

Noise induced hearing loss: An occupational medicine perspective

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Abstract

Purpose of review: Up to 30 million workers in the United States are exposed to potentially detrimental levels of noise. While reliable medications for minimizing or reversing noise induced hearing loss (NIHL) are not currently available, NIHL is entirely preventable. The purpose of this article is to review the epidemiology and pathophysiology of occupational NIHL. We will focus on at-risk populations and discuss prevention programs. Current prevention programs focus on reduction of inner ear damage by minimizing environmental noise production and through the use of personal hearing protective devices.

Recent findings: Noise induced hearing loss is the result of a complex interaction between environmental factors and patient factors, both genetic and acquired. The effects of noise exposure are specific to an individual. Trials are currently underway evaluating the role of antioxidants in protection from, and even reversal of, NIHL.

Summary: Occupational NIHL is the most prevalent occupational disease in the United States. Occupational noise exposures may contribute to temporary or permanent threshold shifts, though even temporary threshold shifts may predispose an individual to eventual permanent hearing loss. Noise prevention programs are paramount in reducing hearing loss as a result of occupational exposures.

Key words: occupational noise induced hearing loss, occupational noise exposure, hearing protection programs

Introduction

Hearing loss is the most widespread disability in Westernized society. Noise exposure is the most common preventable cause of hearing loss; some estimate that one third of all cases of hearing loss can be attributed to noise exposure.¹ Occupational exposure to noise makes up a great proportion of patients affected by noise induced hearing loss (NIHL), making NIHL the most prevalent occupational disease in the United States.² Occupational noise exposure has been documented since at least the 18th century, when it was noted that copper miners developed hearing loss as a result of the noise from hammering on metal.³

Scope of the problem

Worldwide, 1.3 billion people are affected by hearing loss.⁴ It is estimated that 10% of the world's population are at risk for NIHL.² Within the United States, 16-24% of hearing losses can be attributed to occupational noise exposures.^{5,6} In fact, it is estimated that 22 million to 30 million workers in the United States are exposed to potentially detrimental levels of noise.^{2,7}

The effects of occupational noise exposure place a tremendous burden on both the individual and society. The financial burden to society is significant and continues to rise, with an estimated \$242.4 million annual expenditure in compensation for work-related hearing loss in the United States.² Following military service, workers affected by NIHL make up the most populated disability classification. In 2010, the U.S. Department of Veterans Affairs compensated more than 1.4 million veterans for service-connected NIHL and tinnitus, resulting in more than \$1.2 billion of compensation.³

Workers most at risk for occupational NIHL include those employed in construction, manufacturing, mining, agriculture, utilities, transportation, and the military, as well as musicians. A retrospective cohort study evaluated audiograms of 1 million employees exposed to loud noise and found the jobs at highest risk for hearing loss were mining, wood product manufacturing, construction of buildings, and real estate and rental leasing. Within this group of 1 million noise-exposed workers, hearing loss was more prevalent among men than women, and the risk of hearing loss increased with age.⁸

The consequences of occupational NIHL to the individual can be dire. Hearing loss limits an individual's ability to communicate with the surrounding world, which can lead to increased social stress, depression, embarrassment, poor self-esteem, and relationship difficulties. Obstacles in communication are exacerbated by difficult listening situations such as environments with excessive background noise. Hearing loss can affect attention and cognition.² In older adults, a mild hearing loss is associated with a two times increased risk of dementia, while a severe hearing loss is associated with a five times increased risk of dementia.⁹ Associated aural fullness and tinnitus can also be extremely troublesome for some people.

Occupational NIHL has been associated with an increased risk for work-related injuries. Excessive noise hampers the ability to hear warning signals, monitor equipment, respond to environmental sounds, and communicate with other workers. One study of 46,550 workers exposed to noise found a statistically significant increase in risk for injury requiring hospitalization with each decibel of hearing loss. The hazard ratio for injury was 1.01 for each decibel of hearing loss.¹⁰ Another study evaluated 1,062 workers in a tractor manufacturing plant. In this study, workers exposed to sound intensities ≥ 85 dBA averaged over 8 hours had a 1.52 fold increased risk of injury compared to workers exposed to < 85 dBA.¹¹ Undiagnosed hearing loss has been associated with an excess mortality of 10-20% over the course of 20 years.¹²

Noise-induced hearing loss has also been associated with a number of non-auditory sequelae. The most prevalent of these are annoyance, cognitive impairment, sleep disturbance, and adverse cardiovascular health.² A theory has been proposed that noise exposure leads to stimulation of the autonomic nervous system and endocrine system, leading to increased stress hormone release and elevations in blood pressure and heart rate. This, in turn, may lead to an increased risk of hypertension, ischemic heart disease, and stroke.^{13,14} Epidemiological studies have demonstrated a higher prevalence of cardiovascular disease and mortality in populations exposed to high noise levels.^{15,16,17,18,19,20,21}

Pathophysiology

Noise induced hearing loss may be inflicted by short bursts of loud sound or continuously elevated noise levels.² Such exposures lead to cochlear hair cell damage, damage to surrounding supporting cells, and ultimately degeneration of associated auditory nerve fibers. The level of inner ear damage and associated hearing loss are correlated to the intensity and duration of noise exposure.³

Sublethal levels of noise damage may lead clinically to temporary threshold shifts, in which an individual experiences depressed sensorineural hearing that usually recovers within 24 to 48 hours. More severe damage leads to a permanent threshold shift, clinically manifested as an irreversible decrease in hearing thresholds. Recent evidence suggests that even temporary threshold shifts from which an individual recovers are not innocuous, as such insults may accelerate the process of age-related hearing loss.^{22,23,24}

The amount of hearing loss inflicted by a specified noise exposure is variable and specific to an individual. Like many disease processes, the pathophysiology of noise-induced hearing loss represents a complex interaction between genetic and environmental factors. Some authors have estimated that up to 50% of individual variations in hearing loss due to noise exposure may be attributed to hereditary factors.²⁵ In addition, other patient-related factors such as age, pre-existing sensorineural hearing loss, hypertension, diabetes mellitus, smoking history, and

use of ototoxic medications may alter the amount of damage incurred to the inner ear as a result of noise.¹

Noise exposure typically affects the hair cells near the basal turn of the cochlea, leading to a characteristic decrease in hearing thresholds between 3 kHz and 6 kHz. This pattern of hearing loss will produce a distinctive dip at 4 kHz on pure-tone audiometry, often termed the “noise notch” (Figure 1). The natural resonance frequencies of the outer ear, in particular the ear canal, appear to emphasize the damage to this frequency region. The lower frequencies, including the main speech frequencies, are often spared initially. With progressive damage, however, the noise notch will flatten as thresholds decrease at the surrounding frequencies.³ Individuals with severe hearing loss as a result of noise exposure may have audiograms that are indistinguishable from other causes of hearing loss.

Occupational NIHL is typically bilateral and symmetric. There may be, however, some asymmetry in hearing loss, particularly if there is differential exposure to sound between the two ears. When a discrepancy is present, occupational NIHL is commonly more severe in the left ear, though the reasons for this have not been scientifically born out.³ Some have hypothesized that a right-handed machine operator may be more likely to look over their right shoulder, leaving the left ear turned toward a machine engine. In hunters or others who shoot firearms, hearing loss tends to be more severe in the ear closest to the barrel of the gun; the opposite ear is relatively protected by the head shadow effect. Musicians may have discrepancies based on their location within the band or orchestra. Brass players generally produce the greatest amount of noise,²⁶ and musicians seated with one side closer to a noise-producing instrument may have greater exposure in that ear.

Prevention Programs

At the present time, occupational NIHL is not a treatable disease. It is, however, entirely preventable. A focus is placed, therefore, on prevention of NIHL before permanent damage to the inner ear can occur. Prevention strategies may be focused on reducing noise levels in an occupational environment or on reducing the amount of environmental noise that is transmitted to an individual’s inner ear system. Many countries have implemented legal standards regarding occupational noise exposure and the need for protection above a certain exposure level. In the United States, occupational noise exposure is regulated by the Occupational Safety and Health Administration (OSHA, a division of the U.S. Department of Labor) and the National Institute for Occupational Safety and Health (NIOSH, a division of the U.S. Centers for Disease Control and Prevention within the U.S. Department of Health and Human Services). These two governing bodies work together to develop and enforce workplace safety regulations in the prevention of occupational NIHL.

In order to monitor occupational noise exposures, noise levels must be quantified and expressed as a numerical value that can be subjected to standardized regulations. For regulatory purposes, noise levels are generally measured in the

dBA scale and expressed as an 8-hour time-weighted average (TWA). The dBA scale is a decibel scale in which the low frequencies are relatively de-emphasized. This scale is applied when measuring occupational noise exposure because high-frequency noise exposures cause greater threshold shifts than low-frequency exposures of equivalent intensity.²⁷ OSHA and NIOSH have both set forth regulatory guidelines for acceptable occupational noise exposures and recommendations for hearing conservation programs. OSHA regulations carry the weight of law; however, NIOSH recommendations are more stringent and are thought to be backed by stronger scientific evidence.²⁷ Under OSHA criteria, the maximum 8-hour noise exposure is 90 dBA. For noise above 90 dBA, exposure time must be reduced by 50% for every 5 dB increase (e.g. maximum allowable exposure time for 95 dBA noise is 4 hours, etc.). This is referred to as the 5-dB trading rule or 5-dB exchange rate. In contrast, NIOSH recommendations are calculated using a 3-dB exchange rate, in which exposure time must be halved for every 3 dB elevation in noise levels. In addition, NIOSH sets the recommended 8-hour TWA exposure at 85 dBA rather than 90 dBA.²⁷ These changes lead to considerably more protective regulations for workers, as is demonstrated in Table 1.

OSHA mandates that a hearing conservation program be provided for workers who are exposed to noise ≥ 85 dB over an 8-hour TWA. This hearing conservation program must include the following elements: (1) employers must measure noise levels on the premises, (2) free hearing protective devices must be provided if noise exceeds exposure limits, (3) employees must be trained regarding noise exposure and the use of personal hearing protective devices, (4) employees must be provided with baseline and annual audiograms to assess their hearing, and (5) records of all test results must be kept and made available to employees.²⁷ With regard to noise regulation, noise sources or the environment must be altered as much as possible through engineering or scheduling changes to decrease noise levels in the workplace.²⁸ Mechanisms to reduce noise production and exposure through engineering and administrative controls may provide the most effective means for reducing workers' exposure to noise, but often these mechanisms are unrealistic or impossible to impose. When the on-the-job environmental noise levels cannot be brought down to acceptable standards, every employee must be provided with appropriate personal hearing protective devices and instructed on their use. It may be that current regulations, which are based upon recovery of pure-tone thresholds following noise exposure, will, in the future, need to take into account the recently reported findings that even recovery of thresholds following temporary threshold shifts does not truly indicate an undamaged ear.^{22,23,24}

Personal hearing protective devices include both earmuffs and earplugs. Expandable foam plugs provide similar noise attenuation to earmuffs, though other types of earplugs provide less protection. Advertised attenuation levels of hearing protective devices range from 15-28dB when tested in a laboratory setting. In real workplace conditions, most earmuffs and expandable foam plugs will provide 10 to 15dB attenuation. The use of earmuffs and plugs together improves attenuation by 5 to 10dB over either device alone. A protective effect of 10 to 15dB is relevant to a

noise-exposed worker, as 10dB of attenuation will bring noise levels into the acceptable range in more than 90% of exposed workers.²⁷ Data has shown that earplugs may not provide the advertised level of protection if employees are not instructed on their proper use.²⁹ The most important variable in preventing occupational NIHL with personal hearing protective devices is consistent use of the device. Data suggest that personal hearing protective devices are effective in preventing NIHL;^{29,30,31,32} however, studies have demonstrated that many workers do not regularly use these devices.^{33,34,35,36,37} Non-use of hearing protective devices tends to be more prevalent in certain occupations, particularly among agricultural workers.³⁸ Musicians represent another class of workers many of whom do not regularly use hearing protective devices.²⁶

Studies evaluating the success of hearing protection programs have demonstrated conflicting results. A Cochrane Review of 15 studies including 79,986 participants found that the regular use of personal hearing protective devices as part of a strong hearing loss prevention program was associated with less hearing loss.²⁹ Other studies have demonstrated significant continued hearing loss even in the presence of a hearing loss prevention program. Groenewold and colleagues studied audiometric data from 19,911 workers who were exposed to significant noise, stratified into groups based on survey responses into those workers who never used hearing protection and those who always used hearing protection. Data did not show any significant difference in OSHA threshold shifts between the two groups, though there was a significant trend toward increased risk for high frequency threshold shift without use of protective devices.³⁹

Future Directions

Research into the underlying disease processes has produced some headway in achieving preventative and therapeutic treatments for occupational NIHL. Several authors have theorized that oxidative stress plays a role in the pathogenesis of NIHL on a microcellular level.^{40,41} Accordingly, several antioxidant compounds have been explored for their protective properties. In a study looking at the treatment effect of the antioxidant compound magnesium after impulse noise, magnesium-treated guinea pigs had improved hair cell preservation compared to groups treated with methylprednisolone and with placebo.⁴²

The antioxidant precursor of glutathione, N-acetyl-L-cysteine, has been shown to have protective effects against noise exposure in chinchilla studies.⁴³ Human studies on such compounds are preliminary and have had conflicting results. One trial in which 31 normal-hearing participants were treated with N-acetyl cysteine as a protective mechanism against nightclub music exposure did not demonstrate a protective effect.⁴⁴ A double-blind, placebo-controlled crossover study, however, performed on 53 noise-exposed workers did demonstrate that N-acetyl cysteine significantly reduced the incidence of temporary threshold shift.⁴⁵ Interestingly, variability in response to this compound was associated with genetic

polymorphisms. Those workers with the glutathione S transferases null genotype demonstrated increased responses to the treatment compound.

Another compound, D-methionine, may soon undergo clinical trials with the US Army for its use as an otoprotective agent.²⁶ D-methionine is an ingredient of yogurt and cheese that has shown a protective effect against NIHL in animal studies.^{46,47} This agent was effective even when administered after the noise exposure. Each of these compounds has shown promise in preventing or reversing the effects of NIHL, and hopefully in the future will add to the armamentarium of protective tools against occupational NIHL.

Conclusion

Occupational NIHL is the most prevalent occupational disease in the United States. The burden of disease is felt by both the affected individual and by society. Treatment strategies are in the developmental stages; before they become universally available, the mainstay of care is in prevention. Prevention programs to reduce occupational NIHL can be effective, but they require compliance with OSHA and NIOSH standards and consistent use of personal hearing protective devices.

Key Points

- Occupational NIHL is the most prevalent occupational disease in the United States.
- Occupational NIHL can result in psychosocial isolation and an increased risk of dementia in the elderly population, as well as an increased risk of injury, heart disease, and stroke.
- The effects of noise on an individual's hearing threshold are dependent on a complex interaction between genetic and environmental factors that is unique to an individual.
- The most important way to mitigate occupational NIHL is through prevention programs that emphasize exposure reductions and the use of personal hearing protective devices.
- Antioxidant compounds have shown promise as preventative and possible therapeutic agents against NIHL.

Maximum Exposure Level in dBA	Maximum Exposure Duration	
	OSHA HCA (1983) Regulations	NIOSH (1998) Recommendations
85		8 hours
88		4 hours
90	8 hours	2 hours, 31 minutes
95	4 hours	47 minutes, 37 seconds
97	3 hours	30 minutes
100	2 hours	15 minutes
105	1 hour	4 minutes, 43 seconds
110	30 minutes	1 minute, 29 seconds
115	15 minutes or less	28 seconds

Table 1. Comparison of OSHA and NIOSH regulations for noise exposure.

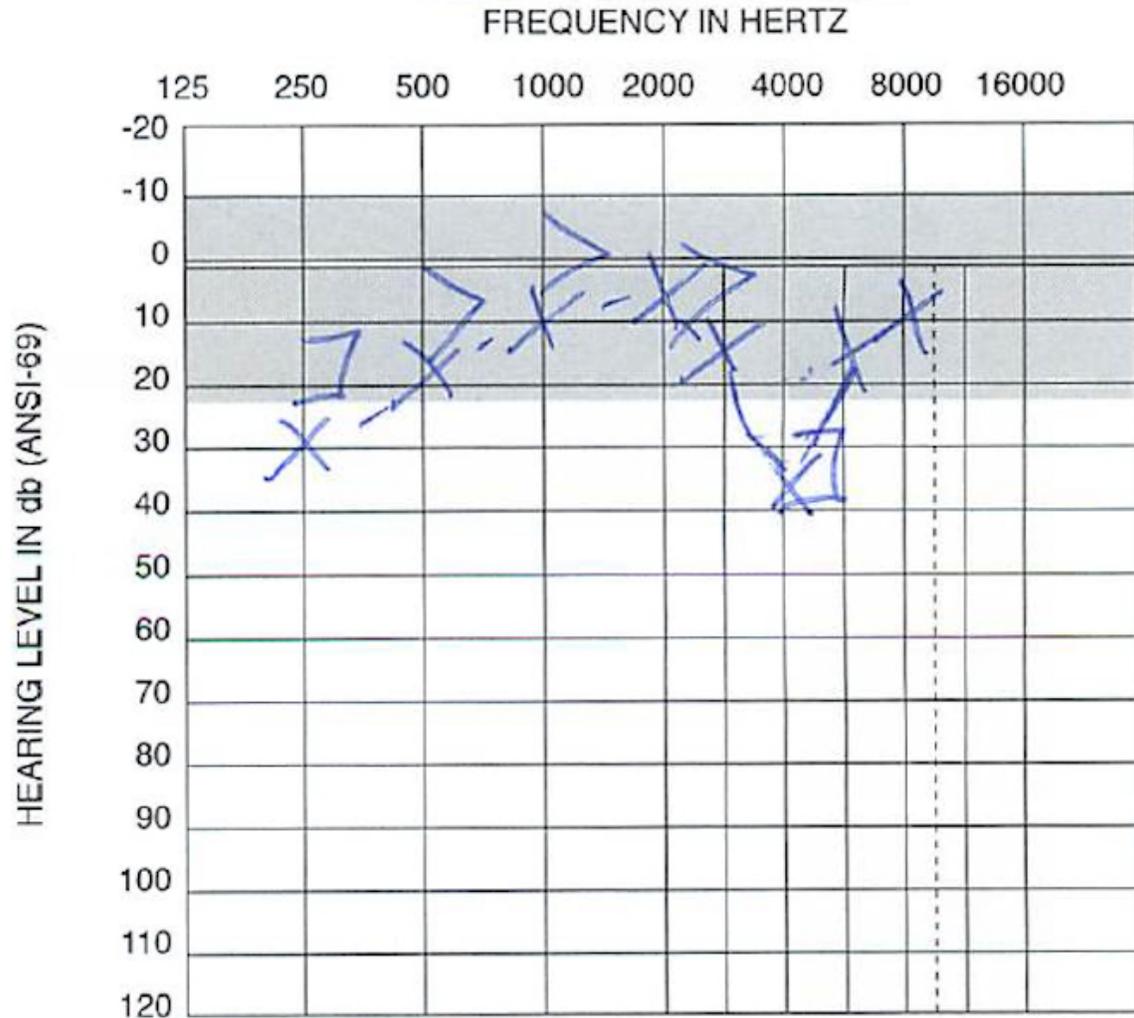


Figure 1. Pure-tone audiogram demonstrating the classic “noise notch” at 4 kHz.

References

- ¹ National Institutes of Health. Consensus Development Conference Statement: Noise and Hearing Loss. Bethesda, MD; U.S. Department of Health & Human Services; 1990.
- ² Basner M, Babisch W, Davis A, et al. Auditory and non-auditory effects of noise on health. *Lancet* 2014; 383:1325-32.
** A detailed discussion of the effects of noise induced hearing loss and their implications for public health.
- ³ Hong O, Kerr M, Poling G, Dhar S. Understanding and preventing noise-induced hearing loss. *Dis Mon* 2013;59:110-8.
** An excellent review of the mechanisms and effects of noise induced hearing loss, as well as prevention programs.
- ⁴ Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2163-96.
- ⁵ Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M. The global burden of occupational noise-induced hearing loss. *Am J Ind Med* 2005;48:446-58.
- ⁶ Tak S, Calvert GM. Hearing difficulty attributable to employment by industry and occupation: An analysis of the National Health Interview Survey – United States, 1997 to 2003. *J Occup Environ Med* 2008;50:46-56.
- ⁷ National Institute for Occupational Safety and Health (NIOSH). National Occupational Research Agenda. Cincinnati, OH: National Institute of Occupational Safety and Health Publications 1996. 96-115.
- ⁸ Masterson EA, Tak S, Themann CL, et al. Prevalence of hearing loss in the United States by industry. *Am J Ind Med* 2013;56:670-81.
- ⁹ Lin FR. Hearing loss in older adults: who's listening? *JAMA* 2012;307:1147-8.
- ¹⁰ Girard SA, Leroux T, Courteau M, Picard M, Turcotte F, Richer O. Occupational noise exposure and noise-induced hearing loss are associated with work-related injuries leading to admission to hospital. *Inj Prev* 2014;Epub ahead of print.
- ¹¹ Amjad-Sardrudi H, Dormohammadi A, Golmohammadi R, Poorolajal J. Effect of noise exposure on occupational injuries: A cross-sectional study. *J Res Health Sci* 2012;12:101-4.
- ¹² Karpa MJ, Gopinath B, Beath K, et al. Associations between hearing impairment and mortality risk in older persons: the Blue Mountains Hearing Study. *Ann Epidemiol* 2010;20:452-9.
- ¹³ Babisch W. Cardiovascular effects of noise. In: Nriagu JO, ed. *Encyclopedia of environmental health*. Burlington: Elsevier, 2011:532-42.
- ¹⁴ Lusk S, Gillespie B, Hagerty BM, Ziemba RA. Acute effects of noise on blood pressure and heart rate. *Arch Environ Health* 2004;59:392-99.
- ¹⁵ Van Kempen EE, Kruize H, Boshuizen HC, Ameling CB, Staatsen BA, de Hollander AE. The association between noise exposure and blood pressure in ischemic heart disease: a meta-analysis. *Environ Health Perspect* 2002;110:307-17.

-
- ¹⁶ Tomei G, Fioravanti M, Cerratti D, et al. Occupational exposure to noise and the cardiovascular system: a meta-analysis. *Sci Total Environ* 2010;408:681-89.
- ¹⁷ Davies H, van Kamp IV. Noise and cardiovascular disease: a review of the literature 2008-2011. *Noise Health* 2012;14:287-91.
- ¹⁸ Huss A, Spoerri A, Egger M, Roosli M, for the Swiss National Cohort Study Group. Aircraft noise, air pollution, and mortality from myocardial infarction. *Epidemiology* 2010;21:829-36.
- ¹⁹ Sorensen M, Hvidberg M, Andersen ZJ, et al. Road traffic noise and stroke: a prospective cohort study. *Eur Heart J* 2011;32:737-44.
- ²⁰ Gan WQ, Davies HW, Koehoorn M, Brauer M. Association of long-term exposure to community noise and traffic-related air pollution with coronary heart disease mortality. *Am J Epidemiol* 2012;175:898-906.
- ²¹ Sorensen M, Andersen ZJ, Nordsborg RB, et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS One* 2012;7:e39283.
- ²² Kujawa SG, Liberman MC. Acceleration of age-related hearing loss by early noise exposure: evidence of a misspent youth. *J Neurosci* 2006;26:2115-23.
- ²³ Campo P, Venet T, Rumeau C, et al. Impact of noise or styrene exposure on the kinetics of presbycusis. *Hear Res* 2011;280:122-32.
- ²⁴ Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after “temporary” noise-induced hearing loss. *J Neurosci* 2009;29:14077-85.
- ²⁵ Sliwinska-Kowalska M. Contribution of genetic factors to noise-induced hearing loss. In: Griefahn B, ed. 10th international congress on noise as a public health problem of the international commission on biological effects of noise, London, UK, 2011. <http://www.icben.org/proceedings.html> (accessed April 25, 2014).
- ²⁶ Sliwinska-Kowalska M, Davis A. Noise-induced hearing loss. *Noise Health* 2012;14:274-80.
- ²⁷ Gelfand, SA. The effects of noise and industrial audiology. In: *Essentials of Audiology*, New York: Thieme, 2001. 33;501-541.
- ²⁸ U.S. Department of Labor (USDL), Occupational Safety and Health Administration (OSHA). Occupational noise exposure: hearing conservation amendment; final rule. *Fed Regist* 1983;48:9738-85.
- ²⁹ Verbeek JH, Kateman E, Morata TC, Dreschler WA, Mischke C. Interventions to prevent occupational noise-induced hearing loss. *Cochrane Database Syst Rev* 2012.
- * A thorough evidence-based review of the efficacy and utility of prevention programs for occupational hearing loss.
- ³⁰ Hong O. Hearing loss among operating engineers in American construction industry. *Int Arch Occup Environ Health* 2005;78:565-574.
- ³¹ Brink LI, Talbott EO, Burks JA, Palmer CV. Changes over time in audiometric thresholds in a group of automobile stamping and assembly workers with a hearing conservation program. *Am Ind Hyg Assoc J* 2002;63:482-7.
- ³² Hessel PA. Hearing loss among construction workers in Edmonton, Alberta, Canada. *J Occup Environ Med* 2000;42:57-63.
- ³³ Neitzel R, Seixas N. The effectiveness of hearing protection among construction workers. *J Occup Environ Hyg* 2005;2:227-38.

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- ³⁴ Hong O, Ronis DL, Lusk SL, Kee GS. Efficacy of a computer-based hearing test and tailored hearing protection intervention. *Int J Behav Med* 2006;13:304-14.
- ³⁵ Kerr MJ, Savik K, Mosen KA, Lusk SL. Effectiveness of computer-based tailoring versus targeting to promote use of hearing protection. *Can J Nurs Res* 2007;39:80-97.
- ³⁶ McCullagh M, Lusk SL, Ronis DL. Factors influencing use of hearing protection among farmers: a test of the pender health promotion model. *Nurs Res* 2002;51:33-39.
- ³⁷ Hong O, Chin DL, Ronis DL. Predictors of hearing protection behavior among firefighters in the United States. *Int J Behav Med* 2013;20:121-30.
- ³⁸ Depczynski J, Challinor K, Fragar L. Changes in the hearing status and noise injury prevention practices of Australian farmers from 1994 to 2008. *J Agromed* 2011;16:127-42.
- ³⁹ Groenewold MR, Masterson EA, Themann CL, Davis RR. Do hearing protectors protect hearing? *Am J Ind Med* 2014. Epub ahead of print.
- ⁴⁰ Le Prell CG, Dolan DF, Bennett DC, Boxer PA. Nutrient plasma levels achieved during treatment that reduces noise-induced hearing loss. *Transl Res* 2011;158:54-70.
- ⁴¹ Le Prell CG, Spankovich C. Healthy diets and dietary supplements: recent changes in how we might think about hearing conservation. In: Griefahn B, ed. 10th International Congress on Noise as a Public Health Problem of the International Commission on Biological Effects of Noise, London, UK, 2011. <http://www.icben.org/proceedings.html> (accessed April 25, 2014).
- ⁴² Abaamrane L, Raffin F, Gal M, Avan P, Sendowski I. Long-term administration of magnesium after acoustic trauma caused by gunshot noise in guinea pigs. *Hear Res* 2009;247:137-45.
- ⁴³ Kopke RD, Jackson RL, Coleman JK, Liu J, Bielefeld EC, Balough BJ. NAC for noise: From the bench top to the clinic. *Hear Res* 2007;226:114-25.
- ⁴⁴ Kramer S, Dreisbach L, Lockwood J, et al. Efficacy of the antioxidant N-acetylcysteine (NAC) in protecting ears exposed to loud music. *J Am Acad Audiol* 2006;17:265-78.
- ⁴⁵ Lin CY, Wu JL, Shih TS, et al. N-acetylcysteine against noise-induced temporary threshold shift in male workers. *Hear Res* 2010;269:42-7.
- ⁴⁶ Samson J, Wiktorek-Smagur A, Politanski P et al. Noise-induced time-dependent changes in oxidative stress in the mouse cochlea and attenuation by d-methionine. *Neuroscience* 2008;152:146-50.
- ⁴⁷ Campbell K, Claussen A, Meech R, Verhulst S, Fox D, Hughes L. d-methionine (d-met) significantly rescues noise-induced hearing loss: Timing studies. *Hear Res* 2011;282:138-44.