	3.27	-54	-30	38	423	3.84	-58	-20	38
	R	2340	6.79	52	-26	50	951	5.76	50	-30	52
							377	4.38	62	-44	8
Angular Gyrus	L	174	8.97	-50	-76	8	446	6.05	-32	-68	52
							263	5.99	-54	-68	8
	R	1082	9.13	48	-80	8	184	5.85	32	-70	52
							84	3.54	48	-50	6
IPS	L	890	6.75	-30	-60	48	1151	5.15	-28	-70	44
	R	1308	6.66	32	-56	54	1380	5.73	44	-40	56
Temporal Pole	L	235	4.17	-60	12	-12					
	R	467	3.95	60	16	-6					
STGa	L										
STGp	L										
	R	359	5.37	52	-36	2	193	3.88	56	-36	4
MTGp	L	267	4	-66	-40	-12	88	3.18	-54	-44	-2
	R	172	6.57	50	-38	-2	149	3.32	56	-36	-2
MTGto	L	1002	9.8	-50	-62	0	1062	4.87	-48	-62	0
	R	1272	12.8	48	-60	0	828	4.13	52	-60	0
STSa	L	285	3.57	-60	4	-8					
STSp	L	577	10.2	-48	-66	4	639	6.64	-52	-66	4
	R	481	9.5	52	-56	2	330	3.32	52	-58	4
Fusiform	L	2006	5.93	-36	-78	-8	1278	4.64	-42	-70	-18
	R	2450	8.09	36	-70	-4	1811	5.04	38	-74	-16
V5	L	877	11.3	-48	-78	0	774	5.49	-48	-76	-2
	R	1000	14.6	48	-72	-2	793	5.56	44	-74	0

Non-Communicative vs. Fixation

		Hearing					Deaf				
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
A1	L										
	R										
IFG	L	1057	4.61	-40	4	22	765	5.39	-54	6	32
							100	4.27	-38	26	-10
	R	1769	5.57	38	10	24	1258	3.95	48	16	26
Precentral	L	1380	4.96	-40	4	22	860	4.7	-54	6	34
	R	4043	7.44	36	-16	64	2603	5.17	42	-6	58
SMG	L	370	3.19	-44	-38	30	283	3.66	-64	-54	12
	R	1491	5.87	66	-44	18	1675	5.2	62	-40	14
Angular Gyrus	L						109	4.79	-26	-72	52
	R	994	9.4	48	-80	8	89	5.21	32	-68	52
IPS	L	855	5.7	-26	-62	48	674	4.73	-40	-54	54
	R	1349	6.28	32	-60	54	1381	5.05	42	-40	56
Temporal Pole	L										
	R	56	4.16	60	16	-6					
STGa	L						21	3.82	66	-4	2
STGp	L						56	3.61	-60	-42	10
	R						39	3.92	56	-36	4
MTGp	L										
	R										
MTGto	L	389	7.6	-48	-62	0	699	4.67	-50	-62	0
	R	1036	12.5	48	-60	0	717	4.47	54	-60	0
STSa	L										
STSp	L	390	8.43	-48	-64	2	417	5.92	-52	-66	4
	R	445	9.45	52	-56	2	321	3.63	52	-58	4
Fusiform	L	2305	7.14	-36	-78	-8	1504	5.09	-38	-64	-20
	R	2487	8.58	36	-70	-4	1823	5.09	34	-74	-2
V5	L	877	12.6	-46	-80	0	719	5.1	-48	-76	-4
	R	999	14.7	48	-72	-2	835	5.77	46	-76	0

Scrambled vs. Fixation

		Hearing					Deaf				
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
A1	L	425	4	-60	-4	0					
	R										
IFG	L	754	4.27	-64	6	4	120	4.88	-52	6	34
	R	1863	6	40	8	26	809	3.82	44	6	46
Precentral	L	3593	5.97	-36	-22	64	953	4.75	-54	6	34
	R	2527	6.87	40	8	26	517	3.84	40	0	46
SMG	L	619	4.69	-52	-30	44	1561	4.97	-48	-46	50
	R	1077	5.34	66	-44	18	383	5.23	64	-38	16
							67	3.47	52	-28	46
Angular Gyrus	L										
	R	372	6.63	46	-58	6	203	4.72	-32	-68	50
		275	9.94	46	-80	8					
IPS	L	773	4.91	-30	-60	48	1474	5.6	-48	-44	50
	R	1447	5.01	30	-68	30	1274	3.96	30	-62	48
Temporal Pole	L	104	4.31	-60	12	-12					
	R	330	4.9	62	14	-4					
STGa	L	94	4.43	-20	-30	-6					
STGp	L						288	3.54	-56	-40	12
	R	56	3.98	66	-36	16	73	4.4	62	-34	16
MTGp	L										
	R										
MTGto	L	24	4.86	-48	-62	0	95	3.78	-48	-62	0
	R	947	11.8	48	-60	0					
STSa	L	181	3.68	-60	0	-6					
STSp	L						83	4.63	-52	-66	4
	R	389	8.22	52	-56	2					
Fusiform	L	1677	6.77	-10	-88	-16	1185	3.98	-30	-66	-20
	R	2656	7.52	22	-78	-16	447	4.03	30	-78	-22
							66	3.15	44	-60	-26
V5	L	735	8.99	-46	-80	0	451	3.63	-48	-76	-4
	R	981	12.9	48	-72	0	549	5.03	46	-76	0

Supplementary Table 2: Summary of clusters within ROIs that showed significant differences between groups, for the contrasts of each stimulus condition with fixation. No areas showed significantly greater activation for deaf people. Abbreviations are as for Supplementary Table 1.

Hearing > Deaf						
Communicative vs. Fixation						
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
Precentral	R	216	4.91	36	-16	64
V5	L	24	3.57	-44	-82	4
	R	627	4.73	50	-70	-4
Non-Communicative vs. Fixation						
Precentral	R	76	4.63	36	-14	64
V5	L	181	4.11	-44	-82	4
	R	673	5.09	48	-70	-2
Scrambled vs. Fixation						
A1	L	50	3.35	-60	-4	2
Precentral	L	62	4.04	-42	-14	60
SMG	R	31	3.67	66	-48	26
V5	L	38	3.22	-40	-82	0
	R	612	4.54	46	-68	-2

Supplementary Table 3: Summary of significant clusters of activation within ROIs, for the contrast of each biological motion condition versus scrambled point-light motion, for each group, as well as for the comparisons between groups. No significant clusters were identified for hearing relative to deaf participants. Abbreviations are as for Supplementary Table 1.

Communicative - Scrambled

		Hearing					Deaf					Deaf > Hearing				
		Voxel					Voxel					Voxel				
ROI	Hemi	Count	Max. *z*	x	y	z	Count	Max. *z*	x	y	z	Count	Max. *z*	x	y	z
A1	R	185	4.37	50	-30	6										
IFG	L						340	3.00	-48	14	12					
Precentral	R	2857	8.23	36	-16	64	1132	4.93	36	-6	62					
							54	3.05	10	-22	72					
SMG	R	790	7.15	52	-26	50	450	5.02	46	-38	56	34	4.04	54	-54	44
		345	6.22	56	-44	8										
Angular Gyrus	L	204	4.73	-56	-64	6	174	4.51	-52	-74	10					
	R	736	5.66	60	-62	4	234	4.94	32	-70	54	696	4.23	54	-54	42
Temporal Pole	L						36	4.12	-38	14	-28					
STGp	R	298	5.89	48	-36	0	167	2.93	-62	-22	-6	229	3.68	-70	-16	0
MTGp	R	151	6.60	48	-38	-2										
MTGto	L	953	6.24	-58	-62	0	207	3.73	-60	-62	8					
	R	1133	6.82	52	-60	0	478	3.27	48	-48	4					
STSp	L	483	5.45	-54	-64	2	220	4.27	-56	-66	8					
	R	470	5.43	52	-56	2	162	3.08	48	-48	4					
IPS	R	159	7.90	44	-40	56	253	5.98	44	-40	56	226	4.19	40	-64	44
Fusiform	L	826	6.13	-40	-44	0	120	4.37	-44	-68	-18					
	R	742	5.81	40	-44	-6	309	3.88	40	-70	-16					
V5	L	812	5.95	-54	-72	0	420	3.59	-48	-76	0					
	R	966	7.95	50	-72	-6	436	3.72	50	-72	-12					

Non-Communicative - Scrambled

		Hearing					Deaf					Deaf > Hearing				
		Voxel					Voxel					Voxel				
ROI	Hemi	Count	Max. *z*	x	y	z	Count	Max. *z*	x	y	z	Count	Max. *z*	x	y	z
Precentral	R	1370	7.1	36	-16	64	748	3.85	42	-20	60					
SMG	R	222	4.96	52	-26	50	448	4.42	46	-40	60					
AngGyr	L	41	4.36	-44	-86	12										
	R	150	4.7	48	-58	6	115	4.39	32	-72	56					
STSa	R						62	3.02	58	-4	-20					
MTGa	R						161	3.27	64	4	-32					
MTGto	L	449	4.77	-50	-62	-2										
	R	549	5.91	50	-60	0										
IPS							92	5.07	44	-40	56					
STSp	L	393	4.32	-48	-62	0										
	R	277	5.25	52	-56	2										
Fusiform	L	1174	7.35	-38	-46		236	4.93	-42	-46	-26					
	R	775	5.62	40	-48		315	3.94	40	-62	-20					
V5	L	886	6.87	-48	-78	-8	251	3.69	-48	-76	-12					
	R	969	7.28	50	-72	-6	272	3.18	42	-70	-8					

Supplementary Table 4: Summary of significant clusters of activation for the direct contrast of communicative versus non-communicative biological motion. There were no significant clusters for this contrast within the deaf group. Abbreviations are as for Supplementary Table 1.

Communicative vs. Non-Communicative						
Hearing						
ROI	**Hemi**	**Voxel Count**	**Max. *z***	**x**	**y**	**z**
A1	L	98	3.91	-56	0	-10
	R	133	3.94	56	-26	8
	R	38	3.23	42	-32	14
Precentral	R	135	3.77	64	-2	32
SMG	R	225	4.12	62	-32	10
Temporal Pole	R	134	4.16	28	10	-28
STGp	R	461	4.21	54	-36	2
MTGa	L	171	3.4	-60	-2	-12
MTGp	R	467	4.46	52	-38	-2
MTGto	R	273	3.86	60	-40	-2
STSa	L	382	3.53	-58	-4	-8
	R	32	3.28	64	-16	-16
STSp	R	42	3.47	56	-44	4