

Noise Interactions at Work, Leisure and Home

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Introduction

Presently, health research is in large characterized by the study of single agents as if they occurred alone in the work environment. Ninety-five percent of the resources in toxicology are committed to single chemical investigations. Exposure standards and recommendations are based on the evidence provided by these focused studies under the assumption that the health effects of combined exposure to two substances can be predicted by adding the adverse effects resulting from exposures to individual substances. A wealth of information was generated through the single-agent approach and serious risks have been identified and controlled because of that. Nevertheless, the limitations of this approach are becoming more obvious, as revealed by recent investigations on mixed exposures. The idea to study exposures in relation to each other is not at all new, but it is likely that it received limited attention because of its complexity. Advances in research and statistical methods, however, have facilitated a recent increase in the number of scientific studies on combinations. The objective of this paper is to stimulate researchers to consider the way that their research may account for possible interactions.

Studying Interactions

Results that have raised serious concerns come from reports on synergism (when the observed effects are greater than the sum of individual effects). Synergism has been reported from combined exposures to substances of widespread use. Three

scenarios of this enhanced response include: a) cosynergism, when two agents enhance the toxicity of each other, b) potentiation, when one agent affects the toxicity of the other with no toxicity itself and, c) coalism is the term for the situation when two agents with no observable toxicity cause, in combination, a toxic effect. Furthermore, there are situations in which the combination of exposure reduces the effects to less than the expected from the single exposures (antagonism).

Investigations that used a more global approach of studying risk factors offer examples that illustrate its importance (Niosh, 2005; Robinson & MacDonnell, 2004). The action of individual components of chemical mixtures can be altered by the presence of other substances. Enhanced effects have been reported in many of the body systems, including nervous, sensory, respiratory and reproductive systems. Potentiation effects from solvent mixtures have been reported involving the liver, kidney and nervous system. Solvents mixtures have also been associated with additive and antagonistic effects on the nervous system. Combinations of hormone-disrupting chemicals are much more powerful than any of the individual chemicals by themselves, even at low-level exposures. One of the chemicals of the specific mixture, which has no ability to disrupt hormones by itself, greatly enhanced the ability of other chemicals to disrupt hormones. Tobacco smoke and asbestos can enhance each other's effects on the lungs. Some of the questions related to the Gulf War Syndrome were only answered by studies of combinations of chemicals, which when acting alone did not cause an effect.

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Studying Physical-Physical or Physical-Chemical Mixed Exposures

Physical agents such as extreme temperatures, noise, vibration and radiation also may interact among themselves and with chemicals in causing various effects. Extreme temperatures and whole body vibration may enhance the effects of noise in causing a hearing loss. The uptake of several chemicals increases with elevated temperatures. Exposure to solvents can enhance the effects of noise on hearing, beyond what would be predicted from their individual effects. Similarly, studies have shown that exposure to radiation enhances the toxicity of certain chemicals in causing tumors and causing developmental effects.

Noise interactions at home

Home is likely to be the place where noise disturbs the most. Noise from cars, trucks, trains, buses, motorcycles, and airplanes are the most common noise sources to affect large populations at their homes. Transportation noise increases with growing populations and urban sprawl (U.S. Environmental Protection Agency, 1981). Other noise sources at home include appliances and lawn care equipment, power tools, pets, neighbors, toys, video games and several electronic music gadgets. The effects from noise exposures at home are mainly non-auditory, but a few household items, such as noisy toys, garden equipment and loud stereos could affect hearing.

As described by Babisch (2002), the noise-stress hypothesis is well understood. Acute and chronic noise experiments have consistently shown changes in the production of stress hormones. These changes affect the body's metabolism. The consequences of such changes could be multiple, however, not all changes necessarily imply in a dysfunction or disorder. Cardiovascular disorders are the most-studied of the non-auditory noise effects because of the seriousness of the condition and the number of people affected by heart disease (Babisch et al., 1993). This outcome is a good example for the need to investigate interactions, since the factors that explain heart disease are known to be numerous. In a recent study multiple factors and the incidence of ischemic heart disease in 3950 middle-aged men were examined (Babisch et al., 2003). The participants' noise exposure history was

gathered along with their medical history. The participants who were highly annoyed or disturbed by noise but free of any chronic disease at the beginning of the follow up intervention had significant odds ratios between 1.7 and 3.0 for heart disease. In the subgroup with chronic diseases, no such noise effects were seen.

Another emerging area for studies on noise interactions at home are respiratory tract diseases. Generally urban environments with high noise pollution also have high air pollution. During sleep, noise signals that are associated with danger (i.e. truck noise) have the potential to trigger stress reactions even if the noise level is low. Usually studies on the effects of road traffic pollution have attributed the effects of combined air pollution and noise pollution solely to one or the other pollutant, without recognizing the relative contribution of each or the interaction of both. Recently has it been shown that in areas of high noise pollution there is an increased incidence of asthma and bronchitis suggesting an interaction between noise and air pollution induced effects (Ising et al., 2003; Ising et al., 2004). Similarly, skin diseases have been studied in association with traffic air and noise pollution (Ising et al., 2003). In a blind interview study, pediatricians' diagnoses of 400 children were analyzed together with their parents' answers on the density of road traffic on their street and several confounding factors. Multiple regression analyses resulted in relative risks of asthma, chronic bronchitis and neurodermitis, which increased significantly with increasing traffic load. A comparison with the literature on these outcomes when looking at air pollution alone showed that night traffic noise might have a modifier effect on the pathogenesis of the respiratory and skin conditions examined.

Noise is most annoying at the times when people expect to rest or sleep. Not only noise can interrupt or delay sleep, but also affect the quality of sleep, for instance, by causing shifts from deeper to lighter sleep stages (Maschke & Hecht, 2004; Maschke et al., 2004; Spreng, 2004). Sleep disorders can have negative health effects when it becomes a chronic problem. Psychological and learning disorders have also been associated with noise exposure, but not many investigations have carefully controlled the contribution of multiple interacting factors (Stansfeld & Matheson, 2003; Matsui et al., 2004; Kjellberg, 1990).

Research is still needed for a better understanding of these effects. Special attention should be given to those who suffer from circulatory and respiratory illnesses and vulnerable groups including children, the elderly, and others who do not have the means to remove themselves from the situation.

Noise interactions during leisure activities

Some entertainment and sports activities are becoming increasingly louder. Arcade game areas in shopping malls, boom boxes, discos, surround-sound movie theaters, loud broadcast at sports stadiums full of thousands of noisy fans are a few examples. People seek these exposures. The effects of such exposures have not been extensively studied, but it is conceivable that they are both auditory and non-auditory. Until now, most publications are on auditory effects; some of them only report the noise levels during leisure activities and discuss potential risks. Publications on people who hunt or practice target shooting are numerous, but they focus exclusively on noise since the exposure is so damaging by itself, that studying interactions is not needed. Comparatively, a smaller number of publications exist on studies conducted with musicians, or on those involved in motor sports.

Evidence from studies with professional musicians indicates a range of responses from no effect to hearing impairment (Ostri et al., 1989; Axelsson et al., 1995; Kahari et al., 2001). The variables that have been hypothesized to play a role in this effect include the characteristics of the exposure (duration, levels, location of the musicians, room acoustics), consumption of drugs, alcohol and tobacco, and attitude or stress level, but no investigation aimed at examining these interactions.

Research conducted on noise from motor sports (motorcycle or car racing, monster truck or motorcross shows, and snowmobile use), reports noise levels and discusses the potential hearing risk, but no hearing database exist on these populations (Morley et al., 1999; Alexander, 1996; Bess & Poyner, 1972; Campen et al., 2005; Gwin et al., 2005). The variables that have been hypothesized to play a role in a hearing effect include the characteristics of the exposure and exposure to vibration and chemicals, such as carbon monoxide.

Noise interactions at work

Work is the setting where noise exposures can be higher and more consistent than in any other setting. Noise exposure at work can interact with other factors in causing both auditory and non-auditory effects. The most studied are the auditory effects. Studies have been conducted on several endogenous and exogenous factors that can interact with noise in causing hearing loss. Endogenous factors are those inherent to the individual, and factors such as general health indicators, genetics, age, gender, race and body temperature have been examined. Exogenous factors include smoking, vibration, ambient temperature, use of medicinal drugs and exposure to chemicals.

Noise exposed workers with vibration white finger syndrome from hand-arm vibration have greater hearing loss than those exposed to a similar noise alone (Seidel et al., 1988; Seidel et al., 1992; Pyykkö et al., 1981; Pyykkö et al., 1986; Iki et al., 1986; Miyakita et al., 1987; Hamernik et al., 1989). A possible reason for more pronounced noise-induced hearing loss in subjects with vibration white finger syndrome is that vibration might operate in both of these disorders through a common mechanism—that is, producing a vasoconstriction in both cochlear and digital blood vessels as a result of sympathetic nervous system activity (Pyykkö et al., 1981). The relationship between the extent of vibration effect and hearing loss is not clear at present and whether whole body vibration enhances risk for hearing loss. The new European Community (EC) directive on noise (2003/10 EC noise) requires that the interaction between noise and vibration be taken into account in the risk assessment of exposed populations. It is still important to examine whether current exposure limits for both vibration and noise protect the individual from hearing loss.

In the past two decades progress has been considerable towards understanding the effects of certain environmental and occupational chemicals on the auditory system and their interaction with noise (Fechter et al., 1987; Liu & Fechter, 1995; Chen et al., 1999; Loquet et al., 1999). Before the 1980's no research program had systematically focused on chemical-induced hearing loss and only isolated studies reported such effects. This scenario started changing following reports from groups dedicated to investigations of the neurotoxic properties of chemicals (Pryor et al., 1983). Chemicals investi-

gated as potential ototoxicants are heavy metals (Abbate et al., 1995; Lille et al., 1988; Osman et al., 1999), pesticides (Teixeira et al., 2002), solvents (Morata et al., 1993) and asphyxiants (Fechter et al., 2000). These are substances with diverse chemical structures, which suggest a number of targets for injury within the auditory system and an array of possible underlying mechanisms (Fechter, 1995). Recently, metabolic processes involving oxidative stress have been shown to contribute to noise-induced hearing loss, or hearing loss from chemicals. The generation of reactive oxygen species (ROS) or free radicals has been associated with cellular injury in different organ systems. Free radicals produce cell damage by binding to macromolecules and by producing lipid peroxidation. It is considered a basic mechanism of toxicity, and is thought to be part of the mechanism underlying acquired hearing losses. Cells have two recognized defenses for limiting the damage induced by radicals; they are enzymes and molecules called antioxidants. Enzymes include glutathione (GSH), superoxide dismutase (SOD), catalase, and antioxidants include ascorbate and tocopherols (vitamin E), and others. Portions of the cochlea that are richer in glutathione are more resistant to different injuries (Usami et al., 1996) and that several substances that promote ROS scavenging can be administered to the cochlea and minimize the effects of ototraumatic exposures that cause oxidative stress (Kopke et al., 2000; Kopke et al., 2002; Lautermann et al., 1997; Rao & Fechter, 2000). The antioxidant glutathione enzyme (GST) which is found in the mammalian cochlea may play a protective role in noise-exposed workers against hair cell damage due to noise or aging (Rabinowitz et al., 2002). This is still an emerging area of research and broadly applicable methods of protecting hearing are being investigated.

It has been shown that if solvent exposures occur in sufficiently high concentrations, hearing may be affected despite the lack of occupational exposure to noise (Lataye & Campo, 1997; Johnson et al., 1988; Campo et al., 1999; Morata et al., 1997; Morata et al., 2002). Others have indicated that chemicals that do not affect the auditory system by themselves may potentiate the effect of noise exposures. Clinical studies have suggested that the effects of certain chemicals are not limited to the cochlea, but can affect more central portions of the auditory (Ödkvist et al., 1982; Möller et al., 1990).

Field studies in Sweden, Denmark, Brazil, Colombia, Taiwan and Poland have shown that hearing losses are more common in work settings where chemical exposures (Morata et al., 2002; Jacobsen et al., 1993; Morata, 1989; Morata et al., 1997; Chang et al., 2003; Sliwińska-Kowalska et al., 2001; Sliwinska-Kowalska et al., 2003). Hearing losses from ototoxicity are moderate to severe, as is the case with noise-induced hearing loss. The audiometric high-frequency “notch” is often present following long-term exposures, although some reports indicate that a wider range of audiometric frequencies are affected when compared to the range of frequencies affected by noise.

The new European Community (EC) directive on noise (2003/10 EC noise) requires that the interaction between noise and ototoxic chemicals be taken into account in the risk assessment of exposed populations. It is still unclear how this new Directive will be implemented in the field. http://europa.eu.int/eur-lex/pri/pt/oj/dat/2003/1_042/1_04220030215pt00380044.pdf

Since 1998, the American Conference of Governmental Industrial Hygienists (ACGIH) in its *Threshold Limited Values and Biological Exposure Indices* (TLVs® and BEIs®) publication, included a note in its Noise Section which states: “In settings where exposure to toluene, lead, manganese or n-butyl alcohol occurs, periodic audiograms are advised and should be carefully reviewed.” Since 1998, the US Army started requiring consideration of ototoxic chemical exposures for hearing conservation program inclusion, “particularly when in combination with marginal noise” (Dept. of the Army Pamphlet 40-501, ¶ 3-3), upon reviewing its Hearing Conservation Guidelines (Morata, 2003). More recently, the US Army (2003) developed a Fact Sheet on Occupational Ototoxins and Hearing Loss, where it argues that since the exposure threshold for ototoxic effects is not known, audiometric monitoring is necessary to know if the substance is affecting the hearing of exposed workers. This document is available at <http://chppm-www.apgea.army.mil/documents/FACT/51-002-0903.pdf>). The Fact Sheet includes recommendations for yearly audiograms for workers whose airborne exposures (without regard to respiratory protection worn) are at 50% or most stringent criteria for recommended limits, either of the Occupational Safety and Health Administration Permissible Exposure Limit or American Conference of Indus-



trial Hygienist Threshold Limit Value) to toluene, xylene, n-hexane, organic tin, carbon disulfide, mercury, organic lead, hydrogen cyanide, diesel fuel, kerosene fuel, jet fuel, JP-8 fuel, organophosphate pesticides, or chemical warfare nerve agents, regardless of the noise level. The 50% cut-off, while somewhat arbitrary, will ensure the collection of data from sub-Occupational Exposure Limit exposures. If there are dermal exposures to these agents and such exposures may result in a systemic dose equivalent to 50% or more of the Occupational Exposure Limit, yearly audiograms were also recommended. If a worker is currently participating in a hearing conservation program due to excessive noise, the reviewers of the audiometric data were recommended to be alert to possible additive, potentiating, or synergistic effects between the exposure to noise and the chemical substance and, if necessary, suggest reducing the exposure to one or both.

Tinnitus

Tinnitus is also a serious outcome from noise exposure, and its interactions. It has been defined as the sensation of noise in the absence of acoustic stimuli. It is described usually as “ringing in the ears.” Tinnitus can be intermittent, from minutes to a few hours, or continuous. It can be a minor annoyance or a serious and nearly intolerable condition.

Tinnitus is not a disease, but rather a condition often associated with many forms of hearing loss. Tinnitus creates considerable distress, and in severe cases it may interfere with daily activities and with sleep. There are no definite answers to explain what is happening within the auditory system to cause the tinnitus, and this uncertainty contributes to the distress caused by this disorder.

Noise exposure is one of the most cited associations with tinnitus. Several other factors can be associated with tinnitus including more than 200 medications as well as dietary, nutritional, hormonal, immunological, and stress factors (www.ata.org) (Ceranik et al., 1998; Kaltenbach et al., 2001).

Conclusions

The Challenge Of Mixed Exposures And Action Been Taken

The examples above not only illustrate the importance of broadening research objectives, design and modeling efforts, but also reveal how challenging this comprehensive approach is. Regarding commercially available chemicals already in the market, for instance, there are just too many (and hundreds more are released each year), to realistically expect that research institutes can test the toxicity of each of them, or each of the possible mixtures. Observations that the same chemical mixture may have both a synergistic and an antagonist effect in different target organs, and that variations in exposure parameters may also cause this opposite patterns of interaction, complicate things further. Nevertheless, Niosh among other research agencies, have identified the investigation of mixed exposures as a priority, which will impact research planning and in future standard setting.¹

The strategies envisioned by researchers to address mixed exposure include:

- surveillance and assessment of potential interactions, which will allow for making decisions on strategic directions and the priorities of mixed exposures research;
- evaluating mechanisms of interaction, which will provide a rational basis for extrapolation of toxicologic information across different mixtures, dose levels, exposure parameters and routes.
- conducting multidisciplinary epidemiologic investigations with careful mixed exposure assessment, preferably including personal monitoring and biomarkers.
- evaluating, in addition to occupational environmental and organizational factors, non-occupational risk factors and individual variability in response to both;
- improving communication between scientists and partners about exposure and risks.

In the meanwhile, what steps can workers, employers and occupational health professionals take? While research continues, Niosh advises workers, employers and occupational health professionals to consider measures to minimize exposures to physical, chemicals and biological agents and improve work organization (Niosh, 2005).

When evaluating risks from a task or process, NIOSH recommends the involved to be alert to the work environment as a whole. When information on the specific combination is not available, it is advisable to search not only for information available on hazards from the individual agents present (on whether they have common target organs), but also from similar combinations.

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