The auditory system is far from being a hard-wired processing machine. It boasts a dynamically connected map of ipsilateral and contralateral pathways that retain a great deal of plasticity throughout adulthood. “Auditory plasticity” is a term used to describe changes in the anatomical and physiological properties of neurons in the brain, following a change in auditory input or experience. Such experience related physiological reorganisation is often accompanied by perceptual changes which may, or may not, be helpful to the individual.

The main cause of auditory plasticity in humans results from a reduction in auditory input to the central auditory system due to a sensorineural or conductive hearing loss. Reintroducing auditory input through hearing aid use or cochlear implantation has been shown to induce secondary plasticity. The implications of long term deprivation and stimulation have been found to affect a range of physiological and perceptual measures including loudness perception, intensity discrimination, the auditory brainstem response and the acoustic reflex threshold (ART). Some of these changes are thought to result from a “central gain mechanism.”

The central gain mechanism can be likened to an internal volume control. Recent neurophysiologic research has proposed that high levels of neural gain are implicated in the disorders of tinnitus and hyperacusis. It has been suggested that a central homeostatic mechanism causes an abnormal increase in central gain in response to auditory deprivation or trauma. Consequently, a possible solution is to find ways to decrease central gain.

Previous research suggests that it is possible to manipulate central gain. Perceptual and physiological changes have been found to occur following short term auditory deprivation. Munro and Blout fitted 11 normal hearing adults with monaural ear plugs. They found that after seven days of regular use the level required to elicit the acoustic reflex in the treatment ear (i.e., the ear with the earplug) decreased by 5–7 dB, relative to pre-treatment levels. Measurements made seven days post-treatment showed that the ART had returned to baseline values. It was hypothesised that because the plugged ear is deprived of input, neural processes increase the gain in order to restore average neural activity and this is revealed by a lower sound level required to elicit an acoustic reflex. When the earplug is removed, gain reverts back to pre-treatment levels and this is accompanied by an increase in the level required to elicit an acoustic reflex.

In addition to physiological changes, previous research has shown that it is possible to induce perceptual changes following short term auditory deprivation and stimulation. Formby and colleagues reported on 10 normal-hearing listeners who wore bilateral

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earplugs (deprivation) or noise generators (stimulation) for 2–4 weeks. After earplug experience, listeners required a decrease in level of around 6–8 dB to match pre-treatment loudness for moderate and high stimulus presentation levels. Conversely, listeners required an increase in level of around 6–8 dB after noise generator experience.

In summary, previous work suggests that short-term auditory deprivation results in a decrease in the ART, an increase in loudness of sounds and thus an increase in central gain. These findings were the motivation for our recent study; is it possible to induce plasticity in response to short term auditory stimulation?

The main aim of the study was to investigate the effect of short-term use of low-gain hearing aids on ARTs and loudness. We recruited 21 normal hearing adults for the study. The participants were fitted monaurally with a Starkey S series, non-occluding, receiver in the canal (RIC) hearing instrument (Figure 1.) The hearing aid settings were adjusted so that real-ear insertion gain such as the difference in response between the aided and unaided conditions, was 0 dB at 0.5 kHz and 15–20 dB at 2–4 kHz. Amplification was given at 2–4 kHz only, so that we could assess whether the treatment could induce frequency specific changes. The participants were asked to wear the device continuously for five days, except while in bed.

ARTs and loudness ratings for the fitted ear and the control ear were made on three occasions over a five day period: immediately before hearing aid fitting (day zero), after three days of hearing aid
use and after five days of hearing aid use (Figure 2.). Ipsilateral ARTs were measured using a 0.5 kHz, 2 kHz and broadband noise (BBN) stimulus. Loudness judgements were obtained with a 0.5 kHz and 3 kHz tone using the Contour Test of Loudness Perception.9 Listeners used a response pad to assign one of seven loudness categories to a train of pulsed warble tones. The loudness categories ranged from “very soft” to “uncomfortably loud.”

The mean change in ART at day five, relative to baseline, is shown in Figure 3. The data presented in this article compares the change at day five only. However, all the findings have been reported in Munro and Merrett.10 For the 0.5 kHz tone, the change was less than 2 dB in the fitted ear. For the 2 kHz tone, the change was greatest at day five, where the mean difference between ears was around 3 dB, primarily due to an increase in ART in the fitted ear. For BBN, there was a mean difference between ears of around 3–4 dB at day five.

The mean level change at each loudness category is shown in Figure 4. For the fitted ear, listeners generally needed more intense stimuli (+3 to +5 dB) after wearing the hearing aid in order to give the same loudness judgements. In contrast, changes for the control ear were small and generally <1 dB.

The effect of amplification-induced stimulation is opposite to the effect of earplug-induced deprivation. Our results show an increase in the ART and an increase in loudness tolerance in the fitted ear following short term amplification. The change in ARTs provides support for a gain control mechanism. Because the fitted ear is provided with an ‘enhanced’ input, the gain is reduced and this is revealed by a higher sound level required to elicit the acoustic reflex.

In comparison to the loudness judgement changes, the changes in the ARTs appear to be relatively frequency specific. This may reflect different characteristics of the gain mechanism at different levels within the auditory system.

The findings in the present study may have implications for patients with tinnitus and/or sound tolerance problems. It is possible that ear plug use may increase neural gain and exacerbate perceptions such as tinnitus and hyperacusis. Computational models have illustrated how auditory deprivation may result in an increase in neural gain as homeostatic plasticity attempts to restore average neuronal activity.11

However, it would be important to replicate the study with a larger sample size, a longer treatment period and to make a comparison between solely high and low frequency stimulation.
REFERENCES