ASSESSMENT OF LEUKEMIA CAUSED DEATHS DUE TO INTERNAL RADIATION EXPOSURE

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ABSTRACT

A problem of finding the number of cancers, which are developed due to internal exposure to radioactive material, is not a trivial task. This problem is generally rather complex, because in case of protracted exposures, latency period may exceed the time of an individual's natural death, i.e. the age at death due to "natural causes". In this paper the model for calculating risk caused by an internal exposure (inhalation or ingestion of radioactive material) is modeled as a continuous irradiation till the end of an individual's life, taking into account natural deaths in the observed population. The basic tool in constructing the model were risk coefficients per unit dose, developed earlier [1]. Since an important role in radiation exposure of the people in South Serbia may play internal exposure to depleted uranium (DU), which was extensively used during the NATO bombing of Yugoslavia, the leukemia was chosen as a stochastic effect which is to be considered. For this purpose, some different (artificial) amounts of DU intake were assumed. In order to present the continuous exposure of the whole population living on the contaminated area, the model separately considers those born after the environmental contamination. Therefore, the overall population is divided into two parts: the one which was alive at the time of the release, (LG – Living Generation), and the second one, born after that (FG- Following Generations). The paper primarily intends to present the model for risk calculation for the LG part of population. However, just for the purpose of demonstration of the overall risk model, the contribution of the FG is added to get an overall risk assessment for the case of leukemia's deaths. Besides cumulative number of cases, which are usually calculated by other models, this model is able to assess differential values, what means it is able to predict the number of cases within a certain specified age and/or time intervals. According to results obtained by the model, some dependencies are shown, for example distribution of leukemia's induced deaths for a given age at death, and distribution of leukemia induced deaths with respect to the beginning of exposure. The model presented in this paper is the part of the HEALTH module of RODOS, new European decision support system for nuclear emergencies.

Key words: internal exposure, leukeia, risk assessment

1. Introduction

After an inhalation and/or ingestion, radioactive material may retain in the body for a long period, and are therefore always considered as continuous exposures. Such kind of exposure pathways are in module HEALTH (which is part of RODOS, a new European decision support system for nuclear emergencies) modeled as a consecutive series of short-term exposures. Internal exposure from inhalation comprises two different processes. The first one is inhalation (IH) of the radionuclides during the passage of the cloud, while the other one comes from the inhalation of radioactive material after its resuspension (IR) in the air. While IH is referred only to those alive at the time of the accident (LG part of population), IR refers to both, LG and FG.

2. Materials and methods

Let us consider the time and age scale, presented on Figure 1. Let us also suppose release of radioactive material occurs at *(t=0). A*t that time an individual from the observed LG population was at the age *a*, where $a \in (0, l_m)$. Due to the release of the radioactive material, an observed individual may incorporate it by inhalation of the contaminated radioactive plume, or resuspended material in the, and on the other hand the radioactive material may be incorporated by ingestion of the contaminated food. In any case, that intake (inhalation or ingestion) may occur at time t_b, where generally $t_b \in (0, \infty)$. However, for the purpose of our short analysis we will consider approximately the first hundred years after the accidental release of radioactive material. More exactly, in our numerical examples, we will consider time period of exactly 95 years, which is intentionally equal with the maximal life expectancy in the observed population. After that single exposure at time t_b, a lethal cancer may occur up to the time t_S with the probability $w(t_S - t_b, a_b, o)$, where *o* stands for the target organ. Number of lethal cancer developed at organ *o*, which will occur at location *l* up to age *A*, and up to time *T*, is given by

$$N(T, A, l, o) = P(l) \int_0^A f(T, a, l, o) q(a) da \quad , \quad A \geq T \tag{1}$$

where the function *f(T,a,l,o)* is given by

$$f(T, a, l, o) = \int_0^{\min(a,T)} dt_b \int_0^{\min(a,T)} \dot{D}(t_b, a_b, l, o) \dot{w}(t_S - t_b, a_b, o) dt_S \tag{2}$$

with t_b - time of exposure [yr], $a_b = a - t_S + t_b$ - age at exposure [yr];, a - age at death [yr], t_S - time of radiation induced death [yr]. The other parameters are defined earlier. In the above expression age at the exposure $a_b = a - t_S + t_b$ is used for shortening (see also Figure 1). The age a_0 from Figure 1, is age at the accident, with

$a_b = a_0 + t_b$. Equation (1) gives cumulative number of deaths expected to occur up to the certain time *T*, and up to the certain age at death *A*. That means it counts all radiation induced deaths up to time T, and up to the age A.

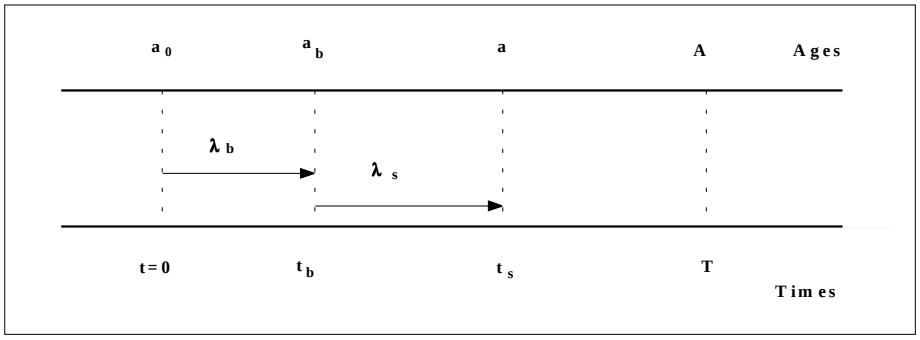

Figure 1Time and age parameters for continuous exposure pathways

Let us further with $\Delta N_A(T_1, T_2, a, l, o)$ denote number of deaths which is expected to occur at location *l* due to cancer *o*, exactly at the age *a*, and within time interval (T_1, T_2). This number is calculated by

$$\Delta N_A(T_1, T_2, a, l, o) = P(l) \quad q(a) \quad \int_{T_1}^{T_2} dt_S \times \int_0^{t_s} \dot{D}(t_b, a_b, l, o)\dot{w}(t_S - t_b, a_b, o)dt_b \quad , \quad a \geq T_1 \tag{3}$$

where again $a_b = a - t_S + t_b$. Let us now consider two opposite requirements in this assessment. From one side, statistical nature of stochastic effects requires rather large number of observed individuals. On the other side, environmental doses, which cause all these effects, may differ significantly from one site to another, and therefore require as small location site as possible. Being in between these two opposite requirements, each of them having its own reason for increasing the confidence in the obtained results, the user of HEALTH can define its own threshold size, below which no risk assessment is being made. So, according to above discussion, the numbers obtained by equation (3) in principle do not give sufficient statistics, and plots, usually $\Delta N_A(T_1, T_2, a, l, o)$ vs. *t* do not offer satisfactory statistics, what means note complete reasonable graphs. From these reasons HEALTH calculates the number of cancers summed up over all locations, i.e.

$$\Delta N_A(T_1, T_2, a, o) = \sum_l \Delta N_A(T_1, T_2, a, l, o) \tag{4}$$

If $T_1 = 0$, from equation (4), the cumulative number of deaths up to time *T* is obtained

$$\Delta N_A(T,a,l,o) = P(l)q(a)\int_0^T dt_b \int_{t_b}^T \dot{D}(t_b,a_b,l,o)\dot{w}(t_S - t_b,o)dt_S \quad (5)$$

where again $a_b = a - t_S + t_b$. At present, in HEALTH observation time extends up to the value of the maximal life expectancy in the observed population. Some earlier risk analyses (see [2] for example) had used more extended time periods, up to 300 years after the accident.

Let us with $\Delta N_T(t,A,l,o)$ denote time dependent number of deaths expected to occur up to the age A, due to lethal cancer o, at location l. HEALTH calculates this number by

$$\Delta N_T(t,A,l,o) = P(l)\int_t^A q(a)da \int_0^t \dot{D}(t_b,a_b,l,o)\dot{w}(t - t_b,a_b,o)dt_b \quad (6)$$

where condition $A \geq t$ is explicitly built into the integration's limits. Let us further $\Delta N_T(t,A_1,A_2,l,o)$ denotes time dependent number of lethal cancer developed on the organ o, which are expected to occur at location l with the condition that all deaths will occur within the age interval (A_1,A_2). HEALTH calculates this number by

$$\Delta N_T(t,A_1,A_2,l,o) = P(l)\int_{A_1}^{A_2} q(a)da \times \int_0^t \dot{D}(t_b,a_b,l,o)\dot{w}(t - t_b,a_b,o)\ dt_b \quad , \quad A_1 \geq t \quad (7)$$

with $a_b = a - t + t_b$. Due to the reasons, given bellow equation (3), HEALTH calculates only the overall number of effects, summed over all locations, according to

$$\Delta N_T(t,A_1,A_2,o) = \sum_l \Delta N_T(t,A_1,A_2,l,o) \quad , \quad A_1 \geq t \quad (8)$$

3. Results

In this paper a part of the model for calculation of number of stochastic somatic health effects is presented. Complete model is a part of the HEALTH module of RODOS, a new European decision support system for nuclear emergencies. For the purpose of the short analysis presented in this paper, one part of the model, which refer to those alive at the moment of the accident (LG part of the observed population) is presented only. As an example, the inhalation of a long-lived nuclide (just as the DU) is observed, knowing that it leads to exposure till the end of an individual's life. On the other hand, due to the possible resuspension of DU in the air, the potential initial exposure by inhalation extends a long period of time. From these reasons, in this paper are presented stochastic health effects in the following generations also, (curves with up-triangle symbols on the Figures), although the FG model itself is not presented in this paper.

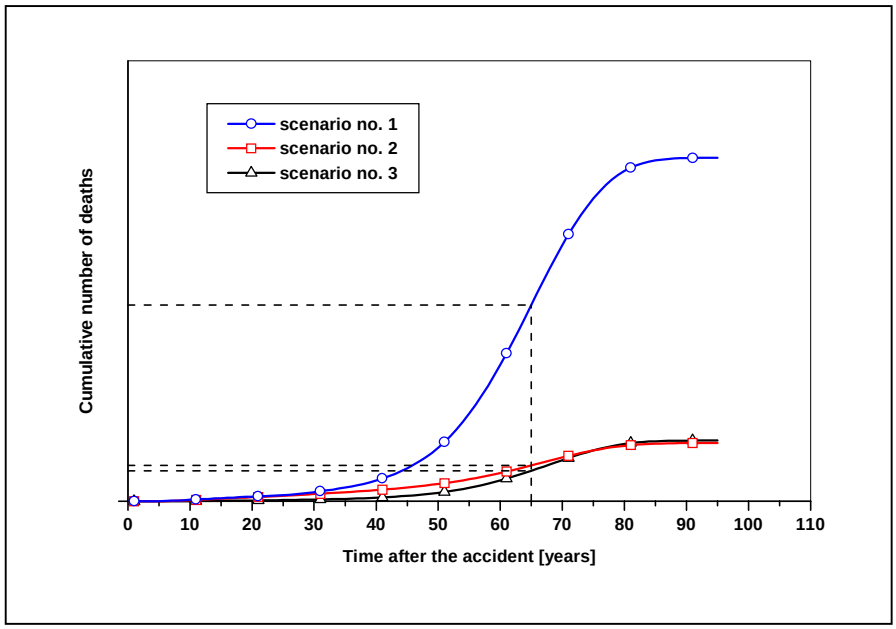

Figure 2. Cumulative number of deaths vs. time after the accident.

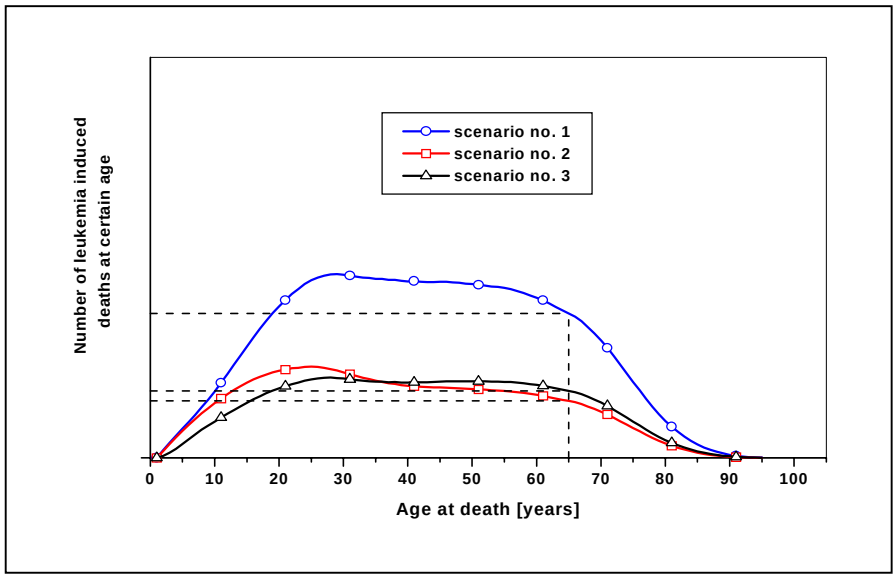

Figure 3. Age distribution of leukemia induced deaths for various scenarios.

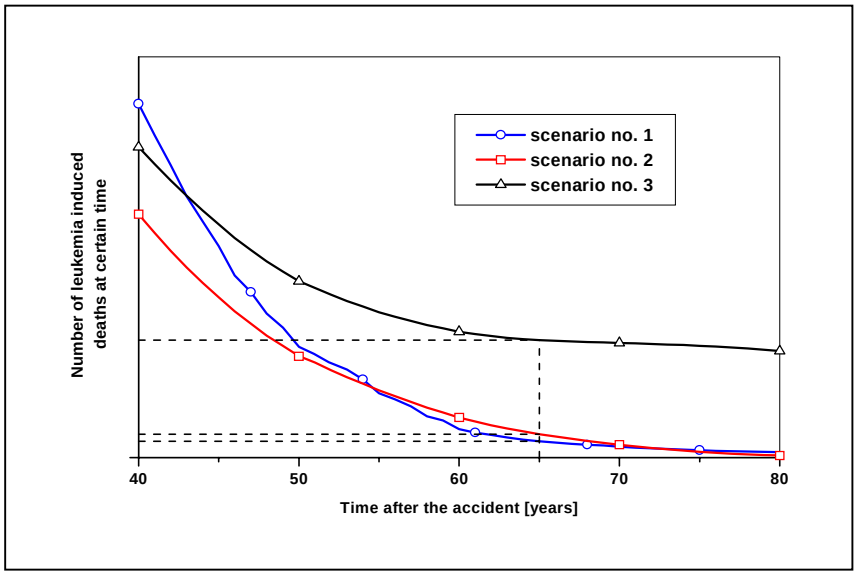

Figure 4. Time distribution of deaths after the accident.

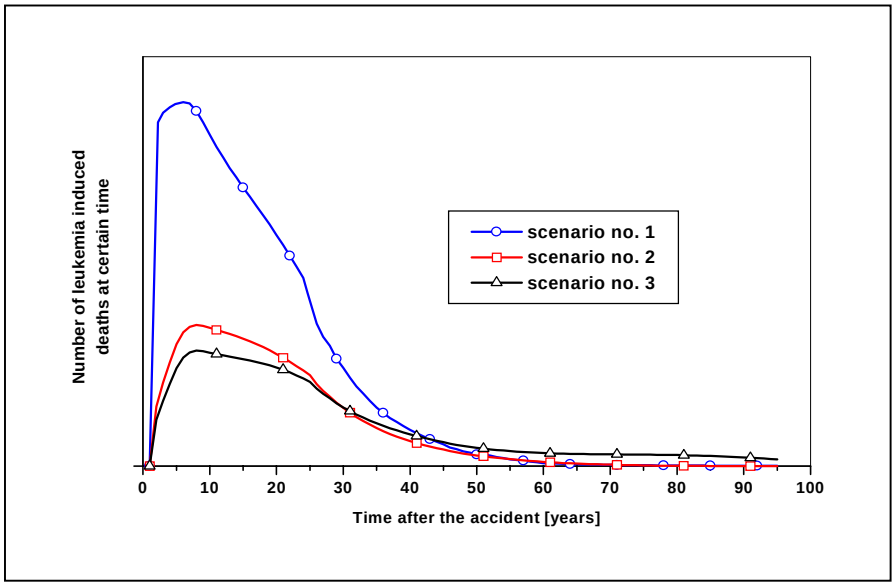

Figure 5. The same as Figure 3 (enlarged)

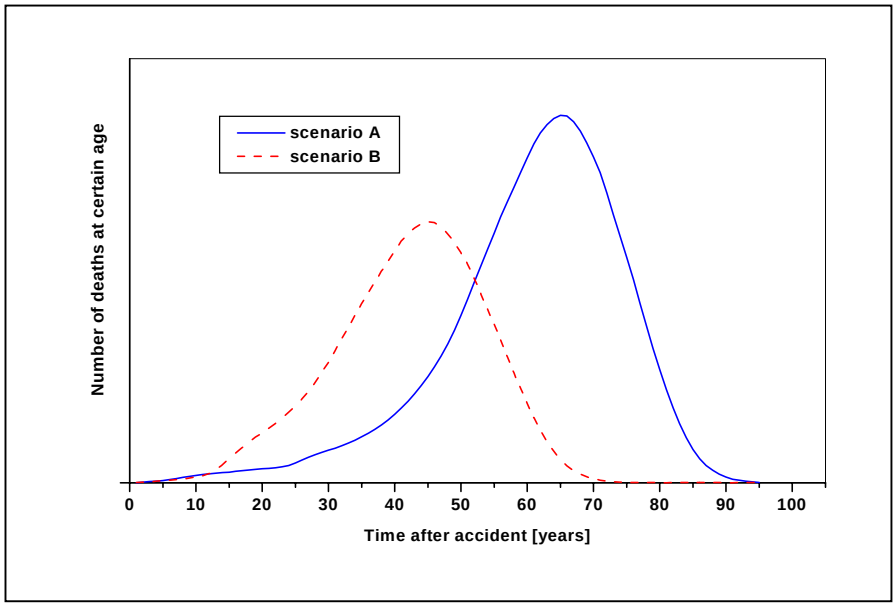

Figure 6. Influence of latency period on the time distribution of deaths.

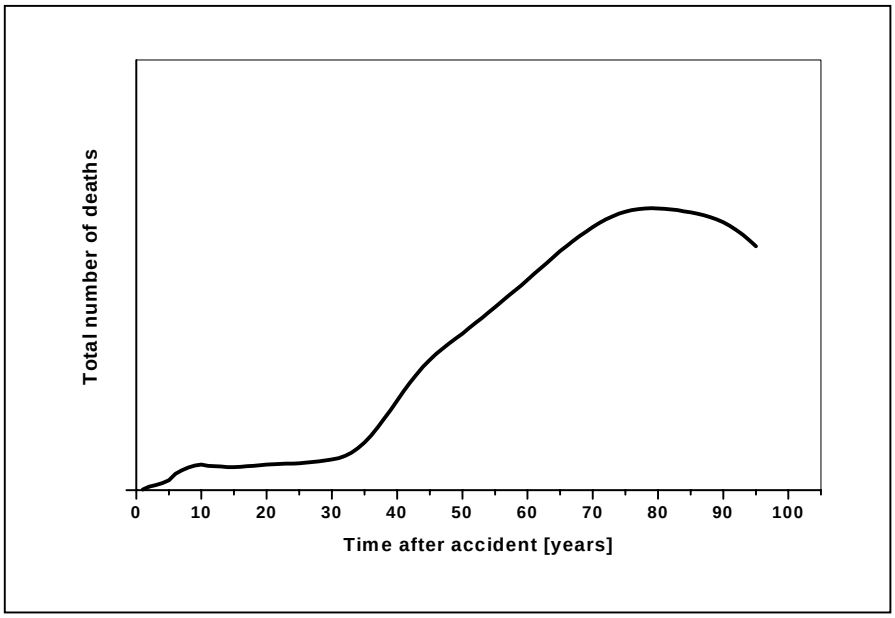

Figure 7. Time distribution of total number of deaths (an artificial case)

The purpose of this paper is to demonstrate some of features of the HEALTH module of RODOS. In order to show the comprehensiveness of possible output's analyses, three different levels of intake were chosen, to demonstrate the number of leukemia caused deaths as a function of time and age at death. Figure 2 shows cumulative number of leukemia's deaths caused by inhalation of a certain amount of depleted uranium (DU). Due to the lack of data, the model was running using artificial data sets, therefore no any conclusions are possible regarding the obtained values. Figures 3 and 4 show differential values. Figure 3 presents the age structure of the leukemia's induced deaths, which are expected in a certain time interval (here just equal to the maximal life expectancy in the observed population, which is set to 95 years). Scenarios number 1 and 2 on Figure 2 represent two different intakes, referring to the LG part of population only. Scenario number 3 refers a smaller intake, but including the following generations also. Figure 4 presents the number of deaths within each consecutive year after the accident (or the beginning of exposure), regardless of the age at death. It should be noted that the age at death interval in the program might be set to one-year interval also. However, in that case numerical experiments have shown rather bad statistics, therefore a larger population, or a broader age at death interval is needed. In the numerical experiments presented in this paper, the maximal age at death interval is used.

In order to give the meanings of the presented curves, and to show as well how differs the number of cases from one case to the another, the age 65 is fixed in all figures. This value on Figure 2 gives the number of individuals, which died up to 65 years after the accident. Three different scenarios obviously give rather different values. On Figure 3, the ordinate values which correspond to 65 year fixed on abscise, give the number of individuals, which will die from leukemia at the age of 65, within the first 95 years after the accident. Figure 5 is given as an enlargement of the corresponding part of Figure 4. The values, which correspond to abscise value of 65 years on Figure 5, give the number of people, which are expecting to die from leukemia exactly within the 65^{th} year after the beginning of the exposure. From Figure 4 it is clear that mostly the leukemia cases occur within the first 20 years, because of the small latency period (between 2 and 5 years). On the other side, if the following generations (FG) are considered also, even after the long period of time there are still observable leukemia deaths (scenario number 3 on Figures 4 and 5).

The latency period influence to the time distribution of deaths, within the LG part of population, is shown on Figure 6. Scenario A from Figure 6 corresponds to the effect with a smaller latency period than that considered by the scenario B. Finally, if the all effects are included, and the whole population is considered, time distribution of deaths has a shape like that presented on Figure 7. At the end of the observed period there is still non-trivial number of cases, which stem from the FG part of population. The curve from Figure 7, which includes all exposure pathways (external and internal), is given as an example only, to demonstrate the HEALTH outputs only.

4. Conclusions

Assessments of stochastic health effects, caused by the accidental exposure to radioactive material in the environment, need an extensive and comprehensive investigation. Based on the formerly developed dose risk coefficients, a new model for assessment of number of stochastic health effects is developed, which supports finding time and age at death distribution of the radiation induced cancers. This model is a part of the RODOS, a new

European computer decision support system for nuclear emergencies. One of possible use of this program could be the assessment of the number of leukemia, which are expected to develop due to the military use of depleted uranium (DU) in this region. The employment of such kind of analyses might be an important tool within *pro* and *contra* public discussions about possible consequences. Collection of field data is one of the crucial steps in such analysis, and if it is properly done, a small effort remains to fit it to the format necessary to run the program. Therefore, as a final conclusion, one should emphasize the need of an international project which should prepare and organize such calculations.

5. References

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