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A PROBABILISTIC METHODOLOGY FOR ESTIMATING RADIATION-INDUCED CANCER RISK*

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ABSTRACT

The RICRAC computer code has been developed at Oak Ridge National Laboratory to provide a versatile and convenient methodology for radiation risk assessment. The code allows as input essentially any dose pattern commonly encountered in risk assessments for either acute or chronic exposures, and it includes consideration of the age structure of the exposed population. Results produced by the analysis include the probability of one or more radiation-induced cancer deaths in a specified population, expected numbers of deaths, and expected years of life lost as a result of premature fatalities. These calculations include consideration of competing risks of death from all other causes. The program also generates a probability frequency distribution of the expected number of cancers in any specified cohort resulting from a given radiation dose. The methods may be applied to any specified population and dose scenario.

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A PROBABILISTIC METHODOLOGY FOR ESTIMATING RADIATION-INDUCED CANCER RISK

The RICRAC (radiation-induced cancer risk analysis code) computer code has been developed at Oak Ridge National Laboratory to estimate the probability that a group of persons exposed to low levels of radiation will eventually experience one or more radiation-induced cancer deaths. The methods are applicable either to a population of any size and age distribution, or to a cohort of any number of persons all simultaneously liveborn. The specified population is subjected to a scenario of radiation dose, and is then observed until all members have died. The maximum individual life expectancy is arbitrarily limited to 110 years, so the analysis is halted at that time. Thus, the RICRAC method essentially considers a snapshot population with a given age distribution, and determines the probability of the occurrence of one or more radiation-induced cancer deaths for individuals of each age at the onset of the analysis period.

The latency period (period following exposure in which there is no risk of radiation-induced cancer) and plateau period (period in which the radiation-induced risk persists) for each type of cancer considered is specified for fetal, child, and adult exposure. Risk factors may vary continuously with age. Radiation exposure may occur at any point(s) within the 110 year period of analysis, or may be continuous over the entire period.

Competing mortality risks (i.e., risks of death from causes other than radiation-induced cancer) are considered through an actuarial life table analysis. Deaths attributed to competing risks, as determined by actuarial data, are removed from the population in each age category

before the incremental mortality risk is computed. Consideration of competing risks is important because of the delay inherent in the induction of radiogenic cancer. During or after radiation exposure, a potential cancer victim may experience years of life in which he is continually exposed to risks of death from daily activities.

In addition to the probability of a given individual eventually dying from incremental radiation exposure, results of the RICRAC code also include estimates of the expected number of radiation-induced deaths, expected years of life lost, and the decrease in the population life expectancy for each age-at-onset group in the population. Estimates are also produced for the probability of one or more radiation deaths in the total population, as well as the expected number of deaths and years of life lost in the total population. Another useful product of the analysis is a probability distribution of the expected number of incremental deaths, normalized to a cohort of 100,000 persons. This probability distribution is usually determined for the group assumed to be born at the onset of the 110 year period.

Estimates of the expected incremental mortality risk from leukemia and from all other cancers due to exposure of a population of one million persons have been computed. In this analysis we have assumed risk factors, latency periods, and plateau periods suggested by the 1972 BEIR report for fetal, child, and adult exposure. The absolute risk model was selected for use in this calculation, but the method is equally applicable to relative risk. The population age distribution and the actuarial mortality data for competing risks approximate those of the 1976 U.S. population.

In the scenario, the population receives a dose of 0.01 sieverts (1 rem) to critical organs during the first year of the 110 year analysis period, and no dose thereafter. This case might represent an accident where only an initial brief exposure is experienced. The estimated incremental mortalities due to this exposure are 29 from leukemia and 119 from all other cancers. The average years of life lost per fatality in the populations is 41.3 for leukemia and 21.7 for all other cancers. A distribution of the expected number of deaths with respect to the age at which exposure was incurred is shown in Fig. 1. A relatively large risk from fetal exposure is observed for each type of cancer considered. For leukemia we observe an essentially continuous decline in risk with increasing age of exposure. For all other cancers, however, risk declines rapidly for childhood exposures, returns to a higher level for early adult exposure, and then decreases continuously with age. This behavior may be explained by the observation that the adult risk factor for all other cancers is larger than that for children by a factor of five. A critical assumption in this calculation is that the risk factor is determined by the age at exposure. We note that the risk factors and other parameters used here differentiate only between fetal, child (0-9 years), and adult (over 10 years) age groups. The results might be considerably different if a more dynamic age dependency in these parameters were employed, allowing for changes in tumor susceptibility and efficiency of cellular repair mechanisms as a function of age.

A probability distribution of the expected number of leukemias resulting from this 0.01 sievert exposure in a cohort of 100,000 newborn infants is presented in Fig. 2. In this case the method predicts that

29 radiation-induced fatalities may be expected during the lifetime of the cohort, and that greater or lesser numbers of cancers are associated with lower probabilities of occurence. A probability distribution of this type may be generated for any specified group in the population.

The RICRAC computer code provides a versatile and convenient methodology for radiation risk assessment. Results produced by the analysis include the probability of one or more radiation-induced cancer deaths in the population, expected numbers of deaths, and expected years of life lost as a result of premature fatalities. The methods may be applied to any specified population and any dose scenario.

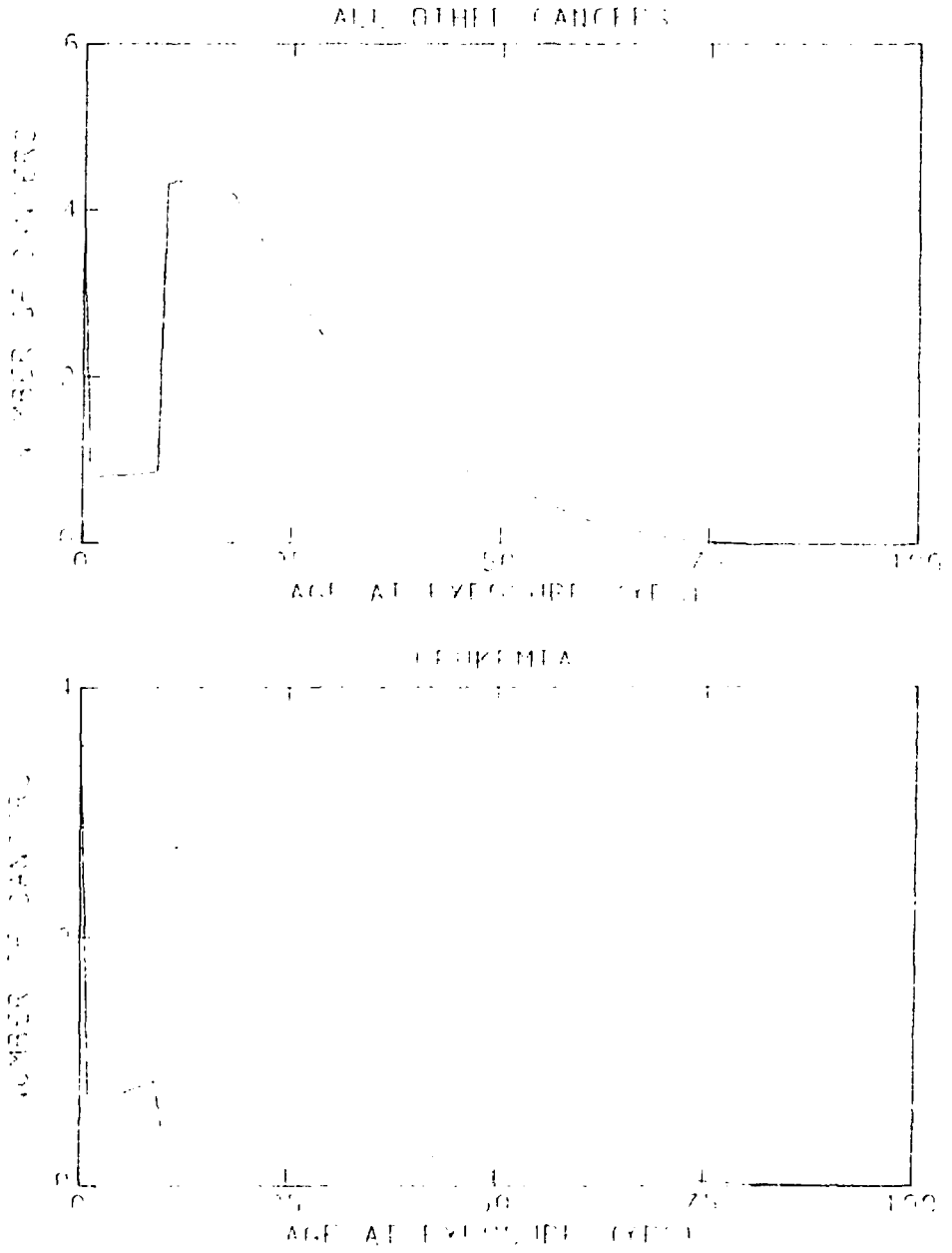

Figure 1. Expected frequency of fatal cancers in a population Of 1 million persons subjected to a 0.01 Sv (1 rem) exposure.

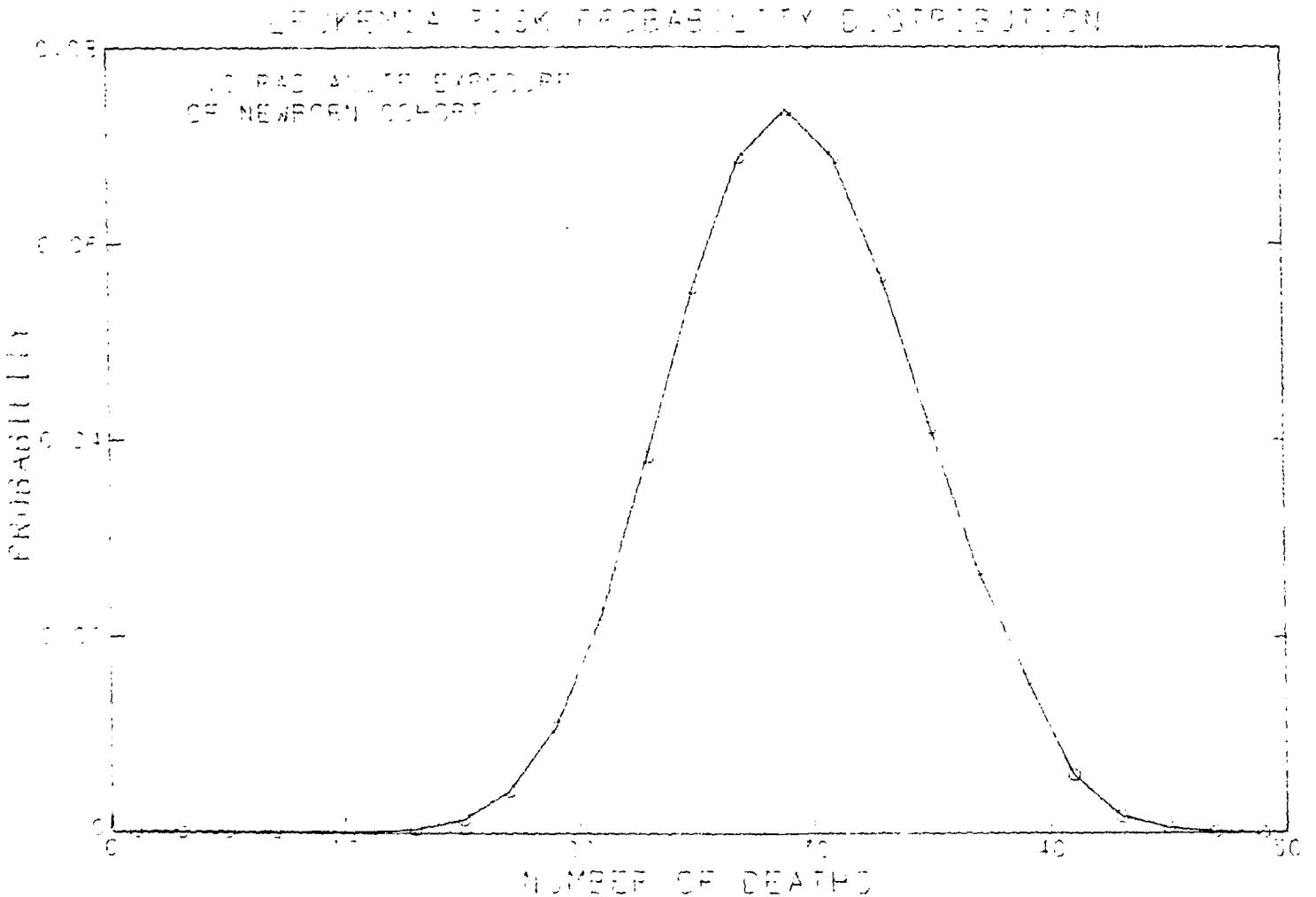

Figure 2. Probability distribution of the number of expected leukemias in a cohort of 100,000 newborn infants subjected to a 0.01 Sv (1 rem) exposure.