Estimates of lung damage from inhaled beta-emitting radionuclides were adequately covered in the RSS. Other useful references for fatalities from inhaled radionuclides are reports by Filipy et al. (1980) and Hahn (1979).

In the RSS, the dose-response relationship for early fatalities is applied to a radiation dose that is the sum of the following:

1. External dose from the passing cloud.

2. External dose from contaminated ground (the duration of exposure to gamma rays emitted by deposited fission products, together with the degree of shielding, depends on the assumed emergency-response strategy).

3. Internal dose received during the first 7 days from inhaled radionuclides.

4. For bone-marrow exposure only, half of the internal dose from inhaled radionuclides received from day 8 through day 30.

5. For lung exposure only, the internal dose from inhaled radionuclides received from day 8 through day 365.

It can be seen that this is a specifically defined dose commitment. The consequence modeler should be cautioned that redefining the dosimetry assumptions used in the analysis would require redefinition of the dose-response relationships for early fatalities. Repair mechanisms may modify the effects of radiation exposure if the exposure is received over an extended period of time.

Injuries

The various types of impairment listed at the beginning of this section are detailed in Appendix VI of the RSS. A sublethal dose, defined as the dose expected to cause a clinical response in 10, 50, or 90 percent of the exposed population, was estimated for the various morbidities. These responses are not as easily determined as fatalities; thus the estimates have some subjectivity and increased uncertainty.

9.3.5.2 Late Somatic Effects

Late somatic effects consist of latent-cancer fatalities, nonfatal cancers, illnesses, and benign thyroid nodules. The RSS model included a latent period during which there was no increase in cancers and a plateau period with a uniform cancer rate for each cancer type.

The estimates of latent cancer calculated by the CRAC code are based on the BEIR I report (NAS-NRC, 1972), with the leukemia and bone-cancer values modified to reflect new data that became available between 1972 and 1975. The RSS developed three estimates of risk. The upper-bound estimate used the linear, no-threshold estimators from the BEIR I report (1972). The central estimate (see Section 9.4.8.4) incorporated a dose-effectiveness
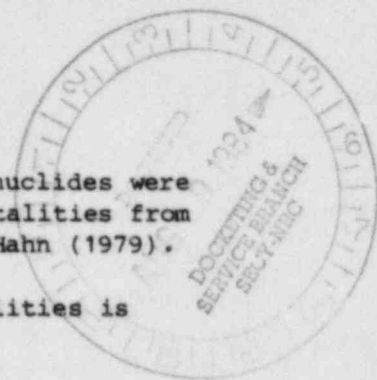
factor for exposures delivered at low dose rates. The lower-bound estimate took into account the large uncertainty in estimating effects from low doses and low dose rates and assumed a threshold of 10 or 25 rem for latent-cancer fatalities. The central-estimate approach is consistent with the BEIR III report (NAS-NRC, 1980), which used a linear-quadratic model to calculate risk estimators for latent-cancer fatalities. In addition, the BEIR III report published ranges that indicate some of the uncertainty associated with these factors. The upper and the lower bounds of the ranges were obtained with the linear model and the pure quadratic model, respectively. The risk estimates, based on the linear-quadratic model, of BEIR III (1980) are approximately 2 times lower than the BEIR I (1972) estimates based on the linear model.

Recently, Loewe and Mendelsohn (1980) conducted some preliminary reassessments of the dose data for people exposed by the atomic bombs at Nagasaki and Hiroshima. Since the BEIR estimates were calculated from these Japanese data, these reassessments could have some impact on the final estimates of latent-cancer risk. The Los Alamos National Laboratory is attempting to redefine the source term from the two bombs. In conjunction with this effort, the Oak Ridge National Laboratory is recalculating dose estimates. Final resolution of the health-effects estimate will likely follow these efforts. It is important that the consequence modeler be aware of these developments.

Except for leukemia, the latent-cancer fatalities presented in Table VI 9-4 of the RSS were calculated for a 30-year plateau period, whereas the BEIR I report (1972) used the remaining lifetime as the plateau period for "solid tumors." A comparison of the values obtained by assuming lifetime and 30-year plateaus is given in Table 9-9. (The lifetime plateau is implemented in the CRAC2 code.)

Table 9-9. Expected latent-cancer deaths per 10^6 man-rem of external exposure

| Type of cancer | Expected deaths per 10^6 man-rem | |
	CRAC health-effects model[a]	CRAC2 health-effects model[b]
Leukemia	28.4	28.4
Lung	22.2	27.5
Stomach	10.2	12.7
Alimentary canal	3.4	4.2
Pancreas	3.4	4.2
Breast	25.6	31.7
Bone	6.9	10.1
Other	21.6	28.0

[a]Based on a 30-year plateau period for all cancers except leukemia.
[b]Based on a lifetime plateau period for all cancers except leukemia.