

Canadian Hearing Report

Revue canadienne d'audition

VOL. 10 NO. 3 2015

TINNITUS!

Pink noise White noise Brown noise
Blue noise Black noise Red noise
Gold noise Green noise Orange noise
White noise Yellow noise Gold noise
Gold noise Blue noise Grey noise
Yellow noise Orange noise Gold noise
Brown noise White noise Yellow noise
Pink noise Grey noise Brown noise
Blue noise White noise Red noise
Gold noise Green noise Orange noise

TINNITUS QUESTIONS

NOTCHED SOUND THERAPY AS A
TREATMENT FOR TINNITUS:
A PRIMER FOR HEARING HEALTH
PROFESSIONALS

CRITICAL REVIEW: EFFICACY OF NOTCHED-SOUND THERAPY FOR NEURAL PLASTICITY MEDIATED TINNITUS TREATMENT

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Hello, hello! Change comes with the seasons, and also with each issue of the *Canadian Hearing Report*. Each has got to be unique. 'Tis the tinnitus issue here. As we all know, tinnitus is highly prevalent in our clinical populations. Tinnitus to us is kind of like allergies are to physicians; we encounter it often, but all too often, many of us don't have a solid grip on how to handle it. We hem and we haw, and use the old saw about background noise, and hope it just goes away.

Tinnitus is thought to arise from an imbalance of afferent (cochlea hair cells – to – brain) input and efferent (brain – back to – cochlea) information in the auditory system. A loss of input from the cochlea to the auditory cortex can give rise to the brain increasing its gain for these expected frequencies and as a result, one experiences tinnitus. Obviously the causes are rather complex and not simply dealt with by medications, etc. As clinicians we all know though, that there are two parts to tinnitus; one is the client's tinnitus itself, and the other is the client's reaction to the tinnitus. Treatments tend to involve two things as well; noise and also, counselling. The main idea is to get the client to realize he/she has some *control over things*. For example, the tinnitus is usually more noticeable at some times compared to other times. This in and of itself is a good thing to consider, because then, maybe the surrounding sound environments can be manipulated so as to mitigate or reduce the tinnitus.

Pulsatile or throbbing tinnitus might be objectively audible, as its cause is likely to be vascular. The vast majority of tinnitus however (and the truly odd thing about

it) is that it is *subjective*, and heard only by the client. It is then a lonely path, accompanied by fears of what might be causing it. This only exacerbates things, which adds to the negative experience of tinnitus. Our first clinical duty here is to rule out these causes, such as VIII nerve tumours, by referring to medical and audiological investigation. Once this has been done, we can help the client find the Holy Grail; that place when the tinnitus can be thought of as simply a nuisance, something that can be lived with, and in short - controlled.

We've got three articles on tinnitus. One is written by a physician (Peter Phua) who describes his approach to tinnitus called, "Notched Sound Therapy." This involves first identifying the specific frequency or narrow band of frequencies of the tinnitus; this specific sound is then notched or removed from either white noise or the client's favourite music. The client then listens to this altered stimulus for several hours a day. The idea is that neurons adjacent to those causing the tinnitus will be stimulated, and that they will thus "laterally inhibit" the neurons that are causing the tinnitus. The hoped for result is that some neuroplasticity will occur; some cortical rewiring will take place, and thus reduce the tinnitus. A big attraction here is the low cost of the approach, as well as the possibility of the client working alone or with his/her clinician.

Every good idea of course, should be tested and examined to see if it is has good clinical "sand." Mark Bennett is an audiologist who follows up with a critical review of the Notched Sound Therapy approach. Here, he examines the efficacy of the approach as a clinical treatment for tinnitus. He compares the results found from no less than five completely separate studies or clinical trials of the Notched approach. All in all, he finds moderate evidence to show that the method works. It's an interesting

follow-up read, and we're happy Mark agreed to submit his article to us.

Our tinnitus triangle is completed by an interview with Robbyn Brodie, an audiologist who specializes in the popular Tinnitus Retraining Therapy (TRT) from Pawel Jastreboff. She describes her own clinical encounters with clients who experience tinnitus. It is heartening to hear her encouragement for others to join her in this endeavour. Moreover, she also endorses the clinical treatment of tinnitus by Hearing Instrument Practitioners (HIPs) too. Did we all know that Richard Tyler PhD is developing a tinnitus treatment protocol with the International Hearing Society (the main HIP organization in the USA)?

For something completely different, have a look at my own missive at the end of this issue. We've been hearing a lot lately about "Hearing loss and dementia." Far too many in the profession however, are being misled (either willingly or blindly) to believe that there is a *cause and effect* thing here. That however, is just not so! A correlation maybe, but cause & effect between hearing loss and dementia has yet to be proved. The mistaken oversight here is one thing, but worse yet would be this oversight being negatively used as a reason to increase sales of hearing aids. As clinicians we all need to earn a living. At the same time however, we also bear in mind that our role is to serve the public. I'll say no more.

OK, here's to you, and have a great summer! It's been a scorcher already here in Victoria BC.

Ted Venema, Editor-in-Chief

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Tinnitus Questions With Robbyn Brodie

Robbyn Brodie, MSc, Registered Audiologist, Tinnitus Training Therapist



About the Author

Robbyn obtained her undergrad degree at UBC and her Master's Degree in Audiology from Dalhousie University in Halifax. She worked at the Children's Hospital of Eastern Ontario (CHEO) and the Ottawa Civic Hospital for five years before moving back to the West Coast and settling in Victoria with her husband and two children. She now works in private practice at NexGen Hearing Clinic (Royal Oak) and has been practicing Tinnitus Retraining Therapy since 2011.

Why or how did you get into audiology?

Growing up, both of my parents had significant hearing loss – my mom is deaf in one ear and my father has severe noise-induced loss – and I watched my grandfather withdraw into isolation as he stubbornly refused to wear his hearing aids. Then, while I was researching different career path options, I was volunteering in a speech-pathology clinic in Vancouver and within this clinic I discovered a tiny audiology department. I worked there for a bit and found it was a perfect fit.

You're originally from White Rock BC but went to Dalhousie to study audiology, right? How come?

There are so few options for places to study audiology (in English) within Canada – UBC, Western, and Dal! I did my undergrad at UBC and wanted a change for my master's degree. Dalhousie had a great program and the East Coast seemed to call to me more than Ontario (no offence Ted!)...little did I know that I would head to Ottawa for five years, right after finishing up at Dal. Both my kids were born in Ontario!

At NexGen Hearing, you are one of the consultants with a specialty in tinnitus; when did you become interested in tinnitus?

As a student, I did a placement with Mark Gulliver at Nova Scotia Hearing and Speech Centre – he led group tinnitus sessions. I learned a lot there. In Victoria, there were few services for those who struggled with their tinnitus. I was seeing many patients whose primary complaint was tinnitus, not hearing loss. I could give them a little information, but felt that I would like to offer more.

As you know, there are lots of different and varying approaches to treating tinnitus; what is yours?

I use Tinnitus Retraining Therapy

(TRT) principles to treat tinnitus. In 2011 I travelled to Maryland to study TRT under Dr. Pawel Jastreboff. TRT is based on the neurophysiological model of tinnitus developed in the late 1980s by Drs. Jastreboff and Jonathan Hazell.

This model of tinnitus suggests that it is the limbic system – the parts of the brain responsible for emotions – that assigns importance to tinnitus sounds (Figure 1). According to this model, if we perceive tinnitus sounds to be a threat or a danger (Figure 2), this provokes an emotional response, which in turn can provoke a stress response from the autonomic nervous system. Our awareness of tinnitus is heightened and so we perceive it to be louder or more persistent. This becomes a vicious cycle.

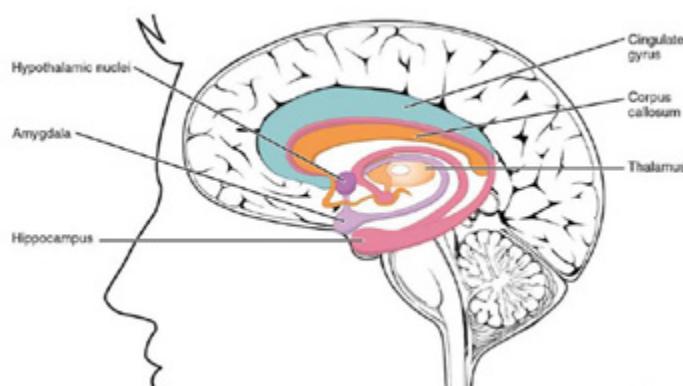


Figure 1. Showing parts of the limbic system from below (from Wikipedia)



Figure 2. The reaction to the tinnitus creates distress! Not the intensity of the tinnitus itself.

TRT combines counselling and low-level sound therapy. When we demystify tinnitus while simultaneously reducing awareness of the tinnitus, over time one's awareness of the tinnitus is reduced, and should only be noticeable when focused upon. This is known as 'habituation' and is the ultimate goal of TRT.

What do you think is the most common cause of tinnitus?

Well, noise-induced hearing loss is a big one, but I find that STRESS is definitely a common denominator in many of those with tinnitus. When someone is in the throes of great stress or anxiety, it is very difficult for them to "tune out" the tinnitus. Stress in turn can cause sleep issues, and when stressed and tired, coping mechanisms often go out the window.

Is there any kind of audiogram you often associate with tinnitus?

Typically a high-frequency loss, such as the typical noise-induced hearing loss pattern (Figure 3) though any shape of loss, conductive, and even perfectly normal hearing, is common too.

So how do you get going in treating clients with tinnitus? What steps do you go through?

First, I start by having them fill out some questionnaires which inquire about how their tinnitus sounds to them, which ear, how bothersome it is, their medical history, stress levels and sleep patterns, and also whether they have hearing loss. I then do a hearing assessment and I measure the tinnitus. Here, I get them to subjectively match the pitch and volume of their tinnitus. I also try to find the minimum masking level, and if there is any residual inhibition (Figure 4). Using all the gathered information, I decide which TRT category they fall into, which determines the appropriate treatment for them.

For some clients, basic information and education about their hearing (whether they have normal hearing or hearing loss)

and tinnitus, and some brainstorming of ideas on use of environmental sound therapy to manage their tinnitus works wonders. Questions I hear often are "Will my tinnitus keep getting louder and louder?" "Will I go deaf?" "Do I have a tumour?" Many are relieved to find out that tinnitus is a fairly common issue and doesn't necessarily mean they have a tumour or disease (of course, I recommend further assessment where required), and this knowledge can be a help in learning to habituate to the tinnitus.

For those with hearing loss and mildly bothersome tinnitus, *often fitting them with hearing aids* (and always the counselling and educational component) brings in enough environmental sound that sound generators may not be required. However, hearing loss accompanied by a moderate or severe degree of tinnitus may require a combination device of hearing aid and sound generator in one.

There are two major components of TRT: educational counselling and sound therapy. The protocol is tailored to the client, as far as number of treatments, direction of counselling,

Noise Induced Hearing Loss (NIHL)

- 2nd most common HL & also most preventable HL
- Most common HL associated with **Tinnitus**

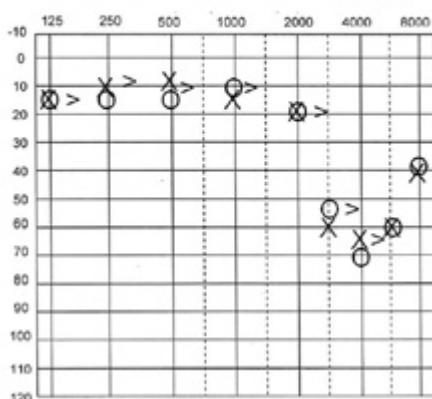


Figure 3.

**Note that the Tinnitus masker is only partial
The objective is to take the edge off the Tinnitus,
not to completely drown it out**

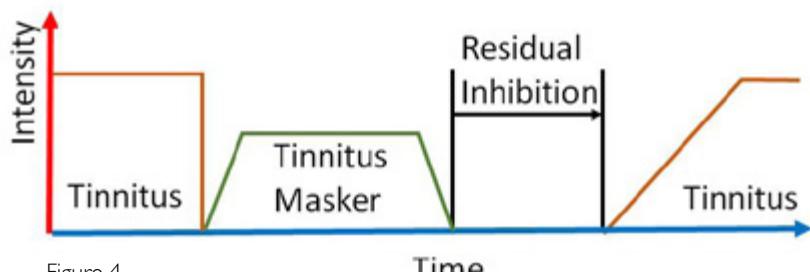


Figure 4.

and type of sound, ear-level device or environmental sound etc., but always involves counselling and sound therapy.

What do you think is the most important thing a client has to do when going for treatment of tinnitus? They need to be willing to believe that their situation can improve. If they believe they have an unknown underlying disease or tumour which is causing the tinnitus, no degree of counselling or sound therapy will get them to habituation. This is why it is important that, when they start TRT, they have seen their doctors, had various tests done (i.e., MRI), have been assured that the tinnitus is not a symptom of something more sinister. The educational component of TRT is about demystifying tinnitus, and if they continue to believe the worst, the treatment will be unsuccessful.

A clinician not specialized in tinnitus has a client with tinnitus; what should they do?

I think that most audiologists and HIPs, with a bit of effort, can be well-equipped to deal with tinnitus. Education and patience are important in equal amounts!

I ask because Richard Tyler is now involved with International Hearing Society (IHS) in developing a tinnitus training program. The more hearing specialists equipped to work with tinnitus patients, the better! I'm all for

it, as long as the professional is educated in treating tinnitus.

About what percentage of your clients complain of tinnitus?

I would speculate that approximately 25% of my clients complain of tinnitus. Tinnitus affects about 15% of the general population, though 75% of this group are not bothered by their tinnitus.

Do most of your clients with tinnitus have it in one or both ears?

Such variation! One ear, both ears, one louder than the other, some hear it in the middle of their head, while others hear it coming from out in front of them somewhere.

I recently attended a talk by Richard Tyler at the Western Canadian Symposium here in Victoria; he said that of clients who have had the VIII nerve severed to get rid of the Tinnitus, 30% still experience it! What do you make of that?

It's fascinating, isn't it? For the lucky patients who found this surgery to be successful, their tinnitus must have been originating in the damaged ear – sending abnormal patterns of activity to the brain. For the remainder of the patients whose tinnitus was not relieved, the tinnitus must be stemming from somewhere else in the brain or body. I have certainly never recommended this option to any of my clients! There is still so much about tinnitus that isn't understood.

Ever experience tinnitus for a brief moment and then it goes away? What do you think that is?

Most of us experience brief episodes of "hearing loss" accompanied by loud high-frequency tinnitus which fades away in less than a minute. This is a normal event, caused by muscle spasms of the tensor tympani, contracting and tightening the eardrum. This is simply a muscle twitch! Don't worry, these episodes are not a sign of auditory damage. They may be associated with caffeine intake or head or neck movement, but they are just as likely to be randomly occurring.

What do you recommend as an inexpensive home-made treatment to ease the negative effects of tinnitus?

Stress-reducing activities. Avoid silence – use a fan, music, TV, one of many free tinnitus apps on a smartphone or iPad, bedside sound generator – 24 hours a day. Understand that the less you react to the tinnitus, the less prominent it will become.

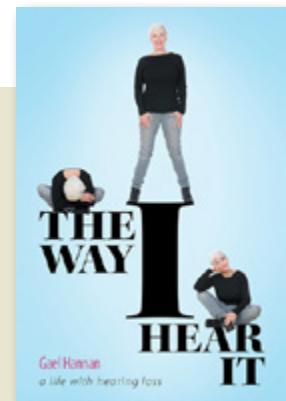
Thanks Robbyn for describing your clinical encounters with tinnitus! I think our readers in the clinical trenches will appreciate your common-sense approach. All in the spirit of learning from each other; Cheers!

New Book – Order Now!

In *The Way I Hear It*, Gael Hannan explodes one myth after another in a witty and insightful journey into the life with hearing loss – at every age. Part memoir, part survival guide, *The Way I Hear It* is an insider account of the frustrations of communicating with hearing loss: pillow talk and other relationships, raising a child, in the classroom and on the job, hearing technology and the everyday things we like to do.

Gael offers advice on how to bridge the gap between consumer and professional in order to get the best possible hearing health care, as well as tips for effective communication, poetic reflections and humorous, poignant stories from the people she has met in her advocacy work throughout North America. This is a book for people with hearing loss—but also for their families, friends and the professionals who serve them.

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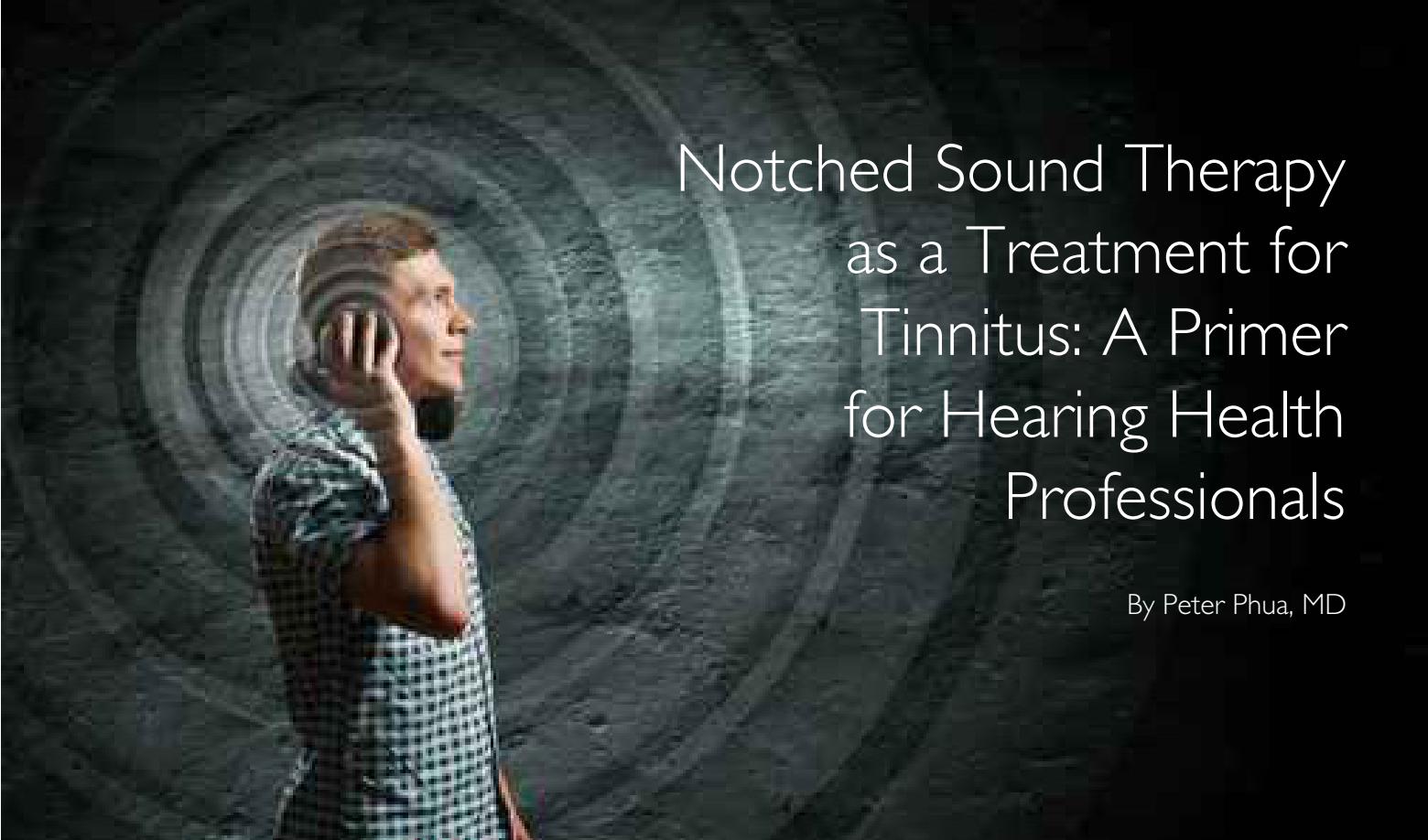
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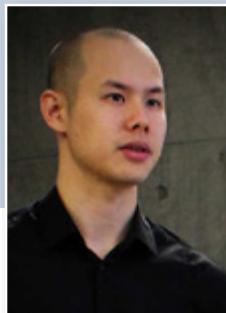
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Notched Sound Therapy as a Treatment for Tinnitus: A Primer for Hearing Health Professionals

By Peter Phua, MD



About the Author

Peter Phua, MD, www.audionotch.com/about is a graduate of McMaster University's medical program, and C.E.O. of AudioNotch www.audionotch.com/ and Tinnitus Treater, www.tinnitustreater.com/, on-line providers of Tailor-Made Notched Sound Therapy for patients with tinnitus.

Tinnitus is a common hearing-related problem that leads patients to seek help from hearing health professionals. It's an extremely prevalent problem, affecting millions of people worldwide.¹ However, only a small fraction of tinnitus sufferers become distressed enough to seek treatment for this problem. Evidence suggests that most people are able to habituate to the tinnitus tone, which ceases to become bothersome to the patient. Those who don't, however, can be severely negatively affected, and may have underlying vulnerabilities that predispose them to become psychologically distressed by tinnitus.

The majority of individuals with tinnitus

have acquired it from hearing loss, although there is a long list of possible causes. The initial evaluation of tinnitus should always determine whether it is a symptom of serious disease requiring medical intervention (for example, an acoustic neuroma growing in the VIII cranial nerve or low brain stem). However, the most common variant of tinnitus is "sensorineural" tinnitus – the consequence of a *neuroplastic* response in the auditory system to a loss of input from the cochlea to the auditory cortex. It's this subtype that the vast majority of audiologists deal with.

I first experienced tinnitus as a medical student, and in a desperate search for answers, I had a difficult time making sense of it all. It was doubly difficult because, as a person in acute

distress, you want to believe all the positive results you read. I eventually stumbled upon Notched Sound Therapy through a professor at my university, which provided me with some acute relief. Fortunately, over the next couple of months I habituated to my tinnitus and didn't really need any more ongoing therapy, but I realized that there was a gap in the market and decided to start a small business to provide this service to others.

"Notched Sound Therapy" is a term derived from how the therapy is made – you take unprocessed audio input such as music or white noise, and "notch out" sound energy at and around the tinnitus frequency of the user. Notched Sound Therapy is audio sound input that has been processed with a "notch" centred

NOTCHED SOUND THERAPY AS A TREATMENT FOR TINNITUS: A PRIMER FOR HEARING HEALTH PROFESSIONALS

at the frequency of a patient's tinnitus.² In this context, a "notch" is made in the audio by a computer algorithm that removes the sound energy at and around the patient's tinnitus frequency. Different researchers have used different notch widths, ranging from one octave to one equivalent rectangular bandwidth. The effects of Notched White Noise has been studied in a randomized control trial (where subjects were randomly assigned to placebo and treatment groups). The effects of Notched Music has been studied in a group of small pseudo-randomized control trials (where the subjects were not randomly assigned to placebo and treatment groups). Both studies have demonstrated that listening to Notched Sound Therapy can directly lower the volume of tinnitus, and in doing so, reduce the psychological annoyance caused by the tinnitus tone.²⁻⁴ The experiments ran with treatment cycles of twelve month's duration, however, significant reductions in tinnitus volume can be seen as early as one week into therapy (provided that it is intense in duration).⁴

The Notched Sound Therapy approach is limited to people who have tinnitus tones that can be successfully localized with either our on-line tuner or with the aid of an audiologist. The mechanism of action isn't understood, but it's thought to occur through strengthening lateral inhibition networks between healthy auditory neurons and the aberrant neurons that spontaneously fire, causing the perception of tinnitus. Thus, a combination of lateral inhibition and subsequent changes in the auditory cortex via cortical plasticity appear to be implicated.² Selectively stimulating the auditory neurons that don't produce the tinnitus frequency appears to laterally inhibit the auditory neurons

that do produce the tinnitus frequency.² Sustained lateral inhibition rewires the connections between neurons in the auditory cortex so that the baseline level of inhibition increases (even when users aren't listening to the music).² Therefore the tinnitus volume decreases.

One of the advantages of Notched Sound Therapy is that the approach has been successfully replicated by two groups of researchers. Replication is one of the most important concepts in experimental science. The basic idea is that if an experiment discovers a treatment to be truly effective, then this finding should be able to be generalized (replicated) elsewhere in the world.

The original research on Notched Sound therapy came out of Germany and the research group of Dr. Christo Pantev. Subsequently, additional research from an analogous (but not identical) form of sound therapy came out of Italy. Whereas Dr. Pantev placed an auditory notch in music and provided this as therapy, the Italian researchers placed a "window" (which, functionally, was a notch) into broadband noise (which sounds like static). <http://www.tinnitusjournal.com/imagebank/pdf/v15n1a05.pdf>. Both experimental groups found a positive treatment effect, sometimes large, in their participants. This example of cross verification is why I believe that, for some people, Notched Sound Therapy does indeed work.

Again, the claims of efficacy around Notched Sound Therapy should be modest. It works for some people, some of the time, and seems to provide a moderate effect in the people that it does help. We have published our internal results here on our blog (choppy and low quality though the

data may be, it's better than nothing). <http://www.audionotch.com/blog/tinnitus/more-on-tinnitus-pitch-matching-a-study-of-interest/>

Notched Sound Therapy represents a new approach to sound therapy for tinnitus. It has two virtues: (1) it has been independently researched with positive therapeutic results, by two different European research groups (only one of which has attempted to commercialize their therapy afterwards), and (2) it is available at low cost to patients via third party proprietors.

HOW IT WORKS

Notched Sound Therapy takes place in three steps.³ In step one, patients determine the frequency of their tinnitus. This can be done in one of two ways: either with the use of a web-based tinnitus tuner, <http://www.audionotch.com/app/tune/> or alternatively, inside of an audiology clinic. In step two, patients choose the audio they wish to have "notched," which includes options ranging from music to white noise.^{2,3} The sound is then "notched" by a special software algorithm. In step three, patients listen to their Tailor-Made Notched Sound Therapy.² This can be done with any device capable of playing MP3 music files. *The lack of a proprietary device is what allows the cost of Notched Sound Therapy to be dramatically lower than its competitors.* After months of listening for several hours per day, a patient's tinnitus volume decreases.³ Again, although the exact mechanism of action is unknown, researchers believe that the therapy utilized cortical neuroplasticity to reduce the spontaneous firing of the neurons responsible for the tinnitus percept by strengthening

existing networks of lateral inhibition from undamaged cochlear hair cells.²

CHALLENGES FOR PARTNERING AUDIOLOGISTS

Determining a patient's tinnitus frequency has classically proven to be of variable difficulty. Patients have a hard time determining the frequency of their tinnitus, and often provide varying readings with little validity. Research has shown that there are two sub-groups of patients – *about 50% can reliably determine their tinnitus frequency, and about 50% cannot.* <http://www.audionotch.com/blog/tinnitus/more-on-tinnitus-pitch-matching-a-study-of-interest/>. In summary, tuning accuracy is critical for the success of notched sound therapy, as the notch must align with the actual tinnitus frequency (there is, however, some margin for error). In our experience, patients

prefer to have their tinnitus frequency detected by an audiologist, instead of using web-based software to do so.

Our web-based service is AudioNotch, but we also have an audiologist partnership program set up under a different brand: Tinnitus Treater. Details are available here on the Tinnitus Treater FAQ for clinics interested in a partnership. <http://www.tinnitus treater.com/audiologists>

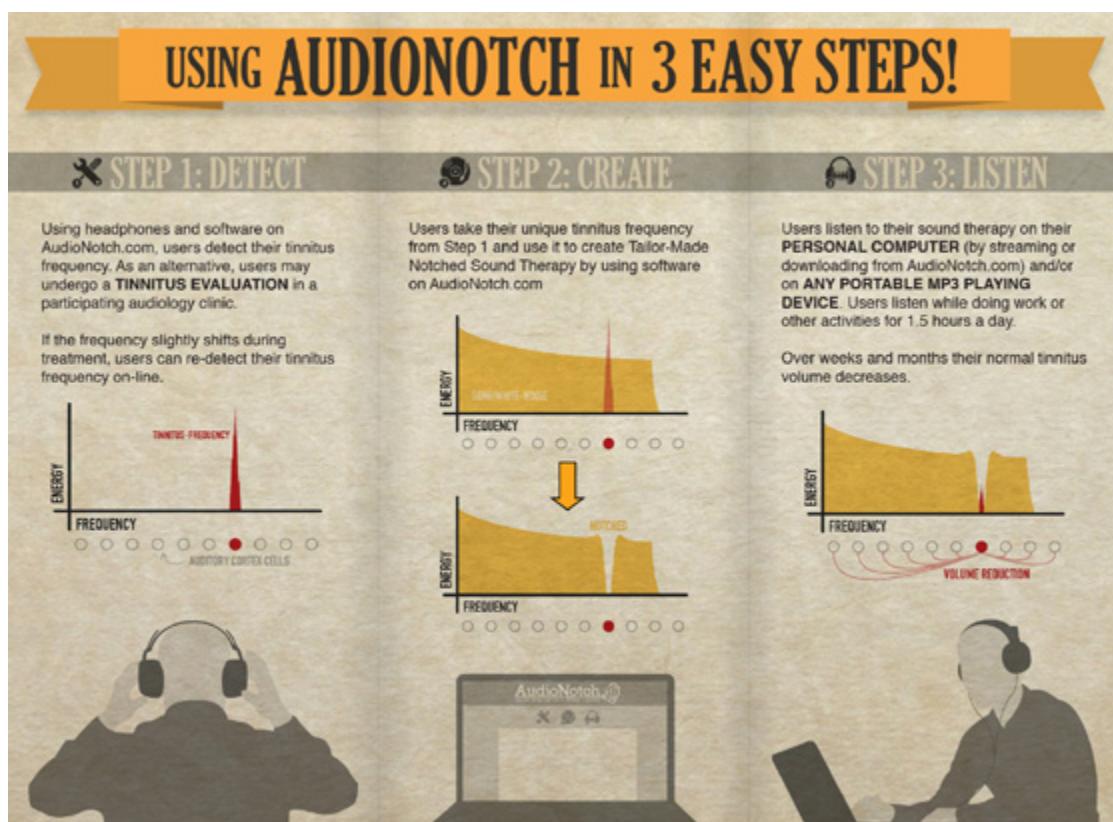
THE STATE OF THE RESEARCH

A critical appraisal of the research on Notched Sound Therapy shows that, although encouraging, the studies suffered from multiple limitations, and more work needs to be done in this area to draw firm conclusions. More studies with larger sample sizes need to be done, and long-term follow-up studies must be performed to determine if the effect

of the therapy persists. However, for patients approaching audiologists and other hearing health professionals, it represents a reasonable treatment option that can be recommended in light of provisional scientific support. For those of you who are interested, I've written a commentary on a critical review of the research, which is available here. <http://thehearingblog.com/archives/2094>

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Critical Review:

Efficacy of Notched-Sound Therapy for Neural Plasticity Mediated Tinnitus Treatment

By Mark Bennett, MCISc



About the Author

Mark Bennett was born and raised in Brantford, Ontario. He developed a passion for community health while pursuing a bachelor's degree in physiological psychology from Brock University. Here, he taught undergraduate health psychology seminars and helped establish rehabilitation programs for the Ontario March of Dimes in the Niagara region.

Mark graduated from The University of Western Ontario with a Master's of Clinical Science in Audiology. He has worked in multiple private hearing clinics across Canada in both adult and pediatric settings.

Most recently, Mark founded and runs the Cobourg Hearing Centre - a comprehensive hearing clinic serving the Northumberland community. His current clinical and research interests are adult hearing-impaired treatment programs, aural rehabilitation, and management programs for tinnitus.

This critical review examines the effects of notched-sound therapy on the human auditory system and its' potential as a novel and effective treatment option for tinnitus. Studies evaluated consisted of one mixed groups randomized clinical trial, two mixed groups pseudorandomized clinical trials, and two mixed groups non-randomized clinical trials. Analyses of these studies revealed moderate evidence for notched-sound's ability to induce changes in the auditory cortices and reduce tinnitus-related symptoms. Future research directions and clinical applications shall be discussed.

INTRODUCTION

It is well documented that the tonotopically-arranged sensory maps in the human auditory cortex are neither static nor permanent in their functional organization. For instance, animal

models have demonstrated that damage to peripheral afferent sensory inputs results in deafferented cortical regions taking over and resuming functions for neighboring cortical sites.¹ This induced deafferentation of auditory pathways results in cortical neurons to shift and broaden their receptive fields outside of the damaged area within hours.² It has been demonstrated in other sensory systems that such cortical plasticity is not limited to cases of permanent lesions or long term sensory deprivation. For instance, restructuring of sensory neurons in the visual system has been documented after reversible "functional" deafferentations whereby the field of vision is temporarily restricted.³ It is proposed that such neural retuning, as a result of either permanent or functional deafferentation, occurs due to several plasticity-based changes in the sensory system. These changes

include: the unmasking of existing cortical connections, lack of local inhibitory connections, alterations in cell membrane excitability and synaptic efficacy.⁴

Subjective tinnitus, the percept of sound in the absence of external acoustic stimuli, affects 5–15% of the human population and is one of the most prevalent symptoms of hearing impairments.⁵ Tinnitus has been shown to be loud and chronic enough to drastically influence quality of life.⁵ In severe cases, it can have negative psychophysiological impacts that result in anxiety, depression, and insomnia.⁶ An effective, universally accepted treatment approach that objectively reduces tinnitus symptoms does not exist. Studies have shown that the perception of tinnitus rises from the auditory cortex and the

generation and persistence of tinnitus is related to maladaptive auditory cortex reorganization.⁵ It is therefore speculated that such maladaptive neural structuring may be reversed by some form of functional deafferentation.

OBJECTIVES

The overlying objective of this critical review is to determine if there is evidence to support that notched-sound therapy is an effective and practical treatment option for tinnitus. Thus, evidence evaluating whether exposure to notched-sound can induce plasticity-based alterations in the human auditory system, and whether such exposure affects tinnitus-related symptoms shall be reviewed.

METHODS

SEARCH STRATEGY

Computerized databases, including, Pubmed, CINHAHL, and Scopus were searched using the following strategy:

- a ((noise) OR (sound) OR (music)) AND ((notched) OR (filtered) OR (windowed)) AND (tinnitus)
- b ((noise) OR (sound) OR (music)) AND ((notched) OR (filtered) OR (windowed)) AND (human auditory cortex))

The results of these two search strategies were combined for evaluation of selection criteria and acceptance into this critical review. The search was limited to articles written in English and those studying humans only.

SELECTION CRITERIA

Studies selected for inclusion in this critical review paper were required to either investigate the impact of notched sound (digitally filtered at a specified

frequency band) on the human auditory system and/or on tinnitus-related symptoms. Limitations were not set for which level of the auditory system was being evaluated (peripheral systems to cortices) or on a particular population of tinnitus sufferers. No limits were set on the demographics of research participants (aside from being human) or outcome measures.

DATA COLLECTION

Results of the literature search yielded the following 5 articles congruent with the aforementioned selection criteria: mixed groups randomized clinical trial (1), mixed groups pseudo-randomized clinical trial (2), mixed groups non-randomized clinical trial (2).

RESULTS AND DISCUSSION

RANDOMIZED CLINICAL TRIAL

Study #1: Lugli and colleagues performed a study evaluating the effects notched noise stimuli on patients' subjective ratings of tinnitus loudness (dB).⁷ Participants recruited were unilateral and bilateral tinnitus patients with high frequency hearing loss of both gender ($n = 43$). Participants were randomly assigned to one of three auditory stimulation regimens: (1) broadband noise containing a notch centered at the individual's tinnitus frequency (WWN); (2) non-notched broadband noise (WN); (3) waterfall noise (Wa). Participants listened to their assigned auditory stimulation for 1.5–3 hours/day over a 1 year span.

Absolute and percentile changes of tinnitus loudness from baseline measures across groups were examined using the Kruskal-Wallis non-parametric analysis of variance. When significant differences between means were found,

a multiple-comparisons procedure of the Kruskal-Wallis test was used to test the difference of mean values between pairs of treatments. Results showed that the mean final absolute change of tinnitus loudness between the three groups differed significantly ($H = 15.6$, $p < 0.001$). Multiple comparisons analyses revealed that final absolute change of tinnitus loudness was significantly larger in the WWN group than in the two controls ($p < 0.5$). There was not a significant difference between the two control groups. The final absolute tinnitus loudness decreased significantly by roughly 12 dB from baseline in the WWN group and non-significantly by roughly 2 dB in the two control groups. Importantly, upon completion of the treatment the perception of tinnitus was entirely eliminated in four of the twenty participants in the WWN group. None of the participants in either control group completely recovered.

Overall, the results of this study provide moderate support for a notched sound exposure as a potential treatment option for tinnitus sufferers and warrants future search in this area. However, the results fail to explain the underlying mechanisms of tinnitus loudness reduction or answer the current research questions. A lack of neurophysiological data limits the ability to correlate a reduction of tinnitus sensation to changes in the neurophysiology of the human auditory system. Moreover, a major limitation to the methodology of the study was that it failed to follow the participants longitudinally posttreatment. Therefore, it is unclear whether the reduction of tinnitus loudness is maintained, and to what degree, after prolonged exposure has ceased.

PSEUDO-RANDOMIZED CLINICAL TRIALS

Study #2: Okamoto et al conducted a study evaluating the effects of notched music on subjective tinnitus loudness and evoked activity in the auditory cortex.⁸ The participants ($n = 23$) matched the following inclusion criteria: (1) chronic tinnitus; (2) unilateral/strongly lateralized tinnitus; (3) tonal tinnitus; (4) tinnitus frequency $< 8\text{kHz}$; (5) no severe hearing impairment; (6) no neurological or psychiatric complications.

Participants willing to participate in music training therapy were pseudorandomly assigned to one of two notched music exposure groups: (1) target notched music, which was filtered centered at the participants' individual tinnitus frequency ($n = 8$); (2) placebo notched music, which was had a moving filter that avoided the individuals' tinnitus region ($n = 8$). Those unwilling to participate in music therapy due to time constraints were assigned to a monitoring group and received no treatment ($n = 7$).

Participants listened to notched music daily for 1 year. Listening times were recorded daily and approximated at 2 hours/day. Tinnitus loudness and magnetoencephalography (MEG) measures were taken at baseline and periodically throughout the study. MEG recordings used both a test stimulus frequency corresponding to the participants' individual tinnitus frequency and a control stimulus of 500 Hz; both N1m and auditory steady state (ASSR) measures were taken.

Methods of statistical analyses are not reported by the authors. The target group showed a significant reduction

in tinnitus loudness after 12 months of treatment compared to baseline ($F[1,7] = 26.1, P = 0.001$). Also, there was a significant interaction between group (target vs. placebo) and time of measurement (baseline vs. average across 7–12 months) ($F[1,14] = 5.9, P = 0.030$). However, the placebo and monitoring groups did not significantly differ from baseline measures of tinnitus loudness.

In the target group, both ASSR and N1m source strength ratios were significantly reduced after 12 months compared to baseline (ASSR: $F[1,7] = 5.9, P = 0.045$; N1m: $F[1,7] = 24.6, P = 0.002$). There was also a significant interaction between group (target vs. placebo) and time of measurement (baseline vs. month 12) for both ASSR ($F[1,14] = 6.1, P = 0.027$) and N1m ($F[1,14] = 13.1, P = 0.003$). There was not a significant difference between baseline and at 12 months for either the placebo or monitoring group for ASSR or N1m signal strength ratios. Reductions of all outcome measures (tinnitus loudness, ASSR, N1m) found in the treatment group were also significant by 6 months. Finally, there was a significant correlation between tinnitus loudness change and auditory evoked response ratio change of the ASSR ($r = 0.69, p = 0.003$), but not for the N1m.

In summary, the treatment group showed significant reductions in both tinnitus loudness and auditory cortex evoked activity relative to baseline. Such results appear to provide plausible evidence for the current research question. The authors conclude that the decreased evoked cortical activity, as represented by the MEG recordings, reflects a reduction of pathological

auditory neural activity corresponding to the tinnitus frequency. Such a reduction subsequently led to reduced tinnitus loudness. The authors do however fail to speculate on nonsignificant correlation between tinnitus loudness and the N1m measures.

Despite these positive findings, the results should be interpreted cautiously. The authors failed to utilize a randomized clinical trial and due to participant refusal, used a monitoring group. Moreover, they fail to specify how the participants were pseudorandomly designated to either the treatment or placebo group. Therefore, possible confounding variables are left unknown and we are forced to render their control groups as meaningless. The authors also failed to provide any description of statistical analyses used. It is also curious that they omitted any reference to data concerning the control carrier frequency of 500 Hz used in the MEG recordings.

Study #3: A paper by Stracke and colleagues presents data that went unpublished from their original paper: the aforementioned Study # 2.⁹ This publication presents the effects of notched music on the perceived annoyance and handicap of tinnitus. As in Study #2, the methods of statistical analyses are not provided by the authors. Compared to baseline, the target group reported significantly less tinnitus annoyance and tinnitus-related handicap upon completion of therapy. Those in the placebo and monitoring groups did not report a significant reduction of these two measures.

In summary, the participants in the treatment group from Study #2 not only had reduced auditory cortical activity and tinnitus loudness, they also

reported less annoyance and handicap experienced by the tinnitus. This supports that such treatment may be effective at improving tinnitus symptoms on a global scale and lends support to such a regimen as a possible form of tinnitus management. Moreover, it also strengthens the association between cortical activity and tinnitus perception. However, these results remain questionable for the aforementioned methodological limitations discussed in Study # 2.

NON-RANDOMIZED CLINICAL TRIALS
 Study #4: Teismann, Okamoto, and Pantev (2011) evaluated the effects of notched music on subjective tinnitus loudness, tinnitus-related distress, and cortical evoked activity.¹⁰ Moreover, the study assessed whether the perceived pitch of tinnitus affects the efficacy of notched music therapy based on the above outcome measures.

Participants ($n = 20$) with chronic, tonal tinnitus who did not have severe hearing losses were recruited for the study. Participants were separated into a low tinnitus frequency group (<8 kHz) and a high tinnitus frequency group (>8kHz) based on pitch matching tasks. Participants were exposed to music notched at their individual tinnitus frequencies. They were instructed to listen for three hours on day 1 and 5 and for six hours on days 2–4. MEG recordings (both ASSR and N1m source strength ratios) were taken using a carrier stimulus corresponding to the participants' individual tinnitus frequency and a 500 Hz control stimulus. For patients with tinnitus frequencies above 8 kHz, auditory evoked fields could not be measured with sufficient quality and

were thus not included in statistical analyses.

Planned *t*-tests were used to compare outcome measures from baseline to specified intervals posttreatment within each group. For the low frequency group, significant reductions were found in at least one outcome measure at all intervals post-treatment. At 3 hours post-treatment there was a significant reduction in tinnitus loudness ($t = -2.3, p < 0.03$), but no other outcome measure. At 3 days post-treatment there was a significant difference reduction in N1m ratios ($t = -2.14, p < 0.02$). There was no significant change in tinnitus loudness, tinnitus-related distress, or ASSR ratio. At 17 days post-treatment there were significant reductions in tinnitus-related distress ($t = -2.11, p < 0.02$), tinnitus loudness ($t = -2.15, p < 0.02$), and N1m ration ($t = -1.97, p < 0.03$). There was not a significant difference in ASSR ratio. At 31 days post-treatment there was a significant reduction in tinnitus-related distress ($t = 2.38, p < 0.01$), but not in any other outcome measure. For the high frequency group, there was not a significant change in tinnitus-related distress or tinnitus loudness at any of the four points measured compared to baseline. A possible reason for the null results in this group, suggested by the authors, is that the music contained little high frequency energy. Future research with this population should include high frequency enriched stimuli.

It is evident from results across the follow-up measurements post-treatment that both the subjective ratings of tinnitus symptoms and MEG measures are inconsistent across days. The authors speculate that tinnitus

loudness reduction did not persist past 3 days because the treatment program was uniquely short. Thus, the neuronal activity reduction, observed by the N1m, was merely functional and thus only temporary. They go on suggest permanent structural changes may require extended treatment periods. This is supported by evidence from treatment programs for other diseases associated with maladaptive brain plasticity such as phantom limb pain. Whether such permanent neural changes can occur through notched sound exposure can occur in the human auditory system remains unanswered. Importantly, whether such structural changes correlate to a reduction in tinnitus-related symptoms remains to be seen. Thus, although the results of this study warrants future research into the relationships between neural reorganization, the auditory cortical tonotopic map, notched sound, and tinnitus treatment, they provide only plausible evidence at this time. Moreover, the study has several limitations such as failure to utilize a randomized, double-blind procedure with a control group and small sample size relative to the large percentage of tinnitus sufferers in the population.

Interestingly, the authors also speculate on the lack of ASSR reduction in all post-treatment measurements. ASSR source strengths were found to decrease after a more lengthy notched sound treatment in study # 2. Therefore, if the primary auditory cortex (ASSR) may require longer periods of treatment for plastic changes to be observed compared to the non-primary auditory cortex (N1m). What is evident from this prediction is that the plastic nature of the human auditory system is not

yet well understood. More research is needed addressing the precise neural changes as a result of notched sound exposure, and what changes, if any, are most associated with the perception of tinnitus.

Study #5: Pantev et al examined the effects of notched music on cortical neural activity.¹¹ Participants ($n = 10$) between the ages of 25–50, with normal hearing and no history ontological or neurological disorders were recruited. Participants listened to 3 hours of music notched at 1 kHz. Immediately before and after music exposure, MEG recordings were taken using both a test stimulus of 1 kHz and a control stimulus of 500 Hz. This procedure was repeated on three consecutive days. Both the global source power averaged over the sensory array (RMS) and the dipole moment (Q) were the principal parameters used in this study.

An analysis of variance evaluated main effects of the variables days (1–3), stimuli (test/control), before/after functional deafferentation. The RMS field values and dipole moment were assessed using pre-planned *t*-tests contrasting test and control stimuli before and after listening to notched music. There was a significant interaction of before/after with stimulus (test/control) was found for Q ($F[1,9] = 6.71, P = 0.029$) *T*-tests of measurements taken before and after notched music were not significant for the control stimulus when applied to Q ($t[9] = 0.894, p = 0.394$) or RMS ($9t[9] = -0.447, p = 0.665$). For the test stimulus, Q diminished significantly after listening to notched music ($t[9] = -3.30, p = 0.009$). Similar results were also significant for RMS, but only after applying higher significance levels

($p < .10$). For the test stimulus there was also a significant interaction between days and before/after $F(2,18) = 3.76$ ($p = 0.043$). It was discovered that the RMS measures decreased after the second and third days of music exposure, but not the first. There were no significant main effect or interactions concerning days and before/after for the control stimulus. Finally, upon completion of this study, two participants repeated the same study except that the control and test frequencies had been reversed. There were no significant effects.

The authors were able to use MEG recordings to measure short-term plastic changes after periods of brief functional deafferentation. These results were both cumulative and temporary. The reduction of cortical activity was more pronounced on days two and three of exposure; however, after each 24 hour period, values returned to baseline. These results are only suggestive and do not provide strong evidence that notched music treatment can induce permanent neural reorganization; or if such reorganization has any impact on tinnitus symptoms. Finally, their null results in experiment two is not surprising due to the number of participants. However, to be considered a treatment options, the method must be flexible to a variety of individual tinnitus frequencies. A counterbalanced, crossover, randomized, controlled study, which uses multiple test frequencies is needed.

CONCLUSION AND CLINICAL IMPLICATIONS

The underlying rationale behind the studies under review are that by targeting auditory cortex neurons that code for tinnitus frequencies,

through customized notched sounds, maladaptive neural structuring that is causing tinnitus perception may be remedied. Indeed, where applicable, the authors of these studies attribute their positive to results to the ability of notched sound to reduce the excitability of hyperactive auditory neurons. This reduction would be caused by strengthening weakened inhibitory networks in the critical tinnitus frequency band. Such suggestions remain purely speculative and rest heavily on studies of animals and other sensory systems. Although these studies provide some evidence that notched sound exposure can reduce auditory cortex activity, MEG measurements represent large areas of the nervous system and should be interpreted with caution. The MEG results across studies were inconsistent. In the studies of notched sound exposure lasting only several days, such reductions in cortical activity were not maintained and in some cases not measurable. In both studies of 1 year, an adequate assessment of cortical activity post-treatment was not obtained. Therefore, it remains unclear whether such reductions can be sustained. Similar to the neurophysiological results, the subjective ratings of tinnitus symptoms were inconsistent across studies and whether a reduction of symptoms in these participants was maintained is unknown. It is also important to consider the overt methodological limitations, which were largely consistent amongst the studies under review. The lack of longitudinal randomized controlled studies, disclosure of statistical methods, and omission of many important statistical data renders the results of these studies questionable.

Indeed, a customized notched sounds treatment, if feasible, would be a cost effective and perhaps even enjoyable (music) form of tinnitus treatment. However, there is not yet enough evidence to support that such a form of treatment is ready for clinical implication. The studies that have attempted to study the efficacy of such a treatment remain too few and inconsistent. The studies under review differed in many ways including: forms of notched sound (noise vs. music), tinnitus loudness and frequency matching, length of exposure, outcome measures, and participant inclusion (hearing thresholds, tinnitus characteristics, and age). The latter point is significant clinically as what population such a treatment option would be successful for, if at all, remains unknown. Important to the audiology profession, if those with both hearing impairments and tinnitus could benefit from this treatment is important. Whether or not such treatment could be used while wearing hearing aids is also an avenue for future research. This of course after the more conclusive evidence to suggest that notched sound can indeed restructure maladaptive neural networks associated with the perception of tinnitus.

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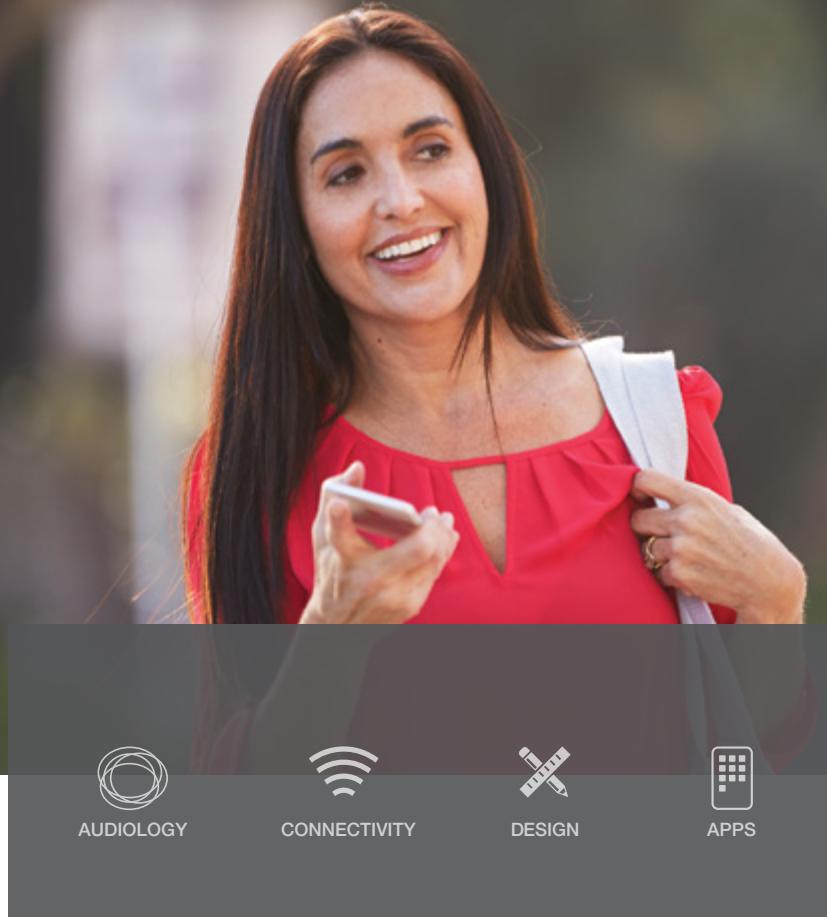
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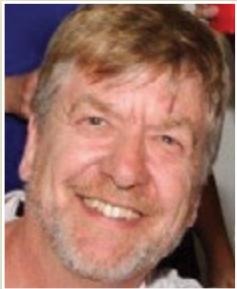
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HEARING LOSS and DEMENTIA

Ted Venema, PhD



About the Author

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Hearing loss, does it cause dementia and Alzheimer's? Around November 2013 I saw an article called "Brain Atrophy & Hearing Loss" (Healthy Hearing, June 25, 2013). Health magazines like these are intended for the general public. Their articles are written in terms that are meant to be digested and understood, and they often draw their sources from latest research in various medical health-related fields. I thought I'd look up the actual source cited in "Brain Atrophy & Hearing Loss"; this turned out to be an article by authors Peele, J., Troiani, V., Grossman, M., and Wingfield, A., titled: "Hearing Loss in Adults Affects Neural Systems Supporting Speech Comprehension," and it appears in the *Journal of Neuroscience*, August 31, 2011 (see Figure 1, left side).

Like many peer-reviewed articles in behavioural research, this one began with an *Abstract* giving an overall summary of the paper, and moved on to its *Introduction* where the topic relevance is described and the purpose of the paper is laid out. It then explained its *Methods*, where the subject pool is described and what kinds of experimentation will take place. It then analyzed its *Results* using statistics, in order to show proof of its findings, and then it concluded with its *Discussion* of the results.

The authors hypothesize that there is a *relationship* or *association* between SNHL and the ability to process auditory linguistic information, and that this processing involves perception and cognition at a neural level. With SNHL, an "impoverished acoustic signal" arrives at the brain to be processed as speech. As time goes on, this will result in actual

changes in the cells of the auditory areas of the cortex. They did two experiments: the first one used Functional Magnetic Resonance Imaging (fMRI) to examine the effects of hearing loss upon actual cortical activity. The second experiment went on to find out what *actual brain atrophy* might have occurred and exactly where this was located. For both experiments,

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MEDICINE
Use It or Lose It: Hearing Loss and
Dementia Linked

Abstract: *Hearing loss in older adults is associated with cognitive decline, particularly in language comprehension. To examine the effects of hearing deficit on the neurophysiological mechanisms underlying language processing in humans, we conducted behavioral and functional magnetic resonance imaging (fMRI) studies in normal-hearing and older adults with sensorineural hearing loss (SNHL) who were matched for age, sex, education, and language proficiency. In the fMRI study, older adults with SNHL showed significantly more brain activity than did normal-hearing older adults. Substantial differences in brain activity predicted the degree of language comprehension in older adults with SNHL. In a second experiment, we measured the relationship of hearing deficit to cerebral cortical integrity using voxel-based morphometry. In a second experiment, we measured the relationship of hearing deficit to cerebral cortical integrity using voxel-based morphometry. These results suggest that increased deficits in temporal auditory activity is a common consequence of progressive sensorineural hearing loss in older adults. Thus, greater sensorineural hearing loss is associated with changes in the brain that may contribute to cognitive decline in older adults.*

Abstract: *To determine whether hearing loss is associated with an increased risk of dementia, we conducted a case-control study of 1,000 community-dwelling elderly subjects (mean age, 75 years) and 1,000 control subjects (mean age, 75 years). Hearing loss was defined as a pure-tone average of 40 dB or greater in either ear. The primary outcome measure was the presence of dementia, as assessed by the Mini-Mental State Examination. We found that older adults with hearing loss had a higher prevalence of dementia than did those without hearing loss (31% vs 21%; P < .001). After adjustment for age, sex, education, race, and other risk factors for dementia, older adults with hearing loss had a 2-fold increased risk of dementia (odds ratio, 1.9; 95% confidence interval, 1.4-2.4; P < .001). The association between hearing loss and dementia was independent of cognitive impairment at baseline. In addition, older adults with hearing loss had a higher prevalence of mild cognitive impairment (15%) compared with those without hearing loss (10%; P = .001). These findings support a causal relationship between hearing loss and dementia.*

Figure 1. Two general articles that have appeared recently are pictured at the top of this slide. In each of these articles a more specific scholarly article is cited and discussed. These are shown respectively, in the bottom of the figure.

the average hearing loss of the subjects was only mild; borderline-normal in the lows to about 30–35 dB HL for the highs. Subjects were presented with sentences that were progressively increased in syntactic difficulty, as well as at different rates of speech presentation. Their task was to indicate whether the speaker was male or female.

In both experiments, they used the statistic of something called “Pearson r.” This family of statistics works to establish if there is a *co-relation*; it does not focus on proving cause-effect (see Figure 2). For example, it’s one thing to say that lung cancer happens to be *associated* with smoking; it’s much stronger to say that the link is direct, that smoking *causes* cancer. The first experiment in their study indeed showed a statistically significant reduction in cortical activity for both right and left primary auditory areas, in the temporal lobes of the brain. The second experiment showed a statistically significant reduction in gray matter volume for the right (not the left) auditory cortex.

At the end of their paper they discuss that peripheral hearing loss may result in *increased effort* in hearing for speech. This would be increasingly evident as hearing loss becomes worse and also as the listening task becomes more difficult. Increased efforts to hear will deprive one from concentrating on, and processing higher cognitive functions. The authors concluded that although they do not have conclusive evidence, they “...think it plausible that changes in older adults’ peripheral hearing ability had a *causal role* (italics mine) in reducing gray matter volume in auditory cortex.” In summary, while they managed to show a *co-relation or association* between HL in the elderly and brain atrophy, they also wondered if there is more, perhaps a cause-effect connection.

One might wonder if hearing loss itself results in brain atrophy, then is this happening in *young* people with hearing loss as well? The authors posit that the effects will be most pronounced for the elderly, because compared to young adults with the same SNHL, the elderly

perform more poorly on speech and linguistic experimentation. As an aside, it is interesting that they found most atrophy for the right temporal auditory areas, because it is normally well known that the *left* auditory cortex is dominant for speech in most people.

Right around the same time as I saw the first article, a second one followed. This one was a news release out of Johns Hopkins University School of Medicine, once again intended for readership by the general public (see Figure 1, right side). Its title was “Use It or Lose It; Hearing Loss & Dementia Linked.” Like the first general article did, it referred to another scholarly article by the authors Lin, F., Metter, E., O’Brien, R., Resnick, S., Zonderman, A., and Ferrucci, L., titled: “Hearing Loss and Incident Dementia” (Archives of Neurology, February 2011).

Once again, I went on the Internet and pulled up the original, detailed article and once again I painstakingly examined it, statistics and all. It is no wonder that the health magazines simplify things; have you ever tried to actually read these kinds of articles? Again like the first article did, right at the outset, at the top of its first page this second paper declares its objective: to determine if there is an *association* between hearing loss and Dementia and Alzheimer disease. In contrast to the first article however, where experimentation was done on a group of subjects, this article is a longitudinal study, one that followed the lives and course of events for subjects over a time span of about 12 years. The subject pool had over 600 people, and in the years between 1990–1994, they were all between the ages of 39–90 years old and were described as “dementia-free.” The demographic characteristics of all the subjects were assessed in terms of their

Scatterplot with 25 Subjects and “Line of Best Fit”

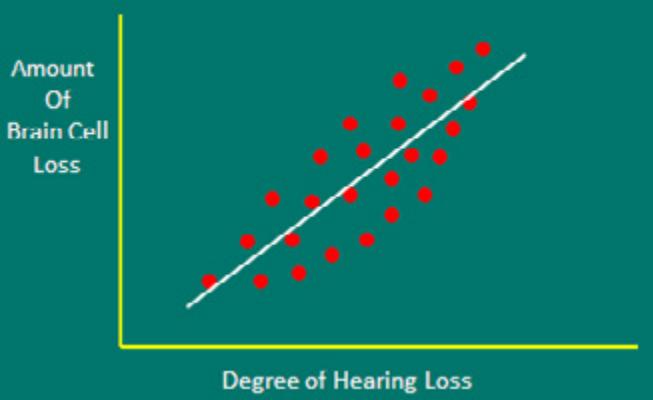


Figure 2. A co-relationship or association is shown here between degree of hearing loss and amount of brain cell loss. Each dot represents one subject in this hypothetical study. The diagonal line represents a “line of best fit,” which shows that there is a positive linear relationship between these two variables; namely, with increased hearing loss there tends to be more brain cell loss.

gender, age, race, education, whether they had diabetes, were smokers, had hypertension, used hearing aids, their scores on a test of dementia, and lastly, whether they developed dementia over the time line of the study. Subjects were then divided into groups of different degrees of SNHL: normal hearing, mild, moderate, and severe.

Results of their analysis showed a statistically significant association between hearing loss and dementia. The study also did a complex assessment of risk of developing dementia due to degree of hearing loss. It found that for hearing loss greater than borderline-normal (25 dB HL), the risk increased as the hearing loss increased. The findings remained the same even after even after the analysis was adjusted for gender, age, race, education, diabetes, smoking, and hypertension, and subjects with a history of aphasia (stroke) were excluded.

Interestingly, they also said that, “There was no evidence to suggest that self-reported hearing aid use was associated with a reduction in dementia risk.” They admit, however, that they did not investigate the number of years or the length of time per day that hearing aids were worn, and consequently, they could not conclude with certainty that hearing aid use could reduce the risk of dementia.

Just as the authors of the first article did, these authors also discuss a possible causal link. Like the first article, they conclude that hearing loss makes listening more difficult and as a result, more cognitive resources are dedicated to listening, and this is to the detriment of other cognitive processes. Okay fine; but it’s that causal thing again...

It is a mistake – and too easy – to believe

articles that show an association between hearing loss and dementia prove that hearing loss *causes* dementia. This may be proven further on down the road, but is hasn’t yet been proven. Maybe that’s never been the intent here behind these articles, but just in case readers think it might be, we all need to step above this.

A third general article about hearing loss and dementia from sources “closer to home” just popped into my email the other day! This one was the announcement of a joint study to be completed late this year (2015) between A. Amlani, an associate professor of speech and hearing at the University of North Texas and B. Taylor, director of practice development and clinical affairs at Unitron. The slant of this third study is *rehabilitative*, in that it seeks to show that the use of hearing aids may be able to delay mild dementia onset. The subjects are adults between the ages of 50–90 years of age, who already have mild dementia, and who have no previous experience wearing hearing aids. The investigators want to assess their abilities for hearing speech in noise, their overall cognitive abilities, as well as their own self-reported improvements in quality of life. Their question is whether improved hearing can help those with dementia to “...lead more active and engaged lives, particularly if hearing loss is identified and treated early.”

Well okay, that makes perfect sense! Isolation from social activities is certainly not going to do any good for someone who is in the stages of developing mild dementia. Providing better hearing sensitivity for these people, so as to increase contact with the on-goings of the outside world, would obviously be a prudent endeavour. *This is a positive way of encouraging clients who need hearing aids to get hearing aids.*

Guess what? We have actually encountered this whole topic before, but from a perspective well ensconced within our own hearing health care field – audiology – as opposed to that of researchers from neuroscience, psychology and medicine. Have a look at an article called, “Hearing aid use, central auditory disorder, and hearing handicap in elderly persons” by Rose Chmiel and James Jerger, in the *Journal of the American Academy of Audiology* in 1996; issue 7(3). The whole May-June issue 7(3) of JAAA deals specifically with aging and hearing loss. The specific paper cited here describes why the very elderly (those 85 years and older) may not always benefit much from *binaural hearing aid fittings*. The reasoning is posited as follows: the whole body ages, not just the cochlear hair cells. This “central presbycusis” may explain why the very elderly brainstem simply cannot fuse together very well the input from each ear; as a result, they cannot always make the best use of binaural amplification. Similar findings are laid out more recently in 2005, when Martin and Jerger published an article titled “Some effects of aging on central auditory processing” (*Journal of Rehabilitation Research & Development*, Volume 42 Number 4, July/August 2005, Supplement 2). Hmm...Maybe people simply grow older...Food for thought...

To conclude here, humans are creatures of communication. Hearing involves communication, and communication keeps us in the game of living. We wear glasses the better to see; similarly, we should acknowledge that we wear hearing aids the better to hear! That’s the take I get from all of these articles. Let’s love our elderly and do all that we can to help them to hear!

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