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The relationship between tinnitus pitch and hearing sensitivity

Giriraj Singh Shekhawat · Grant D. Searchfield · Cathy M. Stinear

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Abstract Tinnitus is the phantom perception of sounds. No single theory explaining the cause of tinnitus enjoys universal acceptance, however, it is usually associated with hearing loss. The aim of this study was to investigate the relationship between tinnitus pitch and audiometry, minimum masking levels (MML), tinnitus loudness, and distortion product otoacoustic emissions (DPOAE). This was a retrospective analysis of participant's records from the University of Auckland Hearing and Tinnitus Clinic database. The sample consisted of 192 participants with chronic tinnitus (more than 18 months) who had comprehensive tinnitus assessment from March 2008 to January 2011. There were 116 males (mean = 56.5 years, SD = 12.96) and 76 females (mean = 58.7 years, SD = 13.88). Seventy-six percent of participants had a tinnitus pitch ≥8 kHz. Tinnitus pitch was most often matched to frequencies at which hearing threshold was 40–60 (T50) dBHL. There was a weak but statistically significant positive correlation between tinnitus pitch and T50 ($r = 0.15$ at $p < 0.05$). No correlation was found between tinnitus pitch and DPOAEs, MML, audiometric edge and worst threshold. The strongest audiometric predictor for tinnitus pitch was the frequency at which threshold was approximately 50 dBHL. We postulate that this may be due to a change from primarily outer hair cell damage to lesions including inner hair cells at these levels of hearing loss.

Keywords Tinnitus pitch · Tinnitus · High frequency audiometry · Hearing

G. S. Shekhawat · G. D. Searchfield (✉)
Section of Audiology, Department of Audiology, University Of Auckland, Tamaki Innovation Campus, Private Bag 92019, 1142 Auckland, New Zealand
e-mail: g.searchfield@auckland.ac.nz

G. S. Shekhawat · G. D. Searchfield · C. M. Stinear
Centre for Brain Research, University of Auckland, Auckland, New Zealand

G. D. Searchfield
Tinnitus Research Initiative, Regensburg, Germany

C. M. Stinear
Department of Medicine, University of Auckland, Auckland, New Zealand

Introduction

Tinnitus is a perceived sound that cannot be attributed to an external source [1]. Tinnitus can be constant or intermittent, and is commonly described as ringing, buzzing, cricket-like, hissing, whistling, and humming [2]. No single theory explaining the cause of tinnitus is universally accepted. Tinnitus can occur due to any form of malfunction occurring along the auditory pathways [3–8]. Chronic tinnitus possibly occurs from a cascade of changes occurring at various cortical [5] and subcortical centres [9] including: dysfunction of cochlear receptors and reduced spontaneous firing rate of the auditory nerve fibers [10] and to compensate for this reduction, there is an increase in central gain by reduction in cortical inhibition leading to tinnitus perception [11].

Tinnitus is usually associated with hearing loss [6, 12]. The range for human hearing is between 20 Hz and 20 kHz [13]. For routine clinical measurement conventional audiometry assesses frequencies from 250 Hz to 8 kHz [14]. However, for disorders which initially affect high frequencies such as noise-induced hearing loss, presbycusis, and ototoxicity, it may be useful to measure the auditory

thresholds at extended high frequencies as it gives in depth and early information about the underlying pathology [14, 15].

High frequency audiometry is also useful for assessment of tinnitus [16]. Roberts et al. [17] showed that 25 % of tinnitus research participants had normal hearing up to 8 kHz yet all revealed hearing loss with extended high frequency audiometry, and experienced residual inhibition to sounds in this high frequency range. Hyun et al. [18] reported similar findings; in their study 66.7 % of tinnitus participants had normal hearing below 8 kHz but when extended high frequency audiometry was conducted all of them had hearing loss at 10, 12, 14 or 16 kHz. The pitch of tinnitus most often corresponded to frequencies above the audiogram edge [19].

Tinnitus pitch is usually associated with frequencies showing hearing loss i.e., high pitch tinnitus is usually associated with high frequency hearing loss and low pitch tinnitus with low frequency hearing loss [11, 20]. However, there is large inter- and intra-session variability associated with pitch matching [21]. Above 3 kHz the tinnitus pitch usually corresponds to the frequency at which the hearing loss becomes clinically significant [19, 22–24]. In addition the shape of the audiogram can also indicate tinnitus, as the steepness of hearing loss is positively correlated with the incidence of tinnitus [19].

Hearing loss can lead to cortical reorganization in animals due to a reduction in the spontaneous outflow of the cochlea [25]. Damage to the inner hair cells (IHC), outer hair cells (OHC), and cochlear neurons give rise to elevated hearing thresholds [26, 27]. Spontaneous activity recorded from the reorganized tonotopic maps is generally higher than that of the normal/un-reorganized map [28]. It has been speculated that spontaneous activity could be the possible neural correlate of tinnitus and the characteristic frequency dominating the reorganized map may constitute the pitch of tinnitus [29]. The maximum amount of cortical reorganization occurs at the transition from good hearing to impaired hearing [30].

OHCs are more vulnerable to damage than the IHCs [31, 32]. However, IHC damage may be a significant contributor or prerequisite for changes in spontaneous afferent output of the cochlea [33] and for tonotopic reorganization [34, 35]. The region in the cochlea where there are no functioning IHCs and/or neurons is referred as the dead region [27]. Using the threshold equalizing noise (TEN) test Weisz et al. [36] demonstrated that 72.7 % of tinnitus suffers had dead regions. Dead regions are often associated with high frequency sloping hearing loss, but it is considered difficult to identify them with just pure tone audiometry [26]. Cochleae of cadavers with cochlear hearing loss have been examined to explore the relationship between audiogram and loss of IHCs [37]. No IHC damage was noticed with thresholds at and below 40 dBHL in any cochlea examined, damage to IHC started appearing after that [37]. Hence 50 dBHL was taken as the cut-off point for suggesting damage of the IHC in the current study.

Robertson [38] used a linear regression model to assess if the audiometric edge of OHC function could predict tinnitus pitch and found a strong positive correlation between audiometric edges and tinnitus pitch in 71 % of participants. The audiogram is generally considered a poor indicator of the degree of cochlear damage [36]. The "edge" of hearing defining a reduction in spontaneous activity (and hence potential plasticity) is not the edge between a normal audiometric threshold and an elevated audiometric threshold it is actually the frequency at which IHC or neural loss begins. From human cadavers IHC loss begins to occur after 50 dBHL. We hypothesized that, if spontaneous output from the cochlea contributes to the so-called edge effect the frequency at which audiometric threshold is approximately 50 dBHL would be more strongly correlated with tinnitus pitch than the frequency at which hearing loss begins according to the audiogram (thresholds at 20 dBHL [T20]) (which may not have any change in spontaneous outflow of the cochlea due to OHC loss not IHC loss). Maximum hearing loss (TW) may (depending on extent of hearing loss) be at frequencies removed from the lowest frequency of IHC damage. The psychoacoustical illusion equivalent to the edge effect (i.e., the perception of sound after a band of noise is the "Zwicker" tone) [39]. The Zwicker tone is most strongly elicited at the low frequency edge of a gap in sound. This has been considered to be the equivalent of the edge of a hearing loss in some models of tinnitus [40].

The aim of this study was to investigate the relationship between tinnitus pitch and audiometry, minimum masking levels (MML), tinnitus loudness, and distortion product otoacoustic emissions (DPOAE). It was hypothesized that the frequency of audiometry equating to a threshold of 50 dBHL would be more strongly correlated to tinnitus pitch than the "edge" frequency of hearing loss or frequency of maximum hearing loss.

Methods

This study was approved by the University of Auckland human participants' ethics committee.

Participants

This was a retrospective analysis of client records from the University of Auckland Hearing and Tinnitus Clinic database. From the database, 300 participants were randomly chosen from March 2008 to January 2011 and those with

incomplete assessment (e.g., unable to match tinnitus pitch, alternative assessment undertaken) were excluded. The sample consisted of 192 participants with chronic tinnitus (more than 18 months) who completed a comprehensive tinnitus evaluation. There were 116 males (mean = 56.5 years, SD = 12.96) and 76 females (mean = 58.7 years, SD = 13.88). For 103 participants the predominant tinnitus was towards the right ear, for 83 it was towards left ear and 6 people found it equally loud in both ears (both ears were included for them in analysis). Participants were excluded if any of the clinical measurements described below were not undertaken.

Procedure

Client records were examined and then pure tone audiometry (250–16,000 Hz), DPOAE, MML, and tinnitus loudness were compared to tinnitus pitch.

All tinnitus assessments had been undertaken in audiometric test booths (ISO 82531-2009) using two-channel audiometers [either GSI-61 audiometer (Grason Stadler) or AC40 (interacoustics)]. While assessing hearing thresholds at extended high frequencies where the audiometer limit was reached, the maximum levels at those frequencies were recorded as the response. Measurements used standard ear phones (TDH-50P telephonics) or insert headphones (E.A.RTONE 3A, 0.25–8 kHz) and high frequency headphones (Sennheiser HDA 200, 8–16 kHz). Audiometry was obtained using the modified Hughson-Westlake procedure (Carhart & Jerger). GSI (Grason Stadler) Tymp star v.2 Immittance audiometers were used and DPOAE were measured using an ILOv 6 (Otodynamics, Ltd.) OAE analyser. Tinnitus pitch, loudness and MML were obtained using the audiometer in the following manner.

Pitch matching

A two-alternative forced-choice (2AFC) method was used, in which pairs of tones were presented based on the audiogram and perceptual feedback from participants regarding tinnitus pitch and participants were asked to identify which one best matched the pitch of their tinnitus. Each tone was presented at a sensation level of 10 dBSL. Once the settings for a given pair of tones were established, the two tones were presented in alternating manner until the participant indicated which one was closest to the pitch of their tinnitus. Pitch match was then compared to tones 1 octave above and below to rule out octave confusion.

The instructions given to participants were "we want you to compare two sounds to your tinnitus. Indicate whether the first or second sound is closest to your tinnitus. Both sounds may not exactly match your tinnitus that is okay, we want to know which is most similar". This was repeated with the following instruction, "we are now going to repeat this comparison, again indicate whether sound 1 or 2 is closest to your tinnitus". If there was a perceived difference in tinnitus loudness between sides of the head, the test ear was chosen to be the ear contralateral to the predominant or louder tinnitus. If the tinnitus was equally loud on both sides or localized in the head, the test ear was the one with the better hearing (if there was no difference between the acuity of the two ears the ear was chosen randomly).

Exceptions to the contralateral rule were:

(a) Contralateral ear had hearing loss in the severe to profound range and it was impossible to present at tinnitus loudness due to the degree of loss and limits of equipment.
(b) Cases of known diplacusis.
(c) Cochlear dead regions in contralateral ear (identified using TEN test or psychoacoustic tuning curves). These tests were not routinely undertaken.

Sensation level matching

Sensation level matching ("loudness" matching) was conducted contralateral to the tinnitus ear as outlined for pitch matching. Air conduction threshold was obtained for the frequency closest to their tinnitus using 1 or 2 dB steps. At the test frequency, the starting level was below threshold and ascended continuously in 1 or 2 dB steps until the participant indicated that it was just as loud as their tinnitus. This measurement was undertaken 3 times and then the average of the 2nd and 3rd response was taken as the loudness match. The sensation level of tinnitus was determined by subtracting the dial dB at threshold from the dial dB at loudness match.

Instructions given were

For threshold: "You will hear a series of tones; we want you to indicate every time you hear the sound, even if it is very quiet."

For sensation level: "You will now hear a series of tones indicate when the sound is equally loud to your tinnitus."

MML

The MML was the minimum sound that "covered" the individual's tinnitus (i.e. rendered the tinnitus inaudible). The patient's threshold for noise (dB dial) was measured and recorded. The level of the noise was then raised in 5 dB increments until the patient reported that the tinnitus was no longer audible (up to the limits of the equipment or the patient's tolerance level, whichever was reached first). The level at which the tinnitus was just rendered inaudible

was recorded. MML in sensation level was the difference between the masked level and threshold for that noise. The MML was tested using narrow band noise at 500 Hz, 1 kHz, 2 kHz, 4 kHz and where possible at tinnitus pitch.

Instructions given were as follow, "You will hear a hissing sound. Indicate each time you hear it, even if it is very quiet. The level of sound will gradually increase. Indicate when it covers your tinnitus. If the sound becomes uncomfortable indicate and it will be stopped".

The test ear was the side with the louder or predominant tinnitus; if there was no difference between the sides, each ear was tested separately. When the masking sound was able to render the tinnitus inaudible, that result was recorded as "complete masking". In some cases, the masking stimulus was only able to make the tinnitus somewhat less audible, and was recorded as "partial masking". In a small percentage of cases, the masking stimulus had no effect on the audibility of tinnitus and was recorded as "not masked".

Analysis

SPSS software (IMB version 19) was used for statistical analysis. *T* tests and correlation analyses were carried out to explore the relationships between tinnitus pitch and other measures (hearing thresholds, MML, and DPOAE). Auditory thresholds were divided into three cut-off frequencies, T20, T50, and TW. T20 was the first frequency at which the hearing threshold crossed 20 dBHL and its consecutive frequency hearing threshold was worse than 20 dBHL. TW was the highest frequency at which auditory threshold was at its highest (poorest hearing) and T50 was the frequency between T20 and TW at which the threshold was equal to or close to 50 dBHL (Fig. 1). It represents the approximate degree of hearing loss required for transition from OHC to IHC loss [37]. This classification was undertaken to study the relationships between the tinnitus pitch and the points at which the hearing is normal (T20), most affected (TW) and the theoretical border between OHC and IHC impairment (T50). Participants were excluded if it was not possible to calculate T20, T50 or TW for any reason.

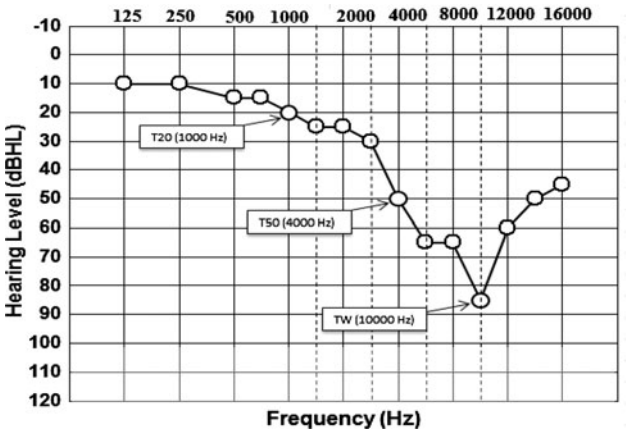

Fig. 1 An example of how T20, T50 and TW were calculated. T20 was the first frequency where threshold crossed 20 dBHL and its consecutive frequency threshold was worse than 20 dBHL; in this case it is 1,000 Hz. TW was the highest frequency at which auditory threshold was at its highest (poorest hearing, 10,000 Hz) and T50 was the frequency between T20 and TW where the threshold was equal to or close to 50 dBHL (4,000 Hz in this case). Although, the threshold is 50 dBHL at 14,000 Hz the lowest frequency between T20 and TW, is at 4,000 Hz

Results

All participants with tinnitus had some degree of hearing loss and the severity of hearing loss was greater in the extended high frequencies. Overall hearing levels were fairly symmetrical between right ears and left ears. Hearing thresholds were below 25 dBHL until 2 kHz, beyond which a sloping deterioration was observed (except at 13 kHz, where thresholds were better compared to adjacent frequencies). No significant difference was seen between the right and left ears' mean threshold up to 12 kHz. Right ear thresholds were worse than those of the left ear at 14, 15, and 16 kHz; however, this difference was not more than 10 dBHL (Fig. 2).

The majority of participant's tinnitus was characterized as being high pitched. A bell-shaped curve skewed towards the high frequencies can be observed across the frequency range for tinnitus pitch with tinnitus most frequent at

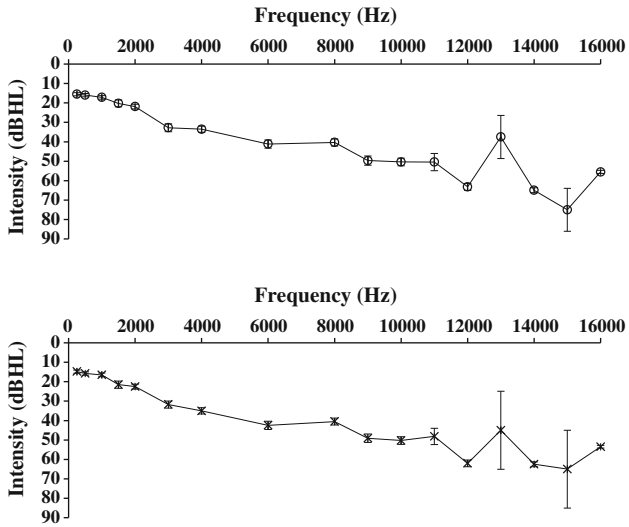

Fig. 2 The mean hearing thresholds for *right* (*circles*) and *left* ear (*crosses*) for participants across the frequency range of 250 Hz to 16 kHz ($N = 192$). The *error bars* represent ± 1 standard error of the mean

9–10 kHz, followed by 8 kHz and 11–12 kHz (Fig. 3). Tinnitus pitch fell between 8 and 10 kHz for 49 % of participants. Tinnitus pitch was most often matched to frequencies at which hearing threshold was 40–60 dBHL (T50). The difference between TP and the estimated pitch at T20, T50, and TW was calculated (Fig. 4). TW resulted in a higher estimate of tinnitus pitch than measured (mean difference = −4,479.65), T20 resulted in a lower estimate (mean difference = 4,595.49), T50 (mean difference = 1,115.98) provided the closest estimate to measured tinnitus pitch. Paired t tests were undertaken to explore the mean difference between T20, T50, TW, and TP (TW–TP, TP–T20 and TP–T50), there was a significant difference between TP–T20 and TP–T50 [t (182) = 18.56, $p < 0.001$] and TW–TP and TP–T50 [t (182) = −5.38, $p < 0.001$], but TW–TP and TP–T20 were not statistically different. Although T50 resulted in the closest estimate of tinnitus pitch, there was still considerable variation (SD = 5,011.05).

There was a small but statistically significant positive correlation between tinnitus pitch and T50 ($r = 0.15$, $p < 0.05$). Tinnitus pitch increased with higher T50 frequency. A similar positive trend was observed at T20 and TW, however, their correlations did not meet the adopted level of statistical significance ($p < 0.05$, Fig. 5).

As the stimulus frequency increased the presence of DPOAE reduced. For the majority of participants the emissions were present at 1 kHz (247 ears), however, at 8 kHz only 11 ears had DPOAEs present (Fig. 6).

Discussion

This study reports the audiological profile of 192 participants with tinnitus from the University of Auckland Hearing and Tinnitus Clinic database. The average hearing loss was normal in the low frequencies sloping to mild at 8 kHz, but moderate to severe hearing loss above 8 kHz (up to 16 kHz). We believe this indicates the importance of high frequency testing in the tinnitus assessment battery [17, 18].

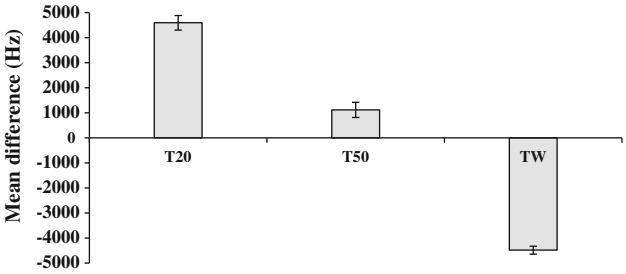

Fig. 4 Mean difference between TP and T20, T50 and TW. The *error bars* represent ±1 standard error of the mean

Seventy-three percent of participants matched their tinnitus pitch between 8 and 16 kHz. The strongest audiometric predictor for tinnitus pitch was the frequency at which threshold was 50 dBHL (T50). This threshold intensity is hypothesized to be important in tinnitus generation as it represents the approximate degree of hearing loss required for transition from OHC to IHC loss [37]. Cochlear deafferentation is believed to be the peripheral driver for central adaptation mechanisms creating tinnitus [36]. The IHCs provide the bulk of afferent input to the central pathways; IHC damage (beginning at approximately hearing thresholds of 50 dBHL) may contribute to tinnitus pitch as a consequence of central plastic changes at the frequency of initial deafferentation.

There have been several efforts to explore the relationship between tinnitus pitch and audiometry. There are two main theories explaining the relationship underpining tinnitus pitch and the audiogram: "edge effect" and "homeostatic" mechanisms. Some studies have demonstrated a positive correlation between tinnitus pitch and edge frequency [19, 41] while others have failed to do so [42, 43]. Proponents of the homeostatic mechanism hypothesis believe that discordant damage to hair cells leads to reduction in sensory input to the auditory nerve. To compensate for this reduced input, homeostatic mechanisms may come into play which increase central gain and reduce cortical inhibition, leading to amplification of neural noises which in turn results in tinnitus [11, 44]. According to this model, the tinnitus pitch

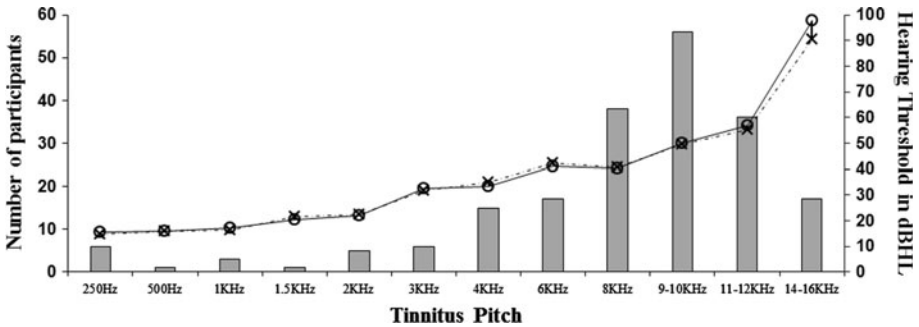

Fig. 3 The numbers of participants reporting tinnitus pitch matches as a function of frequency (*bars*) and mean hearing thresholds corresponding to these frequencies (*symbols*)

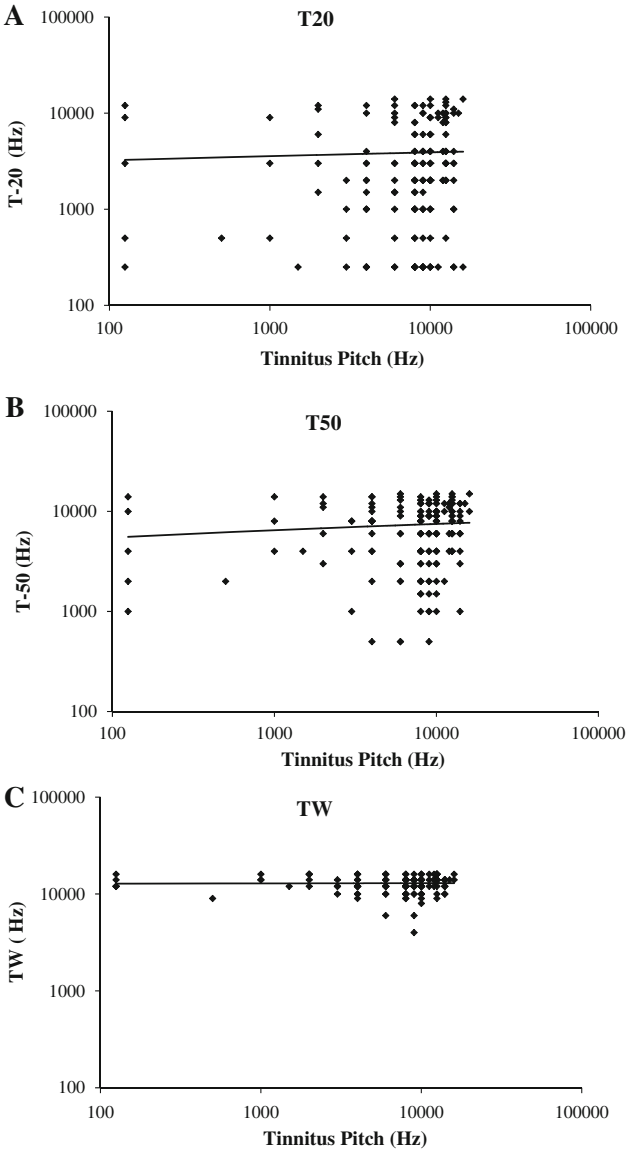

Fig. 5 Correlation between **a** T20 ($r = 0.13$), **b** T50 ($r = 0.15$) and **c** TW ($r = 0.09$) and Tinnitus Pitch

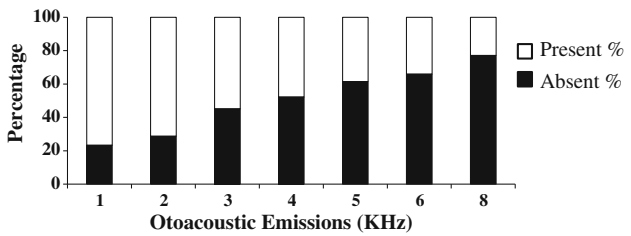

Fig. 6 DPOAEs present and absent as a function of stimulus frequency

should fall in the region of hearing loss. There have been a number of studies supporting this notion [17, 20, 24, 45, 46].

There have been a few studies looking at the relationship between tinnitus pitch and the frequency with maximum hearing loss, with some showing a positive correlation [46] and others not [20, 42]. However, none of the studies have looked at the relationship between tinnitus pitch and T50. The majority of studies attempting to explore the relationship between tinnitus pitch and audiogram have failed to incorporate the high frequency hearing thresholds beyond 8 kHz [19, 20, 41, 42, 46, 47]. The present data indicate that high frequency testing is important for tinnitus assessment, and can provide new insights regarding its mechanisms.

Elsaeid [48] reported that 85 % of tinnitus ears had abnormal TEOAEs, especially at 2, 4, and 8 kHz. Granjeiro et al. [49] found 70.2 and 68.4 % of tinnitus patients showed abnormal TEOAEs and DPOAEs, respectively. In the present study, the percentage of abnormal DPOAEs increased with frequency, 23.29 % had abnormal DPOAEs at 1 kHz, which doubled at 4 kHz (52.16 %) and tripled at 8 kHz (77.08 %) reflecting the dysfunctioning of OHCs especially at high frequencies. The majority of participants in the present study (73 %) matched their tinnitus pitch to 8 kHz and above, that is beyond frequencies associated with OHCs. This suggests that the damage to IHCs accompanying OHC dysfunction may be an important underlying factor or precursor for tinnitus generation. Confirmation of this result would require the use of tests to identify dead regions in tinnitus sufferers, such as use of the TEN test [36] and/or psychophysical tuning curves [50].

Assessment of tinnitus pitch is significant not only for systematic documentation of patients' symptoms, but also for monitoring the impact of interventions and planning tinnitus treatment involving acoustic stimulation such as tinnitus maskers [45]. Although psychoacoustical characteristics of tinnitus (such as tinnitus pitch, loudness, etc.) do not appear to determine tinnitus annoyance or severity of complaint [51], they may be useful as markers for neural plasticity if the tonotopic representation in the central auditory system is modified after treatment.

Feldmann [52] showed that tinnitus can be masked by narrow band noise and other noises (broad band noise, pure tones) in a frequency specific manner similar to masking of external sounds in only 34 % of cases. In the present study, the MML required to mask tinnitus decreased as the frequency increased, with the lowest level occurring at tinnitus pitch. However, no correlation was found between tinnitus pitch and MML.

Undertaking extended high frequency audiometry might also have ramifications for predicting the usefulness of high frequency amplification. Hearing aids may be more effective in treating tinnitus if the tinnitus pitch falls within the stimulated frequency range [53, 54]. A technical

limitation of current hearing aids is that they do not produce sufficient output beyond 5–6 kHz to overcome high frequency hearing loss [55], limiting the beneficial effects for participants with tinnitus pitch falling beyond the range of acoustic stimulation [53]. Further technical advancements in this area could be of significant advantage, especially for people suffering from high pitched tinnitus [56].

In the present study, the edge frequency was defined differently to previous studies [19, 41]. Moore et al. [41] calculated Δn (differences in threshold between successive audiometric frequencies). The lower of the two frequencies for which Δn was largest was assigned as the edge frequency. If there were two equal values of Δn and they were adjacent to one another in frequency, then the lower one was used for calculating edge frequency and if they were not adjacent, two edge frequencies were assumed for those participants. König et al. [19] calculated edge frequency based on the steepest slope in the normal hearing range or if not possible a similar criteria used in our study [(T20) the first frequency at which the hearing threshold crossed 20 dBHL]. Our method was simpler than previous studies [19, 41], but we believe would result in similar estimates to König et al. [19]. The Moore et al. [41] method was applied to mild-moderate high frequency sloping hearing loss in a small sample. Their method for edge calculation would likely result in a relatively higher, or multiple frequencies, of edge compared to our study.

All these methods are limited by the sensitivity of the audiogram to hearing damage, true edges of damage (such as loss of neuronal populations) are not going to be detected using the audiogram [57]. This may account for the variations in study outcomes and variability within the studies. Potential differences in calculation of edge frequency and the interpretations of results (if any) are open for further discussion and research.

Limitations

Only sloping audiograms were included in present study hence, this analysis may not be transferable to patients with other audiogram configurations. The audiogram is a crude measure of mechanisms that may contribute to tinnitus pitch; future research should consider alternative methods which may enhance sensitivity.

Conclusion

The present study highlights the significance of high frequency audiometry and recommends it as a useful test in the tinnitus assessment battery. The most important audiometric predictor for tinnitus pitch was the frequency at which threshold was approximately 50 dBHL. We postulate that this may reflect a transition from primarily OHCs damage to lesions including IHCs at these levels of hearing loss. Further research is needed in this area to confirm these findings.

Conflict of interest The authors declare that they have no conflict of interest.

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