Abstracts

Poster: 0175

Predicting Potentiation of Noise-Induced Hearing Loss by Chemicals: Oxidative Stress as a Mechanism of Hearing Loss

LD Fechter (1) presenting, B Pouyatos (1)

Loma Linda VA Medical Center, Loma Linda, CA, United States (1)

Laboratory animal studies as well as occupational epidemiological studies demonstrate that a broad range of chemical agents can increase the vulnerability of subjects to noise exposure. Consequently, under conditions of workplace exposure to specific chemicals, permanent hearing loss may occur even at permissible noise exposure levels. Predicting which chemicals can promote noise-induced hearing loss is essential both in establishing safe work conditions and developing potential treatment modalities for accidentally exposed workers. The objectives of this research are to determine the biological basis for the interaction between chemicals and noise in order to facilitate prediction of chemical agents that can promote noise-induced hearing loss. Specific chemical agents were selected based both upon their common use in occupational settings and their potential to produce oxidative stress. The specific hypothesis tested was that chemicals, which interfere with normal antioxidant pathways, would render the inner ear particularly vulnerable to noise. A two process model is envisaged: (a) initiation of reactive oxygen species by moderate noise exposure and (b) promotion of oxidative stress through disrupting intrinsic antioxidant defenses. This hypothesis has been tested using carbon monoxide, hydrogen cyanide, and acrylonitrile (ACN); widely used industrial chemicals with clear pro-oxidant potential. Carbon monoxide is the most common air pollutant and is a key contaminant in work settings that involve combustion including transportation, firefighting, and smelting operations. Cyanide is used in metallurgic occupations and as a chemical intermediary. Acrylonitrile is one of the 50 most commonly used chemicals in the United States. ACN is used to make nylon and acrylic fibers, plastics, nitrile rubber, and as a chemical intermediary. In our studies, laboratory rats were exposed to noise alone, selected chemical agents alone (carbon monoxide, hydrogen cyanide, and acrylonitrile), mixed exposure to chemicals and noise, and no experimental treatment (control). In some instances drugs with known antioxidant potential were included to evaluate their protective effects. Hearing was assessed repeatedly using a non-invasive, objective method (distortion product otoacoustic emissions) that is frequently used in testing the function of the human inner ear. Subsequent to measuring auditory thresholds for permanent hearing loss 4-5 weeks following exposure, cochleae were harvested to allow histological evaluation of hair cell loss. The data gleaned from multiple experiments demonstrate that chemical agents capable of interfering with intrinsic antioxidant pathways do, in fact, increase noise-induced hearing loss and produce extensive outer hair cell

death. Such interactions can be seen even when noise exposure by itself produces no hearing loss and when the noise exposure used is at a level permitted under OSHA regulations. Moreover, drugs that can reduce free radicals are protective against the promotion of noise-induced hearing loss by chemicals. These findings advance the fields of hearing research and noise control by establishing the importance of oxidative stress as a mechanism of hearing loss and in identifying a class of chemicals with potential to enhance vulnerability to noise. They identify potential occupational exposures with potential to promote the effects of noise and focus future epidemiological investigations. (Supported in part by grant # OH-03481).

NORA Symposium 2006

Research Makes a Difference

April 18-20, 2006

L'Enfant Plaza Hotel

Washington, DC

NORA

CDC Workplace Safety and Health

NIOSH

National Safety Council