ISSN: 2083-389X
eISSN: 2084-3127

J Hear Sci, 2025; 15(Special Issue): 7–16
DOI: 10.17430/jhs/204835

Received:
October 21, 2024
Revision accepted:
May 8, 2025
Published online:
December 19, 2025

HYPOTHESIS PAPER

LOUDNESS HYPERACUSIS: MECHANISMS OF LOUDNESS PERCEPTION AND THEIR BREAKDOWN

Brian C. J. Moore[AEF]

Department of Psychology, University of Cambridge, UK

Corresponding author: Brian C. J. Moore, Cambridge Hearing Group, Department of Psychology, University of Cambridge, Downing Street, Cambridge CB2 3EB, UK; email: bcjm@cam.ac.uk

Contributions:
A Study design/planning
B Data collection/entry
C Data analysis/statistics
D Data interpretation
E Preparation of manuscript
F Literature analysis/search
G Funds collection

Abstract

This paper is concerned with loudness hyperacusis, a condition where sounds of medium and high levels appear to be louder than normal. The "normal" perception of loudness can be understood using a model that takes into account the processing of sounds in the peripheral auditory system, including the outer ear, middle ear, and cochlea. This model has been modified to take into account the perception of loudness by people with cochlear hearing loss. The model predicts the loudness recruitment typically associated with cochlear hearing loss, and also predicts that hearing loss can sometimes be associated with "over-recruitment," so that some sounds appear louder than normal. However, the model does not account for the fact that loudness hyperacusis can occur for people with normal or near-normal audiograms. This suggests that factors associated with higher levels in the auditory system also need to be taken into account. Here, two such factors are considered: the functioning of the efferent system regulating the active mechanism in the cochlea, and effects of central plasticity and adaptation. Both may play a role in hyperacusis.

Keywords: brain plasticity • hyperacusis • loudness • efferent system • loudness recruitment

NADWRAŻLIWOŚĆ NA GŁOŚNE DŹWIĘKI: MECHANIZMY PERCEPCJI GŁOŚNOŚCI I ICH ROZKŁAD

Streszczenie

Niniejszy artykuł dotyczy nadwrażliwości słuchowej, czyli stanu, w którym dźwięki o średnim i wysokim poziomie wydają się głośniejsze niż normalnie. „Normalne" postrzeganie głośności można zrozumieć za pomocą modelu, który uwzględnia przetwarzanie dźwięków w obwodowym układzie słuchowym, w tym w uchu zewnętrznym, uchu środkowym i ślimaku. Model ten został zmodyfikowany w celu uwzględnienia percepcji głośności przez osoby z ubytkiem słuchu typu ślimakowego. Model przewiduje po pierwsze, rekrutację głośności charakterystyczną dla ślimakowego ubytku słuchu, a po drugie, że ubytek słuchu może czasami powodować „nadmierny efekt wyrównania głośności", tak że niektóre dźwięki wydają się głośniejsze niż normalnie. Model ten nie uwzględnia jednak faktu, że nadwrażliwość słuchowa może występować u osób z audiogramem w normie lub prawie w normie. Sugeruje to, że należy również wziąć pod uwagę czynniki związane z wyższymi poziomami układu słuchowego. Rozważane są tutaj dwa takie czynniki: funkcjonowanie układu eferentnego regulującego aktywny mechanizm w ślimaku oraz efekty plastyczności mózgu i jego zdolności adaptacji. Oba mogą odgrywać rolę w nadwrażliwości słuchowej.

Słowa kluczowe: plastyczność mózgu • nadwrażliwość słuchowa • głośność • układ eferentny • efekt wyrównania głośności

Key to abbreviations	
ART	acoustic reflex threshold
DPOAEs	distortion product otoacoustic emissions
ERB_N	equivalent rectangular bandwidth of the normal auditory filter
HCL	highest comfortable level
HL	hearing level
HQ	Hyperacusis Questionnaire
HTLs	hearing threshold levels
IHC	inner hair cell

Key to abbreviations	
IHS	Inventory of Hyperacusis Symptoms
LDL	loudness discomfort level
LS	loudness suppression
MAF	minimum audible field
MOC	medial olivocochlear
OHCs	outer hair cells
SLT	sound level tolerance
ULL	uncomfortable loudness level

Introduction

Loudness is defined as the intensive attribute of auditory sensation in terms of which sounds can be ordered on a scale extending from soft to loud [1]. Put more simply, loudness is the subjective impression of the strength of a sound. The unit of loudness is the sone. One sone is defined as the loudness of a 1000 Hz sinusoid at 40 dB SPL, presented binaurally from a frontal direction in a free-field as perceived by people with normal hearing. A sound with a loudness of 4 sones is 4 times as loud as a sound with a loudness of 1 sone. A loudness-related quantity that may be easier to interpret is the loudness level, which is decibel-like and has units of phons. The loudness level of any sound in phons is the level (in dB SPL) of the 1000 Hz sinusoid to which it sounds equal in loudness for listeners with normal hearing. For example, if a sound *S* appears to be as loud as a 1000 Hz sinusoid with a level of 45 dB SPL, then the sound *S* has a loudness level of 45 phons. For sounds with levels above about 40 dB SPL, the loudness in sones roughly doubles for each 10-phon increase in loudness level [2,3]. For example, if the loudness level is increased from 40 phons to 60 phons the loudness increases from 1 sone to 4 sones.

For most people with normal hearing, sounds are usually audible and comfortably loud over a wide range of sound levels, from about 0 to 90 dB SPL for mid-range frequencies. However, for some people, sounds become unpleasantly loud when their level is well below 90 dB SPL. This represents a form of hyperacusis. There is no universally agreed definition of hyperacusis, and there may be several varieties of hyperacusis [4]. Here, the focus is on *loudness hyperacusis*, a condition where sounds with moderate levels are perceived as uncomfortably or unpleasantly loud, even though those sounds would not be perceived as unpleasantly loud by most people.

Figure 1 illustrates various ways in which hyperacusis might occur. The figure plots the level of a 1-kHz tone against the loudness level of that tone in phons. For a person with normal hearing the loudness level of the tone is equal to its physical level, as indicated by the solid line in **Figure 1**. A function for a person who is hypersensitive at all levels is illustrated by the short-dashed line in **Figure 1**. This might occur if transmission through the middle ear was especially efficient or the active mechanism in the cochlea was especially efficient at the test frequency. Such cases are sometimes observed, but they are not common. A more common situation associated with loudness hyperacusis is where hearing sensitivity at low levels is normal (i.e., hearing threshold levels, HTLs, are normal) but moderately intense sounds are perceived as louder than normal, as illustrated by the medium-dashed line in **Figure 1**. For this example, a tone with a level of 70 dB SPL would have a loudness level of 90 phons. In other words, the 70 dB SPL tone appears as loud for the person with hyperacusis as a 90 dB SPL tone for a person with normal hearing. Finally, the long-dashed line in **Figure 1** illustrates a case of a person with hearing loss combined with loudness recruitment; the latter is an abnormally rapid growth of loudness once the level of a sound exceeds the detection threshold [5–7]. For levels above about 83 dB SPL, the loudness for the impaired ear is greater

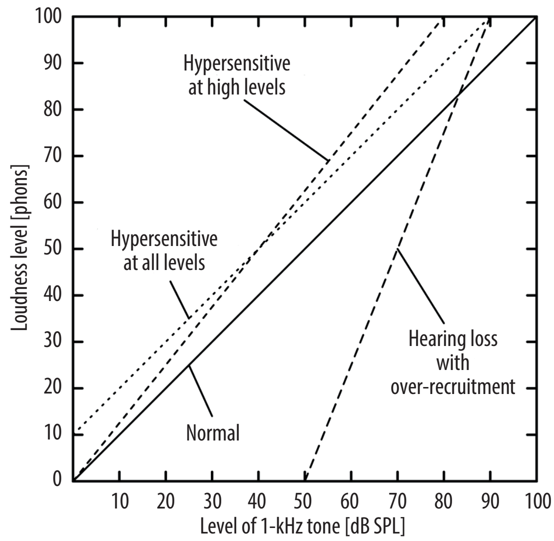

Figure 1. Illustration of various forms of loudness hyperacusis

than would be perceived by a person with normal hearing. This is sometimes called *over-recruitment*. Loudness recruitment is described in more detail in the next section.

Loudness hyperacusis can be diagnosed using a structured clinical interview, using questionnaires, such as the Hyperacusis Questionnaire (HQ) [8] or the Inventory of Hyperacusis Symptoms (IHS) [9,10], or by measurement of the level at which sounds first become uncomfortably loud [11]. Common measures of the last of these are the loudness discomfort level (LDL), the uncomfortable loudness level (ULL), and the highest comfortable level (HCL). Often, these terms are used interchangeably. The precise instructions to the person being tested are important. The British Society of Audiology [12] recommends the following instructions for determining the ULL: "I will gradually make the sound louder in your ear, and you must press the button (or raise your hand) as soon as the sound becomes uncomfortable (uncomfortably loud). This is not a test to find the loudest sound you can tolerate; it is a test to find what level of sound you find uncomfortable. You should press the button (or raise your hand) only when the sound becomes uncomfortable; but make sure you press (raise) it as soon as the sound reaches that level." In the clinic, ULLs are usually measured using sinusoidal tones presented via an audiometer, and the stimulus levels are calibrated in dB HL, rather than dB SPL.

For normal-hearing people without hyperacusis, the average ULL across the audiometric frequencies (usually from 0.5 to 4 kHz, but sometimes averaged over a wider range) usually lies between 86 and 100 dB HL [13–15]. The average ULLs reported for patients with hyperacusis vary widely across studies, from 66.3 dB HL ($SD = 15$) [16], to 77 dB HL [17], or to 83 dB HL ($SD = 17$) [15]. The differences across studies may reflect differences in the patient populations or in the exact instructions given to the patients. Aazh and Moore [18] proposed an overall measure to characterise ULLs for a given patient. ULL values were first averaged across all audiometric frequencies

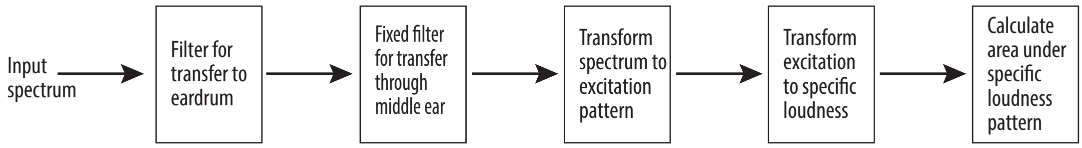

Figure 2. Block diagram of the stages of the model of loudness perception for normal hearing

(0.25, 0.5, 1, 2, 3, 4, 6, and 8 kHz), separately for each ear. The across-frequency average ULL for the ear with lower average ULL is denoted ULL_{min}; this provides an overall measure of loudness tolerance for the more sensitive ear.

The criteria for diagnosing hyperacusis handicap based on HQ scores are not generally agreed upon. Khalfa et al. [8] suggested a cutoff score of 28, Meeus et al. [19] suggested a cutoff score of 26, while Fackrell et al. [20] suggested that the cutoff score of 28 needs to be re-evaluated but did not propose a definite value. Aazh and Moore [18] showed that a diagnosis of hyperacusis handicap based on ULLs could be made consistent with a diagnosis based on HQ scores using the following cutoff values: $ULL_{min} \leq 77$ dB HL and HQ score ≥ 22. With these cutoff values, 95% of patients with ULL_{min} values meeting the criterion also met the criterion based on HQ scores, and vice versa.

Models of loudness perception

This section describes models of loudness that can predict the major features of loudness perception for people with normal hearing and people with hearing loss. The models are primarily based on simulations of the function of the peripheral auditory system. The model for normal hearing [21,22] is based on a modification of a model described by Zwicker and Scharf [23]. Its basic structure is illustrated in **Figure 2**. This version of the model is applicable only to steady sounds and uses the spectrum of the sound as its input. The model has been extended to deal with time-varying sounds [24–26], but that is beyond the scope of this paper.

The first stage of the model is a filter to account for the transfer of sound from the sound source to the eardrum (left-most box in **Figure 2**). The concha and ear canal together lead to a resonance centred near 3 kHz, which increases the level at the eardrum relative to that measured in the sound field by 12–15 dB [27,28]. This largely accounts for the fact that the absolute threshold, specified as the minimum audible field (MAF), is lowest for frequencies near 3 kHz [29].

The second stage of the model is a filter to account for the transfer of sound through the middle ear to the cochlea (second box in **Figure 2**). This transfer is most efficient for mid-range frequencies [30]. Transmission through the middle ear becomes progressively less efficient for frequencies below about 500 Hz, and this partly accounts for the fact that the MAF increases progressively as the frequency is decreased below 500 Hz. There is considerable individual variability in the efficiency of transfer of sound through the middle ear, especially at low frequencies, and this may partly account for the finding that some individuals are especially sensitive to and are bothered by low-frequency environmental sounds, such as those produced by wind generators [31].

The third stage of the model involves the transformation of the spectrum reaching the cochlea into an auditory excitation pattern (middle box in **Figure 3**). The cochlea is conceived of as containing an array of bandpass filters called the auditory filters, with centre frequencies ranging from about 50 Hz [32,33] to 17 kHz [34]. The excitation pattern is defined as the output of the auditory filters as a function of filter centre frequency [35,36], plotted with units of dB versus centre frequency, with the centre frequency transformed to the perceptually relevant ERB_N-number scale [37]. The excitation pattern can be conceived of as a smeared internal representation of the spectrum of the sound reaching the cochlea.

The fourth stage of the model involves transformation of the excitation level at each centre frequency into a quantity called *specific loudness*, which is a kind of loudness density (fourth box in **Figure 2**). Specific loudness has units sones/ERB_N, where ERB_N is the equivalent rectangular bandwidth of the auditory filter at that centre frequency for young listeners with normal hearing [36]. The transformation involves a compressive nonlinearity that simulates the compression that occurs in the cochlea and is mediated by the motor activity of the outer hair cells (OHCs) [38,39]. This is often referred to as the *active mechanism*. The active mechanism results in an amplification of the cochlear response by about 55 dB for low-level sounds, but the amplification decreases as the input level decreases [40]. As a result, when the input sound level increases from 0 to about 90 dB SPL, the response in the cochlea may increase by only 30–35 dB. It is assumed that the overall loudness of a sound is equal to the sum of the specific loudness values across centre frequencies, which is equivalent to the area under the specific loudness pattern (right-most box in **Figure 2**).

The loudness model described above has been modified so as to account for the perception of loudness for people with sensorineural hearing loss [41,42]. The models for impaired hearing are based on the assumption that the hearing loss at each frequency, HL_{TOTAL}, is the sum of the hearing loss due to reduced OHC function, HL_{OHC}, and the hearing loss due to reduced inner hair cell (IHC), synaptic and/or neural function, which for simplicity is denoted HL_{IHC}. This is expressed by the equation:

$$HL_{TOTAL} = HL_{OHC} + HL_{IHC}.$$

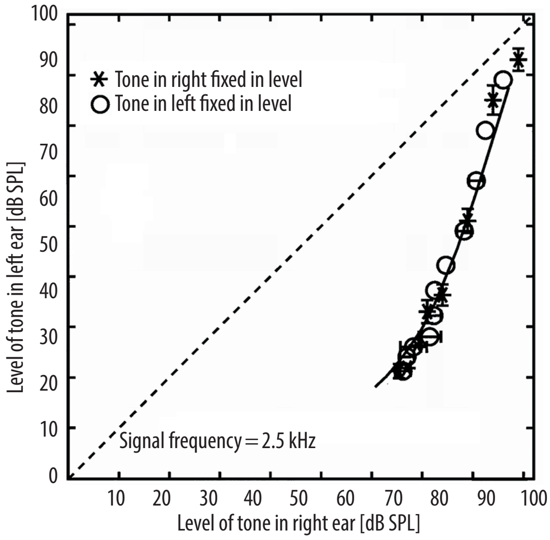

Figure 3. Results of loudness matching of tones presented in alternation to the two ears for a participant with near-normal hearing in the left ear and hearing loss in the right ear

For example, if the total hearing loss at a given frequency is 65 dB, 50 dB of that loss might be due to OHC damage and 15 dB to IHC damage. It is assumed that HL_{OHC} cannot be greater than about 55 dB. The effects of OHC damage are simulated by broadening of the auditory filters (and hence "smearing" of the excitation pattern) and by steepening the functions relating excitation level to specific loudness, both by an amount that increases with increasing HL_{OHC}. The latter mimics the effect of loss of cochlear compression produced by reduced functioning of the active mechanism [40,43]. The effects of IHC dysfunction are simulated by a simple attenuation of the excitation level.

The model can predict with good accuracy the typical effects of loudness recruitment. An example is given in **Figure 3**, which shows the results of loudness matching for tones presented alternately to the two ears via headphones for a participant with near-normal hearing in the left ear and a moderate-to-severe hearing loss in the right ear [7]. At the test frequency of 2.5 kHz the absolute threshold was 27.2 dB SPL for the left ear and 71.4 dB SPL for the right ear. Loudness matches were made with the level of the tone fixed in the right ear and the level in the left ear adjusted to achieve a loudness match (asterisks in **Figure 3**) or with the level fixed in the left ear and the level in the right ear adjusted (circles in **Figure 3**). The dashed line shows where the matches would lie if both ears were completely normal. The solid line shows the predictions of the loudness model [42]. It is clear that the model predicts the loudness matches with good accuracy. For this example, at the highest levels, equal loudness for the two ears occurred when the tones at the two ears were approximately equal in level. In other words, loudness recruitment was near "complete". If the value of HL_{OHC} for the impaired ear is set to its maximum value, the model predicts a small amount (about 5 dB) of over-recruitment; at equal high levels the sound would be louder for the impaired ear than for the normal ear. However, the magnitude of the predicted over-recruitment is small, probably too small to account for the loudness of moderate-level sounds experienced by hearing-impaired people with hyperacusis. Also, the model cannot account for the hyperacusis experienced by some people with normal audiometric thresholds. Hence, there must be factors influencing loudness perception that are not taken into account by the model. Two possible factors are next considered.

The role of the efferent system

The medial olivocochlear (MOC) efferent system is a system of projections from the superior olive to the cochlea [44]. The projections are mainly to the OHCs in the cochlea [44]. There is evidence that activation of the MOC system is driven by the responses of unmyelinated type II spiral ganglion cells in the cochlea, each of which innervates many OHCs [45]. The MOC system can be activated by a sound presented to either or both ears [46,47], and its main effect is to reduce the amplification provided by the cochlear active mechanism [48]. This reduced amplification occurs for both cochleae, even if the activating sound is presented to one ear only. The function of the efferent system is not clear [49]. It has been suggested that it reduces neural saturation effects and hence improves the ability to detect both non-speech and speech signals in the presence of background noise [50,51]. Another possibility is that the efferent system helps to protect the cochlea from the damaging effects of intense noise [52,53]. Whatever its function, it is clear that the efferent system influences the sound-evoked neural activity flowing from the cochlea to the brain, and this would be expected to influence loudness perception. The role of the efferent system is not taken into account in the loudness models described above.

To judge the loudness of sounds, the auditory system must "know" what regulatory signal is being sent to the cochlea from the MOC system at any moment. The neural activity flowing up the auditory nerve can be interpreted correctly only if the MOC signal is taken into account. Similar effects arise in other sensory systems and they are generally explained in terms of what is called *outflow theory* [54]. The idea behind outflow theory is that when a neural command/control signal is sent from the brain or brainstem to a more peripheral structure, a copy of the command/control signal is sent to the part of the brain that is concerned with interpreting signals from that peripheral structure. The copy is called a *corollary discharge* or *efference copy*. The copy is used by the brain to achieve an appropriate interpretation of the neural signals from the peripheral structure.

Outflow theory was proposed by Helmholtz [55] to explain the fact that the visual world appears stable when we move our eyes, despite movements of the image on the retina. He proposed that when the brain sends "instructions" to the eye muscles to move the eyes, an efference copy is sent to the part of the brain dealing with vision, and this copy is used to compensate for the change in retinal image produced by the eye movement. There are two key pieces of evidence supporting the outflow theory in the case of eye movements. Firstly, if the eye is moved by pressing on the eyelid with a finger and wobbling the finger, the world appears to move. This happens because the retinal image moves, but there is no corresponding efference copy, because the brain did not instruct the eye muscles to move. The second piece of evidence is that if the eye muscles are temporarily paralysed, as is sometimes done during eye surgery, then if the person tries to move their eyes, the world appears to move, even though the retinal image stays stable. This happens because the efference copy is not matched by a corresponding change of the image on the retina.

A role for the efferent system in hyperacusis was suggested by Knudson et al. [56]. They assessed what they called *sound level tolerance* (SLT) for four groups of age-matched men, all with near-normal audiometric thresholds: (1) no tinnitus/high SLT; (2) no tinnitus/low SLT; (3) tinnitus/high SLT; (4) tinnitus/low SLT. SLT was assessed by measuring ULLs and also via three questions about sound tolerance in everyday life. MOC function was assessed using the change in magnitude of distortion product otoacoustic emissions (DPOAEs) measured in one ear canal and elicited by broadband noise presented to the contralateral ear. The noise reduced the magnitude of DPOAEs for all groups, as expected, but the reduction was significantly greater for the groups with tinnitus and/or low SLT, indicating hyperresponsiveness of the MOC system compared with the group with no tinnitus/high SLT. The authors concluded that for those with low SLT, "results suggest hyperresponsiveness of the interneurons of the MOC system residing in the cochlear nucleus and/or MOC neurons themselves".

Although these results appear to suggest a role of the efferent system in hyperacusis, they are actually in the opposite direction from what might have been expected if hyperactivity of the MOC system was the only factor affecting hyperacusis. Consider a situation where a moderately intense sound activates the efferent system, and this in turn sends control signals "instructing" the cochleae to reduce the gain provided by the active mechanism (together with an efference copy). If the efferent system is hyperactive in individuals with hyperacusis, as suggested by Knudson et al. [56], the reduction in gain will be greater than expected from the efference copy. In this case, the brain would be expected to interpret the sound as being softer than normal. However, Knudson et al. pointed out that, based on their own and previous data, neural pathways involving every major division of the cochlear nucleus show hyperactivity in humans or animals with tinnitus and/or low SLT. They suggested that top–down neuromodulation drives overactivation of the auditory brain stem generally. Consistent with this, Lauer et al. [49] suggested that MOC enhancement in those with reduced SLT may simply be a byproduct of decreased central inhibition. The roles of top–down influences and central inhibition are discussed in the next section of this paper.

Another possible way in which the efferent system may be linked to hyperacusis derives from the finding that the MOC reflex is driven by type II spiral ganglion cells [45]. Type II afferents are activated when OHCs are damaged [57], and this might lead to an increase in the MOC signal to the cochlea, together with an increase in the efference copy. However, the MOC signal might have only a small effect on the signals flowing from the cochlea because of the reduced ability of the damaged OHCs to alter cochlear gain. The mismatch between the efference copy and the effect of the MOC signal might result in an increased perception of loudness – i.e., hyperacusis.

An example that is consistent with changes in loudness perception resulting from a discrepancy between the control signal and the efference copy comes from a study of patients who had undergone surgery for vestibular schwannoma (also called acoustic neuroma) on one side only [58]. The surgery resulted in deafness on the operated side, because the auditory nerve was severed. All of the patients perceived tinnitus on the deaf side, as is common in such cases. Cope et al. [58] found that that presentation of a noise to the "good" (non-operated) ear resulted in an increase of the loudness of the tinnitus heard on the deaf side, and the higher the noise level the greater was the increase in loudness. Thus, rather than masking the tinnitus, the noise in the good ear increased its loudness. Cope et al. [58] explained this result in the following way. When noise was presented to the good ear, signals from the MOC system would have been sent to the cochleae on both sides, but the signals would not have reached the cochlea on the deaf side, as the MOC system was severed at the auditory nerve level as part of the surgery (and even if the cochlea did respond, there would be no resulting signal at higher levels in the auditory system, as the auditory nerve itself was severed). The efferent control signals would have carried "instructions" to decrease the gain of the active mechanism progressively as the level of the noise in the "good" ear was increased, and efference copies of the control signals would have been sent to the brain. However, the abnormal activity in the auditory pathway that gave rise to the tinnitus was presumably not affected by the signals from the MOC system. The unchanging tinnitus signal in combination with the efference copy of the "instructions" to decrease the gain, may have resulted in the increasing loudness of the tinnitus with increasing noise level in the good ear.

There are many studies of the role of the MOC system in animals using extreme manipulations, such as severing the efferent system surgically, but few if any studies of more graded changes in function of the MOC system [49]. There have been a few studies of patients who have had the MOC system severed during a surgical procedure called vestibular neurectomy, which was used to treat severe vertigo [59,60], but these studies did not assess loudness perception and the patients in the studies had hearing loss, which complicates the interpretation of the results. Hence, the role of the MOC system in loudness perception remains uncertain. Loudness hyperacusis may sometimes occur as a result of changes in the effectiveness of the MOC system, but research is needed to establish whether and how often this is the case.

Brain plasticity and central gain

There is considerable evidence that the perception of loudness can change as a result of auditory experience [61]. In other words, there is plasticity in loudness perception. Most studies of plasticity have assessed changes in loudness perception following changes in auditory input over relatively long time periods, typically weeks or months.

For example, when an earplug is placed in one ear only, say the left, the apparent locations of sound sources initially appear shifted to the right, presumably because sounds in the right ear appear louder than sounds in the left ear. However, with extended experience of using the unilateral earplug, the perceived spatial locations of sound sources shift back towards their veridical locations [62,63]. The effects of a unilateral earplug on loudness perception and other measures related to loudness were assessed by Hutchinson et al. [64]. They measured changes in the acoustic reflex threshold (ART) and loudness perception following two weeks of use of a unilateral earplug. A lower ART means that the sound level required to activate the acoustic reflex has decreased. The ART decreased on average by 8–10 dB, with modest increases in loudness perception after 1 week but no further increases after 2 weeks of earplug use. A similar study by Munro and Blount [65] showed decreases in the ART following 1 week of use of a unilateral earplug.

A complementary approach is to increase the sound level in one ear by fitting a unilateral hearing aid. Munro and Trotter [66] tested 12 participants with symmetrical age-related hearing loss. They measured ULLs for each ear prior to and 3 years after the fitting of unilateral hearing aids. Audiometric thresholds were symmetrical both pre- and post-fitting. Mean ULL values were symmetrical before fitting. The mean ULL values increased for both ears after fitting, but the mean increase was greater for the fitted ear (mean of 2 and 4 kHz = 14.5 dB) than for the non-fitted ear (mean of 7 dB). Several studies have shown that the preferred gain of hearing aids increases with increasing experience of the aids [67] and that the perceived loudness of sounds of a given level decreases with increasing experience of hearing aids [61].

The outcomes of these studies have usually been interpreted in terms of changes in *central gain* [68–71]. The idea is that following decreases (or increases) in the neural activity flowing from the cochlea, mechanisms in the brainstem or auditory cortex apply greater (or lower) "gain" to the signal coming from the auditory nerve to achieve a form of homeostasis. Auerbach et al. [69] described how increased central gain associated with cochlear dysfunction might occur in three ways: (1) via a decrease in inhibitory synaptic responses; (2) via an increase in excitatory synaptic responses; or (3) via alterations to intrinsic neuronal excitability. They presented evidence that all three of these occur. Sustained alterations in inhibitory input following cochlear dysfunction have been demonstrated at levels of the auditory system as peripheral as the cochlear nucleus, and also at more central levels, including the inferior colliculus and auditory cortex. These changes in inhibition often involve the neurotransmitter GABA. Increases in excitatory responses following cochlear dysfunction have been found in the auditory brainstem and midbrain, and often involve the neurotransmitter glutamate. Finally, changes in intrinsic excitability following cochlear dysfunction have been demonstrated in the dorsal cochlear nucleus and pyramidal cells of the auditory cortex. It seems plausible that all three types of change may be associated with hyperacusis following cochlear dysfunction. In addition, there can be plastic changes in the tononotopic mapping of frequency to place in the auditory cortex [72].

Plastic changes in the responses of the central auditory system in response to changes in peripheral input are often referred to as *maladaptive plasticity* when they result in undesired effects such as hyperacusis and tinnitus. It is beyond the scope of this paper to give details of the physiological mechanisms underlying adaptive plasticity. For reviews, see Auerbach et al. [69] and Roberts and Salvi [70].

Consider now how adaptive plasticity would apply to a person with hearing loss combined with loudness recruitment who does not use hearing aids. Such a person hears most sounds (with low and medium levels) as softer than normal. The person may adapt to this: the central auditory system increases the gain to compensate for the reduced signal coming from the cochlea. But this increased central gain may be applied to all sounds, regardless of their level. Hence, moderately intense sounds may be perceived as unpleasantly loud; the person has *loudness hyperacusis*.

It should be noted that the auditory cortex does not just relay or passively respond to information about sound coming from lower levels in the auditory system. Rather, it interprets and assigns meaning to sounds. Altered cortical processing may amplify the emotional or discomfort response to sounds that would otherwise be considered normal. Also, the auditory cortex and brainstem neural centres interact with the limbic system, which is concerned with emotional responses. There is considerable evidence that hyperacusis is associated with increased activity in the limbic system [73–75]. This may account for the distress that can be experienced by people with hyperacusis.

It is less clear how loudness hyperacusis might arise from changes in central gain for people whose audiometric thresholds are normal. One possibility is that that there is some underlying damage that reduces the signal coming from the cochlea but does not affect audiometric thresholds. There is evidence from both animal studies [76] and human studies [77,78] that noise exposure can lead to loss of the synapses that connect the IHCs to the neurons that make up the auditory nerve. This is called *cochlear synaptopathy*. It occurs very rapidly during or after the exposure. Subsequently, the neurons in the auditory nerve may degenerate, an effect called *neuropathy*. Both synaptopathy and neuropathy also occur with increasing age [79–81]. The audiogram can remain normal or near-normal even when there is quite substantial cochlear synaptopathy/neuropathy [82]. Consistent with this, noise exposure is associated with greater measured difficulty in understanding speech in noise [78,83–86] and greater self-reported hearing difficulty [87], even when the audiogram remains within normal limits. Hearing difficulty when audiometric thresholds are normal is often called *hidden hearing loss*, but the term *hidden hearing disorder* is preferable [88] because, by definition, hearing loss is what is measured using the audiogram. Cochlear synaptopathy/neuropathy would reduce the neural signals coming from the cochlea, and this might lead to increased central gain and hence loudness hyperacusis. There is considerable evidence that noise exposure is associated with an increased incidence of loudness hyperacusis [89,90], and that this may be mediated by cochlear synaptopathy/neuropathy [91–94]. It is worth noting that synaptopathy is thought primarily to affect the synapses between IHCs and neurons with low spontaneous rates, which respond only at relatively high stimulus levels [95]. If this is the case for humans, it might explain why hyperacusis is associated with increased loudness for sounds presented at moderate and high levels, but not for sounds at low levels.

It is possible that cochlear synaptopathy/neuropathy occurs to some extent even for people with normal audiometric thresholds who have not been noise exposed. This could be the case for some people who have difficulties understanding speech in noise but have normal audiometric thresholds [96,97]. This might account for the existence of loudness hyperacusis among people with normal audiometric thresholds and no history of excessive noise exposure.

The evidence for plasticity in loudness perception has led to proposals for treatments that might reverse maladaptive plasticity, and hence might alleviate loudness hyperacusis [61]. In a recent study, Formby et al. [98] described the results of a 6-month field trial of an intervention for debilitating hyperacusis. The intervention included structured counselling, promotion of safe sound exposure, and exposure to broadband sound from sound generators. The authors stated that "This intervention is designed to overcome barriers to successful delivery of therapeutic sound as a tool to downregulate neural hyperactivity in the central auditory pathways (i.e., the maladaptive mechanism believed to account for primary hyperacusis) and, together with the counselling, reduce the associated negative emotional and physiological reactions to debilitating hyperacusis." The 12 participants had normal or near-normal audiometric thresholds and ULLs ≤ 75 dB HL at several frequencies. The low-level broadband therapeutic sound was delivered by ear-mounted devices fitted bilaterally with either occluding earpieces and output limiting (with adjustable limiting threshold called *loudness suppression*, LS) or open domes. The LS thresholds were incrementally adjusted across visits based on changes in loudness judgments for running speech. Secondary outcomes included categorical loudness judgments, speech understanding, and questionnaires to assess hyperacusis problems, quality of life, and depression. At the end of the trial there was a large mean change in LS (35 dB), indicating greater tolerance for high-level sounds. Most (11 of the 12) participants achieved a change in LS ≥ 16 dB, consistent with improvements in sound-based questionnaire scores.

While the results of this study are encouraging, the complicated nature of the intervention makes it hard to assess the relative importance of the different components, e.g., counselling versus sound generators. Also, the study did not include a control group, so some of the improvements may reflect a treatment/placebo effect.

Other studies of the use of therapeutic sounds for treatment of hyperacusis have also yielded promising but inconclusive results; for a review see Henry [99]. Clearly, further research is needed to clarify whether controlled sound exposure is effective in the treatment of hyperacusis.

Conclusions

Models of loudness perception for "typical" individuals with normal or impaired hearing, based primarily on peripheral auditory processing, can predict several aspects of loudness perception, including the loudness recruitment associated with cochlear hearing loss. However, these models do not predict the existence of loudness hyperacusis among people with normal audiometric thresholds, and they do not account for the low ULLs that can occur for some people with hearing loss. These limitations probably reflect the failure of the models to take into account central processes that may influence loudness perception. Two such processes are the operation of the efferent (MOC) system and changes in central gain following altered signals from the cochlea.

Loudness hyperacusis may occur when the MOC system fails to result in changes in cochlear gain, perhaps because of damage to the active mechanism in the cochlea. However, there is evidence that hyperacusis is associated with hyperactivity of the auditory system at many levels. The role of the efferent system in loudness perception is not fully understood. More research on this role is clearly needed.

Loudness hyperacusis may also be a result of plasticity in the brainstem or auditory cortex following a reduction of the output from the cochlea. The output from the cochlea may be reduced as a consequence of hearing loss (primarily damage to the OHCs and IHCs). It may also be reduced as a consequence of synaptopathy/neuropathy, which reduces the output of the cochlea without markedly affecting audiometric thresholds. The brain appears to adapt to the reduced signal from the cochlea by increasing the central gain, and this may lead to loudness hyperacusis.

In principle, central gain might be reduced by the use of therapeutic sounds, providing an avenue for the treatment of hyperacusis. While the results of clinical trials evaluating the effectiveness of sound therapies for hyperacusis have been encouraging, there is a dearth of well-controlled clinical trials, so the effectiveness of sound therapy requires further evaluation.

Acknowledgements

I thank two reviewers for their very helpful comments on an earlier version of this paper.

Funding

This research and article did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. ANSI. ANSI S1.1-2013. American National Standard Acoustical Terminology. New York: American National Standards Institute; 2013.
2. Stevens SS. On the psychophysical law. Psych Rev, 1957; 64(3): 153–81. https://doi.org/10.1037/h0046162
3. Hellman RP. Growth of loudness at 1000 and 3000 Hz. J Acoust Soc Am, 1976; 60(3): 672–9. https://doi.org/10.1121/1.381138
4. Tyler RS, Pienkowski M, Rojas Roncancio E, Jun HJ, Brozoski T, Dauman N, et al. A review of hyperacusis and future directions: part I. Definitions and manifestations. Am J Audiol, 2014; 23(4): 402–19. https://doi.org/10.1044/2014_AJA-14-0010
5. Fowler EP. A method for the early detection of otosclerosis. Arch Otolaryngol, 1936; 24(6): 731–41. https://doi.org/10.1001/archotol.1936.00640050746005
6. Steinberg JC, Gardner MB. The dependence of hearing impairment on sound intensity. J Acoust Soc Am, 1937; 9(1): 11–23. https://doi.org/10.1121/1.1915905
7. Moore BCJ. Testing the concept of softness imperception: loudness near threshold for hearing-impaired ears. J Acoust Soc Am, 2004; 115(6): 3103–11. https://doi.org/10.1121/1.1738839
8. Khalfa S, Dubal S, Veuillet E, Perez-Diaz F, Jouvent R, Collet L. Psychometric normalization of a hyperacusis questionnaire. ORL J Otorhinolaryngol Relat Spec, 2002; 64(6): 436–42. https://doi.org/10.1159/000067570
9. Greenberg B, Carlos M. Psychometric properties and factor structure of a new scale to measure hyperacusis: introducing the Inventory of Hyperacusis Symptoms. Ear Hear, 2018; 39(5): 1025–34. https://doi.org/10.1080/14992027.2020.1723033
10. Aazh H, Danesh A, Moore BCJ. Internal consistency and convergent validity of the Inventory of Hyperacusis Symptoms. Ear Hear, 2021; 42(4): 917–26. https://doi.org/10.1097/aud.0000000000000982
11. Fowler EP. Some attributes of "loudness recruitment" and "loudness decruitment". Ann Otol Rhinol Laryngol, 1965; 74(2): 500–6. https://doi.org/10.1177/000348946507400218
12. British Society of Audiology. Recommended procedure: determination of uncomfortable loudness levels. Reading, UK: British Society of Audiology; 2011.
13. Sherlock LP, Formby C. Estimates of loudness, loudness discomfort, and the auditory dynamic range: normative estimates, comparison of procedures, and test–retest reliability. J Am Acad Audiol, 2005; 16(2): 85–100. https://doi.org/10.3766/jaaa.16.2.4
14. Knobel KA, Sanchez TG. [Loudness discomfort level in normal hearing individuals]. Pro Fono, 2006; 18: 31–40 [in Portuguese]. https://doi.org/10.1590/s0104-56872006000100005
15. Sheldrake J, Diehl PU, Schaette R. Audiometric characteristics of hyperacusis patients. Front Neurol, 2015; 6: 105. https://doi.org/10.3389/fneur.2015.00105
16. Blaesing L, Kroener-Herwig B. Self-reported and behavioral sound avoidance in tinnitus and hyperacusis subjects, and association with anxiety ratings. Int J Audiol, 2012; 51(8): 611–7. https://doi.org/10.3109/14992027.2012.664290
17. Anari M, Axelsson A, Eliasson A, Magnusson L. Hypersensitivity to sound: questionnaire data, audiometry and classification. Scand Audiol, 1999; 28(4): 219–30. https://doi.org/10.1080/010503999424653
18. Aazh H, Moore BCJ. Factors related to uncomfortable loudness levels for patients seen in a tinnitus and hyperacusis clinic. Int J Audiol, 2017; 56(10): 793–800. https://doi.org/10.1080/14992027.2017.1335888
19. Meeus OM, Spaepen M, Ridder DD, Heyning PH. Correlation between hyperacusis measurements in daily ENT practice. Int J Audiol, 2010; 49(1): 7–13. https://doi.org/10.3109/14992020903160868
20. Fackrell K, Fearnley C, Hoare DJ, Sereda M. Hyperacusis Questionnaire as a tool for measuring hypersensitivity to sound in a tinnitus research population. Biomed Res Int, 2015; 2015: 290425. https://doi.org/10.1155/2015/290425
21. Moore BCJ, Glasberg BR. A revision of Zwicker's loudness model. Acta Acust United Acust, 1996; 82(2): 335–45.
22. Moore BCJ, Glasberg BR, Baer T. A model for the prediction of thresholds, loudness and partial loudness. J Audio Eng Soc, 1997; 45: 224–40.
23. Zwicker E, Scharf B. A model of loudness summation. Psych Rev, 1965; 72(1): 3–26. https://doi.org/10.1037/h0021703
24. Glasberg BR, Moore BCJ. A model of loudness applicable to time-varying sounds. J Audio Eng Soc, 2002; 50(5): 331–42.
25. Moore BCJ, Glasberg BR, Varathanathan A, Schlittenlacher J. A loudness model for time varying sounds incorporating binaural inhibition. Trends Hear, 2016; 20: 1–16. https://doi.org/10.1177/2331216516682698
26. Moore BCJ, Jervis M, Harries L, Schlittenlacher J. Testing and refining a loudness model for time-varying sounds incorporating binaural inhibition. J Acoust Soc Am, 2018; 143(3): 1504–13. https://doi.org/10.1121/1.5027246
27. Shaw EAG. Transformation of sound pressure level from the free field to the eardrum in the horizontal plane. J Acoust Soc Am, 1974; 56(6): 1848–61. https://doi.org/10.1121/1.1903522
28. Killion MC, Berger EH, Nuss RA. Diffuse field response of the ear. J Acoust Soc Am, 1987; 81: S75. https://doi.org/10.1121/1.2024388
29. ISO 7029. Acoustics: Statistical distribution of hearing thresholds related to age and gender. Geneva: International Organization for Standardization; 2017.
30. Aibara R, Welsh JT, Puria S, Goode RL. Human middle-ear sound transfer function and cochlear input impedance. Hear Res, 2001; 152(1–2): 100–9. https://doi.org/10.1016/S0378-5955(00)00240-9
31. Pedersen CS, Møller H, Waye KP. A detailed study of low-frequency noise complaints. J Low Freq Noise Vib Active Cont, 2008; 27: 1–33. https://doi.org/10.1260/026309208784425505
32. Jurado C, Moore BCJ. Frequency selectivity for frequencies below 100 Hz: comparisons with mid-frequencies. J Acoust Soc Am, 2010; 128(6): 3585–96. https://doi.org/10.1121/1.3504657
33. Jurado C, Pedersen CS, Moore BCJ. Psychophysical tuning curves for frequencies below 100 Hz. J Acoust Soc Am, 2011; 129(5): 3166–80. https://doi.org/10.1121/1.3560535
34. Yasin I, Plack CJ. Psychophysical tuning curves at very high frequencies. J Acoust Soc Am, 2005; 118(4): 2498–506. https://doi.org/10.1121/1.2035594
35. Moore BCJ, Glasberg BR. Suggested formulae for calculating auditory-filter bandwidths and excitation patterns. J Acoust Soc Am, 1983; 74(3): 750–3. https://doi.org/10.1121/1.4955005
36. Glasberg BR, Moore BCJ. Derivation of auditory filter shapes from notched-noise data. Hear Res, 1990; 47(1–2): 103–38. https://doi.org/10.1016/0378-5955(90)90170-T
37. Moore BCJ. An Introduction to the Psychology of Hearing, 6th Ed. Leiden. The Netherlands: Brill; 2012.
38. Ashmore JF. A fast motile response in guinea pig outer hair cells: the cellular basis of the cochlear amplifier. J Physiol, 1987; 388: 323–47. https://doi.org/10.1113/jphysiol.1987.sp016617

39. Hudspeth A. Mechanical amplification of stimuli by hair cells. Curr Opin Neurobiol, 1997; 7(4): 480–6. https://doi.org/10.1016/s0959-4388(97)80026-8
40. Ruggero MA. Responses to sound of the basilar membrane of the mammalian cochlea. Curr Opin Neurobiol, 1992; 2(4): 449–56. https://doi.org/10.1016/0959-4388(92)90179-o
41. Moore BCJ, Glasberg BR. A model of loudness perception applied to cochlear hearing loss. Br J Audiol, 1997; 3(3): 289–311.
42. Moore BCJ, Glasberg BR. A revised model of loudness perception applied to cochlear hearing loss. Hear Res, 2004; 188(1–2): 70–88. https://doi.org/10.1016/S0378-5955(03)00347-2
43. Dallos P. The active cochlea. J Neurosci, 1992; 12(12): 4575–85.
44. Guinan JJ Jr, Backus BC, Lilaonitkul W, Aharonson V. Medial olivocochlear efferent reflex in humans: otoacoustic emission (OAE) measurement issues and the advantages of stimulus frequency OAEs. J Assoc Res Otolaryngol, 2003; 4(4): 521–40. https://doi.org/10.1007/s10162-461002-3037-3
45. Froud KE, Wong AC, Cederholm JM, Klugmann M, Sandow SL, Julien JP, et al. Type II spiral ganglion afferent neurons drive medial olivocochlear reflex suppression of the cochlear amplifier. Nature Commun, 2015; 6: 7115. https://doi.org/10.1038/ncomms8115
46. Guinan JJ Jr. Olivocochlear efferents: their action, effects, measurement and uses, and the impact of the new conception of cochlear mechanical responses. Hear Res, 2018; 362: 38–47. https://doi.org/10.1016/j.heares.2017.12.012
47. Wicher A, Moore BCJ. Effect of broadband and narrowband contralateral noise on psychophysical tuning curves and otoacoustic emissions. J Acoust Soc Am, 2014; 135(5): 2931–41. https://doi.org/10.1121/1.4871358
48. Liberman MC, Guinan JJ Jr. Feedback control of the auditory periphery: anti-masking effects of middle ear muscles vs. olivocochlear efferents. J Commun Disord, 1998; 31(6): 471–82. https://doi.org/10.1016/s0021-9924(98)00019-7
49. Lauer AM, Jimenez SV, Delano PH. Olivocochlear efferent effects on perception and behavior. Hear Res, 2022; 419: 108207. https://doi.org/10.1016/j.heares.2021.108207
50. Micheyl C, Collet L. Involvement of the olivocochlear bundle in the detection of tones in noise. J Acoust Soc Am, 1996; 99(3): 1604–10. https://doi.org/10.1121/1.414734
51. Giraud AL, Garnier S, Micheyl C, Lina G, Chays A, Chery-Croze S. Auditory efferents involved in speech-in-noise intelligibility. Neuroreport, 1997; 8(7): 1779–83. https://doi.org/10.1097/00001756-199705060-00042
52. Maison SF, Liberman MC. Predicting vulnerability to acoustic injury with a noninvasive assay of olivocochlear reflex strength. J Neurosci, 2000; 20(12): 4701–7. https://doi.org/10.1523/jneurosci.20-12-04701.2000
53. Maison SF, Luebke AE, Liberman MC, Zuo J. Efferent protection from acoustic injury is mediated via alpha9 nicotinic acetylcholine receptors on outer hair cells. J Neurosci, 2002; 22(24): 10838–46. https://doi.org/10.1523/jneurosci.22-24-10838.2002
54. Sherrington CS. Further note on the sensory nerves of the eye-muscles. Proc Roy Soc, 1898; 64(402–411): 120–1. https://doi.org/10.1098/rspl.1898.0075
55. Helmholtz HLF. [A treatise on physiological optics]. Leipzig: Voss; 1863 [in German].
56. Knudson IM, Shera CA, Melcher JR. Increased contralateral suppression of otoacoustic emissions indicates a hyperresponsive medial olivocochlear system in humans with tinnitus and hyperacusis. J Neurophysiol, 2014; 112(12): 3197–208. https://doi.org/10.1152/jn.00576.2014
57. Liu C, Glowatzki E, Fuchs PA. Unmyelinated type II afferent neurons report cochlear damage. Proc Natl Acad Sci USA, 2015; 112(47): 14723–7. https://doi.org/10.1073/pnas.1515228112
58. Cope T, Baguley DM, Moore BCJ. Tinnitus loudness in quiet and noise after resection of vestibular schwannoma. Otol Neurotol, 2011; 32(3): 488–96. https://doi.org/10.1097/MAO.0b013e3182096b02
59. Scharf B, Magnan J, Chays A. On the role of the olivocochlear bundle in hearing: 16 case studies. Hear Res, 1997; 103(1–2): 101–22. https://doi.org/10.1016/s0378-5955(96)00168-2
60. Zeng FG, Martino KM, Linthicum FH, Soli SD. Auditory perception in vestibular neurectomy subjects. Hear Res, 2000; 142(1–2): 102–12. https://doi.org/10.1016/s0378-5955(00)00011-3
61. Formby C, Sherlock LP, Gold SL. Adaptive plasticity of loudness induced by chronic attenuation and enhancement of the acoustic background. J Acoust Soc Am, 2003; 114(1): 55–8. https://doi.org/10.1121/1.1582860
62. Florentine M. Relation between lateralization and loudness in asymmetrical hearing losses. J Am Audiol Soc, 1976; 1(6): 243–51.
63. Butler RA. An analysis of the monaural displacement of sound in space. Percept Psychophys, 1987; 41(1): 1–7. https://doi.org/10.3758/bf03208206
64. Hutchison P, Maeda H, Formby C, Small BJ, Eddins DA, Eddins AC. Acoustic deprivation modulates central gain in human auditory brainstem and cortex. Hear Res, 2023; 428: 108683. https://doi.org/10.1016/j.heares.2022.108683
65. Munro KJ, Blount J. Adaptive plasticity in brainstem of adult listeners following earplug-induced deprivation. J Acoust Soc Am, 2009; 126(2): 568–71. https://doi.org/10.1121/1.3161829
66. Munro KJ, Trotter JH. Preliminary evidence of asymmetry in uncomfortable loudness levels after unilateral hearing aid experience: evidence of functional plasticity in the adult auditory system. Int J Audiol, 2006; 45(12): 684–8. https://doi.org/10.1080/14992020600640444
67. Keidser G, O'Brien A, Carter L, McLelland M, Yeend I. Variation in preferred gain with experience for hearing-aid users. Int J Audiol, 2008; 47(10): 621–35. https://doi.org/10.1080/14992020802178722
68. Salvi RJ, Wang J, Ding D. Auditory plasticity and hyperactivity following cochlear damage. Hear Res, 2000; 147(1–2): 261–74. https://doi.org/10.1016/s0378-5955(00)00136-2
69. Auerbach BD, Rodrigues PV, Salvi RJ. Central gain control in tinnitus and hyperacusis. Front Neurol, 2014; 5: 206. https://doi.org/10.3389/fneur.2014.00206
70. Roberts LE, Salvi R. Overview: hearing loss, tinnitus, hyperacusis, and the role of central gain. Neuroscience, 2019; 407: 1–7. https://doi.org/10.1016/j.neuroscience.2019.03.021
71. Zeng FG. Tinnitus and hyperacusis: central noise, gain and variance. Curr Opin Physiol, 2020; 18: 123–9. https://doi.org/10.1016/j.cophys.2020.10.009
72. Wake N, Shiramatsu TI, Takahashi H. Map plasticity following noise exposure in auditory cortex of rats: implications for disentangling neural correlates of tinnitus and hyperacusis. Front Neurosci, 2024; 18: 1385942. https://doi.org/10.3389/fnins.2024.1385942
73. Jastreboff PJ, Jastreboff MM. Tinnitus Retraining Therapy (TRT) as a method for treatment of tinnitus and hyperacusis patients. J Am Acad Audiol, 2000; 11(3): 162–77. https://doi.org/10.1055/s-0042-1748042

74. Chen YC, Li X, Liu L, Wang J, Lu CQ, Yang M, et al. Tinnitus and hyperacusis involve hyperactivity and enhanced connectivity in auditory–limbic–arousal–cerebellar network. Elife, 2015; 4: e06576. https://doi.org/10.7554/eLife.06576
75. Shi L, Zhao R, Li X, Sun W, Liu X. A review of the neurobiological mechanisms that distinguish between loudness recruitment and hyperacusis. Med Sci Monit, 2022; 28: e936373. https://doi.org/10.12659/msm.936373
76. Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. J Neurosci, 2009; 29(45): 14077–85. https://doi.org/10.1523/jneurosci.2845-09.2009
77. Stamper GC, Johnson TA. Auditory function in normal-hearing, noise-exposed human ears. Ear Hear, 2015; 36(2): 172–84. https://doi.org/10.1097/AUD.0000000000000107
78. Liberman MC, Epstein MJ, Cleveland SS, Wang H, Maison SF. Toward a differential diagnosis of hidden hearing loss in humans. PLoS One, 2016; 11(9): e0162726. https://doi.org/10.1371/journal.pone.0162726
79. Wu PZ, Liberman LD, Bennett K, de Gruttola V, O'Malley JT, Liberman MC. Primary neural degeneration in the human cochlea: evidence for hidden hearing loss in the aging ear. Neuroscience, 2019; 407: 8–20. https://doi.org/10.1016/j.neuroscience.2018.07.053
80. Wu PZ, O'Malley JT, de Gruttola V, Liberman MC. Age-related hearing loss is dominated by damage to inner ear sensory cells, not the cellular battery that powers them. J Neurosci, 2020; 40(33): 6357–66. https://doi.org/10.1523/jneurosci.0937-20.2020
81. Wu PZ, O'Malley JT, Liberman MC. Neural degeneration in normal-aging human cochleas: machine-learning counts and 3D mapping in archival sections. J Assoc Res Otolaryngol, 2023; 24(5): 499–511. https://doi.org/10.1007/s10162-023-00909-y
82. Sergeyenko Y, Lall K, Liberman MC, Kujawa SG. Age-related cochlear synaptopathy: an early-onset contributor to auditory functional decline. J Neurosci, 2013; 33(34): 13686–94. https://doi.org/10.1523/jneurosci.1783-13.2013
83. Alvord LS. Cochlear dysfunction in "normal-hearing" patients with history of noise exposure. Ear Hear, 1983; 4(5): 247–50. https://doi.org/10.1097/00003446-198309000-00005
84. Hope AJ, Luxon LM, Bamiou DE. Effects of chronic noise exposure on speech-in-noise perception in the presence of normal audiometry. J Laryngol Otol, 2013; 127(3): 233–8. https://doi.org/10.1017/S002221511200299X
85. Holtegaard P, Olsen SO. Signs of noise-induced neural degeneration in humans. In: Individual Hearing Loss: Characterization, modelling, compensation strategies. Santurette S, Dau T, Dalsgaard JC, Tranebjærg L, Anderson T, Editors. Ballerup, Denmark: The Danavox Jubilee Foundation; 2016, pp. 117–24.
86. Bramhall NF, McMillan GP. Perceptual consequences of cochlear deafferentation in humans. Trends Hear, 2024; 28: 23312165241239541. https://doi.org/10.1177/23312165241239541
87. Tremblay KL, Pinto A, Fischer ME, Klein BE, Klein R, Levy S, et al. Self-reported hearing difficulties among adults with normal audiograms: The Beaver Dam Offspring Study. Ear Hear, 2015; 36(6): e290–9. https://doi.org/10.1097/AUD.0000000000000195
88. Moore BCJ, Sęk A, Vinay, Füllgrabe C. Envelope regularity discrimination. J Acoust Soc Am, 2019; 145(5): 2861–70. https://doi.org/10.1121/1.5100620
89. Lowe D, Moore BCJ. Audiometric assessment of hearing loss sustained during military service. J Acoust Soc Am, 2021; 150(2): 1030–43. https://doi.org/10.1121/10.0005846
90. Fredriksson S, Hussain-Alkhateeb L, Torén K, Sjöström M, Selander J, Gustavsson P, et al. The impact of occupational noise exposure on hyperacusis: a longitudinal population study of female workers in Sweden. Ear Hear, 2022; 43(4): 1366–77. https://doi.org/10.1097/aud.0000000000001194
91. Hickox AE, Liberman MC. Is noise-induced cochlear neuropathy key to the generation of hyperacusis or tinnitus? J Neurophysiol, 2014; 111(3): 552–64. https://doi.org/10.1152/jn.00184.2013
92. Liberman MC, Kujawa SG. Cochlear synaptopathy in acquired sensorineural hearing loss: manifestations and mechanisms. Hear Res, 2017; 349: 138–47. https://doi.org/10.1016/j.heares.2017.01.003
93. Jahn KN, Hancock KE, Maison SF, Polley DB. Estimated cochlear neural degeneration is associated with loudness hypersensitivity in individuals with normal audiograms. JASA Express Lett, 2022; 2(6): 064403. https://doi.org/10.1121/10.0011694
94. McGill M, Hight AE, Watanabe YL, Parthasarathy A, Cai D, Clayton K, et al. Neural signatures of auditory hypersensitivity following acoustic trauma. eLife, 2022; 11: e80015. https://doi.org/10.7554/eLife.80015
95. Furman AC, Kujawa SG, Liberman MC. Noise-induced cochlear neuropathy is selective for fibers with low spontaneous rates. J Neurophysiol, 2013; 110(3): 577–86. https://doi.org/10.1152/jn.00164.2013
96. Parthasarathy A, Hancock KE, Bennett K, DeGruttola V, Polley DB. Bottom-up and top-down neural signatures of disordered multi-talker speech perception in adults with normal hearing. Elife, 2020; 9: e51419. https://doi.org/10.7554/eLife.51419
97. Schirmer J, Wolpert S, Dapper K, Rühle M, Wertz J, Wouters M, et al. Neural adaptation at stimulus onset and speed of neural processing as critical contributors to speech comprehension independent of hearing threshold or age. J Clin Med, 2024; 13(9): 2725. https://doi.org/10.3390/jcm13092725
98. Formby C, Cherri D, Secor CA, Armstrong S, Juneau R, Hutchison P, et al. Results of a 6-month field trial of a transitional intervention for debilitating hyperacusis. J Speech Lang Hear Res, 2024; 67(6): 1903–31. https://doi.org/10.1044/2024_jslhr-23-00360
99. Henry JA. Sound therapy to reduce auditory gain for hyperacusis and tinnitus. Am J Audiol, 2022; 31(4): 1067–77. https://doi.org/10.1044/2022_AJA-22-00127

Brian C. J. Moore, email: bcjm@cam.ac.uk • iD 0000-0001-7071-0671