IRPTC

Scientific Reviews
of Soviet Literature
on Toxicity and Hazards
of Chemicals

Chloroprene

14

UNITED NATIONS ENVIRONMENT PROGRAMME

UNITED NATIONS ENVIRONMENT PROGRAMME (UNEP)

INTERNATIONAL REGISTER OF POTENTIALLY TOXIC CHEMICALS (IRPTC)

USSR STATE COMMITTEE FOR SCIENCE AND TECHNOLOGY

USSR COMMISSION FOR UNEP

Series "Scientific Reviews of Soviet Literature on Toxicity and Hazards of Chemicals"

Chloroprene

Edited by N. F. Izmerov
Corresponding Member
USSR Academy of Medical Sciences

14

CENTRE OF INTERNATIONAL PROJECTS GKNT

Moscow 1982

Compiled by Cand of Sci. (Biol.) L.S. Katosova

The series represents a comprehensive study of Soviet literature on toxicity and hazards of Chemicals and is published by Centre of International Projects, USSR State Committee for Science and Technology under the USSR/UNEP Project "Control of Hazards Posed by Chemicals to Human Health and the Environment" implemented in cooperation with the Research Institute of Industrial Hygiene and Occupational Diseases, USSR Academy of Medical Sciences.

Information carried by the review is in line with the data profile structure elaborated by the International Register of Potentially Toxis Chemicals.

CHLOROPRENE

Chloroprene (2-chlorbutadiene – 1,3; $CH_2 = CCl - CH = CH_2$) is a liquid with a molecular weight equal to 88.54.

At 0°C the density of chloroprene is 0.978 g/cm^3 and its boiling temperature is 59.4°C.

The water solubility of freshly prepared and stabilized chloroprene (if continuously shaken for 15-20 h) is 0.05 ml in 100 ml (20°C) [2].

Stabilizers (neozone D, pyrogallol, pyrocatechin, hydroquinone and other anti-oxidizers) are to be added to keep it from polymerization [1, 2].

Depending on its production process chloroprene contains a variety of impurities, such as chloroprene rectificate, produced by the hydrochlorination of vinylacetylene, containing 99.6 - 99.9 per cent of chloroprene, 0.1 - 0.02 per cent of vinylacetylene, 0.2 - 0.05 per cent of dichlorbutene and the remaining percentage of acetaldehyde.

Chloroprene synthisis from butadiene through 3.4-dichlor-I-butene results in chloroprene containing the admixtures of I-chlor-1.3 butadiene (one per cent) and acetaldehyde (0.2 per cent.). Apart from the main product of butadiene chlorination, some byproducts, such as tetrachlorbutane and others, are also formed [3].

PRODUCTION PROCESS(ES)

Chloroprene can be produced by several methods. Thermally it can be obtained by non-catalytic thermal dehydrochlorination of 1,2,3- or 2,2,3-trichlorbutane at high temperature.

Dehydrochlorination of 1,2-dichlorbutene-3 and 1,3-dichlorbutene also ends with the production of chloroprene.

Chloroprene can be obtained from the addition of vinyl chloride to acetylene.

Broad acceptance throughout the industry goes for the process based on catalytic synthesis of chloroprene from vinylacetylene and hydrogen chloride.

USE

Chloroprene is monomer going into the production of synthetic rubbers and latexes.

Depending on the polymerization process involved, polymers of different modification can be developed from chloroprene, sharply contrasting with one another by their physical and chemical properties, viz.: soft, elastic and soluble in benzene alpha-polychloroprene; and u-polychloroprene an elastic insoluble rubber like polymer; psi-polychloroprene and beta-plychloroprene, a chloroprene, a chloroprene dimer isomer [1].

PATHWAYS INTO THE ENVIRONMENT

Chloroprene is released into the environment during its production as a result of its various uses in industry.

Chloroprene is released into the air in the process of acetylene synthetic rubber production and processing. The penetration of chloroprene into the air occurs during cleaning, opening and disassembling the equipment and in sampling. Chloroprene comes into the atmosphere from the exhausts of ventilation units, sewage hatches and other sources[4].

Chloroprene in the free non-polymerized state in latexes from which it is released into the atmosphere of the workplaces where chloroprene latexes are used.

Chloroprene, together with other chlor-containing unsaturated hydrocarbons, is discharged in considerable quantities with the disposal waters of the acetylene-based synthetic rubber production facilities. The chlor-containing unsaturated hydrocarbons in the waste waters of the monovynilacetylene hydrochlorination and chloroprene rectification department amount to 8,960 mg/l or 556 mg/l in conversion to bromide in the plant's total discharge[7].

CONCENTRATIONS

Chloroprene contents to be found in the atmosphere of the workplaces at a plant using chloroprene latexes to make protective gloves, mittens and radiohoods varies from 1 to 8 mg/m^3 (in different production sections). In a shoemaking factory the chloroprene concentration reached 2-7 mg/m^3 in workplaces where LNT-1 latex was used[6].

In the hygienic evaluation of the atmosphere around the production facility which discharges chloroprene into the air it was noted that a specific chloroprene smell makes itself felt in the atmosphere, finds way into dwellings, and contaminates objects as well as the fruit and vegetables grown in the area. The average single chloroprene concentrations within 250 m to 7 km away from the source of pollution were in the range 8.9 to 2.9 mg/m^3. The average daily concentrations varied from 1.87 to 0.15 mg/m^3. The apricots, peaches

and grapes, grown at the distance of 2-4 km leeward of the source of discharge, revealed a somewhat lower sugar content and higher acidity than in control. A specific chloroprene flavour and smell in the peaches was detected[4].

Chloroprene concentrations of 28.45 to 0.199 mg/m^3 were detected in the atmosphere within the range of 500 to 1500 m from the chloroprene rubber production facility[8].

ENVIRONMENTAL FATE TESTS

Elevated temperatures and light add intensity to the reaction of self-oxidation of chloroprene[1].

In water chloroprene possesses low stability. With the addition into water of 0.1 mg/l of chloroprene its specific smell will no longer be detected already in 15 to 60 minutes. Reduction of the cloroprene quantity in water due to its volatility. Other factors to affect the stability of chloroprene in water involve reaeration, temperature, and so on. After shaking open vessels with water containing 50 and 100 mg/l of chloroprene less than 8 mg/l was left one and half hour later. A high water temperature promotes the fast disappearance of chloroprene.

The threshold of chloroprene smell perception in water at room temperature occurs at the level of 0.1 mg/l [7].

BIOCONCENTRATION/CLEARANCE TINE/MAMMALIAN METABOLITES

The mechanism of chloroprene toxicity has to do a great deal with the formation of peroxides which augment the process of lipid peroxidation. The amount of lipids peroxides in the bodily tissues, particularly the liver increases in response to intoxication with chloroprene.

MAMMALIAN TOXICITY ARRAY

Chloroprene is moderatly toxic for a variety of pathways into the organism. A single subcutaneous injection of 10.000 mg/kg chloroprene caused the death of all experimental mice [13].

CL_{50} of chloroprene for mice is 1300 mg/m^3 (duration of exposure is 2 hours). Under the same conditions the death of all test rabbits and cats resulted from the chloroprene concentration of 19,000 and 11,000 mg/m^3, respectively.

A repeated four-hour inhaltion of chloroprene in the concentration of 500 mg/m^3 caused the death of some part of the test animals after four to eight exposures. The most pronounced changes were those the mice had developed in their lungs (plethora, hemmorhages, edemas and pneumonia). There was stagnant plethora in the other internal organs [14].

An injection of male rats with 4,000 mg/kg chloroprene dose given subcu-

taneously every other day for one month caused despression and the loss of appetite in the animals [14].

When finding its way into the organism, chloroprene causes disorders of various organs and systems.

Chloroprene ingestion with the food in a dose of 15 mg/kg during 6 successive months led to pathomorphological changes in the internal organs in rats (brain, liver, kidneys, etc.) [7].

The inhalation by female rats of the chloroprene concentrations of 1400 + 20;30 + 9 and 1 + 9 mg/m^3 for six months during 6 days each week in a five-hour exposure produced a change in the ultrastructure of the adrenal cortices [15] and the ultrastructure of the blood capillaries in the ovaries of the animals [16].

A decline of liver glycogen was noticed in rabbits exposed to chloroprene with the initial concentration of 100 mg/m^3 being gradually increased to 500-600 mg/m^3 over four months.

The chloroprene concentration of 500-1000 mg/m^3 given for 10 - 45 days during four hours each day to dogs induced a rise of the pyroracemic acid content in their blood [17].

Albino rats were subjected to 60-day round-the-clock poisoning with chloroprene concentrations equal to 0.48, o.22 and 0.83 mg/m^3. The threshold chloroprene concentration according to the effect on the SH-groups of the brain and the adenosinetriphosphatase activity of the liver in albino mice stood at 0.22 mg/m^3 [18].

Chloroprene given intraperitoneally to rats at the rate of 600 mc/mole per 100 g body weight for 7,15 and 30 days resulted in phase changes in the activity of the penthose-cycle enzymes in the liver [13].

Finally, chronic inhalation exposure to chloroprene, its concentration equal to 1.69 + 0.087 mg/m^3 four hours a day was responsible for the inhibition of the nervous system's excitability and modification of the liver function in male rats [5, 19, 20].

The mechanism of the toxic effect of chloroprene is related to the formation of peroxides which intensify the lipid peroxidation. In the case of chloroprene intoxication an amount of lipid peroxides grows in the organism tissues, particularly in the liver [12].

There is vast clinical evidence proving the hazardous impact of chloroprene on the workers. 60 persons occupationally exposed to chloroprene for the period from 1 to 20 years were examined in a production environment where the chloroprene concentration in the atmosphere of the workplaces was 20-24 mg/m^3. There were 56 women and four men aged 18 to 48 years. For the most part, they were found to have nervous system impairments whose severity increased with the workers' record of employment [21]..

Another integrated study into the health of female exployees at a production facility manufacturing chloroprene rubber items covered 103 women from 16 to 50 years and employed for the period from 1 to 20 years.

The chloroprene concentration in the air of the working zone was in the range of 6 to 10 mg/m^3. The females having a many-year experience of contact with chloroprene felt sudden fits of asthenia accompanied with vertigo,

palpitation and unpleasant sensations in the heart. In some of them (4.8 per cent) these fits involved also a loss of consciousness. Of the trophic irregularities there was the shedding of hair in 44.6 per cent of the examined persons and the brittleness of nails in 19.7 per cent. The tendon reflex was found to revive in 39 persons or 37.7 per cent, and the tremor of fingers in 16 persons or 15.5 per cent. Thermoregulation was seen to have been dislocated in one half of those examined and various deviations were found in the regulation of the cardiovascular system.

22.3 per cent of the women showed symptoms suggesting an organic lesion of the nervous system.

The menstrual function was found deficient in 43 per cent of the female employees [22].

The findings of a medical examination of 65 workers from a production facility manufacturing chloroprene latex items, of whom 12 were male and 53 female persons with the duration of employemnt from 5 to 20 years, showed the frequency of complaints over increased fatigueability, headache and pain in the cardiac region to grow with the longer duration of employment. Nearly one half of those screened exhibited subdued heart sounds and one fifth a decrease of the blood pressure. The chloroprene content in the respiratory zone varied among the employees from 2 to 8 mg/m^3.

Note was taken of the diminished hemoglobin and reduced number of erythrocytes in the peripheral blood. A related otorhinolaryngologic examination identified 19 per cent of the workers as having chronic tonsillites, hyperplastic pharyngites and hyperplasias of the lymphoid throat ring. The gynecologic examination disclosed menstrual disorders in 47 per cent of the female employees [5].

Analysis of the data emergent from physiologic examinations of 20 woman workers 19 to 23 years of age and with the duration of employment between 2 and 4 years revealed the paradoxial daily dynamic pattern of blood pressure as its systolic and diastolic levels became lower. The pulse rate at work reached 93.1 + 1.2 per second. Toward the end of their working hours the women in the main group showed an extended latent period of the visual-motor reaction by more than 12 per cent of the initial level. Disturbances were also detected in the function of the olfactory analyser and vestibular apparatus and changes were identified in the cardio-vascular system [5, 19, 23].

SPECIFIC TOXICITY STUDIES

Carcinogenicity. Evidence about the carcinogenic activity of chloroprene is utterly conflicting. A study of the blastomogenic activity of chloroprene was conducted by the accelerated testmethod. Based on the reaction of the sebaceous glands, it was inferred that chloroprene applications to the skin of albino mice, whether singly or repeatedly, for a period of 2 to 30 days, possessed no blastomogenic activity [24].

Investigation of influence by chloroprene that exudes from the polychloroprene latex upon the specific functions of the female organism in women who worked on the shop floor (147 persons) and came in direct contact with

chloroprene, as compared with the staff of the plant management office (105 persons) and a group of women employed at the plant's child-care facility, who served as control (100 persons) disclosed that: tumors occurred in 8.2 per cent of the shop floor workers, as against 1.9 per cent in the office group and 1 per cent in the control group.

Mutagenicity. The mutagenic activity of chloroprene was identified using the method of metabolic activation of the substance in animal organism (mice, line CVA) with the aid of Salmonella tuphimurum 1535 (S.t.). The intraperitoneal administration of 2 ml of the S.t. culture and the intramuscular infection of the chloroprene in dose of 375 mg/kg increased the frequency of mutations three times. With the 750 mg/kg chloroprene dose the mutation frequency leapt up more than eight-fold [26].

On exposure to 48 days of chloroprene inhalation by four hours daily and in the concentrations 3.8 + 0.45 and 39 + 4.7 mg/m^3 albino male rats developed an obvious mutagenic effect in the cells of their sexual and somatic tissues. There followed a noticeable expansion of the total embryonic mortality in the females, coupled with the poisoned males, and a rise of chromosomal aberrations in the bone-marrow cells of the test animals [27].

Following 2.5 month exposure to chloroprene concentrated to 1.69 + 0.087 and 0.15 + 0.0059 mg/m^3, in male rats, it was possible to detect an increased frequency of dominant lethal mutations in the sexual cells of the poisoned animals - quite unlike the changes induced in rats by the chloroprene concentration of 0.051 + 0.0025 mg/m^3 [19].

In the male line C57BL/6 mice the frequency of dominant lethal mutations in the sexual cells was found to increase as a result of two-month exposure to chloroprene concentrations equal to 3.5 + 0.7 and 1.85 + 0.18 mg/m^3. The increasing chromosomal aberrations in the bone-marrow cells were seen to have resulted from the above concentrations and from the concentrations of 0.32 + 0.06 and 1.85 + 0.18 mg/m^3 as well. When used in the concentrations of 0.054 + 0.024 and 0.064 + 0.01 mg/m^3, chloroprene caused no mutagenic effect in the mice [5, 19].

A cytogenic analysis of peripheral blood lymphocytes in 18 workmen engaged in chloroprene production disclosed an excess of chromosomal disorders in them, well above those in control or the level of spontaneous changes of this index. The average chloroprene concentrations at the workplaces was 18 mg/m^3 [19, 28].

A cytogenic examination of 17 women aged 19 to 23 years and exposed to chloroprene latex for from 2 to 4 years, with the chloroprene concentration in the respiration zone equal to 3 - 7 mg/m^3 revealed the expansion of chromosomal aberrations in the employees' blood cells [29].

Neurotoxicity / Behaviour. Like other unsaturated hydrocarbons chloroprene distinctly effects the nervous system. The effect of chloroprene on conditioned reflex activity in rats was investigated by giving 0.005 mg/kg chloroprene to rats in the first group, 1.5 mg/kg to those in the second group and 15 mg/kg to the third group. Chloroprene was administered with food every day less Sundays, for six months. The rats in the third group and some of the rats in the second group exhibited readily apparent changes of their higher

nervous activity. The changes were reversible. In the first group of rats no functional changes of conditioned reflex activity were detected. The impairments of conditioned reflex activity that occurred in some individual cases were attributed to exposure to the 15 mg/kg dose otherwise accompanied also with pathomorphological changes[7].

With a 4.5-month chloroprene exposure in the concentration of $1.69 + 0.087$ mg/m^3 the inhibition of the nervous system's excitability according to the summation-threshold index was observed in the male rats [5, 19, 20].

A study of the action by chloroprene upon the central nervous system in humans as determined by the adaptometric method showed that the inhalation of chloroprene at the rate of 0.25 mg/kg produced no changes in the course of dark adaptation, as compared with the data resulting from the inhalation of clean air. However, when used in the concentration of 0.4 and 0.55 mg/m^3 chloroprene did induce these changes. With these concentrations a process of excitation is seen to emerge in the cerebral cortex, to be irradiated from the olfactory to the visual analyser. Under higher chloroprene concentrations light sensitivity was found to fall off and the inhibition processes to increase accordingly [4].

The evidence obtaining from a five-year observation program of employees who worked at a factory producing synthetic rubber from acetylene has permitted an identification of the initial stages of intoxication with chloroprene which become evident in the form of neurasthenia with enhanced excitability of the subsorticalvegetative organs of the nervous system. Pronounced chloroprene intoxication showed up as toxic encephalopathy and encephalomyelitis with hyporeactivity of the vegetative nervous system [30].

Among 444 chloroprene shop employees who had been under observation in the period of 1956 through 1963, including 92.6 per cent of male workers, there were detected: tremor of the hands and eyelids, sharply and moerately expressed red dermographism, intense perspiration, elevation of the muscular torulus and the presence of trophic disorders. In some cohort members, beyond the functional changes enumeraed above, there were symptoms indicative of an organic lesion of the nervous system in the nature of encephalopathy [31].

60 workers, incluiding 56 females and 4 males in the age bracket from 18 to 48 years, who were occupationally exposed to chloroprene for the period from one to 20 years were examined. The chloroprene concentration being in the order of 20-24 mg/m^3, a variety of functional and in some workers (10 persons) organic lesions of the central nervous system were identified, together with a reduction of the conjunctival and corneal reflexes, revival of the tendon reflexes following the functional pattern, tremor of the fingers and eyelids, general local hyperhydrosis, discruption of thermoregulation and changes of arterial pressure and pulse [21].

In the post-apprenticeship female workers exposed to chloroprene concentrations in the order of 6-10 mg/m^3 one could detect toxic neurosis with vegetative disorders. Twenty three of the 130 persons in the study group, or 22.3 per cent, showed symptoms revealing an organic lesion of the nervous system. The most frequent occurrence was the disappearance or reduction of the

corneal and conjuctival reflexes. Five persons (6.8 per cent) revealed an impairment of the peripheral nervous system in the form of vegetative polyneuritis [22].

Chloroprene concentrations within the range 1.8 mg/m^3 acted upon the workers causing a distubance of the function of the central nervous system which showed itself in an extended latent period of the visual-motor reaction compared with the control group, and diminished olfactory sensitivity [5, 19, 23].

Potentiation. The influence of vitamins on the toxic effect of chloroprene was studied.

Arrival into the test dogs' organism of a one mg quantity of vitamin B_1 with the morning meal during ten days brought down the pyroracemic acid content in the blood of the chloroprene-affected animals [17].

Administration of vitamin E to dogs together with chloroprene promoted the favourable course of chloroprene intoxication [12].

Systematically conducted vitamin therapy with B_1 and C, coupled with balneotreatment abated, to a considerable degree, the severity of pathological developments in the the persons chronically exposed to chloroprene [30].

There is evidence testifying to the nature of a combined effect of chloroprene and the products discharged from various latexes.

Combined action by chloroprene (1.96 mg/m^3), dodecylmercaptan (5.02 mg/m^3) and ammonia (19.8 mg/m^3), and chloroprene (2.8 - 2 mg/m^3) and methylmetacrylate (4.0 - 0.25 mg/m^3), discharged from MX latex, set in train a series of changes in albino rats, not least those occurring in the bone-marrow cells and invloving a significant rise of chromosomal aberrations. The cytogenetic effect became obvious both, after single and four-month exposure to the poison [32].

The cumulative effect of the same poisons but in somewhat lower concentraions, 0.89 - 0.9 mg/m^3 of chloroprene, 0.12 - 0.03 mg/m^3 of dodecylmercaptan, 2.07 - 0.27 mg/m^3 of ammonia and also 0.54 - 0.08 mg/m^3 of chloroprene and 0.74 - 0.39 mg/m^3 of methylmetaacrylate also induced a mutagenic effect in the somatic cells of albino rats.

11 employees were examined after having exposed to the cumulative effect of chloroprene (2 - 7 mg/m^3), dodecylmercaptan (1 - 2.5 mg/m^3) and ammonia (4 - 10 mg/m^3) together with another five workers who were occupationally exposed to a mixture of chloroprene (2 - 22.2 mg/m^3) and methylacrylate (0.5 - 2 mg/m^3). As a resu^{1+}, a greater frequency of chromosomal aberrations was discovered in the blood cells of the employees of both these production divisions [16].

Reproduction. There is ample evidence of the unfavourable impact of chloroprene on the spermatogenesis.

Chloroprene concentrated to 1.69 - 0.087 mg/m^3 induced the atropy of tests in the male rats (5 animals out of eight) upon 4.5 months of exposure. In the animals with non-atrophied tests the dead spermatazoid were expanded, their time of mobility curtailed and their resistance to the acidic medium reduced. This effect was less obvious as induced by the chloroprene concentration of 0.15 - 0.0059 mg/m^3. The total embryotic mortality was seen

to have increased in the females, coupled with the males, suffering from exposure to the poison in the two concentrations selected.

With the inhalation of chloroprene in the concentration of 0.051 - 0.006 mg/m^3 the gonadotropic effect failed to be detected [5, 19, 20].

When acting upon male rats for 48 days during four hours a day with chloroprene concentrations equal to 3.8 - 0.45 and 3.9 - 4.7 mg/m^3 and coupling them with intact females, an excess of the total embryonic mortality was shown in the posterity obtained from the females [27].

It was found in a study addressed to the gonadotropic effect of the poison on mice, line C57Bl/6, that exposure to chloroprene concentrations equal to 3.5 - 0.7 and 0.32 - 0.06 mg/m^3 brought about a change of spermatogenesis by pushing up the number of tubules with peeled-off germinal epithelium. When applied in the concentration of 0.064 - 0.011 mg/m^3 chloroprene had no damaging action on the gonads in mice [19].

In the cloroprene shops where the latter's concentrations were equal to 13.2 - 3.2 and 36.2 - 2.7 mg/m^3 medical examination of workers disclosed functional dislocation of spermatogenesis in those with the duration of employement at the production facility from 6 to 10 years, and morphological disturbances among the workers with the work record of 11 years and above. A questionnaire poll indicated that cases of spontaneous abortions among the workers' wives had three times their frequency in the control group [19, 33].

The impact of chloroprene on the women's sexual sphere was studied.

Inhalation exposure of chloropene in the concentration of 1 - 0.9; 30 - 9 and 1400 - 20 mg/m^3 for a period of six months (5 hour exposure six days a week) resulted in changes in the ultrastructure of the blood capillaries in the ovaries of female albino rats. With a growing chloroprene concentration the degree of irreversible changes (destruction of organellas and cell membranes) in the endothelial cells increased [16].

An examination conducted of 147 women on the shop-floor directly in contact with chloroprene (1st group); 105 female office employees and clerical staff of the plant management office (2nd group), and 100 female exployees of the combined-care facility as control. Upon examination 66 gynecologic cases were identified in the first group, 25 in the 2nd and 7 in control. Primary and seconday infertility was reported as representing 6.1 per cent in the first group, 3.8 per cent in the second group and 2 per cent in the control group [25].

When examining the 109 female shop-floor workforce directly exposed to chloroprene at their workplaces, its concentration being 6 - 10 mg/m^3, a menstrual function disorder became revealed in 47 women, following the type of hypomenstrual sundrome in most of them [22].

Teratogenicity. In the rats which inhaled chloroprene in the concentrations of 28.45, 0.727, 0.523 and 0.199 mg/m^3 some signs of disturbed embryogenesis were identified. Increased death rate of the embryos were established and irregularities found out in the development of the placenta, particularly its weight loss, together with changes of the liver weight in 20-day embryos [8].

The duration of chloroprene action is important for the materialisation of the teratogenic effect.

The administration of chloroprene to pregnant rats given intragastrically

0.5 mg/kg if chloroprene made the embryotropic effect much more prominent under day-to-day exposure than on exposure at particular stages of pregnancy [34].

The inhalation of chloroprene in the concentration of 4 - 0.7 mg/kg all through the period of pregnancy affected rats by inducing the development of malformation in the embryos whereas the same concentration of the poison but given on individual days of preganancy had a teratogenic effect. The largest quantity of cerebral hernias was found on the 5th and 6th days of exposure to the substance [34].

The impact of chloroprene by the inhalation of 3 and 4 mg/m^3 four hours a day for the duration of pregnancy caused a disturbance of the intrauterine development in rats, other changes being the expanded rates of anti-natal mortality, weight loss and a shorter length of the diaphyses of some bones in the embryos' extremeties. The action of chloroprene in the concentrations of 0.13 and 0.6 mg/m^3 resulted in the significant death loss of the posterity. In like manner 3, 4 and 0.6 mg/m^3 of chloroprene brought a weight loss in the posterity and disturbances in some of the functions of the parenchymatuos organs. The quantity of 0.056 mg/m^3 proved to be ineffective according to the indices selected [35].

Privmary Irritation. The irritating action of chloroprene fumes is not distinctly identifiable. The inhalation of 2,000 to 6,000 mg/m^3 of chloroprene for a fleeting one minute caused an almost imperceptible irritation of the mucous membrane of the eyes and throat in humans [13].

EFFECTS OF ORGANISMS IN THE ENVIRONMENT

Used in concentrations above 10 mg/l, chloroprene suppresses the processes responsible for self-purification from organic impurities. With the concentration of 50 mg/l the extent of inhibition of biochemical oxygen comsumption represented 40 per cent compared with control. The threshold concentration was found to be at the level of 1 mg/l and no changes were detected in the growth and death of bacteria in response to chloroprene concentrations varying from 0.1 to 10 mg/l [7].

Note was taken of the lower sugar content and higher acidity of apricots, peaches and grapes cropped at the distance of 2-4 km from the cloroprene discharge source [4].

SAMPLING / PREPARATION / ANALYSIS

The photometric (colorimetric) method for chloroprene determination in air is based on the production of staining as it adds to p-nitrophenyldiazonium. In sample-taking, the air is driven through an immobile silica gel layer at the rate of 0.5 l/min. The extraction of chloroprene from the silica gel is made by thermal desorption, with its subsequent trapping into acetic acid. The determination is sensitive to 5 mcg [10, 11].

Under the gaschromatographic method of determining the chloroprene content in air, when it is present in a mixture with other substances the maximum complete separation of the components is achieved on INZ-600, a solid carrier with the liquid phase of tricresylphosphate applied over it. The determination is performed on a chromatograph with a plazma-ionization detector. The method is sensitive to 1 mg/m^3 for the 10 ml volume of the inserted sample. The sample concentration methodoly contributes to the method's increasing sensitivity, taking it to 0.02 mg/m^3 for the 5-litre volume of the air in transit through it[9].

An advanced method of gaschromatographic chloroprene determination in air has the sensitivity of 0.01 mcg and a determination error of 10 per cent [36].

TREATMENT OF POISONING

Faced with acute intoxications one should carry the victim away from the chloroprene-contaminated room. Tranquilizers and sedatives are indicated. For more serious chloroprene intoxications it is necessary to inhale oxygen, administer cardiac drugs, glucose with ascorbic acid, calcium gluconate or calcium chloride. In sever cases compulsory hospitalization is the key. It is a good practice to use vitamins B_3 and E_1, lipoic acid and calcium panhalate[2].

RECOMMENDATIONS / LEGAL MECHANISMS

For personal protective equipment it is good to use industrial filter respirator moder A or self-contained hose respirator with a forced supply of clean air for high concentrations. Skin protection demands the use of protective working clothes.

Preliminary and regular examinations of those engaged in the production of chloroprene and synthetic rubbers should be planned once in twelve months. The examination team should be manned by a therapeutist, neuropathologist, dermatologist and gynecoligist. The determinations are made with regard to bilirubin, hemoglobin and erythrocytes in the blood and urobilin in the urine. Regular examinations must cover all persons coming in contact with chloroprene or chloroprene-containing products. The recommendations provide for high-protein diet inclusive of an extra 200 g of cottage cheese and 50-60 g of vegetable oil, compulsory vitaminization for one month two times a year (200 g of vitamin C and vitamin B_1, B_6 B_{15}). Direct contact with chloroprene must be denied to all persons under 18 years of age [2, 23].

The maximum allowable concentration of chloroprene in the air of working zone is 0.05 mg/m^3 and the chloroprene MAC for the water bodies used as sources of drinking water or for recreational purposes (the organoleptic danger limit) is 0.1 mg/l [36].

REFERENCES

1. E.B.Badasyan, T.N.Rakhmankova "Chloroprene manufacture". In: Basics of Chloroprene Rubber Synthesis Process, Moscow, Khimiya Publ., 1971, pp.59-94.

2. N.V.Lazarev and E.N.Levina Ed. Hazardous Substances in Industry, 1976, 7th ed., v.1, pp.238-243.

3. N.G.Karapetyan, I.M.Dolgopolsky, A.L.Klebansky. Manufacture of Vinylacetylene and Chloroprene". In: Synthetic Rubber, Leningrad, Khimiya Publ., 1976, pp.710-723.

4. A.V.Mnatzakanyan. Experimental data inputs for determination of maximum allowable chloroprene concentration in the atmosphere" In: Maximum Allowable Concentrations of Atmospheric Pollutants", Moscow, Medgiz Publ., 1961, 5, pp.110-117.

5. Z.A.Volkova, V.N.Fomenko, Yu.M.Bagdinov, N.K.Byalko, L.L.Katosova, N.I.Ponomaryova, E.I.Tolcheva, R.M.Davtyan, Z.N.Zilfyan, T.I.Gurdzhiev, A.Sh.Khairullina. "A contribution to justification of the chloroprene MAC value in the atmosphere of workplaces". Gigiena truda i professionalnye zabolevaniya. 1976, 3, pp.31-36.

6. S.B.Bagramyan, A.S.Pogosyan, E.A.Babayan, R.D.Ovanesyan, S.M. Charyan. "Mutagenic action of low concentrations of volatiles evolving from polychloroprene latexes LNT-I and MX at their combined entry into the organism". Biological Journal of Armenia, Yerevan, 1976, v.XXIX, 4, pp. 98-99.

7. M.K.Khachatryan, T.A.Asmangulyan. "Maximum allowable chloroprene concentrations in water bodies". In: Hygienic Protection of Water Bodies from Contamination with Industrial Waste Waters. Moscow, 1969, 4, pp. 169-176.

8. A.V.Mnatzakanyan, U.G.Pogosyan, K.H.Apoyan, A.S.Kamayan. "Some aspects of embryotropic action on animals by chloroprene rubber productions discharges". In: Questions of Hygiene, Occupational Pathology and Industrial Toxicology at Synthetic Rubber Production Facilities, Ufa, 1972, pp.62-63.

9. K.N.Turusova, V.K.Khanina. "Chromotographic chloroprene determination in air". Gigiena i sanitariya, 1975, 7, pp.81-82.

10. Babina M.D. "Determination of volatile substances evolving into air from production facilities of shoe-making industry". In: New Developments in Industrial Hygienic Chemistry, Moscow, Meditsina Publ., 1969, pp.227-232.

11. M.D.Babina "Chloroprene determination in air". Journal of Analytical Chemistry, 1965, v.XX, II, pp. 1257-1259.

12. L.V.Semerdyan, V.G.Mkhitaryan. "Activity of penthose-cycle enzymes in rat liver under chronic chloroprene toxicosis and the role of vitamin E in the process". Journal of Experimental and Clinical Medicine. Yerevan, 1976, v.XVI, 5, pp.3-10.

13. E.N.Levina. "Toxicity of chlorbutadiene in acute experiments". In: Investigations in industrial toxicology, 1943, 5, pp.95-109.

14. E.N. Levina. "Toxic action of secondary poisoning with chlorobutadiene in mice and rats with special reference to its influence on hair cover". In: Investigations in Industrial Toxicology, 1948, 5, pp.111-112.

15. A.P.Markaryan, V.A.Shakhlamov. "Ultrastructure of adrenal corctices in albino rats under chronic chloroprene intoxication". Journal of Experimental and Clinical Medicine. Yerevan, 1975, v. XV, 5, pp. 27-31.

16. L.P.Markaryan, V.A.Shakhlamov. "Ultrastructure of ovary blood capillaries in albino rats under chronic chloroprene intoxication". Journal of Experimental and Clinical Medicine. Yerevan, 1975, v.XV, 4, pp.46-51.

17. S.V.Nikogosyan. "Influence of 2-chlorbutadiene-1,3 on glycogen content in the liver and on pyroracemic acid in the blood of test animals". Gigiena i sanitariya, 1959, 2, pp.32-34.

18. A.V.Mnatzakanyan. "Data inputs for justification of maximum allowable chloroprene concentration in the atmosphere". Gigiena i sanitariya, Moscow, 1964, 9, pp.13-18.

19. I.V.Sanotsky. Aspects of the Toxicology of Chloroprene: Immediate and Long-term Effects. Environmental Health Perspectives, 1976, v.17, pp.85-93 (in English).

20. R.M.Davtyan. "A contribution to toxicologic description of the action of chloroprene exposure on reproductive function in male rats". In: Toxicology and Hygiene of Products of Petrochemistry and Petrochemical Production Facilities, Yaroslavl, 1972, pp.95-97.

21. R.G.Yakupova, I.V.Chudnovskaya. "Some symptoms of nervous system lesion in workers occupationally exposed to chloroprene". In: Questions of Hygiene, Occupational Pathology and Industrial Toxicology in Synthetic Rubber Production Facilities., Ufa, 1912, pp.73-74.

22. I.V.Chudnovskaya, A.Sh.Khairullina, R.C.Yakupova, R.Sh.Liankuzova. "Integrated health studies of female employees producing chloroprene rubber items". In: Obstetric and Gynecologic Occupational Pathology, Kazan, 1973, v.42, pp.26-29.

23. Yu.M.Bagdinov, Z.A.Volkova, N.I.Ponomaryova, Z.N.Zilfyan "Working conditions and the state of some physiologic functions in female employees in production of chloroprene latex items". In: Obstetric and Gynecologic Occupational Pathology, Kazan, 1973, v.42, pp.12-19.

24. D.H.Gabrielyan, S.A.Papoyan. "Investigation of blastomogenic activity of some chemicals, using accelerated test method". Gigiena i sanitariya, 1977, 8, pp.74-76.

25. A.Sh.Khairullina. "Gynecologic morbility among female employees of a facility producing polychloroprene latex items". In: Obstetric and Gynecologic Occupational Pathology, Kazan, 1973, v.42, pp.83-89.

26. B.S.Fichidzhyan, K.D.Rybakova, S.E.Lokashina. The results of studying mutagenic activity of chloroprene on microorganisms". Journal of Experimental and Clinic Medicine, Yerevan, 1976, v.XVI, 5, pp.39-41.

27. R.M.Davtyan, V.N.Fomenko, G.P.Andreeva. "Influence of chloroprene upon generative function in male mammals". In: Toxicology of New Industrial Chemicals, Moscow, 1973, 13, pp.58-62.

28. L.D.Katossova. "Cytogenetic peripheral blood test of workers engaged in chloroprene production". Gigiena truda i professionalniye zabolevaniya, 1973, 10, pp.30-33.

29. V.N.Fomenko, P.D.Katossova. "Results of cytogenetic peripheral blood tests of female employees exposed to chloroprene latex". In: Obstetric and Gynecologic Occupational Pathology, Kazan, 1973, v.42, pp.33-36.

30. G.T.Muradyan. "Clinic forms of nervous system lesion as a result of chronic exposure to 2-chlorbutadiene". Journal of Neuropathology and Psychiatry, 1958, pp.1238-1240.

31. E.I.Gasparyan. "Extensive observation of health in chloroprene shop employees and prophylactic measures". In: Procs.of 14th Scientific Session in Honour of the 50th Anniv.of Great October Revolution. Yerevan, 1967, pp.111-113.

32. S.B.Bagramyan, E.A.Babayan. "Results of cytogenetic study into mutagenic activity of chemicals isolated from polychloroprene MX and LNT-I latexes". Biological Journal of Armenia, 1974, v.27, 6, pp.102-103.

33. I.V.Sanotsky, R.M.Davtyan, V.I.Glushchenko. "Study of masculine reproductive function as affected by some chemicals". Gigiena truda i professionalniye zabolevaniya, 1980, 5, pp.28-32.

34. L.S.Salnikova, V.N.Fomenko. "Comparative description of the embryotropic effect of chloroprene depending on exposure regime by different routes of entry into the organisms". Gigiena truda i professionalniye zabolevaniya, 1975, 7, pp.30-33.

35. L.S.Salnikova, V.N.Fomenko. "Experimental study of the influence by chloroprene on embryogenesis". Gigiena truda i professionalniye zabolevaniya, 1973, 8, pp.23-26.

36. "Methodological Instructions for Gaschromatographic Determination of Chloroprene in Air". In: Methodological Instructions for Determinations of Hazardous Substances in the Air, Moscow, CRIA, Morflot Publ., 1979, 13, pp.94-96.

Центр международных проектов ГКНТ
Проект СССР/ЮНЕП-МРПТХВ FP/1304-79-01 "Контроль опасности химических веществ для здоровья человека и окружающей среды" (англ.)

Подготовлено для публикации Стройиздатом

Напечатано Производственно-издательским комбинатом ВИНИТИ

Зак. 4483