Multimodal Stimulation Using Bone Conduction for Tinnitus Therapy.

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ABSTRACT

Tinnitus is the perception of sounds with no external source. Several techniques to manage tinnitus exist, including tinnitus masking. This study investigated the potential of using bone conduction to deliver tinnitus masking, instead of through the traditional air conduction route. Auditory and somatosensory perceptions are known to be intricately related including in their central processing. Somatosensory manipulations are also documented to alter tinnitus characteristics for some individuals. Multimodal integration of vibrotactile and auditory sensations using bone conduction may therefore effectively alter the intensity required for the masking noise of an individual's tinnitus. It was hypothesised that the intensity required for tinnitus masking may be reduced when using bone conduction, especially at the lower frequencies where vibrotactile perceptions can be felt.

The group consisted of twelve subjects (mean age of 55.00 years; 6 males, 6 females) with chronic and continuous tinnitus. Ear-specific minimum masking levels at different frequencies ranging from 125 Hz to 8 kHz were determined when using in-ear air conduction transducers and bone conduction transducers. Threshold-adjusted noise prescriptions were calculated to account for the headphones' output variability across the frequency range. A battery of measures were also taken to assess participants' relationship with tinnitus and their hearing to evaluate tinnitus therapy effectiveness by using questionnaires, otoscopy, pure tone audiometry, and psychoacoustic tinnitus tests.

No significant differences were found between the minimum masking levels required when using the two different transducers, including when compared at each frequency. This has implications for clinical practice – as bone conduction transducers are as effective as traditional air conduction transducers, clinicians should consider the potential advantages of bone conduction transducers for tinnitus therapy for clients who prefer not to be fitted with hearing aids, including the ability to maintain awareness of one's sound environment, and the

absence of occlusion that earphones cause. Significant correlations also infer that proportionately lower masking levels were required at frequencies with more hearing loss for both transducers but especially when delivered using bone conduction. Age may also be involved in this relationship; older tinnitus sufferers required less tinnitus masking at these higher frequencies with more hearing loss. Loudness recruitment is likely responsible. Proportionate distributions of sound should therefore be prescribed across frequencies when using hearing aids for sound therapy, especially for older patients.

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LITERATURE REVIEW

1. Hearing pathways

Sound can be propagated from the periphery to the brain using the air conduction (AC) and the bone conduction (BC) routes (Purves et al., 2008). While differing in their initial entry into the auditory pathways, the two transmission pathways later converge at the cochlea to relay auditory information to the brain. A comprehensive exploration of the auditory pathways is beyond the scope of this study hence will be limited to information that supports the study.

1.1 Air conduction

Sound creating objects elicit a disturbance of the particles in the air, causing them to oscillate in a longitudinal wave. This energy is transmitted through the medium until it reaches the ear. The air conduction route involves the contribution of the structures of the outer ear and middle ear. The outer ear consists of the pinna and the external auditory canal. These structures are designed to facilitate the collection of sound energy to focus it at the tympanic membrane for further propagation to the middle ear. Passive resonance allows sounds at around 3 kHz to be boosted in the human auditory canal due to the characteristics of the external auditory canal – which is particularly important for speech perception. The pinna and concha of the outer ear are also used for sound localisation by employing the asymmetrical nature of the pinna (Purves et al., 2008).

Due to the sound energy propagation, the tympanic membrane is displaced (Purves et al., 2008). An ossicular chain is connected to the membrane and hence further transmits this energy: through the malleus, along the incus, and finally to the stapes. The stapes footplate is also attached to the oval window, another membrane that precedes entry into the cochlea. The

ossicles are affixed with various ligaments, tendons (for the stapedius and tensor tympani muscles), and the annular ligament, which connects the stapes footplate to the oval window (Stenfelt & Goode, 2005b).

Once the sound has been transmitted through the outer ear and to the tympanic membrane, the medium of travel is now mechanical (Purves et al., 2008). Because of the difference in impedances between the outer ear and inner ear, the middle ear's function is largely centralised around optimising this energy transfer by mechanically matching the impedances. Firstly, the force from the tympanic membrane – which has a relatively larger diameter – is focused onto the oval window, which has a much smaller diameter (Pickles, 2015). Secondly, the ossicular chain performs a lever function to maximise the energy transfer into the inner ear. This can also be regulated by the middle ear's tensor tympani and stapedius muscles. Upon reflexive contraction of the muscles, the ossicular chain is stiffened and hence the amount of energy propagated is reduced (Purves et al., 2008).

At the inner ear's cochlea, the mechanical acoustic waves are transformed into neural signals that are then propagated to the brain (Purves et al., 2008). When the travelling wave displaces the fluid at the oval window, this energy is transferred along the inner ear's fluid, ultimately displacing the cochlear partition (including the basilar membrane) and fluid at the round window. The manner of transmission contributes to a frequency-tuned system, which is possible due to contributions from a number of designs. The basilar membrane is asymmetrical: the apical end is more flexible and wider, while the basal end is stiffer and narrower. This dictates the direction of energy transfer from the stiffer basal to the more compliant apical end. Upon stimulation, the travelling wave travels in this direction and gradually increases in amplitude while decreasing in velocity before culminating and subsiding. This point of maximal displacement is determined by the frequency: higher frequencies reach their peak

closer to the stiffer base, while for the lower frequencies, this is near the flexible apex. This enables a tonotopic arrangement, which is also facilitated by active biomechanical processes by the outer hair cells' Prestin proteins (Pickles, 2015).

Displacement of the basilar membrane also allows a shearing movement between the basilar membrane and tectorial membrane (Purves et al., 2008). Bending of the stereocilia of the hair cells on top of the basilar membrane can elicit receptor potentials to be converted into an electrical signal. Afferent neurons connecting to the brainstem and central auditory system are predominantly from the inner hair cells, whereas efferent neurons from these structures to the cochlea are largely innervated by outer hair cells (Pickles, 2015).

1.2 Bone conduction

Sound can also directly reach the cochlea via bone, rather than through the external and middle ear. Stimulation of the skull allows vibrations to be transmitted through bone, bypassing the external and middle ear, and travel to innervate the inner ear directly (Purves et al., 2008). This concept can be used to test for conductive components in hearing loss. If a conductive hearing loss is present, this would suggest that the site of lesion is in the outer or middle ear (Steiger, 2015). Upon bypassing the outer and middle ear using bone conduction, if the hearing loss persists, it can be inferred that the pathology lies either in the inner ear's cochlea or the auditory nerve pathways. Alternatively, it can be used to amplify sound for those with inaccessible or pathological middle or outer ears.

1.2.1 Mechanisms of bone conduction

The physiology of bone conduction is more complex than air conduction and hence has not yet been entirely understood (Stenfelt & Goode, 2005b). When a sound is transmitted via bone, vibratory elements are introduced to the skull. These cause vibrations of the skull in all

three planes, as well as rotational forces. There are several pathways by which sound can be transmitted to the cochlea through bone. The extent to which each pathway contributes to hearing is unknown (Dauman, 2013). However, this appears to be somewhat dependent on the frequency of the sound.

Some sound is radiated through the external ear canal via the osseotympanic mode (Stenfelt & Goode, 2005b). This occurs due to the vibration at the skull eliciting a deformation of the ear canal's walls. This causes a sound pressure and hence sound radiation, as it is henceforth propagated to the tympanic membrane as air conducted sound would be. If the sound is of a frequency lower than 1 kHz (approximately the first resonant frequency of the skull), no sound is radiated in the bony portion of the ear canal as the skull's bones move in unison (Hakansson et al., 1994; Stenfelt & Goode, 2005a). Radiation of lower frequencies using the osseotympanic mode will be largely dominated by contributions from the cartilaginous portion of the ear canal, which is also capable of radiating sound (Stenfelt et al., 2003).

Stenfelt & Goode (2005b) concluded that the contribution of the external ear canal component in bone conducted sounds was not significant in an unoccluded ear. However, this is not the case when the occlusion effect is present due to blockage of the ear canal's entrance, causing sound pressure in the canal to be increased for sounds below 1 kHz. In the occlusion effect, the properties of the presenting 'tube' are changed from an open tube to a closed tube, hence its resonance will also change.

Another transmission route for bone conduction is via the middle ear cavity (Stenfelt & Goode, 2005b). This can be through sound pressure in the middle ear cavity that is generated with skull vibrations, similar to the manner of transmission for external ear canal radiation.

However, no evidence has been found that this method provides a significant contribution (Stenfelt et al., 2002).

Alternatively, the inertia of the middle ear's ossicles can be considered (Stenfelt & Goode, 2005b). For low frequency bone conducted sounds, the various ligaments and tendons of the middle ear are moved in phase with the vibratory forces of the skull. However, at higher frequencies, the inertia from the ossicles' mass overcomes the stiffness of these ligaments and tendons. This results in movement of the ossicles relative to the surrounding bone. Specifically, the stapes footplate moves against the oval window, inducing cochlear fluid movement. The contribution of this component in bone conduction perception is especially significant at the resonant frequencies of the ossicles (1 - 3 kHz), although otherwise has minimal influence for low to mid frequency stimuli (Stenfelt et al., 2002).

The cochlear transmission route that provides the most contribution to bone conducted sound is via the inertial forces of the fluids (Stenfelt & Goode, 2005b). When the temporal bone is vibrated, this energy is propagated by the inertia of the cochlea's fluids. Fluids are not compressible hence this must be displaced elsewhere. The membranes on either end of the cochlea (the round window and oval windows) provide compliant barriers that can accommodate these fluid movements by either concaving in or bulging out in synchronicity with each other. Additionally, the pressure gradient formed between these two membranes allows the generation of a travelling wave on the basilar membrane. While this route is a major contributor to bone conducted sound perception at lower frequencies of up to 1 kHz, it appears to be less so for higher frequencies.

Although considerably less significant, the compression of the cochlear walls for bone conducted sounds could also be involved (Stenfelt & Goode, 2005b). When the skull is vibrated due to bone conduction, this causes the bone to be compressed and expanded (Dauman, 2013).

Again, due to the incompressible nature of the cochlear fluid, this bone displacement causes subsequent fluid bulging at the membranes and hence stimulation of the basilar membrane.

Again, while suspected to contribute much less than cochlear fluid inertia, cerebrospinal fluid has been thought to be involved in cochlear bone conduction propagation pathways (Stenfelt & Goode, 2005b). Cerebrospinal fluid exists between the brain and the skull and has a transmission route through the brain's ventricles and aqueduct system. It has been found that a static pressure is exerted from the cerebrospinal fluid to the cochlear fluids via the cochlear aqueduct, and that sound pressure can be passed into the inner ear in this way (Carlborg & Farmer, 1983). However, again, this appears to be a minute contributor from the cochlea to bone conducted hearing, in favour of the role that the inertia of cochlear fluid plays (Gopen et al., 1997; Stenfelt & Goode, 2005b).

This has included a brief explanation of some of the potential contributing routes, although many more are hypothesised. Evidently, bone conduction is a very complex process which can involve multiple routes of transmission.

1.2.2 Bone conduction properties

There are also several properties that are unique to bone conduction as a result of its nature of stimulation. For bone conduction, excitation of the skull stimulates both cochleae (Nolan & Lyon, 1981). Both cochleae are encased in bone cavities of one skull thus vibrations can be transmitted from the site of stimulation across the bones of the skull to stimulate the contralateral cochlea. There are very slight differences in the intensity perception at each cochlea due to its proximity to the stimulation site. This will result in differential damping of sound at either cochlea due to interference by structures, causing transcranial attenuation at the contralateral cochlea. The degree to which sound is attenuated depends on the frequency of the

stimulus, and also has large amounts of variation between individuals due to the differences in skull thickness causing damping (Lee et al., 2016). On average, there appear to be slightly larger amounts of transcranial attenuation at frequencies above 3 kHz than at lower frequencies. However, the interaural attenuation of bone conduction can be considered to be negligible for clinical practice as its contribution is minimal (Nolan & Lyon, 1981). Stimulation of either mastoid for bone conduction is hence deemed to affect both cochleae identically.

Given the vibratory nature of transmission involved in bone conduction hearing, the possibility of vibrotactile sensations also exists (Steiger, 2015). At low intensities, the vibrations delivered by the bone conductor may be sufficient to stimulate the cochlea but not enough to be detected as a somatosensory stimulus. As the transducer is designed to convey mechanical vibrations, these vibrations can become powerful enough at higher intensities to also be perceived on the skin and become multimodal in its perception. The intensity of the threshold at which this occurs depends on a number of factors. This can include variables related to the nature of the stimulus such as type of sound or noise presented, frequency, and site of stimulation (Freden-Jansson et al., 2017). Characteristics related to the individual receiving the bone conduction may also dictate the vibrotactile threshold such as their age (Verrillo, 1977), as does the transducer used including the surface area of the stimulation site and the force at which it is applied (Richter & Brinkmann, 1981; Freden-Jansson et al., 2017). Generally, there appears to be a wide range of vibrotactile thresholds among individuals (even if other variables are kept constant), especially at lower frequencies (Boothroyd & Cawkwell, 1970). Lower frequencies are much more sensitive to vibrotactile stimulation. At 125 Hz, studies by Freden-Jansson et al. (2017) showed that participants were able to report a tactile component at their mastoid at average thresholds of 13 dB HL. Comparatively, the vibrotactile threshold for 1000 Hz was 70 dB HL, and 87 dB HL at 2000 Hz.

Linearity behaviour of bone conduction through the skull is debated. Studies by Khanna et al. (1976) and Arlinger et al. (1978) provide evidence for non-linear sound transmission. They found that at the low frequencies and at louder sensation levels, there were significant harmonic distortions that resulted in non-linearity. To account for this, several explanations were proposed. These included the ideas that the non-linearity could be attributed to the natural asymmetry associated with the cochlear processes or that it may be explained by the non-linearity caused by skin and soft tissues of the area (Khanna et al., 1976). Skin can act as a viscoelastic barrier to sound transfer, differentially attenuating high frequencies of vibration (Stenfelt & Goode, 2005b). Alternative proposals include that disproportionate amplification may arise due to skull resonance characteristics (Arlinger et al., 1978). However, Hakansson et al. (1996) found evidence for the contrary. Bone conduction signals were directly measured *in vivo* by using the titanium fixtures on bone-anchored hearing aids. In doing so, the skin and soft tissues could be bypassed in the analysis of bone transmission. No significant non-linear behaviour was observed between 100 Hz to 10,000 Hz and up to intensities of 77 dB HL. The impedance of the skin at the mastoid, which is typically where bone conductors are placed for audiological practice, also showed no non-linear behaviour. Hakansson et al. proposed that the discrepancy between this study and previous studies could perhaps be attributed to non-linear behaviour of the transducers and measuring methods that had not been accounted for in the prior studies. Despite this finding supporting the linear behaviour of bone conduction, transmission of complex low frequency acoustic stimuli or sounds at high intensities using this pathway may still produce non-linearity (Henry & Letowski, 2007). Signals of low frequency are especially susceptible to temporal distortions, possibly due to inertial movements of the head at these frequencies. Consequently, delivery of a complex signal to either cochlea may differ by as much as 1.5 ms. Intensities exceeding 77 dB HL may also elicit non-linearity. To

minimise this likelihood, should the signal need to be delivered at a high intensity, presentation of signals that are comprised of only mid to high frequency is recommended.

1.3 Comparison of bone conduction and air conduction perception

The similarities and differences between the routes for air and bone conduction in the propagation of sound will be explored in this section. Once the medium for sound conduction – whether bone or air – has been traversed and the sound energy has been transmitted into the cochlea, the fundamental mechanism of stimulation at the initial levels of the auditory pathway appears to be largely similar (Zwislocki, 1953b; Stenfelt & Hakansson, 2002). Excitation generated on the basilar membrane of the cochlea was able to be cancelled out when travelling waves of air and bone conducted origin were generated 180˚ out of phase with each other. This was reported with both subjective cancellation of the auditory stimulus and also objectively via cochlear microphonics (Stenfelt & Hakansson, 2002; Wever & Bray, 1936; Khanna et al., 1976). Evidently, the mechanism by which the basilar membrane is displaced is similar for both air and bone conducted simulations, including that the waves travel from the region of most to least compliance – that is, the cochlear base to the helicotrema – which dictates the point at which the greatest displacement of the membrane will occur for the specific frequency (Stenfelt & Hakansson, 2002). Additionally, two-tone distortion products (indicative of cochlear outer hair cell activity) are able to be produced using either a bone conducted or an air conducted tone (Purcell et al., 1998; Stenfelt & Goode, 2005b).

The frequencies at which air and bone conduction systems are most sensitive to also appear to also be similar (Henry & Letowski, 2007). For both, humans are most sensitive to frequencies that are prevalent in speech: around 1000 Hz to 4000 Hz. Comparatively, they also show less sensitivity to low frequency sounds below 500 Hz. Studies were limited to measuring

bone conduction thresholds up to only 4000 Hz, as audiological equipment is typically only calibrated to measure bone conduction up to this frequency.

However, despite the fundamental similarity in the mechanism of basilar membrane excitation, there also appear to be differences between air conduction and bone conduction stimulations. Differences are present in the sensitivity between air conduction and bone conduction. The thresholds at which low frequency sounds can be detected show some variation in observed differences (Henry & Letowski, 2007). A range from 50 dB to 100 dB of difference has been reported (Zwislocki, 1953a; Henry & Letowski, 2007; Sklare & Denenberg, 1987) These variations could in part be due to variables used in the studies such as frequency of the stimulus used and transducers used. Differences in sensitivity to high frequencies are also present between delivery by bone conduction and air conduction transducers. The frequency range of human hearing is broadened when using a bone conduction transducer to include ultrasonic sounds of up to 100 kHz compared to air conduction, which is limited to around 20 kHz (Lenhardt et al., 1991). There is some speculation that this ultrasonic range of hearing is facilitated by activation of alternative hearing pathways such as by directly stimulating the brain or cerebrospinal fluid within the skull cavity (Henry & Letowski, 2007).

The two modes of acoustic transfer also produce dissimilarities in loudness. This variation is reported using both objective and behavioural data. Comparing the amplitudes of otoacoustic emissions generated by air conduction and bone conduction, for the same increase in stimulus intensity, the emission amplitude showed a larger increase for bone conduction (Rossi et al., 1988). Additionally, for the same reduction in stimulus intensity, the latency of a Jewett wave V on an auditory brainstem response generated by bone conducted sound increased more than for an air conduction stimulus (Schratzenstaller et al., 2000).

Similarly, level differences have been found behaviourally. Military personnel using bone conduction headsets performed better in their speech recognition compared to those using air conduction headsets, regardless of their profile of hearing (Manning et al., 2016). These level differences were also apparent when comparing the perceived air conduction and bone conduction loudness via loudness matching using narrowband noise (Stenfelt & Hakansson, 2002). A difference of 6 - 10 dB was found for the lower frequencies of 0.25 - 0.75 kHz for both normal-hearing participants and for those with mild-to-moderate sensorineural hearing loss. At the higher frequencies of 1 - 4 kHz, the difference was still present but reduced to 4 - 5 dB. This suggests bone conducted noises have comparatively expanded loudness growths for frequencies below 1000 Hz. This is suggested to be attributed to the differences in the mechanisms of bone transmission, rather than due to differences in basilar membrane propagation. The non-linear reluctance of the bone conduction transducers used may allow the introduction of distortion of energy such that the energy becomes distributed over a wider range of frequencies. As a result, this could be perceived as louder, especially when presented at higher intensities at the lower frequencies. However, the reported results indicate that this difference was present throughout the entire range of intensities and frequencies tested, not solely at the parameters where this phenomenon exists. Likewise, vibrotactile responses that employ multimodal stimulation via both somatosensory and acoustic senses to increase the perceived intensity level were explored and subsequently rejected as the sole explanation. While the intensities delivered at the lower frequencies were at predominantly vibrotactile suprathreshold levels, the range of frequencies and intensities tested suggests that vibrotactile responses were not solely responsible for the differences seen, as the higher frequencies (> 1 kHz) were at vibrotactile subthreshold levels (Stenfelt & Hakansson, 2002).

The level differences present between air conduction and bone conduction could be explained by the physiological variations in their transmission paths. While the air conduction

route involves a comparatively straightforward path through the ear canal and middle ear to the cochlea, bone conduction propagation can involve many additional pathways that may include other structures (refer to section **1.2.1 Mechanisms of Bone Conduction**). The stapedius reflex involves the contraction of the stapedius muscle, causing stiffening of the stapes and annular ligament and hence attenuation in energy propagation to the cochlea. This can reduce the air conduction thresholds at frequencies lower than 1 kHz by up to 20 dB and can be activated by narrowband noise at intensities as low as even 40 dB HL (Neuman et al., 1996; Stenfelt & Hakansson, 2002). Otosclerosis is a similar situation of cochlear energy propagation reduction due to ossification of the stapes footplate. Again, for these patients, their sensitivity via bone conduction is almost unchanged while air conduction thresholds become poorer. This disproportionate impedance of energy by activation of the stapedius muscle for the air conduction transmission route only could account for some of the contrast seen in the sound level difference, especially as some of the stimuli were presented at levels that could elicit the stapedius reflex (Stenfelt & Hakansson, 2002). Similarly, contracting the tensor tympani results in ossicular chain and tympanic membrane stiffening, thus attenuating sound propagation for low frequency air conducted sounds, but leaving bone conduction sensitivity largely intact. Notably, this reflex is tactilely activated. Some of the sounds presented were at vibrotactile suprathreshold levels and hence the vibrotactile response may have been elicited and as a result, attenuated the air conduction threshold at lower frequencies (Stenfelt & Hakansson, 2002).

1.4 Central hearing pathways

Information transmitted using air conduction and bone conduction converges at the cochlea (Purves et al., 2008). After the level of the cochlea, auditory information is propagated to the brain to be perceived by the individual. Auditory information encoded via action potentials ascends up the pathways, through the spiral ganglion cells, up the auditory nerve

(cranial nerve VIII), and into the brainstem. Here, innervations branch at the first structure of the central auditory system: the cochlear nucleus (Pickles, 2015). Branches are extended to the antero-ventral cochlear nucleus, the postero-ventral nucleus, and the dorsal cochlear nucleus. The function of this complex is related to sound localisation of the vertical plane using inter-aural timing and level differences. However, the dorsal cochlear is also a major integrative centre for senses, including somatosensory pathways (Basura et al., 2012). From the cochlear nuclei, information is transmitted binaurally to the superior olivary complex (including the lateral superior olivary, medial superior olivary, and the medial nucleus of the trapezoid body) or monaurally to the nuclei of the contralateral lateral lemniscus (Pickles, 2018). The information from these structures is integrated at the inferior colliculus, along with further projections from the cochlear nucleus. The inferior colliculus contains central and external nuclei. Of these, the external nuclei of the inferior colliculus are predominantly involved in multisensory integration. These nuclei receive projections from multiple regions, including the primary somatosensory cortex, trigeminal nuclei, and visual inputs (Wu et al., 2015). Subsequently, the medial geniculate nucleus of the thalamus acts as a relay station, and also reciprocally communicates with the cortical regions. Being a relay station, the medial geniculate nucleus also receives projections from other systems such as the somatosensory and visual systems and may hence facilitate multisensory interactions (Schroeder & Foxe, 2005). Finally, the information reaches the auditory cortices located bilaterally in the upper temporal lobes (Pickles, 2015). Throughout the central auditory pathway, tonotopicity is preserved from the frequency decomposition at the basilar membrane such that the primary auditory cortex is set in a tonotopic array corresponding to the frequency arrangement from the cochlear apex to the base. The belt areas of the auditory cortex are also tonotopically arranged but less precisely, receiving input from both the medial geniculate area's belt areas and the primary auditory cortex (Purves et al., 2008).

2. Introduction to tinnitus

Tinnitus is defined as the involuntary and phantom perception of sounds, despite no external source of sound. Chronic tinnitus – which is characterised as tinnitus that persists for more than 6 months – affects approximately 10 - 15 % of the general adult population (Baguely et al., 2013), with it becoming increasingly prevalent with age (Stohler et al., 2019). Tinnitus may also be accompanied by a range of manifestations, which often may include other auditory effects such as hearing loss or hyperacusis (extreme sensitivity to sound due to distorted loudness perception), as well as affective disorders and psychological distress (Eggermont & Roberts, 2004; Moller, 2011; Stohler et al., 2019). Some patients report a decline in their quality of life as a result of insomnia, anxiety, depression, and social burdens caused by their tinnitus. Others, however, remain unbothered by its presence. It has been thought that the lack of control over one's tinnitus may aggravate these associated negative perceptions, as those with gaze evoked tinnitus generally appeared to not find it as intrusive (Lockwood et al., 2001).

Tinnitus is very heterogeneous in the range of psychoacoustic characteristic presentations, including in its intensity and description (Moller, 2011). Generally, it may be described to be localised either centrally so it appears to originate from within the head, or it may be heard in either one or both ears (Baguely et al., 2013). However, some patients describe their tinnitus to have an external source. The frequency may also vary; complaints of high pitched cicada-like sounds are common, as are ringing, hissing, sizzling, or buzzing noises. Other perceptions of tinnitus are also possible. The tinnitus intensity perceived by patients can also differ vastly (Moller, 2011). However, the loudness of an individual's tinnitus appears to not necessarily be related either to how it affects their quality of life (Han et al., 2020) or perceived severity of tinnitus as reflected using questionnaires (Zagolski & Strek, 2014). It may also be constant or

pulsatile in its presentation, and it may be intermittently or continuously present (Moller, 2011).

Tinnitus percept can be also categorised as being subjective or objective (Han et al., 2020). In most patients, a subjective tinnitus is reported as it can only be heard by the individual with tinnitus. However, objective tinnitus has also been reported wherein their tinnitus can be heard externally by another individual. The aetiology of this may be related to a mechanically induced origin with vascular, muscular, or temporomandibular joint involvement as sounds produced by the body – such as blood turbulence or muscle contraction – are conducted and hence heard by the ear (Han et al., 2020; Moller, 2011).

Subjective tinnitus has been associated with many aetiologies. These include from head or neck trauma, ototoxic medications such as cisplatin and aminoglycosides, and excessive noise exposure (Zagolski & Strek, 2014). Consequently, it has been suggested that tinnitus is not a disease in itself but rather a symptom of another pathology. Risk factors for developing tinnitus are also diverse (Baguely et al., 2013). Hearing loss is most commonly implicated. However, the relationship between hearing loss and developing tinnitus appears to be rather complicated. Many patients with hearing loss do not develop tinnitus, while some patients with bothersome tinnitus have hearing thresholds at all frequencies of the audiogram that are within clinically normal ranges. Other potential risk factors that have been suggested include alcohol consumption, obesity, and smoking.

2.1 Tinnitus mechanisms

Tinnitus is typically associated with a pathology of the auditory nerve or ear. However, studies of auditory nerve ablation to remove peripheral input show that tinnitus percept can still persist (House & Brackmann, 1981). Hence although pathology of the cochlea may be a

catalyst for tinnitus generation, the resulting cascade of changes at higher central auditory structures appears to be more heavily responsible for tinnitus (Baguley et al., 2013). It appears that the phantom noise perception may be generated by neuroplastic mechanisms in response to the auditory deprivation from the periphery, such as when hearing loss is present. The resulting hearing loss reduces or entirely eliminates auditory input to higher centres, and consequently, neural remodelling occurs.

Due to their tonotopic organisation, the affected deafferented neurons will have characteristic frequencies specific to the frequency associated with the hearing loss (Weisz et al., 2007). These neurons will therefore likely be less effective at inhibiting the excitatory input from the undamaged neighbouring neurons. Consequently, the gain to the neighbouring neurons – including in the primary auditory cortex – will be increased via compensatory mechanisms in a frequency-specific manner and will now have an increased firing rate and are over-represented. Increased spontaneous firing rate in those with tinnitus is a well-documented occurrence in those with tinnitus (Baguley et al., 2013; Norena & Eggermont, 2003). Central gain enhancement refers to this idea that as a result of the reduced frequency specific stimuli, compensatory increases will occur at these adjacent frequencies, causing tinnitus (Norena, 2011). Cortical reorganisation can therefore occur: the damaged neurons can no longer optimally activate their target cell and are also not as effective in their inhibition of neighbouring excitatory activity (Eggermont & Roberts, 2004). This is a long-term inhibitory effect that can occur due to the neuroplastic nature of the brain. Initially, this may occur at the level of the auditory cortex but may also spread to also occur at other regions such as the dorsal cochlear nucleus (Kaltenback, 2006; Han et al., 2020). However, consideration of the contradicting timeframes associated with tinnitus and increased spontaneous firing rates casts doubt as to whether this mechanism can be attributed for the generation of tinnitus (Baguley et

al., 2013). Tinnitus onset can be immediate after exposure to a loud noise (Norena & Eggermont, 2003). In contrast, the neural processes causing neuroplasticity that underlie increased spontaneous firing rates and hyperactivity can require between hours to days.

Along with hyperactivity, a neural correlate of tinnitus includes an increase in the neural synchrony. Increased synchrony has been found to be present in the auditory cortex and also the cochlear nerve (Martin et al., 1993; Ridder et al., 2007; Cazals et al., 1998). This is consistent with an increase in burst firing of multiple structures throughout the auditory system in those with tinnitus, including in inner hair cells (Puel et al., 2002), secondary auditory cortex (Eggermont & Kenmochi, 1998), dorsal cochlear nucleus (Chen & Jastreboff, 1998), and the extralemniscal system (Eggermont & Kenmochi, 1998; Ridder et al., 2007). With respect to the timeframe of tinnitus onset, this is more consistent than the possibility of hyperactivity involvement in tinnitus generation. After exposure to a loud noise, immediate temporal synchrony of firing patterns was found (Seki & Eggermont, 2003; Norena & Eggermont, 2003). This was reported to be present in multiple neurons in the primary auditory cortex. In particular, this was noted to more heavily affect the neurons with corresponding characteristic frequencies that had been impacted by the noise. Spatially, the neurons with elevated neural synchrony tend to also have modified frequency tuning characteristics due to aberrant cortical tonotopicity. In those with hearing loss, neurons with characteristic frequencies affected by auditory deprivation tended to instead emulate the tuning characteristics of neighbouring neurons that were not affected as much. Tinnitus generation is hence hypothesised to be dependent on this expansion at the audiometric edge. However, it should be noted that no correlations were noted between the pitch of an individual's tinnitus and their audiometric edge (Shekhawat et al., 2013). Rather, a weak positive correlation was noted between the pitch of their tinnitus and frequency at which the individual's hearing threshold was around 50 dB HL.

The “inhibitory gating mechanism” refers to the idea that tinnitus occurs when the inhibitory feedback loop at the paralimbic level that typically eliminates the tinnitus signal is compromised (Rauschecker et al., 2010; Han et al., 2020). It is thought that this occurs at the level of the thalamus, allowing the resulting tinnitus signal to be transmitted to the auditory cortex and subsequently perceived. The introduction of a triggering factor such as anxiety or emotional stress reduces the threshold at which tinnitus will be perceived (Guitton, 2012). In addition to this, if the inhibitory gating mechanism is disrupted, the tinnitus will become unmasked and is hence perceived. Tinnitus perception in individuals can cause anxiety or other emotional stress, which causes a vicious cycle in which the tinnitus is exacerbated and becomes increasingly more aggravating for the patient. However, if the patient habituates or adapts to the tinnitus such that it no longer causes emotional distress, this cycle is disrupted and the tinnitus perception is attenuated.

Psychological influences on tinnitus perception and emergence affirm the idea of a more central involvement in the generation of tinnitus. Although hearing loss and tinnitus often present together, the onset of tinnitus is frequently delayed and better correlates to a distressing event than the hearing loss. This can be explained when considering the involvement of the brain, rather than purely at a peripheral level. Aberrant auditory signals that would normally be masked out are no longer done so, resulting in this tinnitus perception (Guitton, 2012).

The involvement of comorbidities such as emotional correlates with tinnitus perceptions suggests that the involved brain regions are not exclusive to auditory areas (Han et al., 2020). Using techniques such as electroencephalography and MRI, various studies have found multiple neural networks and regions that are involved (Vanneste & De Ridder, 2012; Besteher et al., 2019). These include the prefrontal cortex, hippocampus, anterior and posterior

cingulate cortices, and precuneus, among many other regions. This highlights the importance of regarding the patient holistically in conjunction with their tinnitus, as the activity and changes of non-auditory neural networks appear to be heavily involved with the auditory networks in tinnitus. As discussed, for tinnitus perception, it is thought a triggering factor such as an undesirable event (emotional stress, physical or mental illness, noise exposure, etc.) may need to occur in conjunction with existing cochlear damage (Han et al., 2020).

Involvement of the limbic system is thought to be crucial for the chronic maintenance of tinnitus. Application of salicylate (a substance which causes tinnitus) resulted in modulation of not only central auditory structures, but also the limbic structures - especially in the amygdala (Wallhausser-Franke et al., 2003).

As tinnitus characteristics, perception, and aetiology can all vary vastly between individuals, the mechanism of its generation is also likely very heterogeneous.

2.2.1 Tinnitus and somatosensation

Tinnitus aetiology has also been found to be related to head or brain injuries and trauma for 8% of those with tinnitus (Henry et al., 2005). This may be due to modulation of the typical extra-lemniscal auditory pathways due to the trauma. Consequently, activity of the trigeminal nerve – which is responsible for somatosensation of the face – may be disrupted thus neural activity to the dorsal cochlear nucleus can be altered. In turn, this can affect the associations between tactile and auditory sensations (Itoh et al., 1987; Shore et al., 2000; Eggermont, 2007).

2.2.2 Somatic tinnitus

For those with tinnitus, some find that the perceptions of their tinnitus characteristics can be modulated in response to physical contact or movement, and hence this has been sub-

categorised as somatic or somatosensory tinnitus. Typically, this somatosensory integration is focused on areas around the neck or head for 80% of tinnitus sufferers (Levine et al., 2003; Eggermont, 2007) – including contraction of the neck, eye, tongue, face – and 33% can alter their tinnitus perception using jaw movements (Rubinstein, 1993). Furthermore, contraction of these areas has been found to be able to elicit tinnitus in 60% of those with no previous complaints of tinnitus, even in participants who are profoundly deaf (Levine et al., 2003).

It is suggested these alterations to tinnitus perception can be attributed to projections from areas of the cochlear nucleus (specifically, the trigeminal and dorsal systems), which is heavily documented to also be otherwise involved in multisensory integration (Wu et al., 2015).

The successful modulation of tinnitus via somatosensory input suggests that not only are there intricate connections between tinnitus, multimodal integrations, and somatosensory systems, but also that the site of tinnitus generation involves the central auditory pathway (Simmons et al., 2008). To investigate the brain regions activated during induced tinnitus, participants that could voluntarily modulate their tinnitus by clenching their jaw were recruited and compared with normal individuals without tinnitus (Lockwood et al., 1998). Cerebral blood flow was analysed with positron emission tomography (PET) imaging. While the areas associated with the jaw movement showed increased activity in all participants, there were certain neural activities that were seen exclusively in the tinnitus participants in this study. A significant increase in activity was seen in the left primary auditory cortex and the area between the medial geniculate bodies for the two participants who experienced an increased loudness in their unilateral right-sided tinnitus perception with the jaw clenching. Conversely, for the two participants who reported a reduction in their tinnitus loudness, decreased activity was found in the contralateral middle temporal gyrus (where both the primary and association

auditory cortices are located) and the left hippocampus. Evidently, propagated information is integrated at higher levels – somatic modulations are translated as and associated with altered auditory perceptions of tinnitus. Lockwood et al. (1998) also noted that while any 'real' external stimulus presented to a single ear caused increased stimulation of bilateral auditory cortices, changes due to tinnitus appear to only affect one hemisphere's activity. It can thus be inferred that the tinnitus generation site occurs at the central level – either the medial geniculate or the auditory cortex.

3. Tinnitus therapy

While a method to eliminate tinnitus completely has yet to be found, tinnitus management and reduction strategies exist. There is some evidence that the efficacy of tinnitus treatments may be reduced in those suffering with tinnitus for more than six years, especially as fundamentally, chronic tinnitus appears to be dissimilar to acute tinnitus (Moller, 2011). Current therapies include educational counselling, cognitive behavioural therapy, tinnitus retraining therapy, masking, and the use of hearing aids (Fuller et al., 2017; Phillips & McFerran, 2010; Sereda et al., 2018). Acoustic therapy such as via noise generators for masking and hearing aids facilitates access to external sounds, which may encourage neural reorganisation due to changes in central auditory system activity and hence long-term attenuation of tinnitus perception (Jastreboff, 1990). A systematic review by Sereda et al. (2018) show that sound therapy delivered with either hearing aids (to provide either solely amplification or both amplification and masking) or sound generators both produce clinically significant reductions in the severity of patients' tinnitus (as measured using the Tinnitus Handicap Inventory questionnaire). However, the benefit provided to tinnitus severity and loudness from using sound therapy in any form (including masking generators and hearing aids) was not significantly different to that from other tinnitus interventions such as solely tinnitus

counselling (Hobson et al., 2012). Due to its pertinence, only masking methods will be further detailed.

Typically, tinnitus masking is delivered in conjunction with educational therapy. Tinnitus masking involves the introduction of an external acoustic stimulus that is played continuously to cover the sound of the individual's tinnitus. This provides a non-invasive and immediate tinnitus relief option by decreasing the tinnitus signal-to-noise ratio to make their tinnitus less conspicuous. Additionally, noise generators allow more control to the patient, which can help to relieve some of the frustrations caused by tinnitus. With time, noise generators can also help the patient to habituate to their tinnitus by teaching them to ignore it so that it becomes gradually less bothersome. In a longitudinal study over the course of 6 - 48 months involving 50 patients that used sound generators for their tinnitus, a significant reduction in Tinnitus Severity Index scores was reported, as well as a significant decrease in their self-rated tinnitus loudness (Folmer & Carroll, 2006).

3.1 Tinnitus masking mechanisms

Masking may be either informational or energetic. Energetic masking occurs due to the masking noise interfering with the auditory processes at the level of the cochlea, which in turn alters the resulting signals perceived at the brain. Informational masking includes the involvement of higher structures rather than solely peripheral interactions, which ultimately contributes to neural interference (Brungart, 2001; Bennett et al., 2013).

Tinnitus masking patterns also appear to show lots of variations. Feldmann (1971) categorised subjects into five different groups according to their masking pattern. The fairly common 'convergence' type appears to be characteristic for those with sloping high-frequency

hearing losses and high pitched tinnitus, and is defined by the convergence of the masking and threshold curves at the tinnitus pitch. At the lower frequencies, a fairly high sensation level was required for the tinnitus to be masked, whereas the higher frequencies were masked at low sensation levels. The rare 'divergence' type showed an increasing distance between the masking and threshold curves with frequency – masking at the lower frequencies tended to require a lower sensation level than at the higher frequencies. Those with the 'congruence' type were able to achieve masking at an intensity that was only slightly higher than their thresholds with either a tonal or narrow-band noise masking stimulus. The 'distance' type was used to describe subjects who required masking to be presented at an intensity that was consistently much higher than their thresholds. This pattern was often seen for those with a pathological middle ear. Finally, the 'resistance' type produced unsuccessful masking regardless of the stimulus presented. This was the case for most subjects with profound sensorineural hearing loss, suggesting that external masking could not overcome the pathology to be perceived.

Masking can be used for tinnitus therapy to achieve different purposes. Tinnitus masking can be used to entirely cover the tinnitus to achieve total masking (Henry et al., 2006). This is possible when sufficient input of the external masking stimulus is delivered so that the tinnitus can no longer be heard. Alternatively, a lower intensity of masking is used to achieve partial masking at the point that provides the greatest tinnitus relief. Partial masking occurs when both the tinnitus and masking stimuli can be heard, but the perceived intensity of the tinnitus is reduced due to the presence of background noise (Tyler, 2006). The amount of masking input that may be delivered creates a spectrum leading to total masking. The mixing point is the point on the continuum where the patient's tinnitus and the external masking begin to mix or interact, which occurs at approximately 90% of the minimum masking level (Searchfield et al., 2012; Huang et al., 2006). This point is proposed to be the most effective

level for tinnitus retraining therapy (Jastreboff & Jastreboff, 2006). Unlike total masking, the objective of sound use in tinnitus retraining therapy is to gradually habituate the patient to their tinnitus by allowing them to hear both the external masking and tinnitus concurrently. However, some evidence also suggests that the mixing point offers no superior benefit compared to a stimulus delivered at a different level (Tyler et al., 2012; Tyler, 2006).

Ultimately, both masking and tinnitus retraining therapy have been found to effectively provide a sense of relief from tinnitus (Henry et al., 2006). Whereas the effects of total masking appear to be immediate and stable, tinnitus retraining therapy provides a more sustained benefit that becomes incrementally more pronounced with continued treatment (Henry et al., 2006). However, total masking may also be implicated with long-term benefits by breaking the vicious cycle of tinnitus and associated anxiety by enabling it to become less obtrusive (Tyler, 2006).

Additionally, some individuals report instances of residual inhibition, wherein the participant retains relief from tinnitus after the conclusion of the presentation of the masking stimulus that is extended for a brief duration (Jonsson et al., 2016). The reported period of tinnitus relief varies, but typically appears to persist for around 5 to 30 seconds (Roberts et al., 2008; Vernon & Meikle, 2003b; Galazyuk et al., 2016). Some patients report residual inhibition from exposure to masking on the contralateral ear or after implantation of a contralateral cochlear implant, suggesting that the mechanism for this likely occurs at a central level (Perez-Carpena et al., 2021). It is thought residual inhibition occurs as it disrupts the mechanism that generates tinnitus by interfering with the synchronicity (Roberts et al., 2008). A systematic review including 1066 patients concluded that 34.5% of participants report complete residual inhibition, in which the tinnitus remains entirely masked after the exposure (Perez-Carpena et al., 2021), and around 80% report at least some residual inhibition (Galazyuk et al., 2017). With short-term external masking exposure, the duration that the patient experiences residual

inhibition appears to increase as the duration of external stimulation increases (Perez-Carpena et al., 2021).

3.2 Bone conduction for tinnitus masking

The presence of multiple transmission pathways for auditory propagation to the brain introduces the potential for using bone conduction as a route to deliver tinnitus masking. Compared to the properties associated with air conducted acoustic transmission, this pathway presents some pragmatic advantages. This pathway remains an accessible option including for those with conductive hearing losses (characterised by pathology of either the outer and/or middle ear), as the manner of transmission bypasses the outermost portions of the hearing system and instead directly stimulates the inner ear's cochlea (Holgers & Hakansson, 2002). As a result, it remains a feasible approach to mask tinnitus, even for those with conductive hearing loss components. Similarly, as the site of excitation is located on the skull and requires no ear moulds or other insertion into the canal, those that physically cannot tolerate foreign objects in the canals due to pain, allergies, chronic discharge, or canal malformations are able to consider this manner of masking (Holgers & Hakansson, 2002). Additionally, due to the lack of inserted ear moulds, the user will experience no sensations of aural fullness or the occlusion effect (in which the wearer's own voice is perceived to be louder due to the vibrations of the voice stimulating the bone and being heard via bone conduction) (Holgers & Hakansson, 2002). Because the canals are not obstructed, sounds can be perceived via bone conduction in a route parallel to sound entering normally via the ear canals. Environmental sound can hence be perceived more clearly as sound energy into the outer and middle portions of the pathway is not attenuated (Holgers & Hakansson, 2002). Hearing protection can also be worn in addition to the bone conduction device if the user is in an environment that requires it (Walker & Stanley, 2005). Furthermore, because sound is propagated through the bones to the cochleae on either

side, masking via bone conduction requires only a single point of contact (although the perceived intensities at either cochlea may be slightly different). This is especially advantageous for those with bilateral tinnitus (Holgers & Hakansson, 2002).

Both tinnitus and bone conduction are often described to be 'heard' from within the head or skull, rather than being perceived as an external stimulus. While the potential implications of this association are yet unknown, this could provide another potential advantage in targeting tinnitus that masking via air conduction cannot (Holgers & Hakansson, 2002). Holgers and Hakansson (2002) showed that masking via bone conduction can be used to deliver effective tinnitus relief. Eight participants with significant conductive hearing losses and bone anchored hearing aids (BAHA) were recruited, and a bone anchored sound stimulator was attached on the implantation site to provide tinnitus masking. The participant with the most severe tinnitus reported the most benefit, especially when using it as a sleeping aid. In contrast, the other seven participants recruited reported only modest benefit. These participants, however, had mild-to-moderate tinnitus and perhaps had already habituated to it. It was also reported that this manner of providing masking was unfavourable and thus most participants did not use the bone anchored sound stimulator. This was because it did not have a microphone. Consequently, participants' access to environmental sounds was severely reduced, especially as all participants had significant conductive hearing losses and very little access to any unaided hearing at all. While this shows that the bone conduction route can be used to provide tinnitus masking, investigation of either an alternative excitation position independent of the BAHA implantation site or incorporating a microphone unit into the bone-anchored sound stimulator may prove beneficial.

Tinnitus masking delivered through air conduction headphones and bone conduction headphones were also compared (Jeong & Jin, 2020). Twenty chronic tinnitus sufferers were randomly assigned to receive sound therapy through one of these two transducers. Broadband noise masking was delivered at the mixing point for three months, with instructions recommending daily therapy in quiet for at least three hours (although participants' compliance with the programme was not reported). Achieving multimodal stimulation by eliciting vibrotactile responses with the bone conduction transducer was not the aim, and the masking stimulus was neither ear-specific nor frequency-specific. Tinnitus relief was measured subjectively using the Korean Tinnitus Primary Function Questionnaire and visual analogue scales before treatment and again three months post-treatment. These analysed the self-perceived effects of participants' tinnitus on categories such as their concentration, emotion, hearing, and sleep. All subcategories and overall scores of these assessments showed significant decreases post-treatment for both air conduction and bone conduction headphones, suggesting that they can both effectively provide tinnitus relief. However, no significant differences were found for the participants' subjective relief between the two transducers used.

Ultrasound tinnitus masking through bone conduction has also been proposed for tinnitus therapy. While sounds at these frequencies cannot be detected by air conduction, responses are possible through bone conduction up to 100 kHz (Lenhardt et al., 1991). The frequency at which tinnitus is most effectively masked is not dictated by a patient's tinnitus frequency – in fact, tinnitus can be masked by noise that does not contain the frequency band of the tinnitus frequency (Kitajima et al., 1987). Ultrasound masking is also advantageous as masking delivered at ultrasound frequencies does not compete with the speech frequencies, allowing patients to engage in both simultaneously (Goldstein et al., 2001).

A number of ultrasound studies were performed using the bone conduction device, the UltraQuiet device. The UltraQuiet device is a commercial product that is targeted at tinnitus patients. It delivers ultra-high frequency masking of greater than 10 kHz (10 - 20 kHz) via bone conduction at the mastoid (Goldstein, Lenhardt, & Shulman, 2005). Participants with hearing thresholds equal to, or less than 50 dB at audiogram frequencies between 10 - 14 kHz are suggested to likely report the most relief in response to this device (Goldstein, Shulman, & Lenhardt, 2005). Significant improvements as per tinnitus questionnaires were noted for all participants after bi-weekly sessions using the device for four weeks (Goldstein et al., 2001). These improvements were noted to vary in the duration of their persistence from 1 hour to 4 weeks. However, it should be noted that statistical analyses for this study were not provided in the article. Long-term efficacy of the UltraQuiet was also specifically examined and it was suggested that participants may retain sustained relief for at least two months after the treatment (Goldstein, Lenhardt, & Shulman, 2005). All fifteen participants showed an increase in tinnitus relief for at least two months afterwards – around 75% of patients reported a benefit from the device, and the remaining participants indicated that their perceived tinnitus intensity or severity was reduced. Generally, for the group, the questionnaires showed significant improvements in tinnitus severity after the intervention, although tinnitus intensity and annoyance did not significantly change. Minimum masking levels also decreased, especially from 3 - 6 kHz. Residual inhibition was also recorded during the sessions for six of the fifteen participants, which lasted around 5 minutes on average.

Neural activity after 5 - 7 weeks of exposure to the UltraQuiet device was also investigated using PET scans (Shulman et al., 2004). Subjectively, similar results were reported as prior studies: significant tinnitus relief of varying amounts was noted when using questionnaires, and the minimum masking levels (MMLs) of the participants were significantly decreased. Pre- and post-treatment PET scans were used to compare for significant differences

in the twelve stated regions of interest (including the bilateral thalami, cerebellums, and areas of the lobes (frontal, temporal, parietal, auditory)). Shulman et al. (2004) reported that no significant differences were found in these examined regions, except at the cerebellum. Here, significant interhemispheric differences were found before the therapy but these were no longer present after. Shulman et al. state that the findings of differences in PET scans, MML, and questionnaires before and after the treatment suggest that the device elicited reprogramming of the cortical matter. However, a critical review by Tucker (2013) noted a statistical incongruency regarding the significance level used by Shulman et al.; both of the results before and after the treatment actually showed no significant interhemispheric differences in the cerebellum. PET scans were also performed on the insula cortex after bi-weekly treatments using the UltraQuiet device for eight weeks (Lenhardt et al., 2008). Again, the majority (five of the six participants) documented relief from their tinnitus as per their questionnaire results, and all demonstrated significant decreases in their MML. Statistical analyses were not provided, but it was reported that there were some differences in the activity of the insula cortex after the treatment. More specific and objective evidence would prove beneficial.

The repeated effectiveness of the UltraQuiet device shows that the bone conduction pathway can be used to provide sound therapy associated with benefits, including in psychoacoustic perceptions via questionnaires, residual inhibition, and also reductions in minimum masking level. However, Tucker (2013) states that the results of these studies should be interpreted with caution due to several flaws in their design. Some examples of these include the general lack of (adequate amounts of) randomisation, control groups, blinding, and their small sample sizes. Tucker also notes a conflict of interest as the authors are affiliated with the commercial UltraQuiet device. Overall, Tucker believes that insufficient clear evidence supporting the beneficial effects of the UltraQuiet device on tinnitus therapy were determined,

and hence a change in the current protocols to accommodate bone conducted ultra-frequency tinnitus therapy appears to not be warranted (Tucker, 2013).

Other studies involving ultrasound delivered to the mastoid bone also show varying success in producing tinnitus relief. A double-blinded experiment and placebo was used in this investigation: one device emitted a low dose of 500 kHz noise while the other appeared to be identical but delivered no signal (Carrick et al., 1986). Each participant was exposed to a device for a ten minute session, followed by another session with another device approximately one to two months later. Results agreed with studies by Goldstein et al. (2001, 2005) and Shulman et al. (2004) that significantly more benefit (determined subjectively) was produced by the ultrasound device than the placebo. However, in contrast, the subsequent follow-up study showed that there were no significant differences between the results after their ultrasound treatment and the placebo treatment (Rendell et al., 1987). The devices used in this study were identical to the ones in the pilot study. However, sessions were instead twenty minutes long. A large proportion of the participants in the pilot study were also lost to follow up, with only twenty eight of the forty initial participants remaining. Rendell et al. (1987) suggest that a factor that may have contributed to these conflicting results could be associated with the fact that participants were informed of a placebo device. Especially as the participants were often tested in small groups, the participants may have been able to discuss, causing bias and compromising the accuracy of results of the pilot study. Regardless, this suggests that a placebo effect can occur. Lenhardt et al. (2001) also mentions that the ultrasound frequency of 500 kHz emitted by this device exceeds the human detection limit of 100 kHz as determined by Lenhardt et al. (1991). However, the previous study by Carrick et al. (1986) produced results supporting the efficacy of the device in tinnitus therapy despite also using ultrasound at 500 kHz.

Evidently, there is conflicting data regarding the effectiveness of using bone conduction for tinnitus masking, especially when ultrasound masking stimuli are used. Tinnitus masking via bone conduction should also be considered specifically at low frequencies that are more susceptible to eliciting vibrotactile responses due to the potential role of multisensory integration in facilitating tinnitus relief with tactile and auditory stimulation, as will be detailed in the following section.

4. Multisensory Processing

The interaction of pathways within the central nervous system introduces the potential for a multisensory approach to tinnitus management. Multisensory processing occurs throughout the neural networks to allow the interaction and filtering of information from the periphery. Multisensory interactions can occur when a neuron is independently stimulated in response to two or more different senses (Wu et al., 2015). Alternatively, the neuron may only be activated by a single sensory modality but input from another sense causes significant modification of the resulting perception, either by attenuating or enhancing the response. By integrating information collected from other senses, the representation of an individual's environment is able to be interpreted with higher integrity. Perception of the surroundings can hence be finetuned using signals from multiple senses that convey information relaying peripheral characteristics that either agree with or conflict with each other (Wu et al., 2015).

Phenomena such as the McGurk effect is evidence of the complex interactions of sensory systems: while visual input alone may produce ambiguous interpretation, the addition of auditory stimuli allows this to be modulated (McGurk & MacDonald, 1976). Similarly, when visual information was provided in conjunction with auditory signals, participants were able to localise sounds with greater accuracy and speed than when solely auditory localisation cues were provided (King, 2009). This is by multiplexing in the auditory neurons that are associated

with multisensory integration, which allows them to use pertinent sensory cues from different modalities to synthesise a single, complex interpretation (Wu et al., 2015). Evidently, multisensory integration allows processing at higher centres to alter the preliminary input.

Prioritisation of processing at the cortex when multiple sensory cues are received appears to be dependent on the salience of the respective stimuli (Wu et al., 2015). The cortical areas that are responsible for the processing of the sensory modality with most salience preferentially process the signal, while the cortical activity at the regions of the weaker sense is inhibited. Gobbele et al. (2003) found that in response to auditory-tactile stimuli with tactile salience, the magnetoencephalographic (MEG) activity of the somatosensory cortex was elevated while inhibition was noted in the auditory cortex. In this study, simultaneous auditory and tactile presentation was noted to cause suppression of MEG responses. Compared to the sum of the responses to independent unimodal tactile and auditory stimuli, responses were lower when bimodal auditory-tactile presentation occurred. Subjectively, participants reported stronger tactile perceptions. Simultaneous auditory-tactile stimulation also produced MEGs that were more similar to responses to unimodal tactile activity than those generated by auditory activity. Consequently, Gobbele et al. (2003) proposed that this stimulus had dominant somatosensory responses so when bimodal auditory-tactile stimuli were delivered, the activity of the somatosensory cortex was enhanced but the auditory cortex was suppressed. The converse was reported when a stimulus with salient auditory responses was presented (Lutkenhoner et al., 2002). MEG activity in the auditory cortex was elevated but there was suppression of secondary somatosensory cortex activity.

4.1 An overview of the somatosensory system

A brief explanation of the somatosensory system will be provided to understand its interaction with the auditory system. The somatosensory system can be separated into three ascending pathways according to the type of information they convey (ten Donkelaar et al., 2020). Broadly generalised, the anterolateral or spinothalamic system conveys pain, temperature, pressure, crude touch, and reflexive information, the spinocerebellar system is specialised for the coordination of motor control, and the dorsal column or lemniscal pathway is used for information about fine tactile discrimination, kinesthesia, proprioception, and vibrotactile detection. Only the lemniscal system will be further explored, as vibratory perception is pertinent due to both the auditory and somatosensory systems being capable of detecting vibrations at low frequencies (Caetano & Jousmaki, 2006). The other somatic functions are beyond the scope of this investigation.

Vibrations are detected at the periphery by mechanoreceptors, which detect deformations of the skin. Pacinian corpuscles are located in the deep layers of the skin and are sensitive to vibrations at the higher frequencies of the spectrum at which humans can detect vibrations. Typically, they respond to frequencies from approximately 100 to 400 Hz (Cheadle et al., 2018), although some suggest that they may be responsible for the detection of frequencies up to 1000 Hz (Henry & Letowski, 2007). Meissner's corpuscles may also be involved, but to a lesser extent as they are responsible for the detection of frequencies of only around 30 to 50 Hz (Cheadle et al., 2018). The behaviour of Pacinian corpuscles is also noted to be similar to primary auditory afferents in their response to stimuli (Caetano & Jousmaki, 2006). Both operate with a temporally phase-locked Poisson discharge, meaning that when presented with more complex stimuli, single afferents alone cannot accurately encode the information. The highest sensitivity of Pacinian corpuscles is seen at 250 Hz, as the smallest amplitude of deformation can produce an action potential at this frequency (Hayward, 2018). At this point,

action potentials are elicited at the same rate as the stimulation rate; information is directly temporally dependent (Caetano & Jousmaki, 2006). At higher frequencies with smaller amplitudes, responses are restricted by refractory limitations. Electrical responses are no longer synchronous with the stimulation rate but instead, random and concerted firing of a population of afferents will be more effective in representing the information. Responses are able to be generated by minute deformations of the Pacinian corpuscles by responding over multiple cycles (Hayward, 2018).

These mechanoreceptors responsible for detecting vibrations respond rapidly to touch and hence are stimulated by and allow the perception of vibrations through nerve fibres (ten Donkelaar et al., 2020). When the dorsal column lemniscal pathway is recruited, information is propagated into the dorsal root ganglion, and then ascends ipsilaterally. For stimulations of the upper portion of the body that are innervated by nerves at and higher than the sixth thoracic nerve, projections are sent to the nucleus gracilis of the lower medulla via the fasciculus gracilis tract. Similarly, the lower half of the body has afferents that traverse through the fasciculus cuneatus tract and to the nucleus cuneatus. After synapsing at the lower medulla, decussation then occurs to allow ascension through the medial lemniscus and termination at the ventral posterolateral nucleus of the thalamus. Third-order neurons extend from the thalamus to the final structure of the ascending pathway, the somatosensory cortex. Vibratory information related to the face may also recruit a separate system: the trigeminal pathway (van der Cruyssen & Politis, 2017). The trigeminal pathway is used to transmit vibratory information regarding the face and top of the head. Afferents from these regions are transmitted directly to the principal trigeminal nucleus of the mid-pons via the fifth cranial nerve, the trigeminal nerve (Gilman, 2002). From here, information is propagated to the ventral posteromedial nucleus of the thalamus through the trigeminal lemniscus. Thalamocortical projections then allow the

transfer of information to the primary somatosensory cortex – as would also be the path for the dorsal column lemniscal tract.

The somatosensory cortices are present on either side of the central parietal sulcus, the postcentral gyrus (Hayward, 2018). These are composed of two main regions: the primary somatosensory cortex and the secondary somatosensory cortex. While the primary somatosensory cortex appears to be responsible for receiving afferents from the periphery with tactile information, the secondary somatosensory cortex is more involved in the storage and processing of this information. Tonotopicity is preserved throughout the system, including at the ventral posteromedial nucleus and ventral posterolateral nucleus of the thalamus, as well as at the primary somatosensory cortex (Gilman, 2002). At the primary somatosensory cortex, information from cervical axons are represented more medially in the region and sacral axons are located more laterally (ten Donkelaar et al., 2020).

4.2 Interaction of the auditory and somatosensory pathways

Integration of the somatosensory and auditory pathways occurs at multiple levels throughout the pathways. At the level of the brainstem, the auditory and somatosensory systems interact at the first and second order neurons (Jonsson et al., 2016). The cochlear nucleus is the first integration centre for the auditory pathway. The dorsal cochlear nucleus in particular, is heavily involved (Wu et al., 2015). A number of nuclei in this area are implicated in relaying and integrating information between auditory and somatosensory systems. Here, the dorsal cochlear nucleus not only receives innervation from its auditory inputs, but it also receives information from the spinal trigeminal nuclei and somatosensory dorsal column (Young et al., 1995). Its functions include the suppression of sounds that are generated by the somatosensory system of the individual, as well as functions that are more related to the proprioceptive

capabilities of the somatosensory system such as relaying information about the position of the ear relative to the body. Neural components of importance in the dorsal cochlear nucleus include the fusiform cells (Wu et al., 2015). Among their many functions, they can receive excitatory projections regarding non-auditory information from the granule cells of the cochlear nucleus (Golding & Oertel, 1997). This information is propagated through the parallel fibres. By employing the circuit associated with the parallel fibres, adaptive filtering can hence occur at the dorsal cochlear nucleus (Wu et al., 2018). A top-down predictive model is compared with the actual afferent input to determine the differences in the environment in order to apply appropriate corrections. For example, somatosensory input regarding the vocal tract can be altered by the fusiform cells in response to vocalisations produced externally before being relayed to the inferior colliculus. Vocalisations elicit activation of the trigeminal pathway, allowing interactions between these auditory structures and the somatosensory network. Further modifications also occur at the granule cells, as well as subsequent suppression of self-produced vocalisations by the external nuclei of the inferior colliculus (Pieper & Jurgens 2003). This convergence of projections at the lower levels of the auditory hierarchy allows the potential for inter-network information processing and transfer to occur to refine signals that are transmitted to higher centres.

At the inferior colliculus, the external nuclei of the inferior colliculus respond to both somatosensory and visual input. Studies of the external nuclei of cats found that around 55% of the units responded to bimodal auditory-somatic stimulation (Aitkin et al., 1978), while studies using guinea pigs were demonstrated to show bimodal responses in around 65% of the units (Jain & Shore, 2006). The external nuclei are also involved in producing an auditory spatial map with information pertaining to the localisation of sound (Aitkin et al., 1981). Along with this, the nuclei also contain a somatosensory spatial map. This region is hence thought to

be important in the integration of spatial information regarding somatotopic and auditory localisation. The external nuclei of the inferior colliculus also projects this information to the superior colliculus (Thornton & Withington, 1996). Despite being predominantly involved in visual information processing, the superior colliculus also contains auditory and somatosensory spatial maps, which serves as another essential centre for the integration of these two sensory modalities (Middlebrooks & Knudsen, 1984; Meredith & Stein, 1986). Presentation of a sensory cue will cause activation in its respective visual, somatosensory, or auditory receptive field (Lutkenhoner et al., 2002). This will activate neurons at the same location of the spatial map of the superior colliculus to indicate its position, irrespective of the modality that activated it. This integration allows a much more enhanced and refined neural activity. The external nuclei also convey information to the area of the posterior ventral thalamus that is responsible for somatosensory information processing (LeDoux et al., 1987). This suggests that the role of the inferior colliculus in multisensory integration may also be associated with the projection process to ascending somatosensory networks.

Of the regions of the medial geniculate body of the thalamus, the magnocellular region is most heavily involved in multisensory activities (Aitkin, 1973). Serving as a sensory relay station, it receives information from a variety of sources (Wu et al., 2018). Some of these include auditory input from the inferior colliculus' central nuclei and external nuclei (Calford & Aitkin, 1983), direct afferents from the dorsal cochlear nucleus and cochlear nucleus (Schofield et al., 2014), as well as from non-auditory structures including somatosensory projections (from the spinothalamic tract, the dorsal column, and trigeminal paths) and visual information from the superior colliculus (Wu et al., 2018). In response to somatosensory stimulation, intracellular recordings of cells in this area produced inhibitory postsynaptic potentials and also resulted in inhibited responses to auditory click presentations (Khorevin,

1980). It can thus be inferred that the thalamus acts as a key sensory integration structure, both receiving and projecting information from a variety of multimodal sources.

At the cortical level, there are several multisensory connections between lower somatosensory and auditory regions. The primary auditory cortex receives projections from the secondary somatosensory cortex (Cappe & Barone, 2005). Caudomedial and caudolateral auditory belt regions also receive afferents from specific areas of the secondary somatosensory cortex (the retroinsular and granular insula regions) (Hackett et al., 2007). In addition, thalamocortical pathways allow multi-sensory information to be relayed to the auditory cortex from thalamic integration centres, acting as the major contributing afferent source of auditory information to the auditory cortex (Wu et al., 2015). These allow connections between the belt and parabelt areas of the auditory cortex and the medial and dorsal regions of the medial geniculate bodies of the thalamus (de la Mothe et al., 2006).

Audiotactile integration is suspected to occur early on in the processing at the human cortex (Foxe & Schroeder, 2005). Consequently, a feedforward concept has been proposed. In response to auditory and somatosensory stimulation, functional magnetic resonance imaging by Foxe et al. (2002) found the activity to converge at a subregion of the superior temporal gyrus of the auditory cortex. They proposed this region to be the human homologue of the generally well-established integration centre of multisensory input for macaque monkeys, the caudomedial belt area of the auditory association cortices. Summed responses of unimodal somatosensory and auditory stimulation produced significantly different responses (elevated activity) of this region compared to simultaneous audio-tactile activation, highlighting the involvement of this region in the integration of somatosensory and auditory information. Consistent with the macaque model, Foxe & Schroeder (2005) suggest that an early cortical

feedforward mechanism occurs in structures that had formerly been presumed to be unimodal. This concept would allow the individual to detect objects earlier and better sound localise.

4.3 Auditory and tactile integration

Three important concepts govern processing via multimodal integration: the ideas of temporal coincidence, inverse effectiveness, and spatial congruence (Stevenson et al., 2012). The spatial rule states that stimuli that are closer in space will produce stronger multisensory interactions (Murray et al., 2004). These neural interactions may occur when stimuli associated with different sensory modalities activate receptive fields that overlap. It is not necessarily a requirement for the stimuli to interact in space externally, as only the receptive fields of the neurons responding to the stimuli need overlap in their neural representations, such as at the superior colliculus. Without any overlap, multisensory interaction cannot occur and often, inhibition of the responses may result. However, it has been suggested that this spatial rule is not applicable to auditory-tactile interactions (Gillmeister & Eimer, 2007). Gillmeister and Eimer (2007) found that the involvement of spatial proximity between presented auditory and tactile signals had no effect in the resulting facilitation of auditory perception by tactile input. Similarly, electrophysiological results by Murray et al. (2005) state that cortical responses and response speed to the bimodal presentation of auditory and tactile stimuli were independent of their spatial location. It is suggested that the reason why the spatial rule does not apply to auditory-tactile interactions is because the sensory modality of vision is not involved (Gillmeister & Eimer, 2007). Compared to vision, both the senses of sound and touch have poorer spatial resolution. Also, these two senses are more heavily bilaterally represented in their central processing: it is suggested that auditory and somatosensory integration centres receive information pertaining to both contralateral and ipsilateral structures.

According to the temporal rule, interactions of stimuli are dependent on the temporal coincidence with which they are processed (Murray et al., 2004). Stimuli with neural responses that overlap are deemed to interact, while those that are processed outside of this window are processed as being asynchronous. Responses to stimuli of different senses with overlapping peak discharge periods will elicit maximal multisensory interactions (Gillmeister & Eimer, 2007). The rule of inverse effectiveness suggests that when multiple weak sensory stimuli contribute to a response, when presented concurrently, the resulting response to their interaction will be more intense than if stronger individual components were contributing (Murray et al., 2004). Studies by Gillmeister and Eimer (2007) illustrate the rules of temporal coincidence and inverse effectiveness in their ability to alter auditory input via multisensory integration. Temporally, behavioural responses were ameliorated when presented with auditory and tactile stimuli. The presentation of tactile stimuli in synchronicity with auditory stimuli was demonstrated to improve the accuracy with which the participants could detect the auditory stimulus that was presented close to the auditory threshold. Comparatively, asynchronous tactile presentations showed worse performances, suggesting the pertinence of temporal principles and multisensory input in auditory detection. However, these benefits were not as pronounced when the presented auditory stimuli were more intense. Consequently, the principle of inverse effectiveness can be inferred: the impact of multisensory integration seems to be most prominent when the presented stimuli is of a weaker intensity. The principles of inverse effectiveness of weaker stimuli and temporal preference appear to be ubiquitous throughout multisensory integration. It is theorised that with multisensory integration, attentional awareness can be distributed to deduce the likelihood that the multimodal innervations share the same source (Gillmeister & Eimer, 2007).

Interaction and inter-communication of the auditory and somatosensory systems is well-documented in research. The skin parchment illusion experiment illustrates this (Jousmaki & Hari, 1998). Participants in the research group were instructed to rub their hands together. An auditory recording was taken during this experiment and presented to the participants. This playback was either identical to the original recording, or had been modified slightly so that the only high frequencies of 2 kHz and above were increased or decreased in intensity. In response to the original and the recording with the enhanced high frequency components, the participants reported that the skin of their hands felt drier than the playback with dampened high frequencies. This suggests that the auditory information and somatosensory information is interacting to produce an overall illustration of the individual's surroundings. By analysing and integrating information from all of the senses involved in forming the image, the brain aims to explain how these senses contributed their relevant information. In addition, the participants in the research group reported that when the high frequencies were accentuated, the sensation of their hands rubbing together became rougher, similar to parchment. Evidently, auditory signals can be presented to alter the sensations associated with other sensory modalities. Changes in the auditory information's characteristics can influence the resulting perception by integrating with the other senses' input (Wu et al., 2015). Due to the integration and communication of various sensory networks at higher centres in the central pathways, their interaction could alter their perception. Evidence for central modulation due to auditory-tactile input can also be seen in those with musical training. Those with musical training were found to have a more pronounced representation in the neural regions corresponding to this multisensory processing due to the effects of plasticity in response to complex acoustic stimuli (Kuchenbuch et al., 2014).

Vibrotactile stimulation appears to also effectively elicit auditory sensation (Levanen et al., 1998). It should be noted that the temporal patterns of vibrotactile and acoustic stimuli appear to be quite similar, which may have implications in their processing by the two systems (Caetano & Jousmaki, 2006). Vibrotactile stimulation was delivered to the left palm and fingers of a congenitally deaf participant (Levanen et al., 1998). MEG recordings showed that not only was the auditory cortex activated by this haptic stimulus, but significant differences between stimulating at 180 Hz and 250 Hz were noted in the activation of the cortices. Cross-modal plasticity due to auditory deprivation occurs and is especially potent in developing brains (Neville et al., 1983). Cross-modal plasticity can result from the stabilisation of other sensory connections in the absence of a particular sense, leading to reorganisation of the cortex accordingly. This suggests that the auditory cortex can respond to vibrotactile stimulations via multimodal interactions at the cortical level, with the ability to become reorganised to do so in those with auditory deprivation (Levanen et al., 1998). It appears that the auditory cortices of humans have the potential to be activated by and process vibrotactile stimulations, which is dependent on the innervations and stimulations it receives.

Evidence for activation of the auditory cortex activation in response to vibrotactile stimulation is also provided in those with normal hearing in studies by Caetano and Jousmaki (2006). Perceptual and neural evidence of auditory-tactile integration was found. Humans with normal hearing reported that auditory sensations were elicited along with weak vibratory sensations when a 200 Hz vibrotactile stimulus was applied onto the right fingertip. These sensations were then terminated when the stimulus was removed. In addition, MEG responses showed either bilateral or ipsilateral activation of the cortical regions associated with auditory perception for all participants in response to the vibrotactile application. An initial transient activation of the auditory region was elicited in the MEG recording, as well as a more sustained but delayed response in some participants. Concurrent activity in the somatosensory regions

(both primary and secondary cortical areas) were also noted. Caetano and Jousmaki (2006) suggest that this provides evidence for the idea that vibrotactile information is integrated at the auditory cortex.

Devices that allow the sensory substitution of sound for touch for deaf or hearing impaired users show the link between these two modalities. Perrotta et al. (2021) recruited 18 participants with severe or profound hearing loss to use a wristband device that converts sound into vibration. When presented with vibratory patterns only (but not the sound) that had been converted from auditory signals such as dogs barking or a smoke alarm, participants were able to correctly identify the source from a three-alternative forced choice with a rate better than would have occurred with chance, even before any training (62% correct for 11 of the 18 participants). Additionally, participants' performances improved significantly throughout the month in which they underwent training. By the end, some participants were able to almost perfectly identify the sound during this test, with the highest score of 94.6% accuracy and an average of around 70% of the stimuli identified correctly. Moreover, similar results were seen in the pattern discrimination task. Participants were presented with two vibratory patterns, which were words that had been converted into vibrations. Results showed that participants were able to significantly differentiate between patterns corresponding to both sets of words that differed by a single phoneme and also between those differing by more than a single phoneme. These results suggest that auditory input and haptic sensation influence each other, such that participants were able to learn to identify and discriminate sounds using vibrations on their wrist. The authors hypothesise that due to this relation and the involvement of neural plasticity, with continuous exposure to the wristband, the deaf participants' auditory cortices may begin to be activated when vibrations are felt on their wrists. Stimulation may also be seen

in cortical areas that are associated with multisensory integration, such as the caudal auditory belt cortex (Perrotta et al., 2021).

The potential for making use of functional tactile senses for those with auditory deprivation has also been explored further in cochlear implant patients. Electro-haptic devices convert auditory information into electrical activity and then translate it into a vibratory pattern via a specific algorithm and can be used by cochlear implant patients to complement their auditory input (Fletcher, 2020). Participants have shown improvements in their performance in speech-in-noise when using these haptic devices that deliver vibratory signals to the wrist (Fletcher et al., 2019). On average, participants recognised 8% more of the presented words in noise – including some that showed an improvement of up to 20% more words recognised. Improvements were also reported in studies of these devices in spatially separated noise – a particularly practical investigation as a majority of cochlear implant patients receive implants on one ear only (Fletcher, Song, & Perry, 2020). A speech stimulus was presented in front of the participant, with a noise signal on either the implanted or non-implanted side. Regardless of the direction of noise input, the threshold for speech reception was found to improve in participants by around 3 dB when using the haptic devices.

Furthermore, the devices proved to be effective at also improving sound localisation ability (Fletcher & Zgheib, 2020). For unilaterally implanted users before training, the device was found to reduce errors in sound localisation, becoming comparable in performance to bilaterally implanted patients. With a short duration of training (around 15 minutes), their performance improved further such that their results became better than those of bilateral hearing aid wearers.

Additionally, when using these haptic devices, the cochlear implanted participants' pitch perception improved significantly (Fletcher, Thini, & Perry, 2020). Despite the

importance of pitch in speech perception, cochlear implant patients often remain impaired in their pitch perception abilities. A haptic device was used that spanned the forearm and stimulated the area corresponding to a specific pitch of a single octave. By combining this information with the auditory input from the cochlear implant, participants were able to perform similarly to those with normal hearing. On average, participants reached a pitch discrimination threshold of 1.5% for the audio-haptic condition, while those presented only with the auditory information reached a median of 43.4%.

These studies using haptic devices that supplement auditory information by recruiting tactile senses are promising. Evidently, a multitude of auditory perceptions – including speech-in-noise performance, spatial discrimination, and pitch perception – can be improved when tactile stimuli is provided concurrently with the auditory information (Fletcher, 2020).

4.4 Auditory and tactile integration for tinnitus management

The application of auditory and tactile stimulation demonstrates varying success in tinnitus attenuation. Bimodal excitation using auditory tone bursts and electrical stimulation delivered at intervals that induced long-term depression of the dorsal cochlear nucleus's fusiform cells was found to reliably decrease tinnitus in both guinea pigs and humans (Marks et al., 2018). In guinea pigs, tone bursts were applied, followed by an electrical stimulation to the neck. When these were delivered at intervals of 5 ms or 10 ms, it was found that long-term depression occurred in the dorsal cochlear nucleus' fusiform cells. The synchrony of firing and spontaneous activity of the fusiform cells were reduced. Additionally, behavioural manifestations of tinnitus in the guinea pigs were diminished, which is consistent with the idea of tinnitus generation by pathological cell hyperactivity. However, neither physiological nor behavioural indications were present when only unimodal stimulation was provided. Furthermore, for some subjects, unimodal somatosensory delivery appeared to exacerbate the

effects of tinnitus rather than diminishing – hence the unimodal somatosensory trial was not repeated in human participants.

Clinical trials were repeated with twenty human participants (Marks et al., 2018). Participants were randomly allocated to either a sham treatment (auditory stimulation only) or active treatment (tone bursts and electrical stimulation of the neck or cheek) daily for 30 minutes. Participants were involved in either sham or active treatment for four weeks' duration, followed by a four-week washout period before switching to the other treatment and another washout period. Resulting findings were similar to those obtained using guinea pigs: when bimodal stimulation was applied, the perceived obstructiveness of their tinnitus was attenuated, as was the intensity of the participants' tinnitus perception by a mean of 8.0 dB over the active treatment phase. However, in the fourth week of active treatment, the average reduction in tinnitus loudness was 12.2 dB. Notably, despite the cumulative effects of the active treatment in reducing tinnitus loudness throughout the duration of treatment, this decrease in tinnitus loudness was not propagated throughout the washout period. On average, participants scored 7.51 points less in their Tinnitus Functional Index questionnaires after the treatment, in addition to anecdotal reports of a tinnitus that was easier to manage and was less intrusive to daily life, including for those that did not benefit from a complete elimination of their tinnitus. This effect on TFI questionnaire was sustained throughout the washout period.

In comparison, delivering unimodal auditory stimulation alone did not produce the results seen when using bimodal stimulation of auditory and tactile senses, and the participants' tinnitus was not suppressed. Evidently, there does appear to be some higher processing that allows the pathways of somatosensory and auditory information to interact and alter the ultimate sensory perception of the individual that is not present when a unimodal sensation alone is presented. This study not only supports the potential of using auditory-tactile

multimodal stimulation to reduce tinnitus, but also reinforces the neuroplasticity of the brain and the ability to alter tinnitus percepts – therapeutic benefits offered by this treatment were sustained for three weeks post-treatment. However, it should also be noted that vibrotactile stimulation and electrical stimulation may involve different pathways hence this study may not be entirely indicative of the effectiveness of the integration of auditory and vibrotactile signals.

Considering the promising avenue of bimodal stimulation for tinnitus inhibition, the Neosensory Tinnitus Programme developed a wristband device that takes advantage of this (Perrotta & Eagleman, 2021). Clients participated in a daily 10 minute programme which involved the client listening to a series of tones that were synchronised with vibrations from the wristband. Neosensory claimed that this will help clients to differentiate external from internal sounds, eventually allowing them to adapt to their internal tinnitus so that it becomes less disruptive. The associated mobile application also encourages clients to monitor their progress using a Tinnitus Functional Index tracker. Results were seen after two months for most, although others needed it for 3 months or longer. To assess its efficacy, twenty-nine participants were recruited to take part in the programme using the Neosensory Duo wristbands. As measured using the TFI, of the participants tested, 87.5% showed clinically significant improvements in their tinnitus symptoms. Additionally, the average severity of the participants' tinnitus decreased by ⅓ by the end of the programme. However, it may also be prudent to review the efficacy of the device with non-affiliated authors due to the associations that Perrotta and Eagleman have in the development of Neosensory. Furthermore, the long-term effects sustained after using this device for treatment have not been elucidated. No control participants appear to have been recruited and no comparisons were performed to suggest that the addition of the vibrations from the wristband allowed additional tinnitus relief on top of that provided from the auditory signal – reported effects could perhaps be due to the auditory therapy only.

However, not all of the evidence suggests that recruiting tactile senses will assist in tinnitus management. The Reltus ear massager had been designed to make use of the modulating capability associated with multisensory integration via auditory and tactile modalities, and it is marketed as an effective method of providing short-term relief from tinnitus (Jonsson et al., 2016). Tinnitus relief was reported from the broadband noise and tactile stimulation combination from the massager in 83% of the participants. However, presentation of solely a broadband noise (equivalent to the noise elicited by the device) produced residual inhibition in 87% of the participants tested. This suggests that there was no significant advantage in presenting concurrent auditory and tactile stimulation, but rather that it may have been purely relief associated with unimodal presentation. Essentially, it can be inferred that the relief from tinnitus using the Reltus ear massager was due to the resulting auditory artefact without contribution from multimodal stimulation. This brings into question whether the addition of tactile stimulation will be effective in reducing the levels of masking required for tinnitus management.

AIMS OF THE STUDY

The current evidence suggests that bone conduction can be used to deliver tinnitus masker sounds, and that there may be additional multisensory benefits associated with using bone conduction as a transmission medium. Evidence also suggests that the intensity of sounds may be perceived to be relatively higher when delivered with bone conduction than air conduction. Consequently, it is worth investigating whether using bone conduction will reduce the intensity of masking required for tinnitus therapy. The present study aimed to investigate whether using bone conduction was more effective than air conduction in providing tinnitus therapy by using

multimodal stimulation (auditory and tactile stimulation). It was hypothesised that due to the increased sensitivity to vibrotactile stimulation at the lower frequencies, with the involvement of multimodal stimulation, the intensity of tinnitus masking required would be lower at these frequencies. Specifically, this study aimed to investigate the following questions:

- Can bone conduction headphones be used to effectively deliver tinnitus masking?
- Does multimodal stimulation by using bone conduction headphones reduce the intensity of masking required for tinnitus therapy?
- Is there a difference in the masking required at the lower frequencies?

METHOD

This study was approved by the University of Auckland Human Participants Ethics Committee on 24th September 2021 for three years.

Participants

Inclusion and exclusion criteria

Twelve participants were recruited via the University of Auckland Hearing and Tinnitus Clinic. To be included in this study, participants were required to be at least 18 years of age, have hearing loss that is moderately-severe or better, be fluent in English, and have continuous and chronic tinnitus (present for at least 6 months). Exclusion criteria for participants included those with intermittent or fluctuating tinnitus, hearing loss that was worse than 70 dB HL (due to masking loudness comfort), and those that were not able to provide reliable responses regarding their subjective tinnitus perception.

Participant demographics

The mean age of the participants was 55.00 years old (*SD* = 20.95, range 23 – 82). Of the group, six participants were male and six were female. Most participants were right handed, with one left handed participant and one ambidextrous participant.

Devices used

For the air conduction transducer, the headphones that were used were a pair of Sennheiser 150 BT wireless headphones, model SEBT2. These headphones have in-ear earbuds on either side of a connecting wire and neckband cable, and are able to connect to up to two devices using Bluetooth 5.0. A control panel with an omnidirectional microphone and a three-button remote to manage calls and music playback was also present on the right side of the

wire but not used for this study. The headphones are also rechargeable with a 100 mAh lithium battery. The manufacturers also supplied three additional interchangeable pairs of silicone ear buds of different sizes to ensure that the appropriate size could be selected for a good seal to reduce acoustic leakage. The frequency response range of the output of the headphones are 20 Hz to 20,000 Hz. The transducers within the headphones are dynamic drivers, which use electromagnets to move the diaphragm of the speakers. The diameter of the drivers (which dictates the sound quality) are 9.7 mm, which is a very standard diameter of driver that is used for in-ear headphones. The total harmonic distortion level that these headphones may produce at high intensities is < 0.5 % (1 kHz, 100 dB SPL). The maximum sound pressure level that these headphones are able to produce is 107 dB ± 3 dB (1 kHz / 0 dB FS) with an impedance of 28 Ohms (Sennheiser, 2020).

For the bone conduction transducer, the Z8 bone conduction headphones by Shenzhen JEDI Technology Co. Ltd. were used. This is a lightweight device that has a connecting band that spans the back of the user's head. Two arches are located on either side of the band, which are designed to curve around the pinnae for stability when being used. The ends of both of these arches contain bone conduction transducing elements that induce vibration of the skull. According to the manufacturer's guidelines, these transductors should be placed on the cheekbones in front of the ears. A control panel is present on the headphones, including light indicators, a microphone, a multifunction button to manage calls and control media, as well as volume and power buttons. However, these control panel functions will also not be used in this study. The headphones are rechargeable, using a 200 mAh lithium battery, charging in around 2 hours. Once fully charged, they have a stream time of up to 6 hours. These headphones also connect to devices using Bluetooth 5.0 and are able to transmit to devices up to 10 m away. The frequency response range for these headphones is 20 Hz to 20,000 Hz with an impedance

of 8 Ohms (Shenzhen JEDI Technology Co. Ltd., n.d.). Additional relevant specifications for these headphones are not available from the manufacturer.

Assessment

Audiological assessment

Otoscopy was performed on all participants at the beginning of the assessment to ensure that there were no contraindications for inserting earphones into the canals such as otorrhea, and that there were no occlusions that might prevent acoustic stimuli from being transmitted. A pure tone audiogram was obtained for both ears separately using a pair of RadioEar DD450 circumaural headphones in a soundproof booth. Testing was performed using the AVANT Stealth audiometer. Each individual's hearing thresholds were obtained at standard audiometric frequencies and extended high frequencies: testing was conducted at 250 Hz, 500 Hz, 1000 Hz, 2000 Hz, 3000 Hz, 4000 Hz, 6000 Hz, 8000 Hz, 9000 Hz, 10000 Hz, 11200 Hz, 12500 Hz, 14000 Hz, and 16000 via the modified Hughson Westlake procedure. If a hearing loss was diagnosed at 500 Hz, 1000 Hz, 2000 Hz, and/or 4000 Hz, bone conduction thresholds were obtained at that frequency using a RadioEar B71W bone conductor to determine whether a conductive or sensorineural hearing loss was present. To take asymmetrical hearing losses into account, an average of the right and left ear's thresholds was also calculated for interpretation of binaural hearing effects.

Psychoacoustic Tinnitus Measures: Pitch Matching and Loudness Matching

Tinnitus pitch matching and loudness matching data were also obtained using the RadioEar DD450 circumaural headphones. The MedRx Tinnometer was used to obtain psychoacoustic data.

Tinnitus pitch matching was performed for each individual using a two-alternative forced choice paradigm. Participants were presented with two identical narrowband noises at an audible level, with clarification that they were at an intensity that was clearly present but not masking their tinnitus. These two noises only differed in their central frequencies. Participants were instructed to indicate whether the first or second noise that was played was more similar to their perceived tinnitus. This procedure was repeated, gradually narrowing the separation of the frequencies that were investigated until a final tinnitus pitch match was obtained that the participant could agree resembled their tinnitus pitch. Afterwards, a tinnitus loudness match was obtained at this frequency was obtained by gradually increasing the intensity of the noise from a level that was barely audible. This was increased in 1 dB increments until the participant indicated that the noise presented was similar in intensity to their tinnitus. This was then repeated to confirm the results, with an average obtained and used if required.

Psychoacoustic Tinnitus Measures with Bone Conduction and Air Conduction Headphones

Optimal physical fit of the transducers was ensured by liaising with the participant to allow for comfort and minimal acoustic leakage. For the air conduction headphones, each participant was asked about the physical comfort of the headphones. If the ear buds felt too loose in the canal a larger pair of ear adapters were offered to prevent acoustic leakage. However, if the earbuds exerted an immediate and uncomfortable pressure in the ear canals, these were exchanged for a smaller set of earbuds. Optimal physical fit of the bone conduction headphones was assessed by palpating to localise the cheekbones to align the bone transduction element, as per manufacturer guidelines (Shenzhen JEDI Technology Co. Ltd., n.d.). Participants were randomly allocated to starting with either the Z8 bone conduction

headphones or Sennheiser air conduction earphones using a random generator to reduce the possibility of order bias.

To account for the variations in the output across frequencies due to the transducer, psychoacoustic adjustments were performed via electroacoustic calibration of the headphones for all subsequent measurements. This was done by determining and applying the threshold-adjusted noise (TAN) prescription. The TAN calculation and process was determined by Lim (2020). The TAN is defined as the sum of the threshold at a certain frequency and 40% of the minimal masking level (dB SL) (Lim, 2020). For this, the transducers of interest were linked to the software programme Adobe Audition via Bluetooth and auditory stimuli were presented to individuals directly through these. Presented noise had a sample rate of 48 kHz and 24-bit source.

Using Audition, the output channels were isolated so that white noise was presented to the left ear only via a mono channel. The master gain was set at a level at which the participant reported that the presented white noise was at a clearly audible but comfortable intensity. The master gain was then incrementally decreased in steps of 1 dB until the participant indicated that it was no longer audible. This was determined to be the overall threshold for this ear and also served as a general baseline of initial presentation levels for subsequent frequencies tested, while also allowing the participant to become accustomed to the procedure. To determine the MML, the participant was instructed to indicate again when the presented noise just began to interact with or mask the tinnitus that could be heard. The intensity of the white noise was then gradually increased again until the MML was reached.

To separate each frequency's threshold response, all frequency bands except for a single band of interest were then decreased to -64 dB HL to ensure that only the frequency band being tested was audible. The narrowband noise centralised around the frequency of interest was then presented 20 dB above the overall threshold for the ear (to ensure it was initially audible and acclimatise the participants to the novel stimulus), then gradually decreased in intensity until the participant indicated that it was no longer detectable. The threshold was taken as an average of three responses. Frequency bands tested were centralised around 125 Hz, 250 Hz, 500 Hz, 1000 Hz, 1500 Hz, 2000 Hz, 3000 Hz, 4000 Hz, 6000 Hz, and 8000 Hz.

To determine the MML for a particular frequency, the intensity of the frequency of interest was gradually increased by 1 dB from the threshold until the participant responded that the noise presented appeared to mask or interact with their tinnitus. If the level at which the participant's tinnitus was masked was not able to be reached due to loudness discomfort, the participant was re-instructed to instead respond when a comfortable listening level was reached instead. This was done for each of the frequencies above from 125 - 8000 Hz. The perceived intensity of the individual's tinnitus (sensation level) was recorded at each frequency as the difference between their tinnitus MML and their narrowband noise threshold at that particular frequency. Three responses were measured and the average was recorded.

Data for the right ear was obtained by repeating the same procedures. The channel on Adobe Audition was switched to present to the right side only. The overall threshold and MML for this ear were obtained, followed by the thresholds and MMLs specific to each frequency band as detailed above. The threshold-adjusted noise prescriptions were also obtained.

This procedure was then repeated for each participant using the second transducer (either the Z8 bone conduction headphones or Sennheiser air conduction earphones). This included ear-specific data, white noise data for the thresholds and MMLs, and frequency-specific data of thresholds and narrowband noises measured at 125 Hz, 250 Hz, 500 Hz, 1000 Hz, 1500 Hz, 2000 Hz, 3000 Hz, 4000 Hz, 6000 Hz, and 8000 Hz.

Binaural MMLs were also calculated for air conduction and bone conduction by using the average MMLs of the right and left ears. This was to account for those with asymmetrical hearing losses and/or tinnitus that was more severe on one side than the other.

Questionnaires

Participants completed the Tinnitus Functional Index (TFI) and Tinnitus Sample Case History (TSCHQ) questionnaires. These were done to obtain the fundamental information regarding the individual's tinnitus to assess the characteristics and history of each individual's tinnitus. The participant completed these either electronically or on a physical hard copy.

Tinnitus Functional Index

The Tinnitus Functional Index questionnaire consists of 25 questions that prompts the participant to numerically grade how debilitating their tinnitus is perceived to be in several scenarios. These help to evaluate the impact of their tinnitus by considering numerous domains assessing severity. These categories contain a number of questions each on subjects related to the effect that their tinnitus has on daily life according to its intrusiveness, sense of control, cognition, sleep, auditory abilities, relaxation, quality of life, and emotional distress (Meikle et al., 2012). These categories each have their own subscale, but overall, the TFI questionnaire can be summarised using a score that considers each of the participant's answers.

Tinnitus Sample Case History Questionnaire

The TSCHQ is used to obtain a comprehensive summary of the participant's tinnitus history and related health information. The questions include those related to the characteristics of their tinnitus percept, the onset and potential causes, the impact of tinnitus on daily life, and other related medical history that may be pertinent in discussion of hearing and tinnitus.

Data analysis

Statistical data analyses were performed using Prism 9 by Graphpad. The data was normally distributed (as per the Kolmogorov-Smirnov test) so parametric tests were used. A Greenhouse-Geisser correction factor was also applied, so it will be assumed that there is no sphericity. ANOVA tests, Tukey multiple comparisons tests, and correlation analyses were used to investigate the relationship between an individual's MML and other variables (transducer type, threshold of hearing, and side of presentation).

Results

Participant Description

A total of 12 participants were recruited for this study. All participants had non-pulsatile and chronic tinnitus, with the duration of their tinnitus ranging from 1.5 years to around 40 years. The mean number of years with tinnitus for the participants was about 16.71 years (*SD* = 14.57). Most participants reported a gradual onset of tinnitus, however three had an abrupt onset – two were related to exposure to a loud noise and one participant was unsure of the cause. All participants had bilateral tinnitus, although two reported that it was worse on the right side and two reported it was worse on the left side. Descriptions of their tinnitus varied but most perceived and categorised it to be of a high frequency. Four described a ringing percept, two a whistling, and another two reported a buzzing. The rest of the participants' descriptions included tinkling noises, tonal sounds, cicada-like noises, and noises "similar to a frying egg". Tinnitus pitch matching showed participants had an average tinnitus pitch of 6.08 kHz, suggesting that the majority of participants had a higher pitched tinnitus. However, there was some range in the tinnitus pitch perceived, from 1.5 kHz to 10 kHz. Participants also reported various amounts of distress resulting from their tinnitus. Tinnitus Functional Index scores ranged from 9.2 to 52.7, with participants scoring 29.73 on average. This is categorised as being a small problem (Peter et al., 2017). The distribution of participants categorised according to their TFI severity can be seen in **Figure 2**.

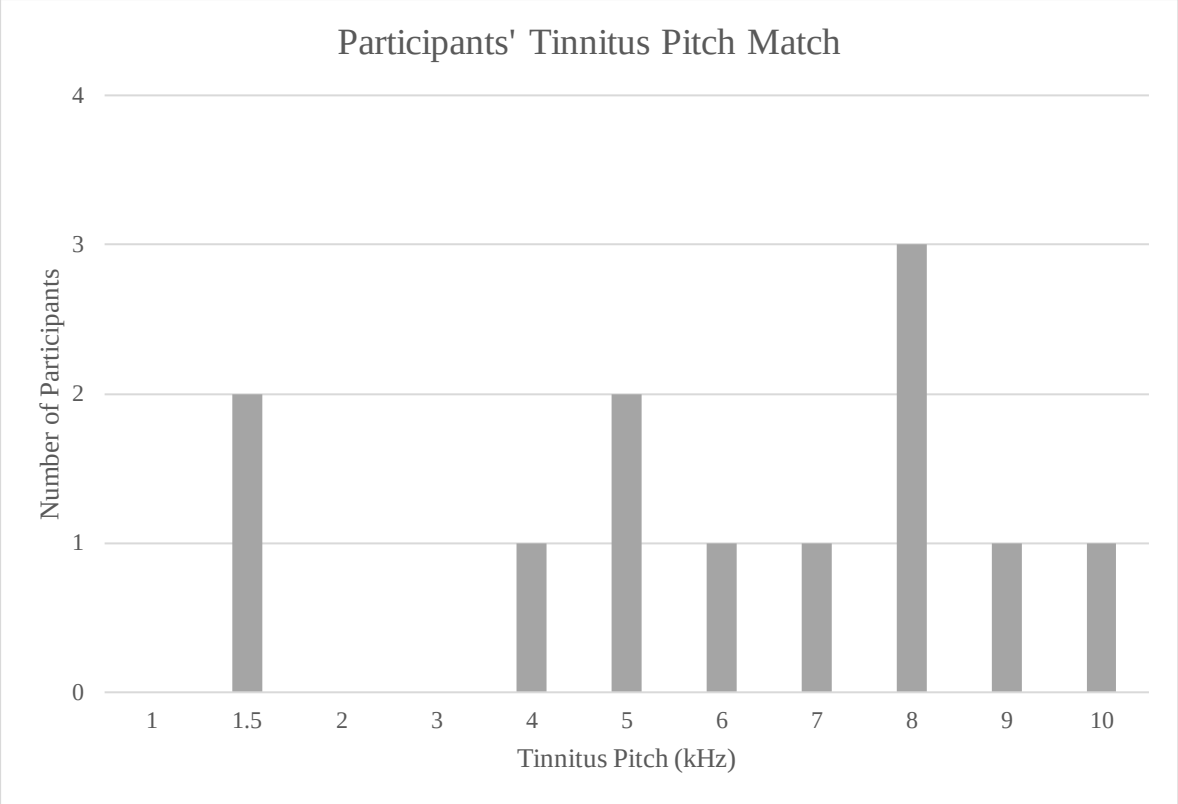

Figure 1 Number of participants according to pitch matching (kHz).

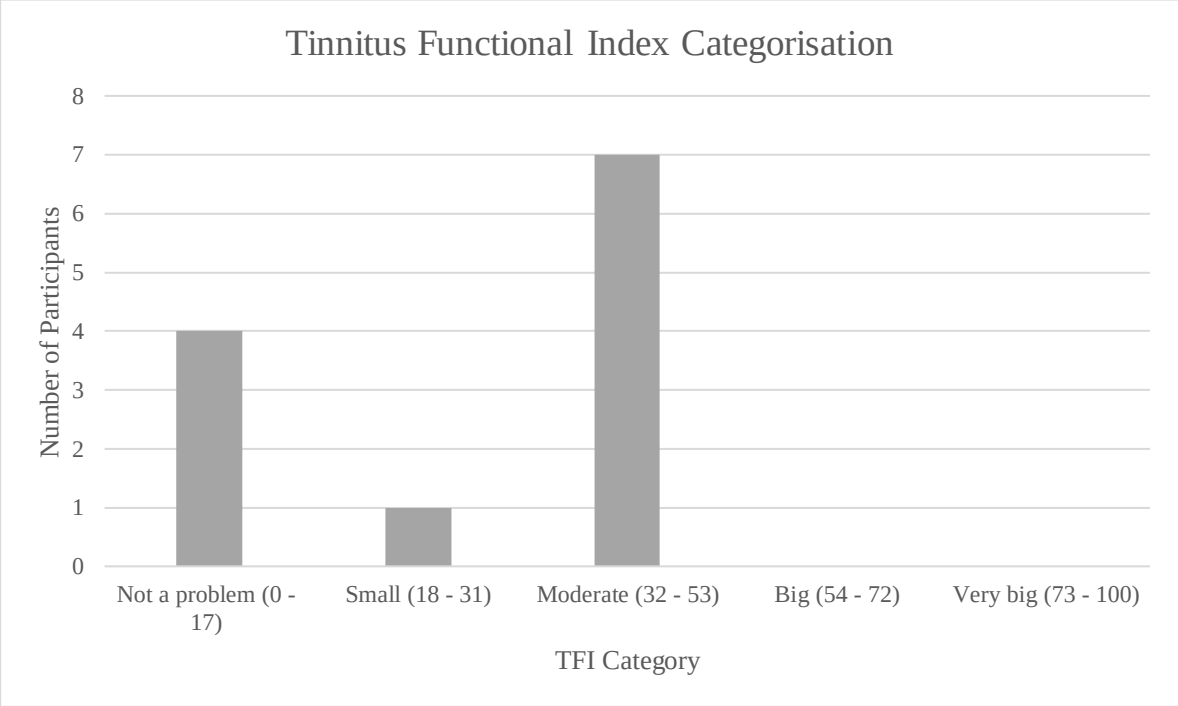

Figure 2 Scores of participants' Tinnitus Functional Index questionnaires calculated and categorised according to severity of the impact that their tinnitus has. Graph indicates the number of participants in this present study that were in each category.

Pure tone audiometry results showed a general trend where on average, the severity of the hearing loss tended to become worse towards the high frequencies, as seen in **Figure 3**. The configuration of the average loss supports effective tinnitus masking, as one of the recommended criteria for successful masking includes good hearing thresholds at the low frequencies (McNeill et al., 2012). Most of the participants had a hearing loss within the standard audiological testing frequencies (0.25 - 8 kHz). However, all participants – including those with normal hearing from 0.25 to 8 kHz – had some hearing loss present at the extended high frequency range (8 - 16 kHz). No response could be recorded at the limits of the audiological equipment for two ears at 12.5 kHz, eight ears at 14 kHz, and four ears at 16 kHz.

Some participants did have slight asymmetries between their two ears. Three participants had clinically significant asymmetries (defined as either ≥ 20 dB HL difference at two adjacent frequencies or ≥ 15 dB HL interaural difference at any two frequencies from 2000 Hz to 8000 Hz (Durakovic et al., 2018)). Two participants had worse hearing in their right ear, and one had worse hearing in the left ear. For two of these patients, this corresponds with the ear in which their tinnitus was perceived to be louder. Some interaural differences can also be seen between the average hearing thresholds of the left and right ears. However, these differences are no larger than 10 dB HL at any frequency. All participants presented with a sensorineural hearing loss.

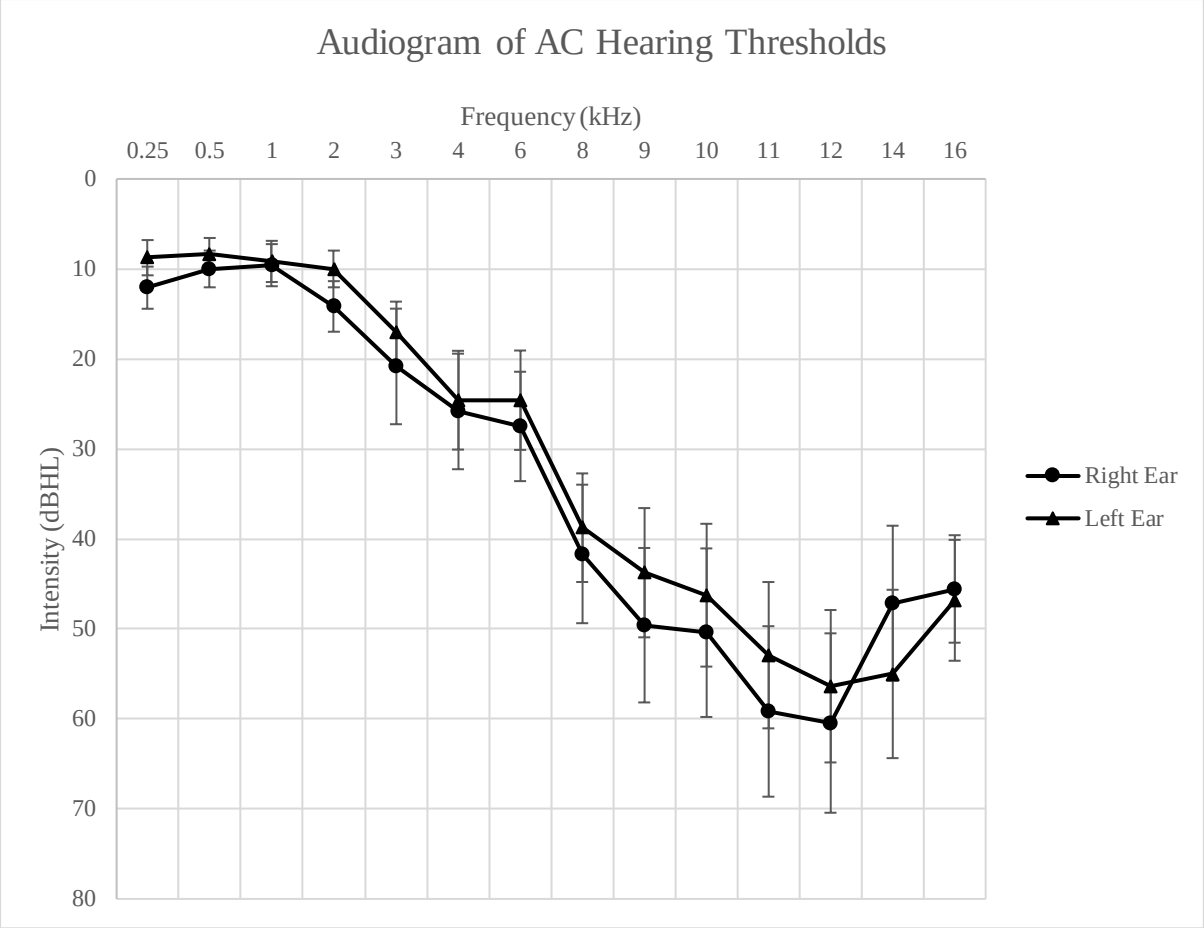

Figure 3 Audiogram of the mean hearing thresholds for all 24 ears of the participants. Results for the right ear are depicted by circles, and for the left ear by triangles. Error bars show ± 1 standard error of the mean.

Differences between Frequencies and Transducer Type

Potential differences in MML (dB SL) were explored by separately considering the results when masking was presented solely to a participant's right ear or to their left ear, and also the average MML of both of their ears to account for inter-aural hearing threshold and tinnitus loudness differences. When considering all MMLs binaurally – whether transmitted using bone or air – there was a significant difference between the frequencies, as determined using a two-way repeated measures ANOVA test ($F(2.26, 24.93) = 3.52$, $p = 0.04$). Comparison of the frequencies just when the masking was delivered with air conduction revealed that a significant difference was still present ($F(2.71, 29.82) = 4.07$, $p = 0.02$). Multiple comparisons

with a Tukey's test demonstrated that this significance was specifically between 0.25 kHz and 1 kHz ($p = 0.02$) and between 0.5 kHz and 1 kHz ($p = 0.03$). However, this was limited to the AC results. Analysis of the binaural MMLs required when using the bone conduction headphones revealed that there were no significant differences between the frequencies ($F(2.58, 28.36) = 2.60$, $p = 0.08$). No significance could also be found when comparing the MML required for a specific frequency when masking was delivered by either air or bone conduction ($F(1.00, 11.00) = 0.41$, $p = 0.54$). This suggests that using the bone conductor headphones was equally effective in masking tinnitus as the air conduction headphones. Furthermore, no significance was found when comparing the MML required between frequencies depending on whether air conduction or bone conduction was used ($F(3.87, 42.58) = 2.13$, $p = 0.10$).

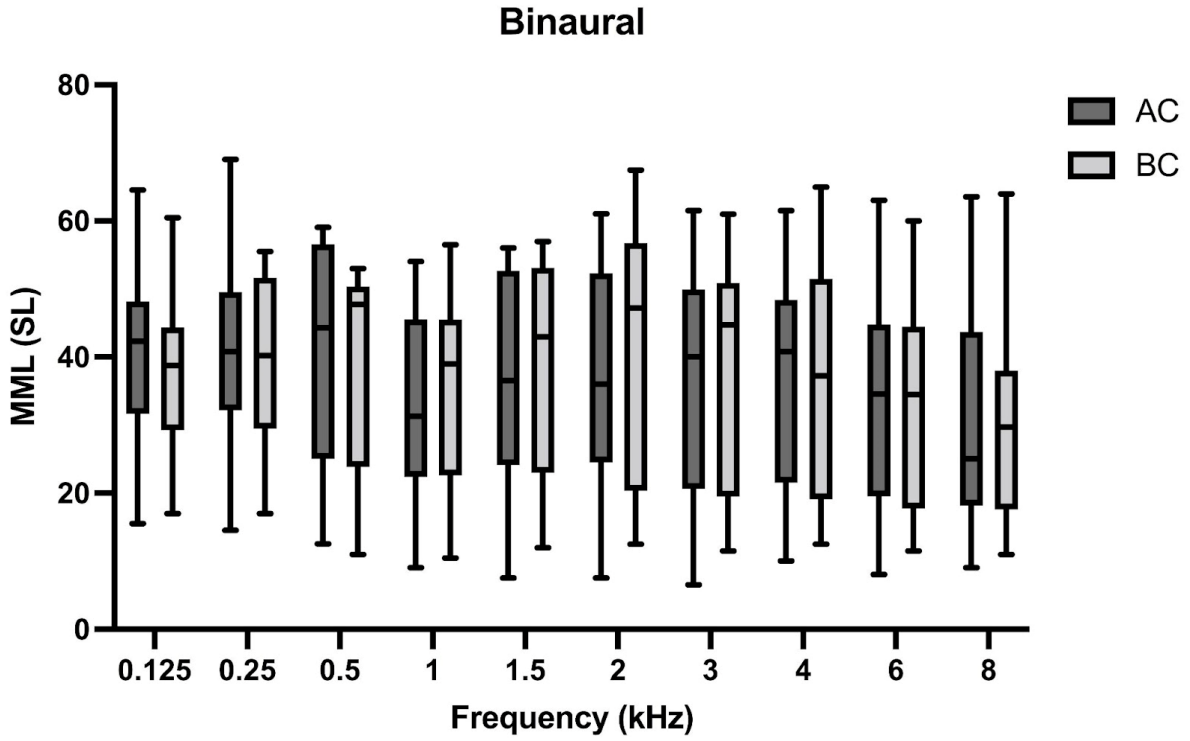

Figure 4 MMLs for binaural data across tested frequencies for both AC and BC.

Similar analyses were performed separately for the left ear and the right ear. For the left ear (LE), no significant differences were found between the MMLs for different frequencies ($F(2.08, 22.84) = 2.25$, $p = 0.13$), when comparing the MMLs when the masking was through

AC or BC (F(1.00, 11.00) = 1.48, p = 0.25), or for frequencies depending on whether the stimulus was delivered by AC or BC (F(4.02, 44.20) = 1.09, p = 0.38).

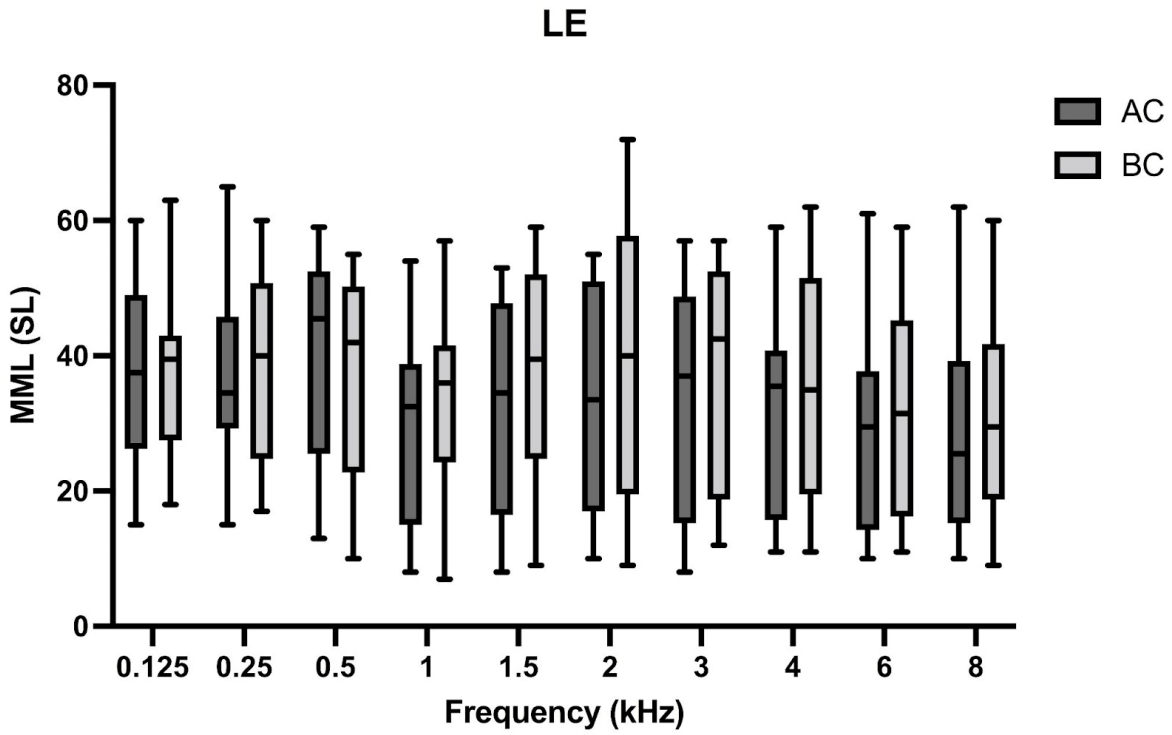

Figure 5 MML for the left ear across tested frequencies for AC and BC.

Another ANOVA for just the right ear (RE) showed that there were significant differences present between the MMLs of different frequencies when AC and BC were considered together (F(2.71, 30.75) = 3.74, p = 0.02). Significant differences in frequencies when using the AC transducer only were determined to be present (F(2.96, 32.56) = 3.55, p = 0.03). Specifically, this was established to be between 0.5 kHz and 1 kHz (p = 0.04). There was also significance in the comparison between frequencies using bone conduction (F(3.38, 37.16) = 3.00, p = 0.04). However, when individual frequencies were compared using a multiple comparisons test, no pairings had significance. Again, results failed to indicate a significant difference between AC and BC MMLs (F(1.00, 11.00) = 0.10, p = 0.75). There was also no significant difference between the frequencies depending on whether AC or BC was used (F(4.39, 48.28) = 1.89, p = 0.12).

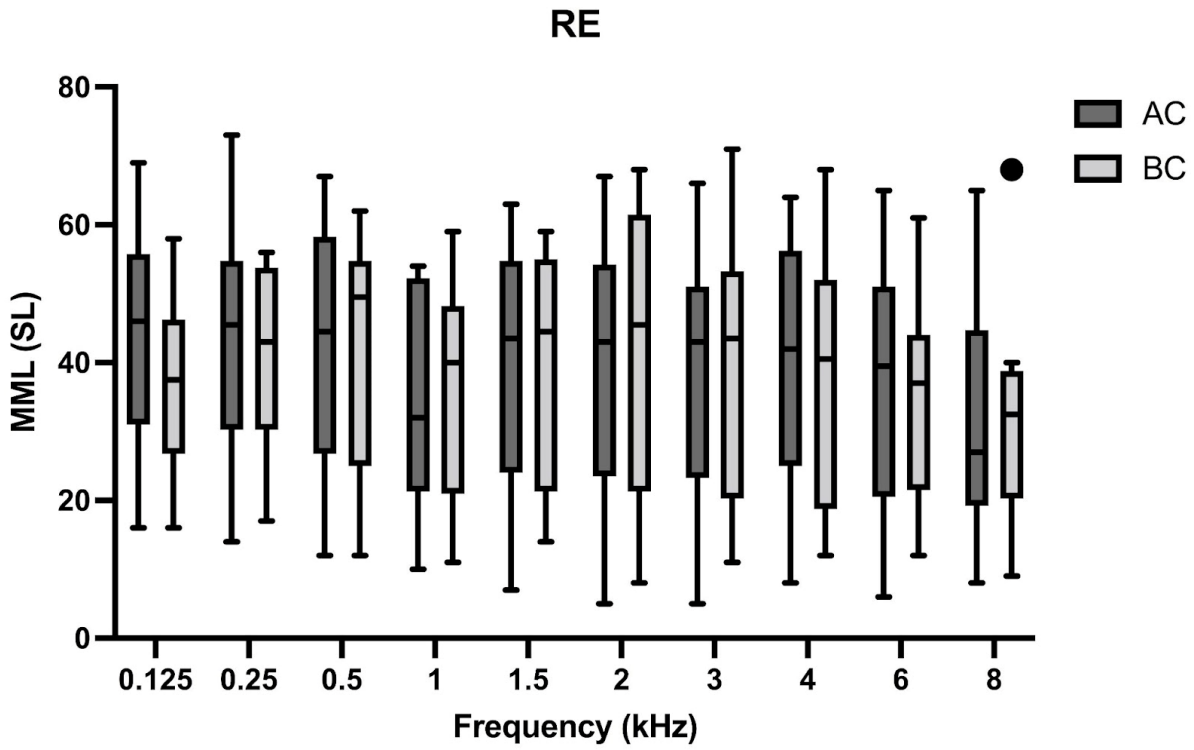

Figure 6 MML for the right ear across the tested frequencies for AC and BC.

Correlations between hearing threshold and MML

Significant correlations were also found between a participant's hearing threshold and the MML required (dB SL) for a particular frequency and when using a specific transducer. These were present for binaural, right ear, and left ear thresholds. All significant relationships can be found in **Appendix A**.

Most frequently, binaural data showed significant relationships between thresholds at high frequencies and MML using BC at high frequencies. There were statistically significant negative correlations between the threshold and MML, where as the severity of hearing loss at a specific frequency increased, the MML required to mask it using a particular frequency decreased. Correlations were found at hearing thresholds of 500 Hz and 4 Hz for the left ear; and 1 Hz and between 8 - 16 kHz binaurally. Generally, significant correlations could be found

between low frequency thresholds and low frequency MMLs, and between high frequency thresholds and high frequency MMLs. The strength of the correlations ranged from weak (r = -0.31) to very strong (r = -0.80). Notably, the strengths of the correlations tended to increase with the frequency of the masking presented. Significant correlations were also present more often when masking was presented using BC.

Similarly, when considering the left ear's thresholds, significant correlations between the hearing threshold and MML at a particular frequency were also present. These trends were typically similar to when binaural data was considered, including in the strengths of correlations with threshold frequency and transducer type. The nature of the correlations were also the same: as the degree of hearing loss at a particular frequency became more severe, the amount of masking required decreased. These relationships were present with the thresholds at 500 Hz, 1000 Hz, 4000 Hz, and between 8000 Hz to 16000 Hz. Strengths of correlation ranged from moderate (r = -0.58) to strong (r = -0.75). Again, results yielded significant correlations when masking was presented with AC or BC, although significant correlations were found at more masking frequencies when masking was delivered with BC.

Comparatively, there were fewer significant correlations for the right ear's hearing thresholds at low and mid frequencies. The only significant relationship was a moderate correlation between the threshold at 1 kHz and the MML using BC at 8 kHz (r = -0.59). There were more significant correlations between higher frequency thresholds (at 8 - 16 kHz) and the MML. The nature of the relationship was also similar to those for the left ear and binaural data: as the hearing loss at a particular frequency worsened, the MML required tended to decrease for all significant relationships. The strengths of correlation ranged from a moderate (r = -0.58)

to a very strong (r = -0.81) relationship. Again, there were also more significant correlations with the hearing thresholds when BC was used for masking compared to AC.

Laterality of Masking Presentation

Comparisons were explored between the MMLs required for a specific frequency when stimuli were presented to either the right ear, the left ear, or the average of both ears. Analysis of MMLs when masking was through AC revealed significant differences between frequencies ($F(2.71, 29.82) = 4.07$, $p = 0.01$). Significant differences were also found between MMLs when presenting with AC masking binaurally, to the left ear, and to the right ear ($F(1.00, 11.00) = 7.93$, $p = 0.02$). Specifically, a multiple comparisons analysis found significant differences at 4 kHz between the MMLs of presentations to the right ear and the left ear, the left ear and binaurally, and the right ear and binaurally ($p = 0.02$). Significance was also found at 6 kHz ($p = 0.02$).

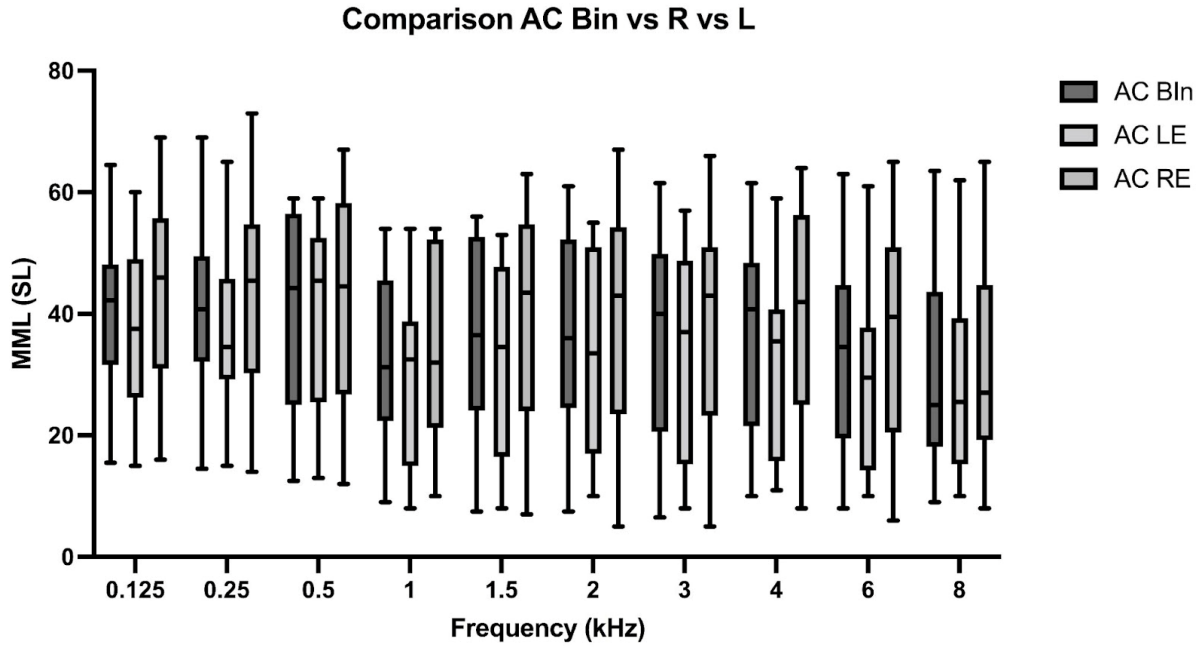

Figure 7 . MML across tested frequencies when masking was presented using AC to the right ear, the left ear, and the binaural average.

However, when using a BC transducer, no significant differences were found in the MML between either frequencies ($p = 0.08$) or when comparing stimuli presented binaurally, to the left ear, or to the right ear ($p = 0.16$).

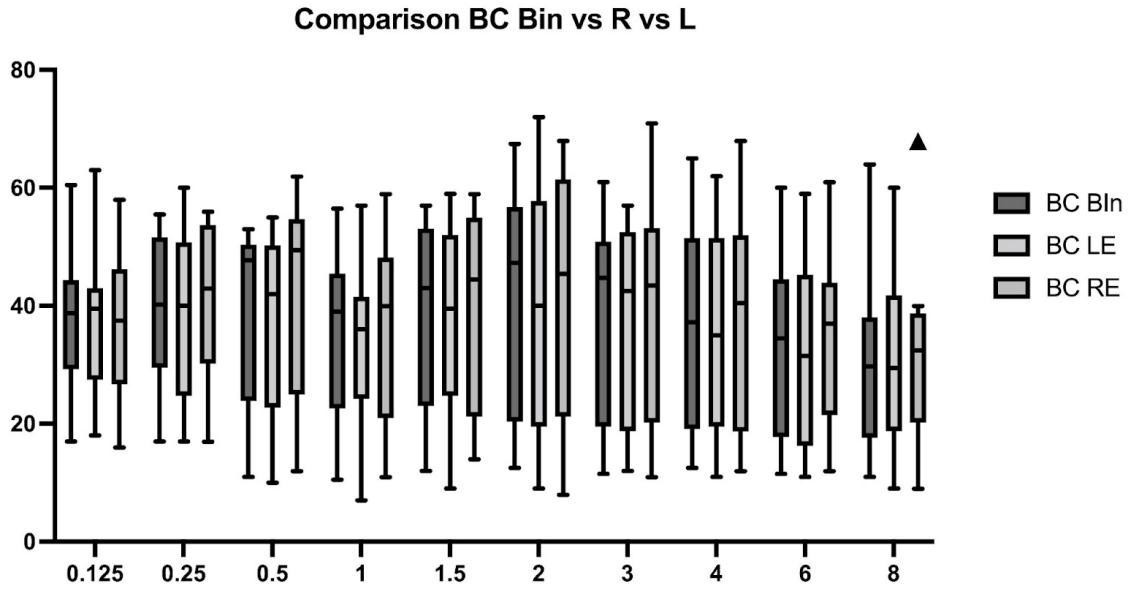

Figure 8 MML across tested frequencies when masking was presented using BC to the right ear, left ear, and the binaural average.

Demographics correlations

Demographic data of the participants was analysed for correlations with the MML required. See **Appendix B** for all significant correlations with demographics. There were no significant correlations found between the pitch perceived by the participant (as determined with pitch matching) and the MML. This was for all frequencies at which masking was delivered and for both air conduction and bone conduction transducers. No significant correlations were found also between MML and the participants' Tinnitus Functional Index scores.

Significance was found with the number of years with which an individual has had tinnitus for only one masking condition. When an 8 kHz masking stimulus was delivered to the left ear using bone conduction, there was a strong negative correlation with the MML required ($p = -0.04$, $r = -0.61$). Participants who had tinnitus for longer appear to require a lower MML.

There were several significant correlations between the age of the participant and their MML. When using AC to mask the tinnitus, significant negative correlations were found with masking noise presented at 8 kHz. There was a strong negative correlation between the MML and participant's age when the masking was introduced to both the right ear ($p = 0.02$, $r = -0.65$) and the left ear ($p = 0.02$, $r = -0.65$). This suggests that for older participants, the amount of masking at 8 kHz using air conduction required at either ear decreases.

There were also several significant correlations between the participant's age and their MML when using BC. For masking delivered to the right ear, there were strong negative correlations with age at 3 kHz ($p = 0.01$, $r = -0.70$) and 4 kHz ($p = 0.02$, $r = -0.67$). Significance was also found with age for masking at 6 kHz. Strong negative correlations were found when masking was delivered to the right ear ($p = 0.03$, $r = -0.62$), and also for the left ear ($p = 0.03$, $r = -0.61$). Similarly, for masking at 8 kHz using BC, significant correlations with age were also present for both ears: a strong negative relationship was demonstrated when masking was input to the right ear ($p = 0.01$, $r = -0.73$), and a strong negative correlation for the left ear also ($p = 0.01$, $r = -0.69$). As for the significant correlations found when using air conduction, these findings support the idea that as the age of the participant increases, the amount of masking required at mid to high frequencies (3 - 8 kHz) when using BC to deliver masking decreases.

DISCUSSION

The present study aimed to investigate whether the use of bone conduction for tinnitus masking would reduce the MML required by recruiting multimodal integration. The MML was not affected by the medium of delivery, whether through air conduction or bone conduction. This was demonstrated when comparing MMLs using the respective transducers with masking output isolated to the right ear, the left ear, and also the binaural average. These results support a study by Jeong and Jin (2020) that also compared tinnitus sound therapy using bone conduction and air conduction headphones. The longitudinal study demonstrated that over three months, despite improvements being noted in participants' subjective relief via questionnaires, no significant differences in improvements were established between the use of the two different transducers (Jeong & Jin, 2020). The present study expanded on this by evaluating behavioural data across frequencies to elicit vibrotactile stimulation at low frequencies to activate multimodal integration, obtaining results that are consistent with the findings by Jeong and Jin.

Other studies report that the intensity of sounds were perceived to be elevated when delivered using bone conduction (Stenfelt & Goode, 2005b; Manning et al., 2016; Henry & Letowski, 2007), which would suggest that less tinnitus masking would be required. This conflict could be attributed to the postulated location of the origin for tinnitus. The two routes of auditory input via air conduction and bone conduction converge at the cochlea with coinciding pathways and mechanisms of stimulation post-cochlea (Bear et al., 2016). However, it is likely that tinnitus originates more centrally, and auditory-tactile integration centres also are located at higher structures of the pathways. Perhaps both sources of auditory information are introduced to the central structures of the pathway identically at lower structures of the auditory hierarchy, such that their ascending course is also processed together and interacts

with tinnitus concomitantly. No differences in their pathways exist after information is collated at the lower structures of the auditory system. Consequently, once MMLs were corrected according to individual hearing thresholds, despite different origins, no significant differences may have been found between the MMLs required.

There are several implications associated with the finding that no significant differences were present between bone and air conducted masking. It suggests that even when facilitated by multimodal stimulation by introducing an additional sensory modality of vibrotactile stimulation, it does not appear to assist in significantly reducing tinnitus MMLs. At the lower frequencies, vibrotactile sensations were likely felt by all participants due to the intensity at which masking was delivered at and the vibrotactile thresholds at these frequencies. These lower frequencies typically required comparatively higher intensities to mask the participants' tinnitus. Several participants also verbally reported that a tactile sensation was felt at the stimulation site. In retrospect, a more objective measurement of participants' vibrotactile perception may have been prudent. Regardless, no significant differences were revealed between the frequencies when masking was delivered through the bone conduction headphones to either the left ear or for the binaural average. It should be noted that significant differences were found between frequencies when using bone conduction to mask the right ear, but no specific frequency pairings had significance. A similar study involving the Reltus ear massager device which emitted vibratory and acoustic signals through the massager also produced no additional benefit from the vibratory element (Jonsson et al., 2016). Tinnitus relief was only provided by the unimodal noise that was produced as an auditory artefact of the massager.

A potential consideration for the presence of no significant differences even when vibrotactile stimulations were felt at the low frequencies could be due to the transducers that were used. The two devices used differ in their interaction with the auditory ear canal. While

the air conduction transducer that was used was an in-ear pair of headphones and blocked the ear canal, the bone conduction headphones left the canal open. The occlusion effect may hence be involved: low frequencies could potentially have been differentially artificially amplified when the air conduction headphones were used, but not for bone conduction. As a result, any possible additional reduction in MML provided by multimodal stimulation at the low frequencies using bone conduction may have been negated. However, as the threshold-adjusted noise prescription was applied, appropriate modifications according to each participant's perceived output intensity would be considered for and hence significant contribution by the occlusion effect seems unlikely.

However, the bone conduction headphones did produce some tinnitus relief, even if it were as a result of unimodal auditory masking relief. The finding that no significant differences exist between MMLs delivered with bone conduction or air conduction does suggest that tinnitus masking can be introduced using either air conduction headphones or bone conduction headphones with equal effectiveness. It can thus be inferred that bone conduction can be used to provide masking for tinnitus patients using MMLs similar to those which would be required if using air conduction. This enables the clinician to liaise with the client to choose a manner of sound delivery that best suits the needs and preferences of the client. Personal music players are equally effective in providing tinnitus relief as conventional tinnitus control instruments and hearing aid sound generators (Fukuda et al., 2011). This makes tinnitus masking very accessible with the widespread use of personal devices, air conduction earphones, and conventional bone conduction headphones, broadening the possible avenues with which one can pursue tinnitus masking. Bone conduction headphones may be preferable due to an un-occluded ear canal and access to environmental sounds. This may prove especially beneficial for hazardous scenarios such as near traffic where remaining vigilant of surroundings is vital,

while also providing relief from tinnitus. As it bypasses the outer and middle ear components of the auditory pathway, it also remains a suitable option for those with conductive hearing losses. In addition to no differences in MML when using bone conduction and air conduction, there were also no significant differences between the laterality of the stimulation site for bone conduction. This allows the possibility of applying bone conduction to either side for those of whom bilateral masking is inaccessible or may find benefit in a single site of stimulation.

Generally, the MML required for older participants tended to become increasingly reduced for higher frequencies compared to the low frequencies, especially when using bone conduction. This may be considered in conjunction with the finding that for frequencies that required lower levels of MML, there also tended to be more hearing loss. The nature of these two sets of correlations were consistent for all significant relationships found between the two variables, regardless of the frequency, side of presentation, and transmission pathway used. It should also be noted that these correlations were present despite the MML being adjusted for according to the severity of each participant's hearing loss using the threshold-adjust noise prescription. Consequently, it can be inferred that older participants may require lower intensities of masking sound to be delivered due to the severity of their hearing loss, especially for higher frequencies. This can be explained when considering abnormal loudness functions and loudness recruitment. Loudness recruitment refers to the idea that if a sound is presented at a level above the absolute threshold for the individual, the rate of growth of the loudness will increase at a higher rate than would be normal compared to the elevation in intensity (Moore & Glasberg, 1996). The extent to which loudness growth and loudness summation impacts each individual differs, which likely is as a result of differences in the physiology underlying the pathology. Due to aberrant cochlear function, the absolute threshold of hearing will increase as a result of pathology of either solely, or due to contributions by, damage to the outer hair

cells and/or inner hair cells. Frequency selectivity may be compromised causing broader auditory filters, which is often largely attributed to damage to the outer hair cells (Moore & Glasberg, 2004). Additionally, pathology of the outer hair cells can also compromise the compressive non-linear characteristics of the basilar membrane (Moore & Glasberg, 1996). Inner hair cell and neuronal function may also be damaged or completely lost, causing dead regions that cannot induce responses. Ears with cochlear damage solely due to pathology localised at the inner hair cells only is unlikely, hence outer hair cell damage will likely be at least partially involved in loudness recruitment (Moore & Glasberg, 1996). At frequencies with hearing losses greater than 50 dB HL, it is suggested that aberrant inner hair cell activity at the cochlea is implicated in addition to outer hair cells (Shekhawat et al., 2013). In response, more abnormal loudness functions may result. Consequently, in the present study, at the higher frequencies of older participants where the absolute threshold tended to be greater, relatively smaller increases in the intensity of masking will produce much larger increases in loudness perception compared to those with normal hearing or milder hearing loss at these frequencies.

The average hearing and tinnitus profiles also support the occurrence of loudness recruitment. Overall demographics of the participants appeared to resemble the 'convergence' masking configuration, as determined using Feldmann's masking types (1971), and generally, the pattern of presentation for individual participants also appeared to be consistent with this. The average pure tone audiogram showed a definite slope towards the higher frequencies, including normal to near-normal frequencies towards the lower pitches. The average tinnitus pitch of the participants was also consistent with a 'convergence' type of masking with an average pitch match of 6.08 kHz. According to this, tinnitus masking at the low frequencies will require relatively large amounts of the masking stimulus to be delivered, while the high frequencies only require small amounts. Again, this is consistent with the idea that loudness recruitment is occurring: because there is more hearing loss present at the high frequencies, the

rate of loudness growth is greater here and thus relatively small amounts of masking presentation will prove to be sufficient in tinnitus masking.

This has implications with respect to the fitting of hearing aids when being used to treat tinnitus more effectively. Previous studies demonstrate the effectiveness of using hearing aids for tinnitus therapy, including that prescribing the required amplification at high frequencies could facilitate the management of high pitched tinnitus (McNeill et al., 2012). For both air and bone conduction transducers, for those with sloped hearing loss configurations, only a relatively small amount of masking needs to be applied at the high frequencies when considering loudness recruitment. In contrast, larger amounts of gain may be required to effectively mask if a low frequency narrow-band noise has been selected. Thus results from this study suggest that those with more hearing loss at a particular frequency will likely require less gain correspondingly. Comparatively, even distributions of gain regardless of hearing configuration will likely have poorer results for the patient's therapy. Presenting a wideband noise that is evenly distributed across the frequencies will not be as effective and may even be dangerous – sufficient intensities may not be delivered at the low frequencies, while the high frequencies may be uncomfortably loud for the user due to the effects of loudness recruitment. Consistent with their abnormal loudness functions, tinnitus sufferers are also often reported to have reduced toleration to loud sounds with low uncomfortable loudness levels (Tyler & Conrad-Armes, 1983). This reinforces the importance of ensuring that appropriate sound therapy is provided. Applying a uniform amount of gain across all frequencies could cause uncomfortably loud sounds to be delivered to the high frequencies, as the present study demonstrates that comparatively low MMLs were required, especially for older participants.

Further investigations could be conducted. Vibrotactile responses (at ethical intensities) from sound presentation are only present at low frequencies but lower masking levels were

required at high frequencies due to loudness recruitment. Some evidence also suggests that bone conduction at very high frequencies may provide tinnitus relief (Goldstein et al., 2005; Carrick et al., 1986). Hence it may be interesting to present high frequencies of masking in conjunction with a separate device that deliberately evokes vibratory conduction. If multimodal stimulation is elicited, this would ensure that the somatosensory system is activated and therefore potentially reduce the masking level required. A similar study by Perrotta and Eagleman (2021) delivered vibrations and auditory tones, but comparisons of tinnitus relief with unimodal stimulations were not provided, hence multimodal benefit cannot be substantiated.

Significant differences were also found at frequencies between the right ear and left ear when using air conduction, but not bone conduction. This could be attributed to the transcranial attenuation associated with bone conduction. As detailed in section **1.2.1: Bone conduction properties**, stimulation at one site would allow the transfer of vibrations through the cranial bones to stimulate the contralateral cochlea with minimal attenuation. In contrast, the interaural attenuation when using air conduction is more significant. The air conduction route relies on transmission including through the outer and middle ear components and hence would traditionally not involve energy transfer through bone to activate the contralateral cochlea, as bone conduction does (Purves et al., 2008). When using circumaural headphones, it is assumed in clinic that a minimum interaural attenuation of 40 dB is present, compared to an interaural attenuation of 0 dB using bone conduction (Snyder, 1973; Smith & Markides, 1981; Yacullo, 2015; Martin & Blosser, 1970). Hence significant differences would be found between stimulations of the two ears when using air conduction only.

Limitations associated with this study should be considered. The group size consisted of 12 participants. Ideally, a larger number of participants would be recruited. Especially for investigations such as when comparing for differences between frequencies of binaural MMLs dependent on which transducer was used. This was approaching significance; a significant relationship may have been able to be found if a larger group had been recruited. This was limited due to the impact of COVID-19 interference and regulations.

While not significant ($p = 0.10$), this may be due to the underpowered study. The study size was limited due to interference with recruitment by COVID-19 lockdowns. It is possible that increasing the group size would reduce variation in the sample, moving the statistic towards significance if the difference in means was maintained.

CONCLUSION

The present study proved that the use of multimodal integration for tinnitus therapy by vibrotactile and auditory responses with bone conduction did not significantly reduce the level required for masking when compared to air conduction. Findings suggest that using bone conduction headphones can be equally effective in providing tinnitus relief. Implications regarding the importance of appropriate amplification of high frequencies can also be inferred, especially for older patients due to the nature and common configuration of age-related hearing loss.

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APPENDICES

Appendix A.

Significant Correlations with Hearing Thresholds and MMLs

This appendix contains all significant correlations that were found between participants' hearing thresholds and their MML. P-values and Pearson r correlation coefficients for all significant relationships are included. Significant correlations were present between participants binaural hearing thresholds and MML. These occurred both when masking was delivered to the right ear, and also for the left ear. Correlations were also present when masking was through air conduction (AC), and across a range of frequencies.

Hearing threshold at 500 Hz

Masking to the left ear:

- AC 0.125 kHz ($p = 0.01$, $r = -0.70$).
- AC 0.25 kHz ($p = 0.02$, $r = -0.68$).
- AC 0.5 kHz ($p = 0.02$, $r = -0.68$).

Hearing threshold at 1000 Hz

Masking to the right ear:

- BC 8 kHz ($p = 0.03$, $r = -0.62$).

Masking to the left ear:

- BC 1.5 kHz ($p = 0.04$, $r = -0.61$).
- BC 3 kHz ($p = 0.04$, $r = -0.59$).

Hearing threshold at 4000 Hz

Masking to the left ear:

- BC 4 kHz (p = 0.04, r = -0.52).

Hearing threshold at 8000 Hz

Masking to the right ear:

- AC 8 kHz (p = 0.03, r = -0.64).
- BC 4 kHz (p = 0.03, r = -0.63).
- BC 6 kHz (p = 0.03, r = -0.63).
- BC 8 kHz (p = 0.004, r = -0.77).

Masking to the left ear:

- BC 8 kHz (p = 0.01, r = -0.70).

Hearing threshold at 9000 Hz

Masking to the right ear:

- BC 4 kHz (p = 0.02, r = -0.67).
- BC 6 kHz (p = 0.03, r = -0.63).
- BC 8 kHz (p = 0.01, r = -0.69).

Masking to the left ear:

- BC 3 kHz (p = 0.04, r = -0.56).
- BC 4 kHz (p = 0.02, r = -0.54).
- BC 6 kHz (p = 0.03, r = -0.56).
- BC 8 kHz (p = 0.009, r = -0.70).

Hearing threshold at 10,000 Hz

Masking to the right ear:

- BC 4 Hz (p = 0.01 r = -0.71).

- BC 6 kHz (p = 0.02 r = -0.67).
- BC 8 kHz (p = 0.008 r = -0.72).

Masking to the left ear:

- BC 3 kHz (p = 0.04 r = -0.59).
- BC 4 kHz (p = 0.02 r = -0.65).
- BC 6 kHz (p = 0.03 r = -0.64).
- BC 8 kHz (p = 0.01 r = -0.70).

Hearing threshold at 11,000 Hz:

Masking to the right ear:

- BC 3 kHz (p = 0.05 r = -0.31).
- BC 4 kHz (p = 0.01 r = -0.71).
- BC 6 kHz (p = 0.02 r = -0.68).
- BC 8 kHz (p = 0.008 r = -0.72).

Masking to the left ear:

- BC 3 kHz (p = 0.04, r = -0.59).
- BC 4 kHz (p = 0.03, r = -0.62).
- BC 6 kHz (p = 0.03, r = -0.64).
- BC 8 kHz (p = 0.005, r = -0.75).

Hearing threshold at 12,000 Hz

Masking to the right ear:

- BC 3 kHz (p = 0.04, r = -0.60).
- BC 4 kHz (p = 0.008, r = -0.72).
- BC 6 kHz (p = 0.01, r = -0.70).

- BC 8 kHz (p = 0.005, r = -0.75).

Masking to the left ear:

- AC 8 kHz (p = 0.03, r = -0.61).
- BC 3 kHz (p = 0.05, r = -0.58).
- BC 4 kHz (p = 0.01, r = -0.68).
- BC 6 kHz (p = 0.02, r = -0.67).
- BC 8 kHz (p = 0.005, r = -0.75).

Hearing threshold at 14,000 Hz

Masking to the right ear:

- AC 8 kHz (p = 0.02, r = -0.64).
- BC 3 kHz (p = 0.03, r = -0.64).
- BC 4 kHz (p = 0.005, r = -0.75).
- BC 6 kHz (p = 0.006, r = -0.74).
- BC 8 kHz (p = 0.002, r = -0.79).

Masking to the left ear:

- AC 8 kHz (p = 0.04, r = -0.61).
- BC 4 kHz (p = 0.03, r = -0.62).
- BC 6 kHz (p = 0.03, r = -0.63).
- BC 8 kHz (p = 0.006, r = -0.74).

Hearing threshold at 16,000 Hz

Masking to the right ear:

- AC 6 kHz (p = 0.05, r = -0.58).
- AC 8 kHz (p = 0.01, r = -0.68).

- BC 3 kHz (p = 0.05, r = -0.58).
- BC 4 kHz (p = 0.01, r = -0.70).
- BC 6 kHz (p = 0.01, r = -0.70).
- BC 8 kHz (p = 0.002, r = -0.80).

Masking to the left ear:

- AC 8 kHz (p = 0.02, r = -0.64).
- BC 3 kHz (p = 0.05, r = -0.58).
- BC 4 kHz (p = 0.01, r = -0.71).
- BC 6 kHz (p = 0.01, r = -0.70).
- BC 8 kHz (p = 0.003, r = -0.77).

Significant correlations were also found between hearing thresholds of the left ear and MMLs delivered to that ear across a range of frequencies using both AC and BC transducers.

Hearing threshold at 500 Hz:

- AC 0.25 kHz (p = 0.03, r = -0.63).
- AC 0.5 kHz (p = 0.17, r = -0.67).
- AC 2 kHz (p = 0.05, r = -0.58).
- BC 0.125 kHz (p = 0.03, r = -0.61).
- BC 0.25 kHz (p = 0.01, r = -0.69).
- BC 0.5 kHz (p = 0.03, r = -0.61).
- BC 1.5 kHz p = 0.04, r = -0.60).

Hearing threshold at 1000 Hz:

- BC 6 kHz (p = 0.03, r = -0.62).
- BC 8 kHz (p = 0.02, r = -0.65).

Hearing threshold at Hearing threshold at 4000 Hz:

- BC 4 kHz (p = 0.03, r = -0.62).
- BC 6 kHz (p = 0.04, r = -0.59).
- BC 8 kHz (p = 0.04, r = -0.59).

Hearing threshold at 8000 Hz:

- AC 8 kHz (p = 0.04, r = -0.61).
- BC 8 kHz (p = 0.02, r = -0.68).

Hearing threshold at 9000 Hz:

- AC 8 kHz (p = 0.04, r = -0.59).
- BC 4 kHz (p = 0.04, r = -0.59).
- BC 6 kHz (p = 0.02, r = -0.64).
- BC 8 kHz (p = 0.01, r = -0.69).

Hearing threshold at 10,000 Hz:

- BC 4 kHz (p = 0.04, r = -0.60).
- BC 6 kHz (p = 0.03, r = -0.63).
- BC 8 kHz (p = 0.03, r = -0.64).

Hearing threshold at 11.2 kHz:

- AC 8 kHz (p = 0.03, r = -0.63).
- BC 6 kHz (p = 0.02, r = -0.64).
- BC 8 kHz (p = 0.01, r = -0.71).

Hearing threshold at 12,500 Hz:

- AC 8 kHz ($p = 0.03$, $r = -0.63$).
- BC 4 kHz ($p = 0.02$, $r = -0.65$).
- BC 6 kHz ($p = 0.02$, $r = -0.67$).
- BC 8 kHz ($p = 0.01$, $r = -0.71$).

Hearing threshold at 14,000 Hz:

- AC 8 kHz ($p = 0.03$, $r = -0.62$).
- BC 4 kHz ($p = 0.04$, $r = -0.61$).
- BC 6 kHz ($p = 0.02$, $r = -0.65$).
- BC 8 kHz ($p = 0.008$, $r = -0.72$).

Hearing threshold at 16,000 Hz:

- AC 8 kHz ($p = 0.03$, $r = -0.64$).
- BC 2 kHz ($p = 0.04$, $r = -0.59$).
- BC 4 kHz ($p = 0.01$, $r = -0.70$).
- BC 6 kHz ($p = 0.01$, $r = -0.71$).
- BC 8 kHz ($p = 0.005$, $r = -0.75$).

Significant relationships were also found between right ears' hearing thresholds and MMLs delivered to the right ear across frequencies for both AC and BC headphones.

Hearing threshold at 1000 Hz:

- BC 8 kHz ($p = 0.04$, $r = -0.59$).

Hearing threshold at 8000 Hz:

- AC 6 kHz (p = 0.05, r = -0.58).
- AC 8 kHz (p = 0.02, r = -0.66).
- BC 3 kHz (p = 0.05, r = -0.58).
- BC 4 kHz (p = 0.02, r = -0.64).
- BC 8 kHz (p = 0.01, r = -0.70).

Hearing threshold at 9,000 Hz:

- BC 3 kHz (p = 0.05, r = -0.58).
- BC 4 kHz (p = 0.02, r = -0.67).
- BC 6 kHz (p = 0.04, r = -0.59).
- BC 8 kHz (p = 0.02, r = -0.66).

Hearing threshold at 10,000 Hz:

- AC 8 kHz (p = 0.04, r = -0.59).
- BC 3 kHz (p = 0.04, r = -0.60).
- BC 4 kHz (p = 0.01, r = -0.70).
- BC 6 kHz (p = 0.03, r = -0.64).
- BC 8 kHz (p = 0.01, r = -0.71).

Hearing threshold at 11.2 kHz:

- AC 8 kHz (p = 0.05, r = -0.58).
- BC 3 kHz (p = 0.03, r = -0.61).
- BC 4 kHz (p = 0.01, r = -0.70).
- BC 6 kHz (p = 0.02, r = -0.65).

- BC 8 kHz (p = 0.008, r = -0.72).

Hearing threshold at 12,500 Hz:

- AC 8 kHz (p = 0.04, r = -0.61).
- BC 3 kHz (p = 0.03, r = -0.63).
- BC 4 kHz (p = 0.01, r = -0.71).
- BC 6 kHz (p = 0.02, r = -0.67).
- BC 8 kHz (p = 0.01, r = -0.75).

Hearing threshold at 14,000 Hz:

- AC 6 kHz (p = 0.04, r = -0.59).
- AC 8 kHz (p = 0.02, r = -0.66).
- BC 3 kHz (p = 0.02, r = -0.69).
- BC 4 kHz (p = 0.01, r = -0.74).
- BC 6 kHz (p = 0.01, r = -0.70).
- BC 8 kHz (p = 0.003, r = -0.78).

Hearing threshold at 16,000 Hz:

- AC 6 kHz (p = 0.04, r = -0.59).
- AC 8 kHz (p = 0.01, r = -0.68).
- BC 3 kHz (p = 0.04, r = -0.60).
- BC 4 kHz (p = 0.01, r = -0.70).
- BC 6 kHz (p = 0.01, r = -0.69).
- BC 8 kHz (p = 0.002, r = -0.81).

Appendix B.

Significant Correlations with Demographics Data

This appendix contains all significant correlations that were found between participants' demographics data and their MMLs.

Significant correlations between age of the participant and air conduction MMLs:

- Masking at 8 kHz delivered to the right ear ($p = 0.02$, $r = -0.65$) and delivered to the left ear ($p = 0.02$, $r = -0.65$).

Significant correlations between age of the participant and bone conduction MMLs:

- Masking at 3 kHz delivered to the right ear ($p = 0.01$, $r = -0.70$).
- Masking at 4 kHz delivered to the right ear ($p = 0.02$, $r = -0.67$).
- Masking at 6 kHz delivered to the right ear ($p = 0.03$, $r = -0.62$) and delivered to the left ear ($p = 0.03$, $r = -0.61$).
- Masking at 8 kHz delivered to the right ear ($p = 0.01$, $r = -0.73$) and delivered to the left ear ($p = 0.01$, $r = -0.69$).

Significant correlations between the number of years with tinnitus and bone conduction masking at the left ear at 8 kHz ($p = 0.04$, $r = -0.61$).