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Systematic Review

# Exploring the Link Between Vitamin D and Calcium Levels and Hearing Disorders: A Systematic Review and Meta-Analysis

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**Abstract**

**Introduction:** Vitamin D deficiency may contribute to auditory dysfunction, including sudden sensorineural hearing loss (SSNHL) and age-related Hearing loss (HL). This study aimed to examine the association between vitamin D, calcium levels, and hearing impairments.

**Methods:** This systematic review and Meta-analysis followed PRISMA guidelines. Eligible studies were critically appraised using the Joanna Briggs Institute tools. A random-effects model was employed to account for heterogeneity across studies. Due to substantial heterogeneity, pooled estimates are interpreted cautiously as averages of widely varying effects.

**Results:** From 2,699 records screened, 33 studies were included in the systematic review, and 14 in the Meta-Analysis. Pooled analysis showed lower vitamin D levels in individuals with HL (standardized mean difference [SMD] = -0.687; 95% CI: -0.832 to -0.542; $P<0.001$; though heterogeneity was extremely high ($I^2=99.22\%$)). Subgroup analysis showed a consistent association for SNHL (SMD = -0.718; 95% CI: -0.873 to -0.564; $I^2=0.0\%$), while no significant association was found for tinnitus. Calcium levels were also low among HL patients (SMD = -0.518; 95% CI: -0.730 to -0.305; $P<0.001$; $I^2=93.79\%$), though findings varied by condition, with one SSNHL study reporting elevated calcium.

**Conclusion:** Vitamin D deficiency appears to be consistently associated with SSNHL, while evidence for tinnitus and conductive loss remains inconclusive and limited by study quality. The predominance of cross-sectional designs precludes causal inference. Future longitudinal and interventional research is needed to establish causality, clarify the role of calcium, and identify populations most likely to benefit from vitamin D screening and supplementation.

## Introduction

Hearing disorders are among the most common chronic conditions, impacting roughly one-fifth of the world's population and recognized as a leading cause of years lived with disability globally.[1] Hearing loss (HL), often defined as the inability to perceive sounds below a certain threshold in the better ear, and tinnitus, the perception of sound in the absence of an external stimulus, are highly prevalent auditory conditions.[2,3] These disorders can lead to severe consequences, including difficulties in communication, mental health problems, compromised cognitive function, reduced quality of life, and financial instability in adults, as well as developmental and educational adverse effects in children.[4,5] While noise exposure is the predominant etiology of preventable HL, it may also result from various medical conditions, including specific infections and ototoxic pharmacological agents.[6] In recent years, the effect of vitamin D levels on audiovestibular health has become an area of interest. Evidence suggests an association between vitamin D insufficiency and a range of otological disorders, including benign paroxysmal positional vertigo (BPPV), tinnitus, sudden sensorineural hearing loss (SSNHL), and age-related hearing loss.[7-10] Vitamin D is a steroid hormone synthesized from 7-dehydrocholesterol in the skin, meeting approximately 90% of the daily requirement.[11,12] Its classical role is to enhance skeletal mineralization by increasing serum calcium and phosphorus levels, preventing rickets and osteomalacia.[13-15]However, non-classic roles have become evident, including the modification of hormone secretion, regulation of the immune system, and adjustment of cellular differentiation and proliferation.[16] Consequently, vitamin D deficiency in adults is associated

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with various autoimmune diseases, metabolic disorders, and malignancies.[17,18] The potential link between vitamin D and hearing may be explained by several mechanisms. Vitamin D receptors are present in the inner ear, and deficiencies can disrupt calcium homeostasis within the cochlea, which is essential for hair cell transduction and synaptic activity.[19,20] Furthermore, vitamin D deficiency may negatively impact cochlear bone density, potentially leading to demineralization and impaired function.[19] Its anti-inflammatory and antioxidant properties may also protect against auditory nerve damage and oxidative stress implicated in conditions like SSNHL.[21,22] Given that vitamin D deficiency is a potentially modifiable risk factor through supplementation or sun exposure, understanding its role in hearing health is of significant clinical interest.

Considering the existing inconsistencies among available studies, we aimed to clarify the association between vitamin D and calcium levels and hearing impairments through a systematic review and meta-analysis, and to delineate whether these deficiencies can be considered established risk factors for hearing problems.

## Materials and Methods

This systematic review and meta-analysis were conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Statement.[23]In 2023, we conducted a comprehensive search for relevant articles across multiple databases, including PubMed, Ovid, ProQuest, Scopus, Web of Science, and Cochrane Library. The reference lists of the retrieved articles were searched, too. The keywords used were: Vitamin D, Cholecalciferol,

Hydroxycholecalciferol, Ergocalciferol, Hydroxycalciferol, Hearing Disorders. The full details of the search strategies are reported in Appendix 1.

### *Inclusion Criteria / Exclusion Criteria*

All clinical trials, cross-sectional studies, and case-control studies in which the effect of serum level of vitamin D was studied on the prevalence of different types of HL and tinnitus were included. The PICO components for this research were: 1) Participants with types of HL or tinnitus, 2) Exposure was the administration or serum levels of Vitamin D, if reported, 3) Comparison was a control group that included patients without any hearing impairment. Studies without a control group were systematically reviewed and excluded from meta-analysis. 4) Outcomes were HL, SNHL, Age-related HL or deafness, Type 2 diabetes mellitus-associated hearing impairment, and tinnitus, which were considered outcomes. Animal studies, studies without any access to full text, study protocols, studies examining the association between vestibular pathologies of the audio-vestibular system with vitamin D deficiency and withdrawn and review studies were excluded. In addition, we excluded case studies and dissertations.

### *Study Selection*

The search results were managed using EndNote software version 20, and duplicates were removed. Subsequently, three independent reviewers (K.S., A.R., T.M.) screened titles and abstracts to evaluate the inclusion criteria. The full text of selected eligible studies was thoroughly assessed by two independent reviewers (Z.S., H.S.). Any disagreements at either stage were resolved through discussion or by involving a third reviewer (Y.J.).

### *Data Extraction*

A custom-designed data extraction table was utilized. Using a modified standard JBI data extraction tool, two independent reviewers (Z.S., S.A.) extracted the data, and a third reviewer (H.S.) resolved any disagreements. This form included details such as study year, design, number of participants, age, sex, outcome (e.g., hearing loss, tinnitus), and levels of Vitamin D, calcium, parathyroid hormone (PTH), and bone mineral density (BMD).

### *Methodological Quality of Studies*

Two independent reviewers critically assessed the quality of eligible studies using the Joanna Briggs Institute (JBI) appraisal tools. Any disagreements between them were resolved through discussion with a third reviewer. Studies scoring 50% or higher on the JBI evaluation questions (available at jbi.global/critical-appraisal-tools) were deemed either "good" or "moderate" quality.

### *Meta-Analysis*

All analyses were performed using Comprehensive Meta-Analysis software (version 3). The random-effects model was used due to anticipated heterogeneity. Between-study heterogeneity was evaluated using the Cochran Q test and $I^2$ statistics. We present the results for subgroups with more than one study and describe single-study findings narratively to provide context, acknowledging that meta-analysis of single studies is not ideal

## Results

### *Study Inclusion*

The initial search retrieved 2699 articles from databases. After removing duplicates (n = 1160), the remaining 1539 records were reviewed by titles/abstracts, and 229 were evaluated by reading the full texts of eligible articles. Finally, 33 studies were systematically reviewed, and 14 were candidates for meta-analysis. Figure 1 presents the details of the screening process.

### *Study Characteristics*

The design of the included studies was cross-sectional[8-10,24-40], case-control[22,41-49], and cohort.[50-52] The total sample sizes varied between 24 and 16408 participants. The cumulative number of participants across all studies was 41,021. For clarity, we refer to groups as "affected" and "unaffected" or "exposed" and "unexposed" where appropriate, rather than uniformly using "case" and "control."

### *Type of Disease*

The disease types were Tinnitus in 10 studies[8,30,31,37,38,40-42,45,46],

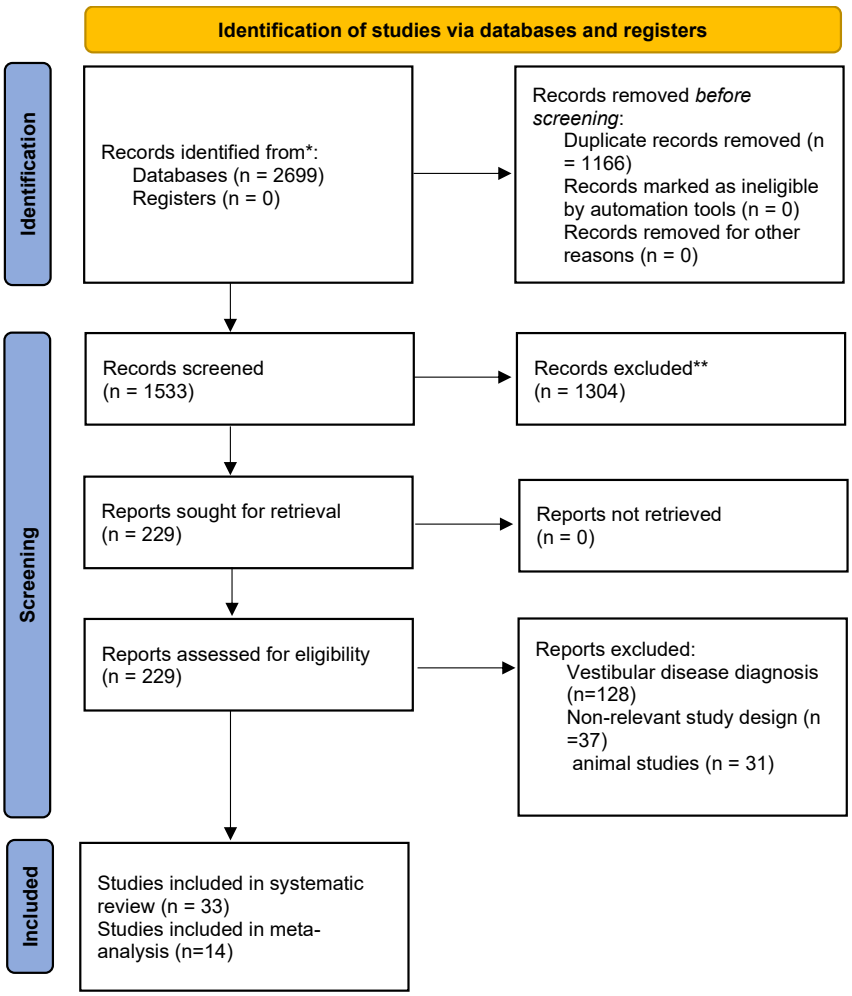



**Figure 1.** The PRISMA flow diagram shows the search and screening processes.

hearing impairment in 12 studies[10,25,27,29,30,32,35,36,51,52], SNHL in 5 studies[9,22,24,34,43,48], Hearing Loss associated with type 2 diabetes mellitus (T2DM) in 2 studies[26,33], Conductive hearing loss in two studies[47,50], disturbed cochlear health in one study[44]and senile deafness in two studies.[28,49]

### *Age and Sex*

Among the included studies, participant age ranged from 6 to 78 years. The variation in age was categorized into three subgroups: those under 60 years old[8,9,22,25,32,33,35,37,38,40-48,50,51] those over 60 years[10,27-31,34,36,49,52] and mixed populations.[24,39] The 60-year cut point was chosen to broadly distinguish studies focusing on age-related hearing loss (typically > 60) from those investigating other forms like SSNHL and tinnitus (often < 60). Researchers frequently studied tinnitus and SSNHL among younger and middle-aged adults. Conversely, age-related hearing loss and general hearing impairment were more commonly reported in the older age groups.[9,28,30-32,36,43-45,47,49]

### *Risk Factors*

Noise exposure was noted in a subset of studies (n = 9)[25,27,31,32,34,40,46,51,52], with rates ranging from 4% to 62%. Type 2 diabetes mellitus (T2DM) and hypertension were frequently reported comorbidities. These factors are relevant as they are established risk factors for hearing loss themselves; their distribution was assessed to contextualize the study populations and consider potential confounding in the observed associations with vitamin D.[27,32]

### *Comorbidities*

T2DM was frequently reported among the included studies.[10,25-29,33,35-37,42,43,51,52] Bener (2018) exclusively studied T2DM and its association with hearing impairment[33]. Furthermore, Hosseini (2020) observed the association between SSNHL and tinnitus and vitamin D deficiency in T2DM[26]. Three Other studies reported moderate diabetes prevalence ranging from 13% to over 60%.[25,36,52]

Hypertension was also frequently reported among the included studies.[10,25,27-29,33,35-37,42,45,51,52] Overall, 12 studies reported the prevalence of HTN among their participants. The highest prevalence was observed in a survey by Handoko (77.8%) and Lee (2024) (~52% in both cases and controls).[25,28] In contrast, Jung (2016) and Han (2024) reported lower rates, with values below 15%.[42,52]

### *PTH and BMD*

Among the included studies, several reported on parathyroid hormone (PTH) and bone mineral density (BMD). Jung et al. reported slightly elevated PTH levels in the group with hearing impairment compared to the unaffected group.[52] Studies by Yoo and Kim stratified participants by BMD

status (normal, osteopenia, osteoporosis), allowing for an analysis of the relationship between bone health, vitamin D, and hearing.[29,36] Table 1 represents the characteristics of the included studies.

### *Meta-Analysis Results*

#### *Vitamin D Deficiency*

The meta-analysis of vitamin D levels examined the association with various conditions of hearing loss in 14 studies. The pooled analysis suggested a significant association between lower vitamin D levels and hearing loss (SMD: -0.687; 95% CI: -0.832 to -0.542; $P<0.001$), with high heterogeneity ($I^2=99.22\%$).

#### *Subgroup Analysis of Vitamin D by Hearing Condition*

- Sensorineural Hearing Loss (SNHL): Four studies, comprising five trials, investigated this relationship. The SMD was -0.718 (95% CI: -0.873 to -0.564; $P<0.001$; $I^2$: 0.0%), indicating a consistent association[9,24,43,48].
- Hearing Loss (Unspecified/General): Four studies were combined (SMD: -0.980; 95% CI: -1.825 to -0.136; $P=0.023$; $I^2=97.31\%$)[25,33,35,52].
- Tinnitus: Three studies were combined, showing no meaningful association (SMD: 0.004; 95% CI: -1.157 to 1.166, $P=0.994$; $I^2$: 99.68%)[29,37,45].
- Conductive Hearing Loss & Disturbed Cochlear Health: Single studies investigated these outcomes, precluding meta-analysis. The study on conductive HL found no significant effect[47], while the study on disturbed cochlear health reported a large effect[44] (Figure 2).

**Table 1.** Characteristics of the included studies

| Study (Year) | Study Design | Participant Number (Case/ Control) | Outcome (Disease) | Key Findings Related to Vitamin D |
|---|---|---|---|---|
| Hosseini[26] | Cross-sectional | 200 T2DM patients (All patients; no separate control group). Groups were defined by Vit D status: Deficient/Insufficient (n=100) vs. Sufficient (n=100). | Hearing Loss (HL) in T2DM | The prevalence of hearing loss was significantly higher in the vitamin D deficient/insufficient group (90%) compared to the sufficient group (80%) ($P=0.04$). Diabetic patients with abnormal vitamin D levels had a 2.25-fold increased risk for developing hearing loss (OR=2.25, 95% CI: 0.99 to 5.09). The severity of hearing loss (mostly slight and mild) was also significantly greater in the deficient/insufficient group ($P=0.003$). |
| Fouad[41] | Case-Control | 75 / 25 | Tinnitus (in Otosclerosis) | Significant tinnitus improvement in cases treated with Risedronate (bisphosphonate)+Vit D/Calcium vs. control (Vit D/Calcium only). After 6 months, 40% of the treatment group had complete tinnitus resolution vs. 0% in the control group. The treatment group showed significant improvement in Tinnitus Handicap Inventory scores (Median: 16 vs. 52 in control). The effect was strongest in patients who had previously undergone stapedotomy (64% complete resolution). |
| Han[42] | Case-Control | 346 / 1247 | Tinnitus | Tinnitus exhibited a significant association with lower 25(OH)D levels (OR=1.028). |
| Zheng[43] | Case-Control | 310 / 154 | Sudden Sensorineural HL (SSNHL) | Vitamin D deficiency in SSNHL patients was more severe than in people with normal hearing. |
| Ghazavi[24] | Cross-sectional | 34 / 34 | SSNHL | Prevalence of vitamin D deficiency was higher in SSNHL patients. Deficient patients had the highest percentage of no response to treatment. |
| Handoko[25] | Cross-sectional | 17 (CKD patients) / 19 | Hearing Impairment | No correlation between serum 25(OH)D and hearing threshold. Not suitable as a biomarker in predialysis CKD. |
| Jung[52] | Cohort | 233 (with OP) / 776 (without OP) | Hearing Impairment | No significant HL risk or difference in hearing thresholds was observed in participants with osteoporosis. Vitamin D levels were not significantly different. |
| Kang[27] | Cross-sectional | 1910 | Hearing Impairment | Higher serum vitamin D concentrations were associated with worse hearing (higher PTA) at mid and high frequencies. |
| Karaer[44] | Case-Control | 30 (Vit D deficient) / 30 | Cochlear Health (via OAE) | Significant association between vitamin D deficiency and disturbed cochlear function. |
| Lee[28] | Cross-sectional | 651 (with HL) / 453 (without HL) | Age-Related HL (ARHL) | Lower serum vitamin D levels were associated with ARHL in the older male population, but deficiency was not a significant independent risk factor (OR=1.224, $P=0.176$). |
| Nowaczewska[45] | Case-Control | 201 / 99 | Tinnitus | Vitamin D level was significantly lower in the tinnitus group. Severe tinnitus (by THI and VAS) was associated with lower 25(OH)D levels (OR=0.11, $P<0.0001$). |

**Table 1.** Continued.

| Study (Year) | Study Design | Participant Number (Case/ Control) | Outcome (Disease) | Key Findings Related to Vitamin D |
|---|---|---|---|---|
| Sharma[46] | Case-Control | 68 / 30 | Tinnitus | Prevalence of Vit D deficiency was significantly higher in tinnitus patients (83% vs 33%), and mean Vit D level was lower. |
| Szeto[10] | Cross-sectional | 954 (Low-freq HL) / 1120 (High-freq HL) | Hearing Impairment | Vitamin D level < 20 ng/mL was associated with greater odds of low-frequency HL (OR: 2.02). |
| Zandi[9] | Cross-sectional | 50 / 50 | SSNHL | Increased prevalence of Vit D insufficiency in SSNHL subjects, suggesting a possible protective role through its antioxidant effect. |
| Yoo[29] | Cross-sectional | Normal: 1299, Osteopenia: 2388, Osteoporosis: 1174 | Hearing Impairment | Vitamin D was included as a covariate in the analysis. Hearing thresholds worsened with decreasing BMD status. |
| Dalton[51] | Cohort | At risk: 2065 / Cases: 317 | Hearing Impairment | No association was found between vitamin D levels and the 10-year incidence of hearing impairment. |
| Acra-Tolari[30] | Cross-sectional | 101 (postmenopausal women) | Hearing Impairment & Tinnitus | Vitamin D was used as a covariate in the analysis. The study reported a high prevalence of HL (64.3%). |
| Berk[31] | Cross-sectional | Control: 31, Osteopenia: 34, Osteoporosis: 34 | Tinnitus | Lower vitamin D levels were observed in the osteoporosis group compared to the control and osteopenia groups. |
| Morais-Moreno[32] | Cross-sectional | 235 (male aviation pilots) | Hearing Impairment | Reported mean vitamin D level was 28.9 ± 8.2 ng/mL in a noise-exposed cohort. |
| Atan[47] | Case-Control | 27 (Otosclerosis) / 30 | Conductive HL (Otosclerosis) | No significant differences in Vit D levels or deficiency prevalence between otosclerosis patients and healthy controls. |
| Kim[36] | Cross-sectional | Normal BMD: 102, Osteopenia: 106, Osteoporosis: 116 | Hearing Impairment | Serum vitamin D was not a significant predictor of hearing impairment (OR: 1.0, *P* value: > .05). |
| Bener[33] | Cross-sectional | 126 (T2DM with HL) / 512 (T2DM without HL) | Hearing Impairment with T2DM | Vitamin D deficiency was a risk factor for HL among diabetic patients (OR: 2.03). |
| Brookes[50] | Observational Cohort | 16 (Otosclerosis) | Otosclerosis | Calcium and vitamin D replacement therapy resulted in significant hearing improvement in 3 of 16 patients. |
| Bigman[34] | Cross-sectional (NHANES) | 3489 | SSNHL | Categorized participants by vitamin D status (Sufficient, Inadequate, Deficient). Specific results for SSNHL not fully extracted in this field. |
| Elhussieny[35] | Cross-sectional | 84 (T2DM) / 32 | Hearing Impairment in T2DM | Serum 25(OH)D level was an independent predictor of hearing loss among diabetic patients (β = -0.605; *P* = 0.041). |
| Abdelmawgoud[8] | Cross-sectional + Intervention | 35 (Tinnitus) | Tinnitus | Vitamin D supplementation (50,000 IU/week for 3 months) significantly improved Vit D levels and reduced Tinnitus Handicap Inventory (THI) scores. |
| Bulla[38] | Cross-sectional | 200 (Tinnitus) | Tinnitus & Hearing Threshold | A complex interaction with age was found. At older ages (> 60), higher vitamin D levels were linked to lower hearing thresholds. |
| Aliyeva[37] | Cross-sectional | 3615 (Tinnitus) / 12793 | Tinnitus | Individuals with deficient vitamin D levels and limited sun exposure had a significantly elevated risk for developing tinnitus. |
| Ikeda[39] | Cross-sectional | 28 | SSNHL | Reported a high prevalence of vitamin D deficiency (82.1%) in SSNHL patients. |
| Yamazaki[49] | case-control | 56 | Senile Deafness | Treatment with 1α(OH)-D3 resulted in hearing improvement in 6 ears in 4 of 12 treated patients. |
| Kose Celebi[48] | Case-Control | 52 / 52 | SSNHL | The mean vitamin D value in the SSNHL patient group was statistically significantly lower than in the control group (*P* < .05). |
| Yilmaz[40] | Cross-sectional | 224 | Tinnitus | 12.1% of tinnitus patients had comorbid metabolic disorders, including vitamin D deficiency. |
| Paprocki[22] | Case-Control | 12 (SSNHL + Vit D) / 12 (SSNHL) | SSNHL | Vitamin D supplementation during HBO therapy reduced oxidative stress markers but did not result in a statistically significant difference in hearing outcomes between groups. |

PTA = Pure Tone Average (measure of hearing threshold); OAE = Otoacoustic Emissions (test of outer hair cell function); THI = Tinnitus Handicap Inventory (questionnaire assessing tinnitus severity); VAS = Visual Analogue Scale (subjective rating scale); DPOAE = Distortion Product Otoacoustic Emissions; HL = Hearing Loss; SNHL = Sensorineural Hearing Loss; SSNHL = Sudden Sensorineural Hearing Loss; BPPV = Benign Paroxysmal Positional Vertigo; T2DM = Type 2 Diabetes Mellitus; PTH = Parathyroid Hormone; BMD = Bone Mineral Density.

### *Serum Calcium Levels*

Across the included studies with reported calcium levels, serum calcium levels in individuals with hearing impairment generally remained within the normal reference range (8.5–10.5 mg/dL)[9,10,30,31,33,35,49]. While one study reported a slightly lower mean in the diabetic hearing-impaired group, most values showed no significant changes[33].

The meta-analysis of calcium levels included four studies. The pooled analysis revealed an overall effect size of -0.518 (95% CI: -0.730 to -0.305; $P<0.001$; $I^2$: 93.79%), suggesting a trend toward lower calcium levels in individuals with hearing loss conditions.

### *Subgroup Analysis of Calcium by Hearing Conditions*

SNHL and Senile Deafness: Single studies reported on these conditions. The study on SNHL reported higher calcium levels in affected individuals[9], while the study on senile deafness reported lower levels[49].

Hearing Loss (Unspecified/General): Two studies[33,35] showed an association with lower calcium levels (SMD: -0.994; 95% CI: -1.326 to -0.662; $P<0.001$) (Figure 3).

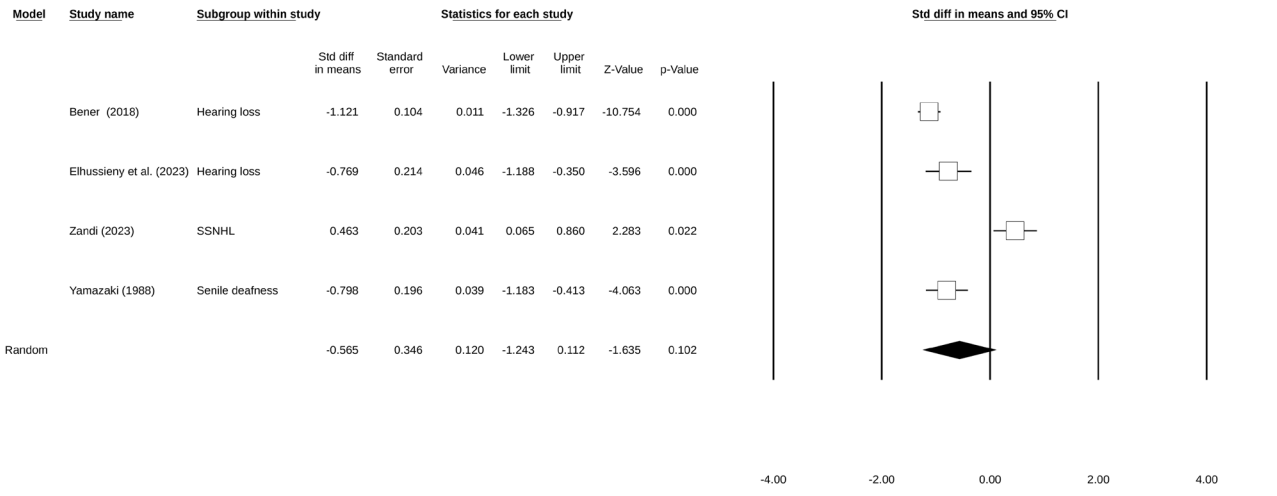



**Figure 2.** The Link Between Vitamin D Level and Hearing Disorders

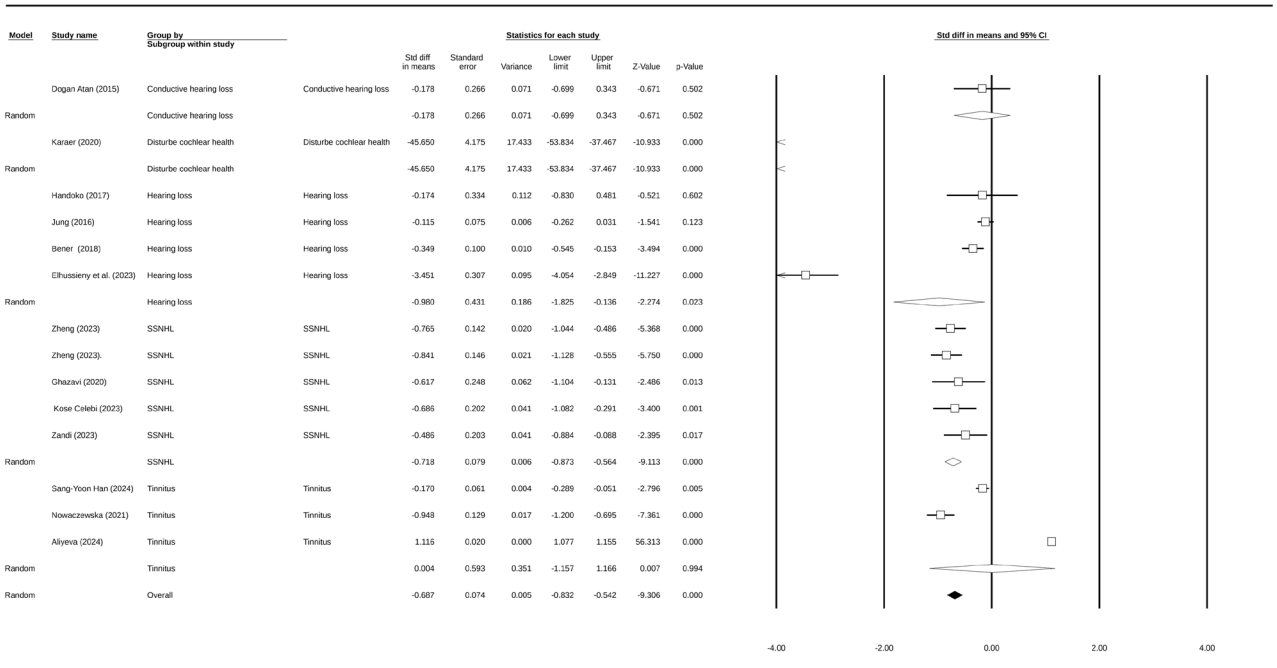



**Figure 3.** The Link Between Calcium Level and Hearing Disorders

### *Quality Assessment of the Included Studies*

The quality assessment, conducted using JBI critical appraisal tools, revealed notable variability. Among cross-sectional studies, most clearly defined inclusion criteria and described study subjects, but limitations were apparent in the identification and handling of confounding factors. For case-control studies, half failed to ensure comparability between groups at baseline. The cohort studies by Jung and Dalton demonstrated robust methodology, while Brookes exhibited significant limitations. These quality issues, particularly regarding confounding, should be considered when interpreting the results (Table 2).

## Discussion

Our results suggest that vitamin D deficiency may be associated with certain types of hearing impairment, although findings vary across conditions. There is notable evidence of a strong association with disturbed cochlear health; however, this is based on a single study. A consistent association was observed in cases of SNHL, with affected individuals exhibiting lower levels of vitamin D. In contrast, no meaningful association was identified for conductive hearing loss. The relationship between vitamin D levels and tinnitus remains unclear, and current evidence does not support a reliable link.

This review also examined the relationship between calcium levels and various hearing conditions. Overall, the findings suggest that lower calcium levels may be associated with hearing loss, with the exception of SNHL. Specifically, individuals with unspecified hearing loss or age-related hearing decline (senile deafness) tended to have reduced calcium levels. However, one study on SSNHL reported an opposite pattern, with higher calcium levels observed in affected individuals. These contrasting findings, along with moderate to high variability across studies, suggest the presence of condition-specific mechanisms or differences in study populations. Several explanations exist for how vitamin D deficiency affects the auditory system.

One key factor is the established role of vitamin D in regulating calcium homeostasis and cochlear function.[20,39] Vitamin D and its active form, $1,25(OH)_2D$, regulate calcium homeostasis by activating the vitamin D receptor (VDR), primarily enhancing intestinal absorption and modulating calcium balance in the kidneys and bones.[53] Then, calcium is essential for cochlear function, including hair cell transduction and synaptic activity, which may explain its association with specific hearing impairments.[54] Notably, vitamin D receptors have been identified within the calcium transport systems of the inner ear, where they play a crucial role in maintaining calcium homeostasis. Additionally, a deficiency in vitamin D can negatively impact cochlear bone density, potentially leading to cochlear demineralization, impaired microcirculation, disrupted calcium metabolism, and structural changes in the cochlea that ultimately result in hearing decline.[20,39] Another potential mechanism involves the ability of vitamin D to cross the Blood-Brain barrier (BBB). The uneven distribution of vitamin D across various brain regions influences the proliferation and differentiation of neural stem cells and progenitors. In the context of vitamin D deficiency, such disruptions can impair neurogenesis, potentially affecting vestibuloauditory neural pathways and contributing to hearing decline.[55-57]

Endocrine disorders, particularly those affecting the thyroid and parathyroid glands, often coexist with hearing disorders. Vitamin D may act as a mediating factor in this relationship. Hypoparathyroidism leads to reduced levels of 1,25-dihydroxyvitamin D, resulting in hypocalcemia. Therefore, it is reasonable to consider both low calcium and vitamin D deficiency as contributing factors in the development of hearing loss. Consequently, dysfunction of the inner ear is likely linked to decreased calcium concentrations in the inner ear fluids and/or insufficient systemic vitamin D levels.[58] Similarly, in hypothyroidism, even with the autoimmune origin as Hashimoto's, patients often report tinnitus (26%) and hearing loss (20%). Then, after thyroxine therapy, many patients exhibited improvements in their symptoms and pure tone audiometry results.[59,60] Although the exact molecular interaction between vitamin D and the thyroid remains unclear, a substantial body of evidence suggests a strong association between low vitamin D levels and an increased likelihood of developing or having hypothyroidism.[61] Considering the frequent coexistence of vitamin D deficiency in individuals with hypothyroidism and its essential role in calcium regulation, maintaining cochlear integrity, and neural function, it is potentially plausible that vitamin D deficiency contributes to auditory symptoms. However, further mechanistic and longitudinal studies are needed to clarify causality.

In addition to its crucial role in regulating calcium balance and metabolism, vitamin D also reduces inflammation and influences immune system activity.[62,63] Vitamin D enhances the innate immune response while suppressing the adaptive immune system, highlighting its potential role in reducing autoimmune and autoinflammatory activities.[64] Vitamin D levels are inversely related to C-reactive protein (CRP), a marker of inflammation, and supplementation has been shown to reduce CRP and other inflammatory mediators. Its anti-inflammatory effects also include suppressing pro-inflammatory cytokines and modulating T-cell activity. These immune-regulating properties may help mitigate the severity of tinnitus associated with otitis media, particularly in individuals with vitamin D deficiency. Therefore, maintaining adequate vitamin D levels may support the management of both otitis and associated tinnitus.[65,66] On the other hand, tinnitus often coexists with sensorineural hearing loss (SNHL). Since neuro-inflammatory mechanisms (neuropathies/neuroepitheliopathies) are involved in hearing loss and tinnitus pathogenesis, the anti-inflammatory role of vitamin D may play a significant role.[67,68] Studies have shown that patients with SSHNL often exhibit lower vitamin D levels and less favorable responses to treatment.[69,70] Antioxidant vitamins such as A, C, and E may serve as supportive treatments for SSNHL. Free radicals, including reactive oxygen species

**Table 2.** Quality assessment of the included studies using JBI critical appraisal checklists

| Study (Year) | Q1 | Q2 | Q3 | Q4 | Q5 | Q6 | Q7 | Q8 | Q9 | Q10 | Q11 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Cross-sectional Studies** | | | | | | | | | | | |
| Ghazavi[24] | Yes | Yes | Unclear | Yes | No | No | Yes | Yes | | | |
| Hosseini[26] | Yes | Yes | Yes | Yes | Unclear | No | Yes | Unclear | | | |
| Handoko[25] | Yes | Yes | Unclear | Yes | Unclear | No | Yes | Yes | | | |
| Kang[27] | Yes | Yes | Unclear | Yes | Yes | Yes | Yes | Yes | | | |
| Lee[28] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Szeto[10] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Zandi[9] | Yes | Yes | Yes | Unclear | No | No | Unclear | Yes | | | |
| Yoo[29] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Acra-Tolari[30] | No | Yes | Yes | Yes | Unclear | No | No | No | | | |
| Berk[31] | Yes | Yes | Yes | Yes | Yes | No | Yes | Yes | | | |
| Morais-Moreno[32] | Yes | Yes | Unclear | Yes | Yes | Yes | Yes | Yes | | | |
| Kim[36] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Bener[33] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Bigman[34] | Yes | Yes | Yes | Yes | Unclear | Yes | Yes | Yes | | | |
| Yilmaz[40] | Yes | Yes | Unclear | Unclear | Unclear | No | Unclear | Yes | | | |
| Elhussieny[35] | Yes | Yes | Yes | Yes | Unclear | Yes | Yes | Yes | | | |
| Abdelmawgoud[8] | Yes | Unclear | Yes | Yes | Unclear | No | Yes | Unclear | | | |
| Bulla[38] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Aliyeva[37] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | | | |
| Ikeda[39] | Unclear | No | Unclear | Unclear | No | No | Unclear | No | | | |
| Questions: 1.Were the criteria for inclusion in the sample clearly defined? 2.Were the study subjects and the setting described in detail? 3.Was the exposure measured in a valid and reliable way? 4.Were objective, standard criteria used for measurement of the condition? 5.Were confounding factors identified? 6.Were strategies to deal with confounding factors stated? 7.Were the outcomes measured in a valid and reliable way?8.Was appropriate statistical analysis used? | | | | | | | | | | | |
| **Case-Control studies** | | | | | | | | | | | |
| Nowaczewska[45] | No | Yes | Yes | Yes | Yes | No | Yes | Yes | Unclear | Yes | |
| Sharma[46] | No | Yes | Yes | Yes | Yes | Unclear | No | Yes | Yes | Yes | |
| Karaer[44] | No | No | Yes | Yes | Yes | No | No | Yes | Unclear | Yes | |
| Han[42] | No | Yes | Yes | Yes | No | No | Yes | Unclear | Yes | Unclear | |
| Zheng[43] | Yes | No | Yes | Yes | Yes | Unclear | No | Yes | Unclear | Yes | |
| Fouad[41] | No | No | Yes | Yes | Yes | No | No | Yes | Unclear | Yes | |
| Kose Celebi[48] | Yes | Yes | Unclear | Yes | Yes | No | No | Yes | Yes | NA | |
| Paprocki[22] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | |
| Atan[47] | Yes | Yes | Unclear | Yes | Yes | No | No | Yes | Yes | NA | |
| Yamazaki[49] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | |
| Questions: 1.Were the groups comparable other than the presence of disease in cases or the absence of disease in controls? 2.Were cases and controls matched appropriately? 3.Were the same criteria used for identification of cases and controls? 4.Was exposure measured in a standard, valid and reliable way? 5.Was exposure measured in the same way for cases and controls? 6.Were confounding factors identified? 7.Were strategies to deal with confounding factors stated? 8.Were outcomes assessed in a standard, valid and reliable way for cases and controls? 9.Was the exposure period of interest long enough to be meaningful? 10.Was appropriate statistical analysis used? | | | | | | | | | | | |
| **Cohort Studies** | | | | | | | | | | | |
| Brookes[50] | NA | NA | Yes | No | No | No | Yes | Yes | Yes | NA | Unclear |
| Jung[52] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Dalton[51] | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Questions: 1.Were the two groups similar and recruited from the same population? 2.Were the exposures measured similarly to assign people to both exposed and unexposed groups? 3.Was the exposure measured in a valid and reliable way? 4.Were confounding factors identified? 5.Were strategies to deal with confounding factors stated? 6.Were the groups/participants free of the outcome at the start of the study (or at the moment of exposure)? 7.Were the outcomes measured in a valid and reliable way? 8.Was the follow up time reported and sufficient to be long enough for outcomes to occur? 9.Was follow up complete, and if not, were the reasons to loss to follow up described and explored? 10.Were strategies to address incomplete follow up utilized? 11.Was appropriate statistical analysis used? | | | | | | | | | | | |

(ROS) and nitric oxide, can damage the auditory system, particularly the outer hair cells. These molecules are naturally produced during mitochondrial respiration but can cause cellular harm when present in excess. Antioxidants help protect cells by neutralizing ROS and reducing oxidative stress.[71-73] In terms of age-related HL, these patients have lower serum BDNF levels, suggesting a neuroinflammatory process. Based on the neuroprotective role of vitamin D, these individuals may also benefit from vitamin D supplementation.[74] More recent studies could be advantageous in this regard.

Our systematic review and meta-analysis found a significant overall association between lower levels of vitamin D and hearing loss, a finding supported by a large, combined sample size. However, the high degree of heterogeneity and variable quality of the included studies necessitate a cautious and nuanced interpretation. The subgroup analyses suggest that this association may be specific to certain types of hearing loss. We observed a consistent and significant association for SNHL, with low heterogeneity across studies. This is biologically plausible given the role of vitamin D in calcium regulation within the inner ear and its neuroprotective and anti-inflammatory properties. In contrast, no significant association was found for conductive hearing loss, which is often caused by mechanical problems in the outer or middle ear, or for tinnitus, a complex condition with diverse etiologies. The single study reporting a strong association with "disturbed cochlear health" requires replication. The relationship between calcium and hearing loss also appears complex. While the overall meta-analysis suggested an association with lower calcium levels, the findings were inconsistent across specific hearing conditions. The opposing result in the SNHL subgroup highlights the need for further research to understand the precise role of calcium homeostasis in different auditory pathologies.

The contrasting finding of elevated calcium in one SSNHL study, while others showed lower levels, merits further consideration. Several mechanisms might explain this discrepancy.

First, acute cellular stress during sudden hearing loss could trigger calcium release from intracellular stores or disrupted cell membranes, leading to transiently elevated serum levels. Second, the timing of calcium measurement relative to symptom onset may be critical initial calcium spikes might be followed by depletion over time. Third, SSNHL is a heterogeneous condition with multiple potential etiologies (viral, vascular, autoimmune), and calcium dysregulation may be relevant only in specific subtypes. Finally, differences in how studies handled potential confounders such as PTH levels, renal function, and medications that affect calcium homeostasis could explain inconsistent findings. Future studies should standardize measurement timing and report detailed clinical phenotyping of SSNHL cases

The quality assessment revealed important limitations in existing literature, that may influence the interpretation of our findings. Among case-control studies, approximately half failed to ensure comparability between groups at baseline, and confounding factors such as noise exposure, diabetes, hypertension, and smoking were inadequately addressed in most studies. This failure to control established risk factors for hearing loss means that the observed effect sizes for vitamin D may be inflated or confounded low vitamin D levels may simply be a marker of overall poorer health or higher comorbidity burden rather than an independent causal factor. Similarly, in cross-sectional studies, the lack of adjustment for key confounders limits the ability to isolate the specific contribution of vitamin D to hearing outcomes. These quality issues should be considered when evaluating the strength of the evidence. Furthermore, the predominance of cross-sectional studies limits any inference of causality; it remains unclear whether vitamin D deficiency contributes to hearing loss or is merely a marker of poor general health associated with hearing decline. The very high heterogeneity observed in our main analyses ($I^2 > 90\%$) warrants cautious interpretation. While the random-effects model accounts for between-study variability, the pooled estimates represent an average of widely differing effects across diverse populations and may not be representative of any single clinical setting. This heterogeneity likely reflects differences in study populations, definitions of hearing loss, measurement methods for vitamin D, and varying control for confounding factors. Therefore, these summary effects should be viewed as hypothesis-generating rather than definitive. The very high heterogeneity in most of our analyses underscores the substantial clinical and methodological diversity among the studies. Differences in population characteristics, definitions of hearing loss, and methods for measuring vitamin D likely contributed to this variability. A critical limitation of the current evidence base is the predominance of cross-sectional designs, which precludes any inference of causality. The observed association between low vitamin D and hearing loss may reflect reverse causality: individuals with hearing loss may have poorer overall health, reduced mobility, and less sun exposure, leading to vitamin D deficiency rather than deficiency causing hearing loss. Similarly, poor nutritional status and multimorbidity in older adults who are already at higher risk for hearing loss, may confound this relationship. For a health promotion audience, it is essential to distinguish between association and causation: while vitamin D deficiency may contribute to hearing pathology, it could equally be a marker of general health status. Longitudinal studies and randomized controlled trials are urgently needed to establish directionality. While the random-effects model accounts for this, it also means the pooled estimate should be interpreted as an average of widely differing effects. Despite these limitations, the potential public health impact is significant. If a causal relationship is established, correcting vitamin D deficiency could represent a simple, low-cost, and low-risk intervention to reduce the burden of hearing loss. The relative safety and systemic benefits of vitamin D supplementation further support the rationale for investigating this link.

Beyond the methodological limitations of this review, several fundamental gaps in primary literature constrain our conclusions. First, the lack of longitudinal data is a critical weakness; without prospective cohort studies tracking vitamin D levels and hearing outcomes over time, we cannot determine temporal relationships or establish causality. Second, outcome definitions vary considerably across studies; hearing loss is assessed using different frequency ranges, thresholds, and diagnostic criteria, making cross-study comparisons challenging. Third, confounding control is consistently inadequate; few studies measure and adjust for all major risk factors for hearing loss, including noise exposure history, occupational exposures, smoking, alcohol use, cardiovascular risk factors, and ototoxic medication use. Fourth, vitamin D assessment methods differ (e.g., various immunoassays vs. mass spectrometry), and timing of measurement relative to disease onset is often unreported. Fifth, publication bias remains a concern, as studies with null findings may be less likely to be published. Finally, the overwhelming reliance on cross-sectional designs from diverse global populations with varying sun exposure, dietary habits, and genetic backgrounds introduces unmeasured confounding that cannot be resolved without more rigorous study designs.

If future high-quality longitudinal studies and RCTs confirm a causal role for vitamin D in hearing health, several public health strategies could be considered. First, promoting vitamin D sufficiency through safe sun exposure (10-30 minutes midday, several times per week) and dietary advice (including fatty fish, fortified dairy products, and supplements where appropriate) represents a simple, low-cost intervention with multiple established health benefits beyond potential hearing protection. Second, targeted screening and supplementation in at-risk populations such as older adults, individuals with diabetes, those with limited sun exposure, and patients presenting with unexplained SSNHL could be evaluated in clinical settings. Third, given the relative safety and low cost of vitamin D supplementation, even modest protective effects on hearing could yield significant population-level benefits. However, we emphasize that such recommendations must await confirmation from adequately powered randomized controlled trials designed specifically to assess hearing outcomes as primary endpoints. In the meantime, maintaining vitamin D sufficiency for overall health remains a prudent recommendation supported by existing evidence.

Future research should prioritize: (1) prospective cohort studies with serial vitamin D and hearing measurements; (2) RCTs of vitamin D supplementation with hearing outcomes as primary endpoints; (3) standardized definitions of hearing loss and consistent measurement methods; (4) adequate control for established confounders including noise exposure, comorbidities, and medications; and (5) investigation of potential effect modifiers such as age, sex, ethnicity, and genetic factors influencing vitamin D metabolism. Such studies will be essential to determine whether vitamin D screening and supplementation should become part of routine hearing health care.

## Conclusion

This review provides evidence suggestive of an association between vitamin D deficiency and sensorineural hearing loss, with a consistent effect size across studies and low heterogeneity in this subgroup. The evidence for other auditory conditions like tinnitus is currently weak and inconclusive. The role of calcium is less clear and may be condition-specific, with the possibility of different mechanisms operating in acute versus chronic hearing disorders.

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### Competing Interests

None.

### Data Availability Statement

Not applicable.

### Ethical Approval

This study obtained an ethics code from Tabriz University of Medical Sciences (IR.TBZMED.REC.1403.400)

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**Appendix 1.** Search strategy

| Search number | Query | Sort By | Filters | Results |
|---|---|---|---|---|
| 3 | #1 AND #2 | | | 288 |
| 2 | ((((((((("Vitamin D"[Mesh]) OR (Vitamin D*[Text Word])) OR (Vitamin-D*[Text Word])) OR (VitaminD*[Text Word])) OR (Vit D*[Text Word])) OR (Vit-D*[Text Word])) OR (Vit. D*[Text Word])) OR (Cholecalciferol*[Text Word])) OR (Hydroxycholecalciferol*[Text Word])) OR (Ergocalciferol*[Text Word])) OR (Hydroxycalciferol*[Text Word]) | | | 112,410 |
| 1 | ((((((((((((((((((((((((("Hearing Disorders"[Mesh]) OR ("Meniere Disease"[Mesh])) OR ("Persons With Hearing Impairments"[Mesh])) OR ("Tinnitus"[Mesh])) OR ("Labyrinth Diseases"[Mesh])) OR ("Endolymphatic Hydrops"[Mesh])) OR (Hearing Disorder*[Text Word])) OR (Distorted Hearing[Text Word])) OR (Dysacusis[Text Word])) OR (Paracousis[Text Word])) OR (Paracusis[Text Word])) OR (Meniere* Disease*[Text Word])) OR (Ménière* Disease*[Text Word])) OR (Hearing Impair*[Text Word])) OR (Hypoacusis[Text Word])) OR (Hypoacuses[Text Word])) OR (hearing loss*[Text Word])) OR (hearing dysfunction*[Text Word])) OR (Deaf*[Text Word])) OR (Hard Hearing Person*[Text Word])) OR (Hearing Disabled Person*[Text Word])) OR (Hearing Impaired Person*[Text Word])) OR (Presbycus*[Text Word])) OR (auditory defect*[Text Word])) OR (auditory deficit*[Text Word])) OR (Tinnitus[Text Word])) OR (Labyrinth Disease[Text Word])) OR (Inner Ear Disease*[Text Word])) OR (Endolymphatic Hydrops[Text Word]) | | | 168,379 |