Bhopal Gas Episode – A Case Study

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Abstract

Industrialization has agreat impact on our lives. Philosophers have been warning us about the danger of industrialization .We realize the price paid for progress only when a nightmarish incident like Bhopal Gas Tragedy involving loss of numerous lives and many after effects occurs .In the present paper we will discuss the incident ,its impact on the health of the victims even today when two and a half decade is over.

Key words – Bhopal gas tragedy, industrialization, impact .

Introduction

On the midnight of 2 and 3 December 1984,the worst industrial disaster was caused by Union Carbide Corporation (USA) in Bhopal ,the capital of Madhya Pradesh. Approximately 40 tonns of Methyl Iso Cyanate spilled over and caused the world's worst disaster (Verma 1986,1989) . The number of people died in the incident was over 3000 and the number of people ranged between 2,00,000 to 6,00,000 (Kumar 1994,Kumar 1995, Sriramachari and Chandra 1997) .

Bhopal Plant (The Monstrous Killer)

The Bhopal plant of Union Carbide India Ltd. Is the second of its own kind in the World and only plant built outside U.S.A . It is one of the leading pesticides units in the country and has a licenced capacity of 5000 tonnes of Pesticides (UCC ,1984) In the procedure for manufacturing the pesticides Sevin and Temik, methyl-isocyanate (MIC) was used as an intermediate. In the beginning, MIC was imported, but in 1979 UCIL built an-MIC unit. The company was offered a site outside the town; but instead on using the existing plant area, close to the railway station. The complex specializes in methyl iso cyanate (MIC) based carbamate pesticide which are claimed by the company to be safer, more effective and which can handle a much larger spectrum of crops. Between 1977 and 1984 , Union Carbide India Limited (UCIL) ,located within a crowded working class neighbourhood in Bhopal was licenced by the Madhya Pradesh Government to manufacture phosgene ,mono methyl amine (MMA),methyl isocyanate (

MIC),and the pesticide carbaryl also known as sevin (Behl et al.,1978,UCC1985 , Singh and Ghosh 1987) .The plant was started in 1979 and the second phase of project was completed in 1980 with actual production of MIC based pesticides (UCC ,1984). The US supplied reactors,distillation towers ,heat exchangers, centrifuges, filters, dryers, valves, control instrumentation safety equipments etc.

Phosgene was manufactured by reacting chlorine, brought to the plant by tanker and carbon monoxide, produced from petroleum coke and oxygen in an adjacent production facility within the plant (Behl et al.,1978, UCC 1985)

The chemical reactions involved are as follows-

$2C + O_2 \rightarrow 2CO$

$2CO \rightarrow 2COCl_2$ (Phosgene)

The mono methylamine also brought in by tanker ,was combined with the phosgene in the presence of chloroform which was used as a solvent throughout the process to produce methyl carbamoyl chloride(MCC) and hydrochloric acid (HCl).HCl was then separated from the MCC so that it could be broken down into MIC and HCl .The MIC was then collected and transferred to stainless steel storage tanks ,while the HCl alongwith residues of MCC ,chloroform , and other unwanted by- products like MMA, carbon tetra chloride ,dimethylallophanoyl chloride ,cyanuric acid,dimethyl urea , trimethylbiurate etc.were collected and recycled back through the process (Behl et al. 1978, UCC 1985)

$COCl_2 + CH_2NH_2 \rightarrow CHNHCOCl + HCl$

(Methylcarbamoyl Chloride)

$CH_3NHCOCl \rightarrow CHNCO + HCl$

(MIC)

MIC was manufactured primarily to make the pesticide Carbaryl (sevin) as well as smaller quantities of Aldicarb (Temic) and butylphenyl methylcarbamate ,all destined for the Indian market (Merkenzie ,1984) Carbaryl was produced by reacting MIC with a slight excess of alphaNapthol in the presence carbontetrachloride (NEERI, 1990) and was sold as the pesticide Sevin.

$COCl_2 + CH_3NH_2 \rightarrow CH_3 NHCOCl + HCl$ (This reaction take place at -20^0 to 6^0)

CH_3 NHCOCl→ CH_3NCO +HCl

Cause of leakage of Methyl Iso Cyanate from tank 610

On the night of the disaster water inadvently entered the MIC storage tank where over two metric tones of MIC were being stored. The addition of water to the tank caused an exothermic chemical reaction resulting in a rapid rise in pressure of and temperature .The heat generated by the reaction ,the presence of higher than normal concentration of chloroform and presence of an iron catalyst resulted into such a fast reaction that the gas formed could not be contained by the safety system. (UCC 1985) As A result, MIC and other reaction products in liquid and vapour form escaped from the plant into the surrounding areas causing devastating effect on the people living in the shanty settlements just over the fence.(UCC 1985, Gupta et. al. ,1988) .The safety systems which in any case were designed for such a run away situation were non functioning .The scrubber designed to neutralise any escaping gas by spraying caustic soda was empty and the flare tower meant to burn off any gases from the scrubber was under repair .Hypothesis for the disaster included sabotage,prolonged bulk storage over 40 tonnes of MIC ,non functioning refrigeration systems, the failure of safety measures and malfunctioning of neutralization facilities (Mackenzie 1984,UCC 1985 ,Milne 1988)

Impacts of the disaster on Human Health

There are many impacts of the tragedy some of them are as follows –

1. Impact on Health

The acute symptoms were burning in the respiratory tract and eyes, breathlessness, stomach pains and vomiting. Those living close to the factory had very severe acute as well as long term symptoms. Several kilo metres away, in the new town, the residents only felt a passing mild irritation in the respiratory passages and eyes. Those who worked with patients or dead bodies suffered from delayed symptoms, even if they had not been exposed to the leakage as such.

The acute clinical picture included a transient irritation and redness of the skin, intense irritation from the eyes including blepharospasm, profuse eyelid oedema and superficial corneal ulcerations(Kulling P, Lorin H. 1987). A soothing and somniferous effect is also reported (Brussels. 1998) From the respiratory tract physical findings were rhinitis, pharyngitis, coughing, respiratory distress including bronchoconstriction, shortness of breath and choking. Many patients died from choking or reflexogenic circulatory collapse. Pulmonary oedema developed in many patients in the acute stage. In others, pulmonary oedema developed later, after a free

interval. All types of complications from the respiratory tract were seen, such as pneumothorax, subcutaneous and mediastinal emphysema, bronchopleural fistulas, secondary infections etc.

The worst hit were children below 2 years, old people and persons with previous pulmonary diseases, like chronic bronchitis and emphysema (Kulling P, Lorin H.1987). The findings during autopsies on victims revealed changes in many organs, but the most pronounced findings were related to the lungs. The lungs were enlarged and oedematous, showed congestion, haemorrhage and consolidation, with microscopic findings such as bronchiolitis and pulmonary oedema. There were focal haemorrhages in the other organs. In addition, the consistency of the brain was softened through cerebral oedema. The kidneys showed congestion and tubular necrosis. In a large number, the liver showed fatty degeneration. In the gastro-intestinal tract, necrotising enteritis was found. At Hamidia hospital, nine cases of partial paralysis were found. Women's reproductive health was affected. Immediately after the gas leak, the stillbirth rate increased by up to 300 % and the perinatal and neonatal mortality rate by 200 %. The spontaneous abortion rate increased three to four times and stayed raised for several years.
The rate of congenital malformations increased.

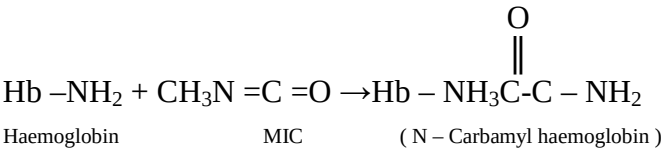
A study prepared by ICMR found that the spontaneous abortion rate following the gas leak was 24.2 per cent, about three times the national average. The stillbirth rate was 26.1 per 1,000 deliveries, compared with a national figure of 7.9 per 1,000. A year after the disaster the infant mortality rate in Bhopal was 110 per 1,000 births, compared with a national average of 65.2 per thousand Cassels J. 1993)

2. Impact on various organs

Most of the information on the medical consequences of Union Carbide disaster in Bhopal has been detained by the Indian Council of Medical Research (ICMR, 1985). The ICMR has established that the toxins from the Union Carbide factory have caused damage to the lungs,brains, kidneys, muscles as well as gastrointestinal , reproductive,immunological and other systems. (Gupta et al.,1988,Rastogi et al. 1988, Saxena et al. ,1988,Bhandari et al.1990,Cullinan et al. 1996,Culillan et al.,1997) Bronchial asthma,Chronic Obstructive Airways disease . ,recurrent chest infections and fibrosis of the lungs. (ICMR, 1987 – 1991) are the principal effects of exposure induced lung injury . The prevelance of pulmonary tuberculosis among the exposed population has been found to be 3 – 4 times than that of national average.

According to six monthly morbidity survey in Bhopal by ICMR ,there were three times more persons with respiratory symptoms in 1991 as compared to 1987.The damage to respiratory system and particularly the lungs comprises the most obvious and significant part of the overall health damage.

The highly reactive group –N = C = O in MIC reacts with biological substrates with active H atoms such as that in hydroxyl and amino groups causing carbamylation .MIC and other isocyanates can carbamylate protein including haemoglobin and DNA (Bhattacharya et al.,1988, Segal et.al.,1989)



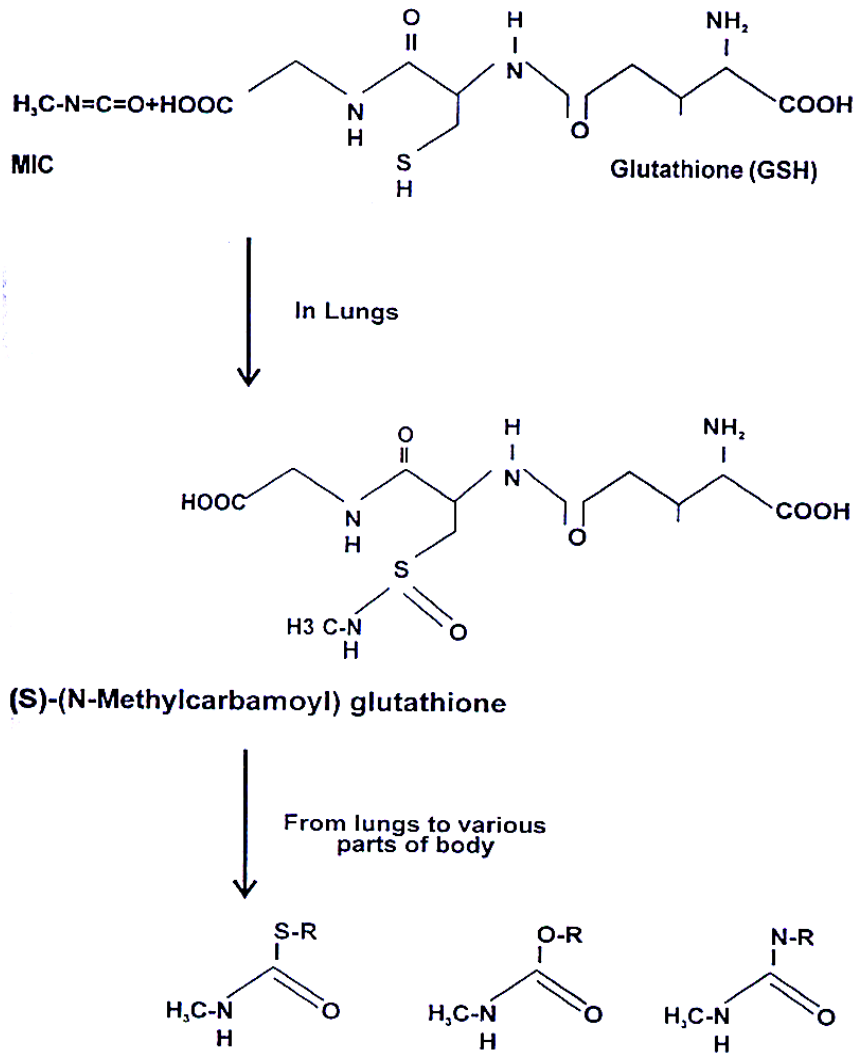
Proteins change,enzymes become inactive ,receptor get stimulated,cells are destroyed and there is inhibition of choline esterase and adenyl cyclase system. There are also evidences of chromosomeal aberrations and increased rates of sister chromatid exchange in Bhopal victims.(Goswami 1986 ,Goswami et al.,1990, Ghosh et al.,1990)

Chemical research by several scientists since the Union Carbide disaster has shown that M IC toxicity does not stop at lungs , as most toxicologists believed ,but the MIC may be transported throughout the body through binding with glutathione. Glutathione isocyanates with thiols extends the half life and toxicity of the iso thio cyanates and serves to distribute toxic chemical to different organs. (Bruggeman et al.,1988,Baille and Slatter 1991).Glutathione is especially abundant in the fluid lining of t he lung.When functioning normally, it mops up toxic chemicals in our blood and tissues and carries them out of the body through the excretory Organ. MIC forms a conjugate with glutathione in fluid lining of lung. GSH transforted MIC can trigger toxic effects through out the body.Conjugate disperses throughout the body and releases MIC.

Baille and Slatter (1991) have described that the glutathione some times acts as an " accessory " to toxic agents instead of flushing them out out of the body. When it is working normally glutathione intercepts toxic contaminants by combining with them to form so called "conjugate " .The conjugate passed round the body and is ultimately discharged harmlessly in bodily waste. They demonstrated in their experiments that glutathione cysteine conjugate were able to harm living cells by "unloading " their toxic cargo of MIC on to protein and membranes in the cells.

It seems that MIC with the help of a molecular coconspirator,is an even more noxious chemical than originally believed. Thus transported MIC can lead to toxic effects all over the body through carbamylation reactions with various enzymes,macromolecules and membrane structure. It is illustrated via Figure . given here.

Fig. 1.4-1 : Carbamylation reactions with various enzymes, macromolecules and membrane structure can lead to toxic effects.



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