

Use of tinnitus masking functions to support or refute the presence or absence of auditory plasticity

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Tinnitus, the perception of sounds that do not have a peripheral correlate, is often hypothesized to be associated with cortical reorganization that over-emphasizes baseline cortical activity and is perceived as these phantom signals. But there are several issues that suggest this explanation may not be universal (if the system is plastic, why can't tinnitus be eliminated by another plastic change?). A potential technique to distinguish tinnitus that may be correlated with auditory plasticity versus tinnitus associated directly with peripheral damage will be evaluated. Narrow bands of noise will be used to determine masking thresholds across frequencies. Thresholds will be plotted relative to the tinnitus pitch to determine whether the frequency of optimal masking is aligned with the frequency of tinnitus, which does not support plasticity, or with adjacent frequencies, supporting the existence of auditory plasticity. Subjects with tinnitus frequency less than 6 kHz will be recruited, and a test battery will be collected, including DPOAE, tinnitus frequency, TEN test to detect possible dead zones, as well as masking thresholds with narrow bands of noise around the tinnitus frequency. Case studies will be presented to demonstrate the threshold functions found in a small sampling of tinnitus patients. Implications for treatment will be discussed.

INTRODUCTION

Auditory plasticity has been defined as a change to the tonotopic arrangement of the auditory cortex caused by lack of stimulation (Engineer *et al.*, 2011). Evidence for plasticity has been caused by ablation of regions of the basilar membrane in animal studies. The evidence shows re-allocation of cortical responses from ablated frequencies to adjacent frequencies that are still audible, so the cortical mapping becomes distorted with larger cortical area allocated to audible frequencies, and less or none allocated to ablated frequencies. This demonstrates how the cortex will map to the nearest frequency with some useful input, since the cells are responding to stimulation from the auditory pathways.

Tinnitus is often hypothesized to be associated with cortical reorganization, where baseline cortical activity is over-emphasized or becomes synchronized when additional cortical areas become associated with frequencies already covered in the standard tonotopic mapping (Engineer *et al.*, 2013). This hypothesis raises several questions such as:

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- Does cortical reorganization require ablation or dead zones?
- If the system is plastic, why can't tinnitus be eliminated by another plastic change?

For this hypothesis to be true at the simplest level, tinnitus should occur at adjacent frequencies compared to dead zones where the tonotopic mapping has been distorted and too many cells respond together at a baseline level. A potential technique to try and distinguish tinnitus that may be correlated with auditory plasticity using clinically available materials has begun to be evaluated to see if some answers are possible.

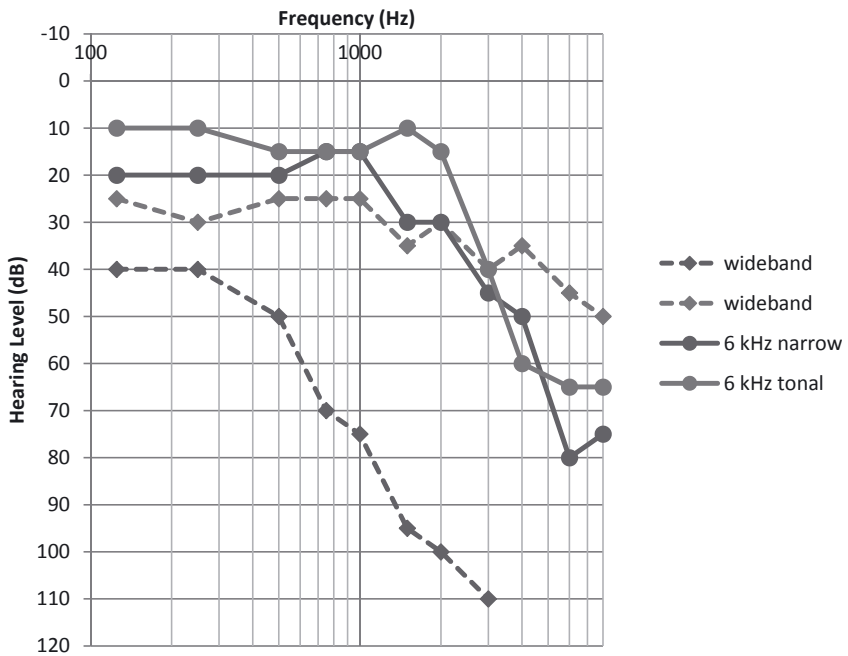


Fig. 1: Audiograms for the four subjects, with the type of tinnitus and characteristic frequency identified in the legend. The colors were chosen to identify the alternate wideband or tonal subject, and are not to identify the ear involved.

METHOD

Subjects were recruited randomly at clinics in Spain with complaints of tinnitus. Standard audiograms were measured from 125 Hz to 8000 Hz, including half octaves, and are reported in Fig. 1 for the ear with the loudest tinnitus. Tinnitus pitch and level in the ear with the loudest tinnitus were determined, as well as masking thresholds using narrowband noise and broadband noise from an audiometer. The masking threshold would help to see what frequencies have the greatest impact on the audibility of the tinnitus, and how these are related to the tinnitus itself. The test battery included distortion product otoacoustic emissions (DPOAEs) and threshold-equalizing-noise (TEN) test results to detect possible dead zones.

Subjects

Four subjects (2 male, 2 female) were recruited with clinical complaints of tinnitus accompanying sensorineural hearing loss (no conductive component). They had a mean age 53 years, with a range from 42 to 64 years of age. The subjects identified the quality of their tinnitus and, where applicable, used pitch matching to identify the nearest audiometric frequency to their tinnitus. The four subjects included two with broadband tinnitus, one with narrowband tinnitus centered at 6 kHz, and one with pure-tone tinnitus centered at 6 kHz.

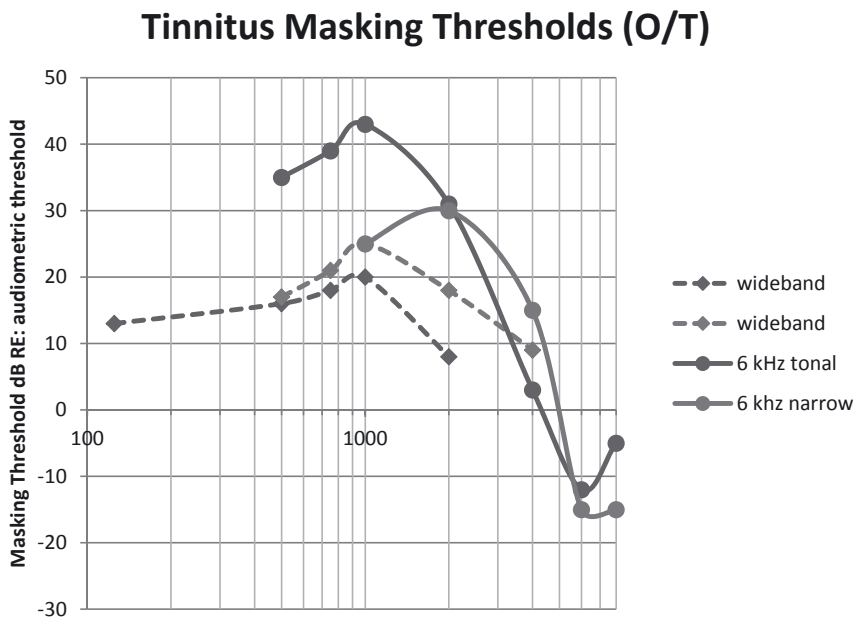


Fig. 2: Plot of Masking Thresholds relative to audiometric thresholds

All showed sloping high-frequency hearing loss of varying degrees. As a note, the tonal tinnitus occurred in the subjects with the best low-frequency hearing, when analyzing audiograms to look for any defining features. For the two subjects with any tonal quality, the tinnitus frequency appears to coincide with the frequency of maximum hearing loss.

ANALYSIS

Thresholds were plotted relative to the tinnitus pitch (Fig. 2) to determine whether the frequency of optimal masking is aligned with the frequency of tinnitus, which does not support plasticity, or with adjacent frequencies, supporting the existence of auditory plasticity.

For the wideband tinnitus, thresholds drop towards higher frequencies, but cannot be measured at the highest frequencies because of equipment limitations along with increasing hearing loss. These subjects do not meet the expectation to support or refute any auditory plasticity hypothesis, but are interesting none the less in the similarity in the function of masking thresholds relative to audiometric frequencies. It appears that masking thresholds, if audible, will fall towards 6 kHz, which would be an interesting finding.

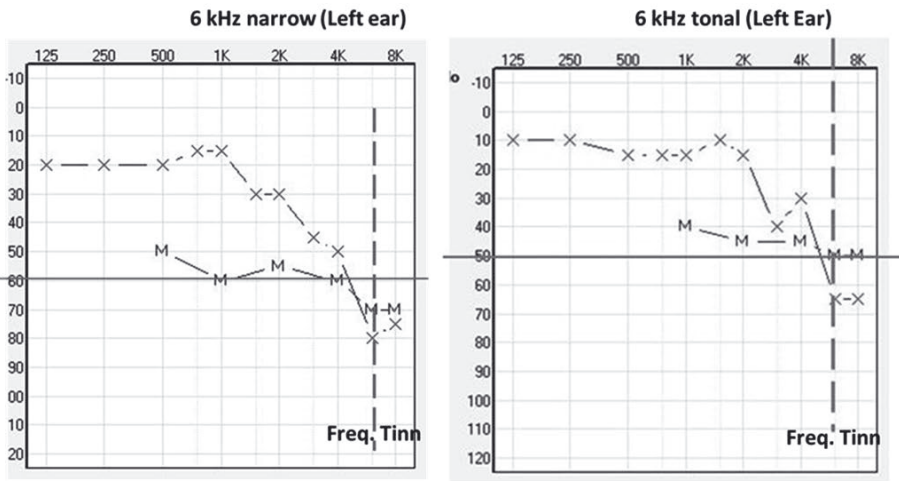


Fig. 3: Audiometric thresholds (X), masking thresholds (M), and the frequency (dashed line) and level (solid line) of the tinnitus in the two tonal subjects tested.

For these two tinnitus subjects with tonal tinnitus (Fig. 3), what is surprising is the fact that thresholds are lower than audiometric thresholds at 6 and 8 kHz. Both show masking by narrow band noise at 6 kHz and 8 kHz at lower levels than hearing thresholds at these frequencies. But this means that the masking signal should be inaudible even though they still have masking of their tinnitus. Stimulation occurring at audible adjacent frequencies from the bandwidth of the noise is not possible based on the adjacent audiometric thresholds and the skirts of the narrow band noise used, so some other explanation is needed.

Both of these subjects have positive results of distortion products in their left ear at 6 kHz, but only at high stimulus levels (70 dB and 75 dB, respectively). These masking thresholds show optimal masking at the frequency of the tinnitus, suggesting that dead zones and reallocation to adjacent frequencies is not likely. The OAEs also support some outer-hair-cell function, so the traditional plasticity model is not possible.

The two patients with broadband tinnitus were not the target for this study, but testing was completed on any subject that could be recruited. In these subjects, masking occurs only at audible levels (Fig. 4), above the audiometric thresholds, but often at a level lower than the tinnitus level. When tested with a broadband masker, broadband masking occurred at a level above the level of the tinnitus.

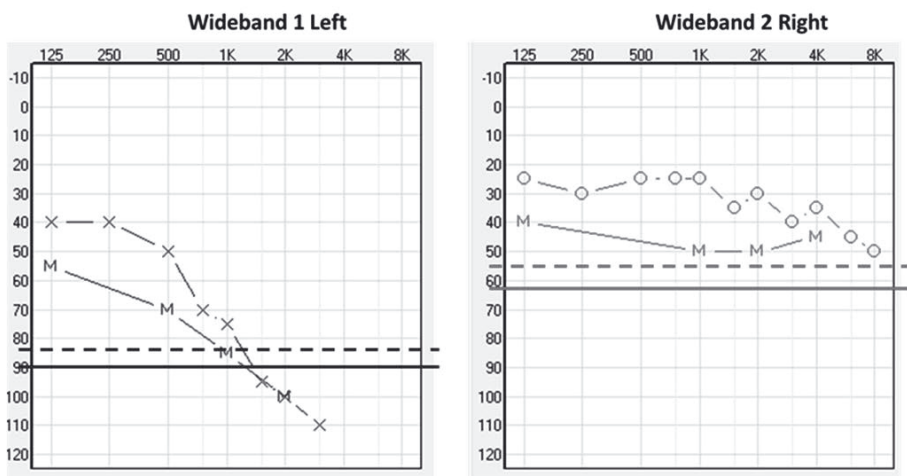


Fig. 4: Audiometric thresholds (X and O), masking thresholds (M), and the level of the tinnitus (dashed line) and level (solid line) of the broadband masking threshold.

Narrowband masking occurs at levels below the level of the tinnitus until the hearing loss exceeds that level and pushes the masking thresholds to very high levels. So this begs the question, which is the best masking noise for these patients? Should it be white noise, because it is similar to the tinnitus noise? Or should it be 1-kHz or 4-kHz narrowband noise, respectively, because those are the bands with the minimum difference between masking and audiometric thresholds? Or should the patient be allowed to alternate between these two (or other) noises? Tinnitus treatment methods have only proven that preferred and optimal sounds have not been shown to be predictable based on clinical measures, but a systematic approach like this highlights some interesting effects. And the results do not explain how the narrowband noise was often lower in level than the broadband noise, as well as the tinnitus, yet still masked the tinnitus. How can this be?

CONCLUSIONS

We raised many questions and did not find conclusive answers. No dead zones were identified (up to the limits of the TEN test at 4 kHz), so should we have expected any cortical reorganization to have occurred? If cortical reorganization does not require cochlear dead zones, then the auditory mapping must constantly be changing, suggesting that tinnitus should disappear as easily as it begins. But this has not been reported in the cases severe enough to seek treatment. In the current data, the tinnitus was aligned with hearing loss, but is this a resolution problem with the audiometric stimuli? It is possible that dead regions may be smaller than audiometric resolution, and therefore an adjacent frequency being associated with tinnitus is just too close to the test frequencies to measure with clinical stimuli.

There are clearly differences in the impact of various simple maskers on the perception of tinnitus, and these differences varied between subjects. The only useful conclusion is that simple clinical measurements may be useful to guide selection of sounds for tinnitus treatment (if masking thresholds are useful to improve treatment), but supporting or refuting cortical plasticity is still not possible. To that end, subject testing will continue.

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