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MIDDLE EAR MUSCLE HYPERACTIVITY AS ONE CAUSE OF TINNITUS: A PUTATIVE ROLE FOR MIDDLE EAR MECHANORECEPTORS

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Abstract

Subjective tinnitus is common yet poorly understood, and existing models do not fully explain its clinical diversity. This article presents a mechanistic hypothesis, based on existing literature, in which tinnitus may emerge not only from reduced auditory input due to inner ear injury but also from increased activity of the middle ear muscles, which may ultimately increase extracochlear, largely somatosensory, input to the cochlear nucleus. In this framework, auditory deafferentation is necessary but insufficient on its own.

We further hypothesise that, in some clinical contexts, somatosensory drive may be amplified by excessive stimulation of putative middle ear mechanoreceptors (MeRs). This stimulation may originate from muscle vibration (tremor) within the tympanic cavity, associated with heightened activity of the stapedius, tensor tympani, and tensor veli palatini muscles. Clinical situations such as loud-noise exposure, temporomandibular disorders, cervical dysfunction, and ear canal irritation may be interpreted within this framework as diverse triggers converging on enhanced middle ear muscle activity and mechanosensory overstimulation.

Although hypothetical, the model yields testable predictions. Functional neuroimaging could reveal abnormal activation of somatosensory cortical areas in tinnitus not directly related to acoustic trauma; middle ear muscle activity could be assessed in animal models using electromyography; and refined impedance-based measurements may help detect and characterise middle ear vibratory phenomena. Clinically, inter-individual variability in the stapedius reflex should modulate transient tinnitus and aural fullness after exposure to loud noise. By integrating auditory deafferentation, somatosensory modulation, and middle ear biomechanics, this hypothesis aims to guide future experimental work and therapeutic strategies.

Keywords: tinnitus • middle ear muscles • cochlear nucleus • mechanoreceptors

NADAKTYWNOŚĆ MIĘŚNI UCHA ŚRODKOWEGO JAKO JEDNA Z PRZYCZYN SZUMÓW USZNYCH: HIPOTETYCZNA ROLA MECHANORECEPTORÓW UCHA ŚRODKOWEGO

Streszczenie

Subiektywne szumy uszne (*tinnitus*) są powszechnym, lecz wciąż słabo poznanym zjawiskiem, a istniejące modele patofizjologiczne nie wyjaśniają w pełni ich różnorodności klinicznej. W niniejszej pracy przedstawiono hipotezę dotyczącą mechanizmu powstawania szumów usznych, opartą na publikacjach naukowych, zgodnie z którymi szumy uszne mogą powstawać nie tylko jako skutek ograniczenia dopływu informacji słuchowych, spowodowanego uszkodzeniem ucha wewnętrznego, lecz również z powodu zwiększonej aktywności mięśni ucha środkowego. Ta ostatnia może prowadzić do nasilenia pozaślimakowego napływu bodźców do jądra ślimakowego, w szczególności somatosensorycznego. W ujęciu proponowanego modelu ograniczenie dopływu bodźców słuchowych do ośrodkowego układu nerwowego stanowi warunek konieczny, lecz niewystarczający do powstania szumów usznych.

Ponadto wysuwamy hipotezę, że w niektórych sytuacjach klinicznych wzmocnienie somatosensoryczne może się zwiększyć wskutek nadmiernej stymulacji mechanoreceptorów znajdujących się w uchu środkowym (MeRs). Źródłem tej stymulacji może być mimowolne drżenie mięśniowe (*tremor*) w jamie bębenkowej związane ze wzmożoną aktywnością mięśnia strzemiączkowego (*stapedius*), mięśnia napinacza błony bębenkowej (*tensor tympani*) oraz mięśnia napinacza podniebienia miękkiego (*tensor veli palatini*). W ramach tego modelu uwarunkowania kliniczne, takie jak: ekspozycja na hałas o dużym natężeniu, zaburzenia czynności stawu skroniowo-żuchwowego, dysfunkcje odcinka szyjnego kręgosłupa czy podrażnienie przewodu słuchowego zewnętrznego, mogą być interpretowane jako czynniki wyzwalające, które prowadzą do zwiększonej aktywności mięśni ucha środkowego i nadmiernej stymulacji układu somatosensorycznego.

Przedstawiony model ma charakter hipotetyczny, jednak pozwala na sformułowanie szeregu wniosków możliwych do zweryfikowania eksperymentalnie. Funkcjonalne badania neuroobrazowe mogłyby ujawnić nieprawidłową aktywację korowych obszarów somatosensorycznych

u pacjentów z szumami usznymi niezwiązanymi bezpośrednio z urazem akustycznym. Aktywność mięśni ucha środkowego mogłaby być oceniana na podstawie modeli zwierzęcych z wykorzystaniem elektromiografii, natomiast udoskonalone metody pomiaru impedancji akustycznej mogłyby umożliwić wykrywanie i charakterystykę zjawisk wibracyjnych zachodzących w uchu środkowym. Z klinicznego punktu widzenia osobnicza zmienność odruchu mięśnia strzemiączkowego powinna wpływać na występowanie przemijających szumów usznych oraz uczucia pełności w uchu po ekspozycji na hałas o wysokim natężeniu.

Ujmując łącznie zjawiska ograniczenia dopływu bodźców słuchowych do wyższych pięter układu nerwowego, modulacji somatosensorycznej oraz biomechaniki ucha środkowego, przedstawiona hipoteza ma na celu wskazanie kierunków przyszłych badań eksperymentalnych i rozwoju nowych strategii terapeutycznych.

Słowa kluczowe: szumy uszne • mięśnie ucha środkowego • jądro ślimakowe • mechanoreceptory

Key to abbreviations	
BA	Brodmann area
CI	cochlear input
CN	cochlear nucleus
DCN	dorsal cochlear nucleus
ECI	extracochlear input
HRMEMS	Hyper-Reactive Middle Ear Muscle Syndrome
LTP	long-term potentiation
MeRs	middle ear mechanoreceptors
OHCs	outer hair cells
PCs	Pacinian corpuscles
SM	stapedius muscle
STDP	spike-timing-dependent plasticity
TMDs	temporomandibular disorders
TTM	tensor tympani muscle
TTTS	Tonic Tensor Tympani Syndrome
TVPM	tensor veli palatini muscle
VGLUT2	vesicular glutamate transporter 2

Introduction

Subjective tinnitus, defined as an auditory perception occurring in the absence of any acoustic stimulation, is a frequent complaint in clinical practice. Its high prevalence in the general population and its potential to markedly impair quality of life [1] underline the need for mechanistic models that can account for its clinical diversity.

Cochlear deafferentation is strongly associated with tinnitus [2,3], yet it appears to be necessary but insufficient. Hearing loss on standard audiometry does not invariably coincide with tinnitus [4], experimental cochlear lesions do not consistently generate tinnitus-like percepts in animal models [5], and tinnitus may occur despite a normal audiogram, consistent with subclinical cochlear damage [6]. Accordingly, although tinnitus secondary to acute or chronic acoustic trauma is the most common clinical presentation, cochlear injury alone does not fully explain why tinnitus develops in some cases and not in others.

Clinical observations suggest that additional, non-auditory factors may influence tinnitus emergence. Tinnitus may occur without obvious noise trauma, as in temporomandibular disorders (TMDs) [7], and may also resolve after removal of a cerumen plug [8]. These observations are difficult to reconcile with a purely cochlear account and instead suggest that clinically diverse triggers may converge on a common pathway. The proposed pathway is summarized in **Figure 1**.

Here, we propose an integrative mechanistic hypothesis, grounded in existing literature. In this framework, cochlear inputs (CIs) refer to auditory information conveyed from the cochlea to the cochlear nucleus (CN) through the auditory nerve. Tinnitus may emerge not only from a reduction in these inputs following inner ear injury, but also from increased activity of the middle ear muscles. More specifically, heightened activity of the stapedius (SM), tensor tympani (TTM), and tensor veli palatini (TVPM) may generate tremor-like vibrations within the tympanic cavity, overstimulating putative middle ear mechanoreceptors (MeRs) and thereby increasing extracochlear, largely somatosensory, inputs (ECIs) to the CN. Cochlear deafferentation, potentially subclinical, is therefore viewed as permissive but insufficient on its own, while abnormal middle ear muscle activity may provide the additional drive required to shift CN processing toward tinnitus-related activity.

The goal of this framework is not to provide confirmatory evidence, but to generate falsifiable predictions and identify experimental approaches to test whether this pathway contributes to subjective tinnitus across etiologies.

Linking middle ear afferents to extracochlear pathways

To assess how middle ear activity might increase ECIs, the key question is whether mechanosensory signals arising from the tympanic membrane, middle ear cavity, or middle ear muscles can reach pathways known to provide extracochlear input to the CN. The tympanic membrane receives a triple innervation [9,10], predominantly via the auriculotemporal nerve (branch of the mandibular division of the trigeminal nerve), with additional contributions from the auricular branch of the vagus nerve and the tympanic plexus, formed by anterior and posterior tympanic branches of the glossopharyngeal nerve, together with sympathetic fibers from the superior and inferior caroticotympanic nerves. Nociception from the tympanic membrane is mediated almost exclusively by the trigeminal nerve [11]. However, global tympanic membrane

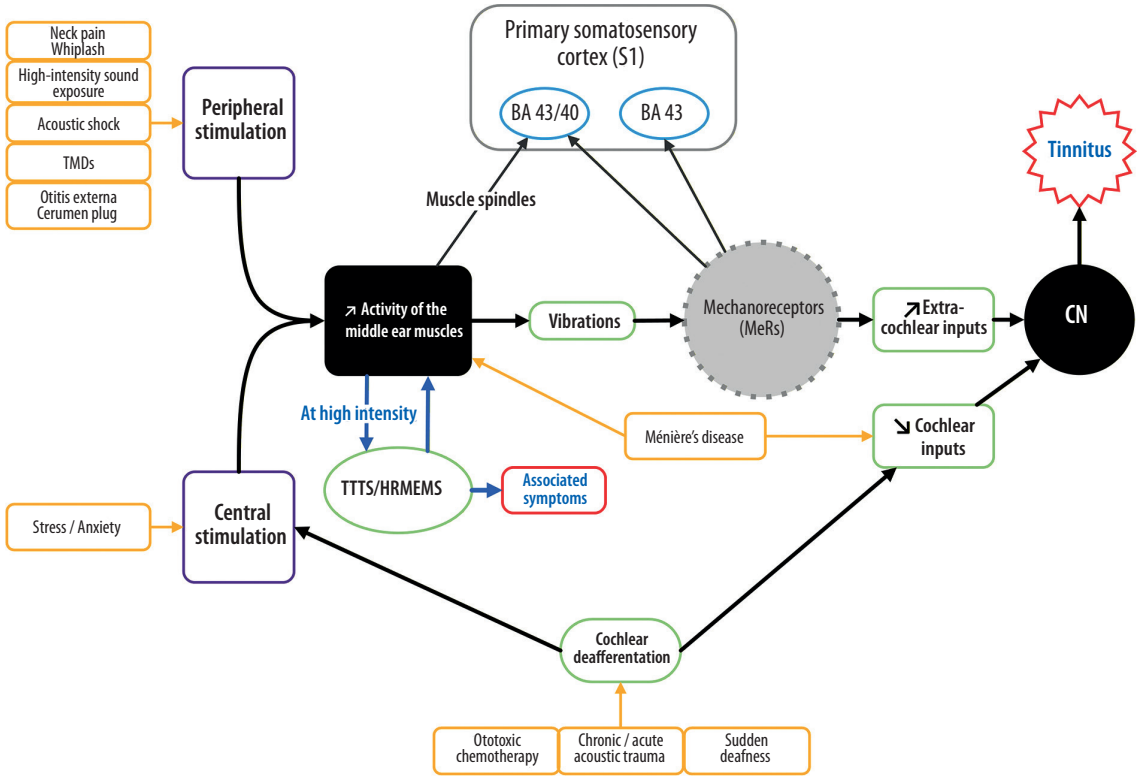

Figure 1. Proposed model linking middle ear muscle hyperactivity to tinnitus via the cochlear nucleus. The diagram summarizes the proposed pathway by which increased activity of the middle ear muscles may contribute to tinnitus. The black shapes indicate key components of the model, including the middle ear muscles and the cochlear nucleus (CN). Tremor-like activity of the stapedius, tensor tympani, and tensor veli palatini muscles may generate vibrations within the tympanic cavity and overstimulate putative middle ear mechanoreceptors (MeRs), shown in the grey circle. This mechanosensory drive may increase extracochlear inputs to the CN, where it may interact with reduced cochlear inputs resulting from cochlear deafferentation. Their convergence may contribute to abnormal CN activity and tinnitus. The same middle ear somatosensory drive may be associated with activation of the primary somatosensory cortex (S1; BA 43 and BA 43/40). At higher levels of middle ear muscle activation, this process may also contribute to Hyper-Reactive Middle Ear Muscle Syndrome (HRMEMS), including cases historically described as Tonic Tensor Tympani Syndrome (TTTS). Etiologies highlighted in yellow, such as acute or chronic acoustic trauma, temporomandibular disorders (TMDs), cervical dysfunction, otitis externa, or Ménière's disease, may act peripherally or centrally to increase middle ear muscle activity, either directly or through cochlear deafferentation, and thereby promote overstimulation of MeRs

displacement induced by moderate pressure changes is typically painless [12], suggesting that tympanic membrane displacement may generate a mechanosensory signal without necessarily engaging a nociceptive response. The mucosa of the middle ear cavity is likewise supplied by the tympanic plexus.

Afferent projections from the tympanic membrane and middle ear mucosa have been traced to trigeminal, facial, glossopharyngeal, vagal, and upper cervical ganglia [13,14]. Centrally, trigeminal thermonociceptive afferents relay through the spinal trigeminal nucleus, whereas discriminative touch is relayed to the principal sensory nucleus of V [15]. General somatic afferents from the facial, glossopharyngeal, and vagal nerves also converge on the spinal trigeminal nucleus. Upper cervical afferents relay through the dorsal column nuclei, including the cuneate nucleus, and can also influence trigeminal and vestibular nuclei [16,17]. Among these pathways, the spinal trigeminal nucleus appears particularly relevant for the present model because it is the only trigeminal nucleus known to contribute directly to ECIs. Upper cervical pathways may likewise be relevant through their convergence with trigeminal and vestibular systems. In light of currently identified sources of ECI to the CN, the most plausible routes by which middle ear signals could access known extracochlear pathways involve the spinal trigeminal nucleus, the C2 dorsal root ganglion, the cuneate nucleus, the trigeminal ganglion, and the vestibular nuclei [18–21].

The SM is innervated by the stapedius nerve, a branch of the facial nerve, whereas the TTM and TVPM are innervated by the medial pterygoid nerve, a branch of the mandibular division of the trigeminal nerve [22]. Muscle spindle afferents from the TTM and TVPM project to the mesencephalic trigeminal nucleus, and a similar pathway has been suggested for the SM [23,24]. No Golgi tendon organs have been identified in middle ear muscles. Because no direct connections have been identified between the CN and the mesencephalic or motor trigeminal nuclei, a dominant proprioceptive route from middle ear muscle spindles appears less likely, although indirect

influences cannot be excluded. Thermal sensitivity is likewise unlikely to play a major role. Clinically, even severely painful acute otitis media does not necessarily produce tinnitus. Taken together, these observations support mechanosensory input arising from the tympanic membrane or middle ear cavity as a plausible route by which middle ear activity could access known ECI pathways and thereby increase ECI drive.

Which muscles respond to what, and why this matters

Somatic maneuvers capable of engaging middle ear muscles have been associated with changes in tinnitus loudness, typically in the direction of worsening [25]. In the present framework, this observation is important because it links tinnitus modulation to stimuli known to activate the middle ear muscles. The respective roles of these muscles in response to different stimuli remain incompletely understood, but several observations indicate that they can be activated by a wide range of acoustic and non-acoustic events. In humans, intense sound stimulation elicits the acoustic reflex, that is, a tonic bilateral contraction of the SM [26]. In contrast with common animal models of tinnitus, where both the SM and TTM may be involved, the human acoustic reflex is predominantly stapedius-mediated [27]. At very high sound intensities, however, both the TTM and SM may contract, and this response has been linked to the startle reflex, although this interpretation is not universally accepted [28–30]. The TTM generally requires higher sound intensities, is more exhaustible, and has a longer latency than the SM [28–31]. The TVPM may also participate in this broader response. Although it lies outside the tympanic cavity, it affects middle ear mechanics through Eustachian tube opening and has been proposed to function as a unit with the TTM [32–34]. Startle-related impedance changes can still be observed in patients lacking a functional TTM, likely reflecting contraction of the TVPM [28]. Because the startle reflex is influenced by vigilance and fatigue, the associated middle ear muscle response may be state-dependent [28].

Middle ear muscle activity can also be triggered by non-acoustic events. Contractions of the TTM and SM have been reported during vocalisation, coughing, yawning, swallowing, laughter, smiling, clenching the teeth, head flexion, and head rotation [25,30,31,35,36]. Cutaneous stimulation of the external auditory canal or adjacent skin can provoke a phasic SM contraction and, in some studies, TTM contraction as well, likely via startle-related mechanisms [30,37]. The external auditory canal and meatus appear to be among the most sensitive sites for this response [37,38], a point relevant to the later discussion of ear canal irritation and cerumen impaction. Electrical stimulation of the tongue and air puffs directed toward the orbital region can also activate the TTM [29,32,38]. In addition, some individuals are able to voluntarily contract the TTM, suggesting a degree of cortical control [38,39].

These observations support a key point of the present model: if putative MeRs contribute to tinnitus, the most likely endogenous source of the relevant mechanical stimulation is the middle ear muscles themselves. The precise nature of this stimulation is considered in the following sections.

Vibrations

In the present model, the relevant mechanical stimulus is not an isolated muscle contraction but tremor-like vibration of sufficient amplitude to increase ECI drive. If middle ear muscles are indeed the endogenous source of this stimulation, force instability during contraction is the most plausible mechanism. In striated muscle, sustained force depends on sequential and largely asynchronous recruitment of motor units, because the contraction of individual fibers is brief [40]. However, motor units are not fully independent and tend to exhibit partial synchronisation, such that two units fire nearly simultaneously more often than expected by chance [40–42]. This phenomenon is thought to arise centrally [41,43]. Partial synchronisation increases force variability and tremor amplitude, and this effect becomes more pronounced as contraction intensity rises [40,44–46]. Importantly, tremor can also occur during low to moderate contractions, for example during simple postural activity [44], and it tends to increase with aging [47].

Although these phenomena have been studied mainly in limb muscles, similar synchronisation has also been demonstrated in facial muscles, which, like middle ear muscles, are controlled by cranial motor nuclei [42]. Several factors can enhance this instability, including fatigue and training [48,49]. Repetitive intense contractions, even of short duration, can durably increase synchronisation, and this effect may persist after prolonged rest [46,48]. Marked inter-individual variability in synchronisation intensity has also been reported, suggesting that comparable levels of middle ear muscle activation may not produce the same degree of vibratory stimulation in all individuals [40,44,46]. Some authors have also suggested that smaller muscles may be particularly prone to synchronised activity, although this remains debated [42,43]. In this respect, the middle ear muscles, which are small striated muscles, may be particularly susceptible to developing tremor capable of overstimulating putative MeRs.

The effect of such vibration on putative MeRs nevertheless depends on two unresolved factors: the amplitude of muscle-generated vibration and the sensitivity of the receptors themselves. The second of these factors is particularly uncertain, because the functional properties of these putative receptors remain unknown. This distinction is central to the present model, because the pathological issue may not be pressure change per se, but rather the delivery of a sufficiently rapid and repeated mechanical stimulus to receptors that are likely to be phasic. The following section therefore examines the anatomical evidence supporting their presence in the middle ear and the limited basis for inferring how they might respond to vibration.

Mechanoreceptors

If vibration is indeed the relevant mechanical stimulus, the next question is whether the middle ear contains sensory structures capable of detecting it. Histological studies have identified encapsulated nerve endings within the middle ear, but their functional properties remain unknown.

Two distinct types of encapsulated formations have been described. The first is located within the tympanic

membrane. Because of its encapsulated morphology, it was initially described by some authors as a *modified Pacinian corpuscle* [10], although its structure differs from that of canonical Pacinian corpuscles (PCs) [50]. The second type consists of encapsulated formations suspended by fine connective tissue strands anchored to the middle ear mucosa. These structures have been observed within the tympanic cavity, particularly near the ossicles, their suspensory ligaments, and the tendons of the TTM and SM [51]. Their onion-like lamellar capsule likewise prompted comparison with PCs, although their ultrastructure again differs from genuine PCs [52]. Notably, this second type has not been observed in children or young adults [52].

Although their function has not been directly characterised, their morphology suggests a mechanosensory role broadly analogous to that of PCs. PCs are rapidly adapting, phasic receptors that respond preferentially to dynamic deformation rather than sustained force and are therefore sensitive to vibration [53]. The structural differences between MeRs and canonical PCs likely alter these response characteristics, but by morphological analogy it remains plausible that MeRs also behave as phasic receptors [54]. If so, vibratory activity within the middle ear would represent a plausible stimulus. This point is central to the present model. If MeRs are indeed phasic receptors, slow or quasi-static pressure changes would be expected to remain within their normal operating range, whereas repetitive tremor-like deformation could recruit them repeatedly and, through temporal summation, generate a supra-physiological ECI drive. In this sense, the relevant variable may not be pressure itself, but the rate and repetition of the mechanical stimulus.

Several indirect observations are consistent with this interpretation. In a study involving 25 healthy volunteers, experimentally induced middle ear vibration produced transient phantom auditory percepts whose occurrence increased with stimulation frequency and became nearly universal at higher frequencies [55]. The fact that this percept became much more frequent as stimulation frequency increased is consistent with the present hypothesis, because it suggests that repeated vibratory stimulation, rather than pressure change alone, is the critical factor. We suggest that these subjects may have experienced a stimulus-induced tinnitus-like percept, potentially engaging mechanisms relevant to tinnitus. Similarly, intense low-frequency sound is known to trigger transient tinnitus [56]. Although a direct effect on cochlear structures cannot be excluded [57], such sounds plausibly generate large-amplitude oscillations within the middle ear.

A second line of evidence comes from the effects of local anesthetics. Agents such as lidocaine, procaine, dibucaine, and tetracaine have long been known to reduce, or even abolish, subjective tinnitus, despite significant adverse effects. This phenomenon has been observed after both intravenous administration [58] and transtympanic application into the middle ear cavity [59]. While traditionally attributed to suppression of cochlear hyperactivity [58], an alternative explanation is possible. Local anesthetics inhibit action potential generation in PCs [60], owing to the presence of unmyelinated neurites. Because MeRs also contain unmyelinated fibers [50], they might be similarly susceptible, which raises the possibility that at least part of the beneficial effect of local anesthetics could involve inhibition of MeRs.

Taken together, these observations are consistent with the hypothesis that the middle ear contains putative MeRs capable of responding to vibration. However, their functional characteristics remain unknown, and direct physiological validation will be required to determine whether they contribute to increased ECI drive in tinnitus.

Clinical correlates of middle-ear muscle hyperactivity

TTTS and HRMEMS

If the proposed pathway is relevant, heightened middle ear muscle activity should have recognisable clinical correlates. Such an association has long been described under the term *Tonic Tensor Tympani Syndrome* (TTTS) [61]. Originally attributed to sustained hyperactivity or hypertonia of the TTM, TTTS encompasses a broad spectrum of symptoms, including tinnitus, aural fullness, otalgia, facial or cervical pain, burning sensations in or around the ear, distorted hearing, vertigo, and nausea [62,63]. It is often described in the context of acoustic shock syndrome, in which a sudden, loud, and emotionally threatening sound triggers this constellation of symptoms [62]. Importantly, TTTS is not exclusively linked to acoustic shock and may occur in patients without such a history. An epidemiological study found that a large majority of patients consulting for tinnitus, with or without hyperacusis, reported at least one TTTS-related symptom, the most frequent being aural fullness, which may be the clinical manifestation most directly related to stimulation of MeRs [63].

More recently, Fournier and colleagues developed a technique that differentiates SM and TTM activity by combining tympanometry with direct recording of air-pressure changes in a sealed external auditory canal [39]. In the same configuration, Eustachian tube opening, primarily mediated by the TVPM, can also be detected. Using this approach, the authors reported involvement of the TTM, SM, and TVPM, alone or in combination, in patients presenting symptoms classically attributed to TTTS [64]. Because the syndrome appears to reflect excessive reactivity rather than purely tonic contraction, they proposed the broader term *Hyper-Reactive Middle Ear Muscle Syndrome* (HRMEMS). Notably, all participants in their study exhibited tinnitus, despite diverse triggering events including stress, sudden sensorineural hearing loss, acoustic shock, otologic surgery, and loud-noise exposure. These observations support the view that abnormal middle ear muscle activation may represent a common denominator across clinically heterogeneous but tinnitus-associated conditions.

Within this framework, isolated tinnitus and tinnitus accompanied by TTTS/HRMEMS symptoms may lie along a continuum, with symptom expression depending on the intensity and persistence of middle ear muscle activation.

Persistence and chronicity

Tinnitus is often transient, but its chronic form is the one that most significantly impairs quality of life. If middle ear muscle activity contributes to persistent tinnitus, the associated mechanical stimulation must itself persist over time. In healthy individuals, transient tinnitus is typically reported to resolve shortly after the putative overstimulation of MeRs ends [55]. As discussed above, muscle tremor may persist for several hours after exertion [65], but in the absence of renewed activation it eventually subsides.

A pathophysiological model proposed by Noreña et al. may help explain how such activity could become self-sustaining after acoustic shock [66]. In this model, excessive TTM contraction leads to local ischemia, metabolic stress, and neurogenic inflammation. This inflammatory milieu lowers activation thresholds in somatosensory afferents and amplifies peripheral input [67,68]. These afferents project to the trigeminocervical complex, a convergence hub for trigeminal and upper cervical inputs [69]. Because the trigeminocervical complex also exerts descending control over head and neck musculature, its sensitisation may increase efferent drive to the middle ear muscles, thereby reinforcing their hyperreactivity and facilitating reflex-related contractions [70,71]. In this way, peripheral inflammation and central sensitisation could establish a positive feedback loop capable of maintaining excessive muscle activation beyond the initial trigger.

At the same time, HRMEMS has not been described by its authors as a permanent state. Rather, it represents hyperreactivity to stimuli, and its apparent intermittence may partly reflect methodological limitations. The available pressure and impedance measures detect only sufficiently large changes in muscle tension. A near-perfectly stable tonic contraction may be poorly captured by such methods [72]. Consistent with this interpretation, in a particularly severe case of TTTS, continuous middle ear impedance monitoring revealed baseline instability in the more symptomatic ear [62]. Such instability could reflect subtle vibration-related changes in the mechanical state of the ossicles and tympanic membrane. This suggests that the hypertonia initially described in TTTS and the hyperreactivity reported in HRMEMS may represent two facets of the same phenomenon, namely an overall increase in neural drive to the middle ear muscular system.

The cochlear nucleus as a site of cochlear and extracochlear convergence

Plasticity in tinnitus models

Current evidence suggests that the earliest tinnitus-specific neural correlate emerges within the CN [73]. The CN is both the first central relay of auditory afferents and an early site of multisensory integration, combining CIs with numerous ECIs [74]. The mechanisms governing this integration remain incompletely understood. The present model proposes that tinnitus arises when reduced CI is accompanied by an abnormal increase in ECI drive.

This interpretation is supported by animal models of tinnitus. Following cochlear deafferentation, reduced auditory nerve activity is observed at several entry points to the CN. However, these changes are not sufficient to explain tinnitus, because they reflect cochlear injury in general and are not restricted to animals that develop tinnitus-like behavior. By contrast, only animals classified as having tinnitus show a marked increase in the spontaneous activity of fusiform cells, the principal output neurons of the dorsal cochlear nucleus (DCN).

Several facilitatory plastic changes affecting ECI pathways have likewise been reported specifically in tinnitus models [73]. These include increased glutamatergic transmission associated with upregulation of vesicular glutamate transporter 2 (VGLUT2) in CN regions receiving ECIs, and a shift in spike-timing-dependent plasticity (STDP) rules toward sustained facilitation and long-term potentiation (LTP) [5,75]. VGLUT2 is specifically associated with ECI terminals [76]. Collectively, these mechanisms increase the relative weighting of ECIs in CN processing.

Our interpretation of these findings is that the plastic changes are more likely to be a consequence of excessive ECI drive than its primary cause. In humans, tinnitus typically begins almost immediately after acute acoustic trauma [77], whereas at least some of the observed plastic changes, particularly VGLUT2-related remodeling, require time to develop. Although timing-dependent synaptic changes might emerge more rapidly [78], the most parsimonious explanation is that tinnitus onset depends on a supra-physiological increase in ECI drive superimposed on reduced CI. The same excessive ECI drive could then induce the plastic adaptations observed in tinnitus models. If this interpretation is correct, the presence of tinnitus-specific plastic changes itself implies that ECI activity must reach a supra-physiological level, since it is sufficient to induce pathway-specific remodeling rather than simple physiological modulation. It also implies that an isolated increase in ECI drive, even in the absence of established structural plasticity, may be sufficient to trigger tinnitus. These plastic changes may subsequently stabilise or maintain tinnitus-related hyperactivity once established.

Extracochlear inputs

In the present model, the middle ear is proposed as a plausible source of the increased ECI drive required for tinnitus. Several observations support this interpretation. First, in tinnitus models, VGLUT2 overexpression in the CN has been associated with increased projections from the spinal trigeminal nucleus, cuneate nucleus, and lateral vestibular nucleus to the CN [18,19]. This pattern is consistent with the anatomical links between the middle ear and the CN described above.

Further support comes from human imaging. In soldiers with tinnitus following acute acoustic trauma, Job et al. reported increased activation in Brodmann area 43 (BA 43) and BA 43/40 on functional MRI [79]. Both regions lie within the primary somatosensory cortex (S1). Notably, BA 43 had previously been activated by deformation of the tympanic membrane induced by pressure changes in the external auditory canal [12], whereas the deeper BA 43/40 region has been linked to somatosensory information arising from the middle ear. In addition, activation intensity in

these regions correlated with tinnitus periodicity and subjective distress. These pressure variations produce a global deformation of the tympanic membrane, whose MeRs are likely to be the primary transducers.

In the present framework, stimulation of tympanic membrane MeRs may therefore plausibly contribute to activation of BA 43, while stimulation of MeRs within the tympanic cavity could, together with neuromuscular spindle afferents, contribute to activation of BA 43/40. In this interpretation, activation of BA 43 and BA 43/40 should be understood primarily as a marker of middle ear somatosensory activity in tinnitus subjects. Taken together, these observations support the middle ear as a plausible source of increased ECI drive in tinnitus.

Clinical contexts potentially associated with increased middle ear muscle activity

In the present framework, the following clinical contexts may be interpreted as situations in which middle ear muscle activity becomes abnormally elevated, thereby providing a possible route to tinnitus.

Loud-noise exposure and other deafferentation-related contexts

Noise exposure is the clearest clinical context in which the present model may operate, because it can combine the two key conditions summarised in Figure 1, namely reduced CI and increased middle ear muscle activity. Temporary tinnitus following loud-noise exposure is common both in individuals without chronic tinnitus and in patients who experience a transient worsening of pre-existing symptoms. Intense sounds elicit the acoustic reflex, that is, a sustained contraction of the SM whose strength increases with sound intensity [80]. Particularly intense, unexpected, or threatening sounds can also recruit the TTM and TVPM, likely through startle-related mechanisms [28,30]. Since tremor-like instability increases with contraction intensity and fatigue, and since repetitive intense contractions may act as a form of training that durably enhances synchronisation, prolonged or repeated loud-noise exposure could plausibly generate sufficient mechanical stimulation of MeRs to trigger transient tinnitus or exacerbate existing tinnitus [28,65].

Other deafferentation-related contexts may also fit the model, because cochlear injury may not only reduce CI but also secondarily increase middle ear muscle activity. A documented case of TTTS developing after sudden sensorineural hearing loss supports this possibility [64]. Moreover, aural fullness is experienced by the vast majority of patients during episodes of sudden hearing loss [81,82]. Two observations are particularly suggestive. In one study, perceived fullness correlated with the severity of the auditory deficit after auditory thresholds had stabilised [82]. In another, patients who initially reported fullness showed better long-term recovery of low frequencies [81], a pattern consistent with a reversible low-frequency threshold elevation caused by middle ear muscle contraction rather than permanent cochlear damage alone [83]. Although the available data are limited, these findings are consistent with the idea that cochlear deafferentation may, in some cases, trigger secondary activation of middle ear muscles. Taken together, acoustic trauma and related deafferentation contexts are especially relevant to the present model because they may combine reduced CI with increased middle ear muscle activity, either simultaneously or through secondary muscle activation following cochlear injury.

External auditory canal

External auditory canal disorders are relevant to the present model because they may precipitate tinnitus without themselves causing direct cochlear deafferentation, and tinnitus often resolves when the local cause is treated. External otitis and cerumen impaction have both been associated with tinnitus [8,84], and resolution commonly follows treatment or wax removal [8]. This observation is difficult to explain solely by reduced sound transmission, since occlusion of the auditory meatus does not itself produce cochlear injury; from the inner ear's perspective, there is no fundamental difference between reduced sound input caused by canal obstruction and a quieter acoustic environment.

Within the present framework, a more plausible explanation is that repeated stimulation of the external auditory canal increases middle ear muscle activity and, in the presence of pre-existing or subclinical cochlear deafferentation, may contribute to tinnitus. As discussed above, stimulation of the external auditory canal can induce changes in middle ear impedance, attributed mainly to SM contraction [30,37]. Recurrent canal irritation, whether from cerumen impaction or otitis externa, could therefore enhance middle ear muscle activation and, in susceptible individuals, promote tinnitus. When the local stimulation ceases, the disappearance of the triggering factor may account for tinnitus remission.

In addition, both external otitis and irritation from a cerumen plug are often accompanied by local inflammation. This may enhance tactile sensitivity [68], strengthen the afferent component of the reflex, and sensitise the trigeminocervical complex, thereby amplifying the efferent response. In this way, external auditory canal disorders can be understood as peripheral triggers of the pathway proposed in **Figure 1**.

Temporomandibular and cranio-cervical disorders

Temporomandibular and cranio-cervical disorders may fit the present model because both can engage trigeminal and upper cervical pathways linked to middle ear muscle activity. Clinically, these conditions are often accompanied by otological symptoms such as aural fullness, otalgia, vertigo, hearing loss, and tinnitus, with substantial overlap in their manifestations [85,86].

Several anatomical and functional links support the possibility that TMDs may contribute to tinnitus through increased middle ear muscle activity [87]. The temporomandibular system and the middle ear muscles share a common embryological origin in the first branchial arch, and phylogenetic analyses suggest a relationship between

the masticatory muscles and the ossicular chain. The TTM and TVPM, like the masticatory muscles, are innervated by branches of the trigeminal nerve, particularly the medial pterygoid nerve. A functional association between the TTM and TVPM has been reported, especially during swallowing [32,36], and dental malocclusion has been linked to Eustachian tube dysfunction [88]. The auriculotemporal nerve, which innervates part of the temporomandibular joint, also supplies the tympanic membrane and external auditory canal, providing a plausible route for symptom overlap. Jaw movements can modulate tinnitus loudness and may even elicit tinnitus in susceptible individuals [25,89], suggesting that middle ear muscle activation could represent a shared contributing factor linking TMDs and tinnitus [87]. In addition, inflammation associated with TMDs may sensitise the trigeminocervical complex [68,90], potentially facilitating increased middle ear muscle activity.

A similar logic may apply to cranio-cervical disorders. Conditions affecting the cervical spine, neck muscles, or the broader cranio-cervical region have long been associated with tinnitus and related otological symptoms [85]. Levine, in a case series of patients with cranio-cervical dysfunction, proposed that somatic afferents from this region could modulate DCN activity and thereby contribute to tinnitus [91]. An additional interpretation is possible: middle ear muscles can contract in response to movements involving the cranio-cervical region, and sustained head flexion, for example, elicits prolonged contraction of both the SM and TTM [30]. Musculoskeletal disorders affecting this region might therefore provoke inappropriate activation of local mechanoreceptors, leading to increased middle ear muscle activity. As in TMDs, associated inflammation could further enhance this pathway by sensitising the trigeminocervical complex. A direct somatosensory influence on the DCN, as originally suggested by Levine, cannot be excluded. However, both TMDs and cranio-cervical disorders are consistent with the present model in that they may converge on increased middle ear muscle activity through trigeminal and upper cervical pathways.

Ménière's disease

Ménière's disease is characterised by episodic vertigo, fluctuating hearing loss, tinnitus, and aural fullness. Beyond the classical endolymphatic hydrops model [92], Bell [93] proposed an alternative hypothesis involving dysregulation and increased activity of the middle ear muscles, particularly the TTM, as a possible contributing mechanism. In this view, excessive contraction of the TTM could exert a mechanical effect on the ossicular chain, increase intra-labyrinthine pressure, and thereby influence outer hair cell (OHC) function and auditory gain, especially at low frequencies.

Several observations are compatible with a possible muscular contribution. First, no clearly established dedicated sensory pathway has yet been identified to detect pressure changes within the inner ear, whereas mechanosensory and proprioceptive feedback exists within the middle ear. Second, voluntary TTM contraction in healthy subjects induces a low-frequency threshold elevation that exceeds what would be expected from changes in middle ear admittance alone [83,94], with similar effects even under bone-conduction testing. Finally, symptomatic improvement, including tinnitus relief, has been reported following TTM tenotomy in selected cases [95], in contrast to the frequently limited efficacy of surgical interventions targeting endolymphatic hydrops [96].

Taken together, these findings suggest that abnormal middle ear muscle activity could contribute to at least some features of the syndrome, including tinnitus.

A possible role of middle ear input in acoustic gain integration

A natural question raised by the present hypothesis is what information putative MeRs could convey, and what functional role such input might play once it reaches the CN through extracochlear pathways. One possibility is that these receptors monitor changes in the mechanical state of the middle ear and therefore convey information about variations in middle ear admittance. MeR-related input could thus provide the CN with information about a precochlear component of acoustic gain. Tinnitus might then reflect, at least in part, a disturbance of mechanisms integrating information about acoustic gain. In this respect, the present hypothesis may be viewed as broadly compatible with central-gain accounts, while extending them by proposing a middle ear source of increased ECI drive. This interpretation is also consistent with Bell's proposal that middle ear muscle activity may influence cochlear sensitivity, thereby linking changes in middle ear mechanics to the regulation of acoustic gain at the cochlear level [97].

Several observations are compatible with this view. First, tinnitus is strongly associated with hyperacusis [63], a condition involving altered processing of sound intensity. Second, following significant acoustic trauma, many cochlear lesions involve the OHCs, a pattern also reported after certain chemotherapies [2,98]. Because OHCs contribute to active cochlear amplification, their dysfunction is also relevant to acoustic gain. It has therefore been proposed that type II auditory fibers, small unmyelinated fibers conveying signals from OHCs to the CN through the auditory nerve, may interact with extracochlear pathways in a process relevant to tinnitus generation [2,98].

Although the functional role of type II fibers remains uncertain [99,100], one may hypothesise that they provide the CN with information about cochlear amplification, whereas MeRs provide complementary information about the mechanical state and admittance of the middle ear. In this framework, the CN would integrate distinct signals related to acoustic gain at both the middle ear and cochlear levels. A disturbance of this integrative process could contribute not only to tinnitus, but also to its frequent association with hyperacusis.

Conclusions

This work proposes that subjective tinnitus may arise when reduced cochlear input is accompanied by abnormally increased extracochlear drive to the cochlear nucleus. In the present framework, heightened activity of the stapedius, tensor tympani, and tensor veli palatini may generate

tremor-like vibrations within the tympanic cavity, overstimulating putative middle ear mechanoreceptors and thereby increasing somatosensory input to the cochlear nucleus (**Figure 1**).

A major limitation of this hypothesis is that the functional properties of these putative mechanoreceptors remain unknown. Progress is therefore most likely to come from direct physiological testing rather than further conceptual expansion. In animals, spontaneous or evoked activity of middle ear muscles could be assessed with electromyography and compared between tinnitus and non-tinnitus animals. In humans, refined admittance or impedance measurements with higher sampling rates could be used to search for rapid middle ear fluctuations consistent with tremor-like activity. Functional neuroimaging could test whether activation of BA 43 and BA 43/40 is also observed in tinnitus unrelated to acoustic trauma. A further prediction is that, among otherwise healthy subjects without clinically evident hearing impairment, individuals lacking a measurable stapedius reflex should show a different susceptibility to transient tinnitus or aural fullness after loud-noise exposure. Direct physiological validation will be required to determine whether this proposed pathway operates *in vivo*.

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