Def Sci J, Vol 37, No 2, April 1987, pp 113-131.

An Overview of the Health Hazards Due to Toxic Exposure in the Indian Work Environment

S.S. Ramaswamy

Director General (Retd.)

Factory Advice Service and Labour Institutes
Ministry of Labour, Bombay

ABSTRACT

Since independence, there has been a phenomenal growth in the chemical industry, the number of units rising from 98 in 1947 to 964 in 1953 and 4364 in 1976. With the national demands ever growing, this trend of growth in chemical industry will continue in future also. The chemical units handle numerous toxic chemicals such as asbestos, benzene, carbon disulphide, carcinogenic dye intermediates, lead, manganese, organophosphorus pesticides, phosgene, vinyl chloride etc.

Being aware of the potential health hazards arising out of exposure to these toxic chemicals necessary safeguards against health hazards have been incorporated in the Factories Act 1948.

With nearly 100,000 tonnes of asbestos, over 100,000 tonnes of benzene, and considerably large quantities of other toxic chemicals being handled in the country, understandably, the random studies and surveys by research agencies have revealed the incidence of definite asbestosis (7 per cent), benzene intoxication in alkaloid extraction units (44.8 per cent), lead poisoning in storage battery units (10.6 per cent), carbon disulphide poisoning in viscose rayon units (20 per cent), mercury poisoning and intoxication in chloroalkali units (22.7 per cent), manganese poisoning in ferromanganese units (24 per cent), silicosis among slate pencil workers (54.7 per cent) etc.

Albeit such a condition, the cases documented in official reports are very few. Even the scattered studies by research institution in occupational health cannot be pooled to evolve a national picture, since, quite often there is no standardised approach in the studies undertaken by different institutions.

After discussing the findings of studies on various toxic chemicals and substances, the paper enumerates the present deficiencies in the current studies and suggest steps for obtaining comprehensive information on health hazards.

Received 10 october 1986

1. INTRODUCTION

Toxic exposure in the industrial work environment arises due to exposure to not only toxic chemicals handled as raw materials, intermediates and end products but also dusts of some natural fibres (for example, cotton dust) and mineral dusts containing free silica, and silicates (asbestos). In this regard, the industries of relevance are manufacturing, mining and to some extent, agriculture in which toxic pesticides are handled. To review the status of the health hazards arising out of such exposure, the following issues need to be considered:

(i) Growth pattern of industries inherent with such hazards.

(ii) Statutory and other safeguards available for controlling such hazards.

(iii) Documented statistics on the magnitude of such health hazards in the typical industries/processes.

(iv) Reasons for the shortfall in our efforts to control such health hazards in the past.

(v) Future strategy to control the hazards, and monitor the levels from time to time.

2. GROWTH OF INDUSTRIES AND TOXIC HAZARDS

Industrial development in the country has been practically a post-independence phenomenon, and its pace has been very rapid in the initial years. Considering the chemicals units alone, from a small number of 98 in 1947, they grew tenfold to 964 units in 1953, and to 4,364 in 1976.[1] As shown in Table[2-4] 1 the increase in the

Table 1. Trend of increase in production in some industries with inherent toxic hazards

Sector	Industry/product	Nature of inherent toxic hazard	Unit of production	Production in 1950-51	Production in 1982-83	Remarks
Manufacturing	Benzene	Benzene poisoning	000 tonnes		100.1	53.2 in 1970-71
	Caustic soda	Mercury poisoning	"	12	577	
	Cotton yarn	Byssinosis	Mn kg	534	999	
	Rayon yarn	Carbon disulphide poisoning	000 tonnes	2.1	124	
	Refractories	Silicosis	"	237	812*	*1978-79
	Storage batteries	Lead poisoning	Mn	0.187	1.58*	
Mining and minerals	Aluminium	Fluoride exposure	000 tonnes	4	211.5	
	Chromite	Chromium poisoning	"	17	336**	**1981
	Coal	Pneumoconiosis	Mn tonnes	32.8	136.9	
	Manganese ore	Manganese poisoning silicosis	000 tonnes	897	1526**	

Source of information – Ref: 2–4

Table 2. Major toxic substances associated with some of the industries which have grown after independence, and the occupational diseases caused by them

Sl. No.	Industry	Major toxic substances handled	Occupational diseases caused	Major symptoms
1.	Asbestos handling units	Asbestos fibre	Asbestosis, lung cancer, mesothelioma of the pleura	Respiratory functions impaired
2.	Solvent extraction units	Benzene	Benzene poisoning – haemopoetic disorders dermatitis	Haemoglobin content of blood reduced
3.	Viscose rayon	Carbon disulphide	Central and peripheral nervous system impairment	Peripheral neuropathy, muscular weakness, tremor, loss of memory
			Cardiovascular disorders and Coronary heart disease	ECG abnormalities Elevated cholestrol in blood
4.	Dyestuff manufacture	Carcinogenic dye intermediates	Bladder cancer	–
5.	Cotton textile	Cotton dust	Byssinosis	Respiratory functions impaired
6.	Manufacture of chromate and bichromates	Chromium (Hexavalent)	Chromium poisoning	Nasal septum perforation, chrome ulcers
7.	Acid storage battery units – other lead process units	Lead	Lead poisoning	Constipation, pallour, abdominal cramps, weakness and fatigue, anorexia and blue line of gums, anaemia
8.	Ferro-manganese plant	Manganese	Manganese poisoning	Lassitude, paraesthesia, sonolescence, dyspnoea, gait disturbance and speech disturbance
9.	Caustic soda (cell units)	Mercury	Mercury poisoning	Loosening and loss of teeth, abdominal discomfort tremors, anorexia
10.	Organic chemicals manufacture	Nitro, amino compounds	–	Mild exposure causes fatigue and headache, weakness, giddiness, vomitting, difficulty in breathing Severe exposure may result in cyanosis
11.	Pesticide manufacture and formulation	Organo phosphorus compounds	–	Cholinesterase activity in blood reduced
12.	Coal mines	Coal dust	Pneumoconiosis	Respiratory functions impaired
13.	Non-coal mines	Silica containing dust	Silicosis	–do–
14.	Slate pencil manufacture	Dust containing free silica	Silicosis	–do–

production of those industries with inherent health hazards was still more striking. Such proliferation resulted in large scale use of many toxic chemicals and other substances such as asbestos, benzene, carbon disulphide, carcinogenic dye intermediates and organo-phosphorus pesticides. Major toxic substances associated with some of the industries and occupational diseases caused by them are presented in Table 2.

3. STATUTORY SAFEGUARDS FOR CONTROLLING HEALTH HAZARDS

In the developed countries toxic chemicals and substances were already known to have seriously affected the health of the workers handling them. Fortunately, from such experience, information was also available as to what type of preventive control and protective measures were to be adopted to control the hazards. The Government of India had incorporated these measures in the major Safety Statute, viz. the Factories Act, 1948. For instance, there are general provisions on :

(i) Effective measures to be undertaken to prevent the accumulation of harmful dusts and fumes in the work places (Section 14),

(ii) Precautions to be taken against dangerous fumes in confined space (Section 36).

Besides, Section 87 of the Act enables the State Governments to make rules to protect the safety and health of the workers engaged in dangerous operations. Such rules cover operations involving exposure to asbestos, benzene, chromium, carbon disulphide, dangerous pesticides, lead, manganese and nitroamino compounds, among others. In respect of workers employed in these processes, pre-employment and periodical medical examinations using special tests, wherever warranted, have been prescribed. Further, under Section 89 of the Act, 22 occupational diseases (including those resulting from exposure to the above toxic substances) have been declared as 'notifiable diseases' and any medical practitioner or management who comes across any worker diagnosed to be suffering from any one of these diseases, has to report to the concerned authorities immediately. Appointment of full time or part time medical officers to carry out medical surveillance work, has been prescribed for a number of dangerous processes.

Realising that a large number of small units may employ less than the prescribed number of workers and hence may not be registered as factories, provision was made under Section 85 that even such small factories, if they are engaged in dangerous operations, can be specially notified and brought under the provisions of the Act.

4. MAGNITUDE OF INDUSTRIAL HEALTH HAZARDS

Functioning of such an elaborate system of safeguards for controlling health hazards all these years should, normally, have provided us with enough documented statistics/evidence on the pattern of changes in the hazard levels taking place, and the resultant health impairment. Unfortunately, no such information is available either from the health records maintained in the factories or from the records of the enforcing agencies. Even the studies and surveys carried out by institutions engaged in occupational health research, with a few exceptions, have been scattered and incomplete. In spite of this, quantification of the trends of the health hazards in industries handling important toxic substances based on the few studies, has been still possible to some extent.

4.1 Asbestos

Of all the toxic substances handled at work places, asbestos, the naturally occurring mineral mainly consisting of silicates in fibrous form is the most hazardous substance. Asbestos fibre, when inhaled and gets dispersed in the lungs, can cause 'asbestosis', a respiratory disease, as well as lung cancer and cancer of the membranes lining the lungs and the abdomen (mesothelioma of the pleura and the peritoneum). The Threshold Limit Value (TLV) which is a Time Weighted Average (TWA), ranges from 2 to 0.2 fibre per cc, for different varieties of asbestos. In 1980, a more stringent standard of 0.1 fibre per cc has been recommended for all varieties. Such hazards have been instrumental for a number of developed countries gradually withdrawing it from use. As for the situation in India, even now nearly 81,000 tonnes are imported every year over and above the nearly 20,000 tonnes mined in the country itself. The very fact that the import of asbestos[5] had gradually increased from nearly 63,000 tonnes in 1978-79 to 81,000 tonnes in 1981-82,[5] is an indication that either the potential hazards in handling this have not been fully appreciated or its use cannot be avoided for the time being and hence, instead of banning it, control measures should be adopted to prevent the actual exposure or at least minimise it.

In the industry both the white greenish 'Chrysotile' which is the serpentine variety, and the bluish 'Crosidolite' which is the amphibole variety, are largely used. Of these two, it is the latter which poses greater health hazard to the exposed individuals.

As with many other substances, the national approach seems to have been more emotional than scientific. The few studies which have been carried out in the last decade or so lack in uniformity in the parameters used, and in many cases are not even complete. From the results of these studies enumerated in Table 3, it appears that incidence of 'definite' cases of asbestosis is around 7 per cent. One should not overlook the fact that there is a considerable incidence of 'possible' cases, and these may, with passage of time, become definite cases. Another observation which should cause concern is that the incidence is related to the length of service and exposure. Hence, as units will become old, many more workers may be affected unless very strict preventive control measures are adopted. Smoking has been noticed to aggravate the condition. None of the studies carried out so far, seems to have probed the incidence of carcinoma of the lungs or mesothelioma.

4.2 Benzene

Because of its miscibility and solubility in lipids, benzene can get stored in the body fat and bone marrow. Due to its easy absorption through the intact skin, the hazard is aggravated in the hot working environments as prevailing in the country. During acute exposure the central nervous and cardiovascular systems are affected. The symptoms include excitation, dizziness, muscular weakness, visual disturbances, tightness in the chest and breathlessness, tremors, cyanosis, convulsions etc., even though these are transitory in nature. Chronic exposure to low concentrations is reported to result in symptoms of tiredness, gastro-intestinal disturbances, giddiness, haemorrhages from mucous membranes of nose and gums, and anaemic conditions. Prolonged exposure to high concentrations can cause leukemia. Of late, cancer due

Table 3. Asbestosis hazards in asbestos handling units as revealed by some recent studies

Sl. No	Study Ref.*	Airborne concentration of asbestos fibres (fibres/cc)	Number of workers studied	Health impairment record	Remarks
1.	N.I.O.H. Ahmedabad (1976)	72 – 119	Exposed – 355 Control – 112	Incidence of: a) X-ray abnormalities– 8.7% in exposed 1.8% in control b) Respiratory ailments– 19% in exposed 10.7% in control c) Lung function 7% lower in exposed	Health parameters covered were not adequate to decide on asbestosis cases
2.	N.I.O.H. (1978)	216 – 418	N.A.	Restricted vital capacity	Incomplete health examination – No conclusion on asbestosis cases
3.	N.I.O.H. (1979)	N.A.	477	Lung capacity of the workers in the stripping department significantly low	– do –
4.	C.L.I. Bombay (1980)	0.02 - 0.80	900	6.5% cases of 'restricted' type of pulmonary functions	Incidence was related to the duration of exposure. Health parameters not adequate
5.	C.L.I. (1981)	0.005–9.02 crocidolite (Blue)	307	Definite asbestosis cases % Smokers – 8.9 Non-smokers – 3.2 Overall – 6.5 Possible asbestosis cases % Smokers – 34.8 Non-smokers – 26.2 Overall – 31.9	All parameters as per international practice. Incidence of cases was related to exposure: Definite asbestosis – 3% in group with exposure upto 5 years 8% in group with 11-15 years exposure 14% in group with 15-20 years exposure
6.	C.L.I. (1983)	0.1 – 10.8	405	Definite asbestosis % Smokers – 5.3 Non-smokers – 7.7 Overall – 7.1 Possible asbestosis % Smokers – 3.2 Non-smokers – 5.8 Overall – 5.2	Effect of smoking contrary to expectation. Incidence was related to duration of exposure: Definite asbestosis cases – 2.9% in group with exposure upto 5 years 6.4% in 10-15 years group 10.9% in 15-20 years group 12.9% in 20-25 years group 17.6% in 25-30 years group

Contd.

Sl. No.	Study Ref.*	Airborne concentration of asbestos fibres (fibres/cc)	Number of workers studied	Health impairment record	Remarks
7.	KEM Hospital	N.A.	789 (from 3 major units)	Nearly 28.2% showed fibrotic shadows in chest chest X-ray (Asbestosis related)	No environmental monitoring was done.

Conclusion : Incidence of definite cases of asbestosis seems to be 6-7%.

* All references unpublished.

to benzene exposure is also feared. Because of such inherent hazards, there is an attempt the world over to replace wherever practicable, benzene by the less toxic higher homologues. In India, its use has been steadily on the increase. Benzene production which was 53,000 tonnes in 1950-51, has almost doubled by 1982-83, and can be expected to go up further. Statutory safeguards to protect the workers have, however, been made through the inclusion of benzene handling units under 'dangerous processes' in many State Factories Rules. Industrial toxicological studies on benzene workers have been very few in the country. In large undertakings where health surveillance of such workers is carried out, the results are not published. In the numerous small unorganised factories, it is not practicable to carry out any study.

The health risk faced by such workers has, however, been brought out in major study in a large alkaloid extracting unit (using large quantities of benzene as solvent), by the Central Labour Institute[6] (CLI) in 1979. In this study, 89 air samples were taken at different locations of the unit, for the determination of benzene concentrations. 96 workers exposed to benzene vapour in different processes and 13 unexposed control were subjected to biological monitoring (determination of urinary phenol, a metabolite). Chemical and haematological examinations were carried out on 81 of the exposed and all the 13 control workers. The findings were as follows.

(i)	Air samples exceeding the TLV of 30 mg/m^3 for benzene	87 out of 89 i.e. 98% (concentrations ranged from 33.3 to 2,797.8 mg/m^3)
(ii)	Cases with urinary phenol values exceeding the normal value of 50 mg/litre	68 out of 96 i.e. 71%
(iii)	Of the above, those recording very high values indicative of state of intoxication'	43 out of 96 i.e. 44.8%
(iv)	Cases with haematological abnormalities	14 out of 96 i.e. 14.6%
(v)	High urinary phenol cases with history of bleeding from nose and gums	12 out of 81 i.e. 14.8%
(vi)	Cases with symptoms of acute effects	58 out of 81 i.e. 71.6%

Ironically, such high toxic exposures were noticed to arise due to such simple causes like inadequate ventilation, lack in personal hygiene among the workers and defective material handling methods. Since such deficiencies may prevail to a still larger extent in smaller units, the hazards will be there, perhaps to a greater extent.

4.3 Lead

Prolonged absorption of lead is known to lead to lead poisoning with general symptoms of fatigue and tiredness, gastro-intestinal disorders like loss of appetite, abdominal discomfort, nausea and constipation, haematological disturbances and neurological disorders. The TLV for lead is 0.15 mg/m.[3] In industry lead poisoning is potential in the lead acid storage battery units, printing presses, battery repair shops etc.

4.3.1 Large Units

The first major study[7] on 'lead hazard' was carried out in 1953, in which 16 storage battery units were surveyed and 492 of the workers were examined. 10.6 per cent of these were diagnosed to be lead poisoning cases. In another extensive survey[8] carried out in 1978-80, apart from environmental monitoring, health examination of 363 workers was also conducted. A comparison of the findings of the two studies reveals that there has been a trend of very slight improvement in the condition over the years (Table 4). However, one has to be cautious while drawing this conclusion, because of the differences between the two studies particularly in (i) the pattern of sampling, location and the number of samples taken, (ii) the criteria for diagnosing

Table 4. Status of lead exposure hazard in acid storage battery units as observed in 1953 and 1981

Sl. No.	Parameter	Values in 1953	Values in 1981
1.	Range of mean airborne concentrations of lead in different processes (mg/m^3)	0.67–2.57	0.13–11.38*
2.	Mean airborne lead concentration (mg/m^3) in :		
	Casting section	1.06	0.13*
	Pasting section	2.37	1.00*
	Assembly section	0.67	0.96*
3.	Percentage of workers actually exposed to lead levels higher than the TLV of 0.15 mg/m^3	70	65.5
4.	Number of workers medically examined	492	363
5.	Percentage of workers diagnosed as lead poisoning cases	10.6	9.1

(Data from Ref: 7, 8).

* Computed from basic data of Ref: 8.

the lead poisoning cases. In the 1953 study, only clinical signs and symptoms such as constipation, pallour, abdominal cramps, weakness and fatigue, anorexia and lead line of gums formed the basis whereas in the 1981 study, both clinical signs and symptoms as well as lead levels in blood and urine, and ALA level in urine formed the basis. In both the cases there seemed to be a certain degree of adhocism.

However, only from the data of 1953 studies, it is possible to draw cause-effect relationships between the percentage incidence of lead poisoning cases on one hand and each of the causative parameters of time intensity factor (exposure duration in months × intensity of exposure to lead), urinary lead level and blood level (Fig. 1) on the other. Unfortunately, no such relationship could be established with the data of the latter study.

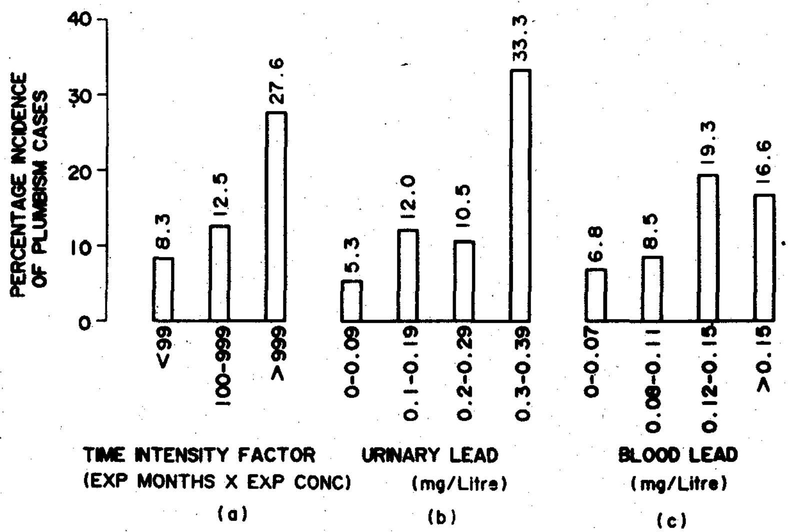

Figure 1 (a,b,c).

4.3.2 Medium and Small Units

Normally, there is an impression that in smaller units, the environmental conditions will be poor, and workers will be exposed to greater health risk. The National Institute of Occupational Health at Ahmedabad studied the lead exposure status of workers in medium scale printing presses (2 units employing 95 workers) and small scale accumulator and battery repair shops (7 shops employing 17 workers).[9] The working environments were also monitored for lead concentration. The findings showed that the concentrations in the printing presses ranged from 0.001 to 0.018 mg/m^3, and in the repair shops from 0.008 to 0.146 mg/m^3. Understandably, the lead content of blood was 36 μg per 100 ml in the printing press workers and 55 μg per 100 ml in the other group. The corresponding urinary values were 50 μg per litre and 130 μg per litre respectively. All these levels of lead concentration were well within permissible limits. The trend of impact of the chronic exposure to even such very low levels of lead has been reflected in the haemoglobin content and RBC being slightly lowered in the exposed workers as compared with the unexposed controls as shown in Table 5.

Table 5. Haemoglobin and RBC contents in exposed/unexposed workers

	Control	Exposed	
		Battery workers	Printing press workers
	(n = 25)	(n = 17)	(n = 87)
Hb (gm %)	15.2 ± 0.88	13.98 ± 0.98	14.81 ± 0.57
RBC (Mn/c. mm)	4.9 ± 1.1	4.48 ± 0.45	4.73 ± 0.88

Even though the data suggests that such workers are not seriously affected, still these are too inadequate to draw a valid generalised conclusion.

4.4 Carbon Disulphide

In the viscose rayon units, the major health hazards arise due to exposure to CS_2 and H_2S. The major effects of CS_2 have been recorded on the central nervous system. Continued exposures to very high concentrations of the order of 300–500 mg/m^3 are reported to give rise to polyneuritis. The early symptoms are the dissociation of tendon reflexes in lower extremities. Recently, coronary heart disease has also been stated to arise out of CS_2 exposure. Hydrogen sulphide is known for its irritant action on eyes and the respiratory tract. The TLV for carbon disulphide is 30 mg/m^3 and for hydrogen sulphide 14 mg/m^3. There are 11 viscose rayon plants in India employing nearly 15,000 direct workers. Having been aware of the toxicity of the chemicals, the Government of India has advised the State Governments to include in their Factories Rules, processes involving these two under 'dangerous' operations and observe strict safety and health measures. Carbon disulphide poisoning is also a 'notifiable disease' under the Factories Act. Consequently, national surveys have been conducted at different points of time, to evaluate the extent of hazard prevailing from time to time.

The first series of studies[10] were carried out in 1958 by the Chief Adviser of Factories (later designated as DGFASLI) in 3 viscose rayon plants. 27 per cent of the 270 workers studied were diagnosed to be suffering from CS_2 intoxication. Incidence of various psychic symptoms ranged from 4–45 per cent. After the units had supposedly carried out improvements as per the recommendations of the report, a follow-up study[11] was carried out in 1969-70, by CLI to evaluate only the environmental changes. This survey concluded that there was a considerable reduction in the concentrations of CS_2 and H_2S. For instance, in the spinning section of the three plants, concentrations of CS_2 were reduced from a range of 45.9 – 94.5 mg/m^3 to 7.2 – 21.3 mg/m^3. Ironically, another study[12] carried out by CLI in 1974-75 in one of the three units, revealed that the concentrations of CS_2 in spinning halls were still very high, being 20.0–84.8 mg/m^3. The percentage incidence of subjective symptoms such as pain in the chest, weakness in the lower limbs, sleeplessness, loss of appetite etc. was considerable, being 20 per cent. The slight fall in the incidence of symptoms from 27 per cent in 1958 to 20 per cent in 1974-75, suggests that the considerable improvement in environmental situation reported in the 1969-70 study was perhaps an artifact. Or, the differences may be due to the differences in the measurement techniques between the two studies. All the studies carried out so far have brought out that exposure to high concentrations of CS_2 prevails mostly in the spinning halls, and the churn rooms of the viscose sections. It can be reduced by encasing the individual machines in the spinning halls so as to improve the efficiency of the central exhaust, and by providing a non-sticky teflon quoting on the interior of the churn. As is evident from Table 6, encasing individual spinning machines has been proved to reduce the concentration of CS_2 and H_2S beyond doubt in the unit studied in 1974-75, after they had carried out this improvement.

Table 6. Reduction in the concentration of pollutants (CS_2 and H_2S due to encasing the spinning machines in a viscose rayon plant

Location	Concentration of CS_2 (mg/m^3)		Concentration of H_2S (mg/m^3)	
	Before encasing	After encasing	Before encasing	After encasing
A	87.0	38.7	23.2	10.7
B	96.0	39.3	24.3	8.6
C	106.0	40.3	30.6	8.2
D	74.0	34.0	14.0	8.1
E	66.0	38.0	18.0	10.6
F	80.0	17.0	19.0	5.6
G	80.0	31.0	23.4	5.6
H	98.0	28.5	20.7	3.9
I	92.0	24.0	18.0	8.5
J	66.0	28.5	12.0	8.8
K	58.0	27.8	14.4	10.2

Permissible limits (TLV)

Carbon disulphide – 30 mg/m^3

Hydrogen sulphide – 15 mg/m^3

Data from Ref: 12

Very recently, the occupational health status of workers in 8 of the 11 plants was studied again going mainly by subjective complaints and neurological and clinical observations.[13] 2,217 workers who are occupationally exposed to the chemical, and 712 workers unexposed control were covered. The results revealed that respiratory and cardiovascular disorders, gastro-intestinal disorders, psychic disorders, and many of the neurological symptoms were all much reduced since 1958 study. Only the tremor in the hands and dissociation of reflexes had gone up. Even at this improved status. the incidence among the exposed workers was much higher than the control group, thereby indicating that the causative factor viz. the environmental concentrations of CS_2 are to be further lowered. The drawback in such comparative studies is that too much weightage is given to subjective symptoms which is questionable. Conditions can be improved if objective measurements are made using appropriate instruments. Where clinical signs and subjective symptoms are unavoidable, it is advisable to go through the health records of the individuals, and make sure of the complaints.

Albeit the general deficiencies in the health parameters used, the rise in the incidence of tremors in the hands is indicative that CS_2 poisoning still exists. This can be brought down considerably by just two source control measures, viz. encasing the spinning machines so as to prevent the dispersal of CS_2 and making the inner surface of the churns non-sticky so that dumping of xanthates can be carried out quickly.

4.5 Mercury

Occupational exposure to mercury vapour in the manufacturing industries occurs mostly in the caustic soda plants operating with mercury cells. In 1985, 75 per cent of the then existing 36 caustic soda units were on mercury process. In the same year, sanction was accorded to another 16 units.[14] It can be presumed that many out of these may also be using mercury cells. Acute exposure to mercury vapour has long been known to cause abdominal pain, vomiting, diarrhoea, mental disturbances, disordered sensation at the extremities etc. Chronic exposure may give rise to disorders in the mouth such as loosening and loss of teeth, oedematous gums, gingivitis, stomatitis, metallic taste etc. Neuromuscular disorders such as vague numbness, tremors of fingers and lips and fatigue and weakness, and mental changes like emotional upset, loss of memory, nervousness, and insomnia are other chronic effects. Considering such serious potentialities, mercury poisoning has been made a 'notifiable disease' under the Factories Act.

Unfortunately, no effort was made to study the possible health risk faced by workers in caustic soda plants until 1973 when Harwant Singh and Gupta[15]examined the problem in 3 caustic soda units. Apart from environmental monitoring for mercury content, 88 workers exposed to mercury vapour and 36 who were not, were screened for typical ailments. Mercury content in their urine and blood samples was also determined. Complaints of large incidence were loosening and loss of teeth, abdominal discomfort, tremors, anorexia, weakness and fatigue, gum disorders, insomnia and stomatitis. There were wide day to day variations in the urinary excretion levels. Even so incidence of the above symptoms was generally accompanied by large excretions in urine. Surprisingly, there were also a few cases with high blood and urinary levels of mercury, but without any symptoms. These indicate the difficulties in deciding on the actual poisoning cases. All the same, 16 were diagnosed as poisoning cases and 4 as intoxication cases. The incidence, thus, works out to be 22.7 per cent. The development of high concentrations of mercury vapour in the work environment was attributed mainly to poor conditions of flooring, and lack of ventilation. These were partly substantiated when the airborne concentrations in one of the plants and urinary levels in the workers came down significantly, merely after the ventilation was increased. Changes in the mercury vapour concentration due to ventilation have been presented in Table 7. Till the housekeeping conditions are toned up in such units, at least mercury intoxication cases will continue to pose a problem.

Table 7. Changes (mostly reduction) in the concentration of mercury vapour in the cell house of a caustic soda plant, due to improvement in the ventilation

Number of locations	Mean concentrations (mg/m^3)	
	During the initial study	During the follow-up study
6	0.115 ± 0.030	0.037 ± 0.015
1	0.05	0.05
1	0.04	0.06

4.6 Nitro-amino Compounds

In the coming years production of aromatic nitro and amino compounds may have to be hiked up to meet the increasing demands of the dyestuff and pharmaceutical industries. This is also evident from estimates of the Planning Commission on the production of nitrobenzene during 1985 and 1990. As per these estimates the production of nitrobenzene will be 26,000 and 37,000 tonnes, and of aniline, 11,000 and 15,000 tonnes respectively.[16] Since exposure to these chemicals may result in cyanosis, anaemia, and carcinogenic effects (amines), manufacture of aromatic nitro or amino compounds is considered to be a dangerous process and in many State Factories Rules, rigorous personnel hygiene and other preventive measures have been prescribed. Regarding health hazards, because of a psychological fear on the part of both the managements and the workers, very few occupational health studies are encouraged to be carried out in such units.

All the same, a major environmental and health study carried out in 1976 by CLI in a large organic chemical unit manufacturing nitro compounds, at the instance of the management itself, has provided an insight into the nature of the health problems prevailing therein (Gupta, V.P., CLI, personal communication). In this study, 115 samples were taken from the nitrobenzene, dinitrobenzene, nitrotoluene and nitrochlorobenzene plants and analysed for the concentrations of respective chemicals. 140 workers from these four plants and the effluent treatment plant, were subjected to clinical, haematological and urinary tests. The findings are presented in Table 8.

Even though the urinary excretion levels of the free compounds were negligible compared to their biological TLVs, elevation in the urinary level due to shift exposure was much higher with nitrochlorobenzene (1.57 mg/l) than with nitrobenzene (0.40 mg/l) and nitrotoluene (nil). Naturally, with such high degree of absorption, 41 per cent of nitrochlorobenzene workers showed statistically low haemoglobin values, and 29 per cent showed elevated methemoglobin. In spite of the overall low levels of absorption, subjective complaints were widely prevalent. 55–90 per cent workers in all the plants had one or more complaints. 'Weakness and fatigue' was the most

Table 8. Environmental findings of organic chemical unit

Chemical	TLV (mg/m^3)	Number of samples	Range of concentrations found (mg/m^3)	Percentage of samples exceeding TLV	TWA* (mg/m^3)
Nitrobenzene	5	13	0.20-2.90	Nil	0.11- 1.79
Dinitrobenzene	1	6	0.10-1.80	33	0.33- 0.60
Nitrotoluene	30	26	Tr- 7.38	Nil	0.52- 6.82
Nitrochlorobenzene	1	70	Tr-23.33	53	0.37-10.64

* TWA – Time Weighted Average for 8 hours, considering the actual exposure duration.

prevalent (36 per cent), followed by 'stomach pain and abdominal disorders' (28.7 per cent), headache (28.5 per cent), skin itches (21.8 per cent), loss of appetite (15 per cent), chest pain (14.3 per cent), and giddiness and vomitting (9.3 per cent). Whereas, as mentioned earlier, one has to consider these subjective findings with a degree of caution, there is also the possibility that because of synergistic considerations (nutritional level and ambient temperature), the TLVs may be lowered, which needs careful verification. The plants studied are located in a sparcely populated open area, where due to prevailing conditions of natural ventilation, build up of concentrations is arrested. The same may not be the case with other units, particularly numerous small ones which are operating in congested localities. In such units, the health problems may be aggravated. Changes in the concentration of nitrochlorobenzene before and after the implementation of control measures are presented in Table 9.

Table 9. Changes (mostly reduction) in the airborne concentrations of nitro chlorobenzene after implementation of control measures

Location	Airborne concentration (mg/m^3) During the first survey Range	Mean	During the follow-up study Range	Mean
A	0.67 – 2.67	1.51	0.75 – 2.31	1.18
B	traces – 2.00	0.66	0.14 – 1.30	0.44
C	traces – 5.33	1.83	0.25 – 1.00	0.54
D	0.50 – 23.33	5.75	0.66 – 7.08	2.48
E	0.56 – 4.44	2.07	1.08 – 3.75	2.08
F	0.17 – 2.44	1.26	0.50 – 5.25	1.70

4.7 Manganese

Manganese with its TLV of 1 mg/m^3 (for fumes) is known for its high-toxicity. The major disorders associated with manganese poisoning are asthenia, mental excitability, speech disorders, clumsiness in movements, changes in facial expression (masklike face), tremors and 'cock walk'. Albeit such toxic effects, manganese production in the country has been steadily going up and a large number of workers are exposed to its dust and fumes. The statutory safeguards to protect the health of the workers are that the processes have been declared 'dangerous processes', and cases of manganese poisoning are to be notified. Surprisingly, not much data is available on the health status of manganese workers. Though a number of animal studies have been carried out by the Industrial Toxicology Research Centre at Lucknow, studies on the effects on Indian workers due to occupational exposure, have been very few. The first and perhaps the only major study was carried out in 1962, by the Chief Adviser of Factories, Ministry of Labour, Government of India, in a ferromanganese plant.[17] Out of the 12 locations monitored, 5 were recording concentrations ranging from 1.3 to 8.4 mg/m,[3] and the rest were within the TLV. 43 of the 179 workers examined were found to be poisoning cases, 13 being completely disabled, 30 partially. There were another 19 with symptoms only. Thus the percentage of incidence was 24. Unfortunately no further study has been reported and there is no means of assessing whether manganese exposure hazard has been reduced since then.

4.8 Chromium

Chronic inhalation of dust and fumes containing hexavalent chromium is known to cause such serious disorders as ulceration of nose and skin and nasal cartilage perforation, apart from dermatitis. The TLV for hexavalent chromium is 0.05 mg/m.[3] The trend of incidence of such diseases was studied way back in 1953 in a study on workers engaged in manufacture of dichromate.[18] Among the 374 workers comprising of roastermen and centrifuge workers, leach and acid tank workers, and grinders and mixers, 101 (27 per cent) suffered from skin ailments, 227 (60.7 per cent) had nasal ulcer and perforation and in 39 (10.4 per cent) nasal involvement and skin ulceration. Obviously some of the workers should have had more than one disorder.

Subsequently, a few studies carried out in Kanpur also reported the prevalence of nasal septum perforation among such workers.[19]

4.9 Pesticides

Information available on the health status of workers in pesticides manufacture or formulation is inadequate. Work on 75 workers employed in a DDT plant revealed that 22.4 per cent of them were recording elevated eosinophils, 56.6 per cent neutropenia (53 per cent polymorphs) and 60.5 per cent lymphocytosis.[20] Another study on 45 workers in a formulation unit exposed to a combination of pesticides including parathion recorded that 40 per cent of them were excreting high quantities of paranitrophenol in urine and 4 per cent suffered from loss of appetite. Reduction in cholinesterase activity of blood was observed in a few, the values going down to as low as 62.5 per cent (Gupta, V.P. - personal communication).

4.10 Vinyl Chloride

Vinyl chloride monomer which is used as a raw material in the PVC industry has been proved to result in angiosarcoma of liver (liver cancer). The TLV is fixed at 10 mg/m.[3] In spite of such a serious health hazard and a large quantity being manufactured in the country, no evaluation of the health risk to the workers has been carried out so far.

4.11 Cotton Dust

Cotton textile workers engaged mainly in blending and scrutching, carding and brush strapping operations and exposed to cotton dust, suffer from the typical disease 'Byssinosis', in which the lung functions are considerably reduced. This problem has been widely studied in the past, at various textile centres in the country. The incidence of byssinosis[21] ranged from 7.3 to 9.0 per cent. The latest study by Kamat[22] et al 1976, in which 814 workers were subjected to a 5 years prospective study, revealed that even in the newly set up plants with modern machinery and air-conditioning, byssinosis is potential, even though it is reduced. The combined incidence of cases of byssinosis, and byssinosis and bronchitis in the old and the semi-modern mills was 21 and 24 per cent respectively, while in the modern unit only 10 per cent. Thus, even though the problem cannot be completely eliminated, it can be considerably brought down by improving the conditions of the machinery and of the work places.

4.12 Coal Dust

The coal miners, being exposed to coal containing minerals, have been established to suffer from pneumoconiosis, which is indicative only of deposits of coal in the lungs with minimal fibrosis. Lung functions may be impaired to some extent. A major study in Jharia and Ranigunj coal fields conducted in 1961, covered 950 miners in 12 coal fields.[23] The incidence of pneumoconiosis was 18.8 per cent. After nearly a decade, another study in 11 coal mines in the same area covered a very large sample of 8,822 miners.[24] Incidence of pneumoconiosis was found to be 10.8 per cent, thus suggesting the possibility of an improvement in the situation over the years. The prevalence among underground workers was 12.7 per cent and among surface workers 5.1 per cent.

4.13 Metallic and Mineral Ores

The metallic and mineral ores contain also free silica apart from the concerned metals and minerals. As such, exposure of the miners to these dusts has been reported to cause silicosis in which there is fibrosis of the lungs and the lung functions are impaired. The studies carried out by the Chief Adviser of Factories in 50s and early 60s revealed the following incidence of such silicosis cases. Mica miners–34.1 per cent (nodular and conglomerate silicosis), lead and zinc miners–30.4 per cent. Apart from such miners, workers in such industries where free silica containing dust is emanated have also been found to be affected. The incidence in pottery and ceramic industry was 15.7 per cent, and in refractories 17.7 to 23.1 per cent. Unfortunately, no repeat studies were carried out in such factories, so as to reflect on the extent of improvements in the situation. An alarming situation was revealed in the small scale slate pencil manufacturing units in Mandsaur in M.P., where 54.7 per cent of the 605 workers examined were found to suffer from silicosis, and 17.7 per cent from conglomerate silicosis, due to inhalation of silica containing slate dust.

4.14 Physical Exertion and Toxic Effects

In some studies, there have been indications that the physical activity level very much influences the toxic effects. For instance, in the study on benzene, for the same levels of exposure, the mean urinary excretion of phenol was only 60.4 mg/l in supervisors, whereas in the active workers, the level[6] was 154.1 mg/l. Similarly, the enhanced paranitrophenol excretion in urine of parathion workers, was found to be related to the physical exertion levels of the workers. The mean values ranged from 'traces' in sedentary workers to 0.151 mg/l in moderately active workers and 0.171 mg/l in active workers.

5. SHORTFALLS AND FUTURE STRATEGIES

(i) There is no uniformity in the design of sampling, as well as in the sampling and analytical techniques. These need to be standardised. Quality assurance is to be ensured.

(ii) While the TLV which is a TWA concept, is used as guideline for comparison, the number of samples taken, and the patterns of sampling do not meet this requirement. With biological samples, there is a very large day to day variation. Hence samples are to be taken on a number of days to arrive at reliable averages. With very few exceptions, (Tables 6, 8 and 9) no repeat studies are carried out in a systematic way so as to evaluate the changes from time to time. This approach is to be adopted in future studies.

(iii) The studies do not cover medium and small units. Future studies are to be made representative.

(iv) There is a reluctance to publish the results of studies (withholding the identity of the units, and thus retaining the confidential aspect of even consultancy studies).

(v) Surveys undertaken by different institutions are mostly on ad hoc basis. It is advisable that an apex body co-ordinates all these activities, so that maximum information can be obtained with minimum efforts.

REFERENCES

1. Labour Bureau, Simla, Statistics of Factories, 1947, 1953, 1976.
2. India 1983 (Publications Division, Ministry of Information and Broadcasting, Government of India), 1983, p. 333.
3. Statistical Outline of India (Vora & Co. Publishers (P) Ltd., Bombay), 1961, p. 21.
4. Statistical Outline of India (Tata Services Ltd., Bombay), 1980, p. 66.
5. Meenakshi, Behara, *Business India,* 17-30 Jan. (1983), 73.
6. Gupta, V.P., Bhar, S. Alam Khan & Panday, G.K. Industrial Hygiene and Occupational Health Studies in the Government Opium and Alkaloid Works at Ghazipur, U.P. (CLI, Sion, Bombay), 1979.

7. Chief Adviser of Factories, Ministry of Labour, Government of India, Environmental and Medical Studies in the Storage Battery Industry (Government of India), 1953.

8. Chakravorti, S. & Bhar, S. Occupational Lead Exposure in India: Lead Acid Storage Battery Manufacturing Industry (CLI, Sion, Bombay), 1981.

9. National Institute of Occupational Health, Ahmedabad, Study of Workers Exposed to Inorganic and Organic Lead in Various Types of Industries (NIOH, Ahmedabad), 1976.

10. Chief Adviser of Factories, Ministry of Labour, Government of India, Survey of Carbon Disulphide, Hydrogen Sulphide and Sulphur Dioxide Hazards in the Rayon Industry in India (Government of India) 1959.

11. Kale, S.C., Gupta, V.P., Dangwal, S. K. & Chakravorti, A.K., Report of the Environmental Studies: Airborne Concentration of Carbon Disulphide and Hydrogen Sulphide in the Viscose Rayon Plants (Ind. Hyg. Laboratory, CLI, Sion, Bombay), 1973.

12. Chakravorti, S., Dangwal, S.K., Bhar, S. & Joseph, J., Environmental, Biochemical and Chemical Studies in the Viscose Rayon Plant, Century Rayon, Kalyan (CLI, Sion, Bombay), 1976.

13. Bhar, S. & Sinha, Y.N., National Project on Occupational Health of Viscose Rayon Workers in India (Ind. Med. Laboratory, CLI, Sion, Bombay), 1985.

14. Indian Chemical Manufacturers Association, 'Reports from other Associations — the alkali industry', *Chemical Industry News,* **XXX** (1985), 288.

15. Singh, H., Gupta, V.P. & Dangwal, S.K., *Industrial Safety Health Bill,* **20** (1977), 11.

16. Varadarajan, S., *Chemical Industry News,* **XXX** (1986), 841.

17. Chief Adviser of Factories, Ministry of Labour, Government of India, An Industrial Hygiene Study in a Ferro-manganese Plant (Government of India), 1962.

18. Chief Adviser of Factories, Ministry of Labour, Government of India, Investigations on the Incidence of Occupational Diseases in the Manufacture of Dichromate and in the Mining and Concentrating of Chromites (Government of India), 1953.

19. Gopalakrishna, Mathur., J.S. & Gupta, R.K., *Ind. J. Med. Res.,* **22** (1976), 866.

20. Chief Adviser of Factories, Ministry of Labour, Government of India, Medical and Environmental Study in a Plant Manufacturing DDT (Government of India), 1965.

21. Thiruvengadam, K.V., et al., *Ind. J. Med. Res.,* **16** (1970), 149.

22. Kamat, S.R., *Ind. Safety Chron.,* **XV** (1984), 102.

23. Chief Adviser of Factories, Ministry of Labour, Government of India, Pneumoconiosis in the Coal Mines in Jharia and Ranigunj Coal Fields (Government of India), 1961.

24. Viswanathan, R., et al, Morbidity Survey of Coal Workers (Rajendra Memorial Research Institute of Medical Sciences, Patna), 1972.