1. Introduction

Studies on occupational hearing loss have focused on noise as the primary cause. While the effect of this physical agent on hearing has been demonstrated, an analysis closer to the site of exposure confirms that the presence of other contaminants, such as chemicals, can interact with noise. This association may influence a temporal variability in the manifestation of an occupational hearing pathology.

In this respect, the term "working conditions" is too ambiguous (i.e., noise in the metal industry) as, in apparently similar conditions, several exposure environments can be identified: machining (noise+fluids, e.g. lathing), manufacture of structures (noise+fumes, e.g. welding) and surface protection (noise+solvents, e.g. painting), among others.

The European Agency for Safety and Health at Work recognises that noise-induced hearing loss is the most common occupational disorder in Europe. It advises that, in order to achieve greater efficiency in its prevention, more attention must be paid to the combined risk factors (multiple exposures) in workers exposed to high noise levels and chemical compounds associated with their work.

Similarly, recent studies conducted in the US (Agrawall et al., 2009) and New Zealand (Thorne et al., 2008) recognise noise-induced hearing loss as one of the most widespread occupational illnesses in these countries. Conclude that traditional noise monitoring and control methods have not achieved the expected results, identifying increasing prevalence in the general working population, and particularly in young people.

This study aims to test the hypothesis of interaction between various physical and chemical pollutants and their influence on hearing. It obtains a complete temporal exposure model, based on survival analysis, which covers the entire working life of an individual between \( t=0 \) (start time) and \( t=50 \) years (maximum period). The study of multiple exposures using a qualitative variable allows the prevention cost associated with hygiene risk assessment (see 2.3.1. point 1) to be sufficiently reduced. This is also the methodology used in the study of other environment related illnesses caused by prolonged exposure to different agents.
The analysis was carried out using as sample data taken from a pre-existing database on occupational health. The aim was to assess the viability of using these historical databases and the quality of the information obtained from them with regard to the interaction between noise and chemicals and the effect of this interaction on hearing.

The characteristics of the archive information determined the design of the study, the definition of the variables and the method of data analysis used. For instance, the instruments used to measure these variables in some cases may have changed over the prolonged time of this study and it is therefore difficult to maintain consistency. These instruments include: audiometers for identifying the decline in the auditory threshold; integrated sound level meters and dosimeters for the measurement of environmental noise; vacuum pumps for taking air samples, and instruments for chemical analysis used for collecting and quantifying environmental chemical contaminants. Consequently quantitative recording was avoided, defining measurements qualitatively (as binary variables) instead. This provided greater flexibility when evaluating variables, eliminating possible discrepancies associated with potential changes in technology and measurement criteria.

Using a minimum amount of information, one discrete quantitative variable (length of time exposed to noise) and the remaining qualitative variables, it was possible to estimate the influence of a particular working environment on hearing in combination with certain personal habits. The results obtained are of descriptive and explanatory interest, providing information on the interactions between the stated variables and their effects on the individual.

The analysis of the data was fast and economical, whereas obtaining pure samples of data would be less so. Furthermore, and as a corollary, average or high frequencies is required in order to give consistency to the analysis. In addition, if a classification is used to record a variable, it has to be entirely discrete. Failure to fulfil these two criteria (frequency and being discrete) can make analysis using the proposed methodology ineffective, as speculation about the data could lead to an unreliable interpretation of that data.

The results obtained show that workers exposed to noise where metalworking fluids are present show a greater delay in hearing alteration than workers exposed only to noise. By contrast, workers exposed to noise where welding fumes are present exhibited an increase in hearing alteration compared to those exposed only to noise. This thereby demonstrates the antagonistic effect of metalworking fluids with noise and the synergic effect of welding fumes.

As a preventative application, there exists a need for combined respiratory and auditory protection in processes that produce welding fumes, and the former should be effective against certain gases and metal components (use of integrated personal protection equipment). Fabric masks (a highly-used protection) do not meet this requirement, and nor do extraction systems. Environments with noise and metalworking fluids have the advantage in that the aforementioned masks can be used as respiratory protection combined with auditory protection.

Based on recognised research for the study of this problem (Gobba, 2003), the study of pathogenic mechanisms, and evaluation of new multiple-exposure thresholds. This paper focuses on the second of these aspects, the purpose being to obtain patterns that allow for the comparison of various populations of workers in multiple-exposure conditions similar to those defined by such patterns.
In view of the above, the aim of this study is to analyse the influence of the combination of different chemical agents and noise on occupational hearing loss within the metal industry, to be aware of the interrelationships between such factors for preventive purposes.

2. Material

2.1 Study design

A descriptive epidemiological study was conducted, using two types of sources: one based on the records of each individual, occupational medical examinations (OME), with a specific noise protocol (SNP), carried out on various dates during the inclusion period, providing their audiometric data, duration of exposure to noise, and personal habits.

The second type involved on-site testing of a selection of job positions, in order to ensure the type and homogeneity of the environmental exposure conditions of the individuals in the companies included in the sample during the period of study, and environmental record of exposure (ERE).

The study design presented is conceptually interpreted as longitudinal, as defined by Rothman (1986), the existence of a time interval between exposure and the onset of illness.

With two observation points, at t=0 (estimated starting point for the specified sources, after having first carried out a strict process of selection of individuals to be included in the study) and at t=n (period in which the first audiometric test was performed).

2.2 Sample collection

The Aragonese population working in the metal industry during the study period 1991-2000, was evaluated using the Industrial Companies Survey (Spanish acronym EIE) conducted by the Spanish National Statistics Institute (Spanish acronym INE), and an average population of 10,802 workers was obtained.

The data was provided by the Spanish National Institute of Safety and Hygiene at Work (Spanish acronym INSHT) and the Aragon Institute of Occupational Safety and Health (Spanish acronym ISSLA), from a list of companies in their files.

The initial sample size represented 10% of the workers, i.e. 1,080 individuals, using a systematic sampling of companies from said list.

From the initial selection, the following were eliminated: individuals not exposed to occupational noise; those who presented alterations in audiometric tests due to causes other than noise; individuals who, prior to their exposure to occupational noise (t=0), had been subjected to noise outside work over a long period of time; individuals exposed to solvents and degreasing agents and products that did not qualify for inclusion. The final study sample included 558 workers.

2.3 Description of variables

A total of six variables were used, which can be divided into two groups. The first group, characterised by not having missing values consists of three variables, which define the cause-effect relationship: time of noise exposure, the atmosphere to which individuals were
exposed and the degree of hearing alteration. The second group of variables, characterised by having missing values, refers to certain personal habits (smoking, exposure to non-occupational noise and use of hearing protection). These can modify the response of the individual to the environmental factors to which they are exposed at work. These variables therefore have to be controlled to achieve the most accurate interpretation of the results.

2.3.1 Exposure or cause-effect variables (Table 1)

1. "Exposure atmosphere" ($A_{\text{EXP}}$). This was a nominal qualitative variable with three categories. Each category was treated as binary. The variable noise was determined using an integrated sound level meter to classify the individuals in terms of their degree of exposure and its duration. Chemicals were assessed by the presence or absence of the corresponding particles of fluids or smoke in the atmosphere at work. The classification of noise intensity, moderate or high, was adopted for this work. Each one of the three atmospheres at work considered were classified: (a) MF= mainly noise of moderate intensity [85-90] dB(A) in the presence of metalworking fluids; (b) N=only noise, of moderate or high intensity $\geq 85$dB(A); (c) WF= mainly noise of high intensity $\geq 90$dB(A) in the presence of welding fumes.

| Variables (Cause-Effect) | n  | %   |
|-------------------------|----|-----|
| EXPOSURE ATMOSPHERE ($A_{\text{EXP}}$) | 558 |     |
| Noise Only, N           | 177| 31.7|
| Noise+Metalworking Fluids, MF | 146| 26.2|
| Noise+Welding Fumes, WF | 235| 42.1|
| EXPOSURE TIME ($T_{\text{EXP}}$) | 558 |     |
| 0-5                     | 57 | 10.2|
| 5-10                    | 41 | 7.3 |
| 10-15                   | 36 | 6.5 |
| 15-20                   | 42 | 7.5 |
| 20-25                   | 85 | 15.2|
| 25-30                   | 116| 20.8|
| 30-35                   | 106| 19  |
| 35-40                   | 50 | 9   |
| 40-45                   | 22 | 3.9 |
| 45-50                   | 3  | 0.5 |
| DEGREE OF ALTERATION ($D_{\text{ALT}}$) | 558 |     |
| H                       | 158| 28.3|
| IAT                     | 196| 35.1|
| AAT                     | 105| 18.8|
| MH                      | 70 | 12.5|
| AH                      | 29 | 5.2 |

Table 1. Exposure variables

2. "Exposure time to noise" ($T_{\text{EXP}}$). This was a discrete quantitative variable expressed in years. It was an estimation of the time that the worker had been exposed to noise throughout his or her working life. It was established by consulting the individual directly. The possibility of using both the age of the workers and the length of time they were exposed to noise as the time variable was assessed. The projection of each together on a
dispersion graph illustrates the variation between them. Age was rejected as a suitable variable, since in addition to not defining the real duration of exposure effectively it had to then be transformed to achieve its lineal distribution, whereas this was not a problem when the length of time exposed to noise was used as the variable.

3. “Degree of hearing alteration” (D\textsubscript{ALT}). This was an ordinal qualitative variable with five modalities. Each modality was treated as binary. The variable identified the degree of hearing alteration, defined as the decline in the auditory threshold according to acoustic frequency, measured using an audiometer. The audiometry studied at times gave rise to two types of problems in relation to the interpretation of the results. These concerned manual corrections to the audiometric profile and the impossibility of observing the audiometric profile. Therefore the degree of hearing alteration was recorded according to a diagnostic code assigned by the doctor responsible for the check-up based on the Klockhoff classification (1973): H=healthy (losses 25 dB); IAT=initial acoustic trauma (losses of between 25 and 40 dB); AAT=advanced acoustic trauma (losses of between 40 and 50 dB); MH=mild hypoacusis (losses of between 50 and 55 dB); AH=advanced hypoacusis (losses > 55 dB). The losses indicated refer to the 4000 Hz frequency. There was also a loss of adjacent frequencies as the degree of hearing alteration increases.

2.3.2 Habits or modifying variables (Table 2)

4. "Smoking habit" (SH). A nominal binary variable. Recorded whether or not the subject smoked.

5. “Noise outside work” (NOW). A nominal binary variable. Recorded whether or not noisy activities were undertaken outside of work.

6. "Hearing protection" (HP). A nominal binary variable. Recorded whether or not hearing protection was used.

| Variables (Modifying)                  | n  | %   |
|---------------------------------------|----|-----|
| SMOKING HABIT (SH)                    | 558|     |
| No                                    | 147| 26.3|
| Yes                                   | 130| 23.3|
| Missing values                        | 281| 50.4|
| NOISE OUTSIDE WORK (NOW)              | 558|     |
| No                                    | 192| 34.4|
| Yes                                   | 35 | 6.3 |
| Missing values                        | 331| 59.3|
| HEARING PROTECTION (HP)               | 558|     |
| No                                    | 103| 18.5|
| Yes                                   | 95 | 17  |
| Missing values                        | 360| 64.5|

Table 2. Habit variables

The events were defined based on the "degree of hearing alteration" variable, treating this as a nominal variable of binary response. Since in reality it is an ordinal variable with five modalities, it was necessary to transform the initial variable in such a way that the code (0)
represented the cases censored or in which the event did not occur, and the code (1) represented the event occurring. The system followed is represented in Table 3. This approach does not allow other reinterpretations of the type of censures to be used as they must necessarily be to the right because the exact decrease in the threshold is not available for each individual. Instead, only a diagnostic code is available, which did not allow us to define a specific decrease in dB and to relate this to the “duration of exposure” variable.

| Event 1:                          | Modalities                  | Event of Cox?     |
|----------------------------------|-----------------------------|-------------------|
| Healthy (code 0)                 | (H)                         | YES               |
| Altered (code 1)                 | (IAT+AAT+MH+AH)             | Temporary effect (IAT) treated as permanent |

| Event 2:                          | Modalities                  | Event of Cox?     |
|----------------------------------|-----------------------------|-------------------|
| Recovered (code 0)               | (H+IAT)                     | YES               |
| Not recovered (code 1)           | (AAT+MH+AH)                 | Permanent effect  |

| Event 3:                          | Modalities                  | Event of Cox?     |
|----------------------------------|-----------------------------|-------------------|
| No falls in conversational freq. (code 0) | (H+IAT+AAT) | YES               |
| With falls in conversational freq. (code 1) | (MH+AH)       | Permanent effect  |

Table 3. Definition of events

3. Methods

The way of initially tackling the analysis of the data was by defining the survival functions. The main focus of this study was to identify the patterns of hearing alteration over time, related to the environmental conditions to which the individuals were exposed and their “habits”. Once the survival functions were defined and examined, the data was analysed using various regression analysis techniques to identify the most suitable method.

The starting point was one quantitative variable with the remaining variables being qualitative. We are in a limiting case when applying regression theory to the data, that as indicated by Martín & Paz (2007).

Due to reason stated above the number of useful regression models was limited. Linear regression models require at least two quantitative variables. Models based on the discriminating function require the normal distribution of variables, an aspect which in this case was not satisfied as the only category contrasting with the rest (healthy) did not follow a normal distribution. The remaining categories of this variable are self-contained and as a consequence they cannot be analysed using this technique. Multivariate analysis of variance is not an alternative to discriminant analysis as it also requires at least two quantitative variables.

Specific regression techniques for the analysis of quantitative variables also present problems. Thus, logistic regression with nominal binary or polynomous response (Silva & Barroso, 2004), does not allow the quantitative variable (taken as independent) to be correlated with the others variables. Ordinal regression (Greenland, 1994) is not operational either as it is an extension of the above.

The most ideal model for the analysis of this situation is Cox’s regression model, which makes it possible to work with only one quantitative variable (Cox & Snell, 1989). It also
makes it possible for both the response variable and the predictor variables to establish a strong dependence relationship with the single variable, thereby obtaining suitable variants of Cox's regression model for this particular case (Cox's regression with a time dependent variable). It is true that the character of this regression applied to the data is fundamentally explanatory as the prediction must be based on the most frequently recorded samples with the objective of ensuring the accuracy of the observations.

The steps that were followed to apply Cox's model (Hosmer & Lemeshow, 1999) were: (1) Ensuring that the events defined were Cox type events: i.e. they occurred only once and after the event occurred it was set permanently; (2) Checking the proportionality and consistency of the risks. A graph was used based on the projection of the survival functions (demonstrated and not demonstrated); (3) Assessing the high multicollinearity or interdependence. Those variables defined prior to the study, with a correlation of above 0.8, were eliminated; (4) Assessing the linearity of the quantitative variable (duration of exposure). A graph was used based on the projection of the duration of exposure of each individual with respect to their partial residual plot (calculated with respect to their age); (5) Assessing the existence of influencing observations. Delta-beta values were used. (Cook's distance applied to Cox's regression). Values above 1 were rejected; (6) To identify any possible confusion and interaction between variables, the method involving changing model coefficients was used; (7) The correlation between beta coefficients was used to assess the stability of each model; (8) The fit of the models was assessed using probability reasoning; (9) The model was validated indirectly as it was not possible to obtain another, different sample with which to assess this aspect. Validation of the latent structure was used, obtained by the analysis of matches for each one of the two halves of the sample.

3.1 Nonparametric reliability models

The Kaplan-Meyer method (K-M) was used to obtain the survival function of a particular event associated with the various covariables, and for the contrast of functions and their meaning the Log-Rank test was used.

Subsequently a Cox regression model was used, with the aim of explaining the relationships between the variables.

3.2 Parametric reliability models

To obtain the reliability functions the normal distribution model was used and for their contrast a U of Mann-Whitney and t-test was used. For this each one of the binary variables was transformed into another equivalent referring to duration of exposure.

The parametric model was only used as a descriptor of the variables and for testing certain controls, hypotheses and predictions, starting with the probability distributions: (1) Tests to establish controls (regarding the population percentage, with reference to one or more alterations, which must not be exceeded). The tests concern establishing a common "cut off point" for the modalities of the variable "degree of hearing alteration"; (2) Hypothesis tests (regarding the development of hearing alteration). These involve the analysis of the differences in probabilities based on a real value and a theoretical value. An individual with a particular duration of exposure experiences a degree of hearing alteration (real value). In turn, this individual, with that duration of exposure, could experience other degrees of
hearing alteration (theoretical values); (3) Tests of predictions. This involves predicting the development of certain exposed populations, based on the previous controls and hypotheses, making it possible to improve preventative management systems.

### 3.3 Comparison of survival models

Survival functions obtained for the data from the sample, using a parametric and non-parametric model, they were represented together in a graph to assess their equivalence. The interesting aspect of this equivalence is the complementarity of the results, allowing them to be used together i.e. where one model is not suitable, the other is. For example, for the initial data, regression is possible in a non-parametric approach but not in a parametric approach.

Factorial methods were also used with the aim of exploring the relationships between variables. The most suitable factorial method was correspondence analysis carried out using a Burt table (Benzecri, 1992). The heterogeneity of frequency distributions between the variables implies a low degree of dependency between them, above all when considering the “habits” variables with respect to the “exposure” variables. This situation makes the final solution (analogous with the regression results) more contrived than deductive, an aspect which limits the formal application of the factorial model. The problem can be solved using differential topological models (Cova, Márquez & Tovar, 2001), based on Thom’s morphogenetic theory (1971), which is a future direction for this research.

### 4. Results

The characteristics of the sample are shown in Tables 1 and 2, which summarise its structure with respect to the various variables considered.

It is interesting to examine the categories within the “habits” variables, where the degree of personal protection, i.e. use of hearing protection, non-exposure to non-occupational noise and not smoking, is related to the atmosphere at work. It can be seen that as the noise level becomes more harmful the individuals tend to protect themselves more (Figure 1). This fact is very interesting when interpreting the effect on hearing of the noise and chemicals combination.

![Fig. 1. Distribution of personal habits according the exposure atmosphere](image-url)

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As can be seen in figure 2, the survival functions obtained through the Kaplan-Meyer method define how hearing alteration appears in individuals by event and for each work atmosphere. They show clear differences between event 1 and the others.

Thus, in event 1 the "noise with metalworking fluids" atmosphere causes a delay in hearing alteration which is significant (p<0.05), whereas the "noise only" atmosphere and the "noise with welding fumes" atmosphere develop in unison, showing no differences between them (p>0.05).

For events two and three, the curves that characterise each atmosphere are separated from one another significantly (p<0.05), indicating the time differences that exposure to each of them represents and for the same period (see variation of the medians and contrasts, Table 4 and 5). It was demonstrated, furthermore, that the 0 to 15 years period of exposure to noise was low risk, in general presenting hearing alteration of less than 10% in the individuals exposed (Figure 2).

The percentage of individuals affected, over this period gradually decreased as the event continued. Thus, event 1 principally characterises the variations in the hearing threshold of the recoverable type (initial trauma), event 2 non-recoverable but without alteration in conversational speech (advanced trauma) and in event 3 non-recoverable variations with losses in conversational speech (hypoacusis). The situation described gives a dynamic to the process of hearing alteration which is characterised by the migration of the set of survival functions to the right. This explains the existence of a lower risk in the initial periods over time, demonstrating the suitability of the model (Figure 2).

Obtaining univariate, bivariate and trivariate models (Table 6), based on the Cox regression, explains the effect of the various variables in the study, based on the hazard ratio.

In considering the “smoking habit” variable it was found that its effect was antagonistic to atmospheres with metalworking fluids, although the hazard are more or less balanced, depending on the event. This indicates uniform action over time, which is different from metalworking fluids atmosphere, which tend to intensify the effects of smoking (Figure 3.2.).

| Atmosphere | Event | Sample | Percentiles |
|------------|-------|--------|-------------|
|            |       | N      | E   | C   | Q_{25} | SE | Q_{50} | SE | CI 95% | Q_{75} | SE |
| MF         | 1     | 146    | 91  | 55  | 38    | 0.94 | 32    | 1.03 | (30,34) | 25    | 1.29 |
| N          | 1     | 177    | 124 | 53  | 33    | 0.70 | 28    | 0.75 | (27,29) | 21    | 1.65 |
| WF         | 1     | 235    | 185 | 50  | 32    | 0.73 | 27    | 0.61 | (26,28) | 22    | 0.77 |
| MF         | 2     | 146    | 35  | 111 | 44    | 2.43 | 40    | 1.69 | (37,43) | 35    | 1.10 |
| N          | 2     | 177    | 51  | 126 | 41    | 2.05 | 34    | 0.88 | (32,36) | 29    | 1.37 |
| WF         | 2     | 235    | 118 | 117 | 36    | 0.90 | 31    | 0.75 | (30,32) | 26    | 0.65 |
| MF         | 3     | 146    | 12  | 134 | 45    | *    | 44    | 3.60 | (37,51) | 40    | 1.15 |
| N          | 3     | 177    | 25  | 152 | 45    | *    | 41    | 1.72 | (38,44) | 32    | 1.62 |
| WF         | 3     | 235    | 62  | 173 | 40    | 0.44 | 36    | 1.49 | (33,39) | 30    | 0.76 |

N=cases; E=events; C=censored; SE=standard error

Table 4. Characteristics of no parametric survival functions
Fig. 2. No parametric survival functions, Kaplan-Meyer
### Table 6. Cox regression models

| Models       | $V_i$ | Event-1 | Event-2 | Event-3 |
|--------------|-------|---------|---------|---------|
|              |       | Hazard  | Density | Wald    | Hazard  | Density | Wald    | Hazard  | Density |
|              |       | CI 95%  | $\chi^2$ |         | CI 95%  | $\chi^2$ |         | CI 95%  | $\chi^2$ |
| MF           | MF    | -0.524  | 0.467-0.748 | 0.000 | -0.926  | 0.274-0.570 | 0.000 | -1.368  | 0.139-0.466 |
| WF           | WF    | 0.389   | 1.208-1.801 | 0.001 | 0.816   | 1.686-3.034 | 0.000 | 1.159   | 2.031-4.998 |
| SH           | SH    | 0.492   | 1.203-2.227 | 0.001 | 0.391   | 0.974-2.245 | 0.065 | -0.263  | 0.347-1.702 |
| NOW          | NOW   | 0.826   | 1.540-3.391 | 0.000 | 0.812   | 1.207-4.201 | 0.010 | 0.789   | 0.915-5.298 |
| HP           | HP    | -0.338  | 0.521-0.974 | 0.033 | -0.647  | 0.323-0.847 | 0.008 | -1.058  | 0.172-0.701 |
| MF / SH      | MF    | -0.492  | 0.429-0.869 | 0.006 | -0.904  | 0.239-0.685 | 0.001 | -1.582  | 0.072-0.584 |
| MF / NOW     | MF    | 0.473   | 1.179-2.185 | 0.002 | 0.347   | 0.930-2.151 | 0.105 | -0.376  | 0.304-1.548 |
| MF / NOW     | MF    | -0.269  | 0.542-1.075 | 0.123 | -0.867  | 0.232-0.762 | 0.004 | -1.514  | 0.079-0.617 |
| MF / SH      | MF    | -0.383  | 0.469-0.988 | 0.043 | -0.864  | 0.219-0.811 | 0.010 | -1.507  | 0.067-0.731 |
| MF / HP      | MF    | -0.271  | 0.554-1.048 | 0.095 | -0.526  | 0.362-0.963 | 0.035 | -0.881  | 0.204-0.843 |
| MF / HP      | MF    | -0.322  | 1.020-1.866 | 0.036 | 0.726   | 1.354-3.154 | 0.001 | 1.196   | 1.550-7.049 |
| WF / SH      | WF    | 0.477   | 1.184-2.194 | 0.002 | 0.349   | 0.933-2.155 | 0.102 | -0.371  | 0.308-1.548 |
| WF / NOW     | WF    | 0.346   | 1.045-1.912 | 0.024 | 1.062   | 1.746-4.795 | 0.000 | 1.450   | 1.976-9.192 |
| WF / HP      | WF    | 0.781   | 1.470-3.243 | 0.000 | 0.652   | 1.030-3.576 | 0.040 | 0.575   | 0.739-4.271 |
| WF / HP      | WF    | 0.411   | 1.076-2.117 | 0.017 | 1.134   | 1.735-5.571 | 0.000 | 1.432   | 1.686-10.39 |
| NOW / HP     | NOW   | 0.939   | 1.654-3.956 | 0.000 | 0.989   | 1.289-5.601 | 0.008 | 1.100   | 1.111-8.117 |
| NOW / HP     | NOW   | -0.375  | 0.502-0.939 | 0.018 | -0.692  | 0.308-0.813 | 0.005 | -1.104  | 0.164-0.671 |
| MF / NOW / HP| MF    | -0.317  | 0.501-1.058 | 0.096 | -0.805  | 0.232-0.863 | 0.016 | -1.446  | 0.071-0.779 |
| MF / NOW / HP| MF    | 0.891   | 1.573-3.780 | 0.000 | 0.867   | 1.140-4.972 | 0.021 | 0.943   | 0.951-6.940 |
| MF / NOW / HP| MF    | -0.321  | 0.526-0.997 | 0.048 | -0.583  | 0.341-0.913 | 0.020 | -0.946  | 0.190-0.794 |
| WF / NOW / HP| WF    | 0.381   | 1.041-2.057 | 0.028 | 1.104   | 1.680-5.413 | 0.000 | 1.396   | 1.622-10.06 |
| WF / NOW / HP| WF    | 0.906   | 1.601-3.826 | 0.000 | 0.875   | 1.156-4.980 | 0.019 | 0.962   | 0.974-7.035 |
| WF / NOW / HP| WF    | -0.259  | 0.555-1.071 | 0.121 | -0.405  | 0.404-1.101 | 0.113 | -0.760  | 0.226-0.969 |
Fig. 3. Risk factor comparison with the personal habits through the hazard and according the event

Smoking in the WF atmosphere produces a synergistic effect, the action of which is minimised over time (Figure 3.1.). It is curious that for event 3 welding fumes and tobacco...
have an antagonistic effect; tobacco loses its effect in relation to welding fumes in the medium term (event 2) and long term (event 3), both with p>0.05.

The effect of non-occupational noise is antagonistic to that of metalworking fluids (Figure 3.4.), accelerating hearing alteration uniformly depending on the event, although it is in event 2 where it is most apparent, decreasing in the following event (p>0.05). By contrast, the effect of MF atmospheres strengthens over time, or to put it another way, the delay in hearing alteration increases with time p<0.05).

| Hearing alteration | Normality | Linearity |
|--------------------|-----------|-----------|
|                    | µ (Xi)    | σ (Xi)    | VC   | K-S (Z) | Sig 2 tailed | µ ln(Xi) | σ ln(Xi) | R² Normal | R² Log Normal |
| H                  | 13.13     | 11.39     | 85.63% | 2.076   | 0.0004 | 2.05     | 1.16    | 0.852     | 0.708         |
| IAT                | 24.73     | 8.72      | 35.26% | 1.001   | 0.2636 | 3.11     | 0.54    | 0.738     | 0.925         |
| AAT                | 27.38     | 8.07      | 29.47% | 0.959   | 0.3163 | 3.24     | 0.42    | 0.702     | 0.938         |
| MH                 | 29.31     | 7.92      | 27.02% | 0.658   | 0.7788 | 3.34     | 0.31    | 0.679     | 0.930         |
| AH                 | 32.62     | 7.30      | 22.38% | 0.926   | 0.3571 | 3.45     | 0.32    | 0.631     | 0.942         |

VC: Variation coefficient; K-S: Kolmogorov-Smirnov test; R²: Determination coefficient

Table 7. Normality and linearity conditions

Fig. 4. Parametric survival functions obtained for all atmospheres (MF+N+WF, N=558)
Hearing Loss

Table 8. Contrast of parametric survival functions (U of Mann-Whitney and t-test)

| Categories   | Normal | Log Normal |
|--------------|--------|------------|
|              | U     | Z   | Sig 1 | t   | CV-t 2 | Sig |
| H-IAT        | 6811  | -9.066 | 0.0000 | 3.7024 | 1.9839 | 0.0003 |
| IAT-AAT      | 8349  | -2.698 | 0.0070 | 0.5595 | 1.9804 | 0.5768 |
| AAT-MH       | 3283  | -1.193 | 0.2325 | 0.4361 | 1.9804 | 0.6635 |
| MH-AH        | 715   | -2.309 | 0.0209 | 0.7795 | 1.9802 | 0.4372 |

*(1, 2)* 2 tailed; CV-t: critical value for t

In WF atmospheres non-occupational noise produces a uniform effect depending on the event. It plays a more active role in event 1 in hearing alteration in relation to smoke (Figure 3.3.).

The use of individual protection equipment produces an effect similar to that of metalworking fluids, although their effectiveness increases over time (Figure 3.6.). This is characteristic when the protection equipment is not used continuously. It also explains the major delay produced in MF atmospheres.

In WF atmospheres the use of individual protection equipment is clearly antagonistic (Figure 3.5.), increasing in effectiveness over time, although the action of the WF atmosphere is much more powerful than the protection equipment.

Subsequently the effect on hearing of exposure to all atmospheres in the metal industry was analysed. To do this the initial sample was subdivided into the 3 atmospheres studied and in turn each one of these was divided into the five phases of hearing alteration. In doing this the frequencies were considerably reduced and as a consequence the analysis was not very consistent. Using the combined analysis of atmospheres to study the various phases of hearing alteration was the most useful option. For this analysis a parametric model (log-normal) was used to obtain the survival functions (Figure 4.3.). This has the advantage over those non-parametric models of the probability distribution of the event using continuous functions. This gives more precision to the distribution of each degree of hearing alteration and as a consequence to the identification of the time of the event (Figure 4, Table 8).

In this case, the survival curves must be understood as the combination of individuals who present a specific hearing alteration, independently of the atmosphere to which they are exposed and their personal habits. Each function associated with a degree of hearing alteration characterises an average value i.e. a theoretical value consisting of the combination of the three atmospheres to which must be added the combination of "habits" of the individuals in the sample (Figure 4.1.).

The conditions of normality and linearity of each degree of hearing alteration, obtained according to time, were assessed (Table 7).

The similarity of the survival functions for the different degrees of hearing alteration was also assessed using parametric and non-parametric methods, with the objective of making both the results and their interpretation homogeneous (Figure 5). It should be noted that except for the group of healthy people who do not follow a normal distribution, the remaining degrees of hearing alteration do follow a normal distribution. It can also be seen that in accordance with the degree hearing alteration the mean value of the distributions are displaced to the right. This confirms the suitability of the sample, which is also corroborated...
by the low frequency of individuals that are affected as the degree of hearing alteration increases. The spread of hearing alteration over time can be seen. Thus, once the level of advanced acoustic trauma is reached, the individual undergoes a more rapid process of hearing alteration. This can be substantiated, because the curves tend to unite more than in the IAT/AAT transition.

Fig. 5. Survival function equivalence obtained by parametric and no parametric models

5. Discussion

The qualitative methodology proposed for the study of the combined influence of noise and chemical pollutants on hearing loss (Conte et al., 2009) differs from that used in traditional
studies on the same topic. These perform a quantitative analysis of decreases in the hearing threshold, an aspect which was replaced by an audiogram classification based on a diagnostic reference. The duration of each individual's exposure to noise was also used, instead of their age, thereby improving the linear behaviour of the temporal variable. Finally, each chemical contaminant was characterised by a binary variable, thus avoiding the use of an environmental measurement value, which provides more general and less restrictive identification than quantitative environmental measurements.

This study shows the influence of noise on hearing alteration, whether temporary (IAT) or permanent (AAT, MH, AH). This situation is consistent with studies conducted on the influence of this physical agent on hearing.

Moreover, chemical agents taken as interacting with noise (MF and WF) have been considered by various researchers as pulmonary toxins (Godderis et al., 2008; Schaller et al., 2007), due to the principal way they enter the body: by inhalation. It is nonetheless true that the influence of these agents on hearing loss, a toxic effect that can be considered indirect, has not been given due attention.

This study confirms the existence of an interaction between physical and chemical factors in the metal industry which influence the alteration of auditory function, and which can be characterised by three different exposure environments, WF with noise, MF with noise, and noise only.

The interaction of the pollutants with the individual determines whether the auditory effects caused by the main risk factor (noise) develop more quickly or slowly in the worker. Thus, it can be identified that metalworking fluids delay the development and worsening of the various stages of auditory alteration, whereas welding fumes speed up the development of same. In this respect, the behaviour of one contaminant with another is antagonistic.

The study also indicates that, in the case of welding fumes, the chemical agent is shown to be more detrimental to hearing. One of the main problems regarding welding fumes in the presence of noise is that, in general, the protection used is effective in muffling noise intensity but not in reducing the effect of the chemical agent. In this situation, cellulose masks or those of similar compounds have little effect, as their capacity to filter particles (such as charcoal) is not effective for gaseous molecules such as carbon monoxide, which is highly ototoxic (Gwin et al., 2005; Morley et al., 1999).

As regards personal habits, there is a growing tendency to use hearing protection as the harmfulness of the environment increases. The interpretation of this fact is due to an increased personal willingness to use protective equipment when the individual feels some discomfort, which may be intuitively associated with the work environment. This study verifies that the increase in using protection is not sufficiently capable of improving auditory health conditions, supporting the negative effects of welding fumes on workers.

With regard to the regression models, it has been demonstrated that the univariate models (MF and WF) are those which best, and more accurately, define each model according to the event. Despite a loss of accuracy, the bivariate models may be more interesting as regards application. For Event1, the variable SH is shown to be the most influential and best represented of the models. For this event, NOW is also considered an acceptable model, along with WF.
For Event2 the ideal models are MF with NOW and with HP, as well as WF with NOW. There is a decline in accuracy with respect to the previous event.

For Event3 only the MF-HP model is considered suitable, with the other two habits losing significance.

This indicates the influence obtained for each habit variable: SH influences IAT; NOW influences the development of AAT; HP is influential as protection at all stages, even if it is ineffective against fumes.

The influence of smoking habits (SH) on the initial auditory alteration recognised in this study coincides with the results obtained by other authors (Pouryaghoub et al., 2007; Ferrite et al., 2005; Mizoue et al., 2003), but indicates the need for further research in order to properly assess this influence.

6. Conclusions

A methodological framework was presented which made it possible to use employment related health databases with limited information. The limitations of the data, resulting from possible changes in the way the data was obtained and recorded during the period under study, led to the use of qualitative, binary response variables and only one quantitative variable, namely the time of exposure to noise.

With this situation as the starting point, it was established that survival analysis is one of the best ways of analysing this type of data, both in relation to defining probability-time functions and their contrasts, and for modelling using Cox regression, in relation to both the application possibilities and the results reached (descriptive-explanatory in character).

This research was aimed at the analysis of the interaction between noise and chemicals and its influence on occupational hearing loss. It was found that in the Aragonese metal sector, which was the focus of this study, there were three main atmospheres: noise with metalworking fluids, noise only and noise with welding fumes.

The analysis made it possible to establish that hearing alteration in individuals was related to the exposure atmosphere. Thus, workers exposed to noise and metalworking fluids, who protected themselves less, experienced slower hearing alteration compared to those who were exposed to only noise, and workers exposed to welding fumes, who protected themselves more, suffered hearing alterations sooner than those who were only exposed to noise.

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Authored by 17 international researchers and research teams, the book provides up-to-date insights on topics in five different research areas related to normal hearing and deafness. Techniques for assessment of hearing and the appropriateness of the Mongolian gerbil as a model for age-dependent hearing loss in humans are presented. Parental attitudes to childhood deafness and role of early intervention for better treatment of hearing loss are also discussed. Comprehensive details are provided on the role of different environmental insults including injuries in causing deafness. Additionally, many genes involved in hearing loss are reviewed and the genetics of recessively inherited moderate to severe and progressive deafness is covered for the first time. The book also details established and evolving therapies for treatment of deafness.

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