



**Finnish Institute of
Occupational Health**

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ABSTRACT

There is large variation in hearing loss among workers exposed to the same noise levels. It would be helpful if we could predict the degree of susceptibility to NIHL for each individual worker. Therefore we conducted a systematic review of studies that evaluated how well individual related factors predict noise-induced hearing loss.

Methods:

We searched multiple databases for follow-up studies that compared the change in hearing level over time in workers exposed to noise louder than 80 dB(A) between groups with and without one or more predictive factors. If the exposure to the predictor varied over time, the factor had to be measured at baseline. We included studies regardless of time or language. We performed a meta-analysis of the mean difference in hearing loss per year or the relative risk of a certain amount of hearing loss per year between those with and without the predictor. When available, we choose the most adjusted risk estimate to be included in the meta-analysis.

Results

We located 12 studies that fulfilled our inclusion criteria which dated from 1966 to 2013 and were mainly carried out in the US. Six studies evaluated age, 4 gender, 4 previous hearing loss, 3 other noise exposures, 2 ethnicity and 1 disease-related factors. There were no studies that evaluated eye colour, smoking or cardiovascular risk factors. We found evidence for an association between higher age and previous exposure to noise and increased hearing loss but the magnitude of the association varied widely. The association was inconsistent for gender, skin colour and hearing levels at baseline.

Conclusions

Based on the currently available studies, no predictive model can be constructed. Older workers with previous and leisure time noise exposure are more at risk than other workers but the evidence for this association is limited. Better large studies are needed in which all here reported predictors are included during a follow-up of at least five years in workers exposed to noise louder than 80 dB(A).

YHTEENVETO

Systemaattinen kirjallisuuskatsaus kuulovaurion syntymistä ennustavista tekijöistä

Melulle altistuneiden työntekijöiden kuulonaleneman suuruus vaihtelee suuresti, vaikka altistus olisi samansuuruista. Olisi hyödyllistä jos voisimme ennustaa jokaisen työntekijän alttiuden melun aiheuttamalle kuulonalenemalle. Tämä tavoite mielessä suoritimme systemaattisen kirjallisuuskatsauksen siitä kuinka yksilölliset tekijät ennustavat melusta johtuvaa kuulonalenemaa.

Menetelmät:

Etsimme useista elektronisista viitetietokannoista seurantatutkimuksia, jotka vertailivat muutosta kuulon tasossa työntekijöillä, jotka altistuivat työssään yli 80 dB(A) melulle ja yhdelle tai useammalle ennustavalle tekijälle niihin työntekijöihin verrattuna, jotka eivät altistuneet ennustaville tekijöille tai altistuivat niille vähemmän. Jos altistuminen ennustavalle tekijälle vaihteli ajan myötä hyväksyttävien tutkimusten tuli olla mitannut sitä lähtötasolla. Hyväksyimme mukaan tutkimuksia riippumatta niiden julkaisuaajasta ja -kielestä. Ajoimme meta-analyysin keskimääräisen vuotuisen kuulonaleneman keskiarvojen erosta tai tietyn suuruisen kuulonaleneman riskistä verratessa niitä, joilla oli tietty ennustava tekijä niihin, joilla ei sitä ollut. Aina kun se oli mahdollista valitsimme eniten sopeutetun riskin arvion mukaan meta-analyysiin.

Tulokset:

Löysimme 12 tutkimusta, jotka täyttivät hyväksymiskriteerimme. Tutkimukset oli julkaistu välillä 1966 - 2013 ja enimmäkseen Yhdysvalloissa. Kuusi tutkimusta arvioi iän vaikutusta kun taas neljä tutki sukupuolen, neljä aiemman kuulonaleneman, kolme muiden meluallisteiden, kaksi kulttuuritaustan ja yksi sairauksiin liittyvien tekijöiden vaikutusta. Emme löytäneet tutkimuksia, jotka olisivat arvioineet silmien värin, tupakoinnin tai sydän- ja verisuonitaudin riskitekijöiden vaikutusta. Löysimme näyttöä korkeamman iän ja aiemman meluallistuksen yhteydestä korkeampaan kuulonalenemaan mutta yhteyden suuruus vaihteli suuresti. Sukupuolen, ihonvärin ja lähtötason kuulotason yhteys kuulonaleneman riskiin oli epäjohtonmukainen.

Johtopäätökset:

Tähän mennessä tehtyjen tutkimusten perusteella ei ole mahdollista rakentaa ennustavaa mallia. Vanhemmilla työntekijöillä, joilla on aikaisempaa melulle altistumista niin työssään kuin vapaa-ajallaan on suurempi riski saada kuulonalenema mutta tätäkin yhteyttä tukeva näyttö on rajallista. tarvitsemme parempia suuria seuranta tutkimuksia, joissa huomioidaan kaikki tässä katsauksessa raportoidut ennustavat tekijät ja joissa seurataan vähintään 80 dB(A) melulle altistuvia työntekijöitä ainakin viisi vuotta.

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1 INTRODUCTION

Noise is a prevalent exposure in many workplaces. Worldwide, 16% of disabling hearing loss in adults is attributed to occupational noise, while it is the second most common self-reported occupational illness or injury in the US (1). In Finland and in the Netherlands, noise induced hearing loss is the most common reported occupational disease. The number of workers that report a work-related cause of hearing loss is quite stable with around 1600 workers per year claiming compensation in the past five years. In the Finnish working conditions survey, approximately 25% of workers report to have been exposed to sound levels of 80 dB(A) or more during work (2).

Exposure to noise is especially prevalent in mining, manufacturing, music and the construction industry. (3) Construction workers are still considered as an underserved population when it comes to hearing loss prevention with one in twenty construction workers estimated to have occupational hearing loss (4, 5). An analysis of the noise exposure on construction sites shows the difficulties for preventive interventions in this industrial sector. Due to the setting and nature of the job, noise exposure varies over time and often there are combined exposures such as chemicals and vibration. Various workers in other trades work in the same environment, which puts also trades at risk that do not involve high exposure levels themselves.

1.1 *Noise-induced hearing loss*

The degree of reduction in ability to apprehend/perceive sounds can be regarded as hearing loss. Human ears can hear from a minimum of 500 KHz to 16 kHz and the ability of an ear to hear sound at a certain sound pressure level (dB SPL) varies across this range. The hearing loss is usually measured at specific frequencies across the audible range and is then presented as an average or composite of all dB values across a range of frequencies, or as the highest loss at any given frequency.

Long-term exposure to noise levels beyond 80 dB(A) carries an increased risk of hearing loss, which increases with the level and duration of noise exposure and ultimately this will lead to hearing impairment in some workers. (6) WHO defined hearing impairment as a hearing loss of "at least 25 dB in the better hearing ear (average over the frequencies 0.5, 1, 2 and 4 kHz)". (7) Since human conversation usually ranges between 0.5 to 2 KHz, a permanent threshold of more than 25 dB at frequencies between 0.5 to 2 kHz is considered to affect normal activities. Such level of hearing loss decreases the capacity to engage in conversation, in meetings or social activities, thus creating a significant barrier in establishing or maintaining emotional relationships and leading to isolation.

Hearing loss due to chronic exposure to noise occurs by causing damage to the outer hair cells in the cochlea in the inner ear. (8) The damage is permanent with no effective cure. (9, 10) However, the risk of noise-induced hearing loss can be greatly minimised if noise exposure is reduced to below 80 dB(A). (11)

1.2 *Effectiveness of preventive measures*

The preventive potential of reducing noise induced hearing loss has led to mandatory hearing loss prevention programmes in many countries. These range from reducing noise levels at the source (such as more silent machinery or shielding workers from the source), dampening of noise by protective equipment (such as earplugs) to mandatory screening (audiometry) or a combination of interventions.

Interventions to reduce noise at the source such as efficient design, retrofitting, and maintenance of equipment or special marks for extra quiet equipment are presented in the literature but these have not been evaluated nor sufficiently implemented (12, 13).

A recent systematic review of studies that evaluated interventions to reduce occupational exposure to noise concluded that even though case studies show an effect, there are no controlled studies indicating that specific interventions can be implemented and maintained at a larger scale. Also, the risk of hearing loss is still substantial despite being covered by a hearing loss prevention program. (14)

In workplaces where it is difficult to reduce noise levels at the source, one has to rely on the noise dampening effects of hearing protection. However, the use of hearing protection is dependent on human behaviour and there is only limited evidence on improving their uptake (15). Evidence indicates hearing protection by workers as part of hearing protection programs does not fully protect them in practice, although it was associated with some benefit. (14)

1.3 *Variation in susceptibility*

There is large variation in hearing loss among workers exposed to the same noise levels. As long ago as 1965, Taylor et al. reported a range from 0 to 60 dB hearing loss at 4 kHz after forty years of exposure to industrial noise at 115 dB(A). This is also reflected in the wide standard deviations for hearing loss in workers exposed to noise. (16) Based on the ISO predictions, Malchaire calculated that after 40 years of exposure to 100 dB(A) still about 40% of workers will not suffer from hearing impairment defined as an average hearing loss higher than 35 dB over frequencies 1, 2 and 3 kHz. (17) This means there is an individual predisposition for hearing loss, or at least a variation in risks.

All the preventive measures reported above are taken regardless of the underlying variation in risk of hearing loss. This means that 40% of workers will undergo these measures in spite of not being at increased risk of hearing loss. On the other hand, it might be necessary to monitor workers that are more susceptible to the damaging effects of noise more frequently. It would, therefore, be helpful if we could predict the degree of susceptibility to NIHL for each individual worker.

This might be possible with a predictive model that includes variables that are easy to assess for example at audiometric testing. There are, however, no valid clinical models available to predict the individual susceptibility for NIHL.

Studying predictive factors is a little different from studying causal or aetiological factors. Where in aetiology studies we are trying to find out if a variable can be reliably considered leading to an outcome, when adjusted for other confounding variables, in prognostic studies we try to use as many independent variables as we can to predict an outcome occurring in the future. (18) All variables associated with an outcome can be used as a predictor whether these are causally related to it or not. Therefore, models including many individually predictive variables are more useful than single factor studies to accurately predict future health or disease outcomes.

1.4 Predictive factors

There are factors that are considered to be associated with NIHL and therefore could be used to predict, as accurately as possible, the likelihood of developing NIHL in a worker exposed to noise over 80 dB(A) and the speed of progression of the NIHL.

These susceptibility factors can be divided into two major categories: person-related factors and work-related factors or occupational factors. Occupational factors additional to noise that are related to hearing loss (such as ototoxic chemicals, the use of hearing protection device (HPD) use, or vibration exposure) can be modified at a work place or group level by implementing protection that would protect all people irrespective of their individual susceptibility. Therefore personal factors become more important when an individual's risk is to be predicted.

Person-related factors can be categorized as follows:

- Personal physical characteristics such as skin and eye colour;
- Behaviour or life style factors such as smoking, noisy hobbies or hearing protective behaviours;
- Hearing related factors such as presence of tinnitus, baseline hearing thresholds/levels, temporary threshold shifts or family history of hearing loss;
- Disease factors such as diseases affecting the circulatory system or the ear, or medication factors such as ototoxic drugs.

An additional category could be genetic variations and predispositions. However current research is inconclusive on the association of any particular genetic mutation with an increased susceptibility for NIHL in humans. (19)

There is a general consensus that greater noise exposure, in duration or in intensity, leads to an increased risk of hearing loss. There is however no consensus over how to summarise noise intensities and durations into one cumulative index. Although consensus now exists among scientists that the exchange rate should be 3 dB (This is the exchange rate used for the ISO-1999 models),

the 5 dB exchange rate is also in use in the USA. The 5 dB exchange rate is a political compromise between safety and economic costs. Thus in the USA, OSHA increases the intensity with 3 dB(A) when the duration of exposure doubles but NIOSH does so with 5 dB(A). These measurement strategies are usually only used for short term measurements and a cumulative noise index over longer time periods of years is seldom used. For example with a 3 dB(A) exchange rate, an 8 hour exposure to 85 dB(A) is equal to an 4 hour exposure to 88 dB(A). With a 5 dB(A) exchange rate, the equivalent 4 hour exposure would 90 dB(A).

Age is an important factor that affects our risk of having lower hearing ability. There is ample data on the effect of increasing age on hearing loss.(20) Hearing loss starts already early in life but occurs at an increasing rate later in life from an age of about 30 to 40 year.

Studies indicate that one or more person related factors (physical characteristics) may be predictive for noise induced hearing loss. Skin colour and pigmentation of the iris, which is indicative of higher melanocyte functioning, have been reported to be associated with a decreased risk of hearing loss. In one study, there was an 8.9 dB difference in average hearing loss at 3, 4 and 6 kHz between dark eyed and fair eyed workers with similar levels of cumulative noise exposure.(21) In another study, individuals with a darker skin had better hearing thresholds across all frequencies than those with a whiter skin adjusted for demographic, medical and noise exposure variables.(22)

A possible network of interaction between the exposure, the effect, and the various predictive factors is presented in a directed acyclic graph below (figure 1). The Pink circles represent the factors that may be predecessors of both exposure and outcome: a variation in these factors affects both the risk of exposure to noise and the risk of NIHL. Blue circles are ancestors of outcome only: these do not affect one's risk of being exposed to noise at work but affect the risk of occupational NIHL. The grey ones are those which are not ancestral to either exposure or outcome but are seen associated with the outcome of NIHL or one of its ancestors and therefore can be predictive. The green lines indicate

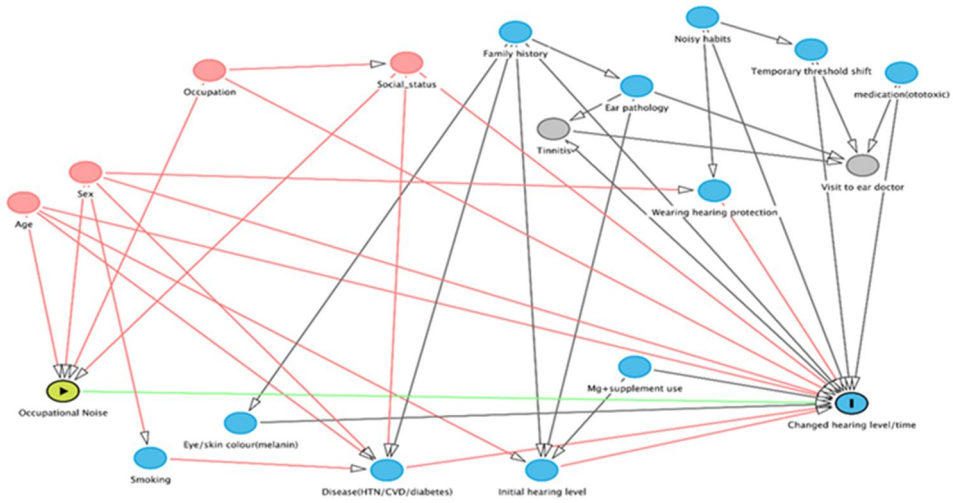


Figure 1. Network diagram to show relationships between various factors and NIHL

Legend: exposure; outcome; ancestor of exposure; ancestor of outcome; ancestor of exposure and outcome; causal path; biasing path

causal paths and the pink lines indicate biasing paths. In a risk factor study all the factors on the biasing path should ideally be adjusted for in primary studies to allow an unbiased estimation of causal influence of the exposure alone on the outcome. In a predictive model however, all these factors could be included whether causally linked or not. There may be more predictive factors which are not included in this diagram.

1.5 Reviews of predictive factors

A recent review considered age, smoking, exposure to organic solvents and carbon monoxide, and genetic risk factors for occupational noise induced hearing loss and recommended that assessments of these factors should be done to diagnose and prevent worsening of the condition in workers. Due to time limitations the authors could not assess some other associated factors such as gender, socioeconomic status, heavy metal exposure, use of certain ototoxic medications, cardiovascular disease and other medical conditions. (23) Another overview of prognostic factors for occupational noise induced hearing loss (24), even though quite comprehensive, did not report a systematic search or formal method to combine the study results. The overview included animal studies as

well, but the extrapolation of the results from animal studies to human beings is problematic because of differences in the shape, size, and hearing range of the hearing organs across species.

Therefore, we conducted a systematic review of human epidemiological evidence to evaluate factors easily assessable in clinical practice in order to predict the risk of noise-induced hearing loss in a worker of a certain age and exposed to a certain level of noise.

1.6 Review Question

To systematically review the strength of predictive factors of noise induced hearing loss (NIHL) for identifying workers at an increased risk

2 METHODS

2.1 *Search methods*

We searched Medline through PubMed, EMBASE, CINAHL, PsycINFO, and OSH Update using a search strategy developed for Medline (appendix A) and adapted appropriately for the other databases.

We checked the references from existing systematic reviews and explored if additional papers have been mentioned in relevant book chapters on NIHL. We contacted subject experts and authors of included studies with a request for information on any unpublished studies.

2.2 *Criteria for including studies*

Type of study/design

We included prospective and retrospective cohort designs. Retrospective cohort refers to studies where data available from employer or administrative databases were used in a post hoc analysis (study design and question were conceived after the data were collected). On the other hand, prospective cohort studies are those for which a pre-planned analysis was done; the study was prospectively designed before data were collected. Of these, retrospectively planned studies have a greater risk of bias than prospective cohort studies which may affect the reliability of their findings. For example such studies often do not show how many participants were initially identified in the database to fit the inclusion criteria but were not included in the analysis (attrition bias).

Since hearing loss due to occupational noise exposure happens over time, we included only studies that had followed up a cohort (retrospective or prospective). The reason for restricting to follow up studies is that these provide adequately reliable data for outcome prediction. (18, 25, 26). Cross sectional studies, although very useful to identify possible links between predictive factors and noise induced hearing loss in workers, present only one point measurements/estimates (distribution) and therefore do not account for the change that may happen due to variables other than the factor of interest over time. For example, it is hypothesized that by some not-well-understood mechanism of melanin functioning dark eyes protect against noise induced hearing loss. Comparing blue eyed and brown eyed people of the same age exposed to the same noise dose for NIHL, the only way we can attribute any difference in NIHL observed between the two groups to eye colour is if we have a baseline-to-follow-up change in NIHL reported for each group. Because it could well be that, by chance or systematically, one group started at a higher mean hearing threshold than the other. For factors that vary over time such as medication

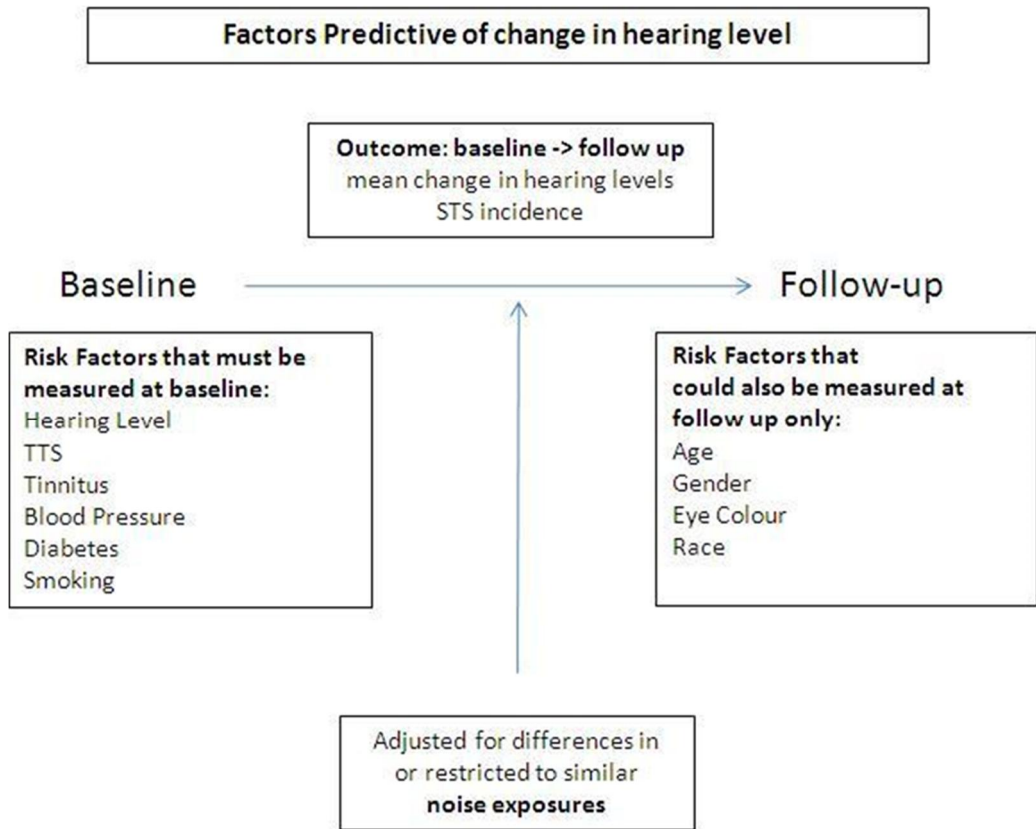


Figure 2. Measurement of variable and invariable predictive factors

use or hypertension, we would require a baseline value of the factor as well for establishing its ability to influence outcome at a later stage. So presence of diabetes or hypertension is to be measured at the start of follow up to be able to predict its effect on the final NIHL. The same is not needed for eye or skin colour because it can be safely assumed that these will be the same at all points in time.

Participants

Workers exposed to noise levels above 80 dB(A);

Exposure to predictive factors

Any predictive factor; we included but did not limit ourselves to studies assessing the following factors. We categorize these into four types:

- Person-related physical characteristics such as skin colour, eye colour, or characteristics such as age, sex and socioeconomic status.
- Behavioural factors such as smoking, noisy occupation or habits outside of work (e.g. music), or hearing protection
- Hearing related factors such as baseline hearing levels, permanent or standard hearing threshold shifts, hearing loss at 4KHz, and tinnitus etc.
- Medical or medicinal factors such as cardiovascular disease, hypertension, diabetes, another condition that may increase susceptibility to hearing loss, using Mg+ supplements or ototoxic medication

Comparison

Absence of exposure to the predictive factor(s) under study, or a lower level of factor exposure.

Primary Outcome

Noise induced hearing loss (NIHL), which is defined as a change in hearing level measured over time by valid audiometric tests, reported at 4 kHz or a range of frequencies and expressed as dB/year. The outcome was to be reported for a minimum of one year of follow up as either a change from baseline (e.g. STS incidence) or both baseline and follow up measurements are made available. Since high noise exposure leads to high risk, a study stating NIHL outcomes for a whole range of noise levels (for example over 80dBA) is not as useful as a study that either adjusted for the various noise exposure levels, or reported risk for each level or category (for example 80- 85, 85-90, and so on)

Effect measures were risk ratios (RR) for dichotomous outcomes and Mean Difference (MD) for continuous outcomes.

Exclusion criteria

A study was excluded if

- the study population included animals, cadavers, dummies, or patients with known anatomical/developmental/other pre-existing sensory-neural hearing loss;
- studies were without a follow up (elicited history of exposure to a factor after development of NIHL had occurred - case control), or studies without a reference group;
- studies were on genetics (DNA characteristics)

2.3 *Data collection and analysis*

Study selection

A pilot tested inclusion criteria template, specific to the research question of this review, was used. Each title and abstract identified from the search was independently checked by two reviewers on whether it fulfils all criteria. All review authors were involved in assessment so that each publication was independently assessed by at least two reviewers. Disagreements on inclusion were resolved by discussion. Personal communication was established with the author(s) for obtaining additional information regarding reports. Studies excluded after full text assessment were tabulated with reasons for exclusion.

Data extraction

Data extraction from included studies was done in duplicate on a pre-piloted standard form. If disagreement occurred it was resolved by discussion or by involvement of a third reviewer. The studies and their characteristics were tabulated in an Excel file by one reviewer (SI) and checked by another (JV) before analysis.

The following data were extracted and described in a table of included studies listed by study ID (first author/ year of publication):

Country of origin; Year of study start and duration of follow up; Study design; Participants' characteristics including age, sex, occupation; Exposure to noise; Outcome(s) hearing loss; Predictive factors; Risk of bias; Method of analysis; Study results RR and OR, adjusted and/or crude; Authors' conclusions.

Since studies in NIHL area often report data in various ways we used the following rules to enable consistent choices: data for 4 kHz frequency were chosen over all others, since this is the frequency at which most often the initial hearing loss is seen. It was therefore considered more important for predicting and preventing further damage to hearing. When data were presented as averaged over various frequencies, we chose the one closest to 4 kHz, for example average of 2, 3 and 4 were chosen over 1 2 and 3. Averaged data over both ears were chosen over single ear data, however when data were presented separately for each ear we chose left ear data always.

Assessment of risk of bias (quality) in included studies

We assessed sources of bias relevant to our question such as bias related to outcome and predictive factor definitions, units of measurement and measurement approaches, and imputation methods for missing data, using the QUIPS tool.(27) The tool has six domains for assessing potential biases and we assessed studies on all these domains in three categories of low moderate or high risk of bias. We categorised the six domains into only low or high risk of bias, however the overall risk per

study was categorised into low moderate or high risk to distinguish studies of very poor quality from relatively better though not ideal quality:

1 Study participation- the goal is to judge the risk of selection bias. This included the following components: description of place, method and time of recruitment, sampling frame, inclusion criteria, adequate participation rate for eligible population, and description of key characteristics of the baseline sample selected.

We marked a study at low risk when inclusion criteria and description of key characteristics of the baseline sample selected were both marked at low risk and none of the other components mentioned above were at high risk. The rest were considered high risk in this domain.

2. Study attrition- the goal is to judge the risk of bias due to differences between completing and non-completing participants. This included assessment of: response rate, reasons for drop out, description of the drop out sample, and attempts to collect missing information. We considered at study at low risk of bias in this domain when two independent authors judged that the loss to follow-up is low (less than 20% overall) and is well described to show little or no difference between the dropping and completing participants. Studies with high drop out and/or no description of dropout rate/ difference between the dropping and completing participants were considered at high risk in this domain.

3. Predictive factor measurement- the goal is to determine if the predictive factor (PF) has been measured adequately enough to sufficiently limit potential bias. This included components of a clear definition, valid and reliable method of measurement which is the same between compared groups, use of appropriate cut-points which are not data dependent, adequate number of sample providing information on PF, and appropriate imputation in case of missing information.

We considered a study at low risk of bias when predictive factors were clearly defined and validly measured for all participants (low risk marked for clear definition and for valid and reliable method of measurement which is the same between compared groups) and the remaining components of this domain were not at high risk.

4. Outcome measurement- the goal was to ensure that the outcome of interest was adequately measured in study participants to sufficiently limit potential bias. This was assessed on the following points: clear definition, valid and reliable measurement method which is similar across groups compared. A study that fulfilled all three criteria was considered at low risk of bias in this domain. If not, it was marked at high risk in this domain.

5. Study confounding- the goal was to assess whether important potential confounders for each PF are appropriately accounted for, limiting potential bias with respect to the relationship between PF and outcome. To this end we listed potential confounders for each PF based on existing evidence from the literature:

- Age = initial hearing levels, disease(CVD, diabetes, ear pathology), previous noisy job

- Gender = HPD use, CVD, smoking
- Melanin indicators = smoking, disease, social status
- Disease = age, gender, social status, family history of hearing loss, medication history
- Ear pathology or symptom = family history of hearing loss, disease, medication history, noisy
- Behaviours (smoking, noisy previous jobs/habits, HPD use, seeing doctor) = age, gender, social status
- Baseline hearing levels = age, gender, previous noisy jobs, disease, family history of HL
- Mg+ supplement use = initial hearing level, previous noisy job,

The list provides all important confounders for a given predictive factor; however it is not an absolute requirement to adjust for all of these: it may be that for a given sample a certain confounder may not be relevant. For example a study that reported age as a predictive factor using 20 to 25 year old participants, it was considered low risk even when disease factors such as CVD were not adjusted for, since in young people these factors are rarely present.

Therefore a study was considered to be at low risk of bias if it was agreed between two authors that the important confounders for the PF reported in that particular study were validly measured and adjusted for. When enough information was not available to judge this or when it was clear that all or some of the important confounders were not accounted for we marked a study as high risk.

6. Statistical analysis and reporting- goal was to assess whether statistical analysis is appropriate for the design of the study, limiting potential for presentation of invalid or spurious results. This included assessing any selective reporting of results, inappropriate analysis methods for the given design, and sufficient description of data and model strategy to rule out risk of bias.

A study was considered at low risk if two of these criteria were fulfilled (marked low risk). A study was marked high risk if less than two of these criteria were fulfilled.

7. Overall risk of bias- Studies were categorized into high, moderate and low overall risk of bias. We decided that the four domains of study attrition, predictive factor measurement, outcome measurement, and study confounding were more important for this review question (major domains for bias) than the two domains of study participation and statistical analysis (minor domains for bias).

Therefore a study was considered at low risk of bias when the four major domains and at least one minor domain were at low risk. When a study had only one major domain at high risk the study was considered at moderate risk of bias. When a study had two of the major domains at high risk the study was considered at an overall high risk.

Sensitivity analyses were done where possible to assess the effect of inclusion of low quality studies and the effect of the overall risk of bias in the studies on our results described.

Dealing with missing data

Missing data from the articles on effect size, and nature of analysis was asked from authors. We imputed missing standard deviation for mean change from baseline for a study (28) from a correlation coefficient derived from the only study that provided information on SD of change from baseline (29), as advised in the Cochrane handbook section 16.1.3 (30).

Data synthesis

Where possible a random effects meta-analysis was performed combining relative risks of yearly rates of hearing loss of a specific amount of decibels or mean differences in hearing loss per year across studies for each outcome. Where data were presented for several years, these were transformed into per year risks (RR/year) and mean differences per year (MD/year) for the analyses. For example, a mean difference of 12 dB in hearing loss over a period of six years will be transformed or recalculated into 2 dB/year hearing loss.

Assessment of reporting biases

We avoided language bias by including studies in any language and outcome reporting bias by contacting authors. Publication bias was avoided by searching for unpublished studies and data, and a formal assessment of publication bias by inspecting Funnel plot was done for comparison 1.1 where four studies were pooled. Eggers test was not done as not enough studies could be statistically combined.

3 RESULTS

3.1 Search results

One hundred and forty four papers were assessed in full text of which 16 were included; representing 12 studies. Excluded studies with reasons are available as appendix B. Fourteen studies are awaiting assessment due to translations needed from Japanese and Russian.

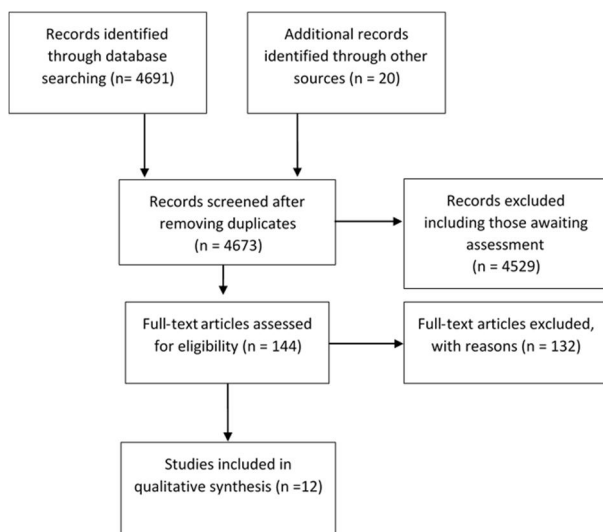


Figure 3. Prisma flow diagram

3.2 Characteristics of included studies

Characteristics of included studies are in table 1.

Study design

A total of 12 studies were included (28, 29, 31-39). Two of these were reported as separate cohorts of males and female within the same publication (37).

All were in English language. Nine studies were from the USA and one each was from Sweden (39), Canada (33) and South Africa (29).

Ten studies were of a retrospective cohort design (collected data already for purposes other than that study were accessed retrospectively for analysis) while two (28, 32) prospectively followed the cohort (data were collected from participants prospectively for the study).

The studies were published between 1966 (34) to 2013 (35). The follow up periods ranged from 3 to 19 years with a median of 6 years.

Participants

Sample size ranged from 14 (34) in the smallest study to 5478 (37) in the largest (Table 1). Most studies were large with eight studies analysing over a thousand participants each with an average of 1560 participants across studies. Both males and females were included in five studies (29, 31-33, 38]. The female participation/inclusion, when present, consisted of between 3 to 10 % of the total. Female only data were reported in one study.(37) Three studies reported data on males only (28, 36, 37), and for another two (33, 39) it was assumed based on the information in the reports. For (35) it was unclear if the data included that of females from the original cohort.

Participants were from textile industry in two studies (28, 31), from mining in one study (29), from food and automotive industry in two studies (37), and from aluminium industry in one study (35). Occupations included welding (2 studies), factory workers (5 studies), various occupations (6 studies), and drillers, grinders and heavy component assemblers (one study each).

Table 1. Characteristics of included studies: design, participants, exposure, outcome and predictive factors.

Study ID (Author Publication year)	Study design	Year start / noise exposure	Location	Industry/ Occupation	N enrolled	N analysed
Brits 2012 (29)	Retro-spec-tive cohort	2001 6 years	South Africa	Gold mining/ Drillers, admin staff	60412	2127 (table 4)
Erlandsson 1983 (39)	Retro-spec-tive cohort	1977 4 years	Sweden	Shipyard/ Weld-ers/ grinders/ heavy compo-nent assembly workers	58	29
Franks 1989 (38)	Retro-spec-tive cohort	1977 NR	USA	Publishing / Va-rious	2373	1693
Heyer 2011-F (37)	Retro-spec-tive cohort	1970 max.1 1 years	USA	Food and auto-motive/ Various	1061	1005
Heyer 2011-M (37)	Retro-spec-tive cohort	1970	USA	Food and auto-motive / Various	5774	5478

		max. 11 years				
Pell 1973 (36)	Retro-spec- tive cohort	1966 5 years	USA	Chemical/ Fac- tory workers	2770 selec- ted	1966 (from tables)
Rabinowitz 2013 (35)	Retro-spec- tive cohort	2006 6 years	USA	Aluminium ma- nufacturer / Fac- tory workers	NR	107
Royster 1984 (28)	Retro-spec- tive cohort	NR 7 years	USA	Textile / Factory workers- spin- ning	100	89
Sataloff 1966 (34)	Prospective cohort	1961 3 years	USA	Paper / Factory workers-machine operators	21	14
Sbihi 2010 (33)	Retro-spec- tive cohort	1978 20 years	Ca- nada	Lumber/ Various	2948	2938
Seixas 2005 (32)	Prospective cohort	1998 6 years	USA	Construction / Various	372	278
Smith 1980 (31)	Retrospec- tive cohort	NR 5 years	USA	Textile / Factory workers	NR	1517

Table 1. Continued.

Study ID (First au- thor Publication year)	Age base- line M (SD) / Range Sex (M/F)	Noise exposure (mean/ range)	Outcome defini- tion	Outcome/effect pre- sentation	Predictive fac- tors
Brits 2012 (29)	37.6 M+F (3.3% female)	108 db(A)	Change in mean hearing level at each ear left or right as average over 0.5, 1, 2; 3, 4, 6 khz,	Mean change from baseline (SD) for each predictive factor Mean (SD) in db over 6 years	TB, no TB, single epi- sode TB (no streptomy- cin); multiple epi- sodes (treated with streptomy- cin)
Erlandsson 1983 (39)	Approx. 40 / 20 to 60 M (assumed)	Leq 84 to 94; Level at ear 90 to 110 db(A)	Change in mean hearing level as aver- age of 0.5 to 2 and av- erage of 2 to 8 khz	Mean (SD) db/year change of total sample	Age

Franks 1989 (38)	36 M+F	85 dba/ 80 to 92 db(A)	Change in mean hearing level (STS > 10db) averaged over 2 3 4 khz	Proportions as % with hearing loss in text, p <0.05 indicated in graphs	Occupational noise expo- sure, non-occu- pational noise exposure, self- reported medi- cal conditions, sex, age
Heyer 2011-F (37)	39.2(10.9) F	Duration stratified by TWA noise intensity (<80, 80 to <85, 85 to <90, 90 to <95, >95 dbA).	Change in mean hear- ing level at 3, 4 or 6 khz	Beta's from GEE indicating more or less hearing loss (MD of change dB/year)	Age, noise ex- posure, base- line hearing
Heyer 2011- M (37)	39.2(10.9)M	Duration stratified by TWA noise intensity (<90, >90 dbA).	Change in mean hear- ing level at 3, 4 or 6 khz	Beta's from GEE indicating more or less hearing loss (MD of change dB/year)	Age, noise ex- posure, base- line hearing
Pell 1973 (36)	39.3 years /17 to 64 M	Categories: I= all bands under 100 in 20-300 Hz. and under 85 dB from 0.3 to 10 kHz; II= under 100 in low freq., 85-94 dB from 0.3-10 kHz; III= all levels in low freq., 95+ from 0.3 to 10 khz	Change in mean hear- ing level at 4 khz	Mean dB change from baseline per group.	Per noise level category, base- line HTL three ordinal catego- ries ranging from 85 to over 95 dB(A) for noise level three ordinal categories: 10 dB or less, 15- 35 dB, 40 dB or more as base- line hearing
Rabinowitz 2013 (35)	49.8 (7.2) M+F (assu- med)	Ambient Lavg: 86.6 dB(A) (IQR74-7 to 88.4)	Change in mean hear- ing level for: frequen- cies 500 to 8000Hz; average of 2, 3, 4 KHz and 3, 4, 6 KHz	MD(SE) of dB/year change over 5 years	Age, occupati- onal noise ex- posure
Royster 1984 (28)	29.75 (9) M	84 to 107 dB(A)	Mean hearing thresh- old at first and 4th an- nual tests at all fre- quencies from 0.5 to 8 (chosen 4 khz per year)	before and af- ter hearing lev- els (measure- ments per group in dB/year)	Ethnicity

Sataloff 1966 (34)	44.5/ 20 to 60+ M+F	93dB to 100dB in 300-2400 octave band widths.	Change in mean per- manent threshold at any frequency from 0.5 to 8 khz; propor- tion of people with 10 dB or more PT and TT at any frequency be- tween 500 to 8000 Hz.	Unclear: results do not factor in the predictive effect of TTS; data not re- ported.	Temporary threshold shift
Sbihi 2010 (33)	NR 90% men	99.7 dB(A)years/ 91.6 to 106.7 dB(A)years	Change in mean hearing level aver- aged across both ears and 0.5 1 2 and 4 khz	Log trans- formed out- comes (beta) for MD of aver- age dB/year change	Age, noise, gender, ethnic- ity, intrinsic fac- tors(med con- ditions), non- occ factors(fire- arms)
Seixas 2005 (32)	27.5 (6.6) M+F (90% M)	Adjusted for HPD 77.3 to 89.5; unad- justed 81.8 to 90.7	Change in mean hear- ing level at 4 khz	dB/year change per group; MD (95% CI) of dB/year change	Gender, age, prior exposure, baseline hear- ing
Smith 1980 (31)	17 to 64 me- dian 41 M+F	Categories: under 85 dB(A) no hearing plugs; > 85 dB(A) cus- tom hearing plugs; 85-89 dB(A), 90-94 dB(A), 95-99.9dB(A), 100-104.9 dB(A); other (intermittent exposure)	Change in mean hear- ing level at 0.5, 1, 2, 3, 4 and 6 khz	Regression coefficient pre- sented only	Job: noise level, job location, plant, years employed; De- mographic : sex, age; Hear- ing: ear history, audiogram type, audio- gram years, baseline hear- ing. Two-way inter- action: Noise level by audio- gram, noise level by sex

Exposure to noise

The noise exposure ranged from 80 to 110 dB(A) in the included studies. Exposure measures for noise used by the included studies varied but the most often reported measure was a range or an average value in dB(A) in six studies (28, 29, 31, 34, 36, 38). Four studies reported Leq values for exposure.(32, 37, 39). Only Shibi 2010 (33) provided a mean cumulative dose of noise exposure over years in dB(A). Rabinowitz 2013 (35) reported a cumulative measure of time weighted Lavg in dB(A).

Predictive factors and comparisons addressed

The set of predictive factors assessed in the included studies is provided in the table 2 below. The most often assessed factor was age assessed in five studies; gender was the second most common - assessed in four studies. This was followed by baseline hearing threshold (BLHTL) in three studies, two as low and high categories and one as a continuous variable. A history of previous noisy jobs was evaluated in two studies.

Personal factors

Studies compared varying categories of the predictive factors. Age was used as a continuous variable in four studies (33, 35, 37), and as a comparison of under 30 versus over 30, and of under 35 and over 40 in one study each.

White ethnicity was compared to black in one study (28) while Chinese and East Indian ethnicity was compared to White in another (33).

Gender was compared in three studies (32, 33, 38). Heyer 2011 (37) presented female and male data separately but did not compare the results of both genders.

Disease factors

Brits 2012 (29) compared no tuberculosis (TB) versus simple (first episode) TB treated with standard therapy and with multiple TB treated with additional streptomycin - a known ototoxic medicine.

Behavioural factors

Noisy habits (non-occupational exposure) were assessed as a comparison of previous fire arm noise exposure versus no such exposure, and comparison of previous blast exposure versus no such exposure in one study (33). Two studies compared previous noisy jobs versus no previous noisy jobs.

Better versus poorer use of hearing protection devices was assessed in two studies.(37)

One study (33) compared a history of a visit to an ear doctor with no history of such a visit.

Hearing test characteristics

Two studies assessed the influence of elevated hearing levels at baseline as a predictor (37). Two studies compared lower than 10 dB hearing loss at baseline with higher than 10 dB baseline categories (32, 36).

Table 2. Predictive factors and number of studies

Predictive category	Predictive factor	Factor levels compared	Number of studies
Personal factors	Age	Yearly increase in age	4 (33, 35, 37)
		Under 30 years vs over 30 years	1 (32)
		Under 35 vs over 40 years	1 (39)
	Ethnicity	White vs black	1 (28)
		Chinese vs East Indian	1 (33)
	Gender	Male vs female	4 (32, 33, 37)
Disease factors	TB	No TB vs TB with treatment 1 vs TB with treatment 2	1 (29)
	Ototoxic medication TB	Tb treatment with streptomycin vs without streptomycin	1 (29)
Behavioural factors	Noisy habits,	Firearm use vs no fire arm use	1 (33)
		Blast exposure history vs no blast exposure	1 (33)
		Previous noisy job	2 (32, 33)
	HPD use	Better HPD use vs poor HPD use	2 (37)
	Doctor visit	Ear doctor visit history vs no visit history	1 (33)
Hearing test characteristics	Baseline hearing threshold level (BLHTL)	Per unit increase in BLHTL	2 (37)
		Low BLHTL vs High BLHTL	2 (32, 36)

Outcome

Studies defined the outcome of interest in more or less similar ways (Table 1). Change in mean hearing levels were reported as an average over a range of frequencies by four studies, separately for a range of frequencies in five studies, and at the 4 kHz frequency in two studies.

The effect of the predictive factor on hearing loss was presented in various ways: mean difference between groups was presented in one study; regression coefficient in one study; change from baseline per group in three studies; beta from predictive modelling in three studies (one used maximum likelihood estimation, while the other two used generalized estimating equations); mean change for the entire sample together was presented in one study; proportions of people with hearing loss were presented in text and in graphs in one study; one study reported number of people with permanent threshold shifts at a certain frequency in text only. We tried to transform all these effect sizes as much as possible to enable meta-analysis but given its wide variation this was only possible to a limited extent.

3.3 Characteristics of excluded studies

One hundred and two studies have been excluded at the full text stage in this review. Of these ten were reviews, and eight method studies with no empirical data. Reasons varied from study design to outcome measurement and studies were excluded for more than one reason also. A lack of follow up and therefore no presentation of change from baseline for the outcome of NIHL was the most common reason for exclusion (49 studies). A short follow up of 2 months was a reason for excluding two studies. Nine studies had not measured the outcome of interest (NIHL), and 19 had no predictive factor assessment reported. Noise was not measured in 35 studies. Additional exposures to chemical or vibration were reported in two studies. Studies with reasons are tabulated in appendix B.

3.4 Risk of bias in included studies

For bias due to differential participation in the study, six studies were at low risk in this domain and six at high risk. Study attrition was not a problem in three studies that were thus judged at low risk here and the remaining at high risk of bias. The measurement of predictive factors was problematic in four studies that were considered at high risk in this domain and eight studies were at low risk of bias. The outcome measurement was valid in all except one study and these were judged at low risk of bias in this domain except the one study. Only half of the studies adjusted correctly for confounding: six studies were at low risk in this domain and an equal number at high risk. Analysis was not appropriately done in most studies: only four studies were at low risk in this domain, and the remaining eight at high risk.

Our judgement of the overall risk of bias resulted in the following:

- two studies were at low risk of bias over all (37)
- three studies were at moderate risk of bias (28, 33, 36)
- seven studies were at high risk (29, 31, 32, 34, 35, 38, 39)

Table 3. Risk of bias in included studies

Study ID	study participation	study attrition	predictive factor measurement	outcome measurement	study confounding	statistical analysis and reporting	OVERALL ROB
Brits 2012	low risk	high risk	low risk	low risk	high risk	high risk	high risk
Erlandsson 1983	high risk	high risk	high risk	low risk	high risk	high risk	high risk
Franks 1989	high risk	high risk	low risk	low risk	high risk	high risk	high risk
Heyer 2011-M	low risk	low risk	low risk	low risk	low risk	high risk	low risk
Heyer 2011-F	low risk	low risk	low risk	low risk	low risk	high risk	low risk
Pell 1973	low risk	low risk	low risk	low risk	high risk	high risk	moderate risk
Rabinowitz 2013	high risk	high risk	high risk	low risk	low risk	low risk	high risk
Royster 1984	high risk	high risk	low risk	low risk	low risk	low risk	moderate risk
Sataloff 1966	high risk	high risk	low risk	low risk	high risk	high risk	high risk
Sbihi 2010	low risk	high risk	low risk	low risk	low risk	low risk	moderate risk
Siexas 2005	low risk	high risk	high risk	low risk	low risk	low risk	high risk
Smith 1980	high risk	high risk	high risk	high risk	high risk	high risk	high risk

3.5 Effects of predictors on rate of hearing loss

Meta-analyses were conducted only for those comparisons where these were sufficiently similar and where outcomes were presented in the same way across studies. The studies and their results are summarized in tables by comparison and outcome in the included studies.

Personal factors

Age

Five studies in total assessed the effect of age as a predictor of noise induced hearing loss. (Table 4)

Table 4 Effect of age on rate of hearing loss

Comparison	Outcome	No. of studies	No. of participants	Effect Size	Effect sizes A positive value indicates worse hearing for the index group
yearly increase in age	dB/year	4	9421	Mean Difference (95% CI)	Shibi 2010 (33): 0.05 (0.05 to 0.06) after exponentiation 1.05 (1.05 to 1.06) Heyer-F (37): 0.08 (0.07 to 0.10) Heyer-M: 0.08 (0.05 to 0.11) Rabinowitz 2013 (35): 0.04 (0 to 0.08)
Over 30 years vs Under 30 years	dB/year	1	278	Mean Difference (95% CI)	Seixas 2005 (32): 3.92 (1.94 to 5.9)
Over 40 years vs Under 35 years	dB/year	1	29	Mean Difference (95% CI)	Erlandsson 1983 (39): 0.64 (0.36 to 0.92)

All four studies in this comparison individually indicate that increase in age increases the susceptibility to NIHL. Older people seemed likely to incur more NIHL per year in all studies than younger people. However, after log back transformation the results in Sbihi indicated a 10- to 25-fold bigger hearing loss increment per year than for the other studies. Therefore we refrained from meta-analysis. Restricting the analysis to the moderate risk of bias studies by excluding Rabinowitz 2013 (35) did not alter these results. We could not find an explanation for the enormous difference in effect size between Sbihi 2010 and the other studies.

Age over 30 years versus under 30 years

Seixas et al (32) comparing under 30 with over 30 workers show that people over 30 have the mean NIHL higher by 3.92 dB (95% CI, 1.94 to 5.9) compared to those younger than 30.

Age over 40 years versus under 35 years

Data from Erlandson et al (39) indicates the older workers (40 plus from figure) to have an average of 0.64 (95% CI, 0.36 to 0.92) higher mean NIHL than younger workers (under 35 from figure).

Ethnicity

Two studies addressed ethnicity as a predictor of NIHL but compared different ethnicities and therefore we did not combine them in a meta-analysis. (Table 5)

Black versus white

Royster 1984 (28) compared white men with black and found that white males had a 0.73 dB/year higher rate of hearing loss than blacks of the same age and noise exposure.

Chinese versus White

Sbihi 2010 (33) addressed this comparison, indicating that Chinese were had a 1.27 dB/year higher hearing loss on average compared to White people

East Indian versus White

East Indians had -1.2 dB/year less hearing loss than Whites but this was not significant. (33)

Table 5 Effect of ethnicity on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes A positive value indicates worse hearing for the index group
Black vs White	dB/year	1	89	Mean Difference (95% CI)	Royster 1984 (28): -0.73 (-1.62 to -0.12)
Chinese vs White	dB/Year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): 0.24 (0.11 to 0.36) after exponentiation 1.27 (1.12 to 1.43)
East Indian vs White	dB/Year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): -0.18 (-0.4 to 0.1) after exponentiation -1.20 (-1.49 to 1.10)

Gender

Three studies reported assessment of gender as a predictor. Two studies indicate men to be at a higher risk and one women but none of the results showed a statistically significant difference. (Table 6)

Male versus female

Outcome- Rate of hearing loss dB/yr

Sbihi 2010 (33) indicated that females had a higher hearing loss than males but this was not significant. Seixas 2005 (32) showed an increased NIHL for men per year but this was also not statistically significant. To be able to combine studies we reversed the comparison in Sbihi by adding a

minus sign to the effect size and the confidence interval. Combined in a meta-analysis the pooled mean difference was 0.27 dB/y (95% CI -2.19 to 2.73) with $I^2 = 28\%$.

Outcome- Risk of STS

Data from Franks 1989 (38) indicated almost 50% higher risk (RR) for men for a STS than women but this was not statistically significant.

Table 6 Effect of gender on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes in dB/y Positive values indicate an increased risk of NIHL
Males versus females	dB/year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): -1.32 (-4.4 to 2.56)
	dB/year	1	278	Mean Difference (95% CI)	Seixas 2005 (32): 1.26 (-1.23 to 3.75)
	Participants with STS	1	1693	RR (95% CI)	Franks 1989 (38): 1.45 (0.71 to 2.98)

Disease - TB

Simple TB versus No TB

A single study (29) assessed this factor showing that under treatment first episode of TB can increase the mean hearing loss by 0.75 dB/year. (Table 7)

Multiple TB versus No TB

Brits (2012) also compared multiple TB episodes (treated appropriately) with No TB and reported an increased mean hearing loss in the TB group of 1.05 dB/yr after multiple treatments.

Table 7 Effect of tuberculosis on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes Negative value means increased risk (worse hearing for index group)
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TB simple treated vs no TB	dB/year	1	1757	Mean Difference (95% CI)	Brits 2012 (29): -0.75 (-0.91 to 0.42)
TB multiple vs no TB	dB/year	1	1207	Mean Difference (95% CI)	Brits 2012 (29): -1.05 (-1.25 to -0.93)

Ototoxic medication

Multiple TB versus simple TB

Brits 2012 (29) comparing first episode TB treated with standard medication and multiple episodes TB treated with additional streptomycin showed that streptomycin treated people had on average the hearing worsened every year but this was not statistically significant. (Table 8)

Table 8 Effect of ototoxic medication on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes
TB treated with additional streptomycin vs TB treated with standard therapy	dB/year	1	1296	Mean Difference (95% CI)	Brits 2012 (29): -0.23 (-0.5 to 0.04) negative value means increased risk (worse hearing)

Behavioural Factors

Previous noise exposure

Two studies addressed this factor Sbihi 2010 (33) and Seixas 2005 (32). (Table 9)

Table 9 Effect of previous noisy jobs on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes Positive means higher risk (worse hearing)
Firearm use history vs no fire arm use	dB/year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): 1.1 (95% CI 1.03 to 1.2)

Blast exposure history vs no blast exposure	dB/year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): 1.6 (95% CI 1.5 to 1.8)
Previous noisy job vs no previous noisy job	dB/year	2	3216	Mean Difference (95% CI)	Sbihi 2010 (33): 1.26 (95% CI 1.1 to 1.35) Seixas 2005 (32): 1.25 (95% CI -0.83 to 3.33)

Fire arm exposure history versus no fire arm exposure

Sbihi 2010 (33) was the only study assessing this factor in two categories: firearm use. They reported a 1.1 dB (95% CI 1.03 to 1.2) higher NIHL in people who have previously used firearms.

Blast exposure history versus no blast exposure

Exposure to blast noise was compared to no such history in one study (33) which indicated 1.6 dB (95% CI 1.5 to 1.8) higher NIHL in people with previous exposure to blast compared to those without such exposure.

Previous noisy job versus no previous noisy job

Previous exposure to noise at work was assessed as a factor in two studies (32, 33). Both reported an increased NIHL in people with history of noisy jobs and the effect sizes were consistent.

Random effects meta-analysis showed a significant increase of 1.26 dB/year (95% CI 1.14 to 1.38) in hearing loss if one had a previous noisy job. The studies controlled for age. There was no statistical heterogeneity ($I^2 = 0\%$).

HPD use

Better HPD use versus poor HPD use

Effect of better use of HPD was reported by two studies Heyer et al 2011 M/ F (37). Men using HPD more appropriately incurred less hearing loss than those who did not, by -0.31 dB/yr. Females with better HPD use were better off by -0.14 dB/yr than their worse HPD use counterparts. The effect was significant in both studies. (Table 10) When pooled together the effect size was -0.24 dB/yr (95% CI -0.40 to -0.07) but with substantial heterogeneity ($I^2 = 81\%$) because the effect was almost twice as big in men compared to women.

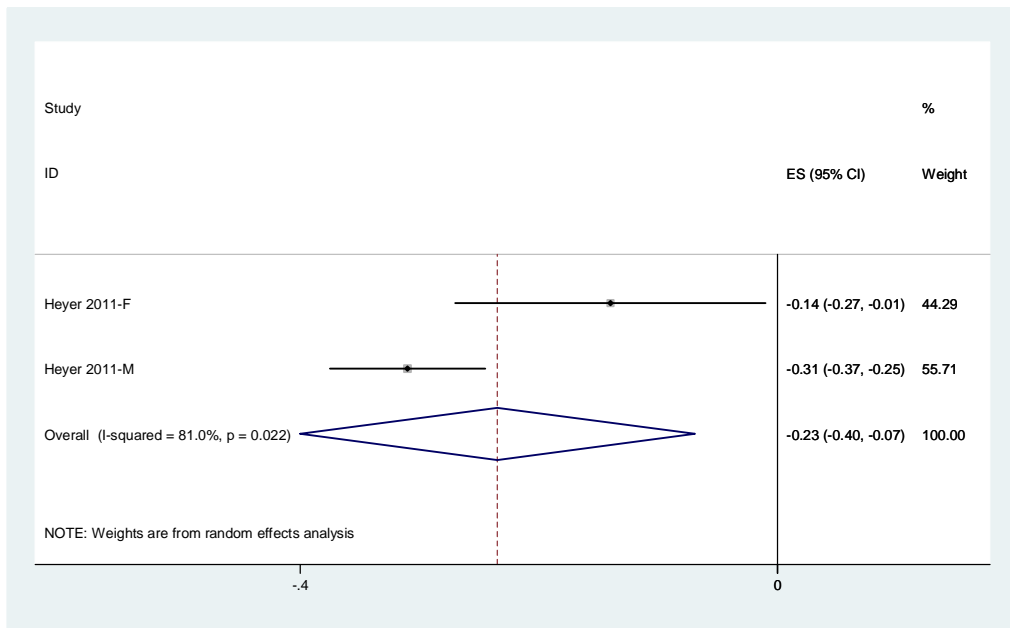


Figure 3 Better HPD use versus poor HPD use; Outcome NIHL (dB/yr); ES= effect size (MD)

Table 10 Effect of hearing protection device use on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes Negative value indicates lower risk(better hearing)
Better HPD use vs poor HPD use	dB/year	2	6483	Mean Difference (95% CI)	Heyer 2011-F (37): -0.14 (-0.27 to -0.01) Heyer-M (37): -0.31 (-0.37 to -0.24)

Visit to ear doctor

History of visit to ear doctor versus no history of ear doctor visit

A visit to an ear doctor in the past was indicative of less hearing loss of 1.12 dB/year according to a single study. (Table 11)

Table 11 Effect of visit to an Ear doctor on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes Positive means higher risk (worse hearing)
history of ear doctor visit vs no visit to ear doctor	dB/year	1	2938	Mean Difference (95% CI)	Sbihi 2010 (33): -1.12 (-1.21 to -1.03)

Hearing test characteristics

Baseline hearing threshold levels (BLHTL)

Three studies assessed this factor. Table 12.

Incremental increase in baseline hearing level

Two studies, Heyer M and F (37) reported similar findings for men and women, a lower hearing loss with higher baseline hearing level. The pooled effect in random effects meta-analysis showed a per unit baseline hearing level increase to lower the hearing loss of -0.03 dB (95% CI -0.041 to -0.021) per dB increase baseline hearing loss with no statistical heterogeneity.

High baseline hearing loss (> 10 dB) versus low baseline hearing loss (< 10dB)

Lower than 10 dB baseline hearing loss was compared to higher than 10 dB baseline hearing loss in two studies (32, 36). They both show that a higher baseline hearing loss significantly increased the hearing loss per year however the magnitude of the effect was ten-fold larger in Seixas than in Pell. The heterogeneity between studies was so big (I^2 almost 100%) that we refrained from combining studies in a meta-analysis.

Table 12 Effect of baseline hearing levels on noise induced hearing loss

Comparison	Outcome	No. of studies	No. of participants	Statistical method	Effect sizes Negative value indicates lower risk (better hearing)
Incremental increase in baseline hearing loss	dB/year	2	6483	Mean Difference (95% CI)	Heyer 2011-M (37): -0.03 (-0.04 to -0.02) Heyer 2011-F (37): -0.05 (0.09 to -0.01)
high (>10 dB) baseline hearing loss versus low (<10)	dB/year	2	2244	Mean Difference (95% CI)	Seixas 2005 (32): 12.4 (11.3 to 13.5) Positive value means worse hearing

dB)baseline hearing loss					Pell 1973 (36): -1.24 (-1.52 to -0.99) Negative value means worse hearing
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4 DISCUSSION

4.1 *Summary of main findings*

We found that higher age and previously being exposed to noise increased the risk of noise induced hearing loss based on multiple studies but it was difficult to draw conclusions about the magnitude of the risk increase. There was also evidence that multiple treatment for tuberculosis increased the rate of NIHL. Similarly, less use of hearing protection and no visits to an ear doctor increased the risk of noise induced hearing loss based on single studies. There was no clear evidence for the effect of having a darker skin, for the effect of gender or for the effect of baseline hearing loss even though there were multiple studies.

Most results were very heterogeneous which means that these results have to be interpreted with caution.

4.2 *Overall completeness and applicability of findings*

The range of the studies is wide in both the populations covered and the factors assessed. However for each assessed factor the number of studies providing data is limited with the maximum number of studies available for the age as a predictor.

Although all studies indicate that older age predicts more hearing loss per year in the future, it is impossible to separate noise-induced hearing loss from presbycusis. The rate of hearing loss increases in general with age and it is therefore difficult to attribute our findings solely to noise-induced hearing loss. A study would be needed that compares both young and old noise-exposed and non-exposed workers over time. It is also disturbing that the effect size was much bigger in Sbihi 2010 and in Seixas 2005. We could not explain the big magnitude of the effect in Sbihi 2010. The authors used log-transformed yearly changes in hearing loss as the dependent variable. Therefore, the results had to be transformed back to be meaningful on a dB hearing loss scale. For other comparisons the back transformation led to very similar effect-sizes with the one for age being the exception. Based on the results of the Sbihi 2010 study the risk of hearing loss increases with about 1 dB per year of age increase. This would mean that a 60-year old worker would have a 30dB extra risk of hearing loss compared to a 30 year old worker given the same exposure duration and intensity. This does not seem to be realistic and practitioners would have noticed these big differences already because they are so big. The other studies yielded a more realistic magnitude of age as a predictor of 0.04 to 0.08 dB per year age increase. This would give a 60 year old worker a 3.6 dB bigger hearing loss than a 30 year old with the same exposure time and intensity. Seixas 2005

found about 4 dB difference between workers over and under 30 year of age. It is however difficult to assess what the meaning of this amount of loss in practice is given that these are all young workers and the study suffered from a high drop out with a short follow up. However the follow up is prospective and is at the start of work careers of the participants, who are very similar across the compared groups, meaning there is minimal confounding. (32) A longer follow up would have shed more light on the relationship of age, exposure to noise and future hearing loss. We believe the predictive effect of age is best obtained with assessing per year increase in age as done in the Rabinowitz 2013 (35), Heyer 2011 (37) and Sbihi 2010 (33) studies and data driven cut off points for categorizing continuous factors should be avoided in the future.

The association of older age with more hearing loss is slightly in contradiction with the ISO standard on noise induced hearing loss that indicates an exponential relationship between duration of noise exposure and noise-induced hearing loss. Here, hearing loss is most accelerated in the first ten years and tends to level off with longer exposure duration. Even though duration of exposure is not similar with age, one would expect a strong correlation and thus a more accelerated hearing loss in younger people.

Being previously exposed to noise at work, increased the risk of hearing loss with about 1.25 dB. This is a small risk increase only but it might indicate that previous exposure makes workers more susceptible. This is also in contradiction with the ISO-1999 model, as discussed in above. Also exposure to non-occupational noise increased hearing loss and this is not unexpected. Both of these factors can be easily assessed and therefore help predict hearing loss due to noise in future. However more data are needed to be sure of the magnitude of the effect of these predictors. To attribute the risks to an exposure accurately, future studies should assess the various levels of exposure to these predictors because in the current evidence all exposure is compared to no exposure. The apparent protection afforded by a history of visit to an ear doctor can possibly be explained by conductive hearing losses as a reason to see the doctor and that then later act as a built-in hearing protector.

Disease and medication factors were assessed in only one included study on South African gold miners. (29) TB and anti-tuberculosis drugs were found to increase noise induced hearing loss. The addition of streptomycin, a known ototoxic drug, was seen to increase this loss further. Streptomycin is given in cases of recurrent TB or when resistance to standard treatment is seen. Thus it reflects longer or more severe disease states which can confound the results. Compared with non TB participants, both first episode and recurrent TB predict a higher hearing loss per year and this

disease and/or its treatment are important predictors of NIHL at least in miners. Future studies need to expand on these findings with other relevant worker populations at risk and for other diseases and medications.

Correct use of hearing protection assessed as a predictor of NIHL showed a differential effect in male and female workers. In males, better use of hearing protection devices led to a much bigger protective effect than in females. This can either be because the sample for females was much smaller as evidenced with wide confidence intervals which include the mean effect in males. It can also be because there was a lower noise exposure in females than in males. It could also be a problem with fitting of the hearing protection devices, as these are developed largely on males and therefore may not adequately fit females who usually have smaller skulls. Finally it could be that an actual difference in effect exists between men and women in terms of NIHL susceptibility because of the gender which confounds the findings with hearing protection use.

Results were inconsistent and non-significant for gender with some studies showing men to be at risk of greater hearing loss and other studies women. However, we noted that females comprised between 0 to 10% of the study samples. Studies did not explain their choice of sample or how they reached a certain sample size. It is possible that this small fraction is a true representation of females in industrial cohorts exposed to noise. We need more studies to evaluate the difference in NIHL between the genders.

Limited data were found for ethnicity. However, the results indicate that blacks are less at risk compared to whites, supporting the melanin protection hypothesis.⁽²⁸⁾ However, Chinese with dark eyes and hair, but fairer skin seems to be worse off compared to whites. For East Indians with darker skin, there was not a significant relation with hearing loss which seems to contradict the melanin hypothesis. This provides a next step for research, to not only confirm these findings in larger samples of non-whites (Chinese and east Indians were 1.5% of the total sample), but to also determine why this difference exists. It may be that not dark eyes or hair but darker skin only is a predictive factor. The study assessing blacks versus whites was deemed relatively free of confounding but was based on a small sample (N=89) in a textile cohort exposed to up to 107 dB(A).⁽²⁸⁾ It is also important to notice that no follow-up studies were found that evaluated the effect of eye-colour as an indicator of melanin status.

Higher baseline hearing thresholds predicted lower NIHL when per unit rise was assessed in the Heyer 2011 studies but an opposite effect was seen when studies compared less than 10 dB loss at baseline to more than 10 dB loss at baseline in Seixas 2005 and Pell 1973. Since baseline hearing

threshold is not a naturally dichotomous variable it is probably best to avoid data-driven single cut off categorization unless previous research indicates such a cut off to be of clinical relevance. The categorization should be a-priori at least, but ideally per unit rise in baseline hearing should be assessed as done in Heyer 2011.

Even though often mentioned in the literature, we found no follow-up studies that evaluated the predictive value of cardiovascular risk factors such as smoking, high blood pressure, or high cholesterol levels.

4.3 *Quality of evidence*

Overall the quality of evidence is low to moderate. The large number of excluded studies based on the lack of follow-up design indicates that prediction of hearing loss is not a topic that is studied according to the state of the art. Because data are easily available, it is also easy to publish cross-sectional studies. However, as indicated before we don't think that these study designs provide valid data for prediction. Only two studies were considered at overall low risk and three at moderate risk while the majority (seven) were at high risk of bias. The most often at risk domains were study attrition, and study analysis and reporting. Reporting bias can often be rectified with author contacts. However, here in most cases author contact could not be established mainly because of the age of the study as many authors have retired. Study attrition is an important issue in large epidemiological studies. Particularly in database linkage studies, where the number of initially identified eligible people and those used in the final analysis were not reported and may have been different. Such studies give an illusion of no loss to follow-up or no missing data when in fact neither is known. Outcome measurement and predictive factor measurement domains were usually at low risk of bias as these were measured and reported satisfactorily in the majority of the studies. This increases our confidence in these measurements and their reported relationship being valid.

In summary the results of the two low risk studies Heyer 2011 M/F, are considered more reliable and those that are moderate and high risk studies should be used with caution in relevant situations.

4.4 *Potential biases in the review process*

We published a protocol before conducting the final review which prevents data-driven findings. There were no deviations from the protocol. We did not exclude studies based on language, but were not able to get them all translated. However, we believe that this has caused only little bias, if any.

The criteria for overall risk of bias were developed after the studies were included and may have been affected by the information from the studies. However, four team members contributed to these criteria when only two were aware of all included studies.

4.5 *Agreements and disagreements with other reviews*

The main difference between this review and previous ones (23, 24) is that we followed PRISMA and the Cochrane methods in conduct of the review. This allowed us to come to clear conclusions on not only the predictive factors and their effects but more importantly also on the quality (reliability) of the evidence available.

4.6 Implications for practice

The practice implications are limited by the limited size and quality of the data available. However, there is evidence that increasing age and previous exposure to occupational or non-occupational noise are factors that can increase occupational NIHL. More specifically, this means that older workers should be targeted for prevention of hearing loss. The finding that tuberculosis and its treatment increase the risk of hearing loss should be a further incentive to prevent tuberculosis in workers. Correct use of hearing protection devices is a factor that appears to protect against occupational NIHL. The results for skin colour and gender are contradictory and do not seem directly useful for prediction of hearing loss. There were no studies on cardiovascular risk factors or smoking.

4.7 Implications for future research

New and better epidemiological studies are needed to confirm the findings of this review and provide reliable information on the factors potentially related to NIHL. A wider range of worker populations must be included and the variation in noise exposure assessed within each study. If data base linkage studies are conducted these should consider what sample size for each factor level is needed for reliable differences to be found. Better reporting of study methods and loss to follow up is also indicated. Understudied factors are eye-colour and cardiovascular risk factors such as smoking or high blood pressure. The most optimal step for future research would be a study to model all important factors using a large set of data following the current recommendations for prognostic studies.

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Appendix A: Search Strategies

Search strategy occupational NIHL prediction 5-06-13

Search Medline

#7	Add	Search (Human[MeSH Terms]) AND ((((((work[tw] OR works*[tw] OR work*[tw] OR worka*[tw] OR worke*[tw] OR workg*[tw] OR worki*[tw] OR workl*[tw] OR workp*[tw] OR occupation*[tw] OR military)))))) AND noise) AND (((("hearing loss" OR "hearing acuity" OR "hearing deficiency" OR "hearing level" OR "hearing threshold" OR "hearing assessment"))) OR nihl))	<u>3188</u>	08:38:06
#6	Add	Search ((((((work[tw] OR works*[tw] OR work*[tw] OR worka*[tw] OR worke*[tw] OR workg*[tw] OR worki*[tw] OR workl*[tw] OR workp*[tw] OR occupation*[tw] OR military)))))) AND noise) AND (((("hearing loss" OR "hearing acuity" OR "hearing deficiency" OR "hearing level" OR "hearing threshold" OR "hearing assessment"))) OR nihl))	<u>3570</u>	08:37:27
#5	Add	Search (((("hearing loss" OR "hearing acuity" OR "hearing deficiency" OR "hearing level" OR "hearing threshold" OR "hearing assessment"))) OR nihl	<u>43851</u>	08:37:21
#4	Add	Search nihl	<u>399</u>	08:37:03
#3	Add	Search (("hearing loss" OR "hearing acuity" OR "hearing deficiency" OR "hearing level" OR "hearing threshold" OR "hearing assessment")	<u>43829</u>	08:36:51
#2	Add	Search noise	<u>90045</u>	08:36:18
#1	Add	Search ((work[tw] OR works*[tw] OR work*[tw] OR worka*[tw] OR worke*[tw] OR workg*[tw] OR worki*[tw] OR workl*[tw] OR workp*[tw] OR occupation*[tw] OR military))	<u>1128416</u>	08:36:0

Search Central

ID	Search	Hits
#1	MeSH descriptor: [Work] explode all trees	257
#2	work* or occupation*:ti,ab,kw (Word variations have been searched)	25581
#3	MeSH descriptor: [Military Personnel] explode all trees	570
#4	military or army:ti,ab,kw (Word variations have been searched)	1380
#5	OR #1-#4	26807

#6	MeSH descriptor: [Noise] explode all trees	284
#7	MeSH descriptor: [Noise, Occupational] explode all trees	35
#8	noise* (Word variations have been searched)	1629
#9	OR #6-#8	1629
#10	"hearing loss" or "hearing acuity" or "hearing deficiency" or "hearing level" or "hearing threshold" or "hearing assessment" (Word variations have been searched)	1449
#11	"NIHL" (Word variations have been searched)	11
#12	MeSH descriptor: [Hearing Loss, Noise-Induced] explode all trees	66
#13	OR #11 - #12	70
#14	#10 OR #13	1451
#15	#5 and #9 and #13	34
#16	#15 in Trials	31

Search Embase

No	Query	Results
#10	(work* OR occupation* OR military) AND ('noise'/exp OR noise) AND ('hearing loss' OR 'hearing acuity' OR 'hearing deficiency' OR 'hearing level' OR 'hearing threshold' OR 'hearing assessment' OR nihl OR 'noise-induced hearing loss') AND 'human'/de AND [embase]/lim	1,312
#9	(work* OR occupation* OR military) AND ('noise'/exp OR noise) AND ('hearing loss' OR 'hearing acuity' OR 'hearing deficiency' OR 'hearing level' OR 'hearing threshold' OR 'hearing assessment' OR nihl OR 'noise-induced hearing loss') AND 'human'/de	2,112

#8	(work* OR occupation* OR military) AND ('noise'/exp OR noise) AND ('hearing loss' OR 'hearing acuity' OR 'hearing deficiency' OR 'hearing level' OR 'hearing threshold' OR 'hearing assessment' OR nihl OR 'noise-induced hearing loss')	2,715
#7	('hearing loss' OR 'hearing acuity' OR 'hearing deficiency' OR 'hearing level' OR 'hearing threshold' OR 'hearing assessment') OR (nihl OR 'noise-induced hearing loss')	40,731
#6	nihl OR 'noise-induced hearing loss'	2,024
#5	'noise-induced hearing loss'	1,984
#4	nihl	467
#3	'hearing loss' OR 'hearing acuity' OR 'hearing deficiency' OR 'hearing level' OR 'hearing threshold' OR 'hearing assessment'	40,701
#2	'noise'/exp OR noise	105,577
#1	work* OR occupation* OR military	2,728,433

Appendix B: Table of excluded studies

Report ID	Reason for exclusion	References
Abbate, 2005	outcome change from baseline not reported	Abbate C, Concetto G, Fortunato M, Brecciaroli R, Tringali MA, Beninato G, et al. Influence of environmental factors on the evolution of industrial noise-induced hearing loss. <i>Environmental Monitoring and Assessment</i> . 2005;107(1-3):351-61.
Adera, 2000	no noise measurement reported	Adera T, Amir C, Anderson L. Time trends analysis of hearing loss: an alternative approach to evaluating hearing loss prevention programs. <i>AIHAJ</i> . 2000;61(2):161-5.
Agrawal, 2010	no follow up, noise measurement	Agrawal Y, Niparko JK, Dobie RA. Estimating the effect of occupational noise exposure on hearing thresholds: the importance of adjusting for confounding variables. <i>Ear Hear</i> . 2010;31(2):234-7.
Attias, 1994	follow up 2 months only	Attias J, Weisz G, Almog S, Shahar A, Wiener M, Joachims Z, et al. Oral magnesium intake reduces permanent hearing loss induced by noise exposure. <i>American journal of otolaryngology</i> . 1994;15:26-32.
Aycicek, 2009	no follow up, no change from baseline for outcome	Aycicek A, Sargın R, Kenar F, Derekoş FS. Can Rh antigens be a risk factor in noise-induced hearing loss? <i>European Archives of Oto-Rhino-Laryngology</i> . 2009;266(3):363-6.
Bauer, 1991	no follow up, no change from baseline for outcome	Bauer P, Körpert K, Neuberger M, Raber A, Schwetz F. Risk factors for hearing loss at different frequencies in a population of 47,388 noise-exposed workers. <i>Journal of the Acoustical Society of America</i> . 1991;90(6):3086-98.
Behar, 1984	methodology study, no empirical data	Behar A, Plener R. Noise exposure--sampling strategy and risk assessment. <i>Am Ind Hyg Assoc J</i> . 1984;45(2):105-9.
Benavides, 1997	no noise measurement	Benavides R. [Neurosensory hearing loss caused by noises: results of a longitudinal study in iron and steel workers]. <i>Rev Med Chil</i> . 1997;125(9):1026-31.
Berg, 2009	noise exposure levels not measured, history of work types obtained in questionnaires.	Berg RL, Pickett W, Fitz RM, Broste SK, Knobloch MJ, Wood DJ, et al. Hearing conservation program for agricultural students: short-term outcomes from a cluster-randomized trial with planned long-term follow-up. <i>Preventive medicine</i> . 2009;49:546-52.
Bergström, 1986	no predictive factors assessed	Bergström B, Nyström B. Development of hearing loss during long-term exposure to occupational noise: A 20-year follow-up study. <i>Scandinavian Audiology</i> . 1986;15(4):227-34.
Bohner, 2002	no follow up	Bohner BK, Page JC, Rovig G, Betts LS, Muller JG, Sack DM. U.S. Navy and Marine Corps Hearing Conservation Program, 1995-1999: mean hearing thresholds for enlisted personnel by gender and age groups. <i>Mil Med</i> . 2002;167(2):132-5.
Bohner, 2004	noise exposure levels not measured	Bohner BK, Page JC, Rovig GW, Betts LS, Sack DM. Navy Hearing Conservation Program: 1995-1999 retrospective analysis of threshold shifts for age, sex, and officer/enlisted status. <i>Mil Med</i> . 2004;169(1):73-6.
Carlin, 1980	no follow up, no noise measurement	Carlin MF, McCroskey RL. Is eye color a predictor of noise-induced hearing loss? <i>Ear Hear</i> . 1980;1(4):191-6.
Champagne, 2010	no follow up, outcome not of interest	Champagne M-P. Development of an audiometric technique to identify individuals' susceptibility to noise. US: ProQuest Information & Learning; 2010.

Chung 1982	cross sectional analysis	Chung, DY; Willson, GN; Gannon, RP; Mason, K.: Individual susceptibility to noise; in Hamernik, Henderson, Salvi, pp. 51 1-519 (Raven Press, New York 1982).
Chung, 2012	additional publication (letter from authors) clarifies cross sectional and not follow up data analysis	Chung IS, Chu IM, Cullen MR. Hearing effects from intermittent and continuous noise exposure in a study of Korean factory workers and firefighters. <i>BMC Public Health</i> . 2012;12:87.
Da Costa, 2008	no follow up, no noise measurement	Da Costa EA, Castro JC, Macedo ME. Iris pigmentation and susceptibility to noise-induced hearing loss. <i>Int J Audiol</i> . 2008;47(3):115-8.
Dacomo, 1994	no predictive factors	Dacomo G, Broich G, Iudica F, Campi M, Calabro F, La Salvia R, et al. [Epidemiologic longitudinal study of a population of workers of the state railroads exposed to noise]. <i>Clin Ter</i> . 1994;144(2):95-8.
Daniell, 2003	comparison of hearing measurement methods only; noise exposure levels not measured; no factors assessed	Daniell WE, Stover BD, Takaro TK. Comparison of criteria for significant threshold shift in workplace hearing conservation programs. <i>Journal of Occupational and Environmental Medicine</i> . 2003;45(3):295-304.
Dempsey, 1985	no follow up, survey, no factors	Dempsey JJ. 6000 Hz as an early indicator of noise-induced hearing loss. <i>Ear Hear</i> . 1985;6(3):159-60.
Dias, 2006	no follow up, no noise measurement	Dias A, Cordeiro R, Corrente JE, Goncalves CG. [Association between noise-induced hearing loss and tinnitus]. <i>Cad Saude Publica</i> . 2006;22(1):63-8.
Dobie, 2006	methodolgy study, no empirical data	Dobie RA. Methodological issues when comparing hearing thresholds of a group with population standards: the case of the ferry engineers. <i>Ear Hear</i> . 2006;27(5):526-37.
Dobie, 2007	no predictive factors	Dobie RA. Noise-induced permanent threshold shifts in the occupational noise and hearing survey: an explanation for elevated risk estimates. <i>Ear Hear</i> . 2007;28(4):580-91.
Dogru, 2003	no follow up, no noise measurement	Dogru H, Tüz M, Uygur K. Correlation between blood group and noise-induced hearing loss. <i>Acta Oto-Laryngologica</i> . 2003;123(8):941-2.
Du, 2004	review on noise leads to HL- chinese translated	Du B, Wang XR. [Risk factors of occupational hearing loss]. <i>Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi</i> . 2004;22(2):150-2.
Fabry, 2011	no follow up, no noise measurement	Fabry DA, Davila EP, Arheart KL, Serdar B, Dietz NA, Bandiera FC, et al. Secondhand smoke exposure and the risk of hearing loss. <i>Tobacco Control: An International Journal</i> . 2011;20(1):82-5.
Fransen, 2008	no follow up, no noise measurement, no predictive factors	Fransen E, Topsakal V, Hendrickx JJ, Van Laer L, Huyghe JR, Van Eyken E, et al. Occupational noise, smoking, and a high body mass index are risk factors for age-related hearing impairment and moderate alcohol consumption is protective: a European population-based multicenter study. <i>J Assoc Res Otolaryngol</i> . 2008;9(3):264-76; discussion 1-3.
Fritze, 1981	no noise measurement	Fritze W. [A method for prediction of permanent threshold shift (PTS) (author's transl)]. <i>Laryngol Rhinol Otol (Stuttg)</i> . 1981;60(10):512-6.

		Fritze W. [An attempt to predict the noise-induced PTS. Beginning a long-time study (author's transl)]. <i>Laryngol Rhinol Otol (Stuttg)</i> . 1978;57(12):1049-52.
Fritze, 1975	no follow up	Fritze W. [The prognosis of the noise-induced permanent hearing-loss (author's transl)]. <i>Laryngol Rhinol Otol (Stuttg)</i> . 1975;54(6):485-9.
Gamez Alguacil, 1990	no frequencies reported for HL measurement	Gamez Alguacil I, Herrera Casado A, Donamayor Hernandez C, Llorente de Lara JM, Lopez Castanier MY. [The time factor in hypoacusis caused by acoustic trauma]. <i>Acta Otorrinolaringol Esp</i> . 1990;41(1):19-23.
Ghazizadeh, 2012	no follow up	Ghazizadeh AH, Bakhshaei M, Mahdavi E, Movahhed R. Hair color and hearing loss: A survey in a group of military men. <i>Iranian Journal of Otorhinolaryngology</i> . 2012;24(69):155-60.
Guest, 2011	chemical exposure	Guest M, Boggess M, Attia J. Relative risk of elevated hearing threshold compared to ISO1999 normative populations for Royal Australian Air Force male personnel. <i>Hearing Research</i> . 2012;285(1-2):65-76.
		Guest M, Boggess M, D'Este C, Attia J, Brown A. An observed relationship between vestibular function and auditory thresholds in aircraft-maintenance workers. <i>Journal of Occupational and Environmental Medicine</i> . 2011;53(2):146-52.
Helfer, 2010	no noise measurement, no follow up indicated, no defined NIHL	Helfer TM, Canham-Chervak M, Canada S, Mitchener TA. Epidemiology of hearing impairment and noise-induced hearing injury among U.S. military personnel, 2003-2005. <i>Am J Prev Med</i> . 2010;38(1 Suppl):S71-7.
Helleman, 2012	no predictive factors	Helleman HW, Dreschler WA. Overall versus individual changes for otoacoustic emissions and audiometry in a noise-exposed cohort. <i>Int J Audiol</i> . 2012;51(5):362-72.
Hepler, 1984	review	Hepler EL, Moul MJ, Gerhardt KJ. Susceptibility to noise-induced hearing loss: Review and future directions. <i>Military Medicine</i> . 1984;149(3):154-8.
Humes, 1991	methodology study, no empirical data	Humes LE, Jesteadt W. Modeling the interactions between noise exposure and other variables. <i>J Acoust Soc Am</i> . 1991;90(1):182-8.
Humes, 1977	review	Humes LE. Review of four new indices of susceptibility to noise-induced hearing loss. <i>J Occup Med</i> . 1977;19(2):116-8.
Ickes, 1982	unclear design, no empirical data, likely method study	Ickes WK, Nader C. Noise-induced hearing loss and stress-prone behavior. <i>Ear Hear</i> . 1982;3(4):191-5.
Ide, 2011	no predictive factors, no noise measurement	Ide CW. Hearing losses in wholetime firefighters occurring early in their careers. <i>Occupational Medicine</i> . 2011;61(7):509-11.
Iki, 1986	no noise measurement, no change from baseline for NIHL	Iki M, Kurumatani N, Hirata K, Moriyama T, Satoh M, Arai T. Association between vibration-induced white finger and hearing loss in forestry workers. <i>Scand J Work Environ Health</i> . 1986;12(4 Spec No):365-70.
	letter to editor on unpublished iki 1986 study	Iki M, Kurumatani N, Moriyama T. Vibration-induced white fingers and hearing loss. <i>Lancet</i> . 1983;2(8344):282-3.

Iki, 1994	no follow up, no noise measurement, outcome is postural stability -not NIHL	Iki M. Vibration-induced white finger as a risk factor for hearing loss and postural instability. Nagoya J Med Sci. 1994;57 Suppl:137-45.
Ishii, 1992	no follow up, no change from baseline on NIHL	Ishii EK, Talbott EO, Findlay RC, D'Antonio JC, Kuller LH. Is NIDDM a risk factor for noise-induced hearing loss in an occupationally noise exposed cohort? Science of the Total Environment. 1992;127(1-2):155-65.
Ishii, 1998	no follow up, no change from baseline on NIHL	Ishii EK, Talbott EO. Race/ethnicity differences in the prevalence of noise-induced hearing loss in a group of metal fabricating workers. J Occup Environ Med. 1998;40(8):661-6.
Jang, 2011	predictive factor (fasting glucose) no change from baseline-BL not reported	Jang TW, Kim BG, Kwon YJ, Im HJ. The association between impaired fasting glucose and noise-induced hearing loss. J Occup Health. 2011;53(4):274-9.
Jerger, 1986	no follow up, no change from baseline	Jerger J, Jerger S, Pepe P, Miller R. Race difference in susceptibility to noise-induced hearing loss. Am J Otol. 1986;7(6):425-9.
Joachims, 1993	2 month follow up	Joachims Z, Netzer A, Ising H, Rebentisch E, Attias J, Weisz G, et al. Oral magnesium supplementation as prophylaxis for noise-induced hearing loss: results of a double blind field study. Schriftenr Ver Wasser Boden Lufthyg. 1993;88:503-16.
Johnson, 1991	review	Johnson DL. Field studies: industrial exposures. J Acoust Soc Am. 1991;90(1):170-4.
Kahari, 2001	no noise measurement	Kahari KR, Axelsson A, Hellstrom PA, Zachau G. Hearing development in classical orchestral musicians. A follow-up study. Scandinavian Audiology. 2001;30(3):141-9.
		Kähäri KR, Axelsson A, Hellström P, Zachau G. Hearing development in classical orchestral musicians. A follow-up study. Scandinavian Audiology. 2001;30(3):141-9.
Kamal, 1989	no factors measured at baseline	Kamal AA, Mikael RA, Faris R. Follow-up of hearing thresholds among forge hammering workers. Am J Ind Med. 1989;16(6):645-58.
Karlovich, 1992	no predictive factors	Karlovich RS. Research project shows importance of pre-employment hearing testing. Occup Health Saf. 1992;61(2):38-42.
Karlsson, 1983	no noise measurement, no change from baseline on NIHL	Karlsson K, Lundquist PG, Olausson T. The hearing of symphony orchestra musicians. Scand Audiol. 1983;12(4):257-64.
Kleinstejn, 1984	no follow up, no change from baseline	Kleinstejn RN, Seitz MR, Barton TE, Smith CR. Iris color and hearing loss. Am J Optom Physiol Opt. 1984;61(3):145-9.
Kraus, 2001	case report	Kraus S, Weidner W. Prolonged exposure to extracorporeal shock wave lithotripsy and noise induced hearing damage. J Urol. 2001;165(6 Pt 1):1984.
Kuronen, 2004	no follow up	Kuronen P, Toppila E, Starck J, Pääkkönen R, Sorri MJ. Modelling the risk of noise-induced hearing loss among military pilot. International Journal of Audiology. 2004;43(2):79-84.
Leensen, 2011	no follow up, no factors	Leensen MC, van Duivenbooden JC, Dreschler WA. A retrospective analysis of noise-induced hearing loss in the Dutch construction industry. Int Arch Occup Environ Health. 2011;84(5):577-90.

Lehto, 1989	no noise measurement	Lehto TU, Laurikainen ET, Aitasalo KJ, Pietila TJ, Helenius HY, Johanson R. Hearing of dentists in the long run: a 15-year follow-up study. <i>Community Dent Oral Epidemiol.</i> 1989;17(4):207-11.
Lutman, 1992	no empirical data, likely a methods study	Lutman ME. Apportionment of noise-induced hearing disability and its prognosis in a medicolegal context: a modelling study. <i>Br J Audiol.</i> 1992;26(5):307-19.
Maccacaro, 2007	no predictive factors	Maccacaro G, Baratieri S, Princivalle A, Perbellini L. [Evidence Based Occupational Medicine: ten year experience with audiometric examination in a handicraft company]. <i>G Ital Med Lav Ergon.</i> 2007;29(3 Suppl):241-3.
MacLurg, 2004	no follow up, no predictive factors, no noise measurement	MacLurg K, McCaughan J, McQuillan P. Hearing Surveillance Chart--a tool for tracking serial audiometry results and predicting future hearing impairment. <i>Occup Med (Lond).</i> 2004;54(8):583-4.
Majzel 1981	No follow up, no control group	Majzel K, Gierek T. [Assessment of the value of temporal shift of hearing threshold in extended range of high frequencies depending on the number of years of occupational exposure to noise (author's transl)]. <i>Otolaryngol Pol.</i> 1981;35(4-6):359-64.
Mahendra Prashanth, 2011	review	Mahendra Prashanth KV, Venugopalachar S. The possible influence of noise frequency components on the health of exposed industrial workers--a review. <i>Noise Health.</i> 2011;13(50):16-25.
Malchaire, 1997	no empirical data, method study	Malchaire J, Piette A. A comprehensive strategy for the assessment of noise exposure and risk of hearing impairment. <i>Ann Occup Hyg.</i> 1997;41(4):467-84.
Malchaire, 1987	review	Malchaire J. Evaluation of the individual risk of hearing loss: Prospective study. <i>International Archives of Occupational and Environmental Health.</i> 1987;59(4):355-62.
Malchaire, 1979	no follow up,	Malchaire JB, Mullier M. Occupational exposure to noise and hypertension: a retrospective study. <i>Ann Occup Hyg.</i> 1979;22(1):63-6.
Manninen, 1979	no follow up,	Manninen O, Aro S. Noise-induced hearing loss and blood pressure. <i>Int Arch Occup Environ Health.</i> 1979;42(3-4):251-6.
Marlenga, 2012	no noise measurement	Marlenga B, Berg RL, Linneman JG, Wood DJ, Kirkhorn SR, Pickett W. Determinants of early-stage hearing loss among a cohort of young workers with 16-year follow-up. <i>Occupational and Environmental Medicine.</i> 2012;69(7):479-84.
Mcllwain, 2009	no follow up, no noise measurement, no predictive factors	Mcllwain S, Sisk B, Hill M. Cohort case studies on acoustic trauma in Operation Iraqi Freedom. <i>US Army Med Dep J.</i> 2009:14-23.
Meinke, 2007	review	Meinke DK, Stephenson MR. Noise-induced hearing loss: Models for prevention. In: Ackley RS, Decker TN, Limb CJ, editors. <i>An essential guide to hearing and balance disorders.</i> Mahwah, NJ US: Lawrence Erlbaum Associates Publishers; 2007. p. 287-323.
Meyer-Falcke, 1993	no follow up	Meyer-Falcke A, Lanzendorfer A, Jansen G. Predictors for noise sensitivity: how to use them for a prognostic test. <i>Schriftenr Ver Wasser Boden Lufthyg.</i> 1993;88:223-37.

Miller, 2004	no noise measurement	Miller JAL, Marshall L, Heller LM. A longitudinal study of changes in evoked otoacoustic emissions and pure-tone thresholds as measured in a hearing conservation program. <i>International Journal of Audiology</i> . 2004;43(6):307-22.
Mohammadi, 2010	no follow up, no noise measurement	Mohammadi S, Mazhari MM, Mehrparvar AH, Attarchi MS. Effect of simultaneous exposure to occupational noise and cigarette smoke on binaural hearing impairment. <i>Noise Health</i> . 2010;12(48):187-90.
Morata, 1995	review	Morata TC, Lemasters GK. Epidemiologic considerations in the evaluation of occupational hearing loss. <i>Occup Med</i> . 1995;10(3):641-56.
Moselhi, 1979	no change from baseline	Moselhi M, El-Sadik YM, El-Dakhkhny A. A six-year follow up study for evaluation of the 85 dB(A) safe criterion for noise exposure. <i>Am Ind Hyg Assoc J</i> . 1979;40(5):424-6.
Mrena, 2009	no follow up, no noise measurement, no NIHL measure	Mrena R, Savolainen S, Kiukaanniemi H, Ylikoski J, Makitie AA. The effect of tightened hearing protection regulations on military noise-induced tinnitus. <i>Int J Audiol</i> . 2009;48(6):394-400.
Muhr, 2011	no noise measurement	Muhr P, Rosenhall U. The influence of military service on auditory health and the efficacy of a Hearing Conservation Program. <i>Noise Health</i> . 2011;13(53):320-7.
Neitzel, 2011	no NIHL measurement	Neitzel RL, Stover B, Seixas NS. Longitudinal assessment of noise exposure in a cohort of construction workers. <i>Ann Occup Hyg</i> . 2011;55(8):906-16.
Paul, 1987	no original data-model of noise induced hearing loss	Paul I. [Individual hearing sensitivity and risk of damage caused by the effect of noise]. <i>Z Gesamte Hyg</i> . 1987;33(5):244-6.
Pawlaczyk-Luszczynska 2010	No follow up, no control group	Pawlaczyk-Luszczynska M, Dudarewicz A, Zamojska M, Sliwinska-Kowalska M. [Risk assessment of hearing loss in orchestral musicians]. <i>Med Pr</i> . 2010;61(5):493-511.
Perłowski 1967	No follow up, no control group	Perłowski H, Szczepanski J. [Attempt of evaluation of the relation of hearing loss to the degree of pneumatization of the mastoid processes (in professional drivers)]. <i>Pol Tyg Lek</i> . 1967;22(36):1372-3.
Pettersson, 2012	no NIHL defined or measured(just calling it impaired hearing)	Pettersson H, Burström L, Hagberg M, Lundström R, Nilsson T. Noise and hand-arm vibration exposure in relation to the risk of hearing loss. <i>Noise Health</i> . 2012;14(59):159-65.
Pyykko, 1981	no follow up	Pyykko I, Starck J, Farkkila M, Hoikkala M, Korhonen O, Nurminen M. Hand-arm vibration in the aetiology of hearing loss in lumberjacks. <i>Br J Ind Med</i> . 1981;38(3):281-9.
Pyykko, 1986	no follow up	Pyykko I, Starck J, Pekkarinen J. Further evidence of a relation between noise-induced permanent threshold shift and vibration-induced digital vasospasms. <i>Am J Otolaryngol</i> . 1986;7(6):391-8.
Pyykkö, 1982	no follow up	Pyykkö I, Starck J. Vibration syndrome in the etiology of occupational hearing loss. <i>Acta Oto-Laryngologica</i> . 1982;SUPPL 386:296-300.
Pyykkö, 2007	no follow up	Pyykkö I, Toppila E, Zou J, Kentala E. Individual susceptibility to noise-induced hearing loss. <i>Audiological Medicine</i> . 2007;5(1):41-53.
Pyykkö, 2000	no follow up	Pyykkö IV, Toppila EM, Starck JP, Juhola M, Auramo Y. Database for a hearing conservation program. <i>Scandinavian Audiology</i> . 2000;29(1):52-8.

Robinson, 1985	No follow up or predictive factors	Robinson DW. The audiogram in hearing loss due to noise: a probability test to uncover other causation. <i>Ann Occup Hyg.</i> 1985;29(4):477-93.
Rosler, 1994	review	Rosler G. Progression of hearing loss caused by occupational noise. <i>Scandinavian Audiology.</i> 1994;23(1):13-37.
Siegelaub, 1974	no follow up	Siegelaub AB, Friedman GD, Adour K, Seltzer CC. Hearing loss in adults: relation to age, sex, exposure to loud noise, and cigarette smoking. <i>Arch Environ Health.</i> 1974;29(2):107-9.
Sokas, 1995	no follow up, no noise measurement	Sokas RK, Moussa MA, Gomes J, Anderson JA, Achuthan KK, Thain AB, et al. Noise-induced hearing loss, nationality, and blood pressure. <i>Am J Ind Med.</i> 1995;28(2):281-8.
Solecki 2004	Outcome is not hearing loss but exposure to noise	Solecki L. [Assessment of annual exposure to noise and risk of occupational hearing loss among private farmers specializing in plant production]. <i>Medycyna pracy.</i> 2004;55(2):175-82.
Sulkowski 1984	No follow up, no control group	Sulkowski W, Kowalska S, Lipowczan A. [A permanent noise-induced shift in the auditory threshold in textile industry workers]. <i>Med Pr.</i> 1986;37(3):175-86.
Szanto, 1990	no predictive factor; additional exposure to vibration	Szanto C, Ionescu M. [Hearing loss of workers in mining--a 6-year longitudinal study]. <i>Z Gesamte Hyg.</i> 1990;36(1):44-7.
Tambs, 2006	no noise measurement	Tambs K, Hoffman HJ, Borchgrevink HM, Holmen J, Engdahl B. Hearing loss induced by occupational and impulse noise: results on threshold shifts by frequencies, age and gender from the Nord-Trøndelag Hearing Loss Study. <i>International Journal of Audiology.</i> 2006;45(5):309-17.
Tambs, 2003	no noise measurement	Tambs K, Hoffman HJ, Borchgrevink HM, Holmen J, Samuelsen SO. Hearing loss induced by noise, ear infections, and head injuries: results from the Nord-Trøndelag Hearing Loss Study. <i>International Journal of Audiology.</i> 2003;42(2):89-105.
Tao, 2013	no follow up, no change from baseline on NIHL	Tao L, Davis R, Heyer N, Yang Q, Qiu W, Zhu L, et al. Effect of cigarette smoking on noise-induced hearing loss in workers exposed to occupational noise in China. <i>Noise Health.</i> 2013;15(62):67-72.
Toppila, 2001	no follow up	Toppila E, Pyykko I, Starck J. Age and noise-induced hearing loss. <i>Scandinavian Audiology.</i> 2001;30(4):236-44.
Wallhagen, 1997	no noise measurement, no NIHL measured	Wallhagen MI, Strawbridge WJ, Cohen RD, Kaplan GA. An increasing prevalence of hearing impairment and associated risk factors over three decades of the Alameda County Study. <i>Am J Public Health.</i> 1997;87(3):440-2.
Ward, 1965	review	Ward WD. The concept of susceptibility to hearing loss. <i>J Occup Med.</i> 1965;7(12):595-607.
Welleschik, 1980	no follow up, no change from baseline	Welleschik B, Korpert K. [Is the risk of noise-induced hearing damage higher for men than for women? (author's transl)]. <i>Laryngol Rhinol Otol (Stuttg).</i> 1980;59(10):681-9.
Willson, 1979	no change from baseline, no noise measurement,	Willson GN, Chung DY, Gannon RP, Roberts M, Mason K. Is a healthier person less susceptible to noise-induced hearing loss? <i>J Occup Med.</i> 1979;21(9):627-30.

Wu, 2009	no predictive factors, no noise measurement, no frequencies of NIHL	Wu CC, Young YH. Ten-year longitudinal study of the effect of impulse noise exposure from gunshot on inner ear function. <i>Int J Audiol.</i> 2009;48(9):655-60.
Wu, 1998	no follow up	Wu T, Liou S, Shen C, Hsu C, Chao S, Wang J, et al. Surveillance of noise-induced hearing loss in Taiwan, ROC: a report of the PRESS-NIHL results. <i>Preventive Medicine.</i> 1998;27(1):65-9.
Yano, 1981	no age adjustment	Yano E, Kaneko T, Koizumi A. A trial prognostic evaluation of occupational hearing loss by homogeneous absorbing Markov chains. <i>Sangyo Igaku.</i> 1981;23(1):84-5.
Zhao, 2010	no predictive factors	Zhao YM, Qiu W, Zeng L, Chen SS, Cheng XR, Davis RI, et al. Application of the kurtosis statistic to the evaluation of the risk of hearing loss in workers exposed to high-level complex noise. <i>Ear Hear.</i> 2010;31(4):527-32.
Zhao, 2004	no follow up, outcome is not NIHL- chinese translated	Zhao YM, Wang LZ. [Effects of hearing susceptibility for noise induced hypertension in fertilizer manufacture workers]. <i>Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi.</i> 2004;22(2):128-30.

Appendix C: Analysis and effects of predictive factors

Study ID	Analysis method presented	Results (most adjusted- chosen for analysis)	Effect estimates/what they mean MD (factor vs no factor) RR (factor vs no factor)
Brits 2012 (29)	ANCOVA and pairwise comparisons	Predictive factor level: mean (SD)change from BL in dB (negative values mean worse hearing) 2-average of 3,4,6 khz in left ear (chosen for analysis) no TB: -3.1 (10.2)= TB single Rx: -7.6 (12.1) TB multiple Rx (streptomycin): -9(13.9)	MD (TB simple vs No TB) 6 years =-7.6 (12.1) minus -3.1(10.2) = -4.50 (-5.54, -3.46) MD (TB Multiple vs no TB) 6 years= -9 (13.9) minus -3.1(10.2) = -5.90 (-7.47, -4.33) MD (TB multiple vs TB simple: -9 (13.9) minus=-7.6 (12.1)= -1.40 (-3.01, 0.21) MD of change in dB/yr= MD 6 yr/6 TB simple vs no TB dB /yr= -0.75 (-0.91 to 0.42) TB multiple vs no TB dB /yr= -1.05 (-1.25 to -0.93) Tb multiple vs TB simple dB/yr= -0.23 (-0.5 to 0.04)
Erlands-son 1983 (39)	A test applied to test significance of hearing loss in the group	Results do not show a relation between hearing loss over time (corrected for age) and age groups. Compared left ear to right in fig 4 Fig 7 data from graph- mean change from baseline- NIPTS mean(SD) low exp/younger group (5-15 yrs noise) =3.6 (6.67) n=15 high exp/ older group (20-40 yrs noise) =31.7 (11.48) n=14	fig 7 is showing exposure duration/employment duration under 15 yrs and over 20 years (from the graph), calling these young versus old. The young have a better hearing. MD 95%CI NIPTS young vs old= -28.10 (-35.00, -21.20) Negative value means better hearing. If we do the opposite old vs young then the old have a worse hearing MD 95%CI= 28.10 (21.20, 35.00)
Franks 1989 (38)	MANOVA. Descriptive stats presented in graphs	Gender: Of the noise exposed employees who showed shift (sts+) 16.4% were males and 11.3% were females. Of the non-exposed employees who showed shift, 17% were males and 9.5% were females. noise level: no difference in noise exposure between shift + and - noise duration.	More males than females were seen with STS change indicating a higher chance of worsened hearing average over 2-4 khz when one is male: RR(95%CI) 16.4% vs 11.3 %= 1.45 (0.71, 2.98)
Heyer 2011-F (37)	Generalized estimating equations (GEEs), were used to fit all models (Proc GENMOD procedure in SAS v 9.1).	Beta's from GEE indicating more (+)or less (-) mean hearing loss(dB) with every increase in noise or Age at 3,4,6 khz Plant 2: 0.04 (-0.62 -0.37), Plant 3:-0.62 (-1.00, -0.24) Baseline threshold -0.03 -0.04, -0.02 (yearly change) Age 0.08 0.07, 0.10 Noise duration (yrs) < 95 dB(A)/<=6 yrs: 0.77 (0.71, 0.82)	As negative coefficients indicate a protective effect, subjects with higher baseline hearing loss tend to experience less NIHL.

		<p>>=95 dB(A)/<= 6 yrs: 1.04 (0.88, 1.20) <95 dB(A)/>6 yrs: 0.79 (0.72, 0.86) >=95 dB(A)/>6 yrs: 0.69 (0.43, 0.95) Better HPD use -0.14 -0.27, -0.01</p>	
Heyer-M (37)	Generalised estimating equations (gees), were used to fit all models (Proc GENMOD procedure in SAS v 9.1).	<p>Beta's from GEE indicating more (+)or less (-) mean hearing loss(dB) with every increase in noise or Age at 3,4,6 khz Plant 2:1.27 (0.24, 1.76) Plant 3:1.00 Baseline threshold -0.05 0.09, -0.01 Age 0.08 0.05, 0.11 Noise duration (yrs) < 90 dB(A):0.39 (0.46, 0.73) Better HPD use -0.31 (-0.37, -0.24)</p>	As negative coefficients indicate a protective effect, subjects with higher baseline hearing loss tend to experience less NIHL.
Pell 1973 (36)	Frequency distributions in tables and graphs	<p>Predictive factor: <u>BLHL yes >40 dB start vs BLHL no (less than 10 dB)</u></p> <p>Mean (sd) N (BLHL no) low noise 5.5 (9.4) 435 middle noise 5 (10.4) 152 high noise 4.5 (10.2) 193 Mean (sd) N (BLHL yes) low noise -3.1 (16.3) 304 middle noise 0.7 (10.4) 108 high noise 0.4 (10.7)81</p>	<p>MD diff (in Mean hearing level dB at 4 khz in right ear over 5 years + 95% CI for 40+ dB BLHL vs less than 10 dB BLHL) noise low: -1.7 (-2.1to -1.4) noise middle: -0.9 (-1.37 to -0,35) noise high: -0.98 (-1.52 to -0.44) All noise yes BLHL vs no BLHL 5 yr: -6.22 (-7.60, -4.84)</p> <p>dB/yr: -1.24 (-1.52 to - 0.99)</p> <p>Negative means worse hearing</p> <p>Reference group: less than 10 dB at 4 khz</p>
Royster 1984 (28)	Tabulation of means and standard deviations	<p>Predictive factor: dB/year before and after for each group (Mean HTL (SD) at first and 4th annual test at 4 khz per year) White males before mean: 21 (15), white males after mean: 24.1(17.5) black males before mean: 17.1 (10.3) black males after mean: 17.3 (10.9) White cfbI= 3.1 (2.5) Black cfbI=0.2 ()</p>	<p>Per year mean difference between white and black (95%CI) white over 4 yrs: 2.90 (2.21, 3.59) (in revman) per year: 0.73 (-1.11 to 6.91) . REFERENCE GRP: black Negative value mean worsened hearing four year results divided by 4 based on assumed r = 0.80</p>
Sataloff 1966 (34)	Tabulation of mean threshold changes in the subjects over various frequencies	<p>No quantitative analysis: 2 out of 14 had TTS greater than 10 but no PTS at 3 kHz 5/14 PTS > 10 dB but no TTS at 8 kHz 3/14 PTS > 10 dB but no TTS at 0.5 kHz 1/14 TTS > 10 dB but no PTS at 1 kHz 2/14 TTS > 10 dB but no PTS at 2 kHz 2/14 TTS > 10 dB but no PTS at 3 kHz 3/14 TTS > 10 dB but no PTS at 4 kHz 2/14 TTS > 10 dB but no PTS</p>	RR not possible as data not enough to compare effect of TTS vs no TTS (the predictive factor)

<p>Sbihi 2010 (33)</p>	<p>Predictive models estimated by using xtreg command in STATA and maximum likelihood estimation, using variables generated by manual backward stepwise regression</p>	<p>Predictive factor level: log transformed outcomes (beta for change in mean hearing level as average of both ears 0.5 1 2 and 4 kHz) Noise exposure in dB(A)*year: 0.004 (0.001, 0.008) Female: 0.28 (-0.94, 1,15) Ethnicity Chinese: 0.24(0.11, 0.36) East Indian: -0.18 (-0.4, 0.1) Age (yrs): 0.05 (0.05, 0.06) Firearm (yes): 0.1 (0.03, 0.18) Exposed to blast (yes):0.5 (0.4, 0.6) Exposed to noisy previous job (yes): 0.23 (0.1- 0.3) Ear doctor visit (yes): -0.11 (-0.19, -0.03)</p>	<p>Communication from author A positive value in the table is worse off, and a negative value is better off.</p>
<p>Seizas 2005 (32)</p>	<p>Longitudinal analyses of changes in hearing level using mixed effects modelling</p>	<p>Predictive factor: MD diff (95% CI) mean change in hearing level at 4 kHz annually : Reference group prev occ noise: 1.25 (-0.83 to 3.33): no prev occ noise age > 30: 3.92 (1.94 to 5.9): < 30 baseline 1 vs 2 vs 3: 12.4 (11.3 to 13.5): <10 male: 1.26 (-1.23 to 3.75): female</p>	<p>A positive coefficient (beta) for the audiometry indicates worse hearing e.g. males have worse hearing than females and so on</p>
<p>Smith 1980 (31)</p>	<p>Stepwise regression analysis</p>	<p>Reg Coef Baseline audiogram= 0.1301 Reg Coef Noise level= 0.0114</p>	<p>Change from baseline in table III is all negative so I assume minus values mean worse hearing, but no clue what it means for the regression coefficients presented in table II.</p>

This systematic review of predictors of noise-induced hearing loss found 12 studies that followed hearing loss of workers exposed to noise over time. Older workers had a higher rate of hearing loss than younger workers with a similar level and duration of exposure. It was not clear if this effect could be fully attributed to the noise exposure. Also previous noise exposure led to a higher rate of hearing loss compared to no previous exposure. For other factors the evidence was inconsistent. At the moment, it is not possible to predict noise-induced hearing loss based on individual characteristics.

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