"ROCKY FLATS TRANSURANICS AND RISK ESTIMATES"

COLORADO DEPARTMENT OF HEALTH

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ROCKY FLATS TRANSURANICS AND RISK ESTIMATES

To assist the Lamm-Wirth Task Force on Rocky Flats in its assessment of the consequences of plant operations, the Occupational and Radiological Health Division, Colorado Department of Health has prepared this document. The Division wishes to express its appreciation for constructive draft reviews of this effort by the U.S. Environmental Protection Agency, Standards and Criteria Branch, Office of Radiation Programs, and the various groups and contractors of the U.S. Energy Research and Development Administration.

This information is based on a considerable volume of the best available data. It is hoped that it will place in perspective the health consequences estimated on the basis of observed environmental data for 1970 through 1973.

A nomenclature section is provided to clarify terms used in this paper.

AIR

The Colorado Department of Health (CDH) air sampling station D-3 is located onsite to the east of location identified as the "lip" and on "highly" contaminated soil (approximately 2,000 dpm/g or 10 uCi/m^2) and has an overall average (1970-73) air concentration of 0.00604 pCi $Pu239+240/m^3$. CDH station D-5 is located at Indiana Street near Woman Creek and is associated with a soil Pu239+240 concentration of approximately 20 dpm/g or 0.1 uCi/m^2. The overall average air concentration (1970-73), adjusted by subtracting the air concentration of 0.00008 pCi $Pu239+240/m^3$ which CDH has identified as due to world-wide fallout during that time, is 0.00005 pCi $Pu239+240/m^3$.

The resuspension terms or factors for both locations, calculated from the above data, approximate $1 \times 10^{-9}/m$. The Health and Safety Laboratory (HASL) of the U.S. Energy Research and Development Administration (ERDA) has identified the same resuspension term (Volchok 1971); however, by the use of a different soil sampling technique (sticky paper) identified a resuspension term a thousand times greater ($1 \times 10^{-6}/m$). Wind and local disturbances resuspend soil deeper than the very topmost soil particles, which would adhere to sticky paper, and thereby dilute the higher contaminated topmost layer.* U.S. Environmental Protection Agency (EPA) suggests the use of a resuspension term of $1 \times 10^{-7}/m$. This would adjust for the quiescent location observation at D-5 from $1 \times 10^{-9}/m$ to a value applicable to conditions where local disturbances were routine and of high frequency.

The air concentration observed at D-5 can be used to calculate the worst case risk estimates associated with habitation of a locale contaminated to an undisturbed surface soil level of 2.0 dpm/g. CDH D-5 is associated with a soil concentration of 20 dpm/g, while the State plutonium-in-soil standard is 2.0 dpm/g. Agricultural plowing of the soil reduces the surface soil concentration by a factor of approximately 10, and the "worst case" resuspension term of $10^{-7}/m$ is a factor of 100 greater than observed at D-5 (or 20 dpm/g x $1 \times 10^{-9}/m$ = 2 dpm/g x 1/10 plowing factor x $1 \times 10^{-7}/m$).

*WASH-1537, Liquid Metal Fast Breeder Reactor Program, Proposed Final Environmental Statement (LMFBR-PFES), ERDA, December 1974, Volume II, Appendix G, Pages 19-23 has an interesting discussion of resuspension. Section 4 of this document and the attendant appendices are appropriate to the Rocky Flats situation.

To determine a risk estimate or health consequence of breathing the suspended materials, the EPA uses a factor of 12 rem/year to the lung per pCi Pu239+240/m^3, a 70 year period* and an estimate of 40 excess deaths/million man-rem over a 70 year period. The resultant estimated risk, using the air concentration observed at D-5 is:

{0.00005 pCi/m^3 x 12 rem/year/pCi/m^3 x 70 years x 40 estimated excess deaths/million man-rem} = 1.68 estimated excess lung cancer deaths/million population over a 70 year period. This estimate is based on the ICRP-TGLM identified below.

ERDA is using two models for estimating excess lung cancer deaths. The first is based on the International Commission on Radiation Protection (ICRP) Report #2 and the second on the ICRP Task Group Lung Model (TGLM). ICRP #2 separates compounds into two categories, soluble and insoluble, without much discussion. In this model, insoluble plutonium has a biological half-life (T_b) of 1 year and the lung mass is 1,000 grams. ICRP-TGLM addresses three classes of compounds based on clearance rates. Class D consists of compounds which are removed from the lung over a period of days. No plutonium or americium compounds fall in this class. Class W compounds are removed from the lungs over a period of weeks and Class Y compounds, over a period of years. The lung mass used in this model is 570 grams. Both ERDA and EPA use a particle size of 0.3 um activity median diameter (AMD) for lung dose calculations.

To determine the risk estimate of breathing the suspended materials containing Pu239, ERDA uses a factor of 177 rem/uCi inhaled (ICRP #2 insoluble basis) and 863 rem/uCi inhaled (TGLM Class Y basis). Using 70 years*, 7,300 m^3/year inhaled and 40 (15-110) estimated excess lung cancer deaths/million man-rem over 70 years, the following risk estimates are calculated:

{0.00005 pCi/m^3 x 1 x 10^{-6} uCi/pCi x 7,300 m^3 inhaled/year x 70 years x 177 rem/uCi inhaled x 40 estimated excess lung cancer deaths/million man-rem over 70 years} = 0.18 estimates excess lung cancer deaths/million population over a 70 year period (using ICRP #2);

and,

{0.00005 pCi/m^3 x 1 x 10^{-6} uCi/pCi x 7,300 m^3 inhaled/year x 70 years x 863 rem/uCi Pu239 inhaled x 40 estimated excess lung cancer deaths/million man-rem over 70 years} = 0.88 estimated excess lung cancer deaths/million population over a 70 year period (using TGLM).

Pu241 is essentially a factor of 10 greater than Pu239 in Rocky Flats plutonium, and the dose for Pu241 + Am241 is 2 rem/uCi Pu241 inhaled; therefore, the dose-risk estimates for it and its daughter, Am241, would be an approximate factor of 40 less than those calculated for Pu239+240 observed. Risk estimates for lung cancer from the Pu238 present in Rocky Flats plutonium would approximate a factor of 30 less than those estimated for Pu239+240.

*70 years is used as an appropriate life-time exposure.

See Appendix A for dose conversion term calculation method.

TABLE I

CLASS Y COMPOUNDS			
Estimated Excess Lung Cancer Risks/10^6 Population Exposed based on 0.00005 pCi/m^3 (Pu239+240) over a 70 year period.			
EPA (TGLM)		1.68	Pu239+240
		0.04	Pu241 + Am241
		0.06	Pu238
	Total	1.78	
ERDA (TGLM)		0.88	Pu239+240
		0.02	Pu241 + Am241
		0.03	Pu238
	Total	0.93	

When materials are inhaled and translocated to other organs, health consequences for other organs must be calculated. This is particularly true of the Class W compounds.

ERDA modeling for Class W compounds uses dose conversion terms of 3,300 and 1,300 rems, respectively, per uCi Pu239+240 inhaled for bone and liver. Respective dose-risk conversion factors are 6 (2-17) estimated excess bone and 2 (1-5) estimated excess liver cancer deaths/million population per rem.

TABLE II

CLASS W COMPOUNDS		
Estimated Excess Cancer Risks/10^6 Population Exposed based on 0.00005 pCi/m^3 (Pu239+240) over a 70 year period.		
ERDA (TGLM)		
	Bone cancer	0.51
	Liver cancer	0.07

ERDA is also using a factor of 46 rem to the gonads over 30 years* per uCi Pu239+240 inhaled, and both ERDA and EPA use the same bio-effects conversion terms, identified below, for estimating maximum genetic risks at equilibrium.

Specific defects	30-300/10^6/rem
Complex defects	6-600/10^6/rem

*30 years is used as the time appropriate for acquisition of an organ burden (gonads) and reproductive processes.

Therefore, the estimates of the maximum number of genetic defects which might appear in the exposed population and all future generations from the inhalation of Class W compounds at a concentration 0.00005 pCi/m^3 (Pu239+240) over 30 years are:

TABLE III

Estimated Maximum Genetic Risks/10^6 Population Exposed based on 0.00005 pCi/m^3 (Pu239+240) 30 years exposure.		
Specific defects	0.02 - 0.20	(all generations)
Complex defects	0.003 - 0.30	(all generations)

Contribution to the estimated risks by Pu241 + Am241 again approximate a factor of 40 less than those stated for Pu239+240 and the estimates for Pu238 are a factor of 30 less than those identified for Pu239+240. Dose and risk estimates based on the intake of radioactivity through the terrestrial food chain are unsignificant when compared to the risk estimates associated with inhalation and are not considered here.

The National Academy of Sciences, Biological Effects of Ionizing Radiations (BEIR) report, under genetic risk estimates, recommends that natural background radiation be used as a standard for comparison. "If the genetically significant exposure is kept well below this amount (natural background), we are assured that the additional consequences will neither differ in kind from those which we have experienced throughout human history nor exceed them in quantity."

The annual average background for Colorado is 0.2 rem/year although some areas which are highly mineralized will have levels half again as high (0.3 rem/year). The Rocky Flats environs has a higher than average background. For use here, 0.2 rem/year is assumed as the annual average background genetic significant dose.

Using a 0.2 rem/year natural background genetic significant dose with EPA's 200 estimated excess deaths/year/10^6 man-rem annual exposure and 200 estimated excess non-fatal cancers/year/10^6 man-rem annual exposure for 70 years, and the specific and complex genetic risk estimates previously identified for a 30 year exposure period, yield the following:

TABLE IV

Risk Estimates for Natural Background 0.2 rem/year genetic significant dose per million population		
excess deaths	2,800	over a 70 year period
excess non-fatal cancers	2,800	over a 70 year period
maximum specific genetic defects	180 - 1,800	all generations (30 year exposure)
maximum complex genetic defects	36 - 3,600	all generations (30 year exposure)

The above estimates are in accord with those generated in AEC WASH-1258, July 1973, Volume 1, Proposed Rule Making Action-As Low As Practical (Annex 5B), attached to this write-up as Appendix B.

To summarize, the risk estimates from the State plutonium-in-soil standard for a million person population at risk, based on the four year overall observed air concentration of radioactive contaminants, are provided in tabular form in Table V with the natural background radiation health consequences for comparison.

TABLE V

Risk Estimates/10^6 Population Exposed at the State Soil Standard (2 dpm Pu/g)

	Due to Rocky Flats Transuranics	Due to Natural Background
70 years exposure		
Excess deaths (EPA)		2,800
Excess lung cancer deaths (EPA)	1.78	
Excess lung cancer deaths (ERDA)	0.93	
Excess bone cancer deaths (ERDA)	0.53	
Excess liver cancer deaths (ERDA)	0.07	
Excess non-fatal cancers (EPA)		2,800
30 years exposure		
Genetic effects at equilibrium (all generations)		
Specific defects	0 to 0.02 - 0.2	0 to 180 - 1,800
Complex defects	0 to 0.003 - 0.3	0 to 36 - 3,600

Note: The above risk estimates for Rocky Flats are overestimates since the same material is assumed to be totally in either Class Y (for lung cancer estimates) and Class W (for bone and liver cancer and genetic effects estimates). (See Appendix C.)

WATER

The City of Broomfield's water supply (potable water) has an overall average (1970-73) Pu239+240 concentration of 0.05 pCi/L. Standard man ingests 1.2 liters/day as liquid and 2.5 liters/day from all sources. If a single source (liquid) is considered, a person would ingest 438.3 liters/year. Based on a 70 year intake the total activity ingested would

approximate 0.00153 uCi. Using ERDA dose conversion terms of 0.7 for bone and 0.3 for liver (rem per uCi ingested) and the previously described risk estimate factors, risk estimates may be calculated.

The risk estimates for Pu238 would be a factor of 30 lower and for Pu241 (without Am241) would be a factor of 100 lower.

As Americium is processed at Rocky Flats, no assumptions can be made regarding the Am241 concentrations in the liquid effluent stream and Broomfield water supply. Dow Chemical's analyses of Broomfield water have identified the levels to be less than 0.02 pCi/L. Total intake for 70 years would be less than 0.00061 uCi. The dose conversion terms provided by ERDA are 25 rem/uCi ingested for bone and 10 rem/uCi ingested for liver and the risk estimate factors of 6 excess bone cancer deaths/10^6 population per rem and 2 excess liver cancer deaths/10^6 population per rem over a 70 year period are used in the risk estimate calculation.

To estimate maximum genetic risk estimates, again a 30 year exposure period is used. The above identified concentration data of 0.05 pCi/L of Pu239+240 and $<$0.02 pCi/L of Am241, the dose conversion factors of 0.01 and 0.4 rem/uCi ingested for the gonads for the respective materials, and the genetic risk factors previously identified are also used in the calculation.

To summarize, the risk estimates for the Broomfield water supply for a million person population at risk, based on the four year overall observed water concentration transuranics are provided in tabular form:

TABLE VI

Risk Estimates/10^6 Population Exposed for the Broomfield Water Supply

	Rocky Flats Transuranics	
70 years exposure	0.05 pCi/L (Pu239+240)	$<$0.02 pCi/L (Am241)
Excess bone cancer deaths (ERDA)	0.006	$<$ 0.09
Excess liver cancer deaths (ERDA)	0.0009	$<$ 0.01
30 years exposure		
Genetic effects at equilibrium (all generations)		
Specific defects	0 to 0.0002 - 0.002	0 to 0.003 - 0.03
Complex defects	0 to 0.00004 - 0.004	0 to 0.0006 - 0.06

The risk estimates due to natural background, previously described in the section on air, are appropriate for comparison.

SUMMARY

In 70 million man-years (1 million persons x 70 years), a population living on soil contaminated initially at the state soil standard, approximately 500,000 persons would die from all causes (7 deaths per 1,000 persons was the overall Colorado mortality rate in 1974), approximately 3,000 persons may die due to natural radiation background and 2 persons might die as a consequence of the plutonium soil contamination. Genetic effects resulting from the plutonium contamination would be at least a factor of 10,000 less than that anticipated due to natural background. In the case of the Broomfield water supply, it is likely that no person would die, and the genetic effects would be at least a factor of 60,000 less than that anticipated due to natural background for an exposure time of 70 million man-years.

The calculations indicate that the health consequences from the breathing of air associated with soil contamination at the State's plutonium-in-soil standard and the drinking of water from the Broomfield supply does not constitute an imminent health hazard to the populace so exposed. While the calculated estimated risks per million population exposed over 70 years are low, continued effort to reduce further the associated consequences of plant releases is indicated. The calculated estimates herein are provided to give perspective to the situation. The calculated values must not be considered absolute as the caveats on dosimetry and risk estimators, included in Appendix C, indicate.

BIBLIOGRAPHY
(Major references used)

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"Occupational Radiation Protection", Training Course Manual, Public Health Service, U. S. HEW, 1965.

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Nomenclature Section
(Used in this presentation)

disintegration = radioactive decay of an atom by the emission of an alpha or beta particle.

dpm/g = disintegrations per minute per gram of material.

uCi = microcurie = one millionth of a curie = 2,220,000 dpm.

uCi/m^2 = microcurie per square meter of surface area.

pCi = picocurie = one millionth of a millionth of a curie = 2.22 dpm.

m^3 = cubic meter of air = 35.3 cubic feet of air.

pCi/m^3 = picocurie per cubic meter of air.

Pu = chemical symbol for plutonium.

Pu239+240 = the radioisotopes of plutonium having atomic weights of 239 and 240.

resuspension term = derived by dividing the air concentration (uCi/m^3) by the soil "concentration" (uCi/m^2).

rem = unit of radiation dose.

man-rem = 1 man x 1 rem = 1 man-rem = one million men x 0.000001 rem.

biological half-life = time required for ½ of a material to be removed biologically from an organ.

um = micrometer = one millionth of a meter = one micron.

AMD = activity median diameter.

10^{-6} = 0.000001 or one millionth.

10^6 = 1,000,000 or one million.

Pu241 + AM241 = parent plus radioactive daughter contribution.

pCi/L = picocurie per liter of water.

liter = L = 0.264 gallons = 0.95 quarts.

Am = chemical symbol for americium.

< = less than.

λeff = 0.693 or ln 2 divided by $\frac{Tp\ Tb}{Tp + Tb}$, where Tp and Tb are the physical and biological half-lives, respectively = the effective decay constant.

APPENDICES

APPENDIX A

General Dose Calculation Equations

$D_o^t = 51\ EI/M$ = rem for period t

51.1 = rem/MeV uCi-day/gram

E = $\sum$ EF(RBE)n = effective energy = MeV

$$I = \frac{vcf_x}{\lambda_{eff}} \left[t - \left(\frac{1}{\lambda_{eff}} \right) \left(1 - e^{-\lambda_{eff} t} \right) \right] = \text{uCi-days}$$

where

v = volume per day
c = concentration = uCi/unit volume
f_x = fraction ingested or inhaled reaching the critical organ
λ_{eff} = effective decay constant (day)
t = time in days

M = mass of critical organ in grams

ERDA dose conversion units.

rem/uCi inhaled or ingested = D_o^t/vct

where t in D_o^t and vct are identical

EPA dose conversion units.

rem/pCi/m^3/year = $D_o^t/c/t'$

where t' = fraction of t

Note: Dose conversion terms and biological effect factors must be for the same time integral for the risk estimate calculation to be valid.

APPENDIX B

Annex 5B, AEC WASH-1258, July 1973

The National Academy of Sciences/National Research Council recently completed an extensive review of information concerning the biological effects of radiation in order to evaluate the present radiation protection guides. A report, "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," by the Advisory Committee on the Biological Effects of Ionizing Radiation was published in November 1972. The Publication is referred to as the BEIR Report.

The BEIR Report discusses two analytical models for estimating risk based on absolute terms and relative or comparative terms. The absolute risk is the excess number of deaths (or cases of disease) due to irradiation. In practice, this is the difference between the risk in the irradiated population and the risk in the nonirradiated population. Relative risk is usually stated as a multiple of the natural risk. Either type of estimate, when applied to dose levels for which human data are lacking, involves assumptions concerning the dose-effect relationship. Either type of estimate also involves assumptions concerning the distribution of effects with time after irradiation, because pertinent information for many types of cancer in human populations is fragmentary at this time. Risk estimates derived from the two analytical models differ; consequently, the uncertainty ranges for risk estimates are greater than

would be found by simply reflecting the uncertainty ranges for parameter values in either of the models.

5B.1 Leukemia And Other Fatal Malignancies

If a population were to receive a single acute radiation exposure resulting in a dose too low for immediate biological effects but sufficiently high that delayed biological effects could be observed, the following typical response sequence may be identified: (1) a latent period during which no biological effect occurs (in excess of those occurring spontaneously from natural causes); (2) a period of elevated incidence of a specific effect (plateau region); and, perhaps, (3) a period where the incidence returns to normal. The duration of the latent period, the duration of the plateau region, and the magnitude of the increased incidence (risk) vary with the age of those irradiated and the particular biological effect observed. Although inferred by the term "plateau," the increased incidence is not necessarily uniform over the time interval defining the plateau region. Further, the plateau region may extend over a lifetime, thereby eliminating the subsequent time interval of normal incidence. Table 5B-1 taken from the BEIR Report presents the assumed values for parameters required to estimate the number of specified somatic effects from the total-body irradiation. Two values are presented for the duration of the plateau region for "all

other cancers" to reflect the uncertainty arising from limited observation periods for human experience. These values were used to demonstrate the differences in quantifying the possible number of deaths annually in the U.S. population from an annual dose of 0.1 rem derived from absolute and from relative risk analytical models. Table 5B-2 presents the results of both analyses. Tables 5B-3 and 5B-4 present the calculated number of deaths annually for each of the incremental age groups required to ascertain the values presented in Table 5B-2 using the absolute risk values and relative risk values respectively.

5B.2 Thyroid Cancer

The risk of thyroid cancer is given in the BEIR Report as 2.5 to 9.3 cases per 10^6 children per year per rad. The duration of the latency period is given as 5 years and the plateau region as 20 years. Values of 30 years and lifetime are assumed for the plateau region elsewhere in the same report. The BEIR Report does not provide thyroid cancer risk values for humans irradiated in utero or as adults. However, the report cites the "lesser susceptibility of the adult thyroid to radiation induced carcinogenesis, as compared with the thyroid of the infant or child." This statement appears to contradict the ICRP Publication No. 14 observation that among the Japanese there was "little if any variation with age at the time of exposure" for total risk from thyroid cancer.

The annual number of thyroid cancers calculated for the U. S. population, if it were to receive 0.1 rem per year, is given in the BEIR Report as 2,000 to 4,000 for assumed 30-year and lifetime plateau regions, respectively, using the relative risk analytical model. Unfortunately, supplemental information is not provided to permit more than limited estimates of the number of thyroid cancers for various exposure situations.

5B.3 Selected Risk Models

Risk estimates derived by summing contributions of small-interval age groups are desirable because significant differences in dose may occur between age groups when a common exposure is experienced, particularly where the dose is from radioactive material taken into the body and the intake of radioactive material as well as the organ mass varies with age. However, in many evaluations it is impractical to determine doses for each small age group, and in view of the overall lack of precision in the analyses, approximations of biological risks are quite sufficient.

The sample estimates presented in the BEIR Report will be used in the evaluations performed for the ES where possible, but where detailed information is lacking, approximations will be made. Where approximations are required, they will take the form of an absolute risk calculation. Factors used in the somatic risk for the ES approximations are presented in Table 5B-5.

Estimates for thyroid cancer cases are much less certain than those for cancer deaths owing to inadequate information. For purposes of this study, it will be assumed that the absolute risk for children (0 to 10 years) is 9.3 cases of thyroid cancer per 10^6 children per year for a lifetime with a 5-year latent period. The additional risk of thyroid cancer from irradiation *in utero* or as adults (10 to 70 years) is much more difficult to estimate owing to insufficient data and apparent discrepancies in available information. In view of the BEIR Report conclusion concerning the "lesser susceptibility" of the adult thyroid, it will be assumed that the absolute risk for adults (10-70 years) is 3 cases of thyroid cancer per 10^6 adults per year for a lifetime with a 5-year latency period.

Applying the foregoing absolute risk approximations to an assumed dose of 0.1 rem to the United States population, the somatic effects presented in Table 5B-6 are obtained.

The values for somatic risks presented in Table 5B-6 compare the various risk approximations used in this study with those in the BEIR Report. For the sample evaluation of a dose of 0.1 rem (100 mrem) per year to the United States population, the calculated number of deaths from leukemia and other fatal neoplasms obtained by using the risk values in Table 5B-5 multiplied by the product of the number of persons in

the age class irradiated and the dose rate (0.1 rem/year) will yield values approximately equal to those in Table 5B-3. The number of thyroid cancer cases calculated for a dose of 0.1 rem to the United States population is ∿2200. The BEIR Report does not offer values for the number of thyroid cancers estimated for population doses using the absolute risk model. However, the values for population thyroid cancer obtained with the risk model in this study are about equal to the lower range of values obtained with the relative risk model in the BEIR Report. A comparison of the risk values derived using absolute and relative models in the BEIR Report (Tables 5B-3 and 5B-4) shows that the risk values for thyroid cancer used in Table 5B-5 appear to be consistent with risk values for leukemia and other fatal neoplasms. It is concluded that the estimates of somatic effects for population exposures reported in the ES are within the range of those suggested in the BEIR Report.

5B.4 Genetic Effects

Four bases for assessment of genetic risks are discussed in the BEIR Report:

1. the risk relative to the natural background radiation (not quantitative);
2. the risk of specific genetic conditions and cytogenetic effects (values are presented in Tables 5B-7 and 5B-8);

3. the risk relative to the current incidence of serious disabilities (values presented in Table 5B-9 include values from Tables 5B-7 and 5B-8); and

4. the risk in terms of overall ill health. The contribution of the mutational component to ill health is arbitrarily taken as 20 percent and a doubling dose between 20 and 200 rem suggests an increase of ill health ranging from 0.1 to 1 percent per rem per generation.

To aid in quantifying the cost associated with biological risk, the BEIR Report presents one rationale which suggests one person-rem of genetically significant population dose would add an amount of illness equivalent in health cost to between $12 and $120 as a consequence of genetic effects. The BEIR Report does not provide a similar cost estimate for somatic effects of one person-rem.

The information presented in Table 5B-9 was used as a basis for calculating the number of cases of genetic diseases from population doses in the ES. In order to make use of the information in Table 5B-9, some adjustment in form was necessary.

There are $\sim 4 \times 10^6$ live births per year in the United States ($\sim 2 \times 10^3$ persons) or $\sim 2 \times 10^4$ live births per year per million persons. The current incidence of dominant disease (Table 5B-9) is given as

10^4 per million live births. Since it is desirable to express incidence per million persons, the normal incidence of dominant disease is:

$$\frac{(10^4 \text{ cases})}{(10^6 \text{ births})} \frac{(2 \times 10^4 \text{ births})}{(\text{million persons})} = \frac{200 \text{ cases}}{\text{million persons}}.$$

The number of cases of dominant disease in the first generation from a dose of 5 rem per generation is given as 50 to 500 (Table 5B-9) per million live births. Since there are $\sim 2 \times 10^4$ live births per year per million persons, there are $\sim 10^6$ live births among 50 million persons. A dose of 5 rem per generation (30 years) is ~ 0.17 rem per year; consequently, 50 to 500 cases are attributed to a population dose of 0.17 x 50 million person-rem (i.e., 8.5 million person-rem) or 6 to 60 cases per million person-rem. Other values in Table 5B-10 were derived in a similar manner from the values in Table 5B-9.

Table 5B-9 presents the range of number of cases by disease classification per million live births for a population. Population total-body doses are presented in units of person-rem. Genetic effects are noted in the progeny of those irradiated; consequently, the information in Table 5B-9 is expressed in terms of population dose, and the adjusted values are presented in Table 5B-10. The total-body doses, as presented in the ES, are calculated for a 5-cm depth of tissue. The genetically significant doses may be slightly

different from the dose at 5 cm owing to the specific "effective" depth of actual genetically significant tissue. No correction has been attempted, and the total-body dose will be assumed to also represent the genetically significant dose.

TABLE 5B-1

VALUES USED IN CALCULATING ESTIMATES OF RISK SHOWN IN TABLE 5B-2 (BEIR REPORT)

Age at Irradiation	Type of Cancer	Duration of Latent Period (years)	Duration of Plateau Region (years)*	Risk Estimate: Absolute Risk** (deaths/10^6/yr/rem)	Risk Estimate: Relative Risk (% incr. in deaths/rem)
In Utero	Leukemia	0	10	25	50
	All other cancer	0	10	25	50
0-9 Years	Leukemia	2	25	2.0	5.0
	All other cancer	15	(a)30 (b)Life	1.0	2.0
10 + Years	Leukemia	2	25	1.0	2.0
	All other cancer	15	(a)30 (b)Life	5.0	0.2

* Plateau region = interval following latent period during which risk remains elevated.

**The absolute risk for those aged 10 or more at the time of irradiation for all cancer excluding leukemia can be broken down into the respective sites as follows:

Type of Cancer	Deaths/10^6/Year/rem
Breast	1.5***
Lung	1.3
GI incl. stomach	1.0
Bone	0.2
All other cancer	1.0
Total	5.0

***This is derived from the value of 6.0, corrected for a 50% cure rate and the inclusion of males as well as females in the population.

TABLE 5B-2

ESTIMATED NUMBERS OF DEATHS PER YEAR IN THE U. S. POPULATION ATTRIBUTABLE TO CONTINUAL EXPOSURE AT A RATE OF 0.1 REM PER YEAR, BASED ON MORTALITY FROM LEUKEMIA AND FROM ALL OTHER MALIGNANCIES COMBINED. (BEIR REPORT)

Irradiation During Period	Absolute Risk Model* Excess Deaths Due to: Leukemia	Absolute Risk Model* Excess Deaths Due to: All other Cancer	Relative Risk Model* Excess Deaths Due to: Leukemia	Relative Risk Model* Excess Deaths Due to: All other Cancer
In Utero	75	75	56	56
0-9 years	164	(a) 73 (b) 122	93	(a) 715 (b) 5,869
10 + years	277	(a) 1,062 (b) 1,288	589	(a) 1,665 (b) 2,415
Subtotal	516	(a) 1,210 (b) 1,485	738	(a) 2,436 (b) 8,340
TOTAL	(a) 1,726 = 0.6% incr. (b) 2,001 = 0.6% incr.		(a) 3,174 - 1.0% incr. (b) 9,078 - 2.9% incr.	

* The figures shown are based on the following assumptions:

(1) 1967 U.S. vital statistics can be used for age specific death rates from leukemia and all other cancer, and for total U.S. population

(2) Values for the duration (a or b) of the latent period (the length of time after irradiation before any excess of cancer deaths occur), duration of risk ("plateau region"), and magnitude of average increase in annual mortality for each group are as shown in Table 5B-1.

TABLE 5B-3

CALCULATION OF ANNUAL NUMBER OF EXCESS CANCER DEATHS IN U.S. POPULATION FROM CONTINUOUS EXPOSURE OF 0.1 REM/YEAR, USING ABSOLUTE RISK MODEL (BEIR REPORT)

Age	1967 U.S. Pop'n (millions)	LEUKEMIA				ALL OTHER MALIGNANCIES						
		Excess Deaths Due to Irrad'n in Period			Total Excess Deaths	Excess Deaths Due to Irradiation During					Total Excess Deaths (a)	Total Excess Deaths (b)
		In utero	0-9 yrs	10+ yrs		In Jtero	0-9 yrs (a)	0-9 yrs (b)	10+ yrs (a)	10+ yrs (b)		
0-4	19.191	36	3	-	39	36	-	-	-	-	36	36
5-9	20.910	39	23	-	62	39	-	-	-	-	39	39
10-14	19.885	-	38	2	40	-	-	-	-	-	-	-
15-19	17.693	-	35	10	45	-	4	4	-	-	4	4
20-24	14.572	-	29	15	44	-	11	11	-	-	11	11
25-29	11.958	-	24	19	43	-	12	12	15	15	27	27
30-34	10.860	-	11	22	33	-	11	11	41	41	52	52
35-44	23.838	-	1	60	61	-	24	24	179	179	203	203
45-54	22.586	-	-	56	56	-	11	23	282	282	293	305
55-64	17.571	-	-	46	46	-	-	18	263	307	263	325
65-74	11.678	-	-	29	29	-	-	12	175	263	175	275
75-84	5.945	-	-	15	15	-	-	6	89	163	89	169
85 +	1.174	-	-	3	3	-	-	1	18	38	18	39
TOTAL	197.863	75	164	277	516	75	73	122	1,062	1,288	1,210	1,485

TABLE 5B-4

CALCULATION OF THE ANNUAL NUMBER OF EXCESS CANCER DEATHS IN THE U.S. POPULATION FROM CONTINUOUS EXPOSURE TO 0.1 REM/YEAR, USING RELATIVE RISK MODEL (BEIR REPORT)

	From 1967 U.S. Vital Statistics		LEUKEMIA					ALL OTHER MALIGNANCIES								
			% Increase Due To Latent Period					% Increase Due To Latent Period								
Age	No. of Leuk. Deaths	Leuk. Risk	In Plane	0-9 yr	10 + yr	Total % Increase	Excess No. of Death	In Plane	0-9 yr (a)	0-9 yr (b)	10 + yr (a)	10 + yr (b)	Total Increase (a)	Total % Increase (b)	Excess No. of Deaths (a)	Excess No. of Deaths (b)
0-4	684	795	3.75	0.45	-	4.2	29	3.75	-	-	-	-	3.75	3.75	30	30
5-9	601	697	3.75	2.75	-	6.5	52	3.75	-	-	-	-	3.75	3.75	26	26
10-14	475	735	-	4.8	0.2	5.0	24	-	-	-	-	-	-	-	-	-
15-19	411	439	-	5.0	1.1	6.1	25	-	0.5	0.5	-	-	0.5	0.5	5	5
20-24	264	1,059	-	5.0	2.1	7.1	19	-	1.5	1.5	-	-	1.5	1.5	16	16
25-29	222	1,221	-	3.75	3.1	6.8	15	-	2.0	2.0	0.05	0.05	2.05	2.05	27	27
30-34	233	2,226	-	1.27	4.1	5.4	13	-	2.0	2.0	0.15	0.15	2.15	2.15	48	48
35-39	312	4,132	-	-	4.9	4.9	15	-	2.0	2.0	0.25	0.25	2.25	2.25	102	102
40-44	436	9,102	-	-	5.0	5.0	22	-	2.0	2.0	0.35	0.35	2.35	2.35	214	214
45-49	598	15,525	-	-	5.0	5.0	29	-	1.5	2.0	0.45	0.45	1.95	2.45	303	380
50-54	726	23,696	-	-	5.0	5.0	36	-	0.5	2.0	0.55	0.55	1.05	2.55	249	604
55-59	935	34,034	-	-	5.0	5.0	47	-	-	2.0	0.60	0.65	0.60	2.65	192	549
60-64	1,223	38,284	-	-	5.0	5.0	61	-	-	2.0	0.60	0.75	0.60	2.75	230	1,053
65-69	1,536	42,567	-	-	5.0	5.0	77	-	-	2.0	0.60	0.85	0.60	2.85	236	1,214
70-74	1,783	42,833	-	-	5.0	5.0	89	-	-	2.0	0.60	0.95	0.60	2.95	263	1,294
75-79	1,697	37,557	-	-	5.0	5.0	85	-	-	2.0	0.60	1.05	0.60	3.05	225	1,145
80-84	1,202	25,062	-	-	5.0	5.0	60	-	-	2.0	0.60	1.15	0.60	3.15	150	739
85-89	600	12,128	-	-	5.0	5.0	30	-	-	2.0	0.60	1.25	0.60	3.25	73	394
90-94	172	3,710	-	-	5.0	5.0	9	-	-	2.0	0.60	1.35	0.60	3.35	22	124
95-99	24	681	-	-	5.0	5.0	1	-	-	2.0	0.60	1.45	0.60	3.45	4	23
100 +	5	89	-	-	5.0	5.0	-	-	-	2.0	0.60	1.55	0.60	3.55	1	3
Total:	14,336	298,647	56	93	589	Total:	738	56	715	5,869	1,665	2,415		Total:	2,436	8,340

5B-LJ

TABLE 5B-5

RISK VALUES SELECTED FOR ESTIMATING BIOLOGICAL EFFECTS FOR RADIATION DOSES IN THE ENVIRONMENTAL STATEMENT

Irradiation Period	Population Fraction	Lifetime Absolute Risk[a] (Deaths/10^6 person-rem) Leukemia	Other Fatal Neoplasms[b]	(Cases/10^6 person-rem) Thyroid Cancer[c]
In Utero	(0.2)	250 (187)[d]	250 (187)	-
0-9 years	0.2	41 (46)	30 (2920)	280
10 + years	0.8	17 (37)	82 (153)	65
Population Weighted Avg.		26 (37)	75 (426)	110 (202)

[a] Risk values for leukemia and other fatal cancers in this table were derived directly from the BEIR Report Table 5B-3 using lifetime plateau values. Risk values for thyroid cancers were derived by calculating risks for incremental age groups (similar to those in Table 5B-3) assuming a 5-year latent period, a lifetime plateau, and risk values of 9.3×10^{-6} cases/year/rem for children and 3.0×10^{-6} cases/year/rem for adults.

[b] The number of cancer cases in this category may be twice the number of cancer deaths.

[c] Thyroid cancers following irradiation (particularly in childhood) have a relatively low probability of being fatal, making them unlike the other types of cancer considered in this table. The survival rate for thyroid cancer is ~ 90% five years after detection.

[d] Values in parentheses are maximum risks based on relative risk calculations and are presented for comparative purposes.

TABLE 5B-6

ESTIMATED NUMBER OF CANCER DEATHS AND CASES OF THYROID CANCER PER YEAR IN THE U. S. POPULATION ATTRIBUTABLE TO CONTINUAL EXPOSURE AT THE RATE OF 0.1 REM PER YEAR USING ABSOLUTE RISK MODEL SELECTED FOR THIS STUDY

			RISK			EFFECTS		
Age Group Irradiated	Number in Class (millions)	Person-rem (millions)	Leukemia Risk (deaths/10^6 person-rem)	Other Cancer Risks (deaths/ 10^6 person-rem)	Thyroid Cancer Risk (cases/10^6 person-rem)	Leukemia (Deaths)	Other Cancer (Deaths)	Thyroid Cancer (Cases)
In Utero	4	0.3	250	250	---	75	75	0
Children	40	4	41	30	280	164	120	1,120
Adults	160	16	17	82	65	272	1,310	1,040
		20.3	Using Age Group Risk Values:			511	1,430	2,160
			Using Population Weighted Risk Values:			520	1,520	2,200
			From BEIR Report:			516	1,485	2,000-4,000*

* Derived from Relative Risk Model in BEIR Report

TABLE 5B-7

ESTIMATED EFFECTS OF RADIATION FOR SPECIFIC GENETIC DAMAGE[a]
(BEIR REPORT)

	Current incidence per million live births	Number that are new mutants	Effect of 5 rem per generation	
			First generation	Equilibrium
Autosomal dominant traits	10,000	2,000	50-500	250-2,500
X-chromosome-linked traits	400	65	0-15	10-100
Recessive traits	1,500	?	very few	very slow increase

a The range of estimates is based on doubling doses of 20 and 200 rem. The values given are the expected number per million live births.

TABLE 5B-8

ESTIMATES OF CYTOGENETIC EFFECTS FROM 5 REM PER GENERATION[a]
(BEIR REPORT)

	Current Incidence	Effect of 5 rem per generation	
		First generation	Equilibrium
Congenital anomalies			
Unbalanced rearrangements	1,000	60	75
Aneuploidy	4,000	5	5
Recognized abortions			
Aneuploidy and polyploidy	35,000	55	55
XO	9,000	15	15
Unbalanced rearrangements	11,000	360	450

[a] Values are based on a population of one million live births. Unbalanced rearrangements are based on male radiation only.

TABLE 5B-9

ESTIMATED EFFECT OF 5 REM PER GENERATION ON A POPULATION OF ONE MILLION LIVE BIRTHS[a]
(BEIR REPORT)

Disease Classification	Current Incidence	Effect of 5 rem per generation	
		First generation	Equilibrium
Dominant diseases	10,000	50-500	250-2500
Chromosomal and recessive diseases	10,000	Relatively slight	Very slow increase
Congenital anomalies	15,000	5-500	50-5000
Anomalies expressed later	10,000		
Constitutional and degenerative diseases	15,000		
Total	60,000	60-1000	300-7500

[a] This includes conditions for which there is some evidence of a genetic component. This table includes values from Tables 5B-7 and 5B-8.

TABLE 5B-10

ESTIMATED RISK OF GENETIC DISEASE ATTRIBUTED TO GENETICALLY SIGNIFICANT CHRONIC POPULATION DOSE IN CALCULATIONS FOR THE ENVIRONMENTAL STATEMENT

Genetic Effect	Spontaneous (Cases per 10^6 persons per year)	Risk* (Cases per 10^6 person-rad per year)**	
		First Generation	Equilibrium
Dominant diseases	200	6 to 60	30 to 300
Chromosomal and recessive diseases	200	(Relatively slight)	(Very slow increase)
Congenital anomalies	300	1 to 60	6 to 600
Anomalies expressed later	200		
Constitutional and degenerative diseases	300		
TOTAL	1,200	7 to 120	36 to 900

* Ranges are for 20 to 200 rem doubling doses.

** Adjustment of values in Table 5B-7 made use of the following factors:
Current U.S. population $\sim 2 \times 10^8$ persons
Current U.S. live births (annual) $\sim 4 \times 10^6$
5 rem/generation $\sim$ 0.17 rem/yr.

APPENDIX C

Caveats

From "Quantitative Health Estimates of Transuranic Releases", Nathaniel F. Barr, Division of Biomedical and Environmental Research, U.S. ERDA, October 1974, HASL-291.

"These estimates of potential risk are subject to great uncertainty for a number of reasons. There are no observations in man or in animals which are directly relevant to estimating potential health effects at the exceedingly low doses and dose rates being considered here. The estimates assume that health consequences are directly proportional to the quantity and residence time of alpha-emitters in the organs of man, and most probably lead to great overstatement of the maximum potential health consequences." (Quote in part)

From "Plutonium: Statement of the Problem", ORP, U.S. EPA, December 1974.

Problems in Dosimetric Modeling For Plutonium After Inhalation

There are a number of problem areas in toxicologic studies of plutonium and models for dosimetry. These include physical, physiological and biological assumptions used in the models.

To some extent the ICRP Task Group on Lung Dynamics Model is conservative in assumptions for inhalation exposure.

The model uses a tidal volume of 750 cc or 1450 cc. These values are adequate for an adult male, however adult females might be 339 cc or 655 cc, and children dropping to 16.7 cc or 32.4 cc in the sleeping newborn.[3] In this aspect the model provides estimates of exposure which are higher than would be expected in a general population.

The TGLD Model has other assumptions which are not conservative.

(1) The model uses a respiratory rate of 15 respirations per minute. For a heterogenous aerosol the percentage of deposition varies with breathing rate. The minimum level of deposition occurs at 15 to 20 respirations per minute and increases on either side of this value.[4] Deposition rates in hard workers, or in sleeping or sedentary individuals would be higher than the model predicts.

(2) No provision is made in the model for the known regional distribution of inhaled gases within the lung. Bates, Ball and Bryan have shown that in the upright individual, the distribution and rates of wash-in and wash-out of gases is different by about 40% between the upper and lower lobes of the lung.[5] This has profound implications in that it affects both the distribution of inhaled aerosols for settling or diffusion within alveolar.

(3) The model is based on laminar flow in tubes at a constant rate, per the calculations of Findaisen and Landahl[1]. A summary of the assumptions in the model that need refining includes[4]:

(a) The pattern of air flow during respiration is not constant, but goes from zero to some maxium and then returns to zero. Information is needed on the effect of this pattern on deposition.

(b) Pulsating air flow is caused within the lung by the filling and emptying of the heart. The effect of this action on local deposition is not known.

(c) Air flow in the lung may be a mixture of laminar and turbulent flows. The extent of this phenomenon and its effect on deposition should be investigated.

(d) The bulk of new air does not mix volumetrically with lung air. Non diffusable particles (>0.5μ) will penetrate only as far as the new air goes, while finer particles (<0.5μ) will be able to penetrate the depths of the lung by diffusion similar to that of a gas molecule. This also has implications for alveolar deposition of particles, particularly as it relates to source terms.

(e) The respiratory tree is not composed of circular tubes but irregular cross section tubes often corregated or folded over. The effects of these irregularities on turbulance and deposition are not known.

(f) The effects of respiratory excursions (coughs) on respiratory clearance are not considered.

Even if the lung model were considered to be accurate enough for practical purposes, difficult problems peculiar to plutonium distribution and retention are not settled. There is evidence that the higher specific activity isotopes of plutonium (^{238}Pu, ^{237}Pu) are translocated from the lung more rapidly than ^{239}Pu and have a different distribution pattern in the body after translocation.

In general the ^{238}Pu has a distribution pattern after translocation from the lung which resembles the pattern of injected monomeric ^{239}Pu.[a] When ^{239}Pu is translocated from the lung, the pattern of distribution within the body resembles that of injected polymeric ^{239}Pu.[b] This suggest that the ^{238}Pu is dissolving and being transported as a transferin complex[c] within the body while the ^{239}Pu is being engulfed and transported as particulate material (P. Durbin, IRPA Congress, 1973).

It has been suggested that the more rapid translocation of ^{238}Pu relative to ^{239}Pu aerosols deposited in the lung is due to the effect of the specific activity on local chemistry after deposition. The higher activity ^{238}Pu produced enough radicals in its aqueous environment (lung mucus or parenchyma) to influence local chemistry and the rate of dissolution of the particle (D. Craig, IRPA Congress 1973). If these statements are correct separate models must be developed for each plutonium isotope.

The available estimates of half times for the translocation of plutonium from alveolar deposits and from lymph nodes are subject to considerable uncertainty. The data on retention, particularly in lymph nodes is based on animal experiments with relatively high levels of exposure.

---

a/ Monomeric plutonium is in the form of single molecules of the plutonium compound, not a large number of particles aggregated.

b/ Polymeric plutonium is a form where a number of molecules of the plutonium compound aggregate together as a colloid.

c/ Transferin is the serum protein which binds iron as an iron-transferin complex to transport the iron throughout the body.

Dogs at Hanford, exposed to $^{239}PuO_2$ had retained lung burdens of 1.5 to 960 nCi/g of lung (100 to 27,000 times the maximum permissible lung burden for man)[6]. One of the more common responses to this high level of exposure is local cell death followed by fibrosis and scarring in the area[7,8]. The radiation induced changes in the tissue at the high levels of exposure used, change the function of the tissues and interfere with normal metabolism. Extrapolation of retentions seen at these dose levels to lower ranges must be considered suspect until confirmatory evidence is available.

Much environmentally distributed plutonium has the unique property of being in the form of very small sub micron particles weakly attached to larger dust particles. Unfortunately the TGLD model does not address the question of extremely small particles bound to 1.0 μ (or larger) particles. This question must be addressed since any EPA risk estimates must consider this problem for plutonium that escapes through HEPA filters.

Problems in Dosimetric Modeling for Ingested Plutonium.

Problems related to predicting the body burden the ingestion of plutonium are slight relative to those for the case of inhaled plutonium. Although ICRP Models use GI Tract absorption coefficents of 3×10^{-5} for insoluble forms like PuO_2 there are suggestions in the literature that these values may be a factor of 10^4 to 10^3 higher in very young animals[2] and a factor of 10 higher for ^{238}Pu in dairy cattle[3]. There is need for further investigation of absorption values for ingested plutonium in various ages of animals and using different forms and isotopes of plutonium.

Problems in Dose-Conversion Factors for Plutonium Models

Distribution of inhaled or ingested plutonium in the body

Although quite a lot is known about distribution and retention of injected plutonium compounds, less information is available on what happens after inhalation or ingestion.

Current evidence suggests the ingested plutonium enters the circulatory system in ionic form and is distributed through the body as a plutonium-transferrin complex much as monomeric injected plutonium. In this case the skeleton; liver; tissue and excreta distribution would be about 60%; 20%; 20%.[2]

Inhaled plutonium may be either absorbed in ionic form or transferred into the circulation in particulate form by phagocytosis, pinocytosis or mechanical transport. If the plutonium is absorbed in ionic form, it follows the transferrin-complex pathway with primary deposition on new bone surfaces, so that skeletal and marrow exposures are important parameters. If the plutonium enters the circulatory system as a particle it is trapped and retained by cells of the reticulo-endothelial system (RES). RES Cells are located primarily in the liver and spleen but also in lymph nodes and bone marrow. Percentages of deposition for circulating particulate plutonium are 60-80% liver; liver; 1-7% spleen; 1-16% skeleton; about 6% tissues and excreta.

As was pointed out earlier there may be relationship between the isotope and the distribution within the body, e.g. ^{238}Pu distributing differently than ^{239}Pu. This may require that separate models be used for each isotope of plutonium.

The question of biological availability of plutonium compounds is not yet answered. The extent to which plutonium is mixed with other elements in forming the particles in an aerosol has not been defined for all sources. If plutonium and some other element are mixed in a particle, differential leaching may increase the surface area of plutonium available for the dissolution processes. This has been suggested as an explanation for the more rapid translocation of plutonium from the lung than expected after accidental human exposure to a plutonium aerosol from a high explosive burst in a weapon safety test.

The forms of plutonium after weathering for some years or after entering the biological food pathways is not known. The chemical forms and valance states and the isotope involved all may affect the metabolism of plutonium, another area where further study is needed.

The distribution of plutonium in the body after inhalation and ingestion has not been studied in enough detail. The lack of estimates for the distribution and retention of plutonium by the gonads in animals has not been reconciled with the percentage of individuals (10-70%) with relatively high gonad burdens seen in autopsy samples from Colorado and New York.[9] In this study when the gonads were assayed they often had a higher activity per gram than lung, liver, lymph nodes, or skeleton. Unless all tissues are examined to determine the distribution of plutonium, risks will continue to be calculated for an animal consisting only of lungs,

lymph nodes, liver, skeleton, kidney and occassionally spleen. A real animal contains many more tissues.

The calculation of dose from inhaled plutonium particles is complicated by the fact that the tissue in which the energy really is deposited is not known. Since plutonium, whether as "hot particles" or as a diffuse aerosol, does not deposit uniformly over lung parenchyma, the dose is unevenly distributed in the lung. Also, even though the range of plutonium alpha particles may be only 50μ, the mass of the whole lung is used in calculating the dose. These inequalities are certain to lead to under estimation of dose to the tissues being irradiated, possibly to a very great under estimate of dose to critical tissues. More data is needed for better dose calculation.

Problems in Using Health Effects Data With Plutonium Models

Even if the inherent uncertainty in health effect estimates calculated by the BEIR Committee is disregarded, care must be taken in applying these estimates to plutonium organ doses. BEIR risk estimates are based on observations of the response of fairly well defined tissues. Although doses can be calculated for many organs or tissues on strictly biophysical considerations, we do not necessarily have an estimate of the risk associated with that dose. About the only risk values which can be used now are risk of bone cancer from plutonium based on the calculated bone dose and risk of leukemia based on a calculated marrow dose from plutonium deposited in marrow and from plutonium deposited in surrounding bone (Effects are on the basis of the exposure dose in rems). The lung risk estimate in BEIR is relatable only to the dose received by the basal cells of the bronchial epithelium, which dose the current lung models users do not calculate. The risks in BEIR can be employed only if the specific organ doses applicable to BEIR risk estimates can be computed. For example if the thyroid dose from plutonium can be calculated, BEIR risk estimates for thyroid related health effects can be used to determine the expected health effects.

There are also unresolved questions in interpreting the observed effects of plutonium in animal health and correlating them with man for whom we have no observations.

(a) Liver and bile duct tumors have been observed in animals, but there is no corresponding data for man.

(b) Lung tumors have been observed in animals but they are generally alveolar in origin. The tumor for which human risk data is given in man is a bronchogenic tumor. There is no concensus yet as how or if the human and animal tumors relate to one another.

(c) Osteosarcomas have never been observed in animals exposed to $^{239}PuO_2$ aerosols, however osteosarcomas have developed in animals exposed to $^{238}PuO_2$ aerosols (D Craig, IRPA Congress 1973). Whether this means that health effects models must also be for individual plutonium isotopes or not is unclear.

(d) The effects on animals of uniform plumonary distribution of isotope versus distribution as high activity particles in "hot spots" in the lungs has not yet been examined.

STATE OF COLORADO DEPARTMENT OF HEALTH
4210 EAST 11TH AVENUE · DENVER, COLORADO 80220 · PHONE 388-6111
R. L. CLEERE, M.D., M.P.H., DIRECTOR

OCCUPATIONAL AND RADIOLOGICAL HEALTH DIVISION

November 17, 1972

Jefferson County Board of Commissioners
Jefferson County Courthouse
Golden, CO

Attention: Pat Mahon, County Attorney

Gentlemen:

By telephone, Mr. Mahon requested information which would allow both the Board of County Commissioners and subdividers to fully comply with the provisions of Chapter 106, CRS 1963 as amended (106-2-34(3)(a) and (c)(iv)). Specific reference to the situation of the U. S. A.E.C. Rocky Flats Plant environs was discussed and covered in a subsequent letter to Mr. Mahon.

Since that time we have met with personnel from the U.S. Atomic Energy Commission (AEC) and U. S. Environmental Protection Agency (EPA) (meeting summary enclosed) regarding standards for Plutonium in Soil for urban situations. No standards, criteria, or guidelines are immediately available. EPA has established a priority for setting standards or guidelines in this regard; however, the target date for finalization is two (2) years distant. The advice given, due to present knowledge (and lack thereof), is to be conservative.

In this regard, the attached "Suggested Interim Guidance" is provided by the Department. Hopefully, the information and review required therein, will allow proper and adequate decisions to be made.

Also provided herein is all pertinent correspondence and information which has been involved.

If there are any questions regarding content, please do not hesitate to contact this office.

Sincerely,

A. J. Hazle, Assistant Director

AJH/ljw
cc: David Foster, Counsel, Colorado Department of Health
Enclosures

SUGGESTED INTERIM GUIDANCE
for
EVALUATION (Nov. 1972)
required under Chapter 106, CRS 1963, as amended
(106-2-34(3)(a) and 106-2-34(3)(c)(iv)

To assist the Board of County Commissioners in fulfilling the requirements of the Act, the following is provided by the Colorado Department of Health.

I. Information to be provided in the required evaluation:
- A. Prior land use history of proposed plat
  - 1. Specify for the proposed plat and any portion thereof, the different land use histories in relation to solid disturbance.
    - a. Situations to be considered
      - (1) Virgin or undisturbed ground
      - (2) Disturbed ground (specify depth of disturbance)
        - (a) Single (date)
        - (b) Routine
          - ((1)) Yearly
            - ((a)) Contour Plowing
            - ((b)) Strip Plowing
          - ((2)) Others (specify)
        - (c) Others (specify)

- B. Method of taking representative samples
  - 1. Detailed description of sample acquisition and its justification in relation to hazard analysis.
    - a. Items to be considered (but not limited to)
      - (1) Depth of sample
        - (a) Disturbed soil situation
        - (b) Undisturbed soil situation
      - (2) Involved surface area of indivudal sample
      - (3) Number of samples taken per unit land area
        - (a) Increase or decrease number due to
          - ((1)) Different land forms
          - ((2)) Water bodies and streams

- C. Method of Analysis
  - 1. Describe in detail, or if documented procedure, reference the procedure and qualify any changes in that referenced procedure.
  - 2. Specify quality control procedures for the analytical procedure employed.
    - a. Number of analyses per sample (replicates)
    - b. Participation in inter-laboratory cross-check programs (AEC or EPA)
    - c. Standards traceable to nationally recognized source

D. Evaluation
   1. Results of analyses to be presented by table and map with specific sampling sites and land use histories identified.
   2. Methods for "averaging" and handling anomalous data specified.
   3. Criteria or guidelines used in the hazard evaluation specifically described or referenced.
   4. Specify all assumptions used and qualify their use.
   5. Specify appropriate methods for control of the radiation hazard.
   6. Specify the name and qualifications of the individual performing the radiation hazard evaluation.

II. A. Prior to a subdivider, or an agent for a subdivider performing or causing to perform the required evaluation, the information required under Item I, with exception of I. D. 1. and I. D. 5., shall be submitted to the Board of County Commissioners for review and comment.

B. The Colorado Department of Health shall assist the Board of County Commissioners in their review of the proposed evaluation. Upon approval as appropriate to fulfill the requirements of the Act, the evaluation may proceed.

III. Upon completion of the required evaluation and submission to the Board of County Commissioners, the Colorado Department of Health shall assist the Board in its deliberations as to the efficacy of the proposed land use in relation to the radiation hazard identified. Disposition of this matter may take various forms:
   A. Unqualified approval
   B. Qualified approval
   C. Postponed or deferred decision (taken under advisement)
   D. Disapproval or rejection.

IV. Areas of Concern (Specific for each county)
   A. General areas of natural deposits of uranium and/or thorium
      1. Locations identified by the U. S. Geological Survey as possessing significant quantities and concentrations or as otherwise identified.

   B. General areas surrounding nuclear facilities
      1. Production and/or utilization facilities, as defined by U. S. A.E.C. regulations.
      2. Uranium and thorium mill towns.
      3. Areas as otherwise identified.

APPENDIX

Area of Concern (under Item IV.B. (3) of the Suggested Interim Guidance-November 1972) for environs of the U.S. A.E.C. Rocky Flats Plant, Jefferson County.

Effective: December 1973

General Area of Concern

Area bounded by Highway 93, Highway 128, Simms Street between Highway 128 and W. 100th Avenue, W. 100th Avenue to Highway 121, Highway 121, W. 88th Avenue (extended) and Highway 72.

The identified area was selected after thorough review of all available radiation evaluation data on this plant's environs. More proximal locations to the plant site will require greater consideration due to the greater potential radiation hazard near the present plant boundary.

JBB/lc
(1/9/74)

AMENDMENT TO THE

State of Colorado Rules and Regulations Pertaining to Radiation Control

Subpart RH 4.21 is added:

RH 4.21 Permissible Levels of Radioactive Material in Uncontrolled Areas

4.21.1 Plutonium. Contamination of the soil in excess of 2.0 disintegrations per minute of Plutonium per gram of dry soil or square centimeter of surface area (0.01 microcurie plutonium per square meter) presents a sufficient hazard to the public health to require the utilization of special techniques of construction upon property so contaminated. Evaluation of proposed control techniques shall be available from the Department of Health upon request.

Adopted: Colorado State Board of Health
March 21, 1973

Effective: May 1, 1973

AJH/ljw
(3/26/73)

AJL

DENVER REGIONAL COUNCIL OF GOVERNMENTS

1776 SOUTH JACKSON STREET • DENVER, COLORADO 80210 • 758-5166

July 31, 1975

RECEIVED
AUG 5 1975
DIVISION OF PLANNING

Mr. Joseph G. Wagner, Director
Department of Housing & Urban Development
Federal Housing Administration
Denver Insuring Office
Title Building
909 - 17th Street
Denver, Colorado 80202

ATTN: Betty Patton

RE: HPR/037-75 DHUD-FHA Number - ASP-1274

Dear Mr. Wagner:

In accordance with OMB-A-95 procedures, the Denver Regional Council of Governments has reviewed the above captioned project. In addition, the DRCOG has notified the affected agencies of the project and has requested their comments. Those comments received by our office are attached to this letter.

The Council of Governments appreciates this opportunity to be of service to you.

Sincerely,

Robert D. Farley
Executive Director

RDF/nl

Enclosures: HPR and Comments

cc: Richard Brown - Colorado State Division of Planning

COMMENTS ON HPR/037-75

DRCOG Staff - This is a multi-use project including single family, multiple family and commercial development. The portion of the project being considered in this filing includes 28 single family units on 5.96 acres, or a density of approximately 4.6 units per acre. The zoning of the area has become PUD through density transfer agreements with the City of Westminster.

Although the proposed units will market within the average single family price range for the Region, they will not meet the needs of low and moderate income families and thus will not contribute to Westminster's "fair share" goal.

The project is consistent with the regional Land Use Plan which indicates that the planned use for the proposed site is to be low density urbanization. Utilities exist at the site with telephone and electric underground, gas, water and sewer prepared for.

There does not seem to be any conflicts with local governments or with plans and policies defined at the local level. However, there is a possible concern with a potential radiation hazard from proximity to the Rockwell - Rocky Flats plant to the west. Jefferson County Health Department has requested a meeting of all concerned and affected agencies and parties to discuss the issue.

A meeting was held on Tuesday, July 22, 1975 at the Denver Regional Council of Governments' Offices. The following persons were in attendance:

Dr. Carl Johnson, M.D.	Jefferson County Health Department
Richard Bell	Jefferson County Health Department
Dan Axlund	Federal Housing Administration
Charles Todd	Witkin Homes
John Franklin	Planner - Westminster
Richard Brown	Colorado Division of Planning
James Arner	Colorado Division of Planning
Claudia VanWie	Department of Housing & Urban Develop.
Walt Kelm	Department of Housing & Urban Develop.
Albert J. Hazle	Colorado Department of Health
Bert L. Crist	Colorado Department of Health
Earl W. Bears	ERDA - Rocky Flats
Robert E. Yoder	Rockwell International - Rocky Flats
Larry J. Borger	DRCOG Staff
David Choate	DRCOG Staff
Nancy Love	DRCOG Staff

The meeting was called because Dr. Carl Johnson, Jefferson Counth Health Department felt that there were some misunderstandings concerning the proper testing procedures for any development that falls in the area of concern as defined by the Colorado Department of Health. Also, there was a problem

in the evaluation of this area and he felt that perhaps the informational meeting might solve some of these problems.

Discussion followed Dr. Johnson's opening presentation.

The conclusion of the meeting was that the Colorado Department of Health would review all of the past evaluations of developments in the areas of concern located adjacent to the project in question and would hold off advising the Federal Housing Administration to approve the project until such time as the reviews had been completed.

The Jefferson County Health Department would recommend to the Federal Housing Administration that before they approve the subdivision that the developer in fact conduct a study of the area in compliance with State regulations.

In the event that the Colorado Department of Health reduces the area of concern after reviewing previous studies, then no test would be required. The Health Department felt that it would take at least two weeks until they would have any conclusive evidence regarding the area of concern.

It was recommended that the two health departments communicate with the developer, the City of Westminster and the Federal Housing Administration prior to any decisions being made.

(See attached for additional comments)

Jefferson County Colorado

NL

July 11, 1975

Ms. Nancy B. Love, Coordinator
Denver Regional Council of Governments
1776 South Jackson Street
Denver, CO 80210

Dear Ms. Love:

This letter is in response to your referral of the proposed federally assisted project known as Kings Mill North, Filing #3, Case No. HPR/037-75.

The area of this proposed project is within the Standley Lake Water and Sanitation District. It is also within the potential radiation hazard area adjacent to Rocky Flats as designated by the Colorado Department of Health.

This Department recommends that prior to any approval of this project and development within this area, the developer submit a radiation evaluation study for Colorado Health Department review.

Additionally, this Department requests a meeting be held with the following agencies in attendance: Witkin Homes, Inc., City of Westminster Planning Department, Denver Regional Council of Governments, Federal Housing Administration, Jefferson County Health Department and the Colorado Health Department.

Should you have any questions regarding this matter, please contact this office.

Sincerely,

Carl J. Johnson, M. D.
Director of Health

CJJ:lk

cc: Charles E. Todd, Vice-President, Witkin Homes, Inc.
John Franklin, Director, City of Westminster Planning Dept.
Al Hazle, Colorado Department of Health
Robert C. Rosenheim, Regional Administrator, Housing & Urban Development

JEFFERSON COUNTY HEALTH DEPARTMENT
260 SOUTH KIPLING STREET • LAKEWOOD, COLORADO 80226
303/238-6301

Return to Nancy Love

LOCAL PLANNING AGENCY REVIEW

PROJECT NOTIFICATION
HPR No: 037-75
~~PNR No.~~:

PLEASE RETURN YOUR COMMENTS BY July 18, 1975

COG Notification:

Applicant:

Name : Witkin Homes, Inc.

Representative: Charles E. Todd - Vice President

Address : 9725 East Hampden Avenue

Denver, Colorado 80231

Telephone No.: 755-4111

COMMENT ON PROPOSED PROJECT (Attach additional sheet if needed)

The Metropolitan Denver Sewage Disposal District No. 1 is presently operating under a discharge permit issued by the Environmental Protection Agency. One of the requirements of this permit is that the increase in the average annual wastewater flow to the Metro Central Plant be limited to no more than the average annual increase experienced over the past four years. This requirement will be in effect until such time that the present plant expansion has been completed. Completion of this project is scheduled for September 1976.

It is therefore requested that regulatory agencies control the activities within their jurisdictions such that the increase in the average annual wastewater flow from their areas, which is tributary to the Metro Central Plant, be limited to no more than the average annual increase experienced from that area over the past four years.

Metropolitan Denver Sewage Disposal District No. 1 — Local Planning Agency

July 14, 1975 — Date

By William E. Korbitz — Name

William E. Korbitz — Signature

Manager — Title