
# Tinnitus, hearing loss and hearing aids

Tinnitus is the perception of a sound in the absence of a corresponding external acoustic stimulus. In more than 90% of the cases, tinnitus is a purely subjective phenomenon, "objective tinnitus" where an actual physical sound source within the body can be identified is rare. An example for objective tinnitus would be pulsatile tinnitus generated by a blood vessel anomaly close to the ear. In this review, we will focus on "subjective tinnitus" where no physical generator is present, and simply refer to it as tinnitus in the following. The phenomenon of tinnitus has already been described in Egyptian, Greek and Roman scripture, and the term tinnitus itself might have been coined by Pliny the Elder. Ancient explanations of tinnitus involved, for example, spirits sending messages or tiny crickets living in the ear. Luckily, our understanding of tinnitus has progressed since then. In the past two decades, the field of tinnitus research has developed considerably, and tinnitus is no longer seen as purely a topic of ENT medicine. The discovery that tinnitus is not generated in the ear, but in the brain itself, has made tinnitus a topic of neuro-science research, and the neuroscientific approach to tinnitus has greatly enhanced our understanding of the neural mechanisms that are involved in the generation of tinnitus in the brain.

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## Hearing loss and tinnitus

Several lines of evidence from tinnitus patients and animal models of tinnitus point towards a causal relation between hearing loss and tinnitus. Firstly, the majority of tinnitus patients also have a hearing loss (Axelsson and Ringdahl, 1989; Nicolas-Puel et al., 2002), the prevalence of tinnitus rises with hearing impairment (Chung et al., 1984), and tinnitus patients have elevated hearing thresholds compared to age-matched controls (Roberts et al., 2008). Also, 75–90% of patients with otosclerosis experience tinnitus (Ayache et al., 2003; Sobrinho et al., 2004). Moreover, even tinnitus patients with normal hearing in the clinical sense, i.e., hearing thresholds ≤20 dB HL up to 8 kHz, do in fact have a certain degree of cochlear damage that is not detected by conventional audiometry. This "hidden hearing loss" has been detected using psychophysical tests (Weisz et al., 2006) and more directly through auditory brain stem response measurements, where tinnitus subjects with normal audiograms had reduced amplitudes of the auditory nerve signal (Schaette and McAlpine, 2011; Gu et al., 2012). Further indication for a relation between hearing loss and tinnitus comes from tinnitus pitch measurements, as tinnitus patients with sensorineural hearing loss usually match the pitch of their tinnitus sensation to frequencies where their hearing is impaired (Norena et al., 2002; König et al., 2006; Sereda et al., 2011). Probably the most direct indication of a causal relation comes from patients with idiopathic sensorineural hearing loss, as around 80% of them also develop tinnitus (Nosrati-Zarenoe et al., 2007). Moreover, phantom sounds can also occur when conductive hearing loss is simulated through an earplug. In a study where 18 normal-hearing volunteers wore an earplug in one ear for 7 days, 14 reported hearing phantom sounds, and 11 perceived a stable phantom sound at day 7. Upon removal of the earplug, the phantom sounds disappeared within a few hours (Schaette et al., 2012). These findings demonstrate that there might be a causal relation between hearing loss and tinnitus, and that tinnitus can be linked to hearing loss in the majority of cases.

The link between hearing loss and tinnitus has been studied in more detail in animal studies. After noise-induced hearing loss, animals show behavioral signs of tinnitus (Brozoski et al., 2002; Kaltenbach et al., 2004; Engineer et al., 2011; Longenecker and Galazyuk, 2011; Middleton et al., 2011), which are correlated to changes in the spontaneous activity of nerve cells in the auditory brain (Brozoski et al., 2002; Kaltenbach et al., 2004; Longenecker and Galazyuk, 2011). Increased spontaneous firing rates and increased synchrony of the activity of neurons in the central auditory system have been interpreted as neural correlates of tinnitus (Eggermont and Roberts, 2004; Roberts et al., 2010). Such aberrant patterns of spontaneous neuronal activity have been observed along the central auditory pathway after hearing loss, but not in the auditory nerve (Eggermont and Roberts, 2004; Roberts et al., 2010). The conclusion from animal models of tinnitus is that damage to structures of the ear and auditory nerve may trigger plastic changes in the brain, which then give rise to aberrant patterns of neuronal activity. As these altered patterns of spontaneous activity resemble sound-evoked activity, they could create the illusion of a sound in the absence of a sound source. The actual tinnitus sensation is therefore generated in the brain, but the corresponding changes in the brain are triggered through an event at the level of the ear.

## Theory of tinnitus development: tinnitus as a side effect of the brain attempting to compensate for hearing loss

One of the remaining open questions is how exactly hearing loss leads to tinnitus, i.e., which functional mechanisms in the brain underlie the development of the auditory phantom. This question has been addressed in theoretical studies using computer models, in order to investigate which of the known mechanisms of neuronal plasticity could account for tinnitus-related changes in the brain. The simulations show that hearing loss reduces the activity of the auditory nerve fibers and of neurons in the central auditory system. In the central auditory system, such a reduction of neuronal activity could activate a mechanism called homeostatic plasticity. This mechanism stabilizes the mean activity of neurons on long time scales, and in that way it sets the basic operating point of nerve cells, ensuring that they are neither inactive nor too active. When homeostatic plasticity tries to restore neuronal activity to its target level after hearing loss, it increases neuronal response gain, i.e., it makes the neurons respond stronger to input. A stronger response to the remaining input from the auditory nerve boosts activity levels in the auditory brain and can thus restore the overall activity to the pre-hearing-loss level. However, this compensation comes at a cost, as the overly excitable neurons then also start amplifying neuronal noise, like, for example, the spontaneous

activity of the auditory nerve. A certain level of spontaneous random neuronal activity is also always present in the healthy auditory system, but this neuronal activity is normally not perceived, but rather represents the neural code for silence. However, when neurons increase their gain to restore normal activity levels after hearing loss, the resulting amplification of meaningless spontaneous input activity can increase the level of spontaneous activity to such a degree that it starts resembling sound-evoked activity. When such a change happens in the first stages of auditory processing in the brain, the remaining stages might erroneously deduce that a sound is present, leading to the perception of a sound in the absence of a sound source. Simulations have shown that this mechanism can account for the development of neural correlates of tinnitus in the auditory brain stem (Schaette and Kempter, 2006, 2008, 2009) as well as the auditory cortex (Dominguez et al., 2006; Chrostowski et al., 2011). When applied to the audiograms of tinnitus patients with noise-induced hearing loss and tone-like tinnitus, our model predicts tinnitus frequencies that are in very good agreement with the patients' pitch-matching results (Schaette and Kempter, 2009). In summary, computer models of tinnitus development suggest that tinnitus could be a side effect of an attempt of the brain to compensate for hearing loss. The mechanisms of activity-dependent neuronal plasticity that are most likely involved in this process work in a bidirectional manner and are thus reversible, which means that tinnitus might be reduced when appropriate input to the auditory system is re-established.

### Tinnitus treatment through restoration of hearing: evidence from surgical treatment of conductive hearing loss

A direct prediction of the computer models of tinnitus is that tinnitus might be abolished by curing hearing loss. While it is currently not possible to cure noise-induced or age-related hearing loss, conductive hearing loss can often be substantially decreased through surgery. And indeed, in patients with otosclerosis, stapedectomy and stapedotomy have shown remarkable results for tinnitus. Two studies have reported that in more than half of the patients, tinnitus was completely abolished after surgery, and another third of the patients experienced improvement (Ayache et al., 2003; Sobrinho et al., 2004). The reduction of the air-bone gap through surgery was correlated to tinnitus reduction, i.e., a better hearing outcome after surgery led to a greater reduction in tinnitus. Similar results have been reported for tympanoplasty, where tinnitus was improved or eliminated in more than 80% of the cases after surgery (Lima Ada et al., 2007). These findings also correspond well to our results for simulation of conductive hearing loss through an earplug, where "curing" the hearing loss by removing the earplug completely abolished the earplug-induced phantom auditory sensations in all participants (Schaette et al., 2012). These findings show that tinnitus can be treated successfully by treating the underlying hearing loss.

### Tinnitus-related distress

So far, we have only been concerned with the tinnitus percept as such and how the phantom sound is generated. A different aspect is the impact tinnitus has on a patient, i.e., the patient's reaction to the tinnitus. Even though many patients feel distressed by the fact that they are hearing a phantom noise, the presence of tinnitus does not automatically lead to distress. In fact, the majority of people with tinnitus manage to habituate to their tinnitus and are not overly bothered by it (Dobie, 2004). An important factor for tinnitus distress is the emotional reaction to the tinnitus sound, for example, when the tinnitus sound is experienced as threatening or potentially harmful. Unfortunately, this process also changes the perception of the sound itself and can make it appear more salient or louder. Tinnitus distress is therefore created in a two-step process, the first step being the detection of the tinnitus sound, and the second step its evaluation and the subsequent reaction to it. The neurophysiological model by Jastreboff and coworkers (Jastreboff et al., 1996) details how a negative reaction to the tinnitus can give rise to a vicious cycle, where a negative reaction to the tinnitus leads to the attribution of importance to the tinnitus signal thus reinforces tinnitus perception. Moreover, negative automatic thoughts about the tinnitus and safety behaviors can impede habituation and contribute to tinnitus distress (Andersson and McKenna, 2006). Interestingly, the degree of tinnitus distress is only weakly, if at all, correlated to measureable aspects of the tinnitus, like loudness-matching results, minimum masking level, or pitch (Andersson, 2003). As current approaches to tinnitus treatment usually cannot treat the origin of the tinnitus, they often focus on changing the reaction to the sound to reduce the distress. Successful treatment therefore first of all turns a tinnitus sufferer into

someone who just has tinnitus. However, when the vicious cycle of tinnitus self-reinforcements is broken that way, a reduction of the tinnitus loudness can follow as well, and in some cases the tinnitus disappears altogether because the normal process of habituation takes over.

**Outcome measures for tinnitus treatment studies**

Before we start reviewing literature on tinnitus treatment, let us quickly cover how the outcome is usually assessed in tinnitus treatment studies. The most widely used tools for measuring the effect of a treatment on tinnitus are questionnaires, visual analog scales, and rating on a number scale, usually 0–10. There are a variety of validated questionnaires for tinnitus, commonly used ones are the Tinnitus Handicap Inventory (THI) (Newman et al., 1996), the Tinnitus Questionnaire (TQ) (Hallam et al., 1988; Goebel and Hiller, 1994; Hiller and Goebel, 2004), the Tinnitus Handicap Questionnaire (THQ) (Kuk et al., 1990), the Tinnitus Reaction Questionnaire (TRQ) (Wilson et al., 1991) and the Tinnitus Functional Index (TFI) (Meikle et al., 2012). All these questionnaires allow the calculation of an overall score that reflects the degree of tinnitus distress. Visual analog scales are often employed to obtain a subjective rating from a patient, for example, for questions like "How loud is your tinnitus?". The response is given by the patient by marking a point on a 10 cm long line with two opposing statements or properties as anchor points at each end of the line, for example, "the softest sound you can imagine" and "the loudest sound you can imagine" for tinnitus loudness. The rating can then be converted to a score by measuring the position of the mark on the line. Similar to visual analog scales, ratings on a 0–10 scale are often employed for patient self-assessment of tinnitus annoyance, loudness, awareness, and other aspects.

**Hearing aids in tinnitus treatment**

Based on the theory of tinnitus as a side effect of an attempt of the brain to compensate for hearing loss, and the astonishing reductions of tinnitus that can be achieved through surgical treatment of conductive hearing loss, one might think that the "perfect hearing aid" that fully compensates for the effects of hearing loss and thus renormalizes auditory nerve activity could be the "silver bullet" of tinnitus therapy. However, it has to be noted firstly that in conductive hearing loss the sensory cells of the inner ear are usually intact, and therefore an almost complete restoration of hearing through surgery is possible in many cases. Secondly, in noise-induced or age-related hearing loss, individual patients present with varying degrees of damage to or even loss of cochlear hair cells and auditory nerve fibers, and it can be difficult or even impossible to compensate for the effects of severe damage through amplification provided by a hearing aid. Thirdly, as outlined above, psychological factors can also have a strong influence on tinnitus and the associated distress. Therefore, treatment effects of hearing aids against tinnitus can be expected to vary from patient to patient, and hearing aids are usually part of a comprehensive treatment approach that also addresses the psychological aspects of tinnitus, depending on the patient's needs.

In the following, we will have a look at studies that investigated the effects of hearing aids on tinnitus. Some of these studies have investigated noise generators as well. Please note that none of the studies has investigated hearing aids or noise generators as the sole treatment, they were usually employed in conjunction with some degree of counseling, tinnitus retraining therapy, or cognitive behavioral therapy, or combinations of elements of these approaches.

Surr and colleagues (Surr et al., 1999) conducted a study in 34 novice hearing aid users who complained of hearing loss and tinnitus. The study participants were either active military servicemen, or retirees. Hearing aids were fitted based on the audiometric test results, not as tinnitus maskers. The tinnitus handicap inventory was used as main outcome measure, and it was administered before and 6 weeks after hearing aid fitting. Overall, the group showed a significant reduction in tinnitus distress scores, however, only 6/34 participants showed a reduction of more than 20%, which was considered a significant individual reduction.

The effect of ear-level devices on tinnitus was studied by Folmer and Carroll (2006) in 150 patients. All patients underwent a comprehensive tinnitus management program including counseling, and follow-up questionnaires (tinnitus severity index and self-rating of tinnitus loudness on a 1–10 scale) were mailed to the patients 6 to 48 months after their initial appointment. 50 patients were fitted with a hearing

aid, 50 received a noise generator, and 50 used no device. At follow-up, all three groups showed a significant reduction of TSI scores and tinnitus loudness ratings. In the hearing aid group, the TSI score decreased from 38.2 ± 8.3 to 29.6 ± 8.4, and the self-rated tinnitus loudness from 7.5 ± 1.7 to 6.3 ± 1.9. 70% of the participants reported that the hearing aid helped their tinnitus a moderate amount or more. In the noise generator group, a similar improvement of tinnitus was seen, with the TSI score decreasing from 39.6 ± 8.9 to 32.8 ± 8.9, and the self-rated tinnitus loudness from 7.6 ± 1.6 to 6.2 ± 1.9. Interestingly, the no-device group showed a significant improvement as well, but the effect was considerably smaller than in the hearing aid or noise-device group, with a TSI score reduction from 38.1 ± 9.0 to 33.8 ± 8.9 and a decrease in loudness rating from 7.1 ± 1.9 to 6.5 ± 1.8. Overall, the hearing aid and the noise generator group showed large treatment effect sizes of 1.0 and 0.8, respectively, whereas in the no-device group, the effect size was only medium (0.5). The authors concluded that "Ear-level devices such as hearing aids or sound generators can help a significant number of patients who experience chronic tinnitus. Both types of devices reduce patients' perception of tinnitus and can facilitate habituation to the symptom. Amplification provides additional benefits of improved hearing and communication."

Trotter and Donaldson reported on the results of fitting tinnitus patients with hearing aids in their clinical practice for 25 years, from 1980 till 2004 (Trotter and Donaldson, 2008). Subjective tinnitus assessment using visual analog scales was performed before and after fitting. All patients received counseling for their tinnitus, which played an integral role in the management of the patients. A very striking feature of the study is that patients showed a significantly greater improvement of their tinnitus after the introduction of a digital hearing aid program in 2000, compared to the patients receiving analogue hearing aids in the years before. In the digital hearing aid group (years 2000–2004), 80% of the bilaterally aided patients showed a tinnitus reduction of more than 50%, as assessed through the visual analog scale, compared to only 30% of the patients receiving bilateral analogue hearing aids. The authors' conclusions were that "Provision of hearing aids in those with demonstrable audiometric loss can play a very important part in tinnitus control. The introduction of programmable digital aids had a summative effect on this improvement in tinnitus control."

In 2010, Searchfield and colleagues reported on the effects of hearing aids as an adjunct to counseling (Searchfield et al., 2010). This retrospective study comprised 58 patients with hearing loss, all of whom received 1–2 hours of counseling for their tinnitus. Amplification was recommended to all patients, but only 29 chose to wear hearing aids afterwards. Both groups had similar audiometric configurations. The tinnitus handicap questionnaire was administered before and on average one year after the intervention. The THQ scores decreased in both groups, from 59.2 to 37.4 in the hearing aid and from 50.8 to 43.6 in the counselling-only group, but the reduction was only significant in the hearing aid group. A subscale analysis of the THQ results revealed that the hearing aids had a therapeutic effect in addition to their benefit for hearing. The authors stated that "It is concluded that patients with hearing loss and tinnitus should trial amplification."

Another study that investigated the combined effects of hearing aids and counseling was done by Forti and colleagues (Forti et al., 2010). 100 tinnitus patients received counseling and were fitted with open-canal hearing aids. The tinnitus handicap inventory (THI) was administered at the beginning of the study and after nine months of treatment. After nine months, there was a highly significant decrease in THI scores from 54.22 ± 20.37 to 28.32 ± 16.50.

Hearing aids and noise generators are also employed for sound therapy in the context of tinnitus retraining therapy (TRT) (Jastreboff et al., 1996). Parazzini and coworkers examined whether the treatment outcome of TRT would depend on the choice of device (Parazzini et al., 2011). 101 patients were included in the study; all of them had symmetrical hearing loss of less than 25 dB at 2 kHz and hearing loss of more than 25 dB at frequencies higher than 2 kHz. Half of them were fitted with bilateral sound generators, the other half with bilateral open-ear hearing aids. Treatment effect was assessed using the THI and self-ratings for tinnitus loudness, awareness and annoyance. These measures were administered before treatment and after 3, 6, 9, and 12 months of therapy. There was a highly significant improvement of both the THI and the rating scores

starting from the first three months and up to one year of therapy. Moreover, also the decreases between each of the time points were significant. TRT was found to be equally effective with hearing aids and noise generators, there were no significant differences in the results between the two treatment groups. In both groups, THI scores were reduced by approximately 50%.

A recent study investigated which factors could influence the effect of tinnitus treatment with hearing aids in a retrospective analysis of 70 tinnitus patients that all received hearing aids (McNeill et al., 2012). All patients received counseling for their tinnitus. In the whole group, the TRQ score reduced from 49 to 34 on average after three months of treatment. Further analysis revealed that treatment outcome differed markedly depending on whether the patients experienced complete, partial, or no masking of their tinnitus when the hearing aid was switched on. The total-masking group had the biggest reduction of tinnitus, with TRQ scores going down from 51.9 to 17.2, in the partial-masking group scores decreased from 53.1 to 34.5, whereas in the no-masking group the TRQ scores were virtually unchanged (38.6 before and 34.5 after three months of treatment). All patients who experienced total masking achieved a clinically significant reduction of the TRQ score. These results are especially interesting since all patients received counseling and hearing aids. The fact that a more responsive subgroup could be identified based on tinnitus masking through the hearing aid indicates that there might have been an interaction of the acoustic stimulation and the tinnitus-related neuronal activity, possibly triggering plasticity. The authors conclude that "The results support the use of hearing aids for tinnitus management, and suggest that masking may be a significant contributor to hearing aid success." Interestingly, the fact that patients experiencing total masking of their tinnitus through the hearing aid showed a greater reduction of tinnitus than those who experienced partial masking is at odds with the recommendation from TRT that advocate setting sound devices to the mixing point where tinnitus is still audible (Jastreboff et al., 1996). However, a recent study found that for TRT with noise generators, mixing point and total masking are equally effective (Tyler et al., 2012).

An interesting trend that can be noticed in the studies on hearing aids and tinnitus is that the amount of tinnitus reduction seems to increase over time, i.e., more recent studies report greater benefit. The oldest study included in this review reported the least benefit (Surr et al., 1999), whereas a more recent study using the same outcome measure reported a reduction of scores of almost 50% (Forti et al., 2010). On the one hand, the initial THI score was higher in the study by Forti and colleagues, so there might have been more room for improvement. On the other hand, the two studies are separated by more than a decade, and hearing aid technology has progressed significantly over that time, so better hearing aids might lead to a greater tinnitus reduction. This effect has also been observed in the study by Trotter and Donaldson (2008), where the introduction of digital hearing aids in 2000 dramatically increased treatment success. These results also demonstrate that hearing aid technology might need to be taken into account when comparing the results of different studies. Our theory of tinnitus development offers a putative explanation for the difference in treatment effects between analog and digital hearing aids: digital hearing aids with their greater flexibility for fitting are better suited for restoring a "normal" input to the auditory system, and according to the theory they can therefore be expected to lead to a greater reduction of tinnitus.

The general conclusions from the studies reviewed above are that hearing aids can have a considerable effect on tinnitus, and that they form an important part of tinnitus treatment. As already mentioned above, none of the studies provided hearing aids as the only treatment, which raises the question about the "active ingredient" of the treatment. So far, two studies have compared a group treated with hearing aids and counseling against a group receiving counseling without any device, and both have reported that hearing aids provided additional benefit over counseling alone (Folmer and Carroll, 2006; Searchfield et al., 2010), suggesting that hearing aids actively contribute to tinnitus reduction. On the other hand, a study on noise generators found that patients who were treated with a combination of noise generators and cognitive behavioral therapy did not show greater improvement than those receiving cognitive behavioral therapy alone (Hiller and Haerkotter, 2005). It should be noted, though, that these studies employed different treatment approaches, the

participants in the studies by Searchfield et al. and Folmer and Carroll only received a relatively limited amount of counseling, whereas the study by Hiller and Haerkotter employed 4–10 sessions of cognitive behavioural therapy, depending on the distress level, suggesting that the absence of additional benefit through the noise generators in the latter study might at least in part be due to a ceiling effect. Nevertheless, the relative contribution of the different treatment components, e.g., hearing aids, noise generators, TRT, counseling, and cognitive behavioral therapy will need to be clarified in future studies.

In most studies, hearing aids were fitted according to the audiogram, to date there are no evidence-based recommendations for how to fit hearing aids for maximum tinnitus relief. One study used hearing aid settings that increased the amplification of low-level sounds, in order to also make ambient background noise audible and thus provide background stimulation (Searchfield et al., 2010). The recommendation to use open fittings whenever possible is based on a similar rationale, as open fittings are more comfortable and do not block out external noise, which is argued to help reducing tinnitus awareness (Del Bo and Ambrosetti, 2007; Parazzini et al., 2011). However, the ideal fitting strategy will first and foremost depend on the kind and severity of hearing loss of an individual patient.

Despite the fact that most patients experience a great degree of tinnitus relief through treatment with hearing aids, around 10% of patients show no improvement of their tinnitus after hearing aid fitting (Trotter and Donaldson, 2008). This seems to be at odds with the theory that tinnitus could be a side effect of the brain's attempt to compensate for hearing loss, which suggests (at least in a naïve interpretation) that all tinnitus patients with aidable hearing loss should benefit from hearing aids. However, individual differences in treatment effect could, for example, be down to differences in cochlear damage, e.g., loss of outer hair cells versus inner hair cell loss. Loss of outer hair cells decreases hearing sensitivity, but does not affect the number of neural elements that can respond to sound stimuli, and can therefore be compensated by amplification to a great degree. Loss of inner hair cells, on the other hand, permanently and irreversibly reduces the signal in the auditory nerve, simply because inner hair cell loss means that the affected auditory nerve fibers lose their input, which cannot be overcome by amplification. However, patients with inner and outer hair cell loss can have quite similar audiograms, and it can therefore be difficult to predict treatment success with hearing aids unless more detailed investigations of the underlying cochlear damage are performed. Another factor that could influence the effects of hearing aids on tinnitus is the frequency range of the devices. Behind-the-ear devices usually have an upper frequency limit somewhere around 6–8 kHz, but tinnitus pitch is frequently matched to even higher frequencies. In that case, neurons in the tinnitus frequency range will receive less effective stimulation through the hearing aid than when the tinnitus pitch is within the frequency range of the device. The relation between the tinnitus pitch and the frequency range of the hearing aid could thus determine how effectively the hearing aid can counteract the pathological neuronal activity that generates the tinnitus. Studies on the effects of hearing aids on tinnitus might therefore comprise two subgroups, one with tinnitus pitch inside the frequency range of the hearing aid, and the other with high-pitched tinnitus that can hardly, if at all, be reached by the hearing aid. Our computer model of tinnitus (Schaette and Kempter, 2006, 2009) predicts that the "low-pitch" group will show a greater treatment response to amplification in that case. The "true effect" in the more responsive subgroup might thus be underestimated when the study outcome is reported as the average over all participants. This caveat has been investigated in two recent studies (Schaette et al., 2010; McNeill et al., 2012). In both studies, tinnitus reduction was larger in the group where the tinnitus pitch was within the frequency range of the device. Moreover, in the "low pitch" tinnitus group, more than half of the participants experienced total masking of their tinnitus by the hearing aid alone, compared to only about 15% in the "high pitch" group (McNeill et al., 2012). Also, the greatest reduction in TRQ scores was obtained in the patients that experienced total masking through the hearing aid, all of whom had a clinically significant TRQ score reduction of more than 40%, whereas those that experienced no masking had unchanged TRQ scores on average (McNeill et al., 2012).

## Summary and conclusions

Current research suggests a direct causal link between hearing loss and tinnitus, in that hearing loss can trigger plastic changes in the brain that produce aberrant patterns of spontaneous neuronal activity, which is then perceived as tinnitus. These changes can be reversible, in fact more than half of the patients with conductive hearing loss experience a complete tinnitus remission when their hearing loss is reduced or abolished through surgery. Similarly, hearing aids can reduce tinnitus loudness and distress to a great degree in many patients. Several recent studies suggest that hearing aids themselves constitute an active component of tinnitus treatment beyond their obvious benefits of improving audibility and communication (Searchfield et al., 2010; McNeill et al., 2012). Hearing aids can therefore be an important part of tinnitus treatment, ideally in combination with treatment elements that address the psychological aspects of tinnitus distress, like, for example, counseling or cognitive behavioral therapy, in a stepped-care approach tailored to the patient's needs (Cima et al., 2012). In conclusion, for tinnitus patients with aidable hearing loss, hearing aids can help reduce tinnitus awareness, annoyance, and distress, as well as improve hearing and communication.

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# Bibliography

**Dr Roland Schaette**

A spider's web is hidden in one ear, and in the other, a cricket sings throughout the night." This is how Michelangelo described his experience of hearing loss and tinnitus**.** The goal of Roland Schaette's research is to find the cricket, to understand how tinnitus arises and how it manifests itself in the brain, in order to find new ways of treating it. A biophysicist by training, Dr Schaette started doing research on tinnitus at Humboldt University Berlin in 2003. In November 2008, he took on the position of British Tinnitus Association Senior Research Associate at the UCL Ear Institute in London, where he is now leading the tinnitus research program. In his research, Dr Schaette uses a multidisciplinary approach that combines studies with human subjects, animal models and computer modeling to investigate the different aspects of tinnitus and derive a comprehensive understanding of the phenomenon.