Archives of Environmental Health: An International Journal

ISSN: 0003-9896 (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/vzeh20

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To cite this article: Thais C. Morata , Derek E. Dunn & W. Karl Sieber (1994) Occupational Exposure to Noise and Ototoxic Organic Solvents, Archives of Environmental Health: An International Journal, 49:5, 359-365, DOI: 10.1080/00039896.1994.9954988

To link to this article: https://doi.org/10.1080/00039896.1994.9954988

Published online: 03 Aug 2010.

Occupational Exposure to Noise and Ototoxic Organic Solvents

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ABSTRACT. The objectives of this study were to review the literature on the effects of occupational exposure to organic solvents on the auditory system and to identify work settings in which exposure to these agents and to noise might occur. The criteria for selecting the chemicals were (a) evidence available that indicated that the chemicals may affect the auditory system and enhance noise effects, and (b) the ubiquity of their use. References to ototoxicity were noted for three proven neurotoxicants, i.e., carbon disulfide, toluene, and trichloroethylene, and for two probable human neurotoxicants—styrene and xylene. The percentages of workers (estimated by NIOSH National Occupational Exposure Survey) exposed to these solvents in each economic sector are shown. Work settings are identified where multiple exposures occur to solvents and noise. The need for future research is discussed.

ONE of the more sinister aspects of ototoxins is that they may interact when administered simultaneously. It has become increasingly clear that the effects of many drugs or agents, when given concurrently, are not necessarily predicted on the basis of knowledge of their individual effects.[1] In such instances, the damage incurred by the agents acting together may exceed the simple summation of the damage each produces alone.[2,3] Two of the most common hazards that occur simultaneously in many work environments are noise and organic solvents. There is documentation that noise interacts synergistically with various drugs and chemicals.[4–11] There has been a growing interest in the effects this simultaneous exposure might have on hearing.[12–19] This research had two objectives: (1) to review the literature pertaining to the effects of organic solvents on the auditory system, and (2) to identify the industries in the United States where simultaneous exposure to noise and these potentially ototoxic chemicals might occur. This survey was designed to assist with planning epidemiologic studies of the effects of solvent exposure on workers' hearing.

Ototraumatic interaction between noise and solvents

Whereas large doses of many solvents are capable of inducing an acute, reversible narcotic state, few solvents can unequivocally induce chronic, long-lasting, or irreversible changes in nervous system structure and/or function such as peripheral neuropathies and toxic encephalopathies.[20,21] Criteria for classifying solvents as neurotoxic have been proposed: "Three questions must be answered affirmatively before a solvent can be accepted as a human neurotoxicant. (1) Does the substance or mixture produce a consistent pattern of neurological dysfunction in humans? (2) Can this entity be induced in animals under comparable exposure conditions? (3) Are there reproducible lesions in the nervous system or special sense organs of exposed humans and/or animals, and do these abnormalities account satisfactorily for the neurobehavioral dysfunction?"[21] A review of individual solvents yielded a list of both proven human neurotoxicants and probable human neurotoxicants. Included in the list of proven or probable neurotoxicants are five solvents (toluene, xylene, styrene, trichloroethylene, and carbon disulfide) that have been shown to affect the auditory system.

In a review paper published in 1984 in which five occupational studies and four cases reports were discussed briefly, an ototraumatic interaction between noise and organic solvents was suggested and its biological plausibility discussed.[12] It was observed that the incidence of sensorineural hearing loss was higher than expected in noise-exposed workers who were also exposed to solvents. Given that organic solvents are known for their neurotoxic effects to both central and peripheral nervous systems, it was argued that solvents could injure the sensory cells and peripheral endings of the cochlea. It was hypothesized that, because solvent-related effects have been found in the brain, a retrocochlear influence on hearing could also be expected.

In a 20-y longitudinal study of hearing sensitivity in 319 employees from different sectors of industry, a remarkably large proportion of the workers in the chemical sector showed pronounced hearing loss (23%), compared with groups from nonchemical environments (5–8%).[13] This effect was found despite the lower noise levels in the chemical sector (80–90 dBA) when compared with other divisions (95–100 dBA). Thus, exposure to industrial solvents (not identified in the article) was implicated as an additional causative factor for those hearing losses.

The hearing of workers exposed to mixtures of aliphatic and aromatic solvents for periods ranging from 9 to 40 y has been examined.[14] Workers' scores on speech audiometry were lower than would be predicted by their pure tone audiogram (38–64% incidence of abnormality) and their cortical responses to frequency glides were abnormal (50–64% incidence of abnormality); however, no effects were observed on the auditory brainstem responses (0–9% incidence of abnormality). Therefore, it was concluded that solvents do not seem to cause measurable cochlear damage in humans, but the auditory system might be vulnerable at cortical levels, as indicated by the speech discrimination test and the cortical responses.

The following sections will review research relating to the ototoxic effects of the five aforementioned toxicants, and the industries in which exposure to these toxicants might occur in combination with noise will be identified. The industries were identified, using data acquired during the National Occupational Exposure Survey (NOES) conducted by the National Institute for Occupational Safety and Health between 1981 and 1983.[22]

Five organic solvents reported to be ototoxic

Carbon disulfide (CS_2). Carbon disulfide can cause both acute and chronic forms of poisoning. The effects of carbon disulfide are nonspecific, making individual diagnosis a matter of probability, based on the confirmation of exposure, the presence of signs and symptoms, and the exclusion of other diseases.[23]

An investigation was conducted to determine the contribution that audiologic and otoneurologic tests could have to the identification of carbon disulfide intoxication.[24] Workers ($n = 259$) exposed to varying concentrations of carbon disulfide were tested. The level of exposure for those workers varied from 100–900 mg/m^3 immediately after World War II to values of 30–35 mg/m^3 in later years. The auditory and otoneurologic test results showed an increased incidence of pathological vestibular symptoms and of sensorineural hearing loss, compared with a group consisting of 60 textile workers with no carbon disulfide exposure and the same noise level exposure (84–88 dBA).

Hearing and balance functions were measured in workers from a viscose rayon factory who experienced simultaneous exposure to noise and carbon disulfide.[16] Continuous noise levels ranged from 86–89 dBA. Carbon disulfide atmospheric concentration ranged from 88–92 mg/m^3. The subjects were interviewed and underwent pure tone audiometry and vestibular screening. It was found that not only was the percentage of hearing loss higher among the workers exposed to both agents (60% versus 50%), but also that hearing losses were more serious and had an earlier onset than if the only environmental factor had been noise exposure. Chi-square analysis indicated that exposure time to carbon disulfide and noise was associated statistically with the occurrence of hearing losses ($p < .01$).

In a study of 115 workers carried out in a viscose rayon factory in Japan, workers exposed to CS_2 ($n = 75$) were divided into three groups according to length of exposure and compared with unexposed controls selected from workers in a nylon filament factory ($n = 40$).[25] Brainstem auditory evoked responses (BAER) records were analyzed, and the results suggested that chronic exposure to CS_2 in humans has an effect on the auditory pathways in the brainstem.

Some animal experiments have shown an effect of carbon disulfide exposure on BAER.[26,27] These data revealed a prolongation of the interpeak latency and indicated a retrocochlear pattern of hearing loss. These results are consistent with the pattern of increased interpeak latency observed in humans.[24] However, another experiment in which rats were exposed to carbon disulfide revealed no change in auditory function.[28]

Toluene ($C_6H_5CH_3$). Disturbance of the equilibrium system is considered an early manifestation of the neurotoxic action of industrial organic solvents and has been associated with toluene exposure.[29,30] Limited published information exists that documents an association between toluene exposure and hearing impairments, however.

Data on toluene effects on hearing originate mainly from animal studies and case reports on toluene abusers. In the studies that focused on the voluntary inhalation of toluene, dramatic hearing loss originating from the central auditory pathways has been reported.[31–34] Although the mechanisms for the lesions are not understood fully, it is accepted that toluene abuse impairs hearing function in humans.

Several studies report high frequency hearing loss and accompanying cochlear changes in weanling rats exposed to toluene. Unfortunately, the noise levels generated by the exposure chambers were not always reported, and the role played by this confounder was not explored. Hearing losses were measured by behavioral methods and confirmed by electrophysiologic testing.[35,36] Morphologic examination of the toluene-exposed rats' cochleas revealed loss of or damage to hair cells in the basal portion of the cochlea. The results indicated also that young rats are more severely affected than older rats.[37]

Toluene has been injected subcutaneously in rats to observe whether noise emanating from the inhalation system was a major contributing factor to hearing loss and to determine whether a noninhalation route would also cause irreversible hearing loss.[38] The results demonstrated that the noise associated with inhalation exposure was not a necessary condition for causing the hearing loss. Thus, it has been argued that the simultaneous exposure to noise and toluene may result in increased susceptibility to the effects of both agents.

Rats have been exposed to toluene alone, to noise alone, or to toluene followed by noise and their auditory functions tested.[15] Toluene alone and noise alone caused a considerable decrease in the auditory sensitivity of rats, particularly at high frequencies ($p < .001$ in both conditions). The most salient feature was that the decrease in auditory sensitivity of rats exposed to toluene followed by noise was greater than the sum ($p < .0001$) of the effects of toluene and noise alone (i.e., a synergistic effect).

Similar findings were reported in an experiment in which toluene was administrated orally to rats.[39] Daily background noise exposure levels were monitored and found to be below 60 dB (SPL). BAERs were recorded prior to and after dosing, and morphologic examination of the cochleas was performed. The authors argued that the ototoxic effects of toluene contrast with those of other known ototoxicants in terms of the position of hair cell lesion in the Organ of Corti and in the pattern of hair cell loss.

Dizziness, lightheadedness, ringing in the ears, and loss of hearing were some of the dose-related subjective symptoms reported by 193 nonsmoking and nondrinking toluene-exposed female workers who were mostly in their 20s.[40] The dose effect was assessed by dividing the workers into four exposure populations. The prevalence of total complaints per person correlated significantly with the time-weighted average intensity of exposure to toluene for each individual.

In a study of the effects of toluene on the balance function of rotogravure printers ($n = 53$), hearing loss was observed in 11 of the 15 workers that had vestibular impairment.[41] A more recent study of 190 workers was carried out with rotogravure printing workers.[42] The hearing and balance functions of a group of printers exposed simultaneously to noise and toluene were compared with (a) a group of printers exposed to noise alone, (b) a group exposed to a solvent mixture, and (c) a group neither exposed to noise nor toluene. The adjusted relative risk estimates for hearing loss were 4 times greater (95% confidence interval [CI] = 1.4–12.2) for the noise group; 11 times greater (95% CI = 4.1–28.9) for the noise and toluene group; and 5 times greater (95 CI = 1.4–17.5) for the solvents group. Acoustic reflex measurements suggested that hearing losses found in the group exposed to both agents might result from lesions in the central auditory system.

Styrene ($C_6H_6CH{=}CH_2$) and Xylene ($C_6H_4(CH_3)_2$). Having identified the auditory dysfunction caused by exposure to toluene, some investigators examined other solvents that also might affect hearing.[43] Mixed xylene (2)ortho-, *meta*-, and *para*-xylene) and styrene were selected as possible ototoxic agents, primarily because of their structural similarity to toluene. Rats were exposed to either mixed xylene or styrene. Both xylene and styrene caused marked hearing loss in the experimental animals. Moreover, both solvents appeared to be more potent ototoxicants than toluene.

Workers exposed to low levels of styrene did not appear to have increased age-dependent hearing loss at high frequencies. However, a comparison within the group of exposed workers between the least exposed and the most exposed revealed a statistically significant difference in hearing thresholds at high frequencies.[44] Routine hearing tests of workers exposed to styrene in a plastic boat plant did not indicate hearing losses resulting from causes other than exposure to noise. However, 7 of 18 workers displayed abnormal results in central auditory system testing.[45]

Trichloroethylene ($ClCH{=}CCl_2$). Individual medical case histories implicate trichloroethylene (TCE) as a toxicant with possible ototoxic and vestibulotoxic properties. A case was reported in which a 33-y-old male metal degreaser who used TCE suffered several attacks of vertigo.[46] Another case was reported on a 54-y-old male dry cleaner with 10 y of occupational inhalation of TCE.[47] The worker voiced many health complaints and suffered from high frequency sensorineural hearing loss. The hearing impairment was considered to be caused by damage to the auditory nerve resulting from to TCE exposure.

Forty workers exposed to TCE were examined, and 26 cases of hearing loss were found.[48] The hearing loss was bilateral, sensorineural, and affected high frequencies. Electronystagmographic investigations indicated lesions of the balance system. It was argued that the detected disorders of hearing and vestibular functions were early signs of decline in the health status of workers exposed to TCE.

When the health status of populations exposed to TCE through contaminated water ($n = 4\ 281$) were investigated in the TCE Subregistry of the National Exposure Registry, a significant increase in reported hearing losses was found for the 0 through 9 y age group (risk ratio = 2.13).[49]

Laboratory studies showed that rats exposed to TCE had a mid- to high frequency hearing loss.[50] Findings from two other experiments were consistent with these results, demonstrating thresholds shifts at 8, 16, and 20 kHz in the exposed group, compared with controls.[51,52]

Assessment of noise and solvent exposure in industry

Information on industries where exposure to ototoxic solvents and noise may occur is important not only to determine the extent of the problem, but also to help direct efforts of future epidemiologic research on the combined effects of these exposures. Useful information is contained within the 1981–1983 National Occupational Exposure Survey (NOES) conducted by the National In-

Table 1.—Percentage of American Workers Exposed to Noise and Potentially Ototoxic Chemicals, 1981–1983 *National Occupational Exposure Survey, by Economic Sector (Manufacturing Not Included)*

Economic sector	SIC	Description	CS_2	TCE	Styrene	Toluene	Xylene	Noise
Agriculture, forestry, fishing	07	Agricultural services	—	X	—	XX	XXX	C,U
Mining	13	Oil and gas extraction	—	—	—	XX	XXX	C,I
Construction	15	General building contractor	—	X	XX	XXX	XXX	C,I
	16	Construction other than building	—	X	X	XX	XX	C,I
	17	Special trade contractor	—	X	X	XXX	XXX	C,I
Transportation, communication, electric, gas, and sanitary services	40	Railroad transportation	—	X	XX	XXXX	X	C
	41	Local and suburban transit and interurban highway passenger transportation	—	—	X	XXX	XXX	C
	42	Motor freight transportation and warehousing	—	X	X	XXX	XX	C,I
	44	Water transportation	—	—	XX	XXXX*	XXX	C
	45	Transportation by air	—	XX	XX	XXXX	XXX	C,I,U
	46	Pipe lines, except natural gas	—	—	—	—	—	C
	47	Transportation services	—	—	—	—	XX	C
	48	Communication	—	XX	X	XX	XX	C
	49	Electric, gas, and sanitary services	—	X	X	XXX	XXX	C,I,U
Wholesale trade	50	Wholesale trade—durable goods	—	X	X	XX	XXX	C,I
	51	Wholesale trade—nondurable goods	—	X	X	XXX	XX	C,I
	55	Automotive dealers and gasoline service stations	—	X	X	XXX	XXX	C,I
Services	72	Personal services	—	X	X	XX	X	C,I,
	73	Business services	X	X	XX	XXX	XXX	C,I,U
	75	Automotive repair, services, and garages	—	XX	XXX*	XXXX	XXXX*	C,I
	76	Miscellaneous repair services	—	X	—	XX	XXX	C,I
	80	Health services	X	X	X	XX	XX	C,I,U
	84	Museums, art galleries, botanical and zoological gardens	XXX*	XXX*	—	XXXX	XXXX	—

Notes: Exposure to solvents: — = no exposure, X = ≤ 2%, XX = 3-10%, XXX = 11-25%, and XXXX = 26–50%. Noise exposure: C = continuous noise, I = impact noise, and U = ultrasonic noise.
*Greatest exposure occurs.

Table 2.—Percentage of American Workers for Each Industry within the Manufacturing Sector Exposed to Noise and Potentially Ototoxic Chemicals, 1981–1983 National Occupational Exposure Survey

Economic sector	SIC	Description	CS_2	TCE	Styrene	Toluene	Xylene	Noise
Manufacturing	20	Food and kindred products	—	X	X	X	X	C,I
	21	Tobacco manufactures	—	X	X	X	X	C
	22	Textile mill products	—	XX	X	XX	XX	C,I
	23	Apparel and other textile	—	X	X	X	XX	C,I,U
	24	Lumber and wood products, except furniture	—	X	X	XXX	XX	C,I,U
	25	Furniture and fixtures	—	X	X	XXX	XXX	C,I,U
	26	Paper and allied products	X	X	X	XXX	XX	C,I
	27	Printing, publishing, and allied industries	—	XX	X	XXX	XXX	C,I,U
	28	Chemicals and allied products	XX	X	XX	XXX	XXX	C,I
	29	Petroleum refining and related industries	XXX	X	—	XXXX	XXXX	C
	30	Rubber and miscellaneous plastics products	X	XX	XX	XXX	XXX	C,I,U
	31	Leather and leather products	—	X	—	XXX	XX	C,I
	32	Stone, clay, glass and concrete products	X	X	XX	XX	XX	C,I,U
	33	Primary metal industries	—	X	X	XX	XX	C,I,U
	34	Fabricated metal products, except machinery and transportation equipment	—	XX	X	XXX	XXX	C,I,U
	35	Machinery, except electrical	X	X	X	XXX	XXX	C,I,U
	36	Electrical and electronic machinery, equipment, and supplies	X	XX	X	XX	XX	C,I,U
	37	Transportation equipment	X	X	XX	XXX	XXX	C,I,U
	38	Measuring, analyzing, and controlling instruments; photographic, medical, and optical goods; watches and clocks	—	XX	X	XXX	XXX	C,I,U
	39	Miscellaneous manufacturing industries	—	X	X	XXX	XX	C,I,U

Notes: Exposure to solvents: — = no exposure, X = ≤ 2%, XX = 3–10%, XXX = 11–25%, and XXXX = 26–50%. Noise exposure: C = continuous noise, I = impact noise, and U = ultrasonic noise.

stitute for Occupational Safety and Health (NIOSH). A job exposure matrix (JEM) has been developed by NIOSH epidemiologists to identify occupations that have common exposures. The matrix is constructed using results from walk-through surveys of individual plants and industries.[53]

The NOES was a field survey intended to describe health and safety conditions in the American work place, and to determine the extent of workers' exposure to chemical and physical agents. The survey sample was representative of all nonagricultural businesses that were covered under the Occupational Safety and Health Act of 1970 and that employed eight of more individuals. The nature of industrial activity was coded using Standard Industrial Classification (SIC) codes.[54]

In Tables 1 and 2 are listed industries where exposure to the selected agents might occur. Industries are listed by economic sector, SIC code, and by exposures observed. In Table 1, the manufacturing industry was not included. The information from this economic sector is displayed in Table 2. The percentage of employees exposed to the selected chemicals, in each economic sector, was interpolated to the total population and it is shown along with the industry's SIC code. The industry category with the greatest exposure to a particular agent is indicated by an asterisk. It also is possible, using Tables 1 and 2, to identify industries where multiple exposures are probable.

In Table 3, the total number of workers estimated to be exposed to the selected solvents is displayed. It was estimated that approximately 5 million workers in the American industry are exposed to the five reviewed chemicals. Of this total, the majority of workers are exposed to xylene (2 145 039) and toluene (2 015 881). More than 400 000 workers are estimated to be exposed to trichloroethylene, more than 300 000 workers to styrene, and 45 000 to carbon disulfide.

There are numerous categories where multiple exposures occur to the five chemicals in question. In addition, noise exposure is usually present whenever exposure to the selected chemicals occurs. The information provided in Tables 1 and 2 can be useful when developing studies

Table 3—Total Numbers of Employees Estimated to be Potentially Exposed to Selected Solvents, 1981–1983 National Occupational Exposure Survey

Solvent	No. employees	Standard error of estimate
Toluene	2 015 881	20 158
Xylene	2 145 039	21 450
Trichloroethylene	401 373	5 418
Styrene	333 212	4 665
Carbon disulfide	45 760	778

of possible interaction between noise and solvents because the data indicate the population that might be at risk of developing hearing impairments (for detailed information on levels of noise exposure by economic sector and by number of noise exposed, see Franks[55]).

Conclusions

Most of the aforementioned studies have been conducted with laboratory animals and different modes of agent administration. Their positive findings indicate the need for further epidemiologic investigations on populations occupationally exposed to these agents. Hearing assessment should be performed, preferably with other tests to complement the information provided by pure tone audiometry. Studies on a variety of other chemical agents that produce neurotoxic effects need to be pursued (e.g., carbon tetrachloride, *n*-hexane, metals, and asphixiants).

Another important issue to be noted is that few of the reviewed investigations examined or controlled for the effect of simultaneous exposure to noise. The probable interaction between those agents and noise needs more attention to determine if it is a serious problem. The underlying physiologic mechanisms to explain the auditory system injury caused by environmental chemicals are unknown and need to be explored.

The association between occupational exposure to solvents and hearing impairment is rarely evaluated. Noise is often present in most occupational settings where solvent exposures occur, and the hearing losses observed in these situations are often attributed to the noise exposure. In addition, by merely looking to a pure tone audiogram, one cannot determine the etiology of a hearing disorder. The audiometric configuration in cases of noise-induced hearing loss and ototoxicity can be identical.

In the United States, as in many other countries, there are no regulations that require the monitoring of workers' hearing who are employed at locations in which occupational exposure to potentially ototoxic chemicals occurs. Consequently, despite the large number of workers exposed to these chemicals in the presence of background noise, few will be required to have regular hearing tests because the noise exposure may not exceed the regulatory guidelines. Given that there is evidence that exposure to these chemicals alone or in combination with noise can produce hearing loss, it is very possible that current hearing conservation practices are not meeting the needs of this population of workers. Some of the other issues these findings raise include the adequacy of pure tone audiometry testing in screening solvent-exposed workers; the appropriateness of the current threshold limits when certain hazards occur simultaneously in the workplace; and finally, the role of hearing assessment as applied to the early identification of those most susceptible to neurotoxic disorders. These are some of the challenges to be addressed by studies of occupational hazards from combined exposure to chemicals and noise.

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Submitted for publication June 29, 1993; revised; accepted for publication December 5, 1993.

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References

1. Murad F, Gilman AG. Drug interactions. In: Gilman AG, Goodman LS, Hall TW, Murad F, Eds. Goodman and Gilman's the pharmacological basis of therapeutics, 7th ed. New York: Macmillan, 1985.
2. Prosen CA, Stebbins WC. Ototoxicity. In: Spencer PS, Schaumburg HH, Eds. Experimental and clinical neurotoxicology. Baltimore: Williams & Wilkins, 1980.
3. Humes LE. Noise-induced hearing loss as influenced by other agents and by some physical characteristics of the individual. J Acoustic Soc Am 1984; 76:1318–29.
4. Dayal VS, Barek WG. Cochlear changes from noise, kanamycin and aging: II. Potentiating effects of noise and kanamycin. Laryngoscope 1975; 85(suppl):8–11.
5. Marques DM, Clark CS, Hawkins JE. Potentiation of cochlear injury by noise and ototoxic antibiotics in guinea pigs. J Acoust Soc Am 1975; 57:51(A).
6. Bone RC, Ryan AF. Audiometric and histologic correlates of the interaction between kanamycin and subtraumatic levels of noise in the chinchilla. Trans Am Acad Ophthalm Otolaryngol 1978; 86:400–04.
7. Brown JJ, Brummet RE, Fox KE. Combined effects of noise and kanamycin. Arch of Otolaryngol 1980; 86:394–400.

8. Mills JH. A review of the environmental factors affecting hearing. In: Hayes AW, Ed. Toxicology of the eye, ear, and other special senses. New York: Raven Press, 1985.
9. Boettcher EA, Henderson D, Gratton MA, Danielson RW, Byrne CD. Synergistic interaction of noise and other ototraumatic agents. Ear and Hear 1987; 8:192–212.
10. Lindgren F. Clinical Investigations of noise-induced temporary hearing loss. Goteborg: University of Goteborg, 1987.
11. Young JS, Upchurch MB, Kaufman MJ, Fechter LD. Carbon monoxide exposure potentiates high-frequency auditory threshold shifts induced by noise. Hear Res 1987; 26:37--43.
12. Barregård L, Axelsson A. Is there an ototraumatic interaction between noise and solvents? Scand Audiol 1984; 13:151--55.
13. Bergström B, Nyström B. Development of hearing loss during long-term exposure to occupational noise. Scand Audiol 1986; 15:227–34.
14. Ödkvist LM, Arlinger SD, Edling C, Larsby B, Bergholtz LM. Audiological and vestibulo-oculo-motor to findings in workers exposed to solvents and jet-fuel. Scand Audiol 1987; 16(2):75–84.
15. Johnson AC, Juntunen L, Nylén P, Borg E, Hoglund G. Effect of interaction between noise and toluene on auditory function in the rat. Acta Otolaryngol 1988; 105:56–63.
16. Morata TC. Study of the effects of simultaneous exposure to noise and carbon disulfide on workers' hearing. Scand Audiol 1989; 18:53–58.
17. Haider M, Kundi M, Groll-Knapp E, Koller M. Interactions between noise and air pollution. Environ International 1990; 16:593–601.
18. Phaneuf R, Hétu R. An epidemiological perspective of the causes of hearing loss among industrial workers. J Otolaryngol 1990; 19(1):31–40.
19. Rybak LP. Hearing: the effects of chemicals. Otolaryngol Head Neck Surg 1992; 106:677–86.
20. Baker EL, Smith TJ, Landrigan PJ. The neurotoxicity of industrial solvents: a review of the literature. Am J Ind Med 1985; 8:207–17.
21. Spencer PS, Schaumburg HH. Organic solvent neurotoxicity. Facts and research needs. Scand J Work Environ Health 1985; 11(suppl):53–60.
22. National Institute for Occupational Safety and Health. National-Occupational Exposure Survey. DHHS (NIOSH) Publication no. 88-106, 89-102, 89-103. Washington, DC: US Department of Health and Human Services, 1988,1990.
23. World Health Organization. Environmental health criteria 10: carbon disulfide. Geneva: WHO, 1979.
24. Sulkowski WJ. Badania nad prxydatnoscia kliniczna audiometrii i elektronystagmografii w diagnostyce przewleklych zatruc dwusiarczkiem wegla. Medycyna Pracy 1979; 30:135–45.
25. Hirata M, Ogawa Y, Okayama A, Goto S. A cross-sectional study on the brainstem auditory evoked potential among workers exposed to carbon disulfide. Int Arch Occup Environ Health 1992; 64(5):321–24.
26. Robert CS, Beeker E. Effects of inhaled carbon disulfide on sensory-evoked potentials of Long-Evans rats. Neurobehav Toxicol Teratol 1986; 8:533–41.
27. Rebert CS, Sorenson SS, Pryor GT. Effects of intraperitoneal carbon disulfide on sensory evoked potentials of Fischer-344 rats. Neurobehav Toxicol Teratol 1986; 8:543–49.
28. Clerici WJ, Fechter LD. Effects of chronic carbon disulfide inhalation on sensory and motor function in the rat. Neurotoxicol Teratol 1991; 13: 249–55.
29. Wilson RH. Toluene poisoning. JAMA 1943; 123(2):1106–08.
30. Arlien-Søborg P, Zilstorff K, Grandjean B, Pedersen LM. Vestibular dysfunction in occupational solvent intoxication. Clin Otolaryngol 1981; 6:285–90.
31. Fornazzari L, Wilkinson DA, Kapur BM, Carlen PL. Cerebellar, cortical and functional impairment in toluene abusers. Acta Neurol Scand 1983; 67:319–29.
32. Lazar RB, Ho SU, Melen O, Daghestani AN. Multifocal central nervous system damage caused by toluene abuse. Neurol 1983; 33:1337–40.
33. Ehyai A, Freeman FR. Progressive optic neuropathy and sensorineural hearing loss due to chronic glue sniffing. J Neurol Neurosurg Psychiatry 1983; 46:349–51.
34. Rosenberg NL, Spitz MC, Filley CM, Davis KA, Schaumburg HH. Central nervous system effects of chronic toluene abuse--clinical, brainstem evoked response and magnetic resonance imaging studies. Neurotoxicol Teratol 1988; 10:489–95.
35. Rebert CS, Sorenson SS, Howd RA, Pryor GT. Toluene induced hearing loss in rats evidenced by the brainstem auditory evoked response. Neurobehav Toxicol Teratol 1983; 5(1):59–62.
36. Pryor GT, Dickinson J, Feeney E, Rebert CS. Transient cognitive deficits and high-frequency hearing loss in weanling rats exposed to toluene. Neurobehav Toxicol Teratol 1983; 5(1):53–57.
37. Pryor GT, Dickinson J, Feeney E, Rebert CS. Hearing loss in rats first exposed to toluene as weanlings or as young adults. Neurobehav Toxicol Teratol 1984; 6(2):111–19.
38. Pryor GT, Howd RA. Toluene Induced ototoxicity by subcutaneous asministration. Neurobehav Toxicol Teratol 1986; 8(1):103–104.
39. Sullivan, MJ, Rarey KE, Conolly RB. Ototoxicity of toluene in rats. Neurotoxicol Teratol 1989; 10:525–30.
40. Lee B-K, Lee Se-H, Lee K-M, et al. Dose-dependant increase in subjective symptom prevalence among toluene-exposed workers. Ind Health 1988; 26:11–23.
41. Coscia GC, Tabaro G, Albera C, et al. Alterazioni vestibolari nell 'esposizione a toluene. Med Lavoro 1983; 74:23–29.
42. Morata, TC, Dunn DE, Kretschmer LW, Lemasters GK, Keith RW. Occupational exposure to organic solvents and noise: effects on hearing. Scand J Work Environ Health 1993; 19:245–54.
43. Pryor, GT, Rebert CS, Howd RA. Hearing loss in rats caused by inhalation of mixed xylenes and styrene. J Appl Toxicol 1987; 7(1):55–61.
44. Muijser H, Hoogendijk EMG, Hooiisma J. The effects of occupational exposure to styrene on high frequency thresholds. Toxicology 1988; 49:331–40.
45. Moller C, Ödkvist LM, Larsby B, Tham R, Ledin T, Bergholtz L. Otoneurological findings in workers exposed to styrene. Scan J Work Environ Health 1990; 16:189–94.
46. Mitchell ABS, Parsons-Smith BG. Trichloroethylene neuropathy. Brit Med J 1969; 1:422–23.
47. Tomasini M, Sartorell E. Intossicazione cronica da infalazione di trieleina commercial com compromissione dell' viii palo de nervi cranici. Clin Lavoro 1971; 62:277–80.
48. Szulc-Kuberska J, Tronczynska J, Latkowski B. Otoneurological investigations of chronic trichloroethylene poisoning. Minerva Otorinolaryngol 1976; 26:108–12.
49. Agency for Toxic Substances and Diseade Registry/Division of Health Studies. National Exposure Registry—Trichloroethylene (TCE) Subregistry—baseline technical report. Washington DC: U.S. Department of Health and Human Services, 1993.
50. Rebert CS, Day VL, Matteucci MJ, Pryor GT. Sensory-evoked potentials in rats chronically exposed to trichloroethylene: predominant auditory dysfunction. Neurotoxicol Teratol 1991; 13:83–90.
51. Jaspers RMA, Muijser H, Lammers JHCM, Kulig BM. Mid-frequency hearing loss and reduction of acoustic startle responding in rats following trichloroethylene exposure. Neurotoxicol Teratol 1993; 15:407–12.
52. Crofton, KM, Zhao X. Mid-frequency hearing loss in rats following inhalation exposure to trichloroethylene: evidence from reflex modification audiometry. Neurotoxicol Teratol 1993; 15:413–23.
53. Sieber WK, Sundin DS, Frazier TM, Robinson CE. Development, use and availability of a job exposure matrix based on National Exposure Survey data. Am J Ind Med 1991; 20:163–74.
54. Executive Office of the President (EOP)—Office of Management and Budget. Standard industrial classification manual. GPO 041-001-00066-6, Washington, DC: Government Printing Office, 1972.
55. Franks JR. Number of workers exposed to occupational noise. Semin Hearing 1988; 9(4):287–98.