Chapter 72
Auditory Training in Tinnitus

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Keypoints

1. We reviewed sensory training studies from the point of view that tinnitus is caused by synchronous neural activity that develops in tonotopic regions of primary auditory cortex deafferented by hearing loss. Studies were classified according to whether training was conducted within the tinnitus frequency region or outside of it, and whether training was active (requiring behavioral responses) or passive (sounds were presented as background signals). Effects of training on the psychoacoustic properties of tinnitus were distinguished from those on the distress behavior that accompanies tinnitus.
2. Studies in all four categories have reported significant reductions compared to untreated controls in tinnitus distress, measured by standardized questionnaires and visual analogue scales at the first compared to untreated controls in-course assessment, with little further change thereafter. Because the particular details of sensory training do not appear to matter, these gains could reflect important nonspecific effects of the treatment procedures.
3. Psychoacoustic measures may more directly assess tinnitus sensations. Reductions in minimum masking level (MML) on the order of 5–10 dB have been reported by several studies, implying that tinnitus has become weaker. Improvements in loudness discomfort levels (LDL) have also been reported, as have changes in the frequency content of tinnitus. Improvements in MML and LDL are more gradual than those on distress behavior assessed by questionnaires, suggesting that neural plasticity may be at work.
4. Several studies reporting improvements in psychoacoustic measures and questionnaire data used passive sound presentation procedures. Hence, active sensory training requiring discriminated behavioral responses is not needed for these changes.
5. Systematic manipulation of the frequency content of trained sounds has been attempted in only a few studies. This step is needed to determine whether sound training induces specific changes in tinnitus. Alternatively, sound therapy may amplify the nonspecific effect of elements common to all tinnitus therapies.
6. Future studies should continue the practice of specifying how many participants of the total recruited contributed to a data analysis, and why and when exclusions occurred. Substantial sample sizes will be needed to establish treatment effects. Neural correlates offer the advantage of comparative immunity to patient expectations and self-report bias. When sounds are used to evoke neural responses, changes in loudness recruitment consequent on rescaling of loudness growth functions by sound exposure are a potential contributing factor.

Keywords Tinnitus • Sensory training • Tinnitus distress • Minimum masking level • Loudness discomfort levels

Abbreviations

A1	Primary auditory cortex
A2	Secondary auditory cortex
ADT	Auditory discrimination training
AOIL	Auditory object identification and localization

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ASSR	Auditory steady-state response
EAE	Enriched acoustic environment
LDL	Loudness discomfort level
LM	Loudness matching
MML	Minimum masking level
QE	Quiet environment
RI	Residual inhibition
THI	Tinnitus handicap inventory
THQ	Tinnitus handicap questionnaire
TRQ	Tinnitus reaction questionnaire
TRT	Tinnitus retraining therapy
TSI	Tinnitus severity index
VAS	Visual analog scales

Introduction

In 1995, Jastreboff [1] proposed a comprehensive model of tinnitus that addressed three clinically prominent features of this condition. These were (a) the tinnitus sensation itself, generated by pathology in the inner ear; (b) the ability of the tinnitus sensation to command attention; and (c) the patient's disturbing emotional reaction to the tinnitus percept. Jastreboff suggested that although elimination of the tinnitus sensation by treatment of cochlear pathology was in most cases not practical, the latter two features of tinnitus were likely modifiable and if treated would benefit the tinnitus patient. Tinnitus retraining therapy (TRT) was devised to foster extinction of attentional and emotional responses by presenting low-level tinnitus-like external sounds that could be filtered out along with the tinnitus by perceptual mechanisms (see Chap. 73). Studies of TRT and clinical experience have confirmed that emotional responses diminish with time for most tinnitus sufferers, as does the extent to which tinnitus sufferers attend to their tinnitus percept [2]. These are important and beneficial effects for tinnitus sufferers. Attempts to reduce or eliminate the tinnitus sensation itself, however, have met with less success.

One approach that has gained attention with respect to the latter goal in recent years is sensory training aimed at modifying the neural basis of tinnitus sounds. The inspiration for this approach was based in part on the discovery that hearing loss induced by noise exposure in animal models leads to a substantial reorganization of tonotopic maps in primary auditory cortex, such that frequencies near the edge of normal hearing come to be overrepresented at the expense of frequencies in the hearing loss region [3–5]. Because hearing loss is a putative cause of tinnitus, it was suggested that this overrepresentation, or changes in the response properties of auditory neurons associated with it, may correspond to the tinnitus percept [6, 7]. A second foundation was laid by experiments conducted in the last 15 years that demonstrated that cortical representations for sound in the primary auditory cortex are not fixed after early development, as was once believed, but can be modified by auditory training well into adulthood [8, 9]. This phenomenon is called "neural plasticity" (see Chap. 12). These two lines of research have converged to ask whether sensory training procedures derived from animal research can be adapted to humans, with the goal of modifying neural representations that appear to underlie tinnitus.

A Framework for Sensory Training Studies

For this goal to be achieved, the neural modifications induced by sensory training must intersect with the neural mechanisms generating tinnitus. In Chap. 13, we reviewed evidence pointing to a role for *neural synchrony* (temporally coupled neural activity) in tinnitus. According to this viewpoint, tinnitus may be generated by synchronous neural activity that develops in reorganized tonotopic regions of primary auditory cortex that receive diminished input from the ear owing to hearing impairment caused by noise exposure, otological disease, or the aging process [7]. Changes in subcortical structures appear to contribute [10] and may account, as well, for some distinct properties of tinnitus including its modulation by somatosensory activity in many individuals [11, 12]. Although the thalamocortical input to the affected neurons in the primary auditory cortex (A1) is altered by hearing loss, their synchronous output remains intact and may be a driving force underlying tinnitus. This output may recruit other brain regions into a network identified by functional imaging studies [13], including frontal and limbic areas that subserve, respectively, the attentional and emotional aspects of tinnitus described by Jastreboff [1].

In this chapter, we use the neural synchrony model as a template for reviewing auditory training studies of tinnitus. This perspective suggests that it is necessary

to reduce synchronous neural activity occurring in regions of A1 that have been affected by hearing loss, in order to reduce the loudness of tinnitus sounds. Training for sounds in the tinnitus frequency region, with the aim of segregating synchronous network activity in this region, would appear to be the most direct approach. Masking sounds presented to this frequency region induce optimal post-masking suppression of tinnitus [residual inhibition, (RI)], confirming that such sounds interact with the tinnitus generating mechanism [14]. Training in the tinnitus frequency region requires that significant residual hearing be present in this region, which is the case for many, but not all, tinnitus patients. Alternatively, training can be delivered outside of the tinnitus frequency region where hearing is generally better preserved. For example, training at or below the edge frequency region may alter neural representations in these regions, which send collateral inputs into the tinnitus region that may disrupt neural synchrony. Lateral inhibition arising from augmented representations below the tinnitus frequency range could also distribute into the tinnitus region and suppress tinnitus percepts. Inhibitory interactions have been demonstrated by human electrophysiological studies [15, 16] and are known to span several octaves in primate A1 [17], suggesting the feasibility of this approach. While the neural synchrony model focuses on A1 as a preferred site of action, several brain structures are active in tinnitus, including regions of the secondary auditory cortex (A2) that may distribute re-entrant feedback into the auditory core region and disrupt neural activity underlying tinnitus [18]. Remodeling of cortical representations in A2 by sensory training appears to proceed normally in the tinnitus brain (see Chap. 13) and may confer a benefit.

Several methodological limitations should be acknowledged in advance of this review. Auditory training procedures are aimed at modifying the neural processes that generate tinnitus sensations. In order to assess whether this goal has been achieved, it is desirable to employ psychoacoustic tools that more or less directly measure the sensory attributes of tinnitus, such as loudness matching (LM), in which the loudness of an external sound in the range of normal hearing is adjusted to equal the loudness of tinnitus, and minimum masking level (MML), the minimum loudness of a masking sound required to just cover tinnitus. Loudness discomfort level (LDL) is another useful psychoacoustic method, which measures loudness growth functions that are frequently elevated in individuals with tinnitus [19], as are their audiograms. Standardized procedures for measuring tinnitus spectra are also available [14, 20] and beneficial for characterizing tinnitus. However, only a minority of studies report such measures. More often, standardized questionnaires such as the tinnitus handicap questionnaire (THQ) [21], tinnitus severity index (TSI) [22], tinnitus handicap inventory (THI) [23], and tinnitus reaction questionnaire (TRQ) [24] are employed in which tinnitus patients rate on subjective scales the loudness and intrusiveness of their tinnitus and its effect on quality of life including mood and anxiety, interference with sleep, concentration, work productivity, and interpersonal relationships. While these questionnaires – often supplemented with tinnitus ratings on visual analog scales (VAS) – likely reflect to some degree the sensory properties of tinnitus, they tend to focus on the distressful consequences of having tinnitus emphasized by Jastreboff [1]. A further limitation is that few studies have controlled for the contribution of procedural elements that are likely common to all therapeutic approaches and may affect outcome regardless of any direct effect of auditory processing on the neural substrate of tinnitus. Examples of such elements include (a) beneficial effects of discussion with informed and sympathetic staff, (b) knowledge about tinnitus, (c) investment by patient and staff in a therapeutic process, and (d) the effect of these components on a hopeful attitude and expectations for success. In this chapter, we will refer to effects of these elements as "nonspecific effects", not to diminish their considerable importance for benefiting patients, but in order to distinguish them from effects attributable to the specific sounds incorporated into an auditory training procedure.

Notwithstanding these limitations, several approaches to auditory training have been tried or are currently under assessment. The results give a picture of the methods used, whether the goal of auditory training can be realized, and if not for all tinnitus patients, which variables may be important for treatment success. Because active training requiring explicit behavioral responses might confer a benefit in tinnitus, we categorize the studies into active procedures that require such responses and passive procedures that do not. We also categorize the studies according to whether sounds are presented to the tinnitus frequency

(hearing loss) region or outside of this region. Animal studies are included where they are relevant. One novel approach is described that does not fit into these categories.

Active Training Within the Tinnitus Frequency Region

Several studies have assessed the effects of auditory training procedures at or near the tinnitus "pitch" (likely resembling the modal pitch in a tinnitus spectrum). Based on their results studying phantom limb pain where it had been shown that the amount of cortical reorganization was positively correlated with amount of pain [25] and that discrimination training in sensory areas adjacent to the deafferented region reduced phantom limb pain [26], Flor et al. [27] trained seven tinntius patients on a frequency discrimination task for tones matched to their tinnitus frequency (proximal frequency group), with an additional seven patients trained at a frequency distant from the tinnitus frequency (distal group). The participants in this study were asked to determine if two tones presented successively were either identical (50% of trials) or different in frequency and were given feedback for correctness. The difficulty of the task was increased with performance improvement across sessions. Training was to be carried out every day for 2 h over a 4-week period. Interestingly, two of the seven distant-frequency participants dropped from the study complaining of increases in tinnitus severity, suggesting an adverse effect of training below the tinnitus frequency region. At the end of training, the proximal and the remaining distal patients did not differ on any outcome measures, so they were combined for analysis. Given the unreliability of tinnitus pitch match procedures [28], some patients in the distal group may still have trained at frequencies within their tinnitus spectrum. No significant training effect on tinnitus severity was found, but not all patients complied with the training requirements. When the participants were separated post hoc into those who trained more ($n=7$) or less ($n=5$) over the 4-week period, the extensive training group showed significant reduction in self-reported tinnitus severity while the limited training group showed a significant increase in tinnitus severity. Cortical reorganization or changes in the psychoacoustic properties of the tinnitus were not assessed. Given that the treatment effect was not limited to the group training on frequencies within the tinnitus frequency region, it appears that potentially nonspecific factors such as focusing attention away from tinnitus might have been responsible for the lessening in severity.

Herriaz et al. [29, 30] described the results of a number of similar procedures, which they collectively referred to as ADT (auditory discrimination training). In all patients, the stimuli to be discriminated fell within the region of hearing loss. However, the procedures differed from those used by Flor et al. in that the discrimination in most cases was relatively easy (for example, discrimination between a broadband noise and an 8 kHz pure tone) and task difficulty did not increase with training (non-adaptive procedure). Training sessions were relatively short in the largest test group ($n=29$), with the participants required to perform 10-min sessions twice daily for a 1-month period. These procedural changes allowed the patients to perform the task at home using an MP3 device. Significant improvements in self-reported tinnitus severity on a VAS scale of loudness and total score on the THI questionnaire were found compared to waitlist controls. However, because no assessments of the psychoacoustic properties of tinnitus (LM, MML, or tinnitus spectrum) were performed, it is difficult to attribute the tinnitus improvement to a reversal of the presumed cortical reorganization. In another study, participants in one group (SAME, $n=11$) trained at frequency discrimination at a pitch judged to be the same as the tinnitus pitch while the second group (NONSAME, $n=11$) trained at a frequency different from the tinnitus pitch but still within the region of hearing loss. The NONSAME group showed a larger reduction in THI score with the difference between the groups being significant. Like Flor et al. [27], these results suggest that training at the "tinnitus pitch" was not a requirement for reduction in tinnitus severity. However, because the trained pitch in the NONSAME group was in the region of hearing loss, some degree of overlap with the tinnitus spectrum was likely.

Norena et al. [20] trained a single individual on a frequency discrimination task for four frequencies within the participant's measured tinnitus spectrum, and also measured the frequency discrimination threshold during training using an adaptive forced-choice staircase procedure. Training occurred in seven sessions over 3 weeks and was performed monaurally

although the participant had bilateral tinnitus. The tinnitus spectrum changed significantly post-training in the trained ear but not the untrained one, showing a marked reduction in likeness ratings at the highest frequencies. This individual reported informally that the tinnitus sensation shifted from the initially more salient trained ear toward the untrained ear. However, the changes in the tinnitus spectrum occurred at the highest measured frequencies rather than at the frequencies used in the training procedure. This raises the possibility that the changes observed in the tinnitus spectrum could be attributed to an improved ability of the participant to make better discriminations at higher frequencies, allowing more refined judgments of the tinnitus spectrum. The unilateral effect of the tinnitus spectrum change supports the idea that the discrimination training process induced changes in the frequency organization in the auditory cortex. Follow-up studies employing more participants are called for.

In a preliminary study of our own (see Chap. 13 and [31]), we departed from the frequency discrimination training paradigm to one requiring detection of targets of increased sound *intensity* that were embedded in a 40-Hz amplitude-modulated tone of 1-s duration (carrier frequency 5 kHz, in the tinnitus frequency region). This type of stimulus evokes the stimulus-driven 40-Hz "auditory steady-state response" (ASSR) that localizes tonotopically to the region of primary auditory cortex and gives a picture of events occurring in this region during auditory training. Previous research with frequency (not intensity) discrimination had shown that acoustic training advanced the phase of the ASSR (a shortened time delay between the 40-Hz stimulus and response waveforms), but the amplitude of the response (signaling a map expansion) did not change [32]. We therefore switched to the intensity discrimination procedure using a single carrier frequency, which reduced competitive interactions that may obstruct map expansions when several carrier frequencies are experienced [33]. If training at 5 kHz strengthened the thalamocortical tuning of the trained neurons, tinnitus might diminish at this frequency as the affected neurons were removed from synchronous network behavior underlying tinnitus. Measurement of the tinnitus spectrum before and after training showed little change at 5 kHz or any other tinnitus frequency after training. However, in individuals with tinnitus, auditory training did not change ASSR phase either ($n=8$ participants, $p=0.44$), although it did so in their age-matched controls ($n=11$ participants, $p=0.006$) suggesting impaired remodeling of primary auditory cortex in the tinnitus group. A different brain response that is known to be neuroplastic [32] and to localize to secondary auditory areas is the P2-evoked auditory potential (latency ~ 180 ms). P2 amplitude increased with training in both groups ([31]; see Chap. 13), suggesting normal remodeling of secondary areas in tinnitus. However, this remodeling had no effect on tinnitus. The results of this study could change as additional participants and groups are tested.

Active Training Outside the Tinnitus Frequency Region

Based on the proposal that the tinnitus percept elicits abnormal levels of attention, Searchfield et al. [34] trained 10 individuals with tinnitus on an auditory object identification and localization (AOIL) task designed to refocus the participants' attention on external stimuli. Training (approximately 30 min per day over 15 days) consisted of up to 20 listening tasks that required subjects to identify and locate in space (left, right, centre) a number of common sounds (e.g., spoken words, owl hooting, coughing, dog barking) against a variety of background noises. The frequency of the sounds and background noises were not explicitly designed to fall below the frequency region of hearing loss or tinnitus spectrum, although the dominant frequencies were likely in this region. Subjects showed a 6-dB reduction in tinnitus loudness assessed by LM, and a significant reduction in pitched matched MML (in eight of ten participants, up to 30 dB in one person). The experiment is noteworthy for its inclusion of psychoacoustic measures. This type of training explicitly targeting the attentional system (but not using sounds focused within the tinnitus region) produced changes similar to those seen in other training procedures that presented stimuli within the tinnitus spectrum.

Another approach similar to active training on sound discrimination is the restoration of behaviorally relevant input via prostheses. There are a number of studies that report cochlear implants having a suppressive effect on tinnitus (see Baguley and Atlas [35] for a review) (see Chap. 77), and hearing aids have also proven to be beneficial (see Chap. 74). Folmer et al. [36]

found that out of 50 patients purchasing and wearing a hearing aid, 46 reported at least "a little" improvement in their tinnitus, with 11 reporting "very much" after 6–48 months. The self-rated loudness of their tinnitus was significantly reduced from 7.5 to 6.3 out of 10 on a VAS. The matched pitch of their tinnitus was 4.3 kHz, which likely means that the aids (which typically have low-frequency amplification profiles) restored little input near their tinnitus frequency. However, Moffat et al. [37] fitted nine subjects with hearing aids with a high bandwidth amplification regime (20 dB threshold reductions at 6 and 8 kHz) and found no changes in the tinnitus spectrum or tinnitus loudness after 30 days. Interestingly, a second group fitted with a low-medium frequency amplification hearing aid showed a significant diminution of low-frequency components of the tinnitus spectrum, with no effect seen at middle or high frequencies. The authors suggested that the perceptual characteristics of tinnitus depend on a contrast between adjacent central auditory regions of more and less afferent activity, which was increased by the low frequency amplification profile. The limited malleability of the tinnitus percept in the high amplification group may be due to the extent of hearing loss in this region and the robustness of neuroplastic changes that give rise to tinnitus. Neither amplification group, however, reported a reduction in tinnitus when assessed by LM.

Passive Experience Within the Tinnitus Frequency Region

Restoration of input via prostheses restores auditory input in a behaviorally relevant manner, which supports classification of these procedures as active training. However, animal data (and training studies in normal hearing humans) suggest sound input need not be behaviorally relevant in order to effect changes. Norena and Eggermont [38] found that tonotopic map reorganization in cats exposed to traumatic noise can be prevented by subsequent immersion in an enriched acoustic environment (EAE) containing background sounds designed to compensate for the frequency-dependent decrease in sensory inputs from the hearing loss region. This procedure also led to a recovery from hearing loss between 16 and 32 kHz in the EAE cats, compared to cats exposed to an identical noise trauma but placed in a quiet environment (QE). The increased spontaneous firing rates and increased neural synchrony, which underlies the neural synchrony model, were also absent in EAE cats [39]. Subsequent research showed that passive exposure to the EAE for 6 weeks can produce tonotopic reorganization in normal adult cats in the *absence* of any noise trauma, *suppressing* sound representations in the EAE frequency region, and without inducing any threshold changes [40]. These findings accord with other data indicating that passive exposure to environmental sounds can lead to neuroplastic changes in the absence of explicit training requirements [41–44].

Is restoring acoustic input in the tinnitus frequency region, even if this input is not behaviorally relevant, sufficient to normalize frequency representations and reduce the neural synchrony possibly underling tinnitus, in subjects for whom significant residual hearing is present in this frequency region? The most direct evidence comes from three studies initiated by Neuromonics (see Chap. 75), a private company (http://www.neuromonics.com) that markets a device that delivers spectrally manipulated music tailored to augment frequencies in the hearing loss region of the patient's audiological profile. Because the tinnitus spectrum typically tracks the hearing loss region [14], this sound (presented at levels covering fully or partially the tinnitus) would be expected to inject feed forward and surround inhibition into the relevant region, disrupting the tinnitus sound. Patients screened for residual hearing in the loss region were instructed to listen passively to the sound for at least 2 hours per day using a high fidelity sound player with ear phones over a treatment period of 12 months. In the initial months, patients were told to set the sound level so that their tinnitus was fully masked, and then in subsequent months to gradually reduce this level, so that tinnitus was intermittently heard. This sound therapy approach was combined with counseling following the method of systematic desensitization in which aversive stimuli (in this case, tinnitus) are experienced gradually and in a context conducive to relaxation. In three studies [45–47], Neuromonics treatment led to a substantial reduction in tinnitus distress measured by the TRQ at the first assessment taken 2 months into the study, with little further improvement and little remission in the 10 months of treatment following thereafter. VAS ratings assessing tinnitus severity, ability to relax, and loudness tolerance also improved, following a course similar to the TRQ data. Notably, psychoacoustic

measurements of MML and LDL were also taken in each study. In each study, MML decreased progressively over the 12-month treatment interval, while LDL levels increased.

In order to assess whether sound therapy contributed to these beneficial results, Davis et al. [46] contrasted questionnaire and psychoacoustic data among groups that received Neuromonics treatment (Neuromonics sound therapy with counseling, $n=21$ subjects), broadband noise masking with counseling ($n=15$), or counseling alone ($n=13$). After 12 months, subjects in the Neuromonics treatment group reported a 66% reduction in TRQ scores, compared to reductions of 22 and 15% reported by subjects in the masking and counseling alone groups, respectively (the differences between the Neuromonics group and other two groups were statistically significant). In agreement with these results, tinnitus severity assessed by VAS was reduced in the Neuromonics group, compared to the two control conditions. The Neuromonics group also reported a reduction of 11.3 dB in MML ($p<0.001$) at 12 months, compared to non-significant reductions of 0.4 and 1.5 dB in the masking and counseling alone groups, suggesting a benefit of Neuromonics treatment on tinnitus loudness. However, an aspect of this study that should be noted is the high proportion of subjects who were either eliminated prior to treatment for failure to meet admission criteria ($n=19/88$) or were excluded from the final analysis for other reasons ($n=24/88$, overall exclusion rate 48.9%). Among the exclusions were subjects with entering TRQ scores lower than 14/100 who typically show little gain from treatment [2, 47]. It should also be noted that while improvements in the psychoacoustic measures in the Neuromonics group suggest that sound exposure mattered, the effect of spectrally enhancing sounds outside rather than inside the tinnitus frequency region has not been investigated. Evidence on this question is needed to determine whether the specific frequency of the sounds that subjects listen to is crucial for therapeutic gains, or whether the experience of sound (regardless of frequency) amplifies nonspecific contributions by increasing patient involvement and treatment plausibility.

Other evidence supports the contention that passive listening to sounds that cover tinnitus frequencies can reduce tinnitus. In a study cited previously, Folmer et al. [36] fitted 50 subjects with in the ear sound generators producing broadband (100–8,000 Hz) noise and found that self-rated tinnitus loudness significantly reduced from 7.6 to 6.2 on a ten-point VAS scale. However, this improvement was about the same as a group fitted with hearing aids that likely did not restore much high-frequency input. TRT provides exposure to a broadband masking stimulus that resembles tinnitus but is presented at lower loudness levels (called the "mixing point") approximating the tinnitus loudness [1] (see Chap. 73). TRT has been found to lead to decreases in tinnitus distress (measured by the TSI, THI, and THQ) that are initially less than improvements produced by masker therapy [2]. However, after 12–18 months of treatment, improvements induced by TRT exceeded those of masker therapy [2], suggesting that the listening protocol may contribute a role.

Whether covering the tinnitus frequencies are crucial remains unclear, however. In a study modeled on animal data reported by Norena and Eggermont [38, 39], Norena and Chery-Croze [19] exposed individuals reporting abnormal loudness recruitment (hyperacusis) to a background sound containing high frequencies spectrally enhanced over the region of hearing impairment, in a manner similar to EAE-exposed cats. The participants in the study listened to the sound in the background for 3 h per day over 15 weeks. Passive listening rescaled loudness growth functions in the direction of normal hearing over this interval, with some regression over a period of 1 month after passive listening ceased. Effects on tinnitus were not assessed, although a majority of subjects with hyperacusis typically report tinnitus as well [48]. The specific frequency content of the sound was not manipulated in this study (all subjects received a high-frequency amplification profile). In a study of individuals with normal hearing, Formby et al. [49] found that loudness growth functions can be bi-directionally rescaled by enhancing or reducing background acoustic environments. These results, which appear to be mediated in part by subcortical mechanisms [50], show that passive exposure can selectively remodel auditory processing in humans. Whether concomitant effects are seen on tinnitus remains to be investigated.

Passive Experience Outside the Tinnitus Frequency Region

Except for the possibility (discussed above) that effects of hearing aid amplification on tinnitus may be

attributable in part to passive exposure to sounds below the tinnitus frequency region, passive sound therapies restricted to this region have not been widely studied. However, a recent study by Okamoto et al. [51] can be discussed here.

These investigators reasoned that because hearing loss is often present in the tinnitus frequency region, auditory training may be more effective if delivered to frequency regions where hearing is better preserved. Their approach was based on an earlier series of studies by their group in normal hearing subjects [16], which showed that notched sound can suppress neural activations in the notched region by distributing lateral inhibition to these regions. Okamoto et al. [51] therefore gave chronic tonal tinnitus patients in a treatment group daily experience with their favorite music that had a one-octave notch around their dominant tinnitus frequency removed. A placebo group listened to similar musical stimuli, except that the notch shifted over the course of training but was never at the tinnitus frequency. Subjects in the treatment and placebo groups listened for about 12 hs/week over 12 months. A further control group (monitoring) received no treatment but participated in the study measurements. Tinnitus loudness measured by a VAS was significantly reduced from baseline in the treatment group, but changes in VAS ratings did not reach significance in the placebo or monitoring groups. A comparison of the VAS changes between the treatment and placebo group ratings was also significant (this comparison was not made for the monitoring group). Notably, the amplitude of the 40-Hz ASSR and the N1m response to tonal stimuli delivered at the tinnitus frequency were also reduced in the treatment group after their sound therapy, relative to these responses evoked by a control frequency (500 Hz). These brain measures did not change in either of the control groups (a comparison of the treatment and placebo groups was also significant in this measure). Hence, evidence for a brain correlate of tinnitus suppression was observed with the notching procedure. This study is notable for inclusion of control conditions designed to evaluate whether the specific frequency content of auditory training is crucial for tinnitus improvement and for carrying out brain imaging measures. A limitation, however, is that of 39 subjects that met the criteria for entry into the study, only 23 contributed data in the treatment ($n=8$), placebo ($n=8$), and monitoring ($n=7$) groups. Subjects were included in the final statistical analyses only if their subjective tinnitus pitch did not change over the study and if the median of repeated pitch matches fell within the notched region for subjects in the experimental group, which are reasonable criteria for a study of this design. Further research is called for to corroborate the findings and assess the limits and magnitude of possible treatment effects.

Other Approaches

Jepsen and her colleagues have proposed an alternative approach to the treatment of tinnitus based on the concept of category training [52]. This approach is modeled on studies by Guenther et al. [53] in normal hearing subjects, which found that training to classify non-speech stimuli within a particular frequency range as members of the same category (frequency categorization training) led to a decrease in discrimination ability for frequencies within the category. In subsequent research [54], frequency categorization training led to a relative decrease in neural activation measured by fMRI for the trained frequencies, whereas conventional training for discrimination among the same frequencies augmented neural activation for the trained stimuli.

Jepsen et al. [52] hypothesized that it would be advantageous to train subjects experiencing tinnitus to assign tinnitus frequencies to a common category, which might lead to a reduction in activation in this area of cortex and presumably a concomitant decrease in the tinnitus sensation. They trained 20 subjects for 30 min per day for 3 weeks, either to categorize tinnitus frequencies into a group or to discriminate among the frequencies, in each case using a take-home training device. The two groups did not differ markedly in their pre–post THI score changes, but did show differences in auditory-evoked potentials. The categorization group showed a reduction in P2-N1 amplitude post-training while the discrimination group showed an increase, which is in line with the observations of Guenther et al. [54]. However, this change was most evident for a control (untrained) frequency rather than the trained frequency, again indicating a more nonspecific effect of training rather than a reduction in cortical activation for the tinnitus region. Category training merits further investigation for its effects on discrimination ability, neural responses, and tinnitus.

Overview and Conclusion

Animal research in the last two decades has established that neural plasticity is a fundamental property of neurons in the auditory and other sensory systems. Evidence has also accumulated that hearing loss (a triggering factor in many if not most people with tinnitus) leads to changes in central auditory pathways, including tonotopic map reorganization and increased neuron firing rates that may be forged by neuroplastic mechanisms into abnormal network behavior generating tinnitus sounds. These findings have spawned renewed research into the question of whether tinnitus can be reduced or eliminated by auditory training specifically designed to normalize aberrant auditory neural representations that are believed to be responsible for tinnitus. For this goal to be achieved, it must be possible to modify auditory representations by acoustic training in individuals with tinnitus, and the neural modifications induced by training must intersect with the underlying tinnitus mechanisms.

In this chapter, we reviewed auditory training studies from the point of view that tinnitus is caused by synchronous neural activity that develops in tonotopic regions of primary auditory cortex that have been deafferented by hearing impairments. Studies were classified according to whether training was conducted within the tinnitus frequency region or outside of it, and whether the trained sounds served as cues for behavioral responses and were therefore processed actively in attention, or whether the sounds were presented passively as background signals. We also attempted to separate the effects of auditory training on two distinct aspects of tinnitus emphasized by Jastreboff [1], namely, effects on the tinnitus percept itself and effects on distress behavior that accompanies tinnitus. The following summary statements appear to be justified.

1. The number of auditory training studies is not large, and the studies do not evenly cover the four categories we used for classifying them.
2. Studies in all categories have reported significant reductions in tinnitus distress measured by standardized questionnaires (THQ, TRQ, TSI) and VAS ratings. These reductions typically achieved their maxima at the first in-course assessment, with relatively little if any gain thereafter. A noteworthy result is that two treatment procedures that manipulated the frequency content of sounds in the tinnitus frequency region in opposite directions [46, 51] reported similar tinnitus reductions in VAS ratings. If the particular details of auditory training do not matter for these improvements, these gains would appear to be attributable to nonspecific effects of the treatment procedure.
3. Because these changes on questionnaires and VAS ratings are beneficial for patients, it is important to identify the factors responsible for them. Benefits may be greater when some form of sound therapy is employed, although further evidence on this point and particular sound therapy used is needed. Another factor relevant to a successful treatment outcome is opportunity for improvement. Several studies have reported that reductions in distress behavior are minimal when tinnitus distress is low at study commencement.
4. Changes in psychoacoustic measures have been reported that may more directly measure tinnitus sensations. Reductions in MML on the order of 5–10 dB have been reported by several studies [34, 45–47], implying that the tinnitus sensation has become weaker. MML may be a better measure of tinnitus loudness than adjusting external sounds to match tinnitus, which is known to be frequency dependent [14]. Improvements in loudness tolerance (LDL) have also been reported [45–47], as have changes in the frequency content of tinnitus [20, 37]. Improvements in MML and LDL are more gradual than those on distress behavior, suggesting that some form of neural plasticity may be at work.
5. Several of the studies reporting improvements in psychoacoustic measures used passive sound presentation procedures. Hence, active training requiring discriminated behavioral responses does not appear to be necessary for changes in psychoacoustic measures. This observation aligns with experiments in normal hearing animals and humans which found that passive exposure to sound can be sufficient to remodel auditory representations.
6. Animal data and the neural synchrony model of tinnitus imply that training for sounds that cover the tinnitus frequency region is likely to be most effective in modifying tinnitus, provided that residual hearing is present in this region. The results on this point are, however, conflicting. With a few exceptions [27, 37, 51], systematic manipulation of the

frequency content of the trained sounds has not been attempted in auditory training studies. Loudness growth curves are rescaled in normal hearing individuals by augmenting or reducing background sound [49], and rescaling occurs in hyperacusis patients exposed to high-frequency complex sounds [39], in both situations with broad frequency selectivity. However, applications of these procedures to tinnitus remain largely untested. Because the measurement of brain correlates often involves presenting sounds, effects of sound therapy on loudness recruitment are potential contributing factors to such measurements in tinnitus.

While these conclusions are less than satisfying, they do give guidance for continuing study. Future research should emphasize psychoacoustic measures, particularly MML and LDL, as well as standardized measures of tinnitus spectra [14, 20] which can obviate some of the unreliability of single-pitch matches, in addition to standardized questionnaires. Systematic variation of trained frequencies between groups or within individuals is highly desirable, including untreated control conditions. Such evidence is needed to determine whether auditory training induces specific changes in tinnitus, or whether it instead amplifies the nonspecific effect of procedures common to all tinnitus therapies. Neural correlates offer the advantage of comparative immunity to response bias. Finally, care should be taken to specify clearly how many participants of the total recruited contributed to a data analysis, and why and when exclusions occurred. Progress toward an optimal auditory training treatment will be limited until replications are reported involving substantial sample sizes.

We also suggest that applications of auditory training will be enriched when we know more about how neural plasticity works in normal hearing individuals and in individuals with tinnitus. Current findings showing that passive exposure to sound is sufficient to remodel auditory representations in people with normal hearing could be good news for tinnitus, since compliance with treatment procedures may improve when performance requirements are minimal. The results reflect the propensity of the human auditory system to extract and represent the features of salient environmental sounds, regardless of behavioral response requirements. However, that passive exposure is sufficient does not preclude the possibility that active processing may yield more long-lasting outcomes [51].

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