# Examination of Conservatism in Early/Latent Fatality Estimation in Level 3 PRA

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#### 1. Introduction

With the recent Fukushima accidents, concern about nuclear reactor accidents and their consequences is highly visible in recent public dialogues surrounding the use of nuclear power. Consequences of a nuclear reactor accident include human health effects and damage to the environment and are characterized by a level 3 PRA. Thus, results from a level 3 PRA will become very important for public communication of risk from nuclear reactor accidents.

Due to the computational model driven-nature of the work, there exist various sources of uncertainty in level 3 PRA. They are related with source release, environmental transport and deposition, human behavior involved in dosimetry, health effect and risk assessment. For instance, a total of 376 parameters have been considered in Probabilistic Accident Consequence Uncertainty Assessment Using COSYMA [1] and the details on the number of parameters in each analysis are listed in Table 1.

Table	1.	Example	of	the	number	of	uncertainty	parameters
related	l wi	ith level 3	PR	A []	1]			

Module	Number of Parameters Considered	Number of Parameters Analyzed
Atmospheric Dispersion and	28	24
Deposition Food Chain	162	35
Internal and External Dosimetry	159	100
Health Effects	27	27
Overall Analysis	376	186

In 2012, the report of NPP accident consequence simulation was distributed by the Korean Federation for Environmental Movement (KFEM) [2]. They insisted that Kori Nuclear Power Plant (NPP) accident would lead to 48,000 early fatalities and 850,000 cancer fatalities in Busan and Hanbit NPP accident would lead to 550,000 cancer fatalities in Seoul. This report exemplifies the misuse of collective dose, that is effective dose multiplied by population and time. Even though very low effective dose is considered, collective dose could give over-conservative estimate when high population and long time period is multiplied. International Commission on Radiological Protection (ICRP) forewarned about the misuse of collective dose, in their ICRP Publication 103 [3], such as applying it to simplified calculation of fatality and risk.

As part of investigation of conservatism in early and latent fatality estimation, the existing methods of early and latent fatality calculation was reviewed and the results from the use of the existing methodology were examined in this study.

# 2. Investigation of Early/Latent Fatality Estimation Method

Early fatality is an acute effect of radiation and is mainly caused by impaired functioning of red marrow (hematopoietic syndrome), lung (pulmonary syndrome), and gastrointestinal tract (gastrointestinal syndrome). Latent fatality is caused by cancer such as leukemia and solid cancers induced by radiation exposure.

Evaluation of early and latent fatality can be performed by using radiation risk models. In this study, reference risk models were chosen based on the stateof-the-art from the literature and were compared to the method used in the MELCOR Accident Consequence Code System 2 (MACCS2) which is widely used for regulatory analysis of level 3 PRA.

#### 2.1 Early Fatality Estimation

### 2.1.1. Reference Method of Early Fatality Estimation

Assessments of lethal dose for early fatality is introduced in the report of Pacific Northwest National Lab (PNNL) [4] and NUREG/CR-6545 report [5]. These reports describe a relationship between total dose/dose rate and fatality rate for hematopoietic syndrome along with the examination of Lethal Dose 50  $(LD_{50})$ value for hematopoietic syndrome, gastrointestinal syndrome, and pulmonary syndrome. The dose/dose rate fatality response introduced in PNNL report and the LD<sub>50</sub> values suggested in both reports are presented in Figure 1 and Table 2, respectively.



Fig. 1. Lethal dose response related to hematopoietic syndrome for 0.2 Gy/hour dose rate without medical treatment [4]

	LD <sub>50</sub> [Gy]					
	5% CI	50% CI	95% CI			
Hematopoietic Syndrome	2.40	4.50	7.00			
Pulmonary Syndrome	21.4	38.1	332			
Gastrointestinal Syndrome	8.79	18.67	38.04			

Table 2.  $LD_{50}$  dose of each early fatality for 0.2 Gy/hour dose rate without medical treatment  $\left[4,\,5\right]$ 

2.1.2. MACCS2 Method of Early Fatality Estimation

Early fatality risk calculation by for MACCS2 is based on the use of the Weibull distribution with a threshold dose as described below [4, 6]. Parameters of the model for risk calculation are listed in Table 3.

$$\begin{aligned} \mathbf{r} &= 1 - \exp(\mathbf{H}), \mathbf{H} = \sum_{i=1}^{n} H_i, H_i = 0.693 X_i^{\beta_i}, \\ X_i &= \begin{cases} \frac{D_{ext,i}}{D_{50,i,t}} + \left[\sum_{t=1}^{n} \frac{D_{inh}}{D_{50,i,t}}\right], \text{ for } \mathbf{D} \geq \mathbf{D}_{\mathrm{TH}} \\ 0, \text{ for } \mathbf{D} < \mathbf{D}_{\mathrm{TH}} \end{cases} \end{aligned}$$

where

 $\beta$  Shape parameter

X Normalized dose

- $D_{ext,i}$  External dose delivered to target i during the emergency phase of accident
- $D_{50,i,t}$  Dose to the organ that if delivered during the time period t would induce the health effect in half the exposed population

 $D_{inh,i,t}$  Dose delivered to target by materials that were inhaled during the emergency phase

*D<sub>TH</sub>* Threshold dose

Table 3. Parameters for early fatality estimation in MACCS2 code [6]

Type Of Early	β	D <sub>Th</sub> [Sv]	$LD_{50}  ext{ or } D_{50}  ext{ [Sv]}$ for time period end point [days]						
Fatality			1	7	10	30	200	365	
Hematopoietic Syndrome	5	1.5	3.8		7.6	15			
Pulmonary Syndrome	7	5	10		160		370	920	
Gastro- intestinal Syndrome	10	8	15	35					

By assuming that the radiation exposure was only by low Linear Energy Transfer (LET) radiation, the lethal dose response model follows the curve shown in Figure 2. Difference between the result from reference method and the result from the method used in MACCS2 code does not exceed confidence interval. Therefore, the method employed in MACCS2 is judged to be accurate.

The calculated values of  $LD_{10}$ ,  $LD_{50}$ , and  $LD_{90}$  for hematopoietic syndrome by using the method

introduced in MACCS2 code are 2.61 Sv, 3.80 Sv, and 4.83 Sv, respectively.



Fig. 2. Lethal dose response related to hematopoietic syndrome based on computational model result for 0.16 Sv/hr

### 2.2 Latent Fatality Estimation

# 2.2.1. Reference Method of Latent Fatality Estimation

Dose responses of latent fatal cancer risk for different kind of cancers are introduced in Biological Effect on Ionizing Radiation (BEIR) report and U.S. EPA report [8, 9]. As relative risk model, BEIR VII report describes cancer risk like below:

$$\lambda = \lambda_0 (1 + ERR)$$

where

 $\lambda_0$  Background Level Risk ERR Estimated Relative Risk

ERR for solid cancer

$$= \begin{cases} D\left(\frac{a}{60}\right)^{-2} , \text{ for female breast} \\ 0 , \text{ for male breast} \\ \beta_s D \exp[\gamma e^*] \left(\frac{a}{60}\right)^{\eta} , \text{ for other solid tumors} \\ 0.53D \exp[-0.083(e-30)] , \text{ for male thyroid} \\ 1.05D \exp[-0.083(e-30)] , \text{ for female tyroid} \end{cases}$$

ERR for leukemia

$$= \begin{cases} 0 & , for \ t \le 2 \\ ERR(t=5) * \frac{\lambda_0(s, e+5)}{\lambda_0(s, e+2)} & , for \ 2 < t \le 5 \\ \beta_s D(1+\theta D) \exp\left[\gamma\left(\frac{e^8}{10}\right) + \delta \log\left(\frac{t}{25}\right) + \Phi e^* \log\left(\frac{t}{25}\right)\right] & , for \ t > 5 \end{cases}$$

where

$$\beta_s = \frac{ERR}{Sv}$$
 at age 30 and attained age 60 by sex (95% CI)

 $\gamma$  Per-decade increase in age at exposure over the range 0 to 30 Years (95% CI)

 $\eta$  Exponent of attained age (95% CI)

 $e^*$  Exposed age factor,  $\begin{cases} e - 30, for \ e < 30\\ 0, for \ e \ge 30 \end{cases}$ 

e Exposed age

- $\theta$  Degree of curative parameter
- $\delta, \Phi$  Time after exposure parameter

*t* Time after exposure (year)

Current research on latent fatal cancer risk suggests linear dose response for solid tumor and linearquadratic dose response for leukemia.

#### 2.2.2. MACCS2 Method of Latent Fatality Estimation

Latent cancer risk is modeled by dose and exposure period in MACCS2. Cancer risk for target organ i  $(r_i)$  is expressed in MACCS2 as follows [6]:





Fig. 3. Dependence of cancer risks on dose implemented in MACCS2 [6]

Figure 3 is describing the latent cancer risk model in MACCS 2 and parameters for each cancer are presented in Table 4.

Table 4. Parameters for latent cancer risk model in MACCS2 [6]

Type of cancer	a (for fatal cancer)	b	с
Leukemia	3.70E-3	0.39	0.61
Bone Cancer	1.50E-4	0.39	0.61
Breast Cancer (Female)	1.70E-2	1	0
Lung Cancer	5.70E-3	0.39	0.61
Thyroid Cancer	7.20E-3	1	0
Gastrointestin al Cancer	2.50E-2	0.39	0.61
Other Cancers	1.30E-2	0.39	0.61

MACCS2 describes latent fatal cancer risk as linearquadratic dose response for all cancers except thyroid cancer and female breast cancer which follows linear dose response. Thus description of certain cancers in MACCS2 may not be truly conservative.

BEIR VII approach of latent fatal cancer estimation appeared to be more conservative than MACCS2

approach and comparison between two models is being investigated.

## **3. Examination of Conservatism in Early/Latent Fatality Estimation**

The method of estimating fatality in MACCS2 is described in the model description [6] as following:

After average individual risks have been estimated using the individual risk models, total cases of a specific health effect  $N_i$  are calculated in MACCS by multiplying the average individual risk  $r_i$  of experiencing an effect i by the number of people who receive similar dose that leads to the risk:

$$N_i = r_i f_i P$$

where

- P: the total exposed population and
- *f<sub>i</sub>*: the fraction of the population that is susceptible to the risk r<sub>i</sub>

In MACCS, this equation is applied to the populations in individual spatial elements on the computational grid. Total cases of a health effect over the entire region covered by the grid are calculated by summing the results obtained for individual spatial elements.

To examine conservatism in fatality estimation, 8 levels of annular grid by distance from the source was designed for both early and latent fatality estimation. The outer boundary was considered at 1.6 km (1 mile) and 16 km (10 mile) from the plant for early and latent fatality estimation, respectively, according to the standard of U.S. Nuclear Regulatory Commission (U.S.NRC). The population density of the whole area was assumed uniform at 430 people/km<sup>2</sup> based on the population data of Gijang-gun where Kori NPP exists [10]. To estimate the distribution of radiation dose to humans as a function of downwind distance, the reference dose was set as 1 Sv at the 0.8 ~ 1 km distance region, and then the doses at other distance regions were calculated by using the Gaussian plume model. The neutral atmospheric stability condition was assumed in this calculation. The dose was assumed to be uniform in the same distance region. Using the estimated dose values, fatality and risk estimation was made as described in the previous section. The result of examination is described in Table 5 and 6.

# 3.1 Early Fatality

Estimation of early fatality in level 3 PRA is based on the use of threshold dose as described in Table 3 and the concept of collective dose is not utilized. However, if the assessment area consists of only few grids, thus the dose distribution within the grid is poorly characterized, uncertainty in the result could be very high and fatality could be highly over-estimated or under-estimated. When we assume the extreme case of using only one grid, the average individual fatality risk r is 1 or 0 depending on whether the inner (0 ~ 0.2 km downwind) or the outer (1.4 ~ 1.6 km downwind) boundary dose is chosen as representative dose of the grid, respectively. This is because of the sharp reduction in the dose as a function of downwind distance within the grid. In comparison, the result of early fatality estimation in Table 5 shows 6.75E-02 risk when the grid was divided into 8 sub-grids for the calculation of human dose.

Therefore, it is important to use sufficient numbers of grids in the estimation of early fatality even though the distance under consideration is relatively short (e.g., 1.6 km). Description of detailed population distribution adjacent to NPP should also be carried out to provide population density data in each grid.

Distance [km]	0 ~ 0.2	0.2 ~ 0.4	0.4 ~ 0.6	0.6 ~ 0.8	0.8 ~ 1.0	1.0 ~ 1.2	1.2 ~ 1.4	1.4 ~ 1.6
Dose [Sv]	1.80E+01	5.10E+00	2.50E+00	1.50E+00	1.00E+00	7.60E-01	5.90E-01	4.80E-01
Hematopoietic Cumulative Hazard (H <sub>R</sub> )	1.65E+03	3.02E+00	8.54E-02	6.64E-03	8.75E-04	2.22E-04	6.25E-05	2.23E-05
Pulmonary Cumulative Hazard (H <sub>L</sub> )	4.24E+01	6.22E-03	0	0	0	0	0	0
Gastrointestinal Cumulative Hazard (H <sub>GI</sub> )	4.29E+00	0	0	0	0	0	0	0
<sup>*</sup> Total Cumulative Hazard (H <sub>EF</sub> )	1.70E+03	3.02E+00	8.55E-02	6.64E-03	8.75E-04	2.22E-04	6.25E-05	2.23E-05
Individual Risk of Total Early Fatality	1.00E+00	9.51E-01	8.19E-02	6.62E-03	8.74E-04	2.22E-04	6.25E-05	2.23E-05
Population	54	162	270	378	486	594	702	810
Early Fatality / Grid	54	154	22	3	0	0	0	0
Average Individual Risk of Early Fatality				6.75	E-02			

Table 5. Estimation of early fatality and risk when the region within site boundary was divided into 8 sub-regions

 $H_{\text{Eearly Fatality}} = H_{\text{Red marrow}} + H_{\text{Lung}} + H_{\text{Gastrointestinal}}$ 

Distance [km]	0~2	2~4	4~6	6~8	8~10	10 ~ 12	12 ~ 14	14 ~ 16
Dose [Sv]	3.30E-01	1.20E-01	6.60E-02	4.40E-02	3.30E-02	2.60E-02	2.10E-02	1.80E-02
Individual Risk of Leukemia	7.22E-04	2.06E-04	1.05E-04	6.79E-05	5.01E-05	3.90E-05	3.13E-05	2.67E-05
Individual Risk of Bone Cancer	2.93E-05	8.34E-06	4.26E-06	2.75E-06	2.03E-06	1.58E-06	1.27E-06	1.08E-06
Individual Risk of Breast Cancer	5.61E-03	2.04E-03	1.12E-03	7.48E-04	5.61E-04	4.42E-04	3.57E-04	3.06E-04
Individual Risk of Lung Cancer	1.11E-03	3.17E-04	1.62E-04	1.05E-04	7.71E-05	6.01E-05	4.82E-05	4.11E-05
Individual Risk of Thyroid Cancer	2.38E-03	8.64E-04	4.75E-04	3.17E-04	2.38E-04	1.87E-04	1.51E-04	1.30E-04
Individual Risk of Gastrointestinal Cancer	4.88E-03	1.39E-03	7.10E-04	4.59E-04	3.38E-04	2.64E-04	2.11E-04	1.80E-04
Individual Risk of Other Cancers	2.54E-03	7.23E-04	3.69E-04	2.38E-04	1.76E-04	1.37E-04	1.10E-04	9.38E-05
Individual Risk of Total Cancer Fatality	1.73E-02	5.55E-03	2.95E-03	1.94E-03	1.44E-03	1.13E-03	9.10E-04	7.79E-04
Population	5399	16198	26997	37795	48594	59393	70191	80990
Cancer Fatality / Grid	93	90	80	73	70	67	64	63
Average Individual Risk of Cancer Fatality				1.74	E-03			

Table 6. Estimation of latent fatality and risk when the region within site boundary was divided into 8 sub-regions

# 3.2 Latent Fatality

Although different dose response models can be utilized in the estimation of latent fatality, no threshold dose is used in the estimation. Thus, possibility of misusing collective dose exists in the estimation of latent fatality. For example, as shown in Table 6, 63 people in the  $14 \sim 16$  km region were estimated to be dead by cancer with 18 mSv of dose. This result is an outcome of multiplying collective dose by high population number in the area under consideration, even though the dose received by the individuals among the population is low.

The conservatism in the result can be, to some degree, mitigated by limiting the distance of assessment (e.g., 16 km) from the source, thus reducing the relative contribution of the far region from the source to the overall result. However, limiting the distance of consideration does not provide a perfect solution. In the case of low dose and high population density in the last grid, the overall estimate will be largely controlled by the large population number in the last grid even though the dose to each individual is very small.

### 4. Conclusions and Future Work

The method of early and latent fatality estimation in level 3 PRA was investigated and the conservatism in the result was examined in this study.

For the purpose of estimating both early and latent fatality, appropriate dose distributions among the affected population are found to be important. This study showed that large conservatism may be involved in the estimated fatality if the distribution of population dose as a function of downwind distance is not appropriately characterized. Early fatality estimation based on the use of threshold dose avoids the misuse of collective dose concept. However, dividing the region of evaluation into sufficient numbers of grid was found to be important to reduce uncertainty in early fatality estimation.

In contrast with early fatality estimation, there is no threshold dose in latent fatality estimation. Thus the chance of overestimation always exists with the use of the collective dose approach. Describing individual dose distributions within the affected region and representing the corresponding risk profile for the population should be considered to reduce conservatism in level 3 PRA.

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