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LETTER TO THE EDITOR

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INVESTIGATING GENETIC OUTCOMES FOLLOWING 1984 TOXIC UNION CARBIDE DISASTER IN INDIA: EPIDEMIOLOGICAL CHALLENGES

The Indian Council for Medical Research [ICMR] has called for proposals to study the long-term effects of 1984 Union Carbide Industrial Disaster at Bhopal, one of the worst industrial disasters ever, which led to the release of poisonous Methyl Iso-cyanate into the environment and had impact on the population living in the vicinity of the disaster site. Proposals are invited to study the long-term effects, if any, of toxic gas exposure on the exposed population. The effects under study include genetic disorders, low birth weight, growth and developmental disorders, and congenital malformations. The aim of this activity is to launch a community-based epidemiological study to investigate the possibility of genetic effects under conditions of a very short environmental exposure to a chemical in a gaseous form.

Designing such a study is exceptionally fret with challenges, starting with exposure assessment. The major question is who is the exposed case. Presently, there is no alternative to the testimonial of the people affected two and half decades back. Secondly, 25 years since the disaster, the population composition within a 2 km radius of the disaster site is so drastically different, largely due to migration processes, that it is practically impossible to assess the dose response relationship in a similar population sample. Moreover, the opportunity to apply for compensation makes many unexposed people claim that they had been exposed. The government list of the 'victims', prepared for legal purposes, is highly political in nature, hence its authenticity is disputable.

Purely from the occupational health point of view and considering the biological plausibility, there is no documented evidence for a chemical (in a gaseous state) to induce genetic effects under conditions of a single exposure (inhalation) for a short-term period (a day or two). Inhalation-related exposure producing health effects such as pneumoconiosis is thought to be caused by either particulate or fibrous form of the agent. Gases are known to induce only immediate reactions ranging from eye irritation to fatal poisoning. However, there has been no precedence of a day or two days long exposure to a chemical in a gaseous form. I should like to stress that the exposure considered cannot be longer than two days since the hazardous gas is expected to be at a high concentration in the atmosphere for only a few hours directly after the industrial disaster, and on the following day, the concentration of MIC in the air is likely to be negligent due to air currents.

With regard to the Bhopal gas disaster, there was no evidence of a heavy fog on December 3–4, 1984, and hence no reason to believe that the fog or smog could have trapped MIC thus leading to high-level exposure of the population living in the vicinity of the disaster site. Neither is the MIC or its effluents radioactive to suspect that the population could be at a continued risk of exposure months and years after it had been accidentally released to the environment.

Repeated studies on the genetic effects of exposure to environmental pollutants have clearly demonstrated that it is only the chronic chemical exposure that can cause genetic damage [2]. Except for the radioactive substances, the chemicals in general have been seen to cause genetic damage especially on the somatic cells, which may stretch as far as the second generation. The chemicals in question are particularly those absorbed through ingestion. The classical case is diethylstilbestrol (DES) taken during pregnancy and the vaginal cancer developing in female offspring of mothers who had been treated with DES in pregnancy. Profound effects like birth defects could also be seen, such as those in the infamous Thalidomide disaster. Please note

that in all the case studies mentioned above, the oral route of absorption was involved.

Even for chronic exposure to chemicals, e.g. to persistent organic pollutants, after decades of lifetime exposures, there has been no conclusive evidence for the genetic effects. One of these chemicals has been DDT, an insecticide (now banned) for which numerous studies on health effects to humans have been carried out since it started to be manufactured in 1938.

Seeking for the genetic effects as the outcome of the Bhopal gas disaster does not seem to be rational because for the mutations to occur, the hazardous agent has to have impact on the germ cells and only mutations with severe effects, such as chromosomal aberrations, can lead to "birth defects". Only in this case there is a potential for the effect to be passed to the offspring. Here, such profound, trans-generation effects cannot be expected in acute exposure lasting for a day or two as is the case in the industrial disaster in Bhopal.

Researchers have been trying to document adverse health effects in long-term cohort studies on humans who have been exposed to a vast number of synthetic chemicals since they entered the market for the first time. By mid 1960s, these individuals began to bear children who were the first generation of humans exposed to hazardous chemicals in utero. About 1980, this generation reached the reproductive age, and although the search for adverse health outcomes in their offspring continues, the period is much too short for studying any genetic effects [3].

Moreover, in the case of the Indian Union Carbide disaster, the unavailability of biological markers for MIC makes it almost impossible to definitely classify the study population into the exposed and unexposed group to study adverse health outcomes.

After 25 years since the disaster, and under conditions of methodological deficiencies which make the negative or positive association equally probable, the findings of such studies will certainly be questioned.

In the past, human epidemiological studies to determine the genetic outcomes of exposure have focused only on the health of the exposed individuals. It is not surprising that many of these studies have failed to elicit any associations between adverse health effects and chemicals (Type II error).

My reason for calling the genetic studies of Bhopal gas disaster unethical stems from the fact that in research ethics, the relevance of the study design, methodology and duration is among the essential requirements that have to be commensurate with the expected outcomes. The call for a Bhopal genetic study lacks any biological justification, and so does searching for genetic effects within an extremely short period of 25 years. All this, coupled with a severely limited budget for research, makes the outcomes of such study highly disputable.

However, there is certainly a need to identify a biological marker for MIC, and investigate adverse health effects of MIC exposure, including reproductive outcomes like miscarriage, decreased human sperm count, and MIC's acting as endocrine disruptor, as well as the high SMR in the exposed population. Studies focused on current health problems among the victims would also be desirable so that new victims affected by the disaster might be brought to light and receive proper medical care and financial compensation if necessary [3].

KEY WORDS:

MIC, Genetic effects, India, Health effects, Bhopal

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