

Hearing Damage at Work: A Public Health Problem

Diana J Gutiérrez-Tinajero^a, Paulina Murphy-Ruiz^a, and María Luz Arenas-Sordo^{b*}

^a Department of Audiology. Instituto Nacional de Rehabilitación. Mexico City, Mexico

^b Department of Genetics. Instituto Nacional de Rehabilitación. Mexico City, Mexico

Introduction Noise is the main labor risk and constitutes a public health problem. The purpose of this study was to examine the relationship between noise at the workplace and acoustic trauma and noise-induced hypoacusia. **Methods** An observational, prospective and transversal study was carried out on 116 workers of an asphalt factory in Mexico City, Mexico. Descriptive statistical analysis was carried out, and Spearman's rank correlation was run to determine the relationship between years of exposure, decibels and acoustic trauma. **Results** Correlation between years of exposure and degree of hypoacusia was low (Spearman's rho 0.3281). The correlation between years of exposure and type of acoustic trauma was also low (Spearman's rho 0.3514). **Discussion** Correlation between years of exposure to noise at the workplace and hearing loss is low. Other factors, such as genetic susceptibility, are important. Hearing loss prevention programs should be instituted in workplaces with high noise levels to prevent noise-induced hearing loss as well as other health effects related to noise exposure.

Keywords: hypoacusia • noise • workers • asphalt factory • public health

Introduction

Noise is the main labor risk and constitutes a public health problem. One third of the world population is affected by some degree of hearing loss caused by exposure to high intensity noise (Krishnamurti, 2005; Kitcher *et al.*, 2012).

Noise causes diverse physical reactions, auditory as well as non-auditory. In the ear, noise contributes to damage in the hair cells within the organ of Corti (Krishnamurti, 2005; Mulders *et al.*, 2011). Noise, depending on its properties (intensity, duration, frequency, tone, timing, etc.), can result in total destruction of the hair cells within the ear (Daniell *et al.*, 2006; Baradarnfar *et al.*, 2012). However, regardless of the extent of damage, there are changes in auditory function (Krishnamurti, 2005; Mulders *et al.*, 2011).

Noise-induced hypoacusia (which is also known as chronic acoustic trauma or noise-induced auditory damage) can be defined as the partial or total diminished auditory capacity that can be temporary or permanent, in one or both ears, and is of sensorineural type. It originates gradually, during and as result of intermittent or continuous exposure to harmful levels of noise

(more than 80 dB SPL) (Krishnamurti, 2005; Brown *et al.*, 2012).

Occupational hypoacusia may lead to increased stress, work accidents, and low labor performance (Mazurek *et al.*, 2010; Baradarnfar *et al.*, 2012). Due to the number of workers exposed to high levels of noise, occupational hypoacusia is one of the most common occupational diseases (Krishnamurti, 2005; Daniell *et al.*, 2006).

Methods

An observational, prospective and transversal study was carried out on 116 workers of an asphalt factory in Mexico City, Mexico. All participants were asked if they agreed to participate and signed an informed consent form. All procedures were in accordance with the Declaration of Helsinki.

All workers had a tonal and verbal audiometry with Orbiter 922 TM audiometer as well as tympanometry and stapedial reflexes with Zodiac 901 TM impedanciometer. Pure tone average was obtained through audiograms. Audiometric curves were classified as acoustic trauma grade I if they presented a fall in 4 kHz, grade II if there was a fall in 4 kHz and another frequency; grade III if three frequencies were affected; and noise-induced hearing loss when there was damage in more than three frequencies.

Accepted: 26 July 2013

Citation: Gutiérrez-Tinajero, DJ, Murphy-Ruiz, P, & Arenas-Sordo, ML. (2013). Hearing Damage at Work: A Public Health Problem. *KJHCM*. 1(1):15–17.

Author contacts: asgk@unam.mx (MLAS*), dgutierrez@inr.gob.mx (DJGT), chinos_pau@gmail.com (PMR)

* Corresponding author. Instituto Nacional de Rehabilitación, Av. México-Chochimilco 289. Col. Arenal de Guadalupe. Tlalpan 14389, México, DF.

Statistical analysis

Descriptive statistical analysis was carried out. As well Spearman's rank correlation was run to determine relationship between the variables years of exposure, decibels and acoustic trauma.

Results

Only one of the 116 corresponded to the feminine sex.

Age range went from 21 to 80 years of age. We grouped the workers by decades. The majority was found in the group range of 41-50 years.

Duration of exposure to labor noise was classified by lustrums (5-years). The range was between 5 and 41 years. The two majority groups were found between 5 to 10 years and from 26 to 30 years.

Based on audiometric classification, 73% of the individuals had hypoacusia due to noise, 22% normal audition and 5% non-noise related hypoacusia. In the logoaudiometry we found that 100 ears presented the maximum phonemic discrimination to 100% in a threshold between 35 and 40 dB. The rest were in the thresholds between 45 to 105 dB. See Table 1.

Table 1: Audiometric profile and auditory threshold

Audiometric profile	Workers		Auditory threshold	Ears			
	n	%		N		%	
				RE	LE	RE	LE
Normal audition	26	22	<20dB	45	45	41	41
ATG1	24	20	21-40 dB	43	42	39	38
ATG2	11	9	41-60 dB	14	17	13	15
NIHD	27	23	61-80 dB	6	4	5	4
Not related to noise	6	5	>81 dB	2	2	1	2

Abbreviations: ATG: acoustic trauma grades 1, 2, & 3; NIHD: noise-induced hearing damage; RE: right ear; LE: left ear; dB: decibels

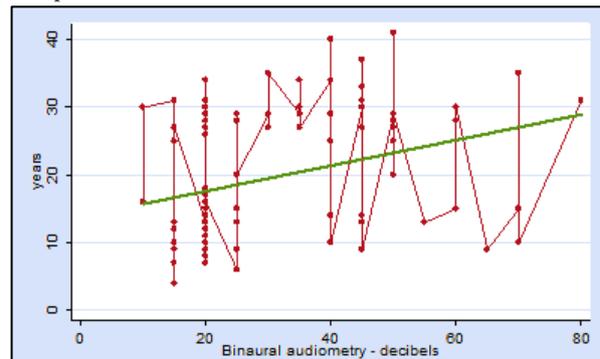
As for the tympanometry, 103 workers had a normal curve of pressure and compliance, which correspond to the type A curve of the Jerger classification. Eleven workers had As curves, one with Ad curve and another with curve C.

The stapedial reflexes in the right ear were present in 78% of cases, of which 52% were normal while in 48% they were in the intensities corresponding to recruitment. In the left ear, 83% had reflexes and 51% of these had 48% they were in the intensities corresponding to recruitment. In the left ear, 83% had reflexes and 51% of these had response in recruitment intensities.

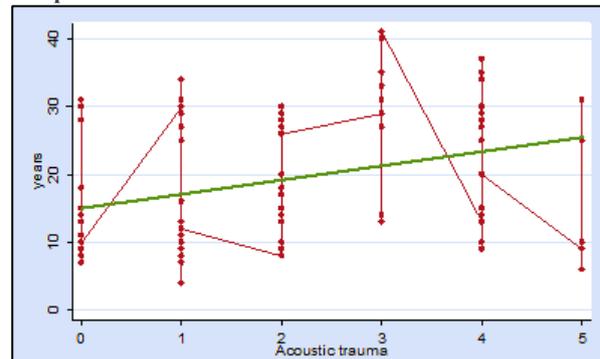
A correlation between the years of exposure to noise and the degree of hypoacusia was carried out, and we found that there is a weak relationship between the duration of exposure and auditory loss (Spearman's rho=0.3281). See Graph 1.

An analysis between the correlation and type of acoustic trauma and the duration of exposure to noise was also carried out. There was a low correlation with a Spearman's rho of 0.3514. See Graph 2.

Graph 1



Graph 2



Discussion

We found in our study that stapedial reflexes were present in 80% of the cases, half of which demonstrated signs suggestive of loudness recruitment, which indicates outer hair cells damage. These findings have not been reported by other authors (Cotanche 2008; Giordano *et al.*, 2008), but corroborates the findings of other earlier studies (Daniell *et al.*, 2006; Kitcher *et al.*, 2012). Outer hair cells within the organ of Corti, depending on the noise stimuli, may have suffered partial or total damage (Le Prell *et al.*, 2007; Bodmer 2008). Chronic exposure to noise also causes damage to inner hair cells and supporting cells of the organ of Corti (Krishnamurti, 2005; Browne *et al.*, 2006).

Following the examination of average audible tones obtained by audiometric testing, for both right and left ears; we found hearing thresholds below 20dB in spite of case history, record background and duration of exposure to noise (around 30 years in most workers). This finding corroborated with the threshold of maximum phonemic discrimination in the logoaudiometry. To our knowledge, this finding has not been reported in the literature.

We found a low correlation between the years of exposure to noise and the magnitude of auditory loss (Spearman's rho 0.3281). The same goes for the correlation between years of exposure and the type of acoustic trauma or acoustic damage (Spearman's rho 0.3514). Both correlations, although low, were positive.

Comorbidities were presented in a small proportion of workers which allowed us to attribute auditory alterations we found, mainly, to exposure to noise (Bodmer, 2008; Giordano *et al.*, 2008). Other factors are also important, including genetic

predisposition in some workers which may have made them more susceptible to auditory damage than others.

Exposure to high levels of noise is a major cause of preventable hearing loss. It has been estimated that worldwide as many as 500 million individuals might be at risk of developing noise-induced hearing loss (Sliwinska-Kowalska & Davis, 2012).

Noise, as an undesired sound in the workplace, is a serious environmental hazard, and it must be controlled and its intensity maintained within acceptable levels at the workplace (Seidman & Standring, 2010; Dube *et al.*, 2011).

Health policies should focus on raising awareness of the risks of noise-induced hearing loss in the workplace and elsewhere. Nowadays, noise-induced hearing loss is irreversible, necessitating as much effort as possible put toward prevention. These efforts should include identifying of high-risk noise exposures, particularly those affecting the young, improving noise regulations and legislations; and enhancing the effectiveness of hearing protectors, especially at the workplace (Finegold *et al.*, 2012; Dube *et al.*, 2011).

In addition it is necessary to implement a hearing conservation program to raise awareness about and to prevent noise-induced hearing loss, especially at the workplace (Finegold *et al.*, 2012).

References

- Baradarnfar, M, Karamifar K, Mehrparvar, A, Mollasadeghi, A, Gharavi, M, Karimi G, Vahidy, M, Baradarnfar, A & Mostaghaci, M. (2012). Amplitude Changes in Otoacoustic Emissions After Exposure to Industrial Noise. *Noise & Health*. **14**(56):28–31.
- Bodmer, D. (2008). Protection, Regeneration and Replacement of Hair Cells in the Cochlea: Implications for the Future Treatment of Sensorineural Hearing Loss. *Swiss Medical Weekly*. **138**:708–712.
- Browne, C, Morley, J & Parsons, C. (2012). Tracking the Expression of Excitatory and Inhibitory Neurotransmission-Related Proteins and Neuroplasticity Markers after Noise-Induced Hearing Loss. *PLoS One*. **7**(3): e33272. doi:10.1371/journal.pone.0033272. Epub 2012 Mar 12.
- Cotanche, D. (2008). Genetic and Pharmacological Intervention for Treatment/Prevention of Hearing Loss. *Journal of Communication Disorders*. **41**(5):421–443.
- Daniell, W, Swan S, McDaniel, M, Camp J, Cohen M & Stebbins, J. (2006). Noise Exposure and Hearing Loss Prevention Programmes after 20 Years of Regulations in the United States. *Occupational and Environmental Medicine*. **63**(5):343–351.
- Dube K, Ingale L & Ingale S. (2011). Hearing Impairment among Workers Exposed to Excessive Levels of Noise in Ginning Industries. *Noise & Health*. **13**(54):348–355.
- Finegold, L, Schwela, D & Lambert, J. (2012). Progress on Noise Policies from 2008 to 2011. *Noise & Health*. **14**(61):307–312.
- Giordano, M, Garzaro, J, Nadalin, G, Pecorari, R, Boggero, P Argentero & R Albera. (2008). Noise-Induced Hearing Loss and Hearing Aids Requirement. *Acta Otorhinolaryngologica Italica*. **28**(4):200–205.
- Kitcher E, Ocansey, G & Tumpi, D. (2012). Early Occupational Hearing Loss of Workers in a Stone Crushing Industry: Our Experience in a Developing Country. *Noise & Health*. **14**(57):68–71.
- Krishnamurti S. (2009). Sensorineural Hearing Loss Associated with Occupational Noise Exposure: Effects of Age-Corrections. *International Journal of Environmental Research and Public Health*. **6**(3):889–899.
- Le Prell, C, Yamashita, D, Minami, S, Yamasoba, T & Miller, J. (2007). Mechanisms of Noise-Induced Hearing Loss Indicate Multiple Methods of Prevention. *Hearing Research*. **226**(1–2):22–43.
- Mazurek, B, Olze, H, Haupt, H & Szczepek A. (2010). The More the Worse: The Grade of Noise-Induced Hearing Loss Associates with the Severity of Tinnitus. *International Journal of Environmental Research and Public Health*. **7**(8):3071–3079.
- McCullagh, M, Raymond D, Kerr M & Lusk S. (2011). Prevalence of Hearing Loss and Accuracy of Self-Report among Factory Workers. *Noise & Health*. **13**(54):340–347.
- Mulders, W, Ding, D, Salvi, R & Robertson, D. (2011). Relationship between Auditory Thresholds, Central Spontaneous Activity, and Hair Cell Loss after Acoustic Trauma. *The Journal of Comparative Neurology*. **519**(13):2637–2647.
- Seidman, M & Standring R. (2010). Noise and Quality of Life. *International Journal of Environmental Research and Public Health*. **7**(10):3730–3738.
- Sliwinska-Kowalska, M & Davis, A. (2012). Noise-Induced Hearing Loss. *Noise & Health*. **14**(61):274–280.