Application of Health Effect Model of NUREG/CR-4214 to the Japanese Population and Comparison with a Latest Model

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Radiation-induced cancer is one of the key issues in a consequence analysis on a Nuclear Power Plant accident. U. S. Nuclear Regulatory Commission (USNRC) developed the estimation model of those risks for the use of accident consequence analysis in 1980s and 1990s. This model is still used as a leading model in this field. In this paper, we aimed to explore the differences between the results of risk prediction from the model of USNRC and those from a latest model. To achieve this aim, radiation-induced cancer risks were projected based on the Japanese population statistics using the models developed by USNRC and the latest model, which was developed by U. S. Environmental Protection Agency (USEPA). As compared to the model of USEPA, the lifetime attributable risks of all cancers projected by the model of USNRC were about 30% higher for male and about 35% lower for female in both morbidity and mortality. When the sex-averaged values were compared between them, the difference is within 10%.

KEY WORDS: accident consequence analysis, health effect model, radiation-induced cancer, NUREG/CR-4214, Japanese population.

I. INTRODUCTION

Health effects due to radiation exposure are one of the key issues in an analysis on consequences due to accidental releases from a nuclear power plant. Radiation-induced cancer is the major health effect, and quantifying those risks is necessary to manage low-dose and low-dose rate exposure situation such as post-accident situations. In 1980s and 1990s, U. S. Nuclear Regulatory Commission (USNRC) developed risk estimation model (hereafter called the NUREG model)1–4) for radiation-induced cancers based on the life span study (LSS) of the Japanese atomic bomb survivors.5) This model is still used as a leading model for projecting radiation-induced cancer risks in the field of accident consequence analysis.6–8) However, because the models are revised continuously with updates of data from the LSS,9–11) a conclusive model has not been established yet. Assessors therefore have to understand the limitations and features of the various estimation models to correctly interpret their results.

One of the aims of this paper is to explore the difference between the results of risk projection from the NUREG model and from a latest model. To achieve this aim, we perform comparative consideration with the NUREG model and the latest estimation model from the U. S. Environmental Protection Agency (USEPA)12) (hereafter called the EPA2011 model). The EPA2011 model was developed in the light of the latest data obtained from the follow-up of the Japanese atomic bombing survivors9–11) and the view of further developing radiation-induced cancer studies.13–18) We apply these two models to the projections of radiation-induced cancer risks for the Japanese population and compare those results. In our comparative considerations, we intend to provide not only the differences between the results of risk projections from the two models but also the features of the Japanese population that figure into the projections of radiation-induced cancer risks.

In addition, we also aim to provide the information about the age-specific risks for the Japanese population. The method of expressing the radiation-induced cancer risks is one of the key elements to manage adequately the post-accident situation taking into account equity among the differences of risks in individuals. Most commonly, the risk of radiation-induced cancer is represented as an integrated value for the period after the exposures occurred, which is called lifetime attributable risk (LAR). LAR is published as nominal values averaged by age and sex in a target population.17, 18) The nominal risk coefficient is a useful tool to optimize planning for exposure situations in accordance with a risk constraint. However, the LAR is not appropriate for dealing with individual- and population-specific issues. From the experiences of post-accident management, when radiation exposures occur, public concern is focused on the protection of children.19) If the information about the differences between the nominal and

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age-specific risks were available, it would be helpful for not only decision-makers trying to optimize the levels of exposure doses but also parents looking to foster a sense of safety and to manage the protection of children.

II METHODS

1. Excess relative and excess absolute risks

The risks of each cancer site are projected based on either excess relative risk (ERR), wherein the excess risk is expressed relative to background risks due to naturally generated cancers, or excess absolute risk (EAR), wherein the excess risk is expressed as the difference in the total risk imposed on people who received radiation exposures. Risk projections for all radiation-induced cancers are obtained by summing the projections of ERR, EAR, or the weighted mean of both for specific cancer sites. Excess risk induced by radiation exposures, \( M \), is expressed by \( \text{ERR} \) and \( \text{EAR} \) as follows:

\[
\text{ERR model} \quad M(s,e,a,D) = \text{ERR}(s,e,a,D) \cdot \lambda_0(s,a), \quad (1)
\]

\[
\text{EAR model} \quad M(s,e,a,D) = \text{EAR}(s,e,a,D), \quad (2)
\]

where \( \lambda_0 \) denotes the background cancer risk at zero dose, \( s \) is the index for sex, \( e \) is age-at-exposure (y), \( a \) is attained age (y), and \( D \) is absorbed dose from gamma-ray exposure (Gy). The projections of risks are made with consideration about the latency and expression period for cancers of each site.

2. NUREG model

EAR and ERR for each cancer site used in the NUREG model are summarized in Table 1. Latency periods for each site are also shown in this table. Cancer sites in the NUREG model are lung, bone, skin, breast, thyroid, leukemia, gastrointestinal system, and residual sites. The gastrointestinal system here includes stomach, colon, esophagus, and liver. In addition, the morbidity of benign thyroid nodules is projected. For all cancer sites except bone and leukemia, the expression period for the risks is the lifetime after exposure. The expression period for bone cancer and leukemia is 25 years after exposure. Lung and breast cancers are estimated with a minimum age at induction of 40 years and 30 years, respectively. In extrapolating cancer risks observed in groups exposed at a high dose rate to low linear energy transfer (LET) radiation, dose and dose rate effectiveness factor (DDREF) is used. In the NUREG model, DDREF of 1 or 2 is used for each cancer site, as shown in Table 1.

3. EPA2011 model

In the EPA2011 model, cancer sites are classified into lung, bone, skin, breast, thyroid, leukemia, stomach, colon, liver, ovary, bladder and prostate, uterus, and residual sites. While the NUREG model was developed based on mortality, the EPA2011 model was based on morbidity from the LSS. Mortality is projected with corrections by fatality. The results of risk projection for each cancer site are given as a weighted arithmetic mean of EAR and ERR to risk transfer among different populations. DDREF for solid cancer is 2. The risk of leukemia is expressed as a linear-quadratic function of radiation dose. Latency periods for solid cancers other than leukemia are 5 years. A period of 2 years is adopted for the latency period of leukemia. For solid cancer sites other than kidney, thyroid, skin, bone, and leukemia, EAR and ERR are functions of sex, age-at-exposure, and attained age:

\[
\text{EAR}(s,e,a,D) \quad \text{or} \quad \text{ERR}(s,e,a,D) = \beta \cdot D \cdot \exp(\gamma \cdot e) \left( \frac{a}{60} \right) \quad (3)
\]
where

\[
\epsilon = \frac{\min(e,30) - 30}{10}
\]  

(4)

and \( \beta \) is excess risk per Gy at age-at-exposure of 30 years and attained age of 60 years for sex \( s \); \( \gamma \) implies that the radiation-induced risk of cancer at age \( e \) falls by about 25% for every decade as age-at-exposure increases up to the age of 30 years; and \( \eta \) implies that the excess risk is almost 20% smaller at attained age of 70 years than at 60 years. Parameter values of \( \beta, \gamma, \) and \( \eta \) are shown in Table 2.

For kidney cancer, excess risks are obtained using the product of excess risks for residual sites and an adjustment factor equal to the ratio of the age-specific kidney cancer background rates divided by the rates for the residual sites. The ERR weighting factor is 0.6 and 0.8 for males and females, respectively. Thyroid cancer incidence risk is derived from the model recommended by the U. S. National Council on Radiation Protection and Measurements. \(^{20}\) The model is expressed as follows:

\[
ERR(s,e,a,D) = \beta \cdot D \cdot A(e) \cdot T(t)
\]  

(5)

where \( \beta \) is excess risk, \( A(e) \) is a multiplicative factor for age-at-exposure, and \( T(t) \) is a multiplicative factor for time-since-exposure. The values of these factors are given in Table 3.

For breast cancer, morbidity is projected by only EAR in the same manner as BEIR VII. \(^{19}\) The EAR for breast cancer is given in the following form:

\[
EAR(s,e,a,D) = \beta \cdot D \cdot \exp \left( \frac{\gamma (e - 25)}{10} \right) \cdot \left( \frac{a}{50} \right) \gamma
\]  

(6)

The parameter values of \( \beta, \gamma, \) and \( \eta \) are shown in Table 2. The mortality of radiation-induced breast cancer is expressed by a site-specific model. If \( da \) represents a small age increment, the probability of a radiation-induced cancer between ages \( a_i \) and \( a_i + da \) is given as follows:

\[
f_{DA}(a_i) \cdot da = M_i(s,e,a,D) \cdot S \cdot \frac{a_i}{S(e)} \cdot da
\]  

(7)

where \( M_i(s,e,a,D) \) is the EAR of breast cancer incidence at the attained age \( a_i \) from age-at-exposure at \( e \). \( S(t) \) is the probability of surviving to age \( t \). In order for radiation-induced breast cancer to result in death at age \( a_M \), the patient has to survive the interval \( (a_M - a_i) \) and then die due to the radiation-induced cancer at age \( a_M \). It is assumed that the relative survival rate depends only on the length of the time interval and the age of diagnosis. The probability of survival with breast cancer for the interval \( (a_M - a_i) \) is \([S(a_M)/S(a_i)] \cdot R(t,a_i)\). Here, \( t \) is the period between diagnosis and death, which equals \( a_M - a_i \). Assuming the breast cancer mortality rate \( h \), the probability of radiation-induced breast cancer death between ages \( a_M \) and \( a_M + da \) can be given as follows:

\[
f_{DA}(a_M) \cdot da = \left( \int_{t=0}^{t=a_M-a_i} \cdot h(a) \cdot M(s,e,a,D) \cdot S \cdot \frac{a_M}{S(e)} \cdot \frac{a_M}{S(a)} \cdot R(t,a) \cdot da \right) \cdot da
\]  

(8)

The value of \( h \) is derived from relative survival rates, \( R(t,a) \), in accordance with the following relationship:

\[
R(t,a) = \exp[-t \cdot h(a)]
\]  

(9)

For the projections of breast cancer mortality, the EPA2011 model used five-year relative survival rates. The EPA2011 model uses both EAR and ERR to project the mortality due to radiation-induced leukemia. The estimation models are given in a linear-quadratic function of exposure dose \( D \), age-at-exposure \( e \), and time-since-exposure \( t \), as follows:

\[
2 \leq t < 5
\]

\[
EAR(s,e,a,D) = ERR(s,e,a,D) = 0
\]  

(10)

\[
2 < t \leq 5
\]

\[
EAR(s,e,a,D) = EAR(s,e, a + 5,D)
\]  

(11)

| Table 2 | Parameters for the risk estimation model developed by USEPA. |
|----------|---------------------------------------------------------------|
| Weighting factor | ERR | EAR |
| Lung | 0.3 | 0.32 | 1.4 | -0.3 | -1.4 | 2.3 | 3.4 | -0.41 | 5.2 |
| Breast | Not used | 9.9 | -0.51 | 3.5 | 1.1 |
| Thyroid | 1 | See text | Not used |
| Leukemia | 0.7 | 1.1 | 1.2 | -0.4 | None | 1.62 | 0.93 | 0.29 | None |
| Stomach | 0.7 | 0.21 | 0.48 | -0.3 | -1.4 | 4.9 | 4.9 | -0.41 | 2.8 |
| Colon | 0.7 | 0.63 | 0.43 | -0.3 | -1.4 | 3.2 | 1.6 | -0.41 | 2.8 |
| Liver | 0.7 | 0.32 | 0.32 | -0.3 | -1.4 | 2.2 | 1 | -0.41 | 4.1 |
| Ovary | 0.8 | 0.38 | -0.3 | -1.4 | 0.7 | 0.41 | 2.8 |
| Bladder | 0.7 | 0.5 | 1.65 | -0.3 | -1.4 | 1.2 | 0.75 | -0.41 | 6 |
| Prostate | 0.7 | 0.12 | -0.3 | -1.4 | 0.11 | -0.41 | 2.8 |
| Uterus | 0.7 | 0.055 | -0.3 | -1.4 | 1.2 | -0.41 | 2.8 |
| Other solid | 0.7 | 0.27 | 0.45 | -0.3 | -2.8 | 6.2 | 4.8 | -0.41 | 2.8 |

(1) For attained age < 50, (2) For attained age ≥ 50.
Table 3 Parameters for thyroid risk estimation model adopted by the EPA2011 model.

| Multiplicative factor | 10.7 |
|-----------------------|------|
| $B$ (Gy$^{-1}$)       |      |
| < 5                   | 1.0  |
| 5–9                   | 0.6  |
| 10–14                 | 0.2  |
| > 15                  | 0.2 · exp[−0.083(e−15)] |
| $A(e)$                |      |
| < 5                   | 0    |
| 5–14                  | 1.15 |
| 15–19                 | 1.9  |
| 20–24                 | 1.2  |
| 25–29                 | 1.6  |
| ≥ 30                  | 0.47 |

$$ERR(e,a,D) = EAR(e,a,D) + 5D \cdot \frac{1}{25}$$

for $t > 5$

$$EAR(e,a,D) = \beta \cdot D \cdot (1 + \theta \cdot D) \cdot \exp[\gamma \cdot e + \delta \cdot \log\left(\frac{t}{25}\right)]$$

The age dependence of LAR for radiation-induced cancer morbidity and mortality is shown in Fig. 1 and Fig. 2 as a function of age-at-exposure. These figures indicate sex-averaged LARs for the sites that have site-specific estimation models in the NUREG model and that contribute largely to the LAR of all cancers.

The projections were performed using the HEINPUT-GUI code, which is developed by the Japan Atomic Energy Agency (JAEA). The NUREG and EPA2011 models are incorporated into HEINPUT-GUI, and its validation of projections was confirmed on the basis of the test calculation for the U. S. population.

### 1. Comparison of Age Dependence of LAR

The age dependence of LAR for radiation-induced cancer morbidity and mortality are shown in Fig. 1 and Fig. 2 as a function of age-at-exposure. These figures indicate sex-averaged LARs for the sites that have site-specific estimation models in the NUREG model and that contribute largely to the LAR of all cancers.

As for leukemia, significant differences were not observed among the results of the NUREG and EPA2011 models. In gastrointestinal system, as mentioned previously, there are differences of cancer site classification among the models. The NUREG model includes esophagus, stomach, colon, and liver; for the EPA2011 model, however, it includes only stomach, colon, and liver. Thus, the results of those cannot be directly compared. However, below the age of about 20 years, the results of the EPA2011 model have a tendency to be lower than those of the NUREG model.

For breast cancer, morbidity and mortality in the EPA2011 model have tendencies to be higher than those in the NUREG model. The main contributor to this tendency is the difference between the models, which means risk coefficients and a minimum induction age. But, for mortality, it was also caused by the new estimation method of the EPA2011 model based on the five-year relative survival rate. In the NUREG model, the fatality rates can be approximated by calculating the ratios of current mortality to current morbidity of breast cancer. However, because the time between breast cancer diagnosis and death is relatively long, diagnostic accuracy and survival rate can rapidly change with developing treatment techniques and social frameworks for the diagnosis. In fact, breast cancer incident rates in Japan are considerably higher than those in the past. Thus, the number of fatalities estimated by the background mortality and morbidity are smaller than those of

Breast cancer morbidity reported in 2010 was 1.5 times higher than in 2003 in Japan.
(1) Morbidity for leukemia is not given by the NUREG model.
(2) Morbidity for all cancer sites were projected as the summation except for benign thyroid nodules.

Fig. 1  Sex-averaged LAR for morbidity by age-at-exposure for the Japanese population.
Fig. 2  Sex-averaged LAR for mortality by age-at-exposure for the Japanese population.

(1) Scale of vertical axis of mortality for G.I. system, lung, breast, residual sites, and all cancer sites are different with those of morbidity.

(2) Scale of vertical axis of mortality for thyroid cancer is one order lower than that of morbidity.
the actual value, which results in the different tendency between the mortality of the EPA2011 model and the NUREG model shown in Fig. 1 and Fig. 2.

For Lung cancer, morbidity and mortality in the NUREG model were lower than those from the EPA2011 model. In this site, there are differences between the models in terms of not only risk coefficients but also whether or not to use a minimum age at induction. For thyroid cancer, repeated increase and decrease were shown in the results of the EPA2011 model. These tendencies are attributed to the ERR risk coefficients, which are shown in Table 3, and the increments of background risks with attained age. In addition, the LARs from the EPA2011 model are highly dependent on the age-at-exposure.

For residual sites, and all cancer sites, there is a slight difference in both mortality and morbidity, which are attributed to the difference of the models. The risks to residual and all sites projected by the NUREG model, which adopted a step function, tended to be higher than those of the EPA2011 model, which adopted a continuous function.

2. Comparison of Nominal Risks for the Population Groups

Age-averaged LARs for radiation-induced cancer morbidity and mortality for the Japanese population are shown in Table 4 and Table 5. Age-averaged LARs for the U. S. population from the EPA report (2) are also shown in these tables. As compared to the EPA2011 model, expect for the contribution of benign thyroid nodules, the LARs of all cancers projected by the NUREG model were about 30% higher for male, and about 35% lower for female in both morbidity and mortality. When the sex-averaged values were compared among them, the difference is within 10%. For male, the dominant contributors of difference were gastrointestinal system and residual sites. The difference in the LARs for female is attributed to breast and lung cancers mainly. These differences between the LARs projected by the NUREG model and the EPA2011 model could be explained by the difference between models such as already mentioned in section III. 1.

In addition, we can see some features in the results of skin cancer risks shown in Table 4. The morbidity of skin from the NUREG model was much higher than those projected by the EPA2011 model. The NUREG model was based on the conservative assumptions (29) that (i) 1/6 of radiation-induced skin cancers would be squamous cell carcinomas (SCC) and remainder basal cell carcinomas (BCC), and (ii) essentially all of the BCC would be curable, whereas about 1% of SCC would be fatal. On the other hand, because the EPA2011 model adopted an assumption that only BCC are induced by low-LET radiation exposure, the projections are lower than those in the NUREG model.

From the aspects of the difference between the Japanese population and the U.S. population, the projections of morbidity and mortality of stomach and liver for the Japanese population are much higher than those for the U. S. population. The differences are caused by the differences of background morbidity and mortality. For stomach and liver cancers, background morbidity and mortality are relatively higher in the Japanese population. Thus ERR of stomach and

| Cancer site               | Male       | Female     | Male       | Female     | Male       | Female     |
|---------------------------|------------|------------|------------|------------|------------|------------|
| Leukemia                  | 750        | 538        | 920        | 690        |
| Bone                      | 24         | 23         | 24         | 23         |
| Breast                    | 1,799      | –          | 2,752      | –          | 2890       |
| Lung                      | 1,542      | 633        | 1,521      | 3,064      | 1,300      | 3,080      |
| Thyroid                   | 601        | 975        | 214        | 506        | 220        | 650        |
| Gastrointestinal system   | 6,101      | 3,636      | –          | –          | –          | –          |
| Stomach                   | 1,461      | 1,586      | 620        | 750        |
| Colon                     | 1,695      | 974        | 1,460      | 920        |
| Bladder                   | 663        | 622        | 970        | 920        |
| Kidney                    | 256        | 176        | 240        | 220        |
| Liver                     | 806        | 436        | 400        | 210        |
| Skin                      | 220        | 235        | 0.5        | 0.3        | 0.5        | 0.3        |
| Ovary                     | –          | –          | 265        | –          | 330        |
| Esophagus                 | –          | –          | –          | –          | –          | –          |
| Prostate                  | –          | 396        | –          | 890        | –          | –          |
| Uterus                    | –          | 0          | 232        | 0          | 230        |
| Residual                  | 3,958      | 1,693      | 2,057      | 2,067      | 2,510      | 2,590      |
| All cancers               | 12,420     | 8,970      | 9,842      | 13,236     | 9,550      | 13,500     |

* Except for the results of projecting benign thyroid nodules.
liver for the Japanese population is several times higher than those for the U.S. population. To adjust the variability of risk estimates among the different populations, latest models\textsuperscript{12–14, 18)} implement risk transfer systems with a weighting factor for ERR and EAR. Although the weighted arithmetic means are adopted to generate the estimates in the EPA2011 model\textsuperscript{12)}, the appropriate way to transfer risk estimates between populations is a general issue yet. If the weighted geometric means were adopted in the EPA2011 model, the projections of stomach for the U.S. population would be about one-half comparing with those gained by the arithmetic weighted approach.\textsuperscript{12)} Therefore, the method of risk transfer leads to a large difference in the results of projections. To improve the accuracy of risk projection, further considerations will be needed about the weighting approach between ERR and EAR.

### 3. Difference of LAR at Each Age to Sex- and Age-averaged Value

Figure 3 shows the ratios of the mortal LAR at age 0, 20, and 60 years to the Sex- and age-averaged value for the Japanese population using the EPA2011 model. For each cancer site, the extent of the ratios obtained from the NUREG model is within the extent shown in Fig. 3. In addition, the ratios had about the same extent between mortality and

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**Table 5** Results of projecting cancer mortality for the Japanese population.

| Cancer site          | LAR ($\times 10^{-4}$ Gy$^{-1}$) | NUREG Japan Male | NUREG Japan Female | EPA2011 Japan Male | EPA2011 Japan Female | U.S. $^{12)}$
|----------------------|---------------------------------|------------------|--------------------|---------------------|----------------------|----------------------
| Leukemia             | 472                             | 483              | 425                | 286                 | 650                  | 500                  |
| Bone                 | 6                               | 6                | 9                  | 8                   | 9                    | 8                    |
| Breast               | -                               | 329              | -                  | 1,232               | -                    | 950                  |
| Lung                 | 1,122                           | 415              | 1,104              | 2,105               | 1,200                | 2,550                |
| Thyroid              | 60                              | 97               | 11                 | 25                  | 11                   | 32                   |
| Gastrointestinal system | 3,018                     | 2,098            | -                  | -                   | -                    | -                    |
| Stomach              | -                               | -                | 601                | 760                 | 320                  | 410                  |
| Colon                | -                               | -                | 649                | 432                 | 670                  | 410                  |
| Bladder              | -                               | -                | 253                | 325                 | 200                  | 260                  |
| Kidney               | -                               | -                | 90                 | 66                  | 83                   | 70                   |
| Liver                | -                               | -                | 585                | 321                 | 310                  | 180                  |
| Skin                 | -                               | -                | <0.1               | <0.1                | <0.1                 | <0.1                 |
| Ovary                | -                               | -                | 137                | -                   | -                    | -                    |
| Esophagus            | -                               | -                | -                  | -                   | -                    | -                    |
| Prostate             | -                               | -                | 81                 | -                   | 140                  | -                    |
| Uterus               | -                               | -                | 98                 | -                   | 64                   | -                    |
| Residual             | 1,653                           | 974              | 1,074              | 1,171               | 1,090                | 1,250                |
| All cancers          | 6,331                           | 4,404            | 4,880              | 6,964               | 4,690                | 6,890                |

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**Fig. 3** Ratio of LAR at each age to Sex- and age-averaged value for the Japanese population projected by the EPA2011 model.
morbidity. Therefore, here, we discussed only insight derived from mortality in the EPA2011 model. As shown in Fig. 1 and Fig. 2, since the LARs generally decreases with age-at-exposure, the ratios of LAR to the averaged nominal risk also decrease with age-at-exposure. The ratios at age-at-exposure of 0 years for thyroid and breast cancers, which are the highest value of all sites, are about 8 and 6, respectively. For leukemia, bone, and skin cancers, the ratios of LAR between 0 years and the averaged are lower than those of other sites. Except for these sites, the ratio at 0 years is about 2.8 on average. On the other hand, the ratios of mortality at 60 years and the averaged values were lower than 1 (excepting leukemia). In particular, the ratio for thyroid and breast is much lower than those at other ages.

For all cancer sites, the ratios of LAR at age-at-exposure of 0, 20, and 60 years is 3.1, 1.3, and 0.7, respectively. These results for the Japanese population are consistent with a general insight suggested by the UNSCEAR report, which stated that lifetime cancer risk for children might be approximately 2–3 times higher than the averaged value in all ages. This tendency is explained by the longer life expectancy and the higher sensitivities of children as compared with those of adults. However, this consideration is made on the basis of the results of analysis on the current LSS data, which is derived as the extrapolation of high-LET radiation exposures due to radiation from atomic bombs. The LSS is an ongoing program, and the surveys of the people who were exposed in their childhood are not yet concluded. To improve the models and to reduce uncertainties in lifetime risk estimates for children, continuous research is needed taking into account the studies not only on the effects from high-LET radiations such as the LSS but also on the effects from low-LET radiations such as high natural background exposures.

IV CONCLUSIONS

Radiation-induced cancer risks were projected based on the Japanese population statistics using the NUREG and EPA2011 models. As compared to the EPA2011 model, the LARs of all cancer sites projected by the NUREG model were about 30% higher for male, and about 35% lower for female in morbidity. Therefore, here, we discussed only insight derived from and mortality. When the sex-averaged values of adults. However, this consideration is made on the basis of the results of analysis on the current LSS data, which is derived as the extrapolation of high-LET radiation exposures due to radiation from atomic bombs. The LSS is an ongoing program, and the surveys of the people who were exposed in their childhood are not yet concluded. To improve the models and to reduce uncertainties in lifetime risk estimates for children, continuous research is needed taking into account the studies not only on the effects from high-LET radiations such as the LSS but also on the effects from low-LET radiations such as high natural background exposures.

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