RESEARCH ARTICLE

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Multimodal integration in tinnitus. Exploratory factor analysis of the relationships between the latent variable tinnitus and the observed clinical variables or indicators

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ABSTRACT

Background: In the auditory system, the integration of information from the cochlea with other sensory modalities begins in the cochlear nucleus. Proper interpretation of this information in patients with tinnitus can enhance our understanding of how auditory signals combine with somatosensory and proprioceptive signals that convey different types of information.

Objectives: The objective of this study is to examine the fundamental mechanisms of multimodal integration by evaluating the intermodal effects of hearing, proprioception, cervical pain, cervical degeneration, and pupillometry on tinnitus using factor analysis.

Methods: A retrospective study of all patients who visited our clinic from February 2010 to September 2025 for tinnitus lasting at least 1 month.

Results: Exploratory factor analysis shows the relationships between the latent variable tinnitus and the observed variables (indicators), such as dizziness, postural instability, cervical pain, hearing loss at 0.125 kHz, hearing loss due to environmental noise (hidden hearing loss), size of anterior osteophyte at the 4th cervical vertebra, and Farfan's measure between the 6th and 7th cervical vertebrae for measuring disc height and baseline pupil diameter (mm), pupillary constriction rate (%) measured by pupillometry.

Conclusion: Tinnitus is a multifactorial condition which is associated with a disinhibition of neurons in the dorsal cochlear nucleus, due to less excitation of the inhibitory part of the cochlear nucleus leading to increasing spontaneous activity in the central auditory system. Loss of inhibition at the fusiform cell in the dorsal cochlear nucleus are especially induced by hidden hearing loss and postural instability.

Keywords: Tinnitus, exploratory factor analysis, indicators, hidden hearing loss, pupillometry, postural instability, cochlear nucleus.

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INTRODUCTION

Tinnitus, the perception of phantom sounds, is a symptom with complex, multifactorial causes, primarily sensorineural, somatosensory, neurovascular, and idiopathic[1,2]. It is often triggered by cochlear damage, which can lead to abnormal neural amplification of the central auditory system to compensate for peripheral sensory loss from the cochlea[3,4]. However, the neural mechanisms behind this altered auditory processing are not fully understood.

The incorporation of auditory information from the cochlea with information of other sensory modalities begins in the cochlear nucleus[5,6]. The sensory modalities include the dorsal column nuclei, the spinal trigeminal nucleus and the cervical nerves. Proper interpretation of this information in patients with tinnitus can enhance understanding of how auditory signals integrate with somatosensory and proprioceptive signals.

Factor analysis is a statistical method used to simplify complex data, discover hidden patterns, and facilitate more detailed analysis[7,8]. The goal of this study is to explore the basic mechanisms of multimodal integration by evaluating the intermodal influences of hearing, proprioception, cervical pain, cervical degeneration, and pupillometry on tinnitus using factor analysis.

MATERIALS & METHODS

Plan

A retrospective study with all tinnitus patients who visited our clinic between February 2010 and September 2025 (n = 912). The Ethics Committee Amsterdam (Amsterdam, the Netherlands) admitted our study (2025.1091, December 23th, 2025).

Subjects

Patients who had tinnitus lasting at least one month were included. No exclusion criteria were effectual. All patients had previously been examined by an otolaryngologist, and in patients with unilateral tinnitus anatomical pathology was ruled out by MRI. Hidden hearing loss (HHL) is defined as extra hearing impairment with ambient noise.

Data assessment

We used data from clinical information, from the audiogram, from measurements of the cervical spine radiographs, and from pupillometry (**Table 1**). Measurements of the radiographs of the cervical spine and the parameters of the pupillometry were described in a previous article (**Figure 1**)[9].

Statistics

Statistical analysis was performed using IBM Corp. Released in 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp. Mean pupillometry values with standard deviations were assessed. Exploratory factor analysis was performed using AMOS (version 24.0), Chicago: IBM SPSS, to evaluate a tinnitus model.

Results

Exploratory factor analysis was carried out using AMOS (version 24.0; Chicago, IBM SPSS). Exploratory factor

Table 1: Pupillometry values.

	N	Mean	Std. Deviation
Baseline pupil diameter (BPD)(mm)	58	3.93	0.85
Pupillary constriction rate (PCR) (%)	58	27.71	8.42
Maximum constriction amplitude (MCA) (mm)	58	3.44	1.55
Latency constriction velocity (LC) (msec)	58	241.62	47.25
Maximum constriction velocity (MCV) (mm/sec)	58	4.22	0.68

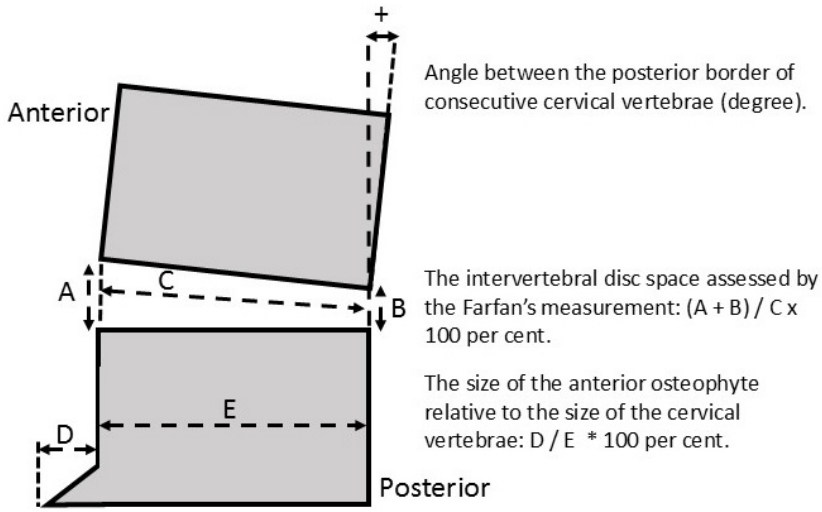

Figure 1: Measurements of the radiographs of the cervical spine.

analysis shows the relationships between the latent variable tinnitus and the observed variables (indicators), such as dizziness, postural instability, cervical pain, hearing loss at 0.125 kHz, HHL, size of anterior osteophyte at the 4th cervical vertebra, Farfan's measure between the 6th and 7th cervical vertebrae for measuring disc height, and baseline pupil diameter (BPD) (mm) and pupillary constriction rate (i.e., the difference between baseline and post-stimulation pupil size, expressed as % of constriction from the baseline value) (PCR) (%) measured by pupillometry (**Figure 2**). All 912 patients reported tinnitus, and since tinnitus was not directly measured, it is treated as a continuous latent variable, referred to as a factor. Tinnitus indirectly affects hearing loss at 0.125 Hz, stemming from exposure to environmental noise. It also indirectly affects the intervertebral disc space between the 6th and 7th cervical vertebrae because of an anterior osteophyte at the 4th cervical vertebra. The indicators are either continuous or categorical variables. The error terms in exploratory factor analysis are assumed to be uncorrelated.

We estimated the standardized regression weights within the same measurement unit, keeping the effect of one observed variable (e.g., cervical pain) fixed at 1 (**Table 2**). We present the standardized total effects, the standardized direct effects, and the standardized indirect effects (**Table 3-5**).

Various goodness-of-fit statistics are calculated. The chi-square fit statistic is 26.81 with 27 degrees of freedom. The model demonstrates a good fit to the data with a non-significant p-value (0.474). The null hypothesis states that the model is correct and cannot be rejected. The normed fit index (NFI) is 0.952, and the comparative fit index (CFI) is 1.0, indicating excellent fit. The root mean square error of approximation (RMSEA) is <0.001 with a p-value of 1.000. The p-value should be above 0.50 to reflect a good model fit. Hoelter's critical n (CN) .05 is 1364. Values above 200 indicate an adequate sample size.

Adding the dependent variables age and gender deteriorated the model's fit. All the regression weights were positive, except for Farfan's measure between the 6th and 7th cervical vertebrae, which was used to measure discus height, BPD, and PCR.

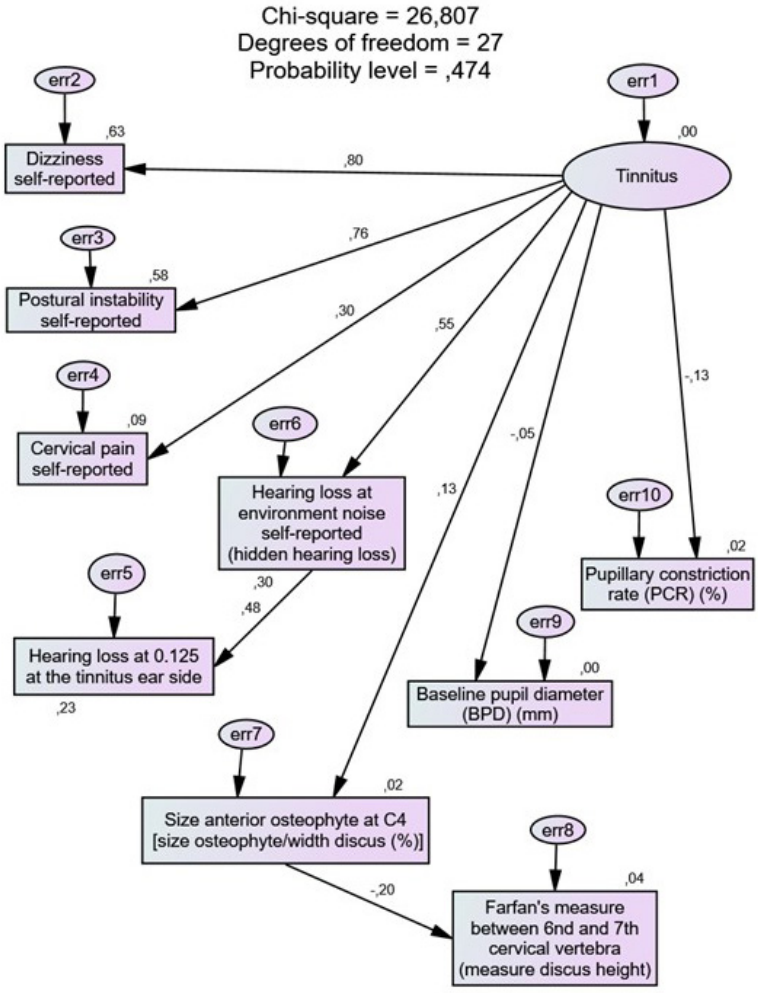

Figure 2: Exploratory factor analysis model for the relationship between tinnitus and dizziness, postural instability, cervical pain, hearing loss at 0.125 kHz, hidden hearing loss, size of anterior osteophyte at 4th cervical vertebra, and Farfan's measure between 6th and 7th cervical vertebrae for measuring discus height and pupillometry measurements, maximum constriction velocity (mm/sec), and baseline pupil diameter (mm). The numbers are explained in the text. In this figure, decimal points are replaced by commas.

Table 2: Standardized Regression Weights.

			Estimate
Size of the anterior osteophyte at C4	<---	Tinnitus	0.130
Hearing loss in environmental noise	<---	Tinnitus	0.549
Dizziness	<---	Tinnitus	0.797
Postural instability	<---	Tinnitus	0.761
Farfan's measure between 6th and 7th cervical vertebrae (discus height)	<---	Size of the anterior osteophyte at C4	-0.202
Cervical pain	<---	Tinnitus	0.301
Hearing loss at 0.125 kHz	<---	Hearing loss in environmental noise	0.476
Pupillary constriction rate	<---	Tinnitus	-0.129
Baseline pupil diameter	<---	Tinnitus	-0.053

Table 3: Standardized Total Effects.

	Tinnitus	Hearing loss at environment noise	Size of the anterior osteophyte at C4
Hearing loss in environmental noise	0.549	0.000	0.000
Size of the anterior osteophyte at C4	0.130	0.000	0.000
Baseline pupil diameter	-0.053	0.000	0.000
Pupillary constriction rate	-0.129	0.000	0.000
Hearing loss at 0.125 kHz	0.261	0.475	0.000
Postural instability	0.761	0.000	0.000
Dizziness	0.797	0.000	0.000
Farfan's measure between 6th and 7th cervical vertebrae (discus height)	-0.026	0.000	-0.202
Cervical pain	0.301	0.000	0.000

Table 4: Standardized Direct Effects.

	Tinnitus	Hearing loss at environment noise	Size anterior osteophyte at C4
Hearing loss in environmental noise	0.549	0.000	0.000
Size of the anterior osteophyte at C4	0.130	0.000	0.000
Baseline pupil diameter	-0.053	0.000	0.000
Pupillary constriction rate	-0.129	0.000	0.000
Hearing loss at 0.125 kHz	0.000	0.476	0.000
Postural instability	0.761	0.000	0.000
Dizziness	0.797	0.000	0.000
Farfan's measure between 6th and 7th cervical vertebrae (discus height)	0.000	0.000	-0.202
Cervical pain	0.301	0.000	0.000

Table 5: Standardized Indirect Effects.

	Tinnitus	Hearing loss at environment noise	Size anterior osteophyte at C4
Hearing loss in environmental noise	0.000	0.000	0.000
Size of the anterior osteophyte at C4	0.000	0.000	0.000
Baseline pupil diameter	0.000	0.000	0.000
Pupillary constriction rate	0.000	0.000	0.000
Hearing loss at 0.125 kHz	0.261	0.000	0.000
Postural instability	0.000	0.000	0.000
Dizziness	0.000	0.000	0.000
Farfan's measure between 6th and 7th cervical vertebrae (discus height)	-0.026	0.000	0.000
Cervical pain	0.000	0.000	0.000

DISCUSSION

Our exploratory factor analysis model investigates the relationships between tinnitus and various factors, including dizziness, postural instability, cervical pain, hearing loss at 0.125 kHz, HHL, the size of anterior osteophytes at the 4th cervical vertebra, and Farfan's measure of disc height between the 6th and 7th cervical vertebrae. It also examines pupillometry metrics like PCR

and BPD. The somatosensory causes of tinnitus, such as dizziness, postural instability, and cervical pain, exhibit high regression weights, indicating their significance. Similarly, the sensorineural causes, including HHL and hearing loss at 0.125 kHz on the tinnitus side, also show high regression weights, emphasizing their importance. Other somatosensory causes of tinnitus, such as the size of the anterior osteophyte at the 4th cervical vertebra and Farfan's measurement between the 6th and 7th cervical vertebrae, were less significant, as shown by their low regression weights. Pupillometry measures also had low regression weights, probably due to either lower relevance or a small number of patients with pupillometry data. These multiple contributing factors affirm that tinnitus is a multifactorial condition. Multifactorial conditions are complex systems with many components. It is essential to study how these components relate to and affect one another.

The neural mechanisms underlying the altered multifactorial auditory processing of tinnitus are unknown and we are in need for more knowledge of the integration of auditory signals with somatosensory and proprioceptive information in patients with tinnitus[10]. In this study, exploratory factor analysis shows an appropriate model of the relationships between the latent variable tinnitus and the indicators, such as dizziness, postural instability, cervical pain, HHL, size of anterior osteophyte at the 4th cervical vertebra, and BPD and PCR measured by pupillometry (**Figure 2**).

Dizziness and Postural Instability. In our study, self-reported dizziness and postural instability are more associated with tinnitus than self-reported HHL, degeneration of the cervical spine and the BPD and PCR measured by pupillometry. Balance and spatial orientation depend on the correct functionality of our vestibular system[11-13]. The vestibular nerve receives signals about the body's motion from hair cells in the vestibular apparatus of the inner ear and sends this information to the vestibular nuclei in the medulla oblongata. The vestibular nuclei send afferent fibers to the lumbosacral, thoracic, and cervical segments of the spinal cord, the cerebellum and the oculogyric nuclei (the nuclei of the cranial nerves III, IV and VI) (**Figure 3**). The cerebellum receives sensory input from the body directly and indirectly[14]. The direct input comes from the inner ear which detects movement of the head. The indirect input is the information reaching to the cerebellum via the brainstem. Within the vestibular cerebellum there are two types of unipolar brush cells (UBCs): ON and OFF[15]. Direct sensory input activates only ON UBCs. Indirect input activates both ON UBCs and OFF UBCs. ON UBCs will have an amplified response of fast GABA mediated inhibitory modulation, whereas OFF UBCs will have a dampened response modulated by slow glycinergic inhibition. The cerebellum exerts its influence through inhibition. Disruption of UBCs, Purkinje cell or cerebellar nuclei activity weakens balance and also contributes to the beginning of tinnitus.

Hidden hearing loss. Tinnitus can be provoked by cochlear damage causing aberrant patterns of neuronal activity in the auditory system[3,4]. Hearing ability is established by using pure-tone audiometry, a measure of dysfunction of the outer hair cells[10,12]. However, the audiogram is much less sensitive to inner hair cell loss and to peripheral neural dysfunction[3,16]. This cochlear synaptopathy, also known as "hidden hearing loss", compromises hearing in a noisy environment and is linked to with tinnitus and hyperacusis. Unfortunately, there is no reliable and validated clinical diagnostic test for the disorder. Inner hair cell deafferentation, rather than outer hair cell loss, represents a primary risk factor for the

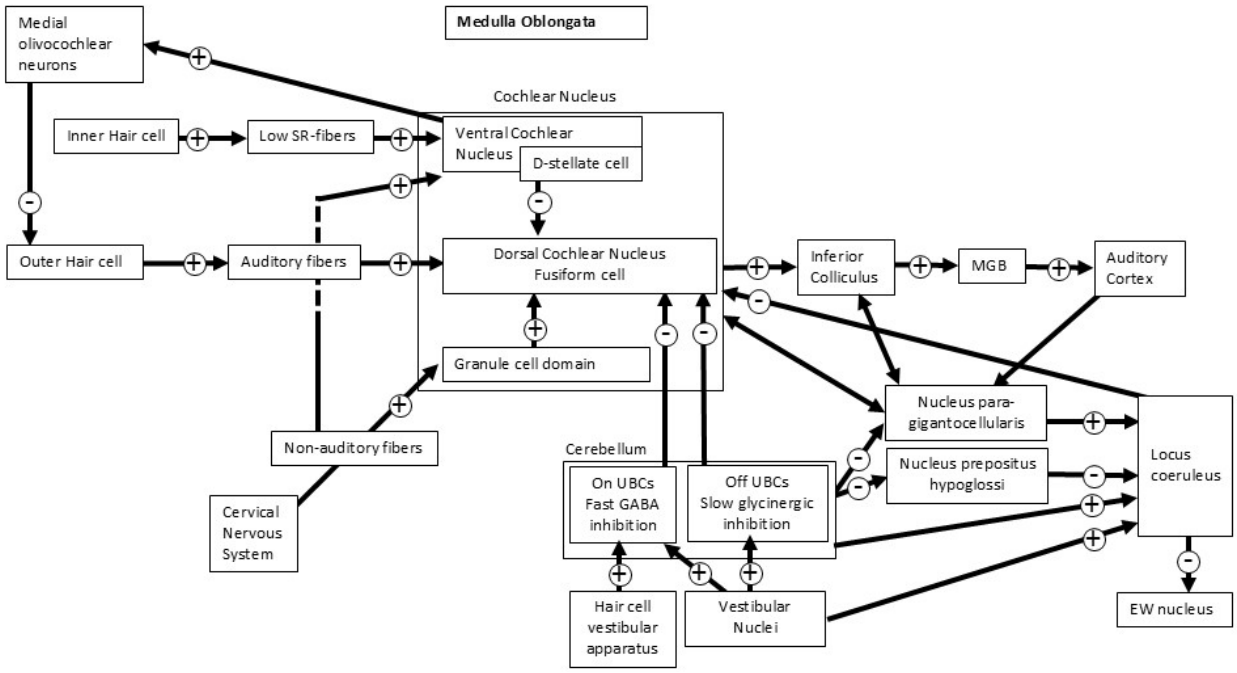

Figure 3: Theoretical model of the mechanisms of multimodal integration at the cochlear nucleus.

generation of tinnitus (**Figure 3**) [17]. The high threshold, low spontaneous rates fibers of the auditory nerve connect the inner hair cells to the ventral cochlear nucleus (VCN)[5,16]. The VCN stellate cell population has two distinct types of multipolar/ stellate cells. The excitatory D-stellate cells of the VCN inhibit fusiform cells in the DCN and the inhibitory T-stellate cells of the VCN are the primary sound-driven inputs to medial olivocochlear (MOC) neurons which send myelinated projections to the outer hair cells of the cochlea. Several studies found MOC efferent pathway dysfunction in the human subjects who had tinnitus[2,18].

Cervical spine disorders. In our study, we found evidence for an association between subjective tinnitus and cervical spine disorders (self-reported cervical pain, size of the anterior spur at the fourth cervical vertebrae and disc degeneration between the sixth and seventh vertebrae). The cochlear nucleus is the first location of multisensory integration in the ascending auditory pathway[5,6]. The cochlear nucleus receives auditory inputs from auditory nerve fibers and nonauditory inputs from several somatosensory nuclei, including the nerves coming from the cervical spine. Non-auditory projections connect with granule cells in the cochlear nucleus granule-cell domain, as well as with bushy cells and D-stellate cells of the VCN 5. These somatosensory inputs excite fusiform cells in DCN by activating granule cells, but they can also inhibit fusiform cells through the inhibitory interneurons, the cartwheel cells (**Figure 3**). Tinnitus was associated with an upregulation of non-cochlear innervation of the VCN and granule cell domain[19].

Pupillometry. The Locus Coeruleus (LC) controls autonomic function[20]. LC activation yield increased sympathetic activity and decreased parasympathetic activity. An excitatory noradrenergic pathway (via the nucleus paragigantocellularis (PGi), an inhibitory GABAergic pathway (via the nucleus prepositus hypoglossi), and fibers arising from the spinal vestibular nucleus provide the input to the LC[21]. The PGi is well connected to the auditory nuclei (cochlear nucleus, inferior colliculus, and the auditory cortex)[22]. Noradrenergic neurons of the LC project to the cochlear nucleus and enhances the inhibition from granule cells to fusiform principal cells in the DCN[20] (**Figure 3**). The LC activity can be indirectly estimated by measuring the pupil[21]. Tinnitus is associated with parasympathetic dysfunction without signs of hyperactivity of the sympathetic nervous system[9].

Neuronal hyperactivity in the posteroventral and dorsal cochlear nucleus, the inferior colliculus, thalamus, and the cerebellum are considered as the hallmark of tinnitus[23]. Neuronal hyperactivity of the auditory system can be the result of excessive excitatory stimulation or loss of inhibition at the fusiform cell of the dorsal cochlear nucleus[24,25] (**Figure 4**). Cervical spine disorders can activate the fusiform cells in DCN[5]. However, cervical spine disorders can also inhibit the fusiform cell by virtue of the cartwheel cell and the D-stellate cell. It is unknown which specific cervical spine disorders are responsible for each route to the fusiform cell in the DCN.

Tinnitus can be associated with a loss of inhibition of the fusiform neurons in the DCN which causes increased spontaneous activity in the central auditory system[16,26]. In our study, loss of inhibition at the fusiform cell in the DCN are especially induced by HHL and postural instability. Especially, the loss of the inhibitory effects of the cerebellum seems important in the pathogenesis of tinnitus. In patients with cervical pain as main complaint,

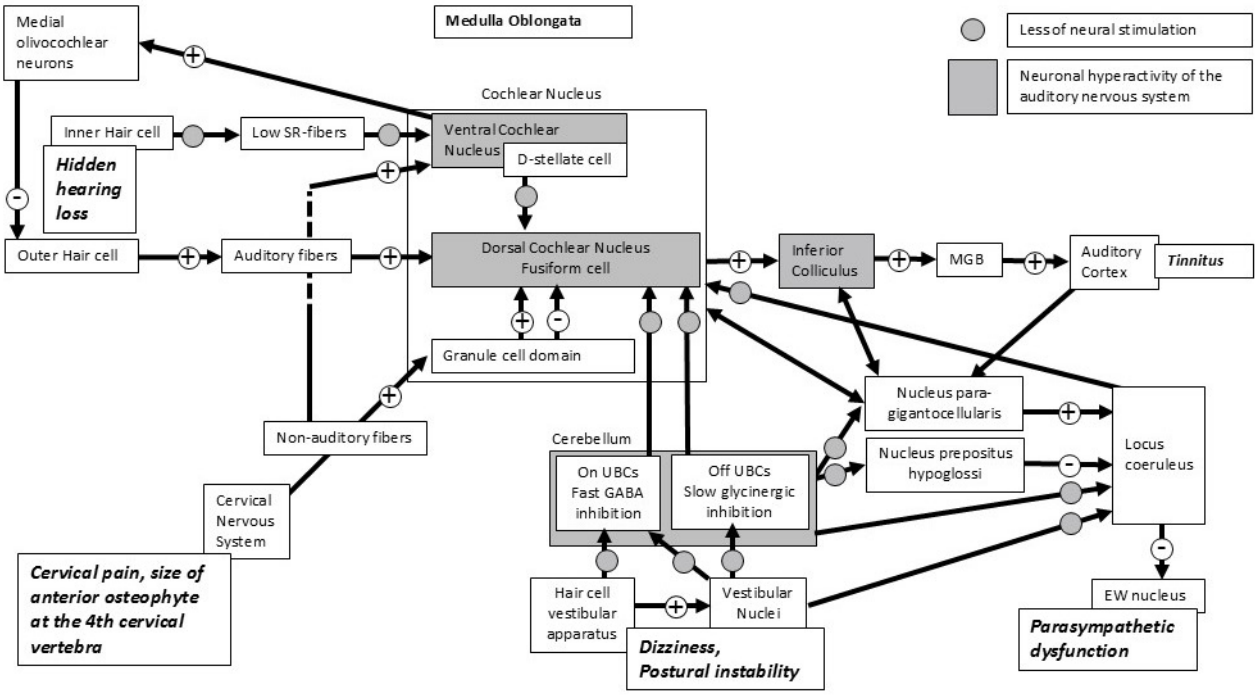

Figure 4: Theoretical model for the pathophysiology of tinnitus based on a factor analysis.

tinnitus was provoked by postural instability and dizziness[27].

Weakened projections from the LC to the DCN may also limit the normal inhibitory control exerted over auditory signal transmission, which may lead to hyperactivation of ascending auditory pathways, resulting in tinnitus. However, pupillometry measures also had low regression weights and we conclude that the influence of the LC on the hyperactivity of the fusiform cell is much less than the impact of HHL, postural instability and cervical spine disorders. Also, loss of inhibition at the fusiform cell of the DCN should be seen with LC dysfunction but pupillometry in patients with tinnitus indicates hyperactivity of the LC. It seems that the influence of the LC in the forthcoming of tinnitus is restricted.

This study has its limitations, such as retrospective study and self-reported symptoms. Dizziness, postural instability, and cervical pain reports were documented after patients experienced these symptoms. Moving forward, researchers should focus on a prospective study with better consistency and accuracy of diagnoses by using Patient Reported Outcome Measures (PROMs), the Dizziness Handicap Inventory (DHI) for dizziness; the Euro QoL 5-Dimension—5-Level (EQ-5D-5L) for postural instability; and the Neck Disability Index (NDI) for cervical pain. Patient-Reported Outcome Measures (PROMs) can be included with the audiogram, such as the Tinnitus Functional Index (TFI) and the Tinnitus Handicap Inventory (THI). PROMS can be incorporated alongside pupillometry, such as in the pupillomotor and gastrointestinal items of the domains of the COMPASS-31.

CONCLUSION

Exploratory factor analysis shows an appropriate model of the relationships between the latent variable tinnitus and the indicators, such as dizziness, postural instability, cervical pain, HHL, size of anterior osteophyte at the 4th cervical vertebra, and BPD and PCR measured by pupillometry. These multiple contributing factors affirm that tinnitus is a multifactorial condition. Tinnitus can be seen as a loss of inhibition of fusiform cells in the DCN leading to increasing spontaneous activity in the central auditory system. Loss of inhibition at the fusiform cell in the DCN are especially induced by hidden hearing loss and postural instability.

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