Alterations in Cognitive and Psychological Functioning after Organic Solvent Exposure

Morrow, L. A., C. M. Ryan, M. J. Hodgson, and N. Robin
Journal of Occupational Medicine, Vol. 32, No. 5, pages 444-450, 42 references May 1990 CODEN: JOCMA7

An evaluation was conducted to assess cognitive functioning in a large sample of solvent exposed workers and to determine whether there were certain cognitive and personality variables that were particularly vulnerable to the effects of solvent exposure. The possibility that certain exposure related variables might predict performance on the cognitive and psychological measures was also evaluated and the relationship between workers' subjective psychological complaints and their performance on the cognitive tests was determined. Thirty two solvent exposed workers participated and were compared to a referent group. Significant differences were observed between the two groups in nearly all of the cognitive domains tested except those measuring general intelligence. These other domains included learning and memory, visuospatial, attention and mental flexibility, and psychomotor speed. The Minnesota Multiphasic Personality Inventories indicated clinically significant levels of depression, anxiety, somatic concerns and disturbances in thinking among the solvent exposed workers. Several exposure related variables were associated with poorer performance on tests of memory and visuospatial ability.

Subclinical Neuropsychiatric Effects of Chronic Low-Level Solvent Exposure in US Paint Manufacturers

Bolla, K. I., B. S. Schwartz, J. Agnew, P. D. Ford, and M. L. Bleecker Journal of Occupational Medicine, Vol. 32, No. 8, pages 671-677, 24 references August 1990 CODEN: JOCMA7

A study of subclinical neuropsychiatric effects associated with low level solvent exposures (painter's syndrome) in paint manufacturing workers was conducted. The study group consisted of 187 male production, maintenance, and warehouse workers, mean age 42 years, at two United States paint manufacturers that produced a variety of paints and coatings. The subjects had been exposed to organic solvents for at least 6 years. They completed a questionnaire package that sought information on demographic characteristics, occupational and medical histories, and rated painter's syndrome according to the Zung Depression Index (ZDI), Scandinavian Questionnaire 16 (Q16), and the Painter's Syndrome Present State Exam (PSPSE). The questionnaire data on occupational history and previously obtained industrial hygiene monitoring data were used to obtain estimates of lifetime weighted average exposure (LWAE) and cumulative lifetime total exposure (CE) for each subject. The total number of neuropsychiatric symptoms reported on the Q16, ZDI, and PSPSE tended to be small and were not significantly related to solvent exposure. Four items on the PSPSE characteristic of depression were significantly associated with LWAE and CE; however, the number of subjects reporting these symptoms was

very small. The authors conclude that the results do not support the association of chronic solvent exposure with the development of painter's syndrome and that the association between certain characteristics of depression and exposure may not be of clinical relevance.

Occupational Phenoxyethanol Neurotoxicity: A Report of Three Cases
Morton, W. E.
Journal of Occupational Medicine, Vol. 32, No. 1, pages 42-45, 7 references January 1990 CODEN: JOCMA7

Cases of occupational exposure to 2-phenoxyethanol (122996) which resulted in damage to the nervous system were reported. The three patients were workers at a fish hatchery in a rural Oregon community where 2-phenoxyethanol was used to partially anesthetize the fish prior to the worker picking them up one at a time from a small dishpan for hand tagging. Larger pans and more phenoxyethanol were used for fin trimming. Each tagger used about one 500 milliliter bottle of 2-phenoxyethanol per day, and the three taggers handled about 4000 to 8000 fish each day. Percutaneous absorption was the principle route of exposure, although a small degree of inhalation probably also occurred. The taggers generally noted headache, lightheadedness, slurred speech, euphoria, grogginess and eventually a drunk feeling while using the 2-phenoxyethanol. After 1 to 2 years of this work they experienced constant irritability, forgetfulness, and difficulty in maintaining concentration. This persistent cognitive impairment and secondary anxiety and depression disrupted their lives, producing a persistent limitation of function and work options. These symptoms eventually resulted in identification of the problem. These effects of 2-phenoxyethanol do not appear to be different from the results of exposure to other organic solvents.
CAS REGISTRY NO.: 122-99-6

Psychotoxicology: The Return of the Mad Hatter
Dumont, M. P.
Social Science and Medicine, Vol. 29, No. 8, pages 1077-1082, 29 references 1989 CODEN: SSCMAW

The psychopathogenic effects of chemical exposures in humans were discussed. The difficulties encountered in describing the behavioral toxicity of substances were considered as resulting from the variability of behavioral measurements. Central nervous system disorders resulting from chronic solvent exposure were classified by severity, from organic affective syndrome, to mild chronic toxic encephalopathy, to dementia resulting from severe toxic encephalopathy. Domestic exposures to psychotoxic agents such as lead (7439921) and organophosphorus insecticides were discussed, as was fetal exposure to neurotoxins and subsequent central nervous system specific birth defects, the psychotoxicity of food additives, and the

prescription of psychotoxic drugs. Three case histories were reported. The first was a 44 year old female presenting with severe agitated depression, who ultimately was found to be exposed daily to lead fumes inside her house. The second was a 23 year old male who developed paranoid symptoms after working in a lacquer factory with high concentrations of toluene (108883) in ambient air. The third was a 39 year old female who developed paranoid symptoms after exposure to neurotoxic pesticides at her workplace. The author recommends that psychotoxicology be incorporated into psychiatric practice by inquiries into occupational and domestic toxic exposures, increasing knowledge of behavioral effects of toxicants, researching the psychopathogenic effects of pollutants, and by more cautiously prescribing teratogenic drugs.
CAS REGISTRY NO.: 7439-92-1; 108-88-3

A Distinct Pattern of Personality Disturbance following Exposure to Mixtures of Organic Solvents

Morrow, L. A., C. M. Ryan, G. Goldstein, and M. J. Hodgson
Journal of Occupational Medicine, Vol. 31, No. 9, pages 743-746, 21 references September 1989 CODEN: JOCMA7

Pattern of psychologic distress among 22 men with histories of occupational exposure to mixtures of organic solvents was evaluated using a psychometrically sound questionnaire (Minnesota Multiphasic Personality Inventory (MMPI)). All participants had been referred to a hospital Occupational Medicine Clinic and had no documented neurologic or psychiatric disorders. Mean length of solvent use was 7.3 years (range 2 months to 19 years). Mean interval between exposure and evaluation was 19.8 weeks (range 1 to 84 weeks). Scores for two or more of the ten clinical scales were more than two standard deviations above the norm and fell in the clinically significant range (scores of at least 70) for 90 percent of the men. The highest elevations were found on the following four clinical scales: excessive bodily concern; depression; somatic concerns coupled with emotional discomfort and social withdrawal; and mental confusion, feelings of alienation, unusual thoughts, or psychologic turmoil. Sixteen men had significant clinical elevations on at least three of the four highest elevated scales while five men had elevations on at least two of these scales. The mean profile pattern for these occupationally exposed men was almost identical in severity and symptomatology to that for a group of former World War-II prisoners of war, half of whom met full criteria for post traumatic stress disorder. The score for MMPI scale eight was significantly correlated with duration of exposure. Profile patterns were not different for workers with versus without a long exposure/evaluation interval or an accidental excessive exposure. A case history representative of the psychological distress pattern for these workers was given. The man first felt strange while working around trichloroethylene (79016) and later experienced personality changes, confusion, and depression. CAS REGISTRY NO.: 79-01-6

Freon: An Unsuspected Problem
Voge, V. M.
Aviation, Space, and Environmental Medicine, Vol. 60, No. 10, Section 2, pages B27-B28 October 1989 CODEN: ASEMCG

A discussion was presented on the inhalation of freon compounds which have frequently been used as unauthorized degreasing agents by aviation ground personnel because they enable the workers to get their jobs done faster. Normally the use of such agents in hanger areas is not hazardous, as most hangers are large with good ventilation. However, attempting to use such agents in poorly ventilated or enclosed areas can result in serious consequences. Freon-113 (76131) has no odor, taste or color and it displaces the oxygen in the immediate breathing area, causing asphyxiation. The victim suffers pulmonary edema and death. Early symptoms may include headache, lightheadedness, dizziness, drowsiness, tingling of the toes and fingers, and signs of central nervous system depression. However, there are often no early symptoms at all. Another danger from the inhalation of high concentrations of the vapors is that the myocardium may become sensitized to adrenaline, causing dysrhythmias, ventricular fibrillation and death. Therapy at the moment consists of the immediate removal of the victim to fresh air. Artificial respiration should be performed if breathing has stopped, but mouth to mouth resuscitation was not recommended if alternative measures were available so as to avoid exposing the would be rescuers to large amounts of freon. Oxygen therapy was suggested. The use of vasopressor drugs was considered to be inadvisable. Guidelines were reviewed for those authorized to use freon.
CAS REGISTRY NO.: 76-13-1

Prenarcotic and Neuraesthenic Symptoms among Dutch Workers Exposed to Organic Solvents
van Vliet, C., G. M. H. Swaen, J. M. M. Meijers, J. Slangen, T. de Boorder, and F. Sturmans
British Journal of Industrial Medicine, Vol. 46, No. 8, pages 586-590, 10 references August 1989 CODEN: BJIMAG

A population of Dutch workers exposed to solvents was compared with a nonexposed population with regard to the prevalence of prenarcotic and neuraesthenic symptoms so that an investigation could be made of the existence of acute and chronic neurotoxic effects due to exposure to organic solvents. The study analyzed the symptoms reported by participants in an earlier case/control investigation of Dutch painters and members of the construction union who were exposed to organic solvents. The symptoms of the exposed and nonexposed populations were compared. Any workers determined in the earlier case/control study to be suffering from mental disorders due to their solvent exposures were not included in this study of symptomatology. Data was collected from 822 individuals. The highest levels of exposure were noted by those 379 persons involved in painting, carpet

laying or road marking. These were classified as the exposed group. The other 443 were labeled nonexposed. Three symptoms were noted more frequently among the exposed subjects: nausea, shortness of breath, and loss of appetite. Half of the exposed workers and 33 percent of the nonexposed workers reported that symptoms ceased over the weekend and holidays. Concerning neuraesthenic symptoms related to fatigue, forgetfulness, concentration difficulties, sleeping difficulties, lability of mood, headache and dizziness, vegetative symptoms and neuropathy, only weak associations with solvent exposure could be identified. The authors conclude that the organic solvent syndrome type-I, which is characterized by the occurrence of these symptoms without any neurological damage, is not a frequent occurrence among Dutch workers exposed to solvents.

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File 161:OCCUPATIONAL SAFETY & HEALTH (NIOSH)_73-91/JULY

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      S2        0  DISULFIDE ..........
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?ss (carbon()disulfide or carbon()disulphide) and neuro?

      S4    12566  CARBON
      S5     1520  DISULFIDE
      S6     1245  CARBON(W)DISULFIDE
      S7    12566  CARBON
      S8      278  DISULPHIDE
      S9      252  CARBON(W)DISULPHIDE
     S10     7759  NEURO?
     S11      216   (CARBON()DISULFIDE OR CARBON()DISULPHIDE) AND
NEURO? ?t s11/8/1


 11/8/1
0165469      NIOSH-00197394
   Special  Nerve  Functions and Colour Discrimination in Workers
with Long Term Low Level Exposure to Carbon Disulphide
   DESCRIPTORS: Synthetic fibers;  Synthetic fibers industry;
 Vision disorders;      Textile    workers;      Nervous    system
disorders;       Occupational  exposure;     Visual  perception;
Neuropathology
  CAS REGISTRY NO.: 75-15-0
?t s11/8/2


 11/8/2
0163064      NIOSH-00194958
   Carbon   Disulfide   Exposure   and Neurotoxic Sequelae among
Viscose Rayon Workers
   DESCRIPTORS:  Solvent  vapors;     Nervous  system  disorders;
Neuropathology; Neurotoxins; Organic solvents; Chemical factory
workers;  Polymerization ;  Occupational exposure
  CAS REGISTRY NO.: 75-15-0; 7783-06-4
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     S14      411  SUICID?
     S15        1  S11 AND SUICID?
?t s15/8/1


 15/8/1
0112003     NIOSH-00151268
  Carbon Disulfide
  DESCRIPTORS: Toxicology; Heart; Lung irritants; Occupational
exposure; Industrial chemicals; Health standards; Exposure
levels; Workers; Industrial emissions
  CAS REGISTRY NO.: 75-15-0
?t s15/5/1


 15/5/1
0112003     NIOSH-00151268
  Carbon Disulfide
  Lilis, R.
   Environmental and Occupational Medicine, pages 627-631, 32
references 1983
   Occupational hazards of carbon-disulfide (75150) exposure are
reviewed. Uses of carbon-disulfide and sources of exposure
are described. Its absorption, distribution, and
biotransformation are discussed. The toxic effects include
central nervous system symptoms resulting in severe acute or
subacute encephalopathy. The clinical picture includes
headache, dizziness, fatigue, excitement, depression, memory
deficit, indifference, apathy, delusions, hallucinations,
suicidal tendencies, delirium, acute mania, and coma. The
outcome may be fatal, or in less severe cases, incomplete
recovery may occur with persistent psychiatric symptoms
indicating irreversible central nervous system injury.
Peripheral neuropathy, electromyographic changes, and
cerebrovascular alterations are described. Exposed workers often
show hypertension, elevated cholesterol and lipoproteins, and
increased mortality from coronary heart disease. Adverse effects
of carbon-disulfide exposure on reproductive function are
considered. Mechanisms of toxicity include reaction with amino
groups to form thiocarbamates which undergo cyclic
transformation to thiazolidines which chelate essential trace
metals. In addition, carbon-disulfide interferes with normal
catecholamine metabolism and with vitamin-B6, and alters liver
microsomal enzymes interfering with cytochrome-P-450
production. Urinary metabolites of carbon-disulfide
catalyze the iodine/azide reaction providing a useful
biological monitor. The morphologic abnormalities underlying
peripheral neuropathy produced by carbon-disulfide in animal
studies are discussed. Axonal degeneration is reported, possibly
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due to covalent binding of highly reactive sulfur to thiol groups of enzymes and proteins essential for the normal function of axonal transport. Engineering controls on exposure, provision of appropriate respiratory protective devices for emergencies, worker education, and medical surveillance are recommended.
DESCRIPTORS: Toxicology; Heart; Lung irritants; Occupational exposure; Industrial chemicals; Health standards; Exposure levels; Workers; Industrial emissions
CAS REGISTRY NO.: 75-15-0
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Set     Items   Description
S1          0   CARO.BON
S2          0   DISULFIDE ..........
S3          0   CARO.BON()DISULFIDE ..........
S4      12566   CARBON
S5       1520   DISULFIDE
S6       1245   CARBON(W)DISULFIDE
S7      12566   CARBON
S8        278   DISULPHIDE
S9        252   CARBON(W)DISULPHIDE
S10      7759   NEURO?
S11       216    (CARBON()DISULFIDE OR CARBON()DISULPHIDE) AND
NEURO? S12          0   SUCI....
S13         0   S11 AND SUCI....
S14       411   SUICID?
S15         1   S11 AND SUICID?
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 12/5/1
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 11/5/1
0165469     NIOSH-00197394
   Special Nerve Functions and Colour Discrimination in Workers with Long Term Low Level Exposure to Carbon Disulphide
  Ruijten, M. W. M. M., H. J. A. Salle, M. M. Verberk, and H. Muijser British Journal of Industrial Medicine, Vol. 47, No. 9, pages 589-595, 28 references September 1990 CODEN: BJIMAG
   An investigation was conducted to determine whether a low level exposure to carbon-disulfide (75150) (CS2) affected the
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peripheral nervous system and color vision, and to evaluate the feasibility of proposed diagnostic methods. All male workers at a viscose rayon facility who had been exposed to CS2 for 10 years or more were examined. A total of 45 exposed workers and 37 comparisons participated in the investigation. Small effects of exposure were demonstrated upon measurement of forced respiratory sinus arrhythmia (FRSA), muscle heart reflex (MHR), and conduction velocity of the slow motor fibers (refractory period). When cumulative exposure was considered, the significant differences in FRSA and MHR disappeared. For indices of motor nerve function, the absolute t-values increased with longer half times. The overall color confusion index as well as those for red and green and for blue and yellow did not differ significantly between the exposed and the comparison workers. The authors suggest that a lifetime exposure of around 4 parts per million appears required to prevent the small observed effects.

DESCRIPTORS: Synthetic fibers; Synthetic fibers industry; Vision disorders; Textile workers; Nervous system disorders; Occupational exposure; Visual perception; Neuropathology

CAS REGISTRY NO.: 75-15-0

11/5/2

0163064 NIOSH-00194958

Carbon Disulfide Exposure and Neurotoxic Sequelae among Viscose Rayon Workers

Aaserud, O., O. J. Hommeren, B. Tvedt, P. Nakstad, G. Mowe, J. Efskind, D. Russell, E. B. Jorgensen, R. Nyberg-Hansen, K. Rootwelt, and L. Gjerstad American Journal of Industrial Medicine, Vol. 18, No. 1, pages 25-37, 36 references 1990 CODEN: AJIMD8

A survey was conducted of carbon-disulfide (75150) (CS2) exposure, the degree of exposure, and the possible presence of neuropathy or encephalopathy in a group of workers from a rayon viscose factory. The investigation was performed in 1986 and included 16 workers from the only existing Norwegian rayon viscose factory which was closed in November of 1982. The subjects had been working in the factory's spinning department for at least 10 years. Two were former heavy drinkers, five had cardiovascular disease, and two had diabetes mellitus. Four workers had a cerebral concussion in their history. Three had received compensation due to CS2 damage. Eight workers had an encephalopathy, probably due to CS2 alone, and six had encephalopathy partly caused by CS2 exposure. There was a neuropathy in seven workers probably caused by CS2 alone, and in three workers CS2 exposure was a partial cause of neuropathy. Results indicated that the long term relatively moderate exposure to CS2, in association with high peak exposures to CS2 and hydrogen-sulfide, (7783064) (H2S) involves a substantial risk of developing neurotoxic disease.

DESCRIPTORS: Solvent vapors; Nervous system disorders; Neuropathology; Neurotoxins; Organic solvents; Chemical factory workers; Polymerization ; Occupational exposure

CAS REGISTRY NO.: 75-15-0; 7783-06-4

11/5/3
0162544 NIOSH-00194428
Health Hazard Evaluation Report No. HETA-89-212-2020, Schlegel Tennessee, Inc., Maryville, Tennessee
Kiken, S., M. Newman, and C. Cox
Hazard Evaluations and Technical Assistance Branch, NIOSH, U.S. Department of Health and Human Services, Cincinnati, Ohio, Report No. HETA-89-212-2020, 17 pages, 15 references March 1990
REPORT NO.: NTIS-PB91-104-604
In response to a request from the company, an investigation was made of possible hazardous conditions at the Schlegel Tennessee, Inc. (SIC-2822), Maryville, Tennessee. Concern had been expressed by union representatives about possible exposures to airborne nitrosamines, and the incidence of cancer in employees. The company produces rubber weather stripping for automobiles. Approximately 70 salaried and 200 production workers were employed by the company. Packers were experiencing dizziness, nausea, tingling lips, headaches, and depression. These problems had been attributed to odors in the workplace. Three employees had abnormal neurologic examinations and two others had reported abnormal urine iodine-azide tests. Current testing did not reveal any employees with 2-thiothiazolidine-4-carboxylic-acid (TTCA) in their urine, indicating that workers were not exposed to more than 0.5ppm carbon-disulfide (75150) (CS2). No personal breathing or area air sampling tests showed CS2 contamination above acceptable levels. No excess of reported cancers was noted following a standardized morbidity ratio analysis compared to the general population of the United States. Detectable levels of nitrosamines were not found. According to the authors, the etiology of medical problems experienced by workers could not be definitively determined. The authors recommend measures to lower potential hazardous exposures at this site. DESCRIPTORS: NIOSH Publication; NIOSH Author; NIOSH Health Hazard Evaluation; NIOSH Technical Assistance Report; HETA 89 212 2020; Hazard Unconfirmed; Region 4; Rubber manufacturing industry; Sulfides; Nitroso compounds; Amines; Organic vapors
CAS REGISTRY NO.: 75-15-0

11/5/4
0162271 NIOSH-00192302
NIOSH Testimony on Neurotoxic Chemicals by J. R. Froines, June 6, 1979 NIOSH
NIOSH, 17 pages June 6, 1979
This testimony summarized information dealing with worker exposure to neurotoxic chemicals. Following a definition of neurotoxicity, the testimony described some of the investigations on workers exposed to such chemicals on the job in which NIOSH has been involved. Included among the examples were the workers in Hopewell, Virginia, exposed to kepone (143500), who suffered loss of muscle coordination, loss of memory, and an eye movement disorder. Another episode of workers exposed to a pesticide involved leptophos (21609905). At the time

of the exposure, leptophos was registered by the Environmental Protection Agency primarily for export. Animals administered a single oral dose showed weight loss, ataxia, and eventual muscle paralysis. Other potentially toxic chemicals were used in the preparation of leptophos including toluene (108883), a neurotoxic solvent. A third incident of worker exposure to pesticides occurred due to the release of o-ethyl-o-p-nitrophenylphenylphosphonothioate (2104645) (EPN), at a manufacturing site in Chicago Heights, Illinois. Neurological signs of distress included muscle weakness and cerebellar signs of toxicity. Cases of worker exposure to the following solvents were also reviewed: methyl-n-butyl-ketone (591786) and carbon-disulfide (75150). Exposures to metals were also reviewed including lead (7439921), arsenic (7440382), and mercury (7439976). Early detection of exposures to neurotoxic agents was briefly considered.

DESCRIPTORS: NIOSH Publication; NIOSH Author; NIOSH Testimony; Froines J R; Nervous system disorders; Neuropathology; Brain damage; Central nervous system; Organic solvents; Chemical manufacturing industry; Agricultural chemicals; Toxic effects

CAS REGISTRY NO.: 143-50-0; 21609-90-5; 108-88-3; 2104-64-5; 591-78-6; 75-15-0; 7439-92-1; 7440-38-2; 7439-97-6

11/5/5

0159832 NIOSH-00191766

The Application of Psychological Methods for Evaluation of Effects of Occupational Exposure to Neurotoxic Substances

Indulski, J. A., and B. Dudek

Polish Journal of Occupational Medicine, Vol. 1, No. 2, pages 154-165, 18 references 1988

The role of psychological methods in evaluating the effects of neurotoxic substances was discussed. Traditional psychological techniques for detecting early (subclinical) symptoms resulting from exposure to neurotoxins were considered. These have been based primarily on administering a battery of tests that evaluated variables such as eye and hand coordination, two hand coordination, palm movement precision, tapping speed, manual dexterity, finger dexterity, and simple and choice reaction time. The results of a study of the neurobehavioral effects of carbon-disulfide (75150) on 126 workers were discussed. The subjects were administered a battery of 12 psychomotor tests. Their performance on nine tests in the battery was significantly impaired. The extent of impairment was greater than that associated with aging. Analyzing the results of psychological studies of persons exposed to neurotoxic substances was discussed. New approaches for applying psychological methods in evaluating the effects of occupational neurotoxin exposure were considered. These included the Neurobehavioral Core Test Battery (NCTB) developed under the auspices of the World Health Organization, microcomputerized test batteries, subjective techniques, and cognitive methods. The authors recommend that the NCTB battery always be used regardless of any other tests administered. This will facilitate comparing data and assist

in developing a database.
DESCRIPTORS: Occupational exposure; Neurotoxicity; Toxic effects; Task performance; Organic solvents; Sulfur compounds; Epidemiology; Mental processes; Psychomotor function
CAS REGISTRY NO.: 75-15-0

11/5/6
0156466 NIOSH-00099407
The Health of Women Workers in the Man-Made Fibers Industry Based on the Findings of Medical Examinations
Petrov, N. V.
Vrachebnoe Delo, No. 10, (Department of Occupational Hygiene and Organization of Public Health), pages 145-148 1975
CODEN: VRDEA5 LANGUAGE(S): Russian
The effects of exposure to carbon-disulfide (75150), caprolactam (105602), and Dinyl (8004135) were studied in 3,624 female workers in the synthetic fiber industry in the Soviet Union. All workers received in depth medical examinations and were divided into three groups. The first group of 2113 workers was comprised of chemical and spinning workers engaged in viscose manufacture and were primarily exposed to carbon-disulfide. The second group of 492 women was composed of chemical and spinning workers engaged in nylon manufacture with primary exposure to caprolactam and Dinyl. The third group of 1,019 women consisted of workers without any chemical exposure. Of the total number of subjects examined, 574 had various health problems with 373 of them in the first group, 85 in the second, and 116 in the third. The frequency of health disorders among all workers increased with age and length of service. Neurological disorders accounted for the majority of health problems in the first and second groups. Most of these disorders were composed of neuroses, vegetovascular dystonia and asthenovegatative syndrome, and diseases of the peripheral nervous system. The second most frequent health disorder among the first and second group of workers were diseases of the urogenital organs, chiefly inflammation of the uterus and appendix. Inflammatory diseases were observed with roughly the same frequency in each of the groups. Respiratory diseases accounted for the third most frequent medical disorder among workers of the first and third group. Diseases of the digestive organs were observed most often among workers of the third group. An increase in the frequency of diseases of the liver and biliary ducts with length of service was identified in workers of the first group. No correlation between length of service and exposure groups and incidence of circulatory disease was noted. The authors conclude that an etiological link between certain pathological conditions among workers in the synthetic fiber industry and specific hygienic features of the work place does exist. (Russian) DESCRIPTORS: TRANS; Sex factors; Chemical exposure; Textile fibers; Textiles industry; Textiles processing; Health surveys; Epidemiological study; Medical examinations; Data collection; Humans; Work environment; Health statistics
CAS REGISTRY NO.: 75-15-0; 105-60-2; 8004-13-5

11/5/7
0155629 NIOSH-00123034
Structural Aspects of Experimental Carbon Disulfide Neuropathy
Szendzikowski, S., J. Stetkiewicz, T. Wronska-Nofer, and I. Zdrajkowska Internationales Archiv fuer Arbeitsmedizin, Vol. 31, No. 2, pages 135-149, 47 references 1973 CODEN: IAANBS

Structural changes in the central and peripheral nervous system of Wistar-white-rats after exposure to carbon-disulfide (75150) (CS2) were studied. Rats were exposed to CS2 vapors at a concentration of 1.5 milligram per liter of air for 5 hours daily, 6 days per week, and killed after 1 to 15 months and prepared for histological and histochemical examination of tissues. Animals retained good general conditions until month 7 of exposure. After 5 to 6 months, histological lesions were observed in the spinal cord and sciatic nerve of rats exposed. Structural changes were observed in myelinated fibers after 1 to 2 months but no indisputable evidence was found of injury to neuronal perikaryons. The authors conclude that histological lesions of neuropathy due to chronic CS2 exposure were observed in rats prior to physical deterioration. DESCRIPTORS: Laboratory animals; Toxic gases; Toxicology; Histology; Nervous system disorders; Central nervous system; Pathology CAS REGISTRY NO.: 75-15-0

11/5/8
0155541 NIOSH-00115603
Study of Neurological and Neurophysiological Impairment in Carbon Disulphide Workers

Gilioli, R., C. Bulgheroni, P. A. Bertazzi, A. M. Cirla, M. Tomasini, M. G. Cassitto, and M. T. Jacovone

Medicina del Lavoro, Vol. 69, No. 2, pages 130-143, 30 references 1978 CODEN: MELAAD

Neurological and neurophysiological effects of carbon-disulfide (75150) exposure were studied. Two hundred and fifty four workers in a viscose rayon factory (SIC-2823) were grouped according to carbon-disulfide (75150) exposure risk category, exposure duration, age, and alcohol consumption history. Fifty four nonexposed workers formed the comparison group. Central nervous system functions and peripheral nerve functioning were evaluated. Central nervous system impairment increased with exposure risk and was manifest by depression and anxiety with reduced vigilance, attention, and memory function. A linear increase in minimal neuropathies also occurred with increased exposure risk categories. Ophthalmoscopic test results indicated a trend toward an induction of normal vascular function with increasing exposure risk. Electroencephalographic results were not related to exposure risk. Alcohol consumption was clearly a factor in the central and peripheral nervous system function decline. Psycho organic disturbances and minimal neuropathy also were evident after 5 years of exposure up to 60 milligrams per cubic meter of carbon-disulfide. The authors conclude that workers exposed to carbon-disulfide are at risk for neurologic disturbances and should be transferred to other work assignments after a maximum of 5 years of exposure. (Italian).

DESCRIPTORS: TRANS; Synthetic fibers industry; Health surveys; Nervous system disorders; Nervous system function; Occupational medicine; Psychological disorders
CAS REGISTRY NO.: 75-15-0; 75-15-0

11/5/9
0154255 NIOSH-00091786
Conduction Velocity in the Peripheral Nerves of Rats with Chronic Carbon Disulphide Neuropathy
Knobloch, K., J. Stetkiewicz and T. Wronska-Nofer
British Journal of Industrial Medicine, Vol. 36, No. 2, pages 148-152, 9 references May 1979 CODEN: BJIMAG
Conduction velocity in the sciatic and tibial nerves of 143 white-rats was measured after exposure to carbon-disulfide (75150) (CS2) vapor at concentrations of 0.9 milligrams per liter (mg/l) or 1.6mg/l of air for periods of 1.5, 3, 6, 9, and 12 months. Rats exposed to 1.6mg/l of CS2 exhibited general physical symptoms of CS2 intoxication after 4 months; rats exposed to lower concentrations appeared healthy throughout the experiment. Electrophysical changes preceded symptoms of intoxication. Measurements of conduction velocity were performed on the day following each exposure period and again 3 and 6 months after the last day of exposure. A reduction in conduction velocity on the day after exposure was observed, the extent of this reduction was related to the concentration and duration of exposure. Complete recovery of conduction velocity was observed after a short intensive period of exposure to CS2. Long term exposure (3, 6, or 9 months) caused a nonreversible reduction in conduction velocity. DESCRIPTORS: Industrial gases; Peripheral nervous system; Laboratory animals; Nervous system disorders; Toxic substances; Electrophysiological measurements; Textile workers; Synthetic fibers; Air contamination; Gas vapors; Neurological reactions
CAS REGISTRY NO.: 75-15-0

11/5/10
0154240 NIOSH-00091771
Clinical and Experimental Studies of Distal Axonopathy -- A Frequent Form of Brain and Nerve Damage Produced by Environmental Chemical Hazards Schaumburg, H. H. and P. S. Spencer
Departments of Neurology, Neuroscience and Pathology (Neuropathology), Albert Einstein College of Medicine, Bronx, New York, Grant No. R01-OH-00535, 34 pages, 27 references 1979
Clinical and pathological studies of nervous system degeneration (distal dying back axonopathy) in man and experimental animals produced by acrylamide-monomer (79061), and hydrocarbon compounds (hexacarbon) are reviewed. Human distal axonopathies include toxic neuropathies induced by the industrial chemicals: acrylamide, n-hexane (110543), cresyl-phosphate (1330785), arsenic (7440382), carbon disulfide (75150), and the pharmaceutical agents clioquinol (130267), nitrofurantoin (67209), and isoniazid (54853). Among the results of the studies were a reappraisal of the dying back hypothesis,

recognition of irreversible subclinical and clinical effects of distal axonopathies on the human central nervous system, a morphological rationale for the wide variety of previously enigmatic clinical phenomena in the human toxic neuropathies, and strong support for the usefulness of neuropathology in the screening of chemicals for neurotoxicity. The authors conclude that it has not been determined with certainty that morphological assays are more sensitive than behavioral testing for detecting the earliest signs of neurotoxicity in the distal axonopathies and that a recently begun collaborative study of behavioral analysis and a simultaneous morphological assay of the nervous system at various stages of intoxication in acrylamide treated rats might clarify the relative uses and merits of these techniques in detecting evidence of neurotoxicity in the distal axonopathies.

DESCRIPTORS: NIOSH Publication; NIOSH Grant; Grant Number R01 OH 00535; End Date 11 30 1981; Neurotoxic effects; Pharmaceutical chemicals; Toxic substances; Spinal cord disorders; Nervous system disorders; Clinical studies; Laboratory animals; Animal studies; Histological reactions; Neurotoxic agents; Pathological studies; Neurophysiological effects

CAS REGISTRY NO.: 79-06-1; 110-54-3; 1330-78-5; 7440-38-2; 75-15-0; 130-26-7; 67-20-9; 54-85-3

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Carbon Disulfide
Lilis, R.
Environmental and Occupational Medicine, pages 627-631, 32 references 1983

Occupational hazards of carbon-disulfide (75150) exposure are reviewed. Uses of carbon-disulfide and sources of exposure are described. Its absorption, distribution, and biotransformation are discussed. The toxic effects include central nervous system symptoms resulting in severe acute or subacute encephalopathy. The clinical picture includes headache, dizziness, fatigue, excitement, depression, memory deficit, indifference, apathy, delusions, hallucinations, **suicidal** tendencies, delirium, acute mania, and coma. The outcome may be fatal, or in less severe cases, incomplete recovery may occur with persistent psychiatric symptoms indicating irreversible central nervous system injury. Peripheral neuropathy, electromyographic changes, and cerebrovascular alterations are described. Exposed workers often show hypertension, elevated cholesterol and lipoproteins, and increased mortality from coronary heart disease. Adverse effects of carbon-disulfide exposure on reproductive function are considered. Mechanisms of toxicity include reaction with amino groups to form thiocarbamates which undergo cyclic transformation to thiazolidines which chelate essential trace metals. In addition, carbon-disulfide interferes with normal catecholamine metabolism and with vitamin-B6, and alters liver microsomal enzymes interfering with cytochrome-P-450 production. Urinary metabolites of carbon-disulfide catalyze the iodine/azide reaction providing a useful biological monitor. The morphologic abnormalities underlying peripheral neuropathy produced by carbon-disulfide in animal studies are discussed. Axonal degeneration is reported, possibly due to covalent binding of highly reactive sulfur to thiol groups of enzymes and proteins essential for the normal function of axonal transport. Engineering controls on exposure, provision of appropriate respiratory protective devices for emergencies, worker education, and medical surveillance are recommended.

Special Nerve Functions and Colour Discrimination in Workers with Long Term Low Level Exposure to Carbon Disulphide
Ruijten, M. W. M. M., H. J. A. Salle, M. M. Verberk, and H. Muijser
British Journal of Industrial Medicine, Vol. 47, No. 9, pages 589-595, 28 references September 1990 CODEN: BJIMAG

An investigation was conducted to determine whether a low level exposure to carbon-disulfide (75150) (CS2) affected the peripheral nervous system and color vision, and to evaluate the feasibility of proposed diagnostic methods. All male workers at a viscose rayon facility who had been exposed to CS2 for 10 years or more were examined. A total of 45 exposed workers and 37 comparisons participated in the investigation. Small effects of

exposure were demonstrated upon measurement of forced respiratory sinus arrhythmia (FRSA), muscle heart reflex (MHR), and conduction velocity of the slow motor fibers (refractory period). When cumulative exposure was considered, the significant differences in FRSA and MHR disappeared. For indices of motor nerve function, the absolute t-values increased with longer half times. The overall color confusion index as well as those for red and green and for blue and yellow did not differ significantly between the exposed and the comparison workers. The authors suggest that a lifetime exposure of around 4 parts per million appears required to prevent the small observed effects.

Carbon Disulfide Exposure and Neurotoxic Sequelae among Viscose Rayon Workers

Aaserud, O., O. J. Hommeren, B. Tvedt, P. Nakstad, G. Mowe, J. Efskind, D. Russell, E. B. Jorgensen, R. Nyberg-Hansen, K. Rootwelt, and L. Gjerstad American Journal of Industrial Medicine, Vol. 18, No. 1, pages 25-37, 36 references 1990 CODEN: AJIMD8

A survey was conducted of carbon-disulfide (75150) (CS2) exposure, the degree of exposure, and the possible presence of neuropathy or encephalopathy in a group of workers from a rayon viscose factory. The investigation was performed in 1986 and included 16 workers from the only existing Norwegian rayon viscose factory which was closed in November of 1982. The subjects had been working in the factory's spinning department for at least 10 years. Two were former heavy drinkers, five had cardiovascular disease, and two had diabetes mellitus. Four workers had a cerebral concussion in their history. Three had received compensation due to CS2 damage. Eight workers had an encephalopathy, probably due to CS2 alone, and six had encephalopathy partly caused by CS2 exposure. There was a neuropathy in seven workers probably caused by CS2 alone, and in three workers CS2 exposure was a partial cause of neuropathy. Results indicated that the long term relatively moderate exposure to CS2, in association with high peak exposures to CS2 and hydrogen-sulfide, (7783064) (H2S) involves a substantial risk of developing neurotoxic disease.

Health Hazard Evaluation Report No. HETA-89-212-2020, Schlegel Tennessee, Inc., Maryville, Tennessee

Kiken, S., M. Newman, and C. Cox
Hazard Evaluations and Technical Assistance Branch, NIOSH, U.S. Department of Health and Human Services, Cincinnati, Ohio, Report No. HETA-89-212-2020, 17 pages, 15 references March 1990
REPORT NO.: NTIS-PB91-104-604

In response to a request from the company, an investigation was made of possible hazardous conditions at the Schlegel Tennessee, Inc. (SIC-2822), Maryville, Tennessee. Concern had been expressed

by union representatives about possible exposures to airborne nitrosamines, and the incidence of cancer in employees. The company produces rubber weather stripping for automobiles. Approximately 70 salaried and 200 production workers were employed by the company. Packers were experiencing dizziness, nausea, tingling lips, headaches, and depression. These problems had been attributed to odors in the workplace. Three employees had abnormal neurologic examinations and two others had reported abnormal urine iodine-azide tests. Current testing did not reveal any employees with 2-thiothiazolidine-4-carboxylic-acid (TTCA) in their urine, indicating that workers were not exposed to more than 0.5ppm carbon-disulfide (75150) (CS2). No personal breathing or area air sampling tests showed CS2 contamination above acceptable levels. No excess of reported cancers was noted following a standardized morbidity ratio analysis compared to the general population of the United States. Detectable levels of nitrosamines were not found. According to the authors, the etiology of medical problems experienced by workers could not be definitively determined. The authors recommend measures to lower potential hazardous exposures at this site.

NIOSH Testimony on Neurotoxic Chemicals by J. R. Froines, June 6, 1979 NIOSH
NIOSH, 17 pages June 6, 1979

This testimony summarized information dealing with worker exposure to neurotoxic chemicals. Following a definition of neurotoxicity, the testimony described some of the investigations on workers exposed to such chemicals on the job in which NIOSH has been involved. Included among the examples were the workers in Hopewell, Virginia, exposed to kepone (143500), who suffered loss of muscle coordination, loss of memory, and an eye movement disorder. Another episode of workers exposed to a pesticide involved leptophos (21609905). At the time of the exposure, leptophos was registered by the Environmental Protection Agency primarily for export. Animals administered a single oral dose showed weight loss, ataxia, and eventual muscle paralysis. Other potentially toxic chemicals were used in the preparation of leptophos including toluene (108883), a neurotoxic solvent. A third incident of worker exposure to pesticides occurred due to the release of o-ethyl-o-p-nitrophenylphenylphosphonothioate (2104645) (EPN), at a manufacturing site in Chicago Heights, Illinois. Neurological signs of distress included muscle weakness and cerebellar signs of toxicity. Cases of worker exposure to the following solvents were also reviewed: methyl-n-butyl-ketone (591786) and carbon-disulfide (75150). Exposures to metals were also reviewed including lead (7439921), arsenic (7440382), and mercury (7439976). Early detection of exposures to neurotoxic agents was briefly considered.

The Application of Psychological Methods for Evaluation of Effects of Occupational Exposure to Neurotoxic Substances
Indulski, J. A., and B. Dudek
Polish Journal of Occupational Medicine, Vol. 1, No. 2, pages 154-165, 18 references 1988

The role of psychological methods in evaluating the effects of neurotoxic substances was discussed. Traditional psychological techniques for detecting early (subclinical) symptoms resulting from exposure to neurotoxins were considered. These have been based primarily on administering a battery of tests that evaluated variables such as eye and hand coordination, two hand coordination, palm movement precision, tapping speed, manual dexterity, finger dexterity, and simple and choice reaction time. The results of a study of the neurobehavioral effects of carbon-disulfide (75150) on 126 workers were discussed. The subjects were administered a battery of 12 psychomotor tests. Their performance on nine tests in the battery was significantly impaired. The extent of impairment was greater than that associated with aging. Analyzing the results of psychological studies of persons exposed to neurotoxic substances was discussed. New approaches for applying psychological methods in evaluating the effects of occupational neurotoxin exposure were considered. These included the Neurobehavioral Core Test Battery (NCTB) developed under the auspices of the World Health Organization, microcomputerized test batteries, subjective techniques, and cognitive methods. The authors recommend that the NCTB battery always be used regardless of any other tests administered. This will facilitate comparing data and assist in developing a database.

Structural Aspects of Experimental Carbon Disulfide Neuropathy
Szendzikowski, S., J. Stetkiewicz, T. Wronska-Nofer, and I. Zdrajkowska Internationales Archiv fuer Arbeitsmedizin, Vol. 31, No. 2, pages 135-149, 47 references 1973 CODEN: IAANBS

Structural changes in the central and peripheral nervous system of Wistar-white-rats after exposure to carbon-disulfide (75150) (CS2) were studied. Rats were exposed to CS2 vapors at a concentration of 1.5 milligram per liter of air for 5 hours daily, 6 days per week, and killed after 1 to 15 months and prepared for histological and histochemical examination of tissues. Animals retained good general conditions until month 7 of exposure. After 5 to 6 months, histological lesions were observed in the spinal cord and sciatic nerve of rats exposed. Structural changes were observed in myelinated fibers after 1 to 2 months but no indisputable evidence was found of injury to neuronal perikaryons. The authors conclude that histological lesions of neuropathy due to chronic CS2 exposure were observed in rats prior to physical deterioration.

Study of Neurological and Neurophysiological Impairment in Carbon Disulphide Workers

Gilioli, R., C. Bulgheroni, P. A. Bertazzi, A. M. Cirla, M. Tomasini, M. G. Cassitto, and M. T. Jacovone

Medicina del Lavoro, Vol. 69, No. 2, pages 130-143, 30 references 1978 CODEN: MELAAD

Neurological and neurophysiological effects of carbon-disulfide (75150) exposure were studied. Two hundred and fifty four workers in a viscose rayon factory (SIC-2823) were grouped according to carbon-disulfide (75150) exposure risk category, exposure duration, age, and alcohol consumption history. Fifty four nonexposed workers formed the comparison group. Central nervous system functions and peripheral nerve functioning were evaluated. Central nervous system impairment increased with exposure risk and was manifest by depression and anxiety with reduced vigilance, attention, and memory function. A linear increase in minimal neuropathies also occurred with increased exposure risk categories. Ophthalmoscopic test results indicated a trend toward an induction of normal vascular function with increasing exposure risk. Electroencephalographic results were not related to exposure risk. Alcohol consumption was clearly a factor in the central and peripheral nervous system function decline. Psycho organic disturbances and minimal neuropathy also were evident after 5 years of exposure up to 60 milligrams per cubic meter of carbon-disulfide. The authors conclude that workers exposed to carbon-disulfide are at risk for neurologic disturbances and should be transferred to other work assignments after a maximum of 5 years of exposure. (Italian).

Conduction Velocity in the Peripheral Nerves of Rats with Chronic Carbon Disulphide Neuropathy

Knobloch, K., J. Stetkiewicz and T. Wronska-Nofer

British Journal of Industrial Medicine, Vol. 36, No. 2, pages 148-152, 9 references May 1979 CODEN: BJIMAG

Conduction velocity in the sciatic and tibial nerves of 143 white-rats was measured after exposure to carbon-disulfide (75150) (CS2) vapor at concentrations of 0.9 milligrams per liter (mg/l) or 1.6mg/l of air for periods of 1.5, 3, 6, 9, and 12 months. Rats exposed to 1.6mg/l of CS2 exhibited general physical symptoms of CS2 intoxication after 4 months; rats exposed to lower concentrations appeared healthy throughout the experiment. Electrophysical changes preceded symptoms of intoxication. Measurements of conduction velocity were performed on the day following each exposure period and again 3 and 6 months after the last day of exposure. A reduction in conduction velocity on the day after exposure was observed, the extent of this reduction was related to the concentration and duration of exposure. Complete recovery of conduction velocity was observed after a short intensive period of exposure to CS2. Long term exposure (3, 6, or 9 months) caused a nonreversible reduction in conduction velocity.

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Schaumburg, H. H. and P. S. Spencer
Departments of Neurology, Neuroscience and Pathology (Neuropathology), Albert Einstein College of Medicine, Bronx, New York, Grant No. R01-OH-00535, 34 pages, 27 references 1979

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