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# A COMPARATIVE STUDY OF THE RISKS OF CANCER MORTALITY FROM IONIZING RADIATIONS AND CHEMICAL POLLUTANTS

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## 1. INTRODUCTION

Quantitative information on the risk of cancer mortality associated with ionizing radiations and chemical pollutants is of great value in the planning of major nuclear and chemical operations. Epidemiological and laboratory studies provide sufficient basis for an attempt to bring out such quantitative data for the purpose of planning. The data on cancer mortality caused by ionizing radiations has been developed over the last fifty years and can be used for similar risk estimates for chemicals. No direct study on the late effects of carcinogenic chemicals has ever been carried out.

As early as 1903 it was shown that X-rays can induce leukemia in mice (1). During the following decade, evidence of increased incidence of cancer among radiologists became available. By 1922 it was found that more than 100 early radiologists had died of occupationally produced cancers and leukemia (2). This early experience was associated with very large exposures, and due to paucity of dose measurement techniques, no dose-effect relationships could be established. During the last two decades data on dose-effect relationships became available through animal experiments, but this could not be used directly for human risk estimates, due to very large doses used in such experiments and large differences in the life span and tissue radiosensitivity of the species.

Extensive data on cancer mortality has been compiled based on the studies carried out on the survivors of Hiroshima and Nagasaki, patients who received large doses from X-rays used for the treatment of ankylosing spondylitis , radium dial painters, the radiologists of early days who used X-rays without precautions and patients treated with radioactive iodine for hyperthyroid. Mortality data for the incidence of lung cancer in uranium miners also became available during the last few decades. Several national and international bodies, notably the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) and the Committee on the Biological Effects of Ionizing Radiations (BEIR Committee) of USA have made extensive use ofthe above information to arrive at quantitative risk estimates for ionizing radiations (3, 4), which will be summarised in this paper for comparison with the risks of chemical pollutants.

Induction of cancer by chemical pollutants has also been recognized for a long time, for example, the incidence of scrotal cancer in chimney

sweepers was first reported in 1775, which was later attributed to the presence of the chemical carcinogen benzo(a)pyrene in the coal soot deposited in the chimneys (5). These workers were in the habit of taking off their clothes during work, and prolonged contact with soot often produced cancer of the scrotum. A large number of chemicals have since been recognized as carcinogenic, mainly those belonging to the family of polycyclic hydrocarbons, heavy metals, asbestos, vinyl-chloride and a variety of organic oxides. For quantitative information on cancer deaths from chemicals, data is available for lung cancer mortality and the likely causative agent benzo(a)pyrene for several industrial cities of the world. Systematic information is not available for a large variety of other chemical carcinogens for the quantitative assessment of dose-effect relationships.

The model proposed in this paper is based on the estimates of cancer deaths per man-rem of radiation exposure and finding of rem-dose-equivalents (RDE) for benzo(a)pyrene which has been shown to correlate with lung cancer mortality.

## 2. CANCER INCIDENCE FOR CONTINUOUS PROTRACTED RADIATION EXPOSURES

On the basis of human data for the radiation exposures mentioned in the previous section, the incidence of cancer death per man-rem has been estimated to be around $200 \times 10^{-6}$. This estimate is based on the slopes of dose-effect curves for the incidence of leukemia and other cancers in the groups of individuals considered. The types of cancer studied include those of the breast, lung, GI tract including stomach, bone and all other sites. The incidence of mortality from all cancers, including leukemia is estimated to be 6 per rem per year and the total cases of cancer per year including the non fatal cases is estimated to be twice this number (BEIR Committee, 1972). For continuous exposure of 1 rem per year for 30 years the mortality rate is, thus, $180 \times 10^{-6}$. This estimate has been rounded off to $200 \times 10^{-6}$ per man-rem for the <u>equilibrium situation</u> reached after a large population has been continuously exposed to a protracted dose for several years. These estimates are based on the assumption of linear dose-effect relationships for low total doses for which the incidence would be independent of the dose rate. In the dose range actually involved in the human exposures considered, linearity may be assumed on the basis of animal experiments, particularly those with low LET radiations in the dose range of 100 rad. Therefore the above risk estimates should be reasonably valid with the reservation that the target tissue has fairly large radiosensitivity for the induction of cancer. These risk estimates have been used as basis for quantitative comparison with lung cancer mortality produced by benzo(a)pyrene.

## 3. QUANTITATIVE DATA FOR CHEMICAL CARCINOGENESIS

The methods of regression analysis have been generally used for obtaining quantitative data for mortality due to chemical pollutants (6, 7, 8). The basic model may be written as

$$X_i = A_o + A_1 P_i + A_2 Q_i + e$$

where $X_i$ is the total cancer mortality rate in the city i, $P_i$ is the mean

concentration of pollutant P and $Q_i$ is the mean concentration of pollutant Q (or some factor Q) in city i. $A_1$ and $A_2$ are regression parameters in the model and e is the unknown error term. The regression parameters are estimated by minimising the quantity

$$\sum_{i=1}^{n} (X_i - A_o - A_1 P_i - A_2 Q_i)^2$$

The coefficients $A_1$ and $A_2$ provide a direct measure of the contribution of the pollutants to the total risk. The validity of the model can be checked by 't' statistics and by '$R^2$' parameter as defined in reference (9). In using the above model it is necessary to use a wide range of the values of X and to ensure that the variables P, Q, etc. are not correlated. For quantitative estimates of the risk of lung cancer, epidemiological data on benzo(a)pyrene and lung cancer incidence has been used in the above model by different authors. For example, Carnow and Meier (8) used the above model for finding the relative contributions of benzo(a)pyrene and cigarette smoking to the incidence of cancer in a population with known lung cancer death rate. They estimated 5% increase in the incidence for an increase of 1 ng/$m^3$ of benzo(a)pyrene, which will be used here for quantitative comparison with radiation risks. Regression analysis also provided an estimate of the contribution of cigarette smoking. For the estimated contribution of benzo(a) pyrene, the lung cancer incidence would be doubled if the pollutant concentration is increased by 20 ng/$m^3$. Similar correlations can be found for different chemical pollutants and the type of cancer they cause if sufficient epidemiological data is available.

## 4. A COMPARATIVE STUDY OF THE RISKS

We are now in a position to compare the risks of ionizing radiations and chemicals. The total risk of all cancers induced by radiations is 200 x $10^{-6}$ per man-rem, as explained earlier. If we take 25% of the total cancers as lung cancers (close to the estimates of BEIR Committee), the risk is 50 x $10^{-6}$ per man-rem for whole body exposure.

Cancer of the lung and respiratory tract has been showing a continuous increase in most industrialised cities of the world, and at the current rate, incidence of 500 x $10^{-6}$ may be taken as a representative world mean. On the basis of 5% increase per ng/$m^3$ of benzo(a)pyrene we get 25 additional cases per ng/$m^3$. The average benzo(a)pyrene levels in the urban areas are in the range of 1-4 ng/$m^3$, the higher value being more prevalent, which would give a contribution of 100 x $10^{-6}$ to the lung cancer mortality. So far no standards have been set for benzo(a)pyrene in most countries.

The ICRP radiation dose standards for radionuclides are set at levels that would limit the whole body dose to 500 mrem/year. For this dose to the lung, the lung cancer mortality rate would be 100 x $10^{-6}$, comparable with the estimated current mortality rate for benzo(a)pyrene in the urban areas. This would imply that for even a single carcinogen benzo(a)pyrene the current levels are comparable with the ICRP dose limit for groups and individuals in the population.

Rem dose equivalent (RDE) of benzo(a)pyrene may thus be written as 8 ng/$m^3$, corresponding to cancer mortality rate of 200 x $10^{-6}$. In

this context it is important to note that recently "Committee-17" of the Environmental Mutagens Society has suggested a unit called Rem Equivalent Chemical (REC) for quantitative assessment of the genetic effects of all environmental mutagens. Thus REC corresponds to the quantity of chemical substance which produces an amount of genetic damage equal to that produced by one rem of chronic exposure to ionizing radiations in the same test system. As an example, the equivalent lifetime dose for the nitrites, which are mutagenic, could be as high as 8 REC (10). The estimates of REC are generally based on laboratory experiments with simple biological systems. The concept of RDE proposed here could be developed on the basis of epidemiological studies. The proposed RDE concept can be extensively used for different cancers and causative agents, particularly cancers of the breast, bone and GI tract, for which human data on induction by radiations is available. This concept would be of great value in the laying of standards for radiations and chemicals on equivalent risk basis.

Experimental evidence shows that the effects of chemical carcinogens are often synergistic, involving two or more causative agents. For example, benzo(a)pyrene in the presence of heavy metals is known to enhance the effect considerably. Therefore, future studies should include data on both, and regression analysis could then be carried out using a single regression parameter for the product of two pollutants. Such analysis would be valid if the individual pollutants show very little effect but when both are present, the effect is enhanced considerably.

To sum up, this paper has highlighted the type of epidemiological data for chemical pollutants and the data on radiation risk estimates from human experience and animal experiments, which could be used for realistic assessment of the risks of cancer mortality. There is an urgent need for extensive surveys to predict the likely hazards to which the world population could be exposed in the absence of adequate control measures.

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