# Auditory effects of combined exposure: interaction between carbon monoxide, noise and smoking

# *Efeitos auditivos da exposição combinada: interação entre monóxido de carbono, ruído e tabagismo*

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## ABSTRACT

**Purpose:** To analyze the auditory effects of the combined exposure to carbon monoxide (CO) and noise, and the impact of smoking. **Methods:** Participants were 80 male workers, smokers and non-smokers, from a steel industry – 40 exposed to CO and noise simultaneously, and 40 exposed only to noise. A retrospective data analysis was conducted regarding the environmental risks (CO and noise) and the file information related to auditory health and to the biological concentrations of CO in the blood (CPHb). The first and the last pure-tone audiometry results were analyzed considering the smoking habits, the type of exposure (CO and noise or noise only), the time of exposure, the level of noise, and age. **Results:** Both the CO concentration and the noise levels were above the tolerance limits provided by the regulatory norm number 15 of the Ministry of Labor and Employment. The group of workers exposed to CO and noise presented a higher rate of noise-induced hearing loss (22.5%), when compared to the group exposed only to noise (7.5%), as well as significant worsening of the hearing thresholds of 3, 4 and 6 kHz. Age, time of exposure, type of exposure, level of noise, and smoking habit significantly influenced the auditory threshold of the participants. Smoking enhanced the effects of both CO and noise on the auditory system. **Conclusion:** The occupational exposure to noise and CO resulted in significant effects on the auditory system of workers from a steel industry.


**Keywords:** Noise effects; Chemical compounds; Occupational exposure; Drug synergism; Hearing loss; Occupational health

## INTRODUCTION

Noise-induced hearing loss (NIHL) is the most prevalent irreversible work-related disease in the world, affecting people, on physical, psychological and social levels. There are several factors that can start it or make it worse, such as smoking, noise, vibration and environmental chemical contaminants (solvents, metals, *asphyxiants*, etc.), among others[1-7].

Among chemical contaminants that may interact with noise, increasing the harmful effects of exposure on hearing and on health in general, carbon monoxide (CO) deserves special attention[3,5 to 13].

From the family of chemical asphyxiants, CO is a colorless and odorless gas that is formed by the incomplete combustion of organic material in the presence of oxygen deficit, accounting for over 50% of the poisonings worldwide and the most common cause of death by poisoning[7,10]. CO is present in industrial processes such as blast furnaces, vehicle emissions, boilers, charcoal kilns, workshops, garages and welding operations[9-12].

The results of some studies on the toxicity of CO in the auditory system show that it has a direct effect on cochlear function[13]. There is evidence showing that CO exposure may facilitate NIHL. Alone, it would not be ototoxic, but it may be when present with noise[13,14].

Moreover, CO in cigarette components can reduce oxygen levels in the cochlea and result in vasoconstriction, increased blood flow, reduced oxygen transport and oxyhemoglobin dissociation problems. Noise exposure also induces cochlear hypoxia, causing injury or interacting with the mechanisms of NIHL. Thus, chronic hypoxia caused by smoking can contribute to hearing loss, particularly in the basal region of the cochlea[15]. Research suggests that smoking associated with noise exposure induces temporary threshold shifts and that this effect is attributed more to CO than to nicotine[16].

Therefore, the aim of this study was to analyze the auditory

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effects of combined exposure to carbon monoxide (CO) and noise, and the impact of smoking.

## METHODS

This study consisted of an exploratory observational study conducted between May and July 2008, through retrospective analysis of documents in a steel foundry specializing in pig iron, located in the metropolitan region of Belo Horizonte (MG), Brazil. The company has 750 employees and has a risk level of 4, according to Regulatory Norm (NR) 4 of the Ministry of Labor and Employment (MTE)[17].

The company does not have a Hearing Conservation Program (HCP). Currently the hearing protection device (HPD) used by study participants are the inserted silicone type, make and model Pomp Plus® and a rating of 17dB (NRRsf). As for the total time of use of an HPD, it was not possible to obtain reliable data, since there are no records for the start of an HPD use for all participants surveyed. Currently, all workers are required to use an HPD as well as the personal protective equipment (PPE), such as respiratory protection masks, shaving aprons, helmets, goggles and gloves when also exposed to CO.

We analyzed the data for 2008 related to environmental hazards (noise and CO) and the information contained in the medical records related to hearing health and the biological concentrations of CO in the blood, verified by laboratory examination of carboxyhemoglobin (COHb).

The information regarding environmental noise analysis contained in the documents of the Program of Prevention of Environmental Risks (PPRA) for the company was analyzed. The noise was evaluated by the Engineering Occupational Safety department of the company in question and the evaluation was performed with samples of workers in all departments, throughout the workday. To this end, we used a Simpson® brand model 897 noise dosimeter, acoustically calibrated before and after the evaluations. The criteria and procedures for the assessment of occupational noise exposure met the standard requirements of the Fundacentro NHO-01 (Level criterion equal to 85dBA, with an 8-hour time criterion and an exchange rate of 3).

Because the concentrations of CO in the environment were not included in the company PPRA documents, they were evaluated by the research team, along with the company's Engineering Work Safety department, with an MSA® brand, model MINI-CO, CO detector. The detector was positioned in front of the mouth of a closed blast furnace, in order to verify the levels of CO in the environment. Evaluation of CO was endorsed by RCFA 1093-093 PPE standards.

The blast furnace is the only equipment at the company that produces CO. The operation of the furnace consists of burning charcoal, which generates at an elevated temperature, causing the iron ore inside the oven to be smelted. Once molten, the pig iron is taken from the furnace and stored in a heated container or solidified into bars. Within the logistics of this process, three types of jobs were found to be exposed to CO and noise in the work environment. Thus, all other jobs analyzed were not exposed to CO and were considered as exposed to noise only.

The 2008 medical records already contained information: identifying information (name, age, job title, industry, daily working hours, working time in the company, etc.), data on smoking, chronic diseases (hypertension, diabetes) and medication use; concentrations of COHb in the blood of participants exposed to CO, and audiometry results.

The COHb examination was conducted in a clinical laboratory, by collecting blood from the worker at the end of the workday. The references of the outcome are: normal up to 3.5% for non-smokers and from 4.0 to 9.0% for smokers[18].

For the documentary analysis, the sample consisted of 80 male participants (40 exposed to CO and noise, and 40 exposed only to noise). To form risk groups, the areas studied were those with environmental exposure to CO and noise (production I, II and III) or only noise (dispatch; assembly and loading). For CO and noise, the positions studied were: furnace operator; furnace operator assistant and caster. For workers exposed only to noise, the positions were: shipping operator; casting box worker; wheel operator; and loader operator.

We excluded workers from the sectors evaluated (n=80) who had, in their audiograms, conductive or mixed hearing loss and/ or with a history of ear pathologies.

As to the audiological evaluation, the audiometric reference and the final audiometric examination of each participant were analyzed, following the parameters of Brazilian legislation[19].

All examinations were performed by an audiologist in a soundproof booth and participants presented an auditory rest of over 14 hours. In the evaluation by air conduction at frequencies of 500 Hz, 1, 2, 3, 4, 6 and 8 kHz were checked, and bone conduction at frequencies of 500 Hz, 1, 2, 3 and 4 kHz in both ears[19].

The analysis was also based on Appendix I of NR 7[19], in which examinations were classified as normal, or within acceptable limits for those with hearing thresholds up to 25 dB HL at all frequencies. Hearing loss, or a state suggestive of NIHL, was considered to be present in audiograms showing bilateral sensorineural hearing loss with hearing thresholds above 25 dB HL, predominantly at the frequencies of 3 kHz, 4 kHz and 6 kHz in both air and in bone. Exams showing hearing loss that was classified as being from other causes not related to the present work were excluded from the study.

Audiometries that, in comparison with the reference audiometry, obtained a difference between the averages of hearing thresholds in the group of frequencies 500 Hz, 1 and 2 kHz, or group of frequencies 3, 4 and 6 kHz, of a value equal to or higher than 10 dB, or equal to or higher than 15 dBHL in frequency isolation were considered to be initiating or worsening of hearing.

The statistical method used was the general linear model, in one way analysis of variance (ANOVA). We used the Student's t test, considering a significance level of 5% (p=0.05).

This study was approved by the Ethics Committee of the Faculty of Management Studies of Minas Gerais (FEAD) involving human subjects, under protocol number 44 and authorized by the individuals through the signing of a consent term.

## RESULTS

Table 1 shows values for the descriptive statistics of the variables: age and working time according to the groups for CO and noise and those for only noise, respectively.

**Table 1.** Description of age and length of service of the participants (n=80)

| Variable | Noise & CO (n=40) | | Noise (n=40) | |
|---|---|---|---|---|
| | n | % | n | % |
| Age (years) | | | | |
| 20 – 30 | 3 | 7.50 | 10 | 25 |
| 31 – 40 | 15 | 37.50 | 7 | 17.50 |
| 41 – 50 | 13 | 32.50 | 14 | 35 |
| >50 | 9 | 22.50 | 9 | 22.50 |
| Time of service (years) | | | | |
| 0 – 5 | 4 | 10 | 8 | 20 |
| 6 – 10 | 28 | 68 | 20 | 50 |
| 11 – 15 | 8 | 20 | 10 | 25 |
| >15 | 0 | 2 | 2 | 5 |

**Note:** CO = carbon monoxide

The assessment of noise levels of the group exposed simultaneously to CO and noise showed: caster (88.7 dBA), furnace operator (93.5 dBA), and furnace operator assistant (93.5 dBA). The group exposed only to noise showed: casting box worker (86.6 dBA), wheel operator (83.3 dBA), shipping operator (106.8 dBA), kettle operator (88.2 dBA), and loader operator (86.7 dBA).

The results of the evaluation of CO in the blast furnace, during a full work day (eight hours), ranged from 200 to 700 parts per million (ppm). All members of the CO and noise group perform their tasks in the same environment, independent of job function, and are also exposed to the same concentrations of CO.

The minimum, maximum, and average plasma levels of carboxyhemoglobin in the blood of smokers and non-smokers exposed to CO and noise are presented in Table 2.

**Table 2.** Dosage of carboxyhemoglobin (COHb) in the blood of smoking and nonsmoking workers exposed to CO and noise

| Result | Non-smokers | Smokers |
|---|---|---|
| Min. value (%) | 0.80 | 0.80 |
| Max. value (%) | 10.20 | 17.30* |
| Mean | 2.96 | 6.30* |

* Significant values (p=0,000) - ANOVA
**Note:** CO = carbon monoxide

Significant differences (p=0.000) were observed in the results of the maximum and average. Workers who smoke showed higher levels of COHb when compared to nonsmokers (Table 2).

The results of the last audiometry showed that 22.5% of participants in the CO and noise group's audiograms suggested some degree of NIHL, while 7.5% of participants in the group exposed to noise showed these characteristics.

Figure 1 shows the results of the test for audiometric threshold worsening depending on the risk groups (%) according to Decree 19 (1998).

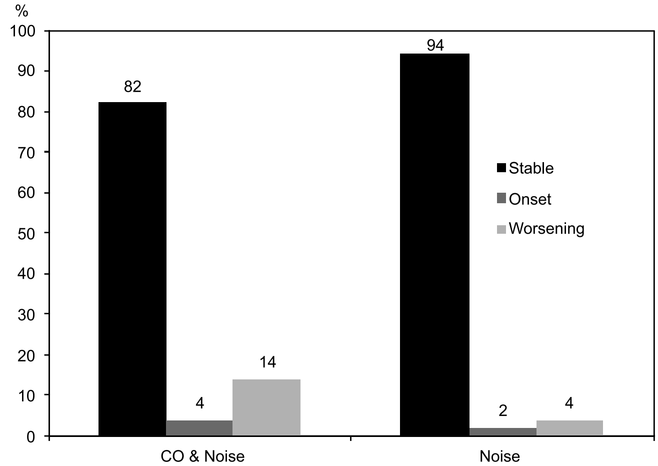
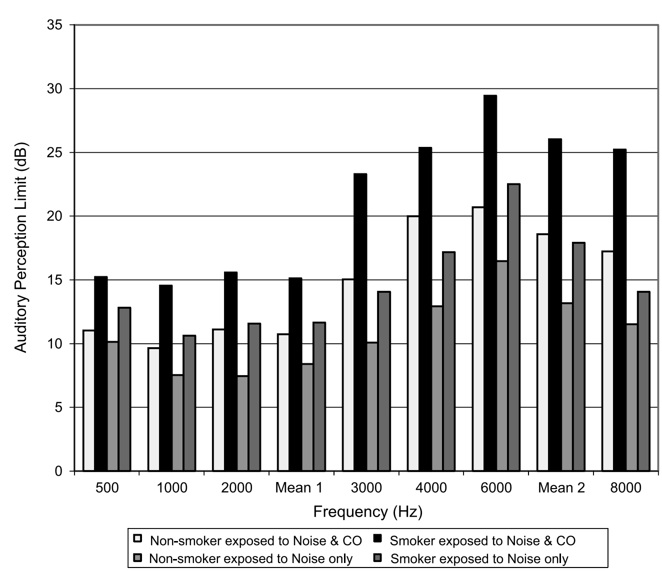



**Figure 1.** Results of the analysis in worsening audiometric thresholds in terms of risk groups (%), according to Ordinance 19 (1998)

The onset of NIHL in the group exposed to CO and noise was observed in 4% of participants and at 2% in the group exposed only to noise. Already worsening NIHL was observed in 14% of the group exposed to CO and noise and 4% for the group exposed only to noise (Figure 1).

Figure 2 shows the mean thresholds as a function of risk groups in relation to smoking, as well as the tritonal average both for low and medium frequencies (mean 1: 500 Hz, 1 and 2 kHz), and for high frequencies (average 2, 3, 4, and 6 kHz).

Smokers had worse thresholds compared to their nonsmoking peers (mean 1 p=0.003 and mean 2 p=0.009). The same effect is observed for the frequency of 8 kHz (p=0.000). Nonsmoking workers exposed to CO and noise showed worse




**Note:** Mean 1 = Mean audiometric thresholds at frequencies of 500 Hz, 1 kHz and 2; Mean 2 = Mean audiometric thresholds at frequencies of 3, 4 and 6 kHz

**Figure 2.** Mean audiometric thresholds (dB HL) by frequency (Hz), depending on risk groups and smoking

auditory thresholds than those for nonsmoking workers exposed only to noise. However, smoking workers exposed to CO and noise presented worse thresholds than those for smoking workers exposed only to noise and than those for nonsmoking workers, regardless of their risk group (Figure 2).

Thus, analysis of variance showed significant differences (p=0.009) between smoking and risk groups (CO and noise or just noise) for low and medium frequencies (mean 1). For high frequencies (mean 2), in addition to the difference between smoking and risk groups (p=0.000), differences were observed between the groups at risk and age (p=0.000). That is, a worsening in audiometric results when smoking was present when participants were exposed to CO and noise and when over 40 years of age.

Table 3 presents the results observed among the variables: age, smoking, noise levels, duration of service, depending on the analyzed risk groups (groups: CO and noise, and noise only) using the Student's t test, considering a significance level of 5% (0.05).

In the analysis of the variance for the frequencies of 500 Hz, 1 and 2 kHz, significant differences were observed between hearing thresholds and: a) smoking b) risk groups (CO and noise, and noise only). In the study of frequencies 3, 4 and 6 kHz there were significant differences between hearing thresholds and: a) smoking, b) age, c) noise level and d) risk groups. In all significant differences observed, there was a worsening of the audiometric results when: a) participants were smokers, b) participants were over 40 years old, c) when participants were exposed to CO and noise, and d) when the noise level was higher than 85 dBA.

The analysis of variance showed that for cases suggestive of NIHL-mean 2 (hearing loss restricted to frequencies of 3, 4, and 6 kHz), significant differences were observed in isolation between age (p=0.003), the noise level (p=0.050), smoking (p=0.009), risk groups (CO and noise), and the association between smoking and risk groups (p=0.003), risk groups and age (p=0.004) and age and noise (p=0.003).

The analysis showed that for a frequency of 3 kHz, age (p=0.050), the noise level (p=0.008), smoking (p=0.019), those at risk due to smoking (p=0.000), age (p=0.023), groups as a function of noise (p=0.050), and age as a function of the noise level (p=0.050), caused the worsening of audiometric results.

The analysis showed that for a frequency of 4 kHz, age (p=0.008), the noise level (p=0.029), groups (p=0.003), smoking as a function of the groups (p=0.003), risk groups according to age (p=0.004), and noise level according to age (p=0.009) influenced the worsening of audiometric results.

The analysis showed that for a frequency of 6 kHz, age (p=0.037), smoking (p=0.003), smoking according to the groups (p=0.007), risk groups according to age (p=0.049), and noise level according to age (p=0.041) influenced the worsening of audiometric results.

The study of the variation of frequencies of 3, 4 and 6 kHz, depending on the risk groups according to age showed a worsening in mean hearing thresholds at high frequencies in the group exposed to CO and noise when compared to the group exposed only to noise and over 40 years of age.

## DISCUSSION

Besides noise, other ototoxic agents that are present in the workplace can affect the auditory system[1-8]. The chemical contaminant in focus in this paper is CO, when combined with noise and smoking caused permanent changes in the auditory system of the participants.

Despite the existence of studies on auditory effects of chronic exposure to CO in the presence of noise in the workplace[4,7,10], the question still needs to be further explored. Possible ototoxic changes induced by CO combined with noise and smoking, chronic exposure, and the magnitude of the exposed population, supported the choice for this study.

The results of this study demonstrated that environmental data, both CO and noise levels (except for the wheel operator) measured in the workplace, are above the tolerance limits set in NR-15[18] and by the ACGIH[20] for the agents in question.

For noise, the maximum allowable levels in NR-15[18], for 8 hours of exposure would be 85 dBA, however, because the participants currently use PPE with a rating of 17 dB, theo-

**Table 3.** Results observed depending on the hearing thresholds of 500 Hz to 8 kHz

| Variables | 500 Hz | 1 kHz | 2 kHz | Mean of high and low frequencies | 3 kHz | 4 kHz | 6 kHz | Mean of high frequencies | 8 kHz |
|---|---|---|---|---|---|---|---|---|---|
| Age | 0.629 | 0.665 | 0.938 | 0.807 | 0.050* | 0.008* | 0.037* | 0.003* | 0.623 |
| Noise lev. | 0.292 | 0.334 | 0.638 | 0.383 | 0.008* | 0.029* | 0.398 | 0.050* | 0.985 |
| Smoking | 0.002* | 0.003* | 0.050 | 0.003* | 0.019* | 0.146 | 0.003* | 0.009* | 0.119 |
| Groups | 0.079 | 0.217 | 0.676 | 0.236 | 0.144 | 0.002* | 0.244 | 0.009* | 0.746 |
| Smoking x Groups | 0.032* | 0.007* | 0.031* | 0.009* | 0.000* | 0.003* | 0.007* | 0.003* | 0.000* |
| Groups x Age | 0.665 | 0.961 | 0.187 | 0.442 | 0.023* | 0.004* | 0.049* | 0.004* | 0.029* |
| Groups x Noise lev. | 0.089 | 0.298 | -0.615 | 0.260 | 0.050* | 0.418 | 0.115 | 0.113 | 0.759 |
| Age x Noise Lev. | 0.559 | -0.580 | 0.923 | 0.688 | 0.050* | 0.009* | 0.041* | 0.003* | 0.739 |

* Significant values (p<0.05) –ANOVA Test
**PS:** High and low frequencies = 500 Hz, 1 and 2 kHz; high frequencies = 3, 4 and 6 kHz
**Note:** Noise lev. = Equivalent noise level

retically noise levels would not be damaging the hearing of participants, except for the shipping operator, that even when using PPE, the noise level was above the recommended level (89.8 dBA). It is necessary to review the hearing protection for these workers.

The ambient concentrations of CO, according to NR-15[18], should not exceed 39 ppm for 8 hours of unprotected exposure. However, even using a HPD, levels were evaluated as far above the recommended health risk.

The same happened with the maximum rate of carboxyhemoglobin some for some nonsmoking workers, who performed higher than recommended (Table 2). Similar data related to the concentration of CO were obtained in a study of forklift operators[21]. It is known that various factors are responsible for rates of carboxyhemoglobin in subjects, for example, the endogenous production of CO, cigarette smoke, exposure to dichloromethane, workload, or exposure to high concentrations of CO in the environment[22].

Thus, in this study it was observed that at least two of the five factors mentioned above, could be responsible for the increased rate of carboxyhemoglobin workers in the group exposed to CO and noise, they are: smoking (for smoking workers) and high concentrations of CO in the environment (especially for nonsmoking workers) (Table 2). However, this analysis is limited and we can only say that these two factors were responsible for the change in rates of carboxyhemoglobin, without investigating the other factors mentioned in the Canadian study[22]. This question deserves to be further explored in future studies.

With respect to audiometric findings (Figure 1), the highest occurrence of the onset and worsening of NIHL and noise in the CO group suggests the potentiating effects of CO. The phenomenon of CO in NIHL facilitation has been shown in studies with laboratory animals[13,14,23]. The authors concluded that exposure to noise caused a cochlear vasoconstriction and anoxia caused by exposure to CO, increased oxygen demand, causing greater NIHL than expected.

Results similar to our findings (Figure 2), were shown in a Canadian study[24], which analyzed 6847 audiometric tests conducted by the National Institute of Public Health of Quebec between 1983 and 1996. We compared two groups: a group of individuals exposed to noise of 90 dBA and to CO, and another group of individuals exposed to noise of 90 dBA only. The results showed significant differences in hearing thresholds between groups exposed to CO and noise and those only exposed to noise, precisely at high frequencies (3, 4 and 6 kHz), indicating the facilitation of NIHL when CO is present in the environment. It is noted that the Canadian study[24] due to sample size, showed more consistent results compared to our findings, since sample size was one of the limitations of this study.

As for smoking, it is known that cigarette components also cause hypoxia and may influence hearing loss[15,16]. It is evident through these findings that smoking may increase the effects of CO as much as noise in the auditory system (Figure 2). In agreement with our findings, a study[25] demonstrated that age and noise exposure alone are positively associated with hearing loss. However, the effect of age combined with exposure to noise was higher than the sum of individual effects to estimate the effect of hearing loss. This combined effect occurred especially for smokers, between 20 and 40 years old that were exposed to noise. The authors concluded that the observed synergistic effect of smoking, noise exposure, and age for hearing loss is consistent with a biological interaction. However, it is possible that toxic substances in the composition of cigarette associated with age, affecting hearing, without the individual being necessarily exposed to noise.

Significant differences were found between age, length of service, risk groups (CO and noise, only noise), noise level, and smoking (Table 3). Thus, all these factors influence risk, isolated or associated with worsening of hearing thresholds for the participants. Consistent with our findings, occupational epidemiological studies also demonstrated positive associations between hearing thresholds and age[25,26], service time[27], noise levels[25-28], risk groups[25, 28] and smoking[25, 29].

Although the findings of our study reveal significant differences between exposure to CO and noise, smoking and hearing loss, age and length of service; the limit for CO exposure, combined with noise levels and smoking, in order to avoid auditory ill effects is still unknown. As for the association of biological effects (age, length of service, lifestyle habits, etc.) with occupational risks (using PPE, HPD, noise, chemicals, etc.), this deserves further investigation, as it may contribute significantly in audiometric findings, as confirmed by this study.

Therefore, it becomes necessary to implement a hearing conservation program[1,3,4] for that segment of industrial workers, regardless of the noise level and the CO concentration to which workers are exposed.

We believe that one of the greatest health challenges for workers is the auditory effects caused by combined exposures to different ototoxic agents. Thus, the example of research institutions such as NIOSH[3] and ACGIH[20], who recommend the implementation of hearing conservation programs, including monitoring the hearing of workers exposed to the chemical industry since 1998[2], we suggest that no ototoxic agent be overlooked and that environmental and biological monitoring of ototoxic agents, in addition to audiological monitoring of all workers exposed to risk agents, be implemented even if noise levels are below the tolerance limits.

Thus, preventive/educational actions[1] are strongly recommended in order for the early identification, avoidance, or worsening of NIHL, and providing a better quality of life for workers. It is necessary to implement measures of collective control of occupational hazards, as well as providing satisfactory use of PPE and HPD, regarding exposure to both noise and chemicals. Educational workshops are important in order to raise awareness for self-care and for improvements in the relationship between work and health[3].

## CONCLUSION

Significant impaired hearing effects were identified in 40 workers exposed to CO when compared to the hearing of 40 workers exposed only to noise. Correlations were found between age, length of service, risk groups (CO and noise and only noise), the noise level, and smoking, influencing the audiometric thresholds of exposure to CO and noise. It was found that smoking may potentiate the effect of CO as much noise in the auditory system.

This study had some important limitations: the lack of data on the start of use of PPE and HPD, thus hindering the establishment of the relationship of exposure to risk and length of service; the lack of information about the extra exposure to occupational ototoxic agents; the lack of analysis to demonstrate how age and length of service influenced the audiometric findings, especially in comparing the auditory reference test with the most recent test. And also, what was the time interval between audiograms to evaluate the onset or worsening of NIHL among risk groups.

However, the results demonstrate the importance of the subject studied, thus encouraging the continuation of this research and the realization of new studies on the subject.

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## RESUMO

**Objetivo:** Analisar os efeitos auditivos da exposição combinada ao monóxido de carbono (CO) e ao ruído, e o impacto do tabagismo. **Métodos:** Participaram da pesquisa 80 trabalhadores fumantes e não fumantes, do gênero masculino, oriundos de uma empresa siderúrgica, sendo que 40 estavam expostos ao CO e ao ruído e 40 somente ao ruído. Realizou-se análise retrospectiva dos dados referentes aos riscos ambientais (CO e ruído) e das informações contidas nos prontuários médicos relacionadas à saúde auditiva e às concentrações biológicas do CO no sangue (COHb). Analisou-se a audiometria tonal de referência e a última, e os limiares auditivos em função do tabagismo, do tipo de exposição (CO e ruído ou somente ao ruído), do tempo de exposição, do nível de ruído e da idade. **Resultados:** Tanto a concentração de CO como os níveis de ruído encontraram-se acima do limite de tolerância previsto na norma regulamentadora de número 15 do Ministério do Trabalho. O grupo exposto ao CO e ao ruído apresentou mais casos de PAIR (22,5%), comparativamente ao grupo exposto somente ao ruído (7,5%) e também apresentou piora significativa nos limiares auditivos de 3, 4 e 6 kHz. Foram encontradas diferenças significativas entre a idade, o tempo de serviço, o tipo de exposição, o nível de ruído e o hábito de fumar influenciando nos limiares auditivos dos participantes. O hábito de fumar potencializou o efeito tanto do CO quanto do ruído no sistema auditivo. **Conclusão:** Efeitos auditivos significativos foram identificados na audição dos trabalhadores de uma siderúrgica expostos ao CO.

**Descritores:** Efeitos do ruído; Compostos químicos; Exposição ocupacional; Sinergismo farmacológico; Perda auditiva; Saúde do trabalhador

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