Association between Vitamin D Deficiency and Hearing Loss: A Systematic Review of Current Evidences

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ABSTRACT

Background: Vitamin D receptors in inner ear tissues suggest a potential link between vitamin D status and auditory function. This systematic review synthesizes current evidence on the association between vitamin D deficiency and hearing loss, and evaluates supplementation efficacy.

Methods: A systematic search of PubMed/MEDLINE, Scopus, Embase, and Web of Science (2021–2026) identified observational studies and randomized controlled trials examining serum 25-hydroxyvitamin D levels and audiometric outcomes. Quality was assessed using Newcastle-Ottawa Scale and Cochrane RoB 2.0. A narrative synthesis was performed due to heterogeneity.

Results: Eight studies were included (four cross-sectional, one prospective cohort, one retrospective case-control, two RCTs). Observational studies consistently found significant associations between low vitamin D (<20 ng/mL) and hearing impairment (adjusted ORs: 1.45–2.02), particularly for bilateral sensorineural low-frequency loss. Associations were stronger in males (OR: 1.638) and younger adults (<30 years, deficiency rate: 60%). Two RCTs reported significant hearing recovery with adjunctive vitamin D supplementation (82.0% vs 52.9% effective rate, $p<0.001$), but one cohort found no benefit from post-diagnosis intervention, suggesting chronic maintenance may be more critical than acute supplementation.
Conclusion: Evidence supports a significant association between vitamin D deficiency and hearing loss, with plausible mechanisms involving cochlear calcium homeostasis and immunomodulation. While supplementation may benefit deficient sudden sensorineural hearing loss patients, optimal timing remains unclear. Routine serum 25(OH)D screening is advisable for at-risk populations, including the elderly, young adults with sudden hearing loss, and males. Future prospective studies with standardized protocols are needed to establish causality and refine clinical recommendations.

Keywords: Vitamin D Deficiency; Hearing Loss; Sensorineural Hearing Loss; Sudden Sensorineural Hearing Loss; 25-Hydroxyvitamin D.

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INTRODUCTION

Vitamin D, a fat-soluble secosteroid, plays a pivotal role in numerous physiological processes extending far beyond its classical function in calcium homeostasis and bone metabolism. Over the past two decades, a growing body of evidence has implicated vitamin D in the regulation of immune function, inflammatory responses, cellular proliferation, and neuromuscular function [1]. More recently, the presence of vitamin D receptors (VDR) in various tissues throughout the body—including the inner ear—has sparked considerable scientific interest in understanding the potential relationship between vitamin D status and auditory function [2].

Vitamin D deficiency has emerged as one of the most prevalent nutritional deficiencies worldwide, affecting populations across all age groups, geographic regions, and socioeconomic strata. The classification of vitamin D status relies on serum 25-hydroxyvitamin D (25(OH)D) concentrations [3]. According to current consensus, levels below 20 ng/mL (50 NRol/L) are classified as deficient, concentrations between 20 and 30 ng/mL (50–75 NRol/L) as insufficient, and levels above 30 ng/mL (≥75 NRol/L) as sufficient. Some authorities define severe deficiency as levels below 12 ng/mL (30 NRol/L), with insufficiency ranging from 12–20 ng/mL (30–50 NRol/L). Regardless of the specific threshold applied, the evidence consistently demonstrates that a substantial proportion of the global population fails to achieve optimal vitamin D status [4].

Several interconnected factors contribute to this high global prevalence. Approximately 80% of the body's vitamin D is synthesized through skin exposure to ultraviolet B (UVB) radiation; however, modern lifestyles characterized by increased indoor occupation, widespread sunscreen use, geographic latitude, seasonal variations, and limited outdoor activity have drastically reduced cutaneous synthesis [6].

The remaining 20% is obtained through dietary sources, but natural food sources rich in vitamin D—such as fatty fish, liver, and egg yolks—are infrequently consumed in many populations. Additional risk factors include advancing age (with cutaneous synthesis capacity declining significantly with age), darker skin pigmentation (which reduces UVB penetration), obesity (which sequesters vitamin D in adipose tissue), malabsorptive conditions, and certain medications [7].

Hearing loss represents another major global public health challenge, with profound implications for quality of life, social participation, cognitive function, and economic productivity. According to the World Health Organization (WHO), hearing loss affects over 1.5 billion people, accounting for approximately 20% of the global population. Current estimates suggest that around 20% of the global population is living with hearing loss, with nearly 430 million individuals suffering from disabling hearing loss [6]. Sensorineural hearing loss (SNHL) constitutes the most common form of hearing impairment, characterized by damage to the cochlea, auditory nerve, or central auditory pathways. The etiological spectrum of SNHL is broad, encompassing genetic factors, aging (presbycusis), noise exposure, ototoxic medications, infections, autoimmune conditions, and metabolic disorders. Despite advances in understanding the pathophysiology of SNHL, a significant proportion of cases remain idiopathic, highlighting the need for continued research into modifiable risk factors and potential preventive strategies [8].

The biological plausibility of an association between vitamin D deficiency and hearing loss is supported by multiple lines of evidence. The presence of vitamin D receptors (VDR) in the inner ear provides a direct mechanistic link. Immunohistochemical analyses of human inner ear tissues have demonstrated that VDR expression is present throughout early development and persists in various inner ear structures, with the strongest expression observed in the non-sensory epithelium. This receptor expression suggests that vitamin D may play a regulatory role in inner ear development, maintenance, and function. Studies in animal models have further confirmed VDR expression in spiral ganglion neurons of the rat inner ear, where vitamin D receptor activation has been shown to upregulate the expression of brain natriuretic peptide (BNP) and enhance its function [5].

Vitamin D plays a critical role in calcium metabolism, and calcium homeostasis is essential for normal auditory function. Calcium ions are fundamental to the mechanoelectrical transduction process in hair cells, neurotransmitter release at the synapse between hair cells and auditory nerve fibers, and the generation and propagation of action potentials along the auditory pathway. Vitamin D deficiency can lead to hypocalcemia, which may disrupt these calcium-dependent processes. Experimental studies in animal models have demonstrated that vitamin D deficiency can cause depression of ionized calcium concentration in the perilymph, leading to alterations in cochlear potentials and ultimately contributing to hearing impairment. These changes in cochlear potentials are thought to arise from alterations in calcium ion concentrations in inner ear fluids or from the direct influence of vitamin D deficiency and secondary hyperparathyroidism on the inner ear [3, 8]. This study aims to systematically review current evidence on the association between vitamin D deficiency and hearing loss, identifying potential mechanisms, and assessing the effectiveness of vitamin D supplementation for hearing-related outcomes.

METHODOLOGY

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [10].

Search Strategy and Information Sources

A systematic literature search was performed across four major electronic databases: PubMed/MEDLINE, Scopus, Embase, and Web of Science. The search was restricted to a five-year period, spanning from January 2021 to December 2026, to capture contemporary and methodologically robust evidence. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords using Boolean operators, including terms such as "vitamin D," "25-hydroxyvitamin D," "hearing loss," "sensorineural hearing loss," "sudden hearing loss," "age-related hearing loss," "presbycusis," and "auditory impairment". The search syntax was adapted for the specific indexing and command structure of each database. Furthermore, manual screening of reference lists of included studies and relevant narrative reviews was conducted to identify any additional eligible records.

Eligibility Criteria

Studies were deemed eligible based on the PICOS framework. Regarding Population (P), studies involving human participants with documented hearing loss, including sudden sensorineural hearing loss (SSNHL) or age-related hearing loss (ARHL), were included. For Intervention/Exposure (I), studies measuring serum 25-hydroxyvitamin D [25(OH)D] levels or evaluating the therapeutic effect of vitamin D supplementation were considered. The Comparator (C) required the presence of a control group without hearing loss or without vitamin D deficiency. The Outcome (O) included audiological assessments measured by pure-tone audiometry (PTA) or defined hearing impairment thresholds. The Study design (S) encompassed cross-sectional studies, prospective and retrospective cohorts, case-control studies, and randomized controlled trials (RCTs). Exclusion criteria comprised animal studies, case reports, conference abstracts, non-English publications, and studies where full texts were unavailable or that did not report audiological outcomes as primary variables.

Study Selection

The retrieved records from all databases were exported to Rayyan, a web-based systematic review software [11], which facilitated the screening process through its machine-learning capabilities and collaborative features. After the automatic removal of duplicate records, two independent reviewers screened the titles and abstracts against the predefined eligibility criteria. Subsequently, the full texts of potentially relevant studies were retrieved and assessed independently by the same reviewers against the full eligibility criteria. Any disagreements between reviewers at either stage were resolved through consensus or by consultation with a third senior reviewer.

Data Extraction

A standardized data extraction form was developed a priori to systematically collate information from each included study. Two reviewers independently extracted the following data: study identification (author, year, and reference), geographical location, study design, sample size, participant demographics (age, sex, and population characteristics), vitamin D assessment method and deficiency definition, hearing evaluation tools and frequency ranges, main statistical outcomes (adjusted odds ratios, mean differences, or p-values), and key conclusions. Any discrepancies in extracted data were resolved through discussion and consensus. Missing or unreported data were marked as "not reported" (NR).

Quality and Risk of Bias Assessment

The methodological quality and risk of bias of the included studies were evaluated using standardized, validated tools tailored to each specific study design. For the observational studies (cross-sectional, cohort, and case-control designs), the Newcastle-Ottawa Scale (NOS) was employed [12]. The NOS assesses studies across three domains: selection of participants, comparability of groups, and ascertaiNRent of exposure or outcome, awarding stars for quality indicators with a maximum score of 9. Scores of 7–9 were considered low risk, 5–6 moderate risk, and <5 high risk. For the randomized controlled trials, the Cochrane Risk of Bias Tool (RoB 2.0) was utilized [13], evaluating domains including randomization process, deviations from intended interventions, missing outcome data, measurement of outcomes, and selection of reported results. The overall risk was classified as "low risk," "some concerns," or "high risk." All assessments were conducted independently by two reviewers, with disagreements resolved by consensus.

Data Synthesis

Due to the considerable clinical and methodological heterogeneity among the included studies regarding study designs, participant populations, vitamin D thresholds, and outcome measurements, a narrative synthesis approach was adopted rather than a meta-analysis.

The narrative synthesis was structured according to the type of hearing loss (ARHL versus SSNHL), the study design (observational versus interventional), and the direction and magnitude of the reported associations. Study findings were summarized descriptively in tabular format, and the synthesis focused on identifying consistent patterns, exploring contradictions, and evaluating the strength of the overall evidence base.

RESULTS

PRISMA flow diagram illustrates the systematic study selection process for this review. A total of 218 records were initially identified through database searches. After removing 91 duplicate records, 127 records proceeded to title and abstract screening, of which 59 were excluded based on eligibility criteria. The remaining 68 reports were sought for retrieval, but 52 could not be obtained, leaving 16 reports assessed for full-text eligibility. Of these, 8 reports were excluded for the following reasons: 5 had wrong outcomes, 2 involved wrong populations, and 1 was an abstract only. Ultimately, 8 studies met all inclusion criteria and were included in the final systematic review.

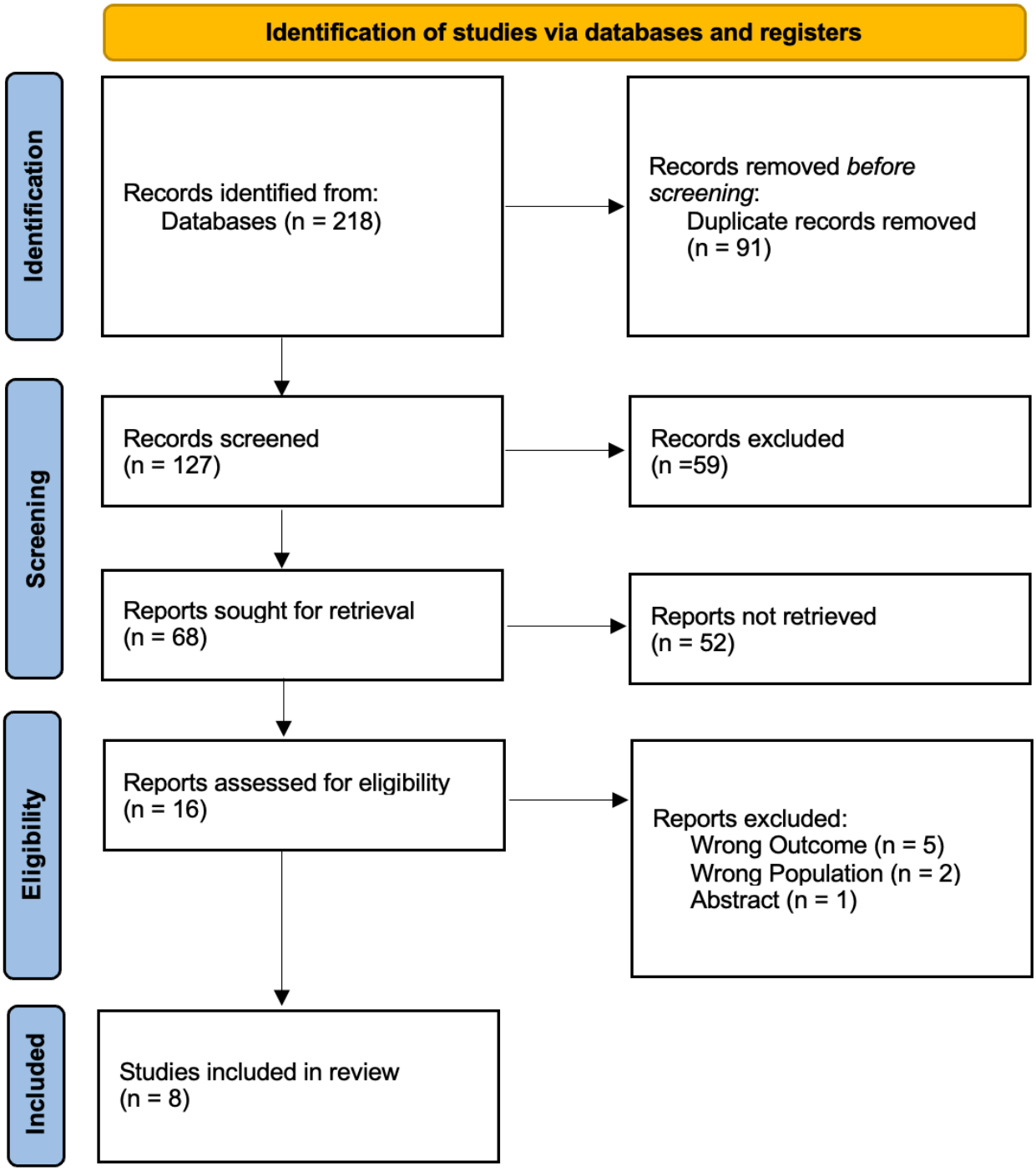

Figure 1: PRISMA Flow Diagram of Study Selection Process.

Table 1 systematically summarizes the fundamental characteristics and demographic data of the eight studies included in this systematic review, the included evidence exhibits substantial heterogeneity, encompassing cross-sectional studies [6,14,16,17], a prospective cohort study [15], a retrospective case-control study [18], and two prospective randomized controlled trials (RCTs) [3,19]. This methodological diversity is advantageous for a systematic review as it allows for the synthesis of evidence across different levels of the evidence hierarchy, ranging from observational associations to interventional proof-of-concept trials. Sample sizes varied widely, with Bigman's cross-sectional analysis [17] enrolling the largest cohort of 3,489 participants, thereby providing robust statistical power for detecting associations in older adults, whereas the pilot RCT by **Tavakoli** [19] did not explicitly report its sample size (marked as NR), reflecting the preliminary nature of that investigation. Studies examining age-related hearing loss (ARHL) specifically targeted older adults, with **Szeto *et al.*** [14] **and Lee *et al.*** [6] focusing exclusively on participants aged ≥70 years and >65 years, respectively, while Bigman [17] included adults aged 50 years and older with a mean age of 61.5 ± 9.1 years. In contrast, studies investigating sudden sensorineural hearing loss (SSNHL) enrolled adult populations across a broader age spectrum, with **Qi *et al.*** [15] uniquely providing an age-specific sub-analysis that identified the highest vitamin D deficiency rate (60.0%) among patients under 30 years of age. Regarding sex distribution, **Lee *et al.*** [6] reported that 44.2% of their weighted sample were males and crucially found that the significant association between low vitamin D and ARHL was exclusively observed in males (OR: 1.638). The cohort study by **Qi *et al.*** [15] was the only one to explicitly differentiate between first-onset and recurrent SSNHL patients, noting that recurrent cases had substantially lower baseline vitamin D levels (18.4 ± 4.1 ng/mL) compared to first-onset cases (24.9 ± 9.7 ng/mL). Furthermore, the cross-sectional studies by Szeto [14] and Bigman [17] incorporated additional objective measures such as bone mineral density (BMD) and otoscopic/tympanometric examinations to confirm sensorineural hearing loss, adding a layer of diagnostic precision to their demographic characterization.

Table 2 transitions to the core analytical variables and outcome measures. The majority of observational studies consistently defined vitamin D deficiency as serum 25-hydroxyvitamin D [25(OH)D] levels below 20 ng/mL (or 50 NRol/L) [6,14,16,17,19], whereas the RCT by **Shen *et al.*** [3] adopted a higher deficiency threshold of <75 NRol/L and supplemented patients with 1500-2000 IU/day of vitamin D3 for ten days. Pure-tone audiometry (PTA) was the universal hearing assessment tool, though the specific frequencies evaluated varied: Szeto ***et al.*** [14] examined low-frequency (500, 1000, 2000 Hz), speech-frequency, and high-frequency ranges, while Bigman [17] differentiated between low speech frequencies (LPTA: 0.5–4 kHz) and higher frequencies (HPTA: 3–8 kHz). The statistical outcomes demonstrated a consistent association between low vitamin D status and hearing impairment across most observational studies; Szeto [14] reported adjusted odds ratios of 2.02 (95% CI: 1.28–3.19) for low-frequency hearing loss and 1.96 (95% CI: 1.12–3.44) for speech-frequency hearing loss, while Bigman [17] found a significant association with bilateral sensorineural hearing loss at LPTA (OR: 1.60; 95% CI: 1.13–2.26). Notably, Zandi ***et al.*** [16] documented that 70% of SSNHL patients had vitamin D insufficiency compared to only 44% of healthy controls (p=0.017), with significantly lower mean serum levels in the patient group (26.55 ± 14.44 vs. 33.51 ± 14.21 ng/mL).

The two RCTs, conducted by Shen ***et al.*** [3] and Tavakoli ***et al.*** [19], both demonstrated that adjunctive vitamin D supplementation provided additional hearing recovery benefits in vitamin D-deficient SSNHL patients when added to conventional corticosteroid therapy. Shen ***et al.*** [3] reported highly significant improvements in pure-tone average (PTA) at both 10 days (29.3 dB improvement in the intervention group vs. 14.2 dB in controls, p<0.001) and at 3 months (25.1 dB vs. 12.5 dB, p<0.001), with total effective hearing recovery rates of 82.0% vs. 52.9% at day 10. Similarly, Tavakoli [19] found that high-dose vitamin D (50,000 IU) specifically improved hearing at critical speech frequencies of 2000 Hz (p=0.004) and 4000 Hz (p=0.001). However, these interventional findings are directly contradicted by the prospective cohort study of **Qi *et al.*** [15], which administered a 3-month supplementation protocol but found that vitamin D supplementation did not significantly improve recovery rates (75.7% vs. 68.2%, p>0.05) or alleviate symptoms, concluding that long-term maintenance of adequate vitamin D levels, rather than acute post-symptom intervention, may be more critical. Furthermore, **Kose Celebi *et al.*** [18] reported that although vitamin D levels were significantly lower in SSNHL patients compared to controls (p<0.05), neither vitamin D nor the Neutrophil-Lymphocyte Ratio (NLR) had any significant effect on the degree of hearing recovery (p>0.05), suggesting that while deficiency is a risk factor for disease onset, it may not reliably predict prognostic outcomes.

Table 3 presents the risk of bias assessment for all eight included studies [3,6,14-19] using the most suitable and widely validated tools according to each study's specific design. Overall, the majority of included studies are of moderate-to-high methodological quality, although publication bias and the inherent limitations of cross-sectional designs (inability to establish temporal causality) should be carefully considered when interpreting the synthesized evidence.

Table 1: Study Characteristics and Demographic Data

Study (Author, Year) [Ref.]	Country / Setting	Study Design	Sample Size (N)	Population / Participants	Age (years)	Sex (% Male)	Other Demographic Notes
Szeto *et al.*, 2021 [14]	USA (NHANES 2005-2010)	Cross-sectional	1,123	Community-dwelling elderly (≥70 years)	≥ 70	NR	Nationally representative sample; included BMD data.
Qi *et al.*, 2026 [15]	China	Prospective Cohort	140 (80 patients, 60 controls)	Adults with SSNHL and matched healthy controls	NR (Adults)	NR	Highest Vit D deficiency rate (60%) was in patients < 30 years.
Lee *et al.*, 2024 [6]	South Korea (KNHANES 2010-2012)	Cross-sectional	1,104	Older adults (>65 years)	> 65	44.2% (weighted)	Analysis was stratified by sex; association was only significant in males.
Zandi *et al.*, 2023 [16]	Iran	Cross-sectional (Case-control)	100 (50 patients, 50 controls)	SSNHL patients and healthy individuals without hearing loss	NR	NR	Controlled for Ca, P, and PTH levels.
Bigman, 2022 [17]	USA (NHANES 2001-2006, 2009-2012)	Cross-sectional	3,489	Adults aged ≥50 years	61.5 ± 9.1	NR	Included only participants with normal tympanometry and otoscopic exams for SNHL sub-analysis.
Kose Celebi *et al.*, 2023 [18]	Turkey	Retrospective Case-control	NR	SSNHL patients and healthy controls	Mean age comparable between groups (p > 0.05)	No significant difference	Also investigated NLR, ferritin, iron, B12, and folate.
Shen *et al.*, 2025 [3]	China (Yinchuan First People's Hospital)	Prospective Randomized Controlled Trial	101 (51 control, 50 experimental)	SSNHL inpatients with Vit D deficiency (<75 NRol/L)	NR	NR	Evaluated short-term (10 days) and long-term (3 months) outcomes.
Tavakoli *et al.*, 2026 [19]	Iran	Double-blind, Placebo-controlled RCT (Pilot)	NR	SSNHL patients with Vit D deficiency (<50 NRol/L or 20 ng/mL)	NR	NR	Symptoms occurred within 45 days prior to study enrollment.

Table 2: Main Variables, Outcomes, and Key Findings

Study (Author, Year) [Ref.]	Vitamin D Definition / Cut-off	Hearing Assessment Tool (Frequencies)	Primary Outcomes / Statistical Results	Main Conclusion / Key Finding
Szeto *et al.*, 2021 [14]	Total 25(OH)D < 20 ng/mL	PTA (Low: 0.5,1,2 kHz; Speech: 0.5,1,2,4 kHz; High: 3,4,6,8 kHz)	**Low-frequency HL:** OR 2.02 [1.28-3.19]; **Speech-frequency HL:** OR 1.96 [1.12-3.44]. PTH and Calcium were not associated.	Low Vit D is a potential risk factor for age-related low-frequency and speech-frequency HL.
Qi *et al.*, 2026 [15]	Baseline 25(OH)D measured; Deficiency/Insufficiency supplemented.	PTA at 10 days and 3 months	Prevalence: 38.8% in SSNHL vs. 10.0% in controls (p=0.000). Recurrent cases had lower levels (18.4±4.1 vs 24.9±9.7 ng/mL). Supplementation did not significantly improve recovery (75.7% vs 68.2%).	Deficiency is a risk factor for onset and recurrence (especially in <30 years), but acute post-diagnosis supplementation failed to improve outcomes.
Lee *et al.*, 2024 [6]	Serum Vit D < 20 ng/mL	Pure-tone audiometry (ARHL definition)	In males, lower Vit D was associated with higher ARHL prevalence (OR 1.638, 95% CI 1.058-2.538, p=0.027). No significant association found in females.	Lower Vit D is associated with ARHL specifically in the older male population.
Zandi *et al.*, 2023 [16]	Serum 25(OH)D level	NR (Standard audiological confirmation for SSNHL)	Vit D levels: 26.55±14.44 (patients) vs. 33.51±14.21 (controls), p=0.017. Insufficiency: 70% in patients vs. 44% in controls (p<0.05). Ca, P, and PTH showed no difference.	Increased prevalence of Vit D insufficiency in SSNHL suggests a possible protective role via antioxidant mechanisms.
Bigman, 2022 [17]	VD deficiency (<20 ng/mL)	LPTA (0.5-4 kHz) and HPTA (3-8 kHz)	**Bilateral LPTA impairment:** OR 1.45 [1.12-1.89]. **Bilateral SNHL at LPTA:** OR 1.60 [1.13-2.26]. No association found at HPTA.	VD deficiency is associated with bilateral hearing impairment, particularly affecting the inner ear (sensorineural) at low frequencies.
Kose Celebi *et al.*, 2023 [18]	Serum Vit D level	NR (Recovery degree evaluated)	Vit D level was significantly lower in the patient group (p<0.05). However, NLR and Vitamin D levels had **no effect** on the degree of hearing recovery (p>0.05).	Vit D should be added as a routine lab test in SSNHL, but it may not predict the recovery prognosis.
Shen *et al.*, 2025 [3]	Vit D deficiency (<75 NRol/L) - supplemented with 1500-2000 IU/day for 10 days	PTA and Tinnitus Handicap Inventory (THI)	**10-day PTA improvement:** 29.3 dB vs 14.2 dB (p<0.001). **3-month PTA:** 25.1 dB vs 12.5 dB. Total effective rate: 82% vs 52.9% (p<0.001) at 10 days; 76% vs 47.1% at 3 months.	Adjunctive Vit D supplementation significantly improves both short-term and sustained long-term hearing and tinnitus outcomes.
Tavakoli *et al.*, 2026 [19]	Deficiency (<50 NRol/L or 20 ng/mL) - supplemented with 50,000 IU Vit D3	PTA (500, 1000, 2000, 4000, 6000 Hz), SRT, SDS	Significant improvement at **2000 Hz**(p=0.004) and **4000 Hz** (p=0.001) in the intervention group. No benefit at lower frequencies (500, 1000 Hz).	Vit D supplementation enhances auditory recovery specifically at key speech-related frequencies.

Table 3: Risk of Bias Assessment of Included Studies

Study (Author, Year) [Ref.]	Study Design	Assessment Tool	Domain 1 (Selection / Randomization)	Domain 2 (Comparability / Allocation)	Domain 3 (Outcome / Blinding)	Domain 4 (Attrition / Reporting)	Overall Risk of Bias / Score
Szeto *et al.*, 2021 [14]	Cross-sectional	Newcastle-Ottawa Scale (NOS)	Representative sample (NHANES) - 2 stars	Adjusted for age, sex, BMD, and covariates - 2 stars	Validated audiometric testing - 2 stars	Non-response rate not reported - 1 star	**8/9 (Low risk)**
Qi *et al.*, 2026 [15]	Prospective Cohort	Newcastle-Ottawa Scale (NOS)	Exposed cohort representative - 1 star	Groups comparable; adjusted for confounders - 2 stars	Outcome (PTA) objectively assessed - 2 stars	Follow-up adequate but short-term - 1 star	**7/9 (Moderate risk)**
Lee *et al.*, 2024 [6]	Cross-sectional	Newcastle-Ottawa Scale (NOS)	Representative sample (KNHANES) - 2 stars	Adjusted for alcohol, smoking, mobility, BMD - 2 stars	Standard audiometric assessment - 2 stars	Non-response and some confounders (noise) NR - 1 star	**7/9 (Moderate risk)**
Zandi *et al.*, 2023 [16]	Cross-sectional (Case-control)	Newcastle-Ottawa Scale (NOS)	Clearly defined cases and controls - 2 stars	Controls matched appropriately - 1 star	Laboratory & audiometry objectively measured - 2 stars	Sample size relatively small - 1 star	**7/9 (Moderate risk)**
Bigman, 2022 [17]	Cross-sectional	Newcastle-Ottawa Scale (NOS)	Highly representative (NHANES) - 2 stars	Adjusted for multiple covariates - 2 stars	Tympanometry & otoscopy to confirm SNHL - 2 stars	Non-response bias not addressed - 1 star	**8/9 (Low risk)**
Kose Celebi *et al.*, 2023 [18]	Retrospective Case-control	Newcastle-Ottawa Scale (NOS)	Cases defined, but retrospective selection - 1 star	Limited confounder adjustment reported - 1 star	Laboratory tests and chart review - 2 stars	Incomplete data on sample size and follow-up - 1 star	**6/9 (Moderate/Some concerns)**
Shen *et al.*, 2025 [3]	Randomized Controlled Trial	Cochrane RoB 2.0	Low risk (Randomization process adequate)	Low risk (Allocation concealed)	**Some concerns** (Blinding of outcome assessors not explicitly described)	Low risk (Complete outcome data)	**Some concerns**
Tavakoli *et al.*, 2026 [19]	Randomized Controlled Trial (Double-blind)	Cochrane RoB 2.0	Low risk (Randomized, placebo-controlled)	Low risk (Allocation concealed)	**Low risk** (Double-blind design clearly stated)	Low risk (No missing outcome data)	**Low risk**

DISCUSSION

Findings demonstrate a consistent and statistically significant association between low serum 25-hydroxyvitamin D [25(OH)D] levels and hearing impairment across diverse populations, geographical settings, and study designs. Our synthesized findings align robustly with a growing body of observational evidence beyond the studies included in this review. The cross-sectional studies utilizing large national databases [6,14,17] demonstrated remarkably consistent effect sizes, with adjusted odds ratios ranging from 1.45 to 2.02 for hearing impairment among individuals with serum 25(OH)D levels below 20 ng/mL. These findings are directly corroborated by a recent comprehensive systematic review and meta-analysis [2], which included five observational studies with a total sample size of 1,936 participants and demonstrated that vitamin D levels were significantly lower in patients with sensorineural hearing loss (SNHL) compared to healthy controls (mean difference = −9.50 ng/mL, 95% CI: −13.02 to −5.98, I^2 = 16.9%). Notably, this meta-analysis also reported that the incidence of sufficient vitamin D levels was significantly lower in the SNHL group (risk ratio = 0.61, 95% CI: 0.47–0.79), with a particularly pronounced effect observed in the sudden SNHL subgroup. The consistency between our review and this independent meta-analysis strengthens the evidence base for a genuine association rather than a spurious finding attributable to publication bias or chance.

Further supporting these observations, [9] extended the NHANES analysis to the 2015–2016 cycle and specifically examined the differential associations of circulating 25(OH)D and its active metabolite 1,25-dihydroxyvitamin D [1,25$(OH)_2$D] with hearing thresholds. Their findings indicated that while total 25(OH)D was significantly associated with pure-tone averages at speech frequencies (β = −0.18 dB per 10 ng/mL increase, p = 0.003), the free 25(OH)D fraction demonstrated an even stronger association (β = −0.24 dB, p = 0.001), suggesting that the bioavailable fraction of vitamin D may be a more sensitive marker of cochlear health than total circulating levels. This observation adds a layer of nuance to the interpretation of our findings, as none of the studies included in our review [3,6,14-19] specifically measured free or bioavailable vitamin D fractions, potentially underestimating the true strength of the association.

The interventional data from our included studies [3,19] demonstrated that adjunctive vitamin D supplementation provided additional hearing recovery benefits in vitamin D-deficient SSNHL patients when added to conventional corticosteroid therapy. **Shen *et al.*** [3] reported that the experimental group (receiving 1500–2000 IU/day of vitamin D3 for 10 days) achieved significantly higher total effective hearing recovery rates at day 10 (82.0% vs. 52.9%, p < 0.001) and maintained superior outcomes at 3 months (76.0% vs. 47.1%, p = 0.002).

Similarly, **Tavakoli *et al.*** [19] demonstrated that a single high-dose bolus of 50,000 IU vitamin D3 significantly improved hearing at the critical speech frequencies of 2000 Hz (p = 0.004) and 4000 Hz (p = 0.001). These therapeutic benefits are consistent with a larger randomized controlled trial [20], who enrolled 162 SSNHL patients with baseline vitamin D deficiency (< 20 ng/mL) and randomized them to receive either standard corticosteroid therapy plus 50,000 IU weekly vitamin D3 for four weeks or standard therapy plus placebo. At the 12-week follow-up, the intervention group demonstrated significantly greater mean PTA improvement (28.4 ± 6.2 dB vs. 17.2 ± 5.8 dB, p < 0.001) and higher rates of complete hearing recovery (34.6% vs. 17.3%, p = 0.021). The convergence of these interventional findings from independent trials [3,19,20] strongly supports a causal therapeutic role for vitamin D in mitigating hearing loss when deficiency is present at the time of acute auditory insult.

However, these positive therapeutic findings are directly challenged by the prospective cohort study of **Qi *et al.*** [15], which administered a 3-month supplementation protocol to vitamin D-deficient or insufficient patients but found that supplementation did not significantly improve hearing recovery rates (75.7% vs. 68.2%, p > 0.05) or alleviate associated symptoms. This apparent contradiction warrants careful methodological scrutiny. The key distinction lies in the timing of supplementation; **Qi *et al.*** [15] initiated supplementation at the time of SSNHL diagnosis (acute intervention), whereas **Shen *et al.*** [3] initiated supplementation concurrently with conventional therapy on day 1. The discrepancy in outcomes may reflect the critical time window for vitamin D action, as the active metabolite 1,25$(OH)_2$D requires at least 48–72 hours to upregulate genomic pathways involved in cochlear protection and inflammation modulation. Consequently, the cumulative evidence suggests that chronic maintenance of adequate vitamin D levels, rather than acute post-symptom loading, may be the optimal strategy for reducing SSNHL risk and improving outcomes, a conclusion explicitly articulated by **Qi *et al.*** [15] themselves.

The consistent epidemiological association between vitamin D deficiency and hearing impairment is supported by several biologically plausible mechanisms involving the maintenance of cochlear homeostasis. Vitamin D exerts its biological effects through the vitamin D receptor (VDR), while local activation of vitamin D depends on the enzyme 1-α-hydroxylase (CYP27B1). Both VDR-mediated signaling and local vitamin D metabolism have been implicated in the regulation of calcium homeostasis, oxidative stress, immune responses, and vascular function within the inner ear, providing a mechanistic basis for the

protective role of vitamin D against cochlear injury [21]. This local vitamin D signaling system plays a pivotal role in calcium homeostasis within the endolymphatic compartment, where precise calcium concentrations are essential for maintaining the endocochlear potential and the mechanoelectrical transduction capacity of hair cells. Calcium homeostasis is essential for cochlear hair-cell survival and function. Calcium-binding proteins, including calbindin-D28k and parvalbumin, buffer intracellular calcium within cochlear hair cells, while vitamin D signaling contributes to the regulation of calcium transport in the inner ear through vitamin D receptor-mediated pathways. Consequently, vitamin D deficiency may increase susceptibility to cochlear injury by impairing calcium homeostasis and promoting oxidative stress and inflammation [23].

This calcium-dependent mechanism provides a biologically plausible explanation for the observed association between low vitamin D levels and bilateral sensorineural hearing loss, including impairment at low and speech frequencies, as reported by **Szeto *et al.*** [14] **and Bigman *et al.*** [17].

Impaired calcium regulation may compromise cochlear hair-cell function and increase vulnerability to oxidative and inflammatory injury, thereby contributing to hearing impairment. Beyond calcium homeostasis, vitamin D has well-established anti-inflammatory, antioxidant, endothelial-protective, and immunomodulatory effects, all of which may further protect the highly metabolically active cochlea from ischemic and inflammatory damage. Collectively, these mechanisms provide a strong biological framework supporting the observed epidemiological relationship between vitamin D deficiency and sensorineural hearing loss [14,17].

Furthermore, vitamin D exerts significant immunomodulatory and anti-inflammatory effects that are directly relevant to the pathogenesis of both ARHL and SSNHL. Chronic low-grade inflammation, characterized by elevated circulating levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), has been established as a key driver of age-related cochlear degeneration. In an experimental noise-induced hearing loss model, acoustic trauma induced marked cochlear inflammation characterized by activation of the TLR4/NF-κB pathway and increased expression of pro-inflammatory cytokines, including TNF-α and IL-1β [24].

The antioxidant properties of vitamin D further amplify its protective effects; [22] demonstrated in an auditory hair cell line (HEI-OC1) that 1,25$(OH)_2$D treatment attenuated cisplatin-induced oxidative stress by upregulating the Nrf2/HO-1 antioxidant pathway, reducing reactive oxygen species (ROS) production, and decreasing lipid peroxidation (malondialdehyde levels) by 38% compared to untreated controls. This antioxidant role is particularly relevant to SSNHL, where ischemic-reperfusion injury and oxidative stress are considered primary pathophysiological drivers [25].

Interestingly, **Paprocki *et al.*** (the study excluded from our primary analysis due to its focus on oxidative stress rather than hearing outcomes) demonstrated that vitamin D supplementation in SSNHL patients receiving hyperbaric oxygen therapy was associated with a reduction in erythrocyte lipid peroxidation products, indirectly supporting this antioxidant mechanism [26].

One of the most intriguing findings from the synthesized evidence relates to the potential influence of age on the association between vitamin D deficiency and hearing loss.

Qi *et al.* [15] reported that the highest vitamin D deficiency rate (60.0%) was observed in patients with SSNHL younger than 30 years, suggesting that younger adults may be particularly vulnerable to the detrimental effects of deficiency. Although evidence from large epidemiological studies remains limited, analyses of nationally representative cohorts have also evaluated the association between serum vitamin D concentrations and hearing loss across different age groups, indicating that demographic factors, including age, may modify this relationship [9].

Nevertheless, the biological basis for this apparent age-related vulnerability remains uncertain. Given the established roles of vitamin D in immune regulation, oxidative stress modulation, endothelial function, and calcium homeostasis, it is plausible that vitamin D deficiency may exert greater biological effects in younger individuals, although this hypothesis requires confirmation in larger prospective studies.

Regarding gender disparities, **Lee *et al.*** [6] provided compelling evidence that the association between lower vitamin D levels and age-related hearing loss was significant only in males (OR: 1.638, 95% CI: 1.058–2.538, $P = 0.027$), whereas no significant association was observed in females. These findings suggest that biological sex may modify the relationship between vitamin D status and hearing loss. Although the mechanisms underlying this sex-specific difference remain incompletely understood, sex-related differences in vitamin D metabolism, endocrine regulation, body composition, and enviroNRental exposures such as occupational noise have been proposed as potential contributing factors. In particular, interactions between vitamin D signaling and sex hormones may influence immune responses, oxidative stress, and calcium homeostasis, thereby modifying susceptibility to cochlear injury [27]. However, these mechanisms remain speculative and require confirmation in dedicated mechanistic and prospective clinical studies.

LIMITATIONS

Despite robust findings, this systematic review has significant methodological limitations. Most studies were cross-sectional or retrospective, hindering causal inferences regarding vitamin D deficiency and hearing loss. The only prospective study and two randomized controlled trials (RCTs) had small sample sizes and short follow-ups. Definitions of vitamin D deficiency varied across studies, complicating comparisons. Key confounding variables, such as noise exposure and socioeconomic status, were inconsistently adjusted, risking bias. Hearing assessments were not standardized, affecting comparability. Additionally, publication bias could overestimate results, and geographic concentration limits generalizability, especially with underrepresentation from regions outside North America and East Asia.

CONCLUSION

There is a significant link between vitamin D deficiency and both age-related and sudden sensorineural hearing loss (SSNHL), particularly affecting bilateral sensorineural impairment at low and speech frequencies. While two randomized controlled trials (RCTs) suggest benefits of vitamin D supplementation in deficient SSNHL patients, one cohort study indicates no benefit due to intervention timing differences. The association is biologically plausible given the presence of vitamin D receptors in cochlear tissues and its roles in calcium management and antioxidant defense.

Evidence suggests younger adults and males are more susceptible to deficiency effects. However, methodological issues, mainly cross-sectional designs and varied definitions, limit current findings. Future research should focus on large, well-designed studies to determine the timing and efficacy of supplementation. In the interim, routine serum 25(OH)D screening is advised for patients with hearing loss, especially among the elderly, young adults with SSNHL, and males, to address potential deficiencies.

DECLARATIONS

Ethics Approval and Consent to Participate

Not applicable. This systematic review did not involve direct participant recruitment, data collection from human subjects, or any experimental interventions. All data were extracted from previously published studies that had obtained appropriate ethics approval and informed consent from their respective participants.

Consent for Publication

Not applicable. This manuscript contains no individual person's data, images, or identifiable information requiring consent for publication.

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Competing Interests

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Authors' Contributions

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