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Review Article

The Risk Factors for Age-Related Hearing Loss: An Integrative Review

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Keywords

- Age - related hearing loss
- Risk factors
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Abstract

Background: Age - related hearing loss (ARHL) is generally a bilateral sensorineural deficit with an incidence that increases at an accelerated rate with age. Strategies to prevent or delay ARHL should focus on identifiable factors.

Objective: To determine risk factors contributing to ARHL

Methods: The Pub Med database was searched using the term "age - related hearing loss." Multiple combinations of terms were searched with the MeSH database using "hearing loss, aged" or "presbycusis" and "risk factors." Then, limits were applied to the literature search: 1) published in English since 2000, 2) human adults, and 3) bilateral sensorineural hearing loss. Review articles were excluded.

Results: 38 studies identified as relevant to the research question. Genetic factors (genes and gender), environmental factors (noise and chemicals), lifestyle factors (smoking and diet), and individual health factors (ototoxic medications, cardiovascular diseases, diabetes, and obesity) influence ARHL. The findings suggest that both environmental and individual health risk factors can accumulate over a lifetime and contribute to the hearing loss experienced by older people. However, research correlating specific risk factors to ARHL has conflicting results.

Conclusion: Many risk factors are modifiable and are possible targets for prevention or moderation of ARHL. Identifying further risk and protective factors for ARHL and developing appropriate interventions are worthy goals for further research.

Implications of Practice: Health providers can play a crucial role in minimizing ARHL by screening for hearing loss in risk populations in the community or clinics, educating older people about potential risk factors and supporting lifestyle changes to delay or moderate ARHL.

INTRODUCTION

Age - related hearing loss (ARHL) is generally a bilateral sensorineural deficit with an incidence that increases at an accelerated rate with age [1]. More than 30% of people aged 40-49 years and approximately half of adults older than 75 years of age experience hearing loss [1]. This emphasizes that ARHL is a public health problem that is documented to have multiple negative effects on an individual's physical, psychosocial, and social status [2].

Although many older adults experience a decline in hearing with age, other risk factors can contribute to ARHL. Some elderly individuals experience more significant hearing problems than others in the same age group depending on one's individual susceptibility and exposure to environmental factors that may damage hearing [3]. Teasing apart the specific contributions of factors that may affect hearing from the effects of chronological age is difficult. For example, an individual may be exposed to noise or ototoxic agents over time and the effects of such exposure is thus associated with chronological age but each of these factors may have an independent, concurrent effect on hearing and will only affect those exposed. Strategies to delay or minimize ARHL should focus on such identifiable factors. The purpose of this paper is to review those risk factors that may interact with age in the development of ARHL as a basis for the development of targeted hearing conservation strategies. Specifically, this literature review will: 1) synthesize the literature examining the associations between risk factors and ARHL; 2) discuss the strengths and limitations of the current literature; and 3) discuss the implications for clinical practice and future research.

METHODS

Literature search

A search of the literature on risk factors for ARHL was performed using the electronic Pub Med database and the keywords "age - related hearing loss" and "risk factors." In addition, multiple combinations of keywords were searched with

the MeSH database using "hearing loss, aged" or "presbycusis" and "risk factors." Psych INFO and the Cochrane Library were also searched with similar terms. In addition, reference lists from every relevant paper were examined to determine whether pertinent studies had been missed through the data base searches.

Inclusion criteria are: 1) published in English since 2000, 2) human adult subjects, 3) bilateral sensorineural hearing loss, and 4) clinical trials, randomized controlled trials or Meta - analyses (Figure 1).

Study quality

The quality of each study was evaluated using a critical appraisal tool developed by Zaza et al., [4]. The evaluation included checking for bias in sampling, measurements, statistical analysis, and interpretation of results. Based on the recommendations of Sanderson, Tatt, and Higgins [5], no total numerical score was given, but each article was classified as moderate to high quality or low quality (Table 1). The criteria for moderate to high quality included: 1) adequate study population sample, 2) valid and reliable measures, 3) appropriate statistical analysis, and 4) appropriate interpretation of results. Additional criteria were: 5) clear study descriptions including potential confounding factors and 6) appropriate description of cases lost to follow-up in longitudinal studies [4].

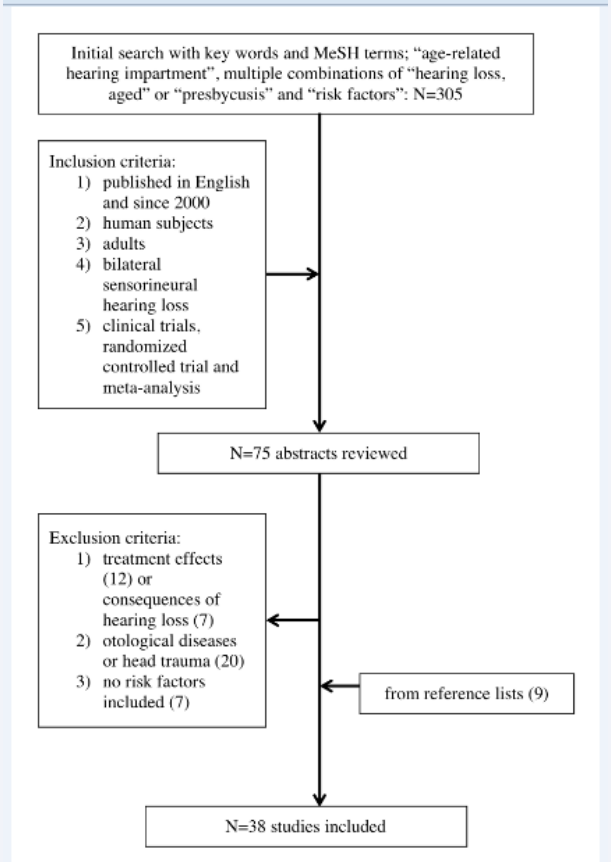

Figure 1 Flow chart showing the selection of studies in the literature review.

RESULTS

As presented in Figure, a total of 305 articles were retrieved from the database using the various combinations of the keywords mentioned above. After applying the inclusion criteria, 75 articles were selected. An additional 26 studies without risk factors and 20 studies focused on specific diseases with unilateral hearing loss were eliminated while nine articles were included from reference lists, yielding 38 research articles to review. A summary of characteristics of the 38 studies included in this review and their major findings by risk factors are summarized in Table (1) and Table (2), respectively. Risk factors are summarized under four categories: genetic, environmental, lifestyle, and individual health. Helzner et al., [6] and Fransen et al., [7] studied for multi risk factors; therefore, their results are presented in different categories when applicable.

Genetic factors

Family history: Human research on ARHL supports that a family history of hearing loss is strongly correlated with ARHL. A maternal family history of hearing loss was more strongly associated in women compared to a paternal family history of hearing loss in men [8]. The heritability contribution to ARHL in older adults was estimated to be 40-50% [9,10].

Genes and sex: Susceptibility genes for ARHL are related to oxidative stress that may be part of the aging process [11]. Glutathione S-transferase (GST) and N-acetyltransferase (NAT) are antioxidant enzymes that detoxify reactive oxygen species in the inner ear, and are comprised of several gene classes. People who have the deletion of these antioxidant enzymes may be more prone to damage by reactive oxygen species and, therefore, more susceptible to ARHL [11]. Van Eyken et al., [12] found the deletion of these two distinct susceptibility genes in the general European and the Finnish populations. In the United States, White subjects with ARHL had more GST deletions and NAT mutations than those without ARHL [13]. The GST deletion was found more frequently in Hispanic Whites than in non - Hispanic Whites in the ARHL group, suggesting that susceptibility genes may vary by ethnicity. In addition, a voltage-gated potassium channel 4 (KCNQ4) and a glutamate metabotropic receptor 7 (GRM7) have been identified as candidate genes for ARHL. KCNQ4 gene was associated with ARHL among Caucasian populations in Europe [14,15]. GRM7 was associated with ARHL in European American older adults [15].

ARHL was more common among men than women, and more common among White participants than Black participants [16]. Low - frequency (500, 1000, and 2000 Hz) hearing loss was most common among White men, followed by White women, Black men, and Black women. High - frequency (2000, 4000, and 8000 Hz) hearing loss was most common among White men, followed by Black men, White women, and Black women [6]. In the past, these findings were attributed partly to the fact that men tended to have more jobs that exposed them to loud noise than women which meant they experienced more occupationally related hearing loss. However, gender differences in hearing loss may be better explained by gender - specific pathophysiologic mechanisms than noise exposure [17].

Table 1: Characteristics of Age-related Hearing Loss Studies by Risk Factors.

Investigator, Year	Country of study	Design	Subjects	Measurement	QL[1]
Genetic Factors:					
Family history					
Christensen, et al., 2001	Denmark	Prospective cohort	Twins from the Danish Twin Registry (3928)	Self-reported HL[2]	-
McMahon, et al., 2008	Australia	Prospective cohort	Adults from BMHS[3] (2669)	HL on PTA[4] at 500, 1000, 2000, 4000 Hz > 25 dB, family history	+
Raynor, et al., 2009	US	Prospective cohort	Adults from EHLS[5] (3510)	HL on PTA at 500 to 4000Hz > 25 dB, family history	+
Genes and Sex					
Van Eyken, et al., 2006	Belgium, The Netherlands	Clinical trial	Adults (664)	PTA thresholds at 125, 250, 5000, 1000, 2000, 4000, 6000 and 8000 Hz, genotypes	+
Van Eyken, et al., 2007	Belgium, UK, The Netherlands, Germany, Denmark, Italy, Finland	Clinical trial	Adults from nine centers (2111)	PTA thresholds at 125 to 8000 Hz, genotypes	+
Pratt, et al., 2009	US	Cross-sectional	Adults from Cardiovascular Health Study (548)	HL on PTA at 250 to 8000 Hz > 25 dB, sex, race	-
Bared, et al., 2010	US	Case-control	HL group (55) vs. No HL group (79)	HL on PTA at 500 to 4000Hz > 30 dB, DNA	+
Newman, et al., 2012	US	Clinical trial	Adults (687)	PTA thresholds at 250 to 8000 Hz, genotypes	+
Environmental Factors:					
Noise exposure					
Gate, et al., 2000	US	Prospective cohort	Men from Framingham Heart Study (203)	PTA thresholds at 250 to 8000 Hz, no notch (< 15 dB), small notch (15-34 dB), and large notch (≥ 35 dB) in the 3000-6000 Hz	+
Albera, et al., 2010	Italy	Cross-sectional	Men (568)	HL on PTA at 2000, 3000, 4000 Hz > 25 dB, occupational noise exposure	+
Cruickshanks, et al., 2010	US	Prospective cohort	Adults from the EHLS (3753)	HL on PTA at 500 to 4000 Hz > 25 dB, occupational noise exposure	+
Ciorba, et al., 2011	Italy	Cross-sectional	Older adults (460)	PTA thresholds at 250 to 8000 Hz, occupational noise exposure	-
Hederstierna, et al., 2016	Sweden	Prospective cohort	Noise-exposed group (84) vs. non exposed group (254)	PTA thresholds from 250 to 8000 Hz, occupational noise exposure	+
Chemical exposure					
Crawford, et al., 2008	US	Cross-sectional	White men licensed private pesticide applicators from the Agricultural Health Study (14229)	Self-reported HL, pesticides exposure	-
Park, et al., 2010	US	Cross-sectional	Men from the Normative Aging Study (448)	HL on PTA at 500 to 4000 Hz > 25 dB, bone lead levels	+
Co-exposure to Noise and Chemical					
Morata, et al., 2002	US	Case-control	Workers at fiberglass product industry (313) Workers at metal products industry (78) Workers at a mail distribution terminal (81)	HL on PTA at 1000 to 8000 Hz > 25 dB, styrene exposure, noise exposure	+
Sliwinska-Kowalska, et al., 2003	Poland	Case-control	Workers at yacht yards and plastic factory (290) Workers with no styrene exposure (223)	HL on PTA at 1000 to 8000 Hz > 25 dB, styrene exposure, noise exposure	+

Table 1: Characteristics of Age-related Hearing Loss Studies by Risk Factors.

Investigator, Year	Country of study	Design	Subjects	Measurement	QL[1]
Chang, et al., 2006	Taiwan	Case-control	Male workers at an adhesive materials manufacturing plant (58) Male workers exposed to noise exposure only (58) Male administrative clerks (58)	HL on PTA at 1000 to 6000 Hz ≥ 25 dB, toluene exposure, noise exposure	-
Lifestyle Factors:					
Cigarette smoking and alcohol consumption					
Itoh, et al., 2001	Japan	Case-control	Adults with HL (496) Age-matched adults without HL (2807)	HL on PTA at 4000 Hz > 40 dB, smoking and alcohol intake	-
Gopinath, et al., 2010	Australia	Cross-sectional	Adults from the BMHS (2815)	HL on PTA at 500 to 4000 Hz > 25 dB, smoking status, alcohol intake	+
Dawes, et al., 2014	UK	Cross-sectional	UK Biobank (164770)	Speech-in-noise test (The Digit Triplet Test), smoking status, alcohol intake	+
Nutrition					
Park, et al., 2006	US	Randomized, controlled trial	Older adults (93)	HL on PTA at 500 to 4000 Hz > 25 dB, vitamin B-12 status	+
Gopinath, et al., 2010	Australia	Cross-sectional	Adults from the BMHS (2956)	HL on PTA at 500 to 4000 Hz > 25 dB, glycemic load value	+
Gopinath, et al., 2011	Australia	Cross-sectional	Adults from the BMHS (2956)	HL on PTA at 500 to 4000Hz > 25 dB, cholesterol level, dietary fat	+
Spankovich, et al., 2011	Australia	Cross-sectional	Adults from the BMHS (2111)	HL on PTA thresholds from 250 to 8000Hz, TEOAE[6], dietary status	+
Individual Health Factors:					
Ototoxic medications					
Curhan, et al., 2010	US	Longitudinal	Men from the Health Professionals Fellow-up Study (26917)	Self-reported professionally diagnosed HL, regular use of aspirin, NSAIDs[7] and acetaminophen	+
Curhan, et al., 2012	US	Longitudinal	Women from the Nurses' Health Study II (62261)	Self-reportedHL, regular use of aspirin, NSAIDs and acetaminophen	+
Cardiovascular disease (CVD)					
Torre et al., 2005	US	Cross-sectional	Adults from the EHLS (1501)	Cochlear function on DPOAEs[8], self-reported CVD	+
Gopinath, et al., 2009	Australia	Cross-sectional	Adults from the BMHS (3654)	HL on PTA at 500 to 4000 Hz >25 dB, stroke	+
Hutchinson, et al., 2010	US	Cross-sectional	Youth (26), Young adults (27), Middle-aged adults (26), Old adults (22)	PTA thresholds at 1000 to 4000 Hz and DPOAEs, cardiovascular fitness levels by VO_2 peak parameters	-
Diabetes					
Frisina, et al., 2006	US	Case-control	Diabetic type II group (30) vs. Non diabetic group (30)	PTA at 250 to 8000 Hz, otoacoustic emissions, gap detection, speech perception, sentence perception	+
Vauhan, et al., 2006	US	Prospective cohort	Diabetic veterans (342) vs. non-diabetic veterans (353)	HL on PTA at 250 to 4000 Hz > 25 dB	+
Mitchell, et al., 2009	Australia	Prospective cohort	Diabetic participants (210) vs. non-diabetic participants (1648) from the BMHS	HL on PTA at 500 to 4000 Hz > 25 dB	+
Uchida, et al., 2009	Japan	Cross-sectional	Adults (2306)	HL on PTA at 500 to 4000 Hz > 25 dB, diabetes	+
Obesity					
Hwang, et al., 2009	Taiwan	Cross-sectional	Adults (690)	PTA thresholds at 250 to 8000 Hz, waist circumference, BMI[9]	+
Multi risk factors					

Table 1: Characteristics of Age-related Hearing Loss Studies by Risk Factors.

Investigator, Year	Country of study	Design	Subjects	Measurement	QL[1]
Helzner, et al., 2005	US	Cross-sectional	Older adults (2052)	HL on PTA of 500, 1000, 2000 Hz > 25 dB and of 2000, 4000, 8000 Hz > 40 dB, ototoxic medication use, occupational noise exposure, lifestyle factors, and medical history	+
Fransen, et al., 2008	Belgium, UK, The Netherlands, Germany, Denmark, Italy, Finland	Cross-sectional, multicenter study	Adults from nine centers (4083)	Hearing thresholds at 250 to 8000 Hz, BMI, smoking, CVD, noise, chemical exposure, alcohol consumption, and medications	+

(Footnotes)
1 QL=quality Level, +: moderate to high quality, -: low quality
2 HL=hearing loss
3 BMHS=Blue Mountains Hearing Study
4 PTA=pure tone average
5 EHLS=Epidemiology of Hearing Loss Study
6 TEOAE=transient evoked otoacustic emissions
7 NSAIDs= nonsteroidal anti-inflammatory drugs
8 DPOAEs=distortion product otoacoutic emissions
9 BMI=body mass index

Table 2: Major Findings by Risk Factors for Age-related Hearing Loss.

Risk factors	Investigator, Year	Major findings
Family history of hearing loss	Christensen, et al., 2001	Monozygotic twin pairs compared to dizygotic twin pairs (OR[1] 4.5; 95% CI[2] 2.7-6.8 vs. OR 1.5: 95% CI 1.0-2.2)
	McMahon, et al., 2008	• Maternal family history in women (OR 3.0; 95% CI 1.6-5.6) • Paternal family history in men (OR 2.0, 95% CI 1.01-3.9)
	Raynor, et al., 2009	• Family history in all (OR 4.69) and in women only (OR 5.49)
Genes and sex	Van Eyken, et. al., 2006	• KCNQ4[3] ($p < 0.05$)
	Van Eyken, et al., 2007	• GSTT1[4] ($p < 0.01$) and GSTM1[5] ($p < 0.01$) in the Finnish population • NAR2*6[6] ($p = 0.035$) in the general European population
	Pratt, et al., 2009	• Men and White participants ($p < 0.01$)
	Bared, et al., 2010	• GSTT1 null genotype (OR 2.84; 95% CI 1.4-5.9), GSTM1 null genotype (2.43; 1.2-5.1), and NAT2*6 mutant genotype (2.88; 1.4-6.1)
	Newman et al. 2012	• GRM7[7] ($p < 0.05$)
Noise exposure	Gates, et al., 2000	• Greater mean 15-year threshold change in large notches (N2) group only at 2000 Hz as compared to a small notch (N1) and absence of a notch (N0) groups ($p < 0.01$) • Greater change at 8000 Hz in the N1 group than the N0 or N2 groups ($p < 0.01$)
	Albera, et al., 2010	• Progressive hearing loss after the first 10-year of noise exposure was more related to age than to duration of noise exposure ($p < 0.01$)
	Cruickshanks, et al., 2010	• Noise exposure was not significantly correlated with the 10-year cumulative incidence of hearing loss.
	Ciorba, et al., 2011	• No difference of hearing thresholds after adjusting age and sex.
	Hederstierna, et al., 2016	• No difference of hearing thresholds between noise-exposed group and non-exposed group.
Chemical exposure	Crawford, et al., 2008	• The highest quartile of insecticides exposure (OR 1.19; 95% CI 10.4-1.35) and organophosphate insecticides exposure (1.17; 1.03-1.31)
	Park, et al., 2010	• Tibia lead and patella lead for lowering hearing thresholds ($p < 0.05$) • Patella lead (OR 1.14; 95% CI 1.14-1.91)
Co-exposure to noise and chemical	Morata, et al., 2002	• Noise exposure (OR 1.18; 95% CI 1.01-1.34) and styrene exposure (2.44; 1.01-5.89)
	Sliwinska-Kowalska, et al., 2003	• Styrene (OR 5.2; 95% CI 2.9-8.9), noise only (3.4; 1.7-6.4), styrene and noise (10.9; 4.9-24.2), styrene and toluene (13.1; 4.5-37.7), and styrene, toluene, and noise (21.5; 5.1-90.1)
	Chang, et al., 2006	• Toluene and noise exposure (OR 22.6; 95% CI 7.8-65.6) and noise only (5.4; 1.8-15.6)
Cigarette smoking and alcohol consumption	Itoh, et al., 2001	• Current smoking (OR 2.10; 95% CI 1.53-2.89) • No increased risk for heavy drinkers
	Gopinath, et al., 2010	• Current smoking (OR 1.63; 95% CI 1.01-2.64)

Table 2: Major Findings by Risk Factors for Age-related Hearing Loss.

Risk factors	Investigator, Year	Major findings
	Dawes, et al., 2014	• Current smoking (OR 1.15; 95% CI 1.09-1.21) • Passive exposure to smoking (OR 1.28; 95% CI 1.21-1.35) • No association with alcohol consumption
Nutrition	Park, et al., 2006	• Higher mean serum MMA[8] concentrations and prevalence of B12 deficiency ($p < 0.01$)
	Gopinath, et al., 2010	• Higher mean dietary glycemic load dietary (OR 1.41; 95% CI 1.01-1.97)
	Gopinath, et al., 2011	• Highest quartile of dietary cholesterol intake (OR 1.33; 95% CI 1.01-1.74)
	Spankovich, et al., 2011	• Higher cholesterol, fat and retinol intakes with poorer TEOAE and PTA[9] ($p < 0.05$)
Ototoxic medications	Curhan, et al., 2010	• Aspirin (OR1.12; 95% CI 1.04-1.20), NSAIDs[10] (1.21; 1.11-1.33), acetaminophen (1.22; 1.07-1.39) in regular user (≥ 2times/week), compared with user less than 2 times per week
	Curhan, et al., 2012	• Ibuprofen for use 2-3 days/week (RR[11] 1.13; 95% CI 1.06-1.19), for use 4-5 days/week (1.21; 1.11-1.32), for use ≥ 6 days/week (1.24; 1.14-1.35) • Acetaminophen for use 2-3 days/week (RR 1.11; 95% CI 1.02-1.19), for use 4-5 days/week (1.21; 1.07-1.37), for use ≥ 6 days/week (1.08; 0.95-1.22) • No significance with aspirin.
Cardiovascular disease	Torre et al., 2005	• Self-reported history of myocardial infarction with cochlear impairment in women (OR 2.0; 95% CI 1.15-3.46), but not significant in men.
	Gopinath, et al., 2009	• Stroke (OR 2.04, 95% CI 1.20-3.49
	Hutchinson, et al., 2010	• Low cardiovascular fitness in the old age group (49-78 years) with worse pure-tone hearing at 2000 and 4000Hz ($p < 0.05$)
Diabetes	Frisina, et al., 2006	• Diabetic group with lower hearing ability, wideband noise and speech reception thresholds and otoacoustic emissions ($p < 0.05$)
	Vauhan, et al., 2006	• Diabetic participants with aged younger than 60 years only in the highest frequencies ($p < 0.05$)
	Mitchell, et al., 2009	• Diabetes (OR 1.55, 95% CI 1.11-2.17) • Newly diagnosed diabetes with progression of hearing loss over 5 years (OR 2.71; 95% CI 1.07-6.86)
	Uchida, et al., 2009	• Diabetes on hearing thresholds in the younger group (40-64 years) at the high frequencies ($p < 0.01$), but no difference in the older group (65-86 years)
Obesity	Hwang, et al., 2009	• Waist circumference with PTA-low (250, 500,1000 Hz) ($p=0.034$) and PTA-high (2000, 4000, 8000 Hz) ($p=0.024$), but BMI[12] with PTA-low ($p < 0.001$) and PTA-high ($p = 0.035$) only in men younger than 55 years
Multi risk factors	Helzner, et al., 2005	• Age (OR 1.88; 95% CI 1.57-2.24), White (1.63; 1.30-2.04), diabetes (1.42; 1.10-1.83), cerebrovascular disease (1.56; 1.12-2.18), current smoking (1.68; 1.11=2.54), and occupational noise exposure (1.55; 1.24-1.94) • In White men, higher diastolic blood pressure (OR 1.27; 95% CI 1.07-1.50), diabetes (2.12; 1.29-3.48), cerebrovascular disease (2.29; 1.17-4.49), and occupational noise exposure (2.18; 1.52-3.11); in White women, cerebrovascular disease (1.90; 1.03-3.50), and diabetes (1.89; 1.07-3.35) • In Black men, cardiovascular diseases (OR 3.23; 95% CI 1.20-8.72); in Black women, current smoking (2.86; 1.20-6.84)
	Fransen, et al., 2008	• Occupational noise exposure, smoking, high BMI ($p < 0.05$)

(Footnotes)
1 OR= odds ratio
2 CI= confidence interval
3 KCNQ4= voltage-gated potassium channel 4
4 GSTT1=Glutathione S-transferase theta 1
5 GSTM1=Glutathione S-transferase mu 1
6 NAT2*6=N-acetyltransferase 2*6A
7 GRM7=glutamate metabotropic receptor 7
8 MMA= methylmalonic acid
9 PTA=pure tone average`
10 NSAIDs=non-steroidal anti-inflammatory drugs
11 RR=relative risk
12 BMI=body mass index

Environmental factors

Noise exposure: Noise - induced hearing loss (NIHL) has been studied extensively [18], but less attention has been given to the contribution of noise exposure to the development of ARHL. It is difficult to distinguish between NIHL and ARHL in people with lifelong noise exposure because NIHL is also a bilateral sensorineural loss that develops slowly over several years [19]. Both are related to deterioration of the cochlear structures, particularly hair cells [20].

A European study found that there was a significant association between history of occupational noise exposure for more than one year and worse on average hearing threshold levels (2000, 4000, and 8000 Hz) [7]. However, Cruickshanks et al., [21] showed that noise exposure was not significantly correlated with the 10 - year cumulative incidence of hearing loss in older adults. These authors noted that hearing loss tends to worsen with current noise exposure, but progressive hearing loss may be mainly due to aging. Albera, Lacilla, Piumetto, and Canale [22] also concluded that progressive hearing loss after noise exposure for at least 10 years was related more to age itself than to the duration of noise exposure. Ciorba et al., [23] supported these findings in an Italian sample exposed to occupational noise for at least 3 years. They found that the difference in hearing threshold was only related to age after adjusting for age and gender. An additional longitudinal study by Hederstierna and, Rosenhall [24] also found that a decrease in hearing was not related to occupational noise exposure. There was no difference in the pattern of ARHL between older adults with noise exposure and without noise exposure for those aged 70-75 years. However, this study was based on self - reported previous occupational noise exposure which included the duration of the exposure but not the level of the noise.

One cohort study evaluated the 15-year change in audiometric thresholds among men [25]. NIHL is typically characterized by a discrete increase (notch) in the hearing thresholds in the 3000 to 6000 Hz region in the audiogram, while ARHL is usually characterized by a progressive increase across the frequencies of 4000 to 8000 Hz [26]. This study found fewer threshold changes over the 15 years in the notch frequencies but significant changes in the adjacent frequencies at 2000 Hz and 8000 Hz among notch groups. Threshold increases at frequencies around the notch seem to catch up with aging. However, notched audiograms should be interpreted cautiously because a notch may not be caused solely by noise [27].

It is difficult to conclude from the data whether noise exposure contributes to ARHL in older adults because there is great variation across the studies in the types of noisy jobs, the age at first exposure, the noise levels individuals are exposed to, the duration of exposure, and the definition of hearing loss used. However, over time, age appears to be a stronger underlying factor in hearing loss than noise exposure.

Chemical exposure: In addition to noise, industrial chemicals are considered environmental risk factors for ARHL. Industrial chemical toxins reach the inner ear via the bloodstream and damage inner ear structures and their hearing functioning. Furthermore, more central effects have been seen, notably with solvent exposures [28].

A cross - sectional study conducted with male workers in Taiwan to investigate the combined effect of toluene and noise exposure on hearing loss [29]. They found that workers exposed to toluene and noise had greater risk than those exposed to noise only. There was no significant dose effect of toluene exposure. Sliwinska - Kowalska et al., [30] found that workers exposed to styrene had significantly greater risk of hearing loss than unexposed workers. Those exposed to both styrene and noise had greater risk than the styrene or noise only exposure groups. The group exposed to styrene, toluene and noise had 22 times greater risk than unexposed group. Morata et al., [31] also studied the effect of styrene on workers' and found that styrene was significantly related to bilateral high frequency (6000 and 8000 Hz) hearing loss, consistent with the previous study [30].

Exposure to pesticides is also a risk factor for hearing loss. A study conducted with pesticide applicators found that lifetime days of any pesticide use were significantly associated with developing hearing loss [32]. In addition, a longitudinal study of veterans found that patella lead levels were significantly correlated with poor high frequencies hearing, while tibial lead levels were significantly correlated with a progressive increase in hearing thresholds over 20 years [33].

Most chemical exposure studies have been conducted in the occupational settings. Multiple exposures to noise and chemicals may have synergistic ototoxic effects on hearing [29,30]. However, there is little research investigating the effect of occupational chemical exposure on ARHL in the general population after retirement or termination of exposure.

Lifestyle factors

Cigarette smoking and alcohol consumption: It has been suggested before that smoking and alcohol consumption negatively impact cardiovascular health, which then contributes to the risk for hearing loss [7]. Several studies found a significant association between current smoking and prevalent hearing loss among older adults, but no significant association between pack-years of smoking and hearing loss [34,35]. However, Fransen et al., [7] found that the number of pack - years of smoking was significantly associated with hearing loss among ever -smokers aged 53-67 years. Dawes et al., [36] also found a significant dose - response effect among smokers. They examined the effect of second hand smoke on hearing loss and found that non-smokers who were exposed to smokers had a higher risk of hearing loss with a dose dependent effect than those without exposure [36]. Smoking itself may cause oxidative damage in the inner ear because nicotine reduces the blood supply to the cochlea [37].

In contrast to smoking, light to moderate alcohol consumption (≤ 2drinks/day or < 30g/day) had a significant positive correlation with hearing function in older adults [34,35]. The apparent protective effect of regular moderated alcohol consumption on hearing is similar to its effects on the cardiovascular system [7]. Interestingly, cross sectional studies from Europe, Japan, and UK found that there was no increased risk for hearing loss with heavy alcohol consumption [7,34-36].

Nutrition: Nutrition may play a role in the pathogenesis of auditory disorders, and poor nutrition among older adults may affect ARHL [38]. Vitamin B-12 is a common problem

among older adults and may impair cellular metabolism, nerve function, myelin synthesis, and vascular function in the auditory system among older adults [39]. A randomized controlled trial by Park et al., [40] found that participants with hearing loss had a higher prevalence of vitamin B-12 deficiency than those with normal hearing. However, three - month of vitamin B-12 supplementation did not significantly improve hearing levels in vitamin B-12 deficient individuals.

Several studies examined the association between diet and ARHL among the participants of the Blue Mountain Hearing Study. Higher glycemic load, cholesterol, carbohydrate and sugar intake were significantly related to the incidence of hearing loss in several studies [41,42]. In contrast, Spankovich et al., [43] found higher carbohydrate intake associated with better hearing. In addition, fat and retinol intakes were related to poorer hearing ability [43].

Several possible mechanisms may explain the association between diet and ARHL. High carbohydrate and cholesterol diets are associated with cardiovascular diseases and diabetes, which cause disruption to the cochlear blood flow and are considered risk factors for ARHL [44]. Atherosclerotic diets result in damaging the cochlear in the animal models [45]. Additionally, oxidative stress generated by hyperglycemia could damage the cochlear [46]. However, it may be difficult to evaluate the effects of individual nutrients on hearing as people often take combinations of nutrients daily.

Individual health factors

Ototoxic medications: Although review articles state that ototoxic medications (amino glycoside antibiotics, chemotherapeutics, salicylates, and loop diuretics) frequently contribute to ARHL [19], there is little human research on this topic, especially for older adults, and less evidence to support these conclusions. Helzner et al., [6] reported that current use of salicylates, quinine, and loop diuretics was not associated with a higher risk of hearing loss. Conversely, salicylates had a protective effect on hearing loss in the cohort. Two large population - based studies focused on the association between hearing loss and the use of analgesics such as aspirin, non - steroidal anti -inflammatory drugs (NSAIDs), and acetaminophen [47,48]. Curhan et al., [47] conducted a study with men using self - reported professionally diagnosed hearing loss. The risk of hearing loss in regular users of analgesia ≥ 2 (times/week) was higher for aspirin, NSAIDs, and acetaminophen than in those who used these medications less than 2 times per week. The risk of hearing loss was higher in men younger than 50 years for aspirin, NSAIDs, and acetaminophen. Also, the risk of hearing loss was positively associated with duration of regular use and the use of multiple analgesics. The same authors did a similar study with women using self - reported hearing loss [48]. The risk of hearing loss was higher with the regular use of ibuprofen and acetaminophen than with the use of these medications less than 2 times per week. There was no association between aspirin use and hearing loss in this latter study. The risk of hearing loss was greater in younger (< 50 years) women who used ibuprofen more than 6 days per week. However, there was no information on the analgesic doses that participant took in either study. In general, the results of human studies on the toxic impact of ototoxic drug use on hearing loss remain controversial.

Cardiovascular disease (CVD): CVD may affect the micro vascular system that provides a large capillary blood supply to the cochlear [49]. Women with a history of myocardial infarction (MI) had nearly twice the risk of cochlear function impairment than those without history of MI, but a similar risk was not found in men [50]. Hutchinson, Alessio, and Baiduc [51] also evaluated the relationship between hearing function and CV health measured by peak oxygen consumption (VO_2 peak). They found that participants with poor CV health in the older age group (49-78 years) had significantly more hearing loss than those in the same age group with good CV health. Cochlear function was also better among older persons with good CV health than those with medium or poor CV health. In addition, Gopinath and collegues [52] showed that participants with hearing loss had more history of stoke.

Diabetes: Diabetes may affect auditory function by microangiopathy, cellular changes to the peripheral nervous system, and metabolic changes from the generation of reactive oxygen species within the cochlear [53,54]. Mitchell et al., [54] evaluated the relationship between diabetes and ARHL. Fifty percent of those with diabetes had ARHL compared to 38% of those without diabetes. Participants with newly diagnosed diabetes showed significant progression of hearing loss over 5 years compared to those without diabetes. Frisina et al., [53] found that hearing function was worse in pure - tone thresholds, wide-band noise thresholds, speech reception thresholds, and otoacoustic emissions among older adults with diabetes as compared to those without diabetes.

In addition, veteran's aged 60 years or younger with diabetes as compare to those without diabetes had greater hearing loss [55]. A similar relationship was not found in veterans older than 60 years of age. Similar differences were found with participants in Japan [56]. Diabetes appears to speed up the progression of ARHL in younger populations, but age still appears to play a significant role.

Obesity: Obesity itself may be an independent risk factor for ARHL by obesity - related oxidative stress beside obesity - induced systemic diseases [57]. Waist circumference was positively correlated to hearing threshold levels [57]. Men younger than 55 years with a larger waist circumference had poorer hearing after controlling for body mass index, diabetes and CVD. Women older than 55 years had the same association between waist circumference and hearing threshold levels at high frequencies (2000, 4000, and 8000 Hz). In addition, Fransen et al., [7] found that participants with higher body mass index had worse hearing function after controlling CVD and smoking.

DISCUSSION

Extensive research has been undertaken to explain the contribution of risk factors to ARHL. The findings of this review support that environmental, lifestyle, and individual health risk factors can accumulate over a lifetime and contribute to the hearing loss experienced by older people. Genetic factors and the aging process itself are important factors associated with hearing loss. Furthermore, the interaction of intrinsic - extrinsic

factors, such as the combined exposures to chemicals and noise and CVD risk factors related to diet, has been discussed to reveal the underlying complex associations of risks for ARHL.

The most consistently strong risk factors for ARHL across the studies are genetics, current smoking, diabetes, CVD, and obesity. Noise was related to early development of hearing loss but its relationship to the progression of ARHL remains unclear. Occupational exposure to organic solvents such as styrene and toluene has been found to relate to hearing loss, with ototoxic effects exacerbated with co - exposure to noise, but most studies were limited to working population in occupational settings. Diet and ototoxic medications showed conflicting results for ARHL, but are considered potential risk factors for ARHL.

There are a number of limitations in the literature that need to be addressed. The studies relying on self - reported historical information could be affected by recall bias in the medical or personal histories of the participants. Also, information from self - administered questionnaires may attenuate the association between risk factors and hearing loss because older adults may accept hearing loss as a natural part of the aging process.

Attempts to correlate specific risk factors to ARHL have resulted in conflicting results. This may be because each study used different inclusion and exclusion criteria to define ARHL. Also, the adjusted confounding factors used in the analyses varied among studies that used the same outcome. Studies used various definition of hearing loss using different frequencies of hearing measurement. Therefore, some studies may have failed to find an association due to their use of a different definition of hearing loss.

Implications for practice and research

The need to treat AHRL is often unrecognized by older adults and health care providers as it is thought to be part of an inevitable aging process and is not a life - threatening condition [58]. However, ARHL should be considered a disorder that may be prevented or delayed and treated like any other chronic disease that has preventable features [59].

Although genetic factors cannot be changed, many risk factors in this literature review are modifiable and potential targets for delaying ARHL. The effect of risk factors seems greater in the younger population. Early detection of hearing loss through screening could help adults benefit from delaying ARHL and further progression. Health providers can screen hearing in populations with risk factors and educate them regarding lifestyle changes, such as smoking and diet, that may minimize hearing loss. Screening hearing should be a part of health checklist, especially for people who have CVD and diabetes, just like annual eye exams. Health providers should consider ototoxicity as an adverse effect of medications. Also, they need to refer high - risk populations for hearing loss to audiologist for further evaluation and treatment.

In the setting where no hearing test is available, a single global question like “Do you have a hearing problem?” may be useful to capture subjective hearing problems and to identify people who need a referral for formal audiometric testing. Using a simple question in the community setting is inexpensive, quick, and easy to administer without special equipment or training. Several simple objectives measures such as whispered voice test may be useful along with the single question in the identification of hidden or unrecognized hearing loss [60].

Although many risk factors associated with ARHL have been described, some may yet be discovered. It is still not known how many environmental and genetic factors contribute to the etiology of ARHL, how they interact with each other, or what their individual contributions are. Many studies for hearing loss still include only young adults or occupational settings for noise and chemicals. Retired older adults need further evaluation for the long - term effects of risk factors for ARHL. Further, several studies found the effects of diet, and medications were still controversial. Because it is difficult to know whether risk factors preceded hearing loss or vice versa in cross -sectional studies, longitudinal studies that can assess the impact of long - term exposure to various risk factors are needed to clarify their association with hearing loss in older adults. Identifying further risk factors for ARHL and improving appropriate interventions will be a worthy goal for further research.

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