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A STUDY ON CHROMOSOME ABBERATIONS CAUSED BY BHOPALGAS TRAGEDY

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ABSTRACT

The findings of the investigation revealed two patterns. Despite the fact that persons who were significantly exposed to the gas today have fewer aberrant cells, the frequency of aberrations within these cells has grown with time. Another leading to a rise in chromosomal abnormalities in persons who had not been subjected to the gas or had just been mildly sensitized to it. Methyl isocyanate, which was emitted by Union Carbide, has been shown to cause DNA damage in humans when it interacts with proteins. Studies conducted by the Indian Council of Medical Research in the years thatfollowed the gas spill revealed that such harm had occurred. The study showed the aberrations according to the sexes of the populace exposed and unexposed to the gas.

KEYWORDS: Chromosomal, Gas, Methyl Isocyanate, Aberrations, Cell.

I. INTRODUCTION

An industrial accident at Union Carbide India Limited (UCIL), Bhopal, in 1984 claimed the lives of thousands of people instantly and many more over the long-term. The clastogenic potential of MIC has been established by genetic investigations on exposed individuals and experimental animals. Radiolabeled MIC has demonstrated its ability to penetrate lung membranes and spread throughout the body. Chronic impacts on reproductive outcomes, pulmonary function, and the ocular system have shown a long-term impact on survivors. Since there was a dearth of information about how harmful microorganisms (MICs) were, the urgent action plan dealing to rehabilitating survivors, environmental sampling, and creating an exposure index could not be implemented. In just over two hours, almost 27 tonnes of MIC were dispersed across an area of 40 square kilometres by a gentle breeze from the UCIL facility. Due to the uneven terrain topography and winter fog, it was predicted that exposure and air gas density would vary. In spite of this, people who left their homes and were exposed to a dense gas cloud in the surroundings were severely harmed.

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That classification was disputed by the International Medical Commission on Bhopal (IMCB), which utilised distance from UCIL as a proxy for exposure ten years later, based on the mortality of December 3–6, 1984. Despite this, neither national nor international agency have any plans to follow up on genetic impacts. For starters, MIC was left in bulk storage (42 tonnes) for too long, the refrigeration system wasn't working,and there were no basic safety precautions in place like tracking the pressure and temperature of each tank or having adequate or working facilities for neutralising a highly reactive chemical product, all of which contributed to the disaster. All in all, the catastrophe appears to have been initiated by an unexpected surge in water through a malfunctioning valve. Uncontrollable runaway response may have been caused by one or more of these reasons, as shown in the Official Report of Varadarajan and associates (1985). Bhopal was caused by a variety of faults, not just those related to the 'hardware' but also those related to "operator, information, and systemic error categories," according to Bowonder's 1987 report, "Analysis of the Bhopal Accident." It's the failure of safety management systems and processes at the corporate level that is the most crucial factor in the incidence of these mistakes, according to him.

II. HUMAN MORTALITY & MORBIDITY:

According to all accounts, the Bhopal Gas Tragedy claimed many human (and animal) lives in a matter of minutes. After only a few hours, the death toll quickly grew to over 200, and the toll continued to grow for the remainder of the day. The MLI mortuary in Bhopal received 311 bodies on December 3, 1984, accompanied by another 250 on December 4, 1984, however it is possible that not all of the bodies had made it there. Afterwards when, the death toll decreased. In December 1984 alone, 731 corpses were received; in 1985, 103; in 1986, 90; in 1987, 44; and in 1988, 22. Bhopal's morgue data may not include all of the city's dead. More than 2000 people lost their lives in the first few days, which is more than the official government death toll of 1900. An enormous majority of human life has been lost, as well as the long-term incapacity of many survivors. As a result, many of the survivors were either newborns or small children, and in other cases, the entire family had perished. As many as a hundred thousand people were found to be suffering from various health problems for weeks or even months after the disaster. The MLI continued to conduct gas-affected victim autopsy in later years, but at a much decreased rate. As to how long the symptoms would stay and if the ocular manifestations of more than 90 percent would lead to substantial vision impairment, there were several important questions.

III. MATERIAL AND METHODS

The 130 people evaluated were divided between those who had been left vulnerable to the gas and those who had not been exposed, meaning those who lived in locations where the gas had not dispersed. A random computer-generated selection of listed survivors was used to choose the group ofthose who were exposed. The 'control group' was drawn from the overall population and matched for gender and age to the experimental group. Gravity sedimentation was used to separate plasma from peripheral venous blood in heparinized vials. RPMI-1640 medium (GIBCO, Grand Island, NY) was supplemented with 20% heat-inactivated foetal calf serum (Sera Lab., England) and phytohemagglutinin (M Form) at 0.2 mL/5 mL of culture after an hour. 6.0,ug of 5-bromo-2- deoxyuridine (BrdU, Sigma Chemical Co., St. Louis, MO) was added per 1.0 mL of culture media in the dark to avoid

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photoinactivation in the set for sister chromatid exchange studies.

Each individual had four cultures grown in two duplicate sets at 37°C in the dark. Chromosome aberrations were studied for 48 hours, whereas SCEs were incubated for 72 hours. Colchicine (40, ug/mL) was given to each culture two hours before the culture was to be terminated. A second incubation period of two hours was followed by centrifugation of the cells, which were then subjectedto 37°C treatments of hypotonic solution (0.09 percent NaCl in deionized water) and fixative solution (methanol acetic acid 3:1). Centrifuged three times, fixative was used to resuspend the cells. Giemsa slides were prepared and stained using the normal process, with adjustments where necessary.

The slides were coded and evaluated by two observers who were not aware of each other's identities. Each patient had 100 first-cycle scattered metaphases (M1), 50 scattered complete second-cycle metaphases (M2), and 200 metaphases recorded for chromosomal abnormalities (including damaged cells; total aberrations with or without gaps, breaks per injured cell). These included the RI and the metaphases in the first, second, and third cycles of the cell cycle. Student's t test was used to compare exposed and unexposed populations of both sexes' deviations for the first three sets of parameters.

IV. RESULTS AND DISCUSSION

Chromosome aberrations occurred more often in the exposed group than in the control group, particularly among female patients (Table 1). Chromosome splits, gaps, dicentrics and rings were among the anomalies that were documented. Even after 1120 days, the chromosomes and quadriradialstructures were still present (Figs. 1-4). A larger number of fractures per cell was found among females exposed to radiation than those who had not been exposed. In men, there was no statistically significant difference. This contradicts the report of greater incidence of micronuclei in male mice exposed in vivo to MIC.

Table 1. Sex-related chromosomal aberrations can be impacted by the use of methyl isocyanate.

Sex	Exposed type	Number of subjects	Cells scored	damaged cellS	Breaks/celia		Aberration/ damaged cella
					+ Gaps	- Gaps	
Male	Exposed	40	4150	5.80±1.99	0.080±0.024	0.064±0.023	1.27±0.32
	Unexposed	20	1890	5.50±1.83	0.070±0.031	0.056±0.025	1.22±0.24
Female	Exposed	42	3970	6.77±2.40	0.100±0.027	0.077±0.025	1.44±0.37
	Unexposed	28	2340	5.50 ± 2.16*	0.070±0.032k	0.050±0.021k	1.15 ± 0.36t
Male	Exposed	40	4146	5.80± 1.99	0.080±0.024	0.064±0.023	1.27±0.32
Female		42	3970	6.77±2.40	0.100±0.027k	0.077±0.025*	1.44±0.37*

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Male	Unexposed	20	1872	5.50±1.83	0.070±0.031	0.056±0.025	1.22±0.24
Female		28	2340	5.50±2.16	0.070±0.032	0.050±0.021	1.15±0.36

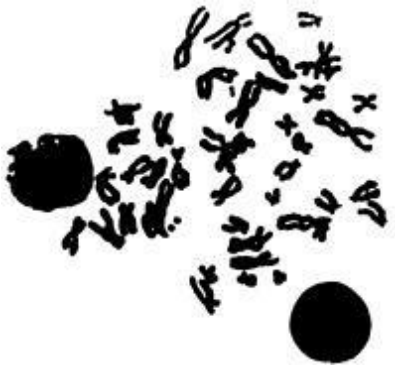

Figure 1. Replicating minutes.

Figure 2. Quadriradial and dicentric configurations.

Figure 3. Endoreduplication.

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Figure 4 Sister chromatid exchange

Table 2. Sympathetic consequences of methyl isocyanate-SCE exchanges on adolescent girls.

Sex				Total SCEs	Range of	SCEs/cell	
	Exposure type	Number of subjects	Total cells scored	counted	Mean SCEs	Mean + SD	
Male	Exposed	40	1540	15730	5.40 - 19.17	10.69	± 3.41
	Unexposed	20	930	10800	6.46 - 17.67	11.61	± 1.92
Female	Exposed	40	1480	15390	5.94 - 19.82	10.87	± 3.49
	Unexposed	30	1160	13800	6.60 - 23.39	12.54	± 3.57
Male		40	1540	15730	5.40- 19.17	10.69	± 3.41
Female	Exposed	40	1480	15390	5.94 - 19.82	10.87	± 3.49
Male		20	930	10800	6.46- 17.67	11.61	± 1.92
Female	Unexposed	30	1160	13800	6.67 - 23.39	12.54	± 3.57

No significant difference in the frequency of SCEs was seen among those who had been exposed and those who hadn't. For the most part, the range of 4 to 14 cells per cell was above the previously stated baseline range of 4 to 14. A person's exposure or sex did not influence the number of SCEs, the number of breaks per cell, or the proportion of cells with SCE (Table 2). There is no evidence to substantiate an earlier claim of an increase in the incidence of SCEs in those who were exposed to MIC during the Bhopal tragedy. In a separate communication, no correlation was found between variables such as smoking, drinking, and pregnancy. Between the exposed and unexposed populations, there was no significant difference in the replicative index.

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Fetal loss may have been caused by MIC exposure in some of the females with persistent chromosomal abnormalities. The only known clastogenic substance they've been exposed to is a chest X-ray. Male day labourers outnumber female housewives by a wide margin in this study. There is still a lot more work to be done before the findings are complete. It is important to consider a person's gender, as well as their physiological and nutritional health, when considering the impacts of harmful compounds like MIC. Other aspects, such as genetic makeup, must also be considered. People exposed to MIC may be more susceptible to chromosomal damage because of the persistence of abnormalities even after 1120 days.

V. CONCLUSION

Women exposed to the gas appear to have an increased risk of chromosomal abnormalities 1120 days after exposure, according to a new research. The history and clinical symptoms (if any) of these instances have not yet been completely correlated to the findings because the selection was made as double blind to permit independent verification by two observers. Clastogenic effects may remain even after 1120 days, based on the occurrence of chromosomal changes such dicentrics, rings, and quadriradial configurations. Damaged lymphocytes may survive in circulation for a long time since chemical damages are largely S dependent for manifestation in the ensuing divisional cycle, and these abnormalities can only be detected in vitro. Pdioxane's effects are analogous to those of contact.

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