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ARTICLE

What is the function of auditory cortex without auditory input?

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Commentary On Ding et al., 2015 for *Brain* - 2014-01833.R2

When a sensory input is absent during development, regions of the brain usually dedicated to processing input from that modality can be engaged to process input from a replacement sense. This is referred to as crossmodal plasticity, and studying it can provide rich and unique insights into the biological versus environmental constraints that act on brain development and brain function (Merabet and Pascal-Leone, 2010). For example, numerous studies report greater activation of typically 'auditory association cortices' in the superior temporal gyrus (STG), sulcus and planum temporale, in those born profoundly deaf than in their hearing peers when processing visual or somatosensory input (e.g, Karns et al., 2012). There is also a wealth of research reporting enhanced *behavioural* performance on visuo-spatial tasks in deaf versus hearing participants (Bavelier et al. 2006). Although it is tempting to make the intuitive assumption that these two findings must be linked, no studies with humans have yet demonstrated a clear link between the extent of crossmodal plasticity in auditory cortices in those born deaf and enhanced performance on visuo-spatial tasks. This is the question addressed by Ding and co-workers in this issue of *Brain* (2015).

In their study, Ding e*t al*. asked hearing and congenitally deaf participants to perform a visuo-spatial working memory task while fMRI data were collected. Deaf participants showed faster responses than hearing participants, although there was no group difference in task accuracy. In support of previous studies, Ding *et al*. report evidence of crossmodal plasticity (greater activation in deaf than hearing participants) in auditory association areas. The novel finding in their work is that deaf participants showed greater activation than hearing participants in auditory association regions, not only when complex visual stimuli were

displayed, but also during the *maintenance phase*, during which only a static crosshair was visible on the screen. They also report correlations between amplitude of response in STG and task performance in deaf but not hearing participants, and argue therefore that auditory association cortices play an important role in visuo-spatial working memory in those born deaf.

This is an interesting finding and one that highlights the critical question for future research in this field – what is the *specificity* of the link between crossmodal plasticity and enhanced processing of non-auditory inputs in those born deaf? In research with congenitally deaf cats, Lomber *et al*., (2010) have made impressive progress in localizing specific visual functions (e.g., localization and motion detection) to discrete regions of the auditory cortex. They have shown that after sensory deprivation, cortices maintain their higher order function – regardless of sensory input. For example, the portion of the auditory cortex that processes auditory localization in hearing cats is sensitive to *visual* localization in cats that were born deaf. Furthermore, the recruitment of this region leads to better performance on visual localization tasks. Similarly, Ding *et al*. argue that in deaf humans functional specialization of the posterior STG for spatial processing is maintained following early auditory deprivation.

But how specific is this link? The relationship between STG activation and some measures of behavioural performance in deaf but not hearing participants is key to the Ding *et al*. argument. However, a significant difference in strength of correlation between deaf and hearing groups was not reported and would have offered stronger support for this position. More importantly, numerous previous studies of deaf individuals have shown activation in parts of STG in response to a wide range of visual and somatosensory stimuli and tasks. Notably, the few studies that have *not* reported crossmodal plasticity in STG have used passive stimulus presentation with no task requirements (e.g. Hauchal et al., 2014). This raises the possibility that allocation of attention is a critical factor in the recruitment of this region. Some support for this position comes from the finding that crossmodal responses in STG are found during timeframes that correspond to higher order processing, and not early sensory processing (Leonard et al., 2012).

Future studies of crossmodal plasticity need to set out clear predictions about the involvement (or not) of particular portions of the STG in a range of cognitive skills and across modalities in those born deaf. For example, a basic prediction from the Ding *et al*. conclusion is that activation in posterior STG would not differ between deaf and hearing participants when presented with a simple visual crosshair. However, activation *would* differ if visuo-spatial maintenance was required during the display, as in the current study.

A fundamental question for understanding cortical plasticity is to establish whether it is driven by bottom up or top down input received by the 'deprived' cortex. Ding and co-workers argue that their data lend support to a top-down mechanism rather than the typically assumed 'bottom-up' process. This conclusion is based on Granger causality analysis, which was used to examine functional connectivity between brain regions. The results showed increased connectivity from the frontal eye fields, known to be involved in working memory tasks, to the STG. Ding *et al*. argue against a bottom-up mechanism since there was no difference between deaf and hearing groups in connectivity between V1 and STG. However, it can be argued that the temporal characteristics of the fMRI BOLD signal are unsuitable for drawing such causal inferences from the Granger causality technique (Smith et al., 2011). Furthermore, given that a 'visual advantage' in deaf over hearing participants has been observed in domains such as motion processing and peripheral visual processing, connectivity between regions involved in these aspects of vision (V5/MT and parietal cortices) and STG may well differ between groups – thus supporting a bottom-up account. Future studies that use different approaches to test functional and effective connectivity, and which test models including a range of different regions, will shed more light on the underlying causes of crossmodal plasticity in humans.

Although research in this field is typically hampered by small sample sizes, Ding *et al*. were able to recruit an impressively large number of deaf participants. However, the one factor that they were not able to control for was language experience. In deaf humans, crossmodal cortical reorganisation is a consequence not only of absent auditory input, but also of language being acquired almost exclusively in a visual modality (Cardin et al., 2013). Ding *et al*. acknowledge that a group of hearing signers would need to be tested to tease apart the influence of sign language knowledge and auditory deprivation.

Research with deaf participants is more complex still, since the influence of language delay and language proficiency must also be considered. Approximately 95% of deaf children are born to hearing non-signing parents. For these children, spoken language exposure is often late and incomplete. This can influence language proficiency (Mayberry, 2007) and also the neural basis of language processing (MacSweeney et al., 2008). It is very likely that the participants in the Ding *et al*. study fell into this group. This then raises the possibility that some of the group differences in activation, and in activation/task performance correlations, are not the result of sensory deprivation *per se*, but a secondary effect of late and insecure language acquisition. For example, activation in STG during the maintenance period was positively correlated with the age of onset of hearing aid use, and negatively correlated with the percentage of lifetime hearing aid use. Given that age of onset of hearing aid use is likely to also index spoken language proficiency, and that the STG is known to be involved in

language processing in deaf individuals, these findings can be interpreted in two ways: a) greater crossmodal activation is a result of reduced auditory experience (as suggested by Ding *et al*.) or b) late and insecure language acquisition triggers compensatory mechanisms for cognitive processing, including the recruitment of the STG for visual working memory processing.

Research with deaf animal models and humans can provide unique insights into cortical plasticity and further our understanding of the general function of the superior temporal cortices, across modalities. Experiments with animal models (Lomber et al., 2010) can lead to the generation of clear hypotheses to be tested in humans, but they can only take us so far. Studies with humans must then also grapple with the difficult question of how auditory deprivation and language acquisition (in the absence of sound) interact and influence brain structure and function, and indeed behaviour. So far we have only scratched the surface of the plastic potential of the human brain.

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