TECHNICAL APPENDICES

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APPENDIX A

Summary of the Fernald Dosimetry Reconstruction Project's Dose and Risk Estimates.

I. Summary of Fernald Dose Reconstruction Project's Dose and Risk Estimates

A. THE FERNALD DOSIMETRY RECONSTRUCTION PROJECT

The Fernald Dosimetry Reconstruction Project (FDRP) was initiated in 1990 in response to community concerns about the possible health effects resulting from the operation of the Feed Materials Production Center (FMPC). The project was completed in 1996 and draft findings of this project were reported to the community in August 1996. A draft report was provided at that time. (RAC, 1996). Since that time, the draft report has undergone extensive review by the National Academy of Sciences and others. The final FDRP report was published in September 1998. This appendix summarizes the methods used in the dose reconstruction project and the results of dose and risk estimation for nine hypothetical exposure scenarios that are available in the FDRP final report.

The FMPC was a government-owned, contractor-operated facility. It is located about 15 miles NW of Cincinnati, Ohio. From the early 1950s through 1988, the FMPC produced uranium metal for the U.S. defense program. The primary purpose of the Fernald Dosimetry Reconstruction Project was to estimate the quantities of radioactive materials released to the environment during operation of the FMPC. However, the Project also estimated potential doses from exposures to uranium and thorium isotopes and their decay products (hereafter referred to collectively as uranium) and radon-222 and its decay products (hereafter referred to collectively as radon) and the health impacts of these exposures on residents of a surrounding 10 kilometer region (the assessment domain). To do this, nine profiles or "exposure scenarios" were created to represent hypothetical community members. The scenarios were developed to represent a wide range of possible exposures and were defined to emphasize factors likely to impact the dose estimate. These factors include date of birth, gender, locations of home, work and school, the years spent within these locations, diet, and other lifestyle factors such as percent of time spent swimming in contaminated water. Tables A1 – A9 summarize the scenario definitions in terms of the values assigned each of these relevant factors. These summaries are obtained directly from Appendix J of the draft Fernald Dosimetry Reconstruction Report (RAC, 1996). A more detailed discussion of each of the factors is presented in Appendix C of this report and in the final Fernald Dosimetry Reconstruction Report, entitled "Task 6: Radiation Doses and Risk to Residents from FMPC Operations from 1951-1988." (RAC, 1998).

Table A1. Scenario 1

Gender F
Born 1946
Home 1.7 km NE
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work In the home
Contaminated water sources Great Miami River (GMR)

	Period 1 1946-1951	**Period 2 1951-1957**		**Period 3 1957-1964**		**Period 4[a] 1964-1965**	**Period 5 1965-1989**
Location	Home	Home	EE	Home	HCR	Home	Home
Time indoors (%)	80	67	14	67	14	83	83
Indoor activity index[b]	0.5	1	1	1	1	1	1
Time outdoors (%)	20	15	4	15	4	17	17
Outdoor activity index[b]	1.5	1.5	1.5	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover (hour^{-1})	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none	none	none
Contaminated irrigation water source	none	none	none	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0	0.5	0.5
Contaminated vegetables (% of intake)	50	50	0	50	0	50	50
Contaminated milk (% of intake)	100	100	0	100	0	100	100
Contaminated beef (% of intake)	50	50	0	50	0	50	50
Contaminated poultry (% of intake)	50	50	0	50	0	50	50
Contaminated eggs (% of intake)	100	100	0	100	0	100	100
Contaminated fish (% of intake)	50	50	0	50	0	50	50
Contaminated water source for fish	GMR	GMR	none	GMR	none	GMR	GMR
Swimming (% of time)	2	2	0	2	0	0	0
Contaminated water source for swimming	GMR	GMR	none	GMR	none	GMR	GMR
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1	0.05	0.05

[a] Pregnancy
[b] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

Table A2. Scenario 2

Gender M
Born 1/1/1951
Home 2 km W
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work In Hamilton, 12 km N (outside of assessment domain)
Contaminated water sources Paddy s Run (PADRUN), Great Miami River (GMR)

	Period 1 1951-1956	**Period 2 1956-1962**		**Period 3 1962-1969**		**Period 4 1969-1989**	
Location	Home	Home	EE	Home	HCR	Home	Work[a]
Time indoors (%)	83	67	14	67	14	60	24
Indoor activity index[b]	0.5	1	1	1	1	1	0.5
Time outdoors (%)	17	15	4	15	4	15	1
Outdoor activity index[b]	1.5	1.5	1.5	1.5	1.5	1.5	1
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover ($hour^{-1}$)	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none	none	none
Contaminated irrigation water source	PADRUN	PADRUN	none	PADRUN	none	PADRUN	none
Irrigation volume (Lm^{-2} day^{-1})	0.25	0.25	0	0.25	0	0.25	0
Contaminated vegetables (% of intake)	50	50	0	50	0	50	0
Contaminated milk (% of intake)	50	50	0	50	0	50	0
Contaminated beef (% of intake)	0	0	0	0	0	0	0
Contaminated poultry (% of intake)	50	50	0	50	0	50	0
Contaminated eggs (%of intake)	100	100	0	100	0	100	0
Contaminated fish (% of intake)	0	0	0	0	0	0	0
Contaminated water source for fish	none	none	none	none	none	none	none
Swimming (% of time)	1	2	0	2	0	0	0
Contaminated water source for swimming	PADRUN	GMR	none	GMR	none	GMR	none
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1	0.05	0.05

[a] Work location is outside the assessment domain: thus no dose is calculated for this location.
[b] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

Table A3. Scenario 3

Gender M
Born 1/1/1951
Home 2 km S
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work 1.5 km SE (WORK3)
Contaminated water sources Paddy s Run (PADRUN), Great Miami River (GMR), and Well 15 (WELL)

	Period 1 1951-1956	**Period 2 1956-1962**		**Period 3 1962-1969**		**Period 4 1969-1989**	
Location	Home	Home	EE	Home	HCR	Home	Work3
Time indoors (%)	83	67	14	67	14	60	15
Indoor activity index[a]	0.5	1	1	1	1	1	1
Time outdoors (%)	17	15	4	15	4	15	10
Outdoor activity index[a]	1.5	1.5	1.5	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover (hour^{-1})	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none[b]	none[b]	none	WELL	none	WELL	WELL
Contaminated irrigation water source	none	none	none	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0	0.5	0
Contaminated vegetables (% of intake)	50	50	0	50	0	50	0
Contaminated milk (% of intake)	100	100	0	100	0	100	0
Contaminated beef (% of intake)	0	0	0	0	0	0	0
Contaminated poultry (% of intake)	50	50	0	50	0	50	0
Contaminated eggs (% of intake)	100	100	0	100	0	100	0
Contaminated fish (% of intake)	0	0	0	0	0	0	0
Contaminated water source for fish	none	none	none	none	none	none	none
Swimming (% of time)	1	2	0	2	0	0	0
Contaminated water source for swimming	PADRUN	GMR	none	GMR	none	GMR	none
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1	0.05	0.1

[a] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).
[b] Groundwater plume did not leave the FMPC site during this period.

Table A4. Scenario 4

Gender F
Born 7/15/1960
Home 4 km ENE
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work N/A-left the assessment domain in 1978
Contaminated water sources Great Miami River (GMR)

	Period 1 1960-1965	**Period 2 1965-1971**		**Period 3 1971-1978**	
Location	Home	Home	EE	Home	HCR
Time indoors (%)	83	67	14	67	14
Indoor activity index[a]	0.5	1	1	1	1
Time outdoors (%)	17	15	4	15	4
Outdoor activity index[a]	1.5	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7
Air turnover ($hour^{-1}$)	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none
Contaminated irrigation water source	none	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0
Contaminated vegetables (% of intake)	10	10	0	10	0
Contaminated milk (% of intake)	10	10	0	10	0
Contaminated beef (% of intake)	0	0	0	0	0
Contaminated poultry (% of intake)	0	0	0	0	0
Contaminated eggs (% of intake)	10	10	0	10	0
Contaminated fish (% of intake)	0	0	0	0	0
Contaminated water source for fish	none	none	none	none	none
Swimming (% of time)	0	2	0	2	0
Contaminated water source for swimming	none	GMR	none	GMR	none
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1

[a] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

TABLE A5. Scenario 5

Gender M
Born 1/1/1951
Home 8 km N
Schools Morgan Elementary (ME) 6.2 km NW
Work In Hamilton, 12 km N (outside assessment domain)
Contaminated water sources None

	Period 1 1951-1956	**Period 2 1956-1962**		**Period 3 1962-1969**		**Period 4 1969-1989**	
Location	Home	Home	ME	Home	HCR	Home	Work[a]
Time indoors (%)	83	67	14	67	14	60	24
Indoor activity index[b]	0.5	1	1	1	1	1	0.5
Time outdoors (%)	17	15	4	15	4	15	1
Outdoor activity index[b]	1.5	1.5	1.5	1.5	1.5	1.5	1
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover ($hour^{-1}$)	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none	none	none
Contaminated irrigation water source	none	none	none	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0	0.5	0
Contaminated vegetables (% of intake)	0	0	0	0	0	0	0
Contaminated milk(% of intake)	0	0	0	0	0	0	0
Contaminated beef (% of intake)	0	0	0	0	0	0	0
Contaminated poultry (% of intake)	0	0	0	0	0	0	0
Contaminated eggs (%of intake)	0	0	0	0	0	0	0
Contaminated fish (% of intake)	0	0	0	0	0	0	0
Contaminated water source for fish	none	none	none	none	none	none	none
Swimming (% of time)	0	0	0	0	0	0	0
Contaminated water source for swimming	none	none	none	none	none	none	0
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1	0.05	0.05

[a] Work location is outside the assessment domain; no dose has been estimated for exposure incurred there.
[b] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

TABLE A6. Scenario 6

Gender F
Born 1/1/1946
Home 3 km ESE
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work Family farm
Contaminated water sources Great Miami River (GMR)

	Period 1 1946-1951	**Period 2 1951-1957**		**Period 3 1957-1964**		**Period 4[a] 1964-1965**	**Period 5 1965-1989**
Location	Home	Home	EE	Home	HCR	Home	Home
Time indoors (%)	80	67	14	67	14	83	83
Indoor activity index[b]	0.5	1	1	1	1	1	1
Time outdoors (%)	20	15	4	15	4	17	17
Outdoor activity index[b]	1.5	1.5	1.5	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover (hour^{-1})	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none	none	none
Contaminated irrigation water source	GMR	GMR	none	GMR	none	GMR	GMR
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0	0.5	0.5
Contaminated vegetables (% of intake)	50	50	0	50	0	50	50
Contaminated milk (% of intake)	100	100	0	100	0	100	100
Contaminated beef (% of intake)	50	50	0	50	0	50	50
Contaminated poultry (% of intake)	50	50	0	50	0	50	50
Contaminated eggs (% of intake)	100	100	0	100	0	100	100
Contaminated fish (% of intake)	50	50	0	50	0	50	50
Contaminated water source for fish	GMR	GMR	none	GMR	none	GMR	GMR
Swimming (% of time)	2	2	0	2	0	0	0
Contaminated water source for swimming	GMR	GMR	none	GMR	none	GMR	GMR
Ingested soil (g day^{-1})	5	0.1	0.1	0.1	0.1	0.05	0.05

[a] Pregnancy
[b] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity.).

TABLE A7. Scenario 7

Gender M
Born 1/1/1951
Home 10 km S (near boundary of assessment domain).
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work In Miamitown near home (WORK7)
Contaminated water sources Paddy s Run (PADRUN), Great Miami River (GMR)

	Period 1 1951-1956	**Period 2 1956-1962**		**Period 3 1962-1969**		**Period 4 1969-1989**	
Location	Home	Home	EE	Home	HCR	Home	Work7
Time indoors (%)	83	67	14	67	14	60	24
Indoor activity index[a]	0.5	1	1	1	1	1	0.5
Time outdoors (%)	17	15	4	15	4	15	1
Outdoor activity index[a]	1.5	1.5	1.5	1.5	1.5	1.5	1
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Air turnover (hour^{-1})	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none	none	none
Contaminated irrigation water source	GMR	GMR	none	GMR	none	PADRUN	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0	0.5	0
Contaminated vegetables (%of intake)	50	50	0	50	0	50	0
Contaminated milk (% of intake)	10	50	0	50	0	50	0
Contaminated beef (% of intake)	0	0	0	0	0	0	0
Contaminated poultry (% of intake)	50	50	0	50	0	50	0
Contaminated eggs (% of intake)	10	100	0	100	0	100	0
Contaminated fish (% of intake)	50	50	0	50	0	50	0
Contaminated water source for fish	GMR	GMR	none	GMR	none	GMR	none
Swimming (% of time)	2	2	0	2	0	0	0
Contaminated water source for swimming	GMR	GMR	none	GMR	none	GMR	none
Ingested soil (g day^{-1})	5	0.1	0	0.1	0	0.05	0

[a] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

TABLE A8. Scenario 8

Gender M
Born 1/1/1990
Home 4 km ENE
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work None before end of exposure period (high school graduation in 1988)
Contaminated water sources Great Miami River (GMR)

	Period 1 1975-1981		**Period 2 1981-1989**	
Location	Home	EE	Home	HCR
Time indoors (%)	67	14	67	14
Indoor activity index[a]	1	1	1	1
Time outdoors (%)	15	4	15	4
Outdoor activity index[a]	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7
Air turnover ($hour^{-1}$)	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none
Contaminated irrigation water source	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0	0.5	0
Contaminated vegetables(% of intake)	10	0	10	0
Contaminated milk (% of intake)	10	0	10	0
Contaminated beef (% of intake)	0	0	0	0
Contaminated poultry (% of intake)	0	0	0	0
Contaminated eggs (% of intake)	10	0	10	0
Contaminated fish (% of intake)	0	0	0	0
Contaminated water source for fish	none	none	none	none
Swimming (% of time)	0	0	0	0
Contaminated water source for swimming	GMR	none	GMR	none
Ingested soil (g day^{-1})	0.1	0	0.1	0

[a] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

TABLE A9. Scenario 9

Gender M
Born 1/1/1951
Home 10 km NE (near boundary of the assessment domain)
Schools Elda Elementary (EE) 4.7 km ENE, Hamilton-Cleaves Middle School, Ross High School (HCR) 6 km NE
Work None; left the area after high school graduation in 1969
Contaminated water sources None

	Period 1 1951-1956	**Period 2 1956-1962**		**Period 3 1962-1969**	
Location	Home	Home	EE	Home	HCR
Time indoors (%)	83	67	14	67	14
Indoor activity index[a]	0.5	1	1	1	1
Time outdoors (%)	17	15	4	15	4
Outdoor activity index[a]	1.5	1.5	1.5	1.5	1.5
Indoor particulate factor	0.7	0.7	0.7	0.7	0.7
Air turnover (hour^{-1})	0.4	0.4	0.4	0.4	0.4
Contaminated drinking water source	none	none	none	none	none
Contaminated irrigation water source	none	none	none	none	none
Irrigation volume (Lm^{-2} day^{-1})	0.5	0.5	0	0.5	0
Contaminated vegetables (% of intake)	0	0	0	0	0
Contaminated milk (% of intake)	0	0	0	0	0
Contaminated beef (% of intake)	0	0	0	0	0
Contaminated poultry (% of intake)	0	0	0	0	0
Contaminated eggs (% of intake)	0	0	0	0	0
Contaminated fish (% of intake)	0	0	0	0	0
Contaminated water source for fish	none	none	none	none	none
Swimming (% of time)	0	0	0	0	0
Contaminated water source for swimming	none	none	none	none	none
Ingested soil (g day^{-1})	5	0.1	0	0.1	0

[a] Varies continuously from 0 (resting) to 1 (light activity) to 2 (vigorous activity).

As part of the Fernald Dosimetry Reconstruction Project, an algorithm was developed that enabled estimation of annual organ-specific doses for individuals residing within 10 kilometers (6.2 miles) of the FMPC facility during its years of operation, 1951 through 1988. Annual estimates of absorbed dose (in Grays) were developed for uranium and radon by exposure pathway (inhalation, ingestion, and direct external exposure) and by low- and high-linear energy transfer (LET) components. Details are available in Appendices I and K of the final Fernald Dosimetry Reconstruction Report. (RAC, 1998). Lung dose equivalent (in Sieverts) was estimated by summing weighted low- and high-LET components of the dose. For this estimate, quality factors of 1 and 20 were used to weight the low- and high-LET components, respectively. Monte Carlo simulation was used to derive probabilistic estimates of lung dose that incorporate the uncertainties in its derivation (e.g. uncertainty in dose conversion factors or atmospheric dispersion model parameters). The values of the 5th, 50th and 95th percentiles of the distribution of the lung dose from uranium and radon for each scenario are presented in Tables A10 – A11 as they appear in the final Fernald Dosimetry Reconstruction Report. (RAC, 1998). (These estimates will vary slightly from those developed by CDC staff and presented later in Appendix C because of sampling in the Monte Carlo analyses; however, both sets of dose estimates are based on the same dose estimation algorithm.) The uncertainty concerning possible values for the lung dose resulting from exposure to radon, as measured by the range from the 5^{th} and 95^{th} percentile, is about twice as large as the corresponding uncertainty measure concerning uranium dose. For these scenarios, most of the dose to the lungs results from exposure to radon and its decay products. Location appears to have the greatest impact on dose with estimates decreasing with increasing distance from the facility.

Table A10. The 5th, 50th and 95th percentile distribution of dose (Sv) to the lungs from uranium[a] for scenarios 1-9.

Scenario	*Percentiles of cumulative dose (Sv)*		
	5th	*50th*	*95th*
1	0.14	0.41	0.99
2	0.049	0.14	0.43
3	0.042	0.13	0.31
4	0.017	0.05	0.13
5	0.011	0.041	0.11
6	0.08	0.22	0.55
7	0.012	0.039	0.092
8	0.003	0.008	0.019
9	0.025	0.076	0.22

a Includes uranium and thorium isotopes and their decay products

Table A11. The 5th, 50th and 95th percentile distribution of dose (Sv) to the lungs[a] from radon[b] for scenarios 1-9.

Scenario	*Percentiles of cumulative dose (Sv)*		
	5th	*50th*	*95th*
1	0.98	3.6	14
2	0.98	3.6	13
3	0.89	2.6	9.8
4	0.4	1.5	7.2
5	0.11	0.42	1.9
6	0.53	2.2	9.2
7	0.12	0.38	1.5
8	0.095	0.42	2.2
9	0.18	0.84	4.8

a Lung dose (Sv) represents the dose to the tracheobronchial epithelium
b Includes radon and its decay products

C.

LIFETIME RISK OF FATAL LUNG CANCER

Lifetime risk of fatal lung cancer was estimated for each of the nine exposure scenarios. Risk was estimated separately for radon and uranium using coefficients of risk per unit dose obtained from different populations: radon-exposed underground miners for radon and atom bomb survivors for uranium. Results were then combined to obtain estimates of risk from all radioactive materials emitted from the facility. Results of these analyses are provided in Tables A12 through A14. For radon, the risk coefficient was derived from the analysis of radon-exposed underground miners published in BEIR IV (1988) (350 excess deaths per 10^6 people per working level month (WLM) in a lifetime). Conversion from risk per WLM to risk per Sievert (Sv) was accomplished by (1) dividing the BEIR IV radon risk coefficient by a value for dose in rads to the bronchial epithelium per unit exposure (0.65 rad/WLM) obtained from NCRP Report No. 78 (NCRP, 1984); (2) converting to gray; and (3) dividing by a factor of 20 (the Relative Biologic Effectiveness (RBE)). The resulting risk coefficient, 0.27×10^{-2} Sv^{-1}, was then multiplied by the FMPC cumulative lung dose estimate to obtain estimates of the lifetime risk of lung cancer mortality. As in the dose estimation process, Monte Carlo techniques were used to generate an uncertainty distribution for risk.

A coefficient for lifetime risk of fatal cancer is not readily available for uranium. Occupational data for uranium millers provides limited information on health effects from internal dose to the lung. Recent evidence on small subgroups of a worker population was not indicative of a dose response for lung cancer risk (Dupree, et al., 1995). Therefore, estimates of lifetime lung cancer risk in the nine scenarios were developed using the lung cancer risk coefficient derived by the ICRP (1991) from atom bomb survivor studies. This risk coefficient, 0.85×10^{-2} Sv^{-1}, is commonly used for radiation protection purposes for the general population. As was done with radon, the risk coefficient for uranium was multiplied by the FMPC dose estimate to obtain estimates of the lifetime risk of lung cancer mortality due to community-based uranium exposures from the FMPC facility. Monte Carlo techniques were used to propagate uncertainties. (The methods used to derive these estimates and the rationale for the methods are explained in greater detail in Appendix S of the final Fernald Dosimetry Reconstruction Report. [RAC, 1998].)

Table A12. The 5th, 50th and 95th percentile distribution of lifetime risk of fatal lung cancer due to exposure to radon[a] from the FMPC for scenarios 1-9.

Scenario	*Lifetime Risk of Fatal Lung Cancer x 10 $^{-2}$*		
	5th	*50th*	*95th*
1	0.096	0.87	8.3
2	0.11	0.94	6.4
3	0.1	0.67	4.7
4	0.055	0.35	2.7
5	0.015	0.099	0.78
6	0.078	0.54	3.7
7	0.015	0.1	0.86
8	0.018	0.1	1.1
9	0.022	0.2	2.3

[a] Includes radon and its decay products

Table A13. The 5th, 50th and 95th percentile distribution of lifetime risk of fatal lung cancer due to exposure to uranium[a] from the FMPC for scenarios 1-9.

Scenario	*Lifetime Risk of Fatal Lung Cancer x 10 $^{-2}$*		
	5th	*50th*	*95th*
1	0.079	0.37	1.8
2	0.025	0.11	0.48
3	0.025	0.11	0.44
4	0.0067	0.038	0.17
5	0.0068	0.03	0.15
6	0.042	0.19	0.83
7	0.0067	0.032	0.13
8	0.0012	0.0058	0.025
9	0.012	0.065	0.27

[a] Includes uranium and thorium isotopes and their decay products

Table A14. The 5th, 50th and 95th percentile distribution of lifetime risk of fatal lung cancer due to exposure to radon[a] and uranium[a] from the FMPC for scenarios 1-9.

Scenario	*Lifetime Risk of Fatal Lung Cancer x 10 $^{-2}$*		
	5th	*50th*	*95th*
1	0.24	1.3	9.6
2	0.18	1	6.8
3	0.15	0.8	4.9
4	0.08	0.41	2.9
5	0.025	0.16	0.9
6	0.15	0.75	4.2
7	0.026	0.15	1
8	0.02	0.11	1.1
9	0.046	0.31	2.5

[a] Includes uranium, thorium and radon isotopes and their decay products

Results of these analyses indicate that radon exposure was the greatest contributor to risk in all nine scenarios. The largest absolute increase in lifetime risk was observed for Scenario 1, an "individual" defined as living 1.7 km northeast of the facility during its entire period of operation. The median value of this risk estimate was 1.3% with a forty-fold range in risk from the 5th to the 95th percentiles. Relative to the background risk of lung cancer mortality (about 6% based on data from the National Cancer Institute's Surveillance, Epidemiology and Ends Results Program [Ries, et al., 1997]), this median estimate represents about a 20% increase in the lifetime risk.

D. IMPACT OF SMOKING ON RISK ESTIMATES

Data from the underground miners studies show that the risk of lung cancer related to radon exposure is modified by cigarette smoking status and that the interaction between these two risk factors is submultiplicative. (Lubin and Steindorf, 1995; Moolgavkar et al, 1993; Hornung et al, 1995). However, smoking status was not considered in the development of the nine exposure scenarios for use in the Fernald Dosimetry Reconstruction Project's dose and risk analyses. Risk estimates by smoking status were obtained in the final Fernald Dosimetry Reconstruction Report by applying adjustment factors of 1.41 for smokers and 0.71 for nonsmokers to the scenario risk estimates presented in Table A14. These adjustment factors derive from work done by Lubin et al (1994) on radon-exposed underground miners.

Lubin et al., applied an "ad hoc modification to [the miners] risk model to account for differences in effects by smoking status" and found about a twofold difference in lung cancer deaths attributable to radon among smokers as compared to nonsmokers. (Lubin and Steindorf, 1995). In the final Fernald Dosimetry Reconstruction Report, it was assumed that dose to the lung as a result of inhalation of alpha-emitting uranium dust particles interacts with smoking in a similar manner to alpha-emitting radon decay products. The results of adjusting the median values (50^{th} percentiles) of the risk estimates due to FMPC exposures for smoking status are presented in Table A15.

Table A15. The median value (50^{th} percentile) of lifetime risk of fatal lung cancer due to exposure to radon[a] and uranium[a] from the FMPC for scenarios 1-9 by smoking status

Scenario	*Median Value of Lifetime Risk of Fatal Lung Cancer x 10^{-2}*	
	Smoker	*Nonsmoker*
1	1.83	0.92
2	1.41	0.71
3	1.13	0.57
4	0.58	0.29
5	0.23	0.11
6	1.06	0.53
7	0.21	0.11
8	0.16	0.078
9	0.44	0.22

[a] Includes uranium, thorium and radon isotopes and their decay products

E. CONCLUSION

The median values (50th percentiles) of the risk estimates due to exposure to all radionuclides (Table A14) range approximately ten-fold across the scenarios: From 0.01% for Scenario 8 to 1.3% for Scenario 1 (unadjusted for smoking status). For all nine scenarios, radon contributes at least two thirds of the risk of lung cancer mortality due to FMPC exposures. As stated previously, location appears to have the greatest impact on dose and thus, risk.

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APPENDIX B

Derivation of the Estimated Number of Lung Cancer Deaths due to Exposures to Radioactive Material Released from the Former Fernald Feed Materials Production Center.

I. Derivation of the Estimated Number of Lung Cancer Deaths

This Appendix provides a description of the methods used to estimate both the number of lung cancer deaths and the percentage increase in lung cancer mortality resulting from environmental exposure to radioactive materials released from the Feed Materials Production Center (FMPC) near Ross, Ohio. These estimates apply only to the population that resided within 6.2 miles (10 kilometers) of the site during the years of plant operations, 1951 through 1988. Our goal is to estimate the lung cancer mortality experience of this population from the period of first potential exposure, 1951, until the last group of potentially exposed citizens has the opportunity to reach the age of 100 years in 2088. To make these mortality projections, we used existing epidemiologic evidence concerning the lung cancer mortality risks associated with exposure to the radionuclides released from the site. This knowledge was applied using a life table approach to model the development of lung cancer and the general survival of a collection of cohorts that comprise this population. Cohorts were defined by year of first exposure to site contaminants, age at that first exposure, location of residence relative to the site, and gender. Within each cohort, the time-specific number of FMPC-related lung cancer deaths was estimated using modeled values for the time- and age-dependent cumulative lung dose, the risk of lung cancer development resulting from that dose, and the size of the population at risk for dying of lung cancer during the time interval being considered. Estimates of the number of FMPC-related lung cancer deaths, the number of lung cancer deaths due to background risk, and the number of deaths due to causes other than lung cancer were estimated within five-year age and time periods. We used these five-year periods instead of modeling the number of deaths for every year from 1951 through 2088 due to limitations on the temporal resolution of some of the data needed to make the estimates and to simplify the computational algorithm. This risk estimation is focused on the probability of lung cancer death potentially resulting from exposure to radon and radon decay products and uranium, thorium and other radionuclides released from the FMPC site. For simplicity, the dose to the lung resulting from these exposures will be referred to as the radon and uranium dose. Based on available epidemiologic evidence, we assumed that an interval, or lag, of five years exists between the time of exposure to radon and the beginning of the time at risk for lung cancer death resulting from that exposure. Similarly, we assumed that a ten-year latency period exits between exposure to uranium and the initiation of the time at risk for lung cancer death resulting from that exposure. The FMPC-related lung cancer mortality experience was modeled for all cohorts in the population from age at first exposure until members of each cohort had the opportunity to reach the age of 100 years. As a result, the

estimates presented in this report reflect, not only the past influence of FMPC exposure on the affected population's risk of lung cancer death, but also the impact of historical releases on the population's future risk.

It must be noted that the true values of the components required to estimate the number of FMPC-related lung cancer deaths, that is, lung dose, the resulting risk, and the size of the population at risk, are uncertain, leading to uncertainty in the estimated cancer burden. In the discussion that follows, therefore, we emphasize the methods and inherent assumptions that were applied to quantify the magnitude of the uncertainty associated with the lung cancer mortality projections.

A. DESCRIPTION OF LIFE TABLE METHODOLOGY

The number of lung cancer deaths associated with exposure to radioactive material released from the FMPC during its years of operation was estimated for the years 1951 through 2088 within subgroups, or cohorts, of the assessment population using a life table approach. These cohort-specific estimates were then summed to provide an estimate of the total number of lung cancer deaths potentially caused by exposure from the FMPC.

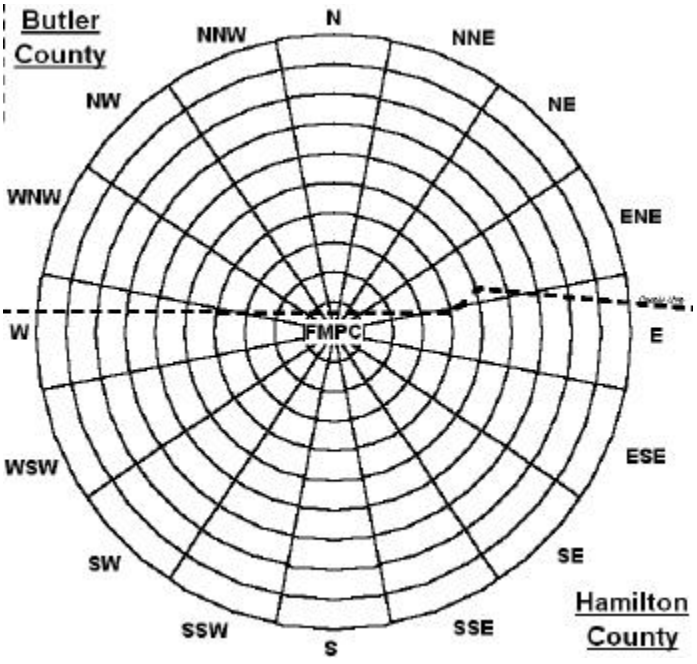


Figure B1 – The location of the 160 cells that comprise the assessment domain

The population for which these estimates apply is defined to be all individuals who resided within 6.2 miles (10 kilometers) of the center of the FMPC production area for some length of time between the years 1951 and 1988. This group is the population for whom dose estimates can be made using the results of the Fernald Dosimetry Reconstruction Project (RAC, 1998). The cohorts into which this population is divided are defined by the year of first exposure to radioactive material released from FMPC, age at which this first exposure occurred, gender, and location of residence within the assessment domain. Location of residence is defined by a collection of geographic cells delineated by the intersection of one-kilometer radius rings centered at the site production area and 16 wind directions. The location of the 160 cells that comprise the assessment domain is illustrated in Figure B1.

Year of first exposure was defined in terms of five-year periods, starting with 1950-1954, which contains the first year of plant operations, and proceeding through to the period 1985-1989, which contains the final year of plant operations. It may be that some amount of radioactive material continued to be released from the FMPC after cessation of plant operations in 1988 but the magnitude of post-1988 releases of uranium is likely small relative to releases that occurred during the production years. Moreover, corrective measures were installed to the K-65 silos, the source of the radon releases, in 1979 that significantly reduced the likelihood of offsite exposure to radon decay products in later years. As a result, lung cancer mortality risk potentially resulting from exposure to radioactive material released from the FMPC site after 1988 is not addressed in this evaluation.

Age at first exposure and in the years subsequent to first exposure is also defined in five-year intervals beginning at the age at which first exposure occurred and continuing until attainment of age 100 years. Therefore, for individuals first exposed as infants, the age intervals considered in the risk estimation are: 0-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85-89, 90-94, and 95-99 years.

To illustrate the procedure used to estimate the number of FMPC-related lung cancer deaths, we will consider each of the time periods of first exposure in succession. The first such period of potential exposure is 1950-1954. Given an estimate of the number of persons residing within the assessment domain during this interval, we estimated the number of persons within each of the cohorts comprising this group by subdividing those first exposed in 1950-1954 by location of residence, age, and gender. The methods by which we made these subdivisions will be discussed in detail later in this Appendix. For now, suppose that n_{ij1950} is the estimated number

of, for example, males in age class *j* in the time period 1950-1954 who were first exposed during that interval and who resided in cell *i*. Because we assume a five-year lag between exposure and initiation of time at risk due to radon exposure and a ten-year lag for uranium exposures, we estimate no FMPC-related lung cancer deaths among this group during the time interval 1950-1954. However, some of these men could die from lung cancer due to background lung cancer risk. By background risk, we are referring to the probability of lung cancer death among the group that exists in the absence of FMPC-related exposures. In addition, some of these men likely died during the time interval due to causes other than lung cancer. Let LC_{ij}^{0} be the estimated background number of lung cancer deaths among the n_{ij1950} males during the interval 1950-1954. In addition, let D_{ij}^{*} be the estimated number of deaths in this group due to causes other than lung cancer during the interval. Again, the specific methods used to estimate these numbers of deaths will be discussed later. However, given these estimates, the number of males who were first exposed in the period 1950-1954 and survived until 1955 can be estimated as

$$n_{i(j+5)1950}^{1955} = n_{ij1950} - LC_{ij}^{0} - D_{ij}^{*} \quad .$$

During the next time interval, 1955-1959, the assumed five-year lag between exposure to radon and initiation of time at risk implies that the males who survived until 1955 are now at risk of lung cancer death as a result of FMPC-related radon exposures received from 1950 to 1954. Using methods outlined later in this Appendix, suppose we develop an estimate for the number of FMPC-related lung cancer deaths that could occur among the $n_{i(j+5)1950}^{1955}$ males during the interval 1955-1959 and we designate this estimate as $LC_{i(j+5)}^{F}$. If $LC_{i(j+5)}^{0}$ is the background number of lung cancer deaths and $D_{i(j+5)}^{*}$ is the number of deaths due to causes other than lung cancer estimated to occur in this group during the interval 1955-1959, then the number estimated to survive until the 1960-1964 period is given by

$$n_{i(j+10)1950}^{1960} = n_{i(j+5)1950}^{1955} - LC_{i(j+5)}^{F} - LC_{i(j+5)}^{0} - D_{i(j+5)}^{*} \quad .$$

The estimated number of FMPC-related lung cancers in the following five-year period, 1960-1964, will now reflect the radon exposure incurred by this group from 1950 through 1959 and, under the assumed ten-year lag for uranium exposure, the lung dose resulting from exposure to uranium during the period 1950-1954. This process of estimating the number of FMPC-related lung cancer deaths, the number of background lung cancer deaths, and the number of deaths due to other causes is continued across five-year periods until that time period in which the age of the group reaches 100 years. For example, if the group being modeled was in the 0-4 year age class in the 1950-1954 period, then their survival is modeled from that initial period until the year 2054. The sum of the estimated numbers of FMPC-related lung cancer deaths in each time interval from age at first exposure through age 100 provides an estimate of the total number of FMPC-related lung cancer deaths among the n_{ij1950} males first exposed at age j in cell *i* between 1950 and 1954. Using an identical approach, the total number of FMPC-related lung cancers among females who were first exposed in 1950-1954 at age *j* in cell *i* can also be estimated. In addition, the total number of FMPC-related lung cancer deaths among the entire portion of the population that was first exposed in 1950-1954 can be estimated by combining similar cohort-specific estimates across genders, age classes, and geographic cells.

Now consider those individuals who were first exposed to FMPC-related radioactive material in the time period 1955-1959. This group is comprised of two classes of persons: those who were born to the survivors of the group first exposed in 1950-1954 period, and those who immigrate into the cell during the period. For the sake of illustration, we will consider only those who attained membership in this cohort through birth. Our attempts to model immigration into and emigration out of cells will be described in detail later in this Appendix. Using the number of individuals in a given cell who were first exposed in 1950-1954 and who are predicted to survive into the period 1955-1959 and appropriate population-based birth rates, we can estimate the number of births occurring in any cell during the period 1955-1959. Still ignoring immigration, this group of births comprises the cohort considered to have been first exposed in 1955-1959. The FMPC-related lung cancer mortality experience of this group is then modeled in the same manner as was used for those first exposed in 1950-1954. This group is followed in the life table process, however, until the year 2059 to allow all members the opportunity to reach an age of 100 years. By combining the estimated total number of FMPC-related lung cancer deaths among those born in the 1955-1959 period with the estimated number of such deaths among those first exposed in 1950-1954, we derived an estimate for the total number of FMPC-related lung

cancer deaths among residents of the assessment domain who were first exposed between 1950 and 1959.

This process was repeated to estimate the total number of FMPC-related lung cancer deaths among those first exposed in the time periods: 1960-1964, 1965-1969, 1970-1974, 1975-1979, 1980-1984, and 1985-1989. By combining these values across time period of first exposure groups, we derived our estimate for the total number of lung cancer deaths in the population resulting from exposure to radioactive material released from the FMPC facility during its operational years.

The process we describe for estimating the potential number of FMPC-related lung cancer deaths is dependent on estimation of the time and age period-specific lung dose, lung cancer mortality risk resulting from that dose, and the number of individuals experiencing that risk. The estimators we used to derive these components are uncertain and we attempted to propagate these uncertainties through to the estimates of FMPC-related lung cancer mortality using a Monte Carlo process (Vose, 1996). In this Monte Carlo approach, the uncertainty associated with the components of the risk estimation process was modeled to produce a collection of plausible values for these parameters. The life table approach described above was then repeated many times, each time with a sampled set of components drawn from the modeled collection of plausible values. The end result of the Monte Carlo simulation process is a probabilistic representation of the possible values for the total number of FMPC-related lung cancers among the population that resided within the assessment domain. The range of values represented by this collection reflects the combined uncertainty of the constituent components. In the remainder of this Appendix, we describe the methods and assumptions used to estimate the dose, risk, and population components needed to implement the life table estimator. In addition, we outline our efforts to quantify and propagate the uncertainty associated with these components through to the resulting estimates of the number of FMPC-related lung cancer deaths.

B.

ESTIMATION OF UNCERTAIN VALUES FOR LUNG DOSE EQUIVALENT

The values for the dose equivalent to lung, which we will refer to as lung dose, were estimated for our risk projection using the methodologies developed in the Fernald Dosimetry Reconstruction Project (RAC, 1998). These estimates were produced in the dose reconstruction by developing a series of mathematical models to mimic the mechanism by which radioactive materials were released from the site, the environmental transport of this material within the assessment domain, and the lung dose resulting from these exposures. The results of this modeling effort indicated that the most prevalent offsite exposures were due to releases of radon progeny from the K-65 silos in which radium-bearing waste was stored. To a lesser extent, the population in the assessment domain was also exposed to airborne particles contaminated with uranium and thorium as a result of actual plant operations. Estimates of lung dose equivalent, in sieverts, were produced separately for that resulting from exposure to radon and for the dose resulting from exposure to uranium and thorium. For convenience, we will refer to these doses as radon dose and uranium dose respectively.

Using the Fernald Dosimetry Reconstruction Project algorithm, we can estimate the time period-specific average cumulative lung doses for any of the cohorts used in the life table estimation. To derive these estimates, we combined the doses produced by the Fernald Dosimetry Reconstruction Project algorithm into a series of tables, which we refer to as nominal dose estimate tables, such that a separate table was produced for each gender within each of the cells of the assessment domain. An example of one of these nominal dose estimate tables is shown in Table B1. This particular table contains radon dose estimates for females who resided in the cell centered 4.5 kilometers northeast of the FMPC site.

Table B1. Nominal[1] five-year cumulative radon-related lung dose equivalent (sieverts) for females who resided 4 Kilometers northeast of the FMPC site during its years of operation (1951 through 1988) by age class and time period of exposure.

Years	Age group							
	0-4	***5-9***	***10-14***	***15-19***	***20-24***	***25-29***	***30-34***	***35-39*** [2]
1950-1954	0.11	0.15	0.14	0.10	0.08	0.08	0.08	0.08
1955-1959	0.24	0.34	0.36	0.26	0.20	0.20	0.20	0.20
1960-1964	0.28	0.39	0.42	0.31	0.23	0.23	0.23	0.23
1965-1969	0.28	0.39	0.42	0.31	0.23	0.23	0.23	0.23
1970-1974	0.28	0.39	0.42	0.31	0.23	0.23	0.23	0.23
1975-1979	0.25	0.35	0.39	0.28	0.21	0.21	0.21	0.21
1980-1984	0.04	0.05	0.06	0.04	0.03	0.03	0.03	0.03
1985-1989	0.02	0.02	0.03	0.02	0.01	0.01	0.01	0.01

1 The nominal dose estimates were produced by setting all uncertain components of the dose estimation algorithm to their median values.

2 Nominal lung dose estimates for those over 40 years of age are equal to the estimates in the 35 to 39 year old age class.

The rows in the nominal dose table correspond to the time periods of potential exposure defined in the life table analysis, while the columns reflect the age class of the individuals being exposed. Thus, if $\hat{d}^{r}_{ijk}$ represents the estimated cumulative five-year radon dose to a female in age class *j* who resided in cell *i* during time period *k*, then the cumulative dose estimate for any female who resided in this cell during the years of plant operations can be obtained by specifying the year and age of first exposure and summing the appropriate elements of the nominal dose estimate table. Because there are 160 geographic cells in the assessment domain and a nominal dose estimate table is calculated for each gender, 320 nominal dose estimate tables were produced using the dose reconstruction algorithm reflecting radon exposures and 320 tables for uranium dose.

The magnitude of the estimates of lung dose comprising these tables depends on the location of the cell, the time period of exposure, and the age and gender of the individuals being considered. In addition, other variables such as the percentage of time spent outdoors, the average level of activity, and the percentage of consumed vegetables that may be contaminated were also included in the dose estimation process. Because our goal was to develop dose estimates for the entire population that resided in the assessment domain, we needed to examine the impact on the dose estimates of fixing these additional variables to a standard set of values for the entire population. The results of a sensitivity analysis to examine the impact on the dose estimates of our use of these standard assumptions are presented in Appendix C of this report. In general, after accounting for time period of exposure, location of residence, age and gender, our assumptions concerning the lifestyle characteristics of the assessment population had negligible impact on both the estimated radon and uranium lung doses. Therefore, by accounting for time period of exposure, age at which this first exposure occurred, place of residence, and gender, we were able to produce a collection of nominal dose estimate tables that are likely to be reflective of the exposure experience of the entire population in the assessment domain.

A great deal of emphasis was placed on modeling the uncertainty associated with the dose estimates in the dose reconstruction project. This modeling was accomplished using a complex Monte Carlo algorithm that produced a range of possible lung doses reflecting uncertainty in the components of the dose estimation algorithm. The dose estimates contained in what we call the nominal dose estimate tables correspond to the similarly named nominal dose estimates from the dose reconstruction project (RAC, 1998). These nominal values correspond to the single dose estimate that is produced when each uncertain input parameter in the dose estimation process is set equal to its median value. In general, the nominal estimates provided a reasonably close approximation to the median of the collection of possible doses produced by the Monte Carlo algorithm. Due to computational constraints resulting from both the structure of the dose reconstruction computer software and the number of cohorts for which we required collections of dose estimates, we could not simply extend the dose reconstruction Monte Carlo process through to the production of multiple realizations of lung cancer deaths for the entire population that may have resided within the assessment domain. As an alternative, we chose to model the uncertainty of lung dose about the nominal estimates.

We first describe our attempt to model the uncertainty associated with the radon component of the lung dose. We assumed that the uncertainty in the true value of the time period- and age

class-specific five-year cumulative radon dose, such as those shown in Table B1, could be modeled as

$$d_{ijk}^{r} = \hat{d}_{ijk}^{r} * g \quad . \qquad \text{(B1)}$$

In equation B1, d_{ijk}^{r} is the true five-year radon dose, $\hat{d}_{ijk}^{r}$ is the nominal estimated dose from the nominal dose table and g is a common uncertainty factor reflecting the combination of uncertainty and variability separating the true and estimated dose. By assuming a constant value for g across cells, age classes, and time periods, we can model the correlation structure among the uncertainties of the entire collection of doses. For example, suppose the true radon lung dose for males age 30-34 in a specified cell during the interval 1960-1964 is greater than the nominal value due to the source term, that is the amount of radium contained in the K-65 silos being greater than the assumed value. In other words, the value of g in equation B1 is greater than one. It is logical to assume that the underestimation of the true source term will result in the nominal radon dose estimate for the next time interval, 1965-1969, also being lower than the true dose as would be the nominal radon dose estimate for males aged 35-39 years and for females of similar age. In addition, uncertainty concerning the concentration of radon exposure in the entire collection of geographic cells within the assessment domain is simultaneously affected by the uncertainty in the source term and environmental transport components of the dose reconstruction algorithm. As a result, if the true value of this exposure and the resulting true radon dose in a given cell is greater than the nominal value, then the true exposure and dose in the other cells of the assessment domain are also likely to be greater than the corresponding nominal estimates.

We used a Monte Carlo approach based on equation B1 to attempt to capture this correlation structure among the uncertainties associated with the possible true values of radon dose. For each realization of the Monte Carlo process, a possible value for g was generated under the assumption that this common dose uncertainty factor follows a lognormal distribution such that

$$\ln(g) \sim N(0,0.9) \quad . \qquad \text{(B2)}$$

The assumed value for the variance of the log transformed uncertainty factor, 0.9, was derived based on the uncertainty distributions of the lifetime radon dose estimates developed for nine

representative scenarios in the Fernald Dosimetry Reconstruction Project (see Table A11 in Appendix A of this report). The selected value for the variance implies roughly a factor of 5 between the median and the 5th and 95th percentiles of the distribution for g given in equation B2. This range for the 90% credibility interval is consistent with the corresponding uncertainty range estimated for lifetime radon doses of the nine scenarios.

Given the sampled value for g , a realization for the full collection of five-year doses for all cohorts in the assessment population was generated using equation B1. This approach implies that the uncertainty concerning a given five-year cumulative radon dose can be summarize using a lognormal distribution with expected value equal to the nominal dose estimate and variance determined by the sampled value for g . This process of sampling a common value for the radon dose uncertainty factor based on equation B2 and then generating a collection of possible five-year radon doses for the entire assessment domain was repeated at each step in the Monte Carlo process. Within each step, the collection of possible radon doses was used to estimate the number of lung cancer deaths potentially resulting from these exposures in the assessment population. The methods use to make these estimates are described in the next section of this Appendix. By using this strategy, the correlation inherent in the uncertainty associated with true radon dose can be included in the estimation of the uncertainty associated with the resulting lung cancer mortality risk.

A collection of 320 nominal dose estimate tables was also produced to estimate the lung dose resulting from FMPC-related uranium exposure. An identical strategy for modeling the uncertainty in the true uranium lung dose was used as was employed in modeling the uncertainty in the radon doses. For uranium dose, however, the common uncertainty factor used in each realization of the Monte Carlo process, d , was generated from a lognormal distribution such that

$$\ln(d) \sim N(0,0.45) \quad . \qquad \text{(B3)}$$

The value used for the variance of the log transformed uranium dose uncertainty factor was derived based on the estimated uncertainty in the lifetime uranium dose estimates of the nine scenarios used in the Fernald Dosimetry Reconstruction Project (see Table A10 in Appendix A of this report). The assumed value for this variance , 0.45, implies a factor of approximately 3 between the median and the 5th and 95th percentiles of the assumed distribution of d . This 90% credibility interval is, again, consistent with the collection of uncertainty ranges estimated for the

nine scenarios. For each stage in the Monte Carlo process, a common value for d was generated using equation B3. Given the sampled uncertainty factor, a possible collection of five-year uranium doses for all cohorts in the assessment population was generated using the model

$$d_{ijk}^{u} = \hat{d}_{ijk}^{u} * d \qquad \text{(B4)}$$

where d_{ijk}^{u} is the generated value for the true five-year uranium dose in cell *i*, age class *j*, and time period *k* and $\hat{d}_{ijk}^{u}$ is the corresponding nominal uranium dose estimate from the nominal dose estimate table.

C. ESTIMATES OF LUNG CANCER RISK DUE TO FMPC-RELATED EXPOSURES

Once a collection of time- and age-specific lung dose estimates were derived, the next step in implementing the life table approach was estimation of the corresponding time- and age-specific lung cancer mortality rate. Derivation of these rate estimates requires specification of a model to represent the likely increase in the expected lung cancer mortality rate per unit dose to the lung. We considered candidate models to estimate this increase separately for radon and uranium dose. We first consider estimation of time- and age-dependent lung cancer mortality resulting from exposure to radon progeny. The primary sources of information on the association between lung cancer risk and radon exposure are the results of a series of epidemiologic studies of underground miners who were occupationally exposed to radon and its decay products (NAS, 1998; Lubin et al., 1994). Despite the fact that the exposure levels in these studies are substantially greater than those estimated for the assessment population, the models relating lung cancer mortality rate to the level of radon exposure derived from these analyses are likely to be the most applicable to estimating the lung cancer mortality risk resulting from FMPC-related radon exposures. In fact, the mortality rate models resulting from the analysis of the miners' data have been used repeatedly to estimate the impact of indoor exposure to naturally occurring radon (NAS, 1988; Lubin et al, 1997; Lubin et al., 1995, Steindorf et al., 1995; Puskin, 1992). In addition, the observed lung cancer mortality rate in the subset of miners who experienced the lowest levels of radon exposure was found to be consistent with the estimated rate for this group based on the model derived from the full exposure range cohort (Lubin et al., 1995). Another rationale for using the underground miner rate models in our risk estimation is the

general agreement between mortality trends implied by this model and the observed patterns of lung cancer mortality in studies of populations experiencing indoor exposure to naturally occurring radon (Boice, 1997).

Using the most recent analyses of the combined miner cohorts available (NAS, 1998; Lubin et al., 1997), we estimated the time- and age-dependent lung cancer mortality rate resulting from exposure to radon decay products released from the FMPC site using the model

$$r^r = r^0 * b * l * f * g * (d_{5-14} + q_{15-24} d_{15-24} + q_{25+} d_{25+}) . \qquad \text{(B5)}$$

In equation B5, r^r is the estimated FMPC-related lung cancer mortality rate during the specified time period, r^0 is the age, gender, time-period specific background mortality rate for lung cancer mortality, b is the increase in the mortality rate per unit exposure to the lung, l is an adjustment to this increase based on smoking status, f is an adjustment to the rate reflecting attained age, g is an adjustment associated with the duration of exposure and q is an adjustment for time since exposure. We define the background mortality rate, r^0, as the lung cancer mortality rate we would expect in a group of individuals in a specified gender and age class during the time interval being considered in the absence of FMPC-related exposures. The term $(d_{5-14} + q_{15-24} d_{15-24} + q_{25+} d_{25+})$ in equation B5 represents the subdivision of the cumulative radon dose during the time period in which the mortality rate is being estimated into relevant periods of exposure. In the model, d_{5-14} reflects that portion of the cumulative dose received 5 to 14 years prior to the time period being considered, d_{15-24} is the portion of the cumulative dose delivered 15 to 24 years prior, and d_{25+} is that portion of the cumulative dose accrued 25 or more years prior to the time period in which the rate is being estimated. Table B2 contains the estimated values of the parameters of equation B5 resulting from the most recent analysis of the miner cohorts (NAS, 1998).

The particular rate model we use is referred to as the Exposure-Age-Duration model in the National Academy of Sciences' BEIR VI report (NAS, 1998). The committee that prepared the BEIR VI report actually had two preferred models for estimating the increase in lung cancer mortality rate resulting from radon exposure. In addition to the Exposure-Age-Duration model in equation B5, the committee also used a model referred to as the Exposure-Age-Concentration model. The difference between the committee's preferred models depends on

how the estimated increase in the lung cancer mortality rate is adjusted to reflect the rate at which the radon exposure occurs. In the Exposure-Age-Duration model, this adjustment is made based on the duration of the period of exposure while in the Exposure-Age-Concentration model the adjustment reflects the actual average exposure rate to radon. Because estimates of these exposure rates for the assessment population were not readily obtainable from the dose reconstruction algorithm, we selected the Exposure-Age-Duration model for use in this assessment. The result of this adjustment is an increase in the estimated rate of lung cancer mortality for those with longer periods of exposure. In our risk estimation, duration of exposure is defined as the number of years separating the specified year of first exposure and 1988, the final year of plant operations. In addition to adjustment for duration for exposure, the mortality rate model also implies a decrease in the impact of lung dose depending on the time lapse between an individual's current age and the age at which the exposure occurred.

Before proceeding to a description of our attempts to model the uncertainty associated with the mortality rate estimates derived from equation B5, we need to specifically define the actual portions of the cumulative dose that were assigned to $d_{5-14}, d_{15-24},$ and d_{25+}. Because we are considering five-year periods of time and age at risk, we needed to define a summary value for cumulative radon dose to represent each period. We considered individuals in a cohort to be at risk for the entire five-year period at the cumulative dose associated with the last year in the period. While a more appropriate indicator of representative cumulative dose during the interval might be the cumulative dose at the midpoint of the time interval, our use of the cumulative dose accrued by the last year in the interval as the period representative dose significantly simplified accounting for the assumed five-year lag between time of exposure and initiation of the resultant time at risk. In addition, due to the continuous nature of the exposure, the values of d_{5-14}, d_{15-24} and d_{25+} would likely vary little between the two candidate definitions of the representative cumulative dose for the period.

The range of uncertainty associated with using the estimated increase in the rate per unit exposure obtained from analysis of the miner's studies has been estimated to correspond to a factor of about 2 to 2.5 (Lubin et al. 1994, Lubin et al. 1995, Puskin 1992). We chose to favor the higher end of this range by assuming that a reasonable factor for this uncertainty is 2.5. In addition, we assumed that the distribution of possible true values for b is approximately lognormal with median $\hat{b}$ where $\hat{b}$ is the estimated value of the increase in the rate per unit of exposure parameter given in Table B2. We modeled the uncertainty associated with b by

assuming that the range of values that results when $\hat{b}$ = 0.0055 is multiplied and divided by the uncertainty factor of 2.5 corresponds to a 90% credibility interval for the true value of b. Under these assumptions the uncertainty distribution for b used in our risk estimation is given by

$$\ln(b) \sim N(\ln(0.0055), 0.3). \qquad \text{(B6)}$$

Table B2: Parameter estimates for model used to estimate the increase in the lung cancer mortality rate[1] resulting from FMPC-related exposure to radon and its decay products

Parameter	***Estimated Value*** [2]
Increase per unit exposure b	0.0055
Adjustment for Attained Age f	1, Age < 55 0.53, 55 ≤ Age < 65 0.28, 65 ≤ Age < 75 0.13, 75 ≤ Age
Adjustment for Duration of Exposure g	1, Duration < 5 2.78, 5 ≤ Duration < 15 4.42, 15 ≤ Duration < 25 6.62, 25 ≤ Duration < 35 10.2, 35 ≤ Duration
Adjustment for Time Since Exposure (15 to 24 Years Since Exposure) q_{15-24}	0.72
Adjustment for Time Since Exposure (25 or More Years Since Exposure) q_{25+}	0.44
Adjustment for Smoking History l	2.0, Never Smoker 0.9, Ever smoker

[1] Equation B5 in this Appendix

[2] Parameter estimates were derived in the most recently published analysis of the lung cancer mortality experience of a collection of underground miner cohorts (NAS,1998)

Notice that we have not yet specifically defined the dose units associated with d_{5-14}, d_{15-24}, and d_{25+}. This omission reflects the fact that the metric of exposure in the miner's studies was working level months (WLM) as opposed to the dose metric of lung dose equivalent in sieverts

that is produced by the dose estimation algorithm. Therefore, to use this model in our risk estimation, we must convert the possible values for b, obtained by sampling from the distribution in equation B6, from the increase in the mortality rate per WLM to the increase in the rate per sievert. The National Council on Radiation Protection (NCRP) has suggested conversion factors from WLM to rads of 0.7 for males and 0.6 for females for domestic exposure versus 0.5 for miners, a correction made partly for differences in breathing rate (NCRP, 1984). Using these values, we converted the increase per WLM to increase per sievert using the equation

$$\frac{Increase}{Sievert} = \frac{Increase}{WLM} * [g^{-1} \frac{WLM}{rad} * RBE^{-1} \frac{rads}{rem} * 100 \frac{rems}{sievert}]$$

where g equals 0.7 for males and 0.6 for females and *RBE* is the relative biological effectiveness for high versus low LET radionuclides. Using a value of 20 for the *RBE*, the conversion factor described above would be 7.1 for males and 8.3 for females. To capture some portion of the uncertainty in making the conversion from increase in rate per unit exposure to increase in rate per unit dose, we define an uncertainty range for the *RBE* for lung cancer, as defined by a 90% credibility interval, to be [10,40] (RAC, 1998). In addition, we assume the distribution of possible true values of *RBE* is lognormal with median value 20. Using these assumptions, candidate values for the conversion factor, which we will call *CF*, that translate the increase in the lung cancer mortality rate per WLM to the increase per sievert can be generated by assuming that

$$\ln(CF_{Males}) \sim N(\ln(7.1), 0.175) \qquad \text{(B7)}$$

for males and

$$\ln(CF_{Females}) \sim N(\ln(8.3), 0.175) \qquad \text{(B8)}$$

for females.

Therefore, for each realization of the Monte Carlo process used to reflect the uncertainty in the estimated number of FMPC-related lung cancer deaths, the uncertainty associated with the increase in the mortality rate per sievert of lung dose was generated in two stages. First a possible value for the increase in the rate per WLM, b in equation B5, was generated from the lognormal distribution of equation B6. This generated value was then converted from the increase in the rate per WLM to the increase per sievert using a possible value for the conversion factor generated from equation B7 for males and B8 for females.

When the results of the epidemiologic investigations of miners have been used to estimate the increase in lung cancer mortality rates due to indoor exposure to naturally occurring radon, the increase in the rate per WLM is multiplied by a factor, sometimes called the *K* factor, to represent differences in lung dose that would result from comparable levels of radon exposure in houses as opposed to mines. The methods used to estimate lung dose equivalent in the Fernald Dosimetry Reconstruction Report dose reconstruction were based on methods that accounted for the majority of differences in home versus mine exposure environments (RAC, 1998). As a result, we did not use a separate *K* factor type adjustment in our estimation of FMPC-related lung cancer mortality.

Repeated analysis of the underground miner experience has indicated that the impact on lung cancer risk resulting from exposure to radon progeny was elevated among miners who smoked as opposed to miners who were non-smokers. The form of the joint impact of smoking and radon exposure on the relative risk of lung cancer appears from these analyses to be intermittent between additive and multiplicative. To model this association, the National Academy of Sciences (NAS, 1998) has suggested adjusting the estimated increase in lung cancer mortality rate per unit exposure to radon dependent on smoking status as defined by being an ever versus never smoker. An individual is considered an ever smoker if he or she has smoked 100 or more cigarettes. The adjustment for smoking used in our risk estimation, the parameter l in equation B5, reflects the submultiplicative association between smoking status and radon-related lung cancer risk suggested in the BEIR VI report.

The final component needed to estimate the FMPC-related lung cancer mortality rate in a given time period, is the background lung cancer mortality rate experienced by the cohort during that interval, r^0 in equation B5. The average annual lung cancer mortality rates occurring in the five year intervals corresponding to the time-periods used in the risk estimation were obtained for the state of Ohio from the CDC's National Center for Health Statistics' Detailed Mortality Tape

Table B3. Average annual age-specific mortality rates[1] for lung cancer [2] and all causes other than lung cancer for the State of Ohio by gender. 1962-1990.

Years	Age group	Lung cancer		All causes other than lung cancer	
		Males	Females	Males	Females
1962-1964	0-4	0.2	0.2	1045.8	807.6
	5-9	0	0	93.3	62.9
	10-14	0	0	95.1	49.2
	15-19	0.1	0.1	193.5	81.3
	20-24	0.6	0	269.6	104.4
	25-29	0.2	0.5	227.9	134.3
	30-34	4.5	1.2	274.4	172.9
	35-39	13.1	4.3	436.2	284.3
	40-44	32.9	11.3	685.6	422.7
	45-49	66.6	11.6	1076.2	627.2
	50-54	139.9	23.7	1803.8	969.63
	55-59	216.8	25.5	2735.5	1417.58
	60-64	305.0	33.5	3962.5	2156.76
	65-69	378.1	40.9	6182.5	3460.24
	70-74	404.2	53.5	9674.8	5898.8
	75-79	376.5	55.4	14997.6	10208.8
	80-84	267.4	58.3	23642.7	17787.5
	85+	209.1	88.8	39856.8	32913.9

[1] Rates are expressed in deaths per 100,000 population

[2] Includes the following ICD codes: 1962-67:163; 1969-78: 162.1-162.2; 1979-90: 162.1-162.9

Table B3, continued

Years	Age group	Lung cancer		All causes other than lung cancer	
		Males	Females	Males	Females
1965-1969	0-4	0.2	0.2	998.2	752.1
	5-9	0	0	102.2	68.0
	10-14	0	0	90.1	56.5
	15-19	0.1	0.1	264.9	107.9
	20-24	0.3	0.4	339.8	120.0
	25-29	1.0	0.1	303.3	139.1
	30-34	3.5	1.8	333.0	190.0
	35-39	18.2	7.3	498.4	309.7
	40-44	43.4	13.9	814.8	494.6
	45-49	81.7	25.5	1241.0	726.6
	50-54	158.8	38.5	1996.4	1077.0
	55-59	268.0	46.5	3112.2	1586.2
	60-64	412.9	54.7	4622.5	2361.2
	65-69	507.1	63.4	6878.4	3817.3
	70-74	599.2	71.1	11062.9	6283.8
	75-79	579.1	73.8	16748.5	10835.1
	80-84	439.8	74.2	25454.9	18043.0
	85+	314.0	97.2	41237.9	33893.4
1970-1974	0-4	0.04	0.1	454.5	356.4
	5-9	0.04	0	45.6	33.1
	10-14	0.2	0	46.3	28.4
	15-19	0.2	0.04	141.3	53.3
	20-24	0.05	0.1	199.4	68.0
	25-29	0.2	0.2	172.0	77.1
	30-34	1.8	1.3	187.9	100.5
	35-39	7.2	3.1	262.9	156.0
	40-44	24.5	9.9	409.2	246.6
	45-49	54.2	19.5	647.7	379.9
	50-54	99.4	31.5	1009.5	567.6
	55-59	174.5	42.7	1627.0	848.1
	60-64	255.9	52.0	2518.1	1315.3
	65-69	350.9	52.3	3820.2	2043.9
	70-74	388.5	50.2	5816.4	3273.9
	75-79	379.8	50.7	8702.6	5443.5
	80-84	351.1	59.2	12811.8	8750.0
	85+	230.2	51.9	20906.5	16802.1

[1] Rates are expressed in deaths per 100,000 population

[2] Includes the following ICD codes: 1962-67:163; 1969-78: 162.1-162.2; 1979-90: 162.1-162.9

Table B3, continued

Years	Age group	Lung cancer		All causes other than lung cancer	
		Males	Females	Males	Females
1975-1979	0-4	0.1	0.1	375.9	294.4
	5-9	0	0	36.3	26.2
	10-14	0	0.04	40.4	23.3
	15-19	0.04	0.04	131.3	51.7
	20-24	0.1	0.1	192.3	58.9
	25-29	0.2	0.4	164.8	66.0
	30-34	1.9	1.2	166.6	85.9
	35-39	7.3	4.6	225.5	127.5
	40-44	19.3	10.4	346.6	206.2
	45-49	54.9	25.0	566.5	315.8
	50-54	115.1	39.9	911.5	505.6
	55-59	181.4	59.8	1386.3	763.8
	60-64	287.9	74.3	2215.5	1186.5
	65-69	389.9	87.2	3449.3	1821.8
	70-74	457.6	79.0	5164.9	2826.2
	75-79	480.1	77.7	7844.0	4718.5
	80-84	433.6	73.1	11617.0	7612.2
	85+	332.4	76.0	19114.2	14929.7
1980-1984	0-4	0.05	0.1	313.2	249.1
	5-9	0	0	31.6	23.8
	10-14	0	0	35.2	19.3
	15-19	0.04	0.04	115.1	47.3
	20-24	0.1	0.1	160.2	54.1
	25-29	0.5	0.2	159.2	57.3
	30-34	1.1	0.7	158.0	77.9
	35-39	5.5	3.9	196.8	109.4
	40-44	19.2	10.2	293.3	171.5
	45-49	47.9	26.5	486.0	288.2
	50-54	112.3	48.7	789.0	451.9
	55-59	205.8	73.1	1247.2	717.7
	60-64	295.0	103.6	1943.5	1121.7
	65-69	417.7	125.9	3090.1	1715.0
	70-74	537.9	125.5	4843.7	2698.9
	75-79	586.9	109.8	7306.5	4255.8
	80-84	518.5	102.1	10933.4	7166.3
	85+	447.9	91.0	18632.2	14450.0

[1] Rates are expressed in deaths per 100,000 population

[2] Includes the following ICD codes: 1962-67:163; 1969-78: 162.1-162.2; 1979-90: 162.1-162.9

Table B3, continued

Years	Age group	Lung cancer		All causes other than lung cancer	
		Males	Females	Males	Females
1985-1989	0-4	0.1	0.1	271.9	207.5
	5-9	0	0	26.7	19.1
	10-14	0	0.1	31.0	19.3
	15-19	0.1	0.1	103.0	43.1
	20-24	0.1	0.2	146.2	51.3
	25-29	0.4	0.3	152.9	59.9
	30-34	1.7	0.6	161.4	73.8
	35-39	4.6	2.9	202.0	102.7
	40-44	14.2	8.6	274.8	154.4
	45-49	41.2	25.9	407.1	260.5
	50-54	97.5	48.8	689.3	421.9
	55-59	190.3	87.1	1099.6	658.0
	60-64	311.0	128.8	1830.1	1073.4
	65-69	422.3	156.3	2796.2	1635.9
	70-74	534.7	182.8	4458.2	2593.8
	75-79	598.1	163.4	6810.7	4072.0
	80-84	590.7	141.5	10799.3	6883.3
	85+	472.6	112.3	18905.1	14929.7
1990	0-4	0	0	261.9	213.8
	5-9	0	0	25.9	17.8
	10-14	0.3	0	35.7	18.8
	15-19	0	0	100.8	44.7
	20-24	0.3	0	137.8	51.0
	25-29	0.2	0.2	156.8	61.3
	30-34	1.5	0.8	175.6	73.0
	35-39	5.8	3.0	214.6	92.0
	40-44	14.0	8.5	277.8	155.9
	45-49	35.6	25.1	410.1	244.0
	50-54	92.7	53.2	630.3	386.2
	55-59	199.7	87.3	1022.2	615.3
	60-64	312.9	138.5	1639.7	1003.3
	65-69	411.7	184.8	2630.8	1594.7
	70-74	521.5	200.9	4054.13	2474.2
	75-79	610.3	215.9	6547.0	3877.2
	80-84	622.2	173.0	10099.8	6679.2
	85+	483.4	130.5	18610.6	14315.0

[1] Rates are expressed in deaths per 100,000 population

[2] Includes the following ICD codes: 1962-67:163; 1969-78: 162.1-162.2; 1979-90: 162.1-162.9

(CDC, 1962-1990). Due to data limitations, specifically lack of computerized vital statistics data on the specific health outcome we are modeling, we did not derive state-level mortality rates for the time periods 1950-1954 and 1955-1959. We, therefore, assumed that the background lung cancer mortality rates during these periods equaled the rates observed in the 1962-1964 period.

We used state-level mortality rates as opposed to county-specific values for a number of reasons. First, while the assessment domain is likely to be more similar in background mortality experience to Butler County than to Hamilton County, which contains the city of Cincinnati, the observed rates in this less populated county were highly unstable due to the small population size and, as a result, may not provide a good indication of the true background risk (Devine et al., 1994). Second, the possibility exists that increases in lung cancer mortality rates due to FMPC exposures might be reflected in the observed Butler County rates making them unacceptable estimators for background risk. For these reasons, we used the average annual Ohio lung cancer mortality rates for r^0 in the risk estimation. The observed values for these rates are shown in Table B3. Modeling all cohorts through age 100 years required projection of future background lung cancer mortality rates. We assumed that the age- and gender-specific rates for time periods beyond 1990 would be equal to the rate observed at that time. This assumption implies that the background lung cancer mortality rates among members of the population who survive past 1990 will remain constant. Given the downward trend in lung cancer mortality currently being experienced among males and the expected downward shift in lung cancer mortality for females, this assumption may lead to overestimation of the background lung cancer mortality rates in the later years of the analysis.

Because we are adjusting the estimated exposure-related lung cancer mortality rate to reflect smoking status, background lung cancer mortality rates must be estimated separately for ever and never smokers. The age- and time period-specific lung cancer mortality rates were adjusted to reflect smoking status rate using the relationship (Steindorf et al, 1995)

$$r^0 = P * r_S^0 + (1 - P) * r_{NS}^0$$

where r_S^0 is the background lung cancer mortality rate among ever smokers, r_{NS}^0 is the background rate among never smokers, and P is the proportion of the population that are ever smokers. Therefore, given r^0 and the relative risk of lung cancer death due to being an ever smoker, RR_S, we can derive the smoking status-specific background rates as

$$r_{NS}^{0} = \frac{r^{0}}{P * RR_{S} + (1 - P)} \qquad \text{(B9)}$$

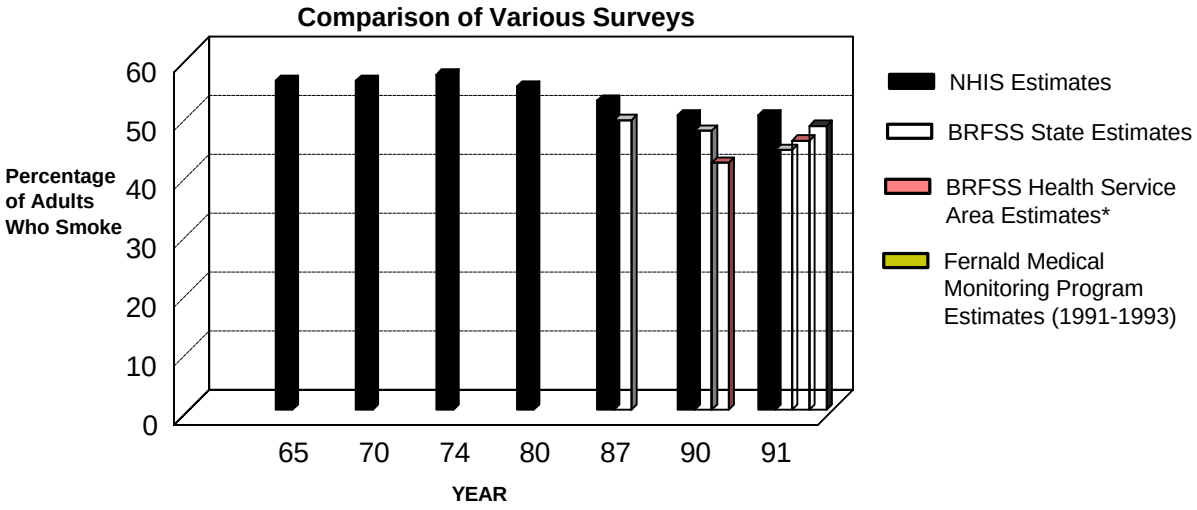
and

$$r_{S}^{0} = RR_{S} * r_{NS}^{0} \quad . \qquad \text{(B10)}$$

The relative risk for lung cancer mortality due to being an ever smoker was estimated to be 12 for males and 5 for females. These estimates were derived by weighting the relative risk estimates for current and former smokers (CDC, 1996) by the proportion of current and former smokers in the population and summing across smoking categories.

Notice that to obtain the estimates of the background lung cancer mortality rates within the time intervals and gender classes required for the life table estimation, we need estimates of the proportion of individuals who ever smoked within these strata. Smoking prevalence estimates for the required combinations of time period and gender were based on national data collected in the Centers for Disease Control and Prevention's (CDC) National Health Interview Survey, 1965-1990 (NHIS) (Giovino et al., 1994). We used national estimates of smoking prevalence, over more localized estimates, because they provide smoking data for the early time periods we are considering. To determine if the national estimates might not be representative of smoking prevalence within the assessment domain, we compared the national values, averaged across genders, to the more localized smoking prevalence estimates for the years when both measures where available. Figure B2 shows the national estimates and those potentially more applicable to the population residing within the assessment domain. State level values were derived using data from CDC's Behavioral Risk Factor Surveillance System (BRFSS) (CDC, 1987, 1990, 1991), which provided estimates for the State of Ohio since 1985. The BRFSS also provides estimates of smoking prevalence at the county level for the State of Ohio in the 1990s. Because of small sample sizes, however, county-level data were aggregated into Health Service Areas for this analysis. (CDC, 1990 and 1991). The Health Service Areas estimates shown in Figure B2 are measured in an eight-county region that included Hamilton and Butler Counties. The final smoking prevalence data to be compared with the national estimates was derived from the Fernald Medical Monitoring Program (FMMP) (Susan Pinney, personal communication). The FMMP is a voluntary medical monitoring system open to individuals who resided within 5 miles of the FMPC site for a minimum of two years during the years of plant operations. While the

FMMP estimates of smoking prevalence may be the most applicable to the population residing within the assessment domain, they are only available since 1991.



* Health Service Area = Hamilton, Butler, Warren, Clermont, Clinton, Brown Highland, and Adams Counties

Figure B2 – Comparison of percentages of ever-smokers among adults obtained from various surveys at the national, state and local level

Care needs to be taken in comparing these estimates of smoking prevalence. Differences in survey methodology and in how smoking is defined reduce their comparability. Despite these limitations, however, there does not appear to be a striking difference between the national and local estimates of smoking prevalence across the limited number of years when multiple survey measures are available. Therefore, we used the national time- and gender-specific smoking prevalence estimates for *P* in equation B9. The smoking prevalence estimates used in the risk estimation are shown for the relevant gender and time periods in Table B4. Some of the time period values were derived by extrapolation between years when the NHIS was conducted. Because the NHIS only includes respondents aged 18 and older, smoking prevalence was assumed to be zero until the cohort attained the age of 20 years in the life table analysis[3]. From

[3] It should be noted that data available indicate that smoking initiation usually occurs during adolescence. (USDHHS, 1989) Average age at initiation reported by ever smokers in the 1978-80 NHIS was 17.2 for men and 19.1 for women. (USDHHS, 1989) However, since national surveys providing information on adolescent smoking vary in the time periods covered as well as in a number of other factors (e.g., definition of smoking), which affect their comparability with each other and with the NHIS, we have included only prevalence of smoking as an adult in our risk estimation.

age 20 onward, appropriate values for smoking prevalence were taken from Table B4 according to the gender of the cohort and the time period being considered. Because we are estimating lung cancer risk for all cohorts through age 100 years, we needed to estimate the smoking prevalence values relevant for the years beyond 1995, the last year for

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Table B4 : Percentage of Ever Smokers Among U.S. Adults* by sex, 1950 – 1995**

Year	***% Ever Smokers***	
	Males	***Females***
1950	72	42
1955	72	42
1960	72	42
1965	72	42
1970	71	44
1975	66	45
1980	65	45
1985	61	45
1990	58	42
1995	58	42

*Age 18 years and older

** Data from National Health Interview Survey ; Percentages are rounded to integers. Estimates prior to 1965 at set equal to the 1965 values. Estimates from nonsurvey years between 1965 and 1995 are derived by interpolation.

which NHIS estimates are available. Projected values for smoking prevalence for all time periods beyond 1995 were set to the 1995 gender-specific estimate. Note that this implies the smoking prevalence among cohort members will remain constant at the 1995 level through the year 2088, the last year in which risk estimates are derived. Given the general trend toward

decreasing smoking in most age groups, this assumption will likely result in overestimation of smoking prevalence in the later years being modeled.

In summary, we modeled the increase in the lung cancer mortality rate due to FMPC-related radon dose within a collection of cohorts defined by the time period of first exposure, age at first exposure, and gender for all time periods until each cohort attained an age of 100 years. The mortality rate due to FMPC radon exposure was modeled for ever smokers as

$$r_S^r = r_S^0 * b * 0.9 * f * g * (d_{5-14} + q_{15-24} d_{15-24} + q_{25+} d_{25+}) \quad \text{(B11)}$$

while the mortality rate for never smokers was estimated using the model

$$r_{NS}^r = r_{NS}^0 * b * 2.0 * f * g * (d_{5-14} + q_{15-24} d_{15-24} + q_{25+} d_{25+}) \quad . \quad \text{(B12)}$$

In equations B11 and B12, the background lung cancer mortality rates among smokers and nonsmokers were derived from the observed average rate using equations B9 and B10. Uncertainty in the excess rate estimate was modeled by sampling possible values for the increase in the rate per unit exposure and the conversion factors to translate this increase into per unit dose. This sampling process was conducted for each realization of the Monte Carlo estimation of the number of FMPC-related lung cancer deaths. The distributions representing the assumed uncertainty in these parameters are given in equations B6, B7, and B8 respectively.

We now consider estimation of FMPC-related lung cancer mortality due to exposure to uranium, thorium and other radionuclides. The dose resulting from these exposures was dominated by that resulting from exposure to uranium (RAC, 1998). The lifetime risk of lung cancer death resulting from exposure to these radionuclides was estimated among the nine scenarios considered in the Fernald Dosimetry Reconstruction Project (RAC, 1998) using the lung cancer experience of A-bomb survivors. The rationale for this approach was that, given the absence of conclusive epidemiologic evidence on the magnitude of the risk resulting from internal exposure to uranium, "there is no reason to assume that, for exposure to uranium, the risks derived from the A-bomb survivors study are not broadly applicable." (RAC 1998) We also used the A-bomb survivor lifetime fatal lung cancer risk estimate of 0.0085 per sievert (NCRP, 1993) to model the possible lung cancer mortality risk resulting from FMPC-related uranium releases within the assessment domain. However, in our life table approach, we require

a time period-specific estimate of the increase in risk as opposed to a lifetime estimate. We developed time period-specific estimates of uranium-related lung cancer mortality risk by apportioning the estimated lifetime risk for each cohort into time periods based on the proportion of the lifetime cumulative dose at which the group was at risk during the interval being considered. In other words, we modeled the risk of lung cancer death that may be due to FMPC-related uranium exposure during time period *k* for each cohort in the life table as

$$P_k^u = (0.0085 * d^u) * \frac{w_k}{W} \qquad \text{(B13)}$$

where P_k^u is the time period-specific lung cancer mortality risk , w_k is a weighting factor that depends on the proportion of the lifetime cumulative dose, d^u, at which the cohort is at risk during the interval, and *W* is the sum of these weighting factors across all time periods from age at first exposure until the cohort attains age 100 years. The value for w_k used in our evaluation was the cumulative uranium dose at which the cohort was at risk during the *k*th interval. Under the assumed ten-year lag between time of exposure to uranium and initiation of time at the resulting risk of lung cancer death, the value of w_k equals the estimated cumulative uranium lung dose accrued by the cohort up to the interval ending ten years prior to the interval being considered. Notice that the sum of the collection of time period-specific lung cancer risk estimates produced using equation B13 for a given cohort, across all time intervals from that in which first exposure occurred until the cohort reaches age 100 years, equals the estimated lifetime lung cancer mortality risk.

We attempted to quantify the uncertainty associated with our estimates of the time period-specific uranium-related lung cancer mortality risk by considering the true lifetime risk coefficient, LR_u to be an unknown realization of the model

$$LR_u = 0.0085 * e \qquad \text{(B14)}$$

where e is a random factor representing the uncertainty and variability associated with the A-bomb survivor lifetime risk estimate. An estimated 90% credibility interval for possible values of e was developed in the Fernald Dosimetry Reconstruction Project (RAC, 1998) to address a variety of uncertain components of the estimate. The resulting estimated uncertainty can be reasonably modeled by assuming the e is a realization from a lognormal distribution such that

$$\ln(e) \sim N(0,0.4) \quad . \quad \text{(B15)}$$

Therefore, for each realization of the Monte Carlo process, a collection of possible cumulative uranium doses to the lung were generated for all cohorts in the assessment domain using equation B4. Based on these estimates, the resulting time period-specific lung cancer mortality risk was estimated using equation B14 with uncertainty reflecting imprecise knowledge of the true lifetime risk coefficient generated using equation B15.

To reflect a possible interaction between the risk of lung cancer death resulting from exposure to uranium and smoking history, uranium-related risk estimates were multiplied by 1.4 for ever smokers and by 0.7 for never smokers. (RAC, 1998).

Let P_{NS}^{u} be the estimated time period-specific lung cancer mortality risk for a given cohort among never smokers in our life table analysis and P_{S}^{u} be the corresponding estimate among ever smokers. Uranium risk estimates were converted to average annual time period-specific mortality rate estimates in order to combine the effect of radon and uranium exposures into an overall FMPC-lung cancer mortality rate estimate. This conversion from uranium-related lung cancer mortality risk to mortality rate estimates, which we will designate as r_{S}^{u} and r_{NS}^{u} for ever smokers and never smokers respectively, was accomplished using the relations

$$r_{S}^{u} = \frac{\ln(1 - P_{S}^{u})}{-5}$$

for ever smokers, and

$$r_{NS}^{u} = \frac{\ln(1 - P_{NS}^{u})}{-5}$$

for never smokers.

The estimated time period-specific lung cancer mortality rates resulting from exposure to radioactive materials released from the FMPC was then modeled for all cohorts by

$$r_{S}^{F} = r_{S}^{r} + r_{S}^{u}$$

for ever smokers and

$$r_{NS}^{F} = r_{NS}^{r} + r_{NS}^{u}$$

for never smokers. In these equations, r_S^F is the estimated lung cancer mortality rate among ever smokers due to exposure to both FMPC-related uranium and radon and r_S^r is the radon-related lung cancer mortality rate estimate based on equation B11. Similarly, r_{NS}^F is the lung cancer mortality rate within a given time-interval for a given cohort estimated to result from the combination of exposure to radon, r_{NS}^r from equation B12, and uranium, r_{NS}^u, among never smokers.

To derive estimates of the number of FMPC-related lung cancer deaths, we needed to translate the combined estimated increase in lung cancer mortality rates into measures of the risk of lung cancer death in a specified period. To illustrate this translation, let $r_{jk(S)}^F$ be the estimated lung cancer mortality rate due to FMPC-related exposures for ever smokers who are members of age class *j* during time period *k* and $r_{jk(NS)}^F$ be the corresponding estimate for never smokers. In addition, let $r_{jk(S)}^0$ and $r_{jk(NS)}^0$ be the background lung cancer mortality rates among ever and never smokers respectively. To estimate age- and time period-specific risk, we also require the mortality rate for all causes other than lung cancer. These rates were estimated for the assessment domain using the state-level mortality data observed within the appropriate time and age classes. Again, these data were obtained from the National Centers for Health Statistics' Detailed Mortality Data Tapes (CDC, 1962-1990) (Table B3). For the reasons given in our description of estimation of the background lung cancer mortality rates, rates for all causes of death other than lung cancer for the time periods 1950-1954 and 1955-1959 were estimated using the rates observed in 1962-1964. Again, when mortality was projected beyond the year 1990, future rates were assumed to equal the collection of age-specific rates observed in 1990.

Because we are modeling the number of FMPC-related lung cancer deaths separately for ever and never smokers, and because background mortality rates due to causes other than lung cancer likely differ between these groups, we estimated gender, age, and time dependent mortality rates for causes other than lung cancer separately for ever and never smokers. Using data from the Surgeon General's 1989 report on the health effects of smoking (U.S. Department of Health and Human Services, 1989), we estimated the relative risk due to smoking for causes of death other than lung cancer to be about 1.8 for males and about 1.6 for females. Using the prevalence values in Table B4, gender, age, and time dependent estimates for mortality rates due

to causes other than lung cancer were derived for both smoking categories in an identical manner as was used for the background lung cancer mortality rates. We will designate the mortality rate for causes other than lung cancer among ever smokers in age class *j* during time period *k* as $r^{*}_{jk(S)}$. Similarly the mortality rate for causes other than lung cancer among never smokers in age class *j* time period *k* will be designated as $r^{*}_{jk(NS)}$.

Given an estimate of the dose dependent lung cancer mortality rate resulting from FMPC exposure and the background lung cancer and non-lung cancer mortality rates, the probability of an ever smoker who has survived to the beginning of time period *k* dying during the interval due to any cause is estimated by

$$P_{jk(S)} = 1 - \exp[-5 * (r^{*}_{jk(S)} + r^{0}_{jk(S)} + r^{F}_{jk(S)})] \ . \qquad \text{(B16)}$$

Similarly, the probability of a never smoker who has survived to the beginning of the time period dying during the interval is

$$P_{jk(NS)} = 1 - \exp[-5 * (r^{*}_{jk(NS)} + r^{0}_{jk(NS)} + r^{F}_{jk(NS)})] \ . \qquad \text{(B17)}$$

Given these estimates, the probability of dying of lung cancer related to FMPC exposures during the time interval was estimated as

$$P^{F}_{jk(S)} = P_{jk(S)} * \frac{r^{F}_{jk(S)}}{r^{*}_{jk(S)} + r^{0}_{jk(S)} + r^{F}_{jk(S)}} \qquad \text{(B18)}$$

for ever smokers and

$$P^{F}_{jk(NS)} = P_{jk(NS)} * \frac{r^{F}_{jk(NS)}}{r^{*}_{jk(NS)} + r^{0}_{jk(NS)} + r^{F}_{jk(NS)}} \qquad \text{(B19)}$$

for never smokers.

Similarly, the background probability of dying from lung cancer during period *k* for ever smokers and never smokers was modeled as

$$P^{0}_{jk(S)} = P_{jk(S)} * \frac{r^{0}_{jk(S)}}{r^{*}_{jk(S)} + r^{0}_{jk(S)} + r^{F}_{jk(S)}} \qquad \text{(B20)}$$

and

$$P^{0}_{jk(NS)} = P_{jk(NS)} * \frac{r^{0}_{jk(NS)}}{r^{*}_{jk(NS)} + r^{0}_{jk(NS)} + r^{F}_{jk(NS)}} \ . \qquad \text{(B21)}$$

Finally, the probability of dying during the time interval from a cause other than lung cancer among ever smokers and never smokers was modeled as

$$P^{*}_{jk(S)} = P_{jk(S)} * \frac{r^{*}_{jk(S)}}{r^{*}_{jk(S)} + r^{0}_{jk(S)} + r^{F}_{jk(S)}}$$

and

$$P^{*}_{jk(NS)} = P_{jk(NS)} * \frac{r^{*}_{jk(NS)}}{r^{*}_{jk(NS)} + r^{0}_{jk(NS)} + r^{F}_{jk(NS)}} \ .$$

To reflect the uncertainty in the components of the estimated probabilities, for each realization of the Monte Carlo process used to estimate the number of FMPC-related lung cancer deaths, different values of $P^{F}_{jk(S)}$, $P^{F}_{jk(NS)}$, $P^{0}_{jk(S)}$, $P^{0}_{jk(NS)}$, $P^{*}_{jk(S)}$, and $P^{*}_{jk(NS)}$ were generated reflecting different samples of the uncertain values of cumulative lung dose and the uncertain parameters used in estimating $r^{F}_{jk(S)}$ and $r^{F}_{jk(NS)}$.

D. ESTIMATION OF THE NUMBER OF FMPC-RELATED LUNG CANCER DEATHS

We estimate the number of lung cancer deaths resulting from offsite exposure to radioactive material released from the FMPC site by applying a life table approach to a series of cohorts. These cohorts are defined by the time period of first exposure, the age at first exposure, cell of residence within the assessment domain, and gender. Using the methods described in previous sections of this Appendix, we can estimate the time- and age-specific probabilities of death due to lung cancer and all other causes among members of these cohorts as they age from the year of first exposure through age 100 years. To translate these probability estimates into estimates of the actual number of lung cancers, however, we must also estimate the number of individuals within each cohort for all time periods being modeled. We now will describe the methods used to estimate the changing number of individuals at risk of dying of lung cancer over age and time periods.

We first consider the population present in the assessment domain during the time interval 1950-1954. The first step in determining the relevant population sizes for this group was to estimate the number of residents in each cell during this period. We made these estimates using the methods described by Rogers and Killough (1997) by using U.S. Geological Survey quadrant topographical maps (Ohio Department of Natural Resources, 1992). In addition, to illustrating the usual USGS map features, these maps also contained symbols indicating the location of structures based on surveys of the area conducted between 1949 and 1955 and again between 1979 and 1981. Using an overlaid map of the assessment domain, we were able to separately enumerate the number of structures within each geographic cell of the domain present in years close to 1950 and 1980. The cell-specific structure counts were then reduced by 15% to account for unoccupied buildings such as barns, warehouses, and garages (Rogers and Killough, 1997).

Let $\hat{S}_{i1950}$ be the estimated number of structures in 1950 within cell *i* of the assessment domain. Using this value, we estimated the total population size within cell *i* in 1950 as

$$\hat{n}_{i1950} = \hat{S}_{i1950} * \hat{O}_{i1950} \qquad \text{(B22)}$$

where $\hat{O}_{i1950}$ is an estimate for the occupancy rate within cell *i* in the period 1950-1954. The average Butler County occupancy rates from the 1950 census were used to estimate the

occupancy rates for all cells so that $\hat{O}_{i1950} = \hat{O}_{1950}$ for all *i*. There are at least two potential sources of uncertainty in the population estimates derived using equation B22. The first is uncertainty concerning the true number of structures in a cell given the number enumerated from the map, $\hat{S}_{i1950}$. The second potential source of uncertainty is the sampling variability inherent in using the average county occupancy rate as an estimate for the cell-specific average number of occupants per structure. We attempted to quantify the first uncertainty component by comparing the map-based structure counts to counts derived using local government tax and real estate records. Due to the resources required for the record-based structure estimation, only three cells were used to make these comparisons. Table B5 shows the estimated number of structures for the three test cells based on the map counts and the analysis of tax and real estate records. It should be noted that a number of subjective decisions were required to develop the records-based estimates, for example determination of tax codes most indicative of an inhabited structure. However, we believe that comparison of the records-based and map-based approaches should, at a minimum, indicate the magnitude of the uncertainty associated with $\hat{S}_{i1950}$.

Let S_{i1950} be the true number of structures in cell *i* during the time period. We assume that the uncertainty associated with S_{i1950} follows a lognormal distribution such that

$$\ln(S_{i1950}) \sim N(\ln(\hat{S}_{i1950}), e^2) \qquad \text{(B23)}$$

where e^2 represents the uncertainty concerning the true value which is assumed to be constant across all cells and years. Based on the ratios of the record-based to map-based number of structure estimates in Table B5, we used a value 0.1 for e^2 to reflect the average uncertainty of the map-based structure counts. Note that this selection for the value of e^2, along with the assumption that the distribution of S_{i1950} about $\hat{S}_{i1950}$ is lognormal, implies a 90% credibility interval for $S_{i1950} / \hat{S}_{i1950}$ of 0.6 to 1.7. The width of this interval is generally consistent with the range of values for the ratio of the records-based to map-based estimators for the number of structures shown in Table B5.

Table B5. Comparison of the number of residential structures obtained from United States Geological Survey Maps Containing the Fernald Assessment Domain with those obtained from Butler County Property Tax Records for three cells. 1955 and 1979.

Cell Number	Number of Residential Structures			
	1955		1979	
	USGS Map[1]	Property Tax	USGS Map[1]	Property Tax
7	26	18	49	77
34	161	151	316	531
158	41	12	99	86

[1] Number reduced by 15% to account for nonresidential structures

The sampling variability associated with using the mean county occupancy rate to estimate the cell-specific occupancy was quantified using the observed distribution of occupancy rates collected in the 1990 Census within the two Census Tracts that comprise a large portion of the assessment domain. We assumed that the random variable corresponding to the cell-specific occupancy rate could be described with the model

$$O_{i1950} = \hat{O}_{i1950} * l$$

where l is a random variable, which we will call a variability factor. Based on the Census occupancy rate data, we assume the l follows a lognormal distribution such that

$$\ln(l) \sim N(0,0.5) \quad .$$

Figure B3 shows the agreement between the empirical cumulative distributions of the variability factors estimated from the Census occupancy data and the corresponding distribution based on our assumed distribution for l. Note that the assumptions on the distribution of l , along with the assumed median occupancy rate of 3 persons per household, results in a lognormal uncertainty distribution for the true 1950 cell-specific occupancy rates such that

$$\ln(\hat{O}_{i1950}\ ) \sim N(\ln(3),0.5) \ . \qquad \text{(B24)}$$

Let n_{i1950} be the true number of residents in cell i during the period 1950 – 1954. Based on equation B22 and the uncertainties implied by B23 and B24, we generated possible values for the cell-specific population size for each realization of the Monte Carlo process from a lognormal distribution such that

$$\ln(n_{i1950}) \sim N(\ln(\hat{n}_{i1950}),0.6) \quad . \qquad \text{(B25)}$$

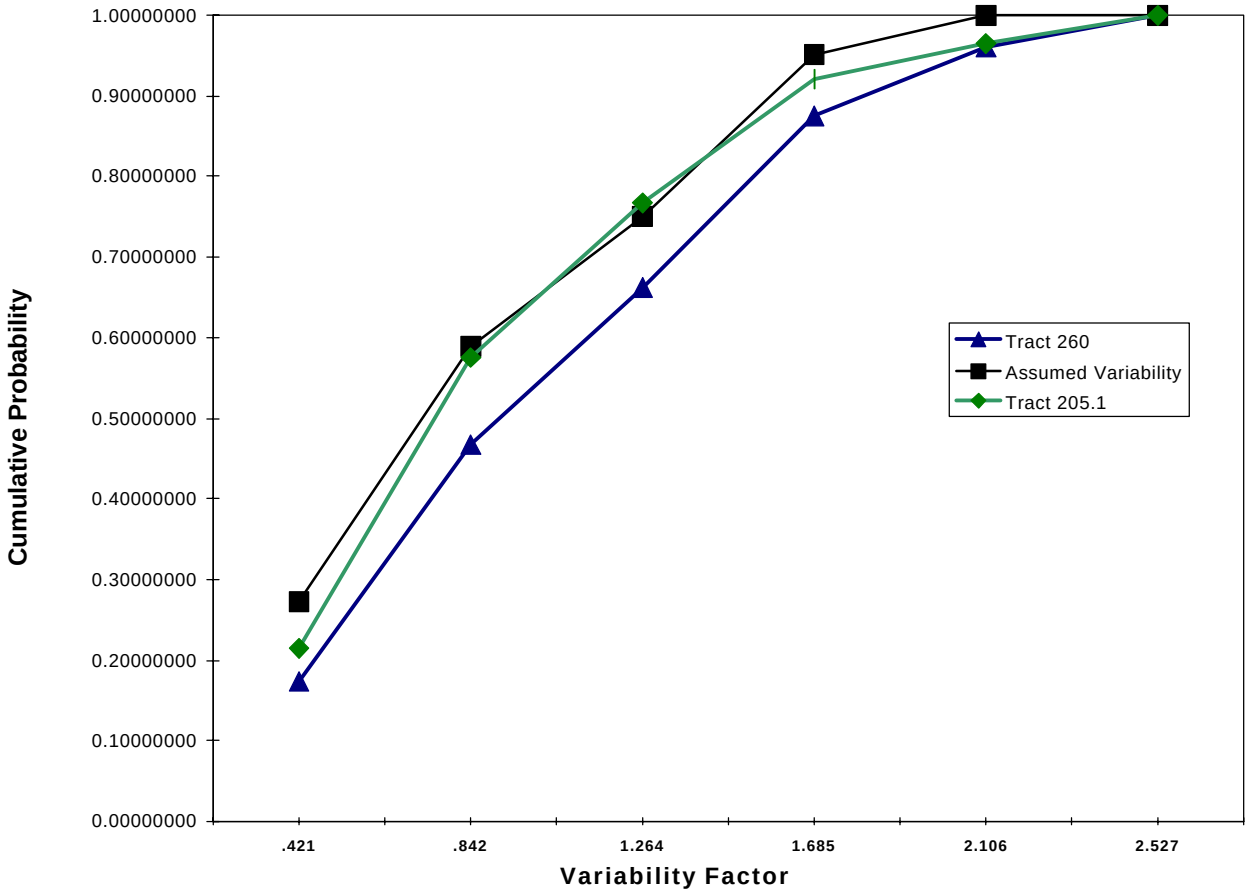

Figure B3 – Comparison of the empirical cumulative distributions of the factor representing variability in the number of persons per structure for U.S. Census Tracts 260 (Crosby and Harrison Townships) and 205.1 (Colerain Township) as estimated from 1990 U.S. Census data with the empirical distribution of the assumed sampling variability.

Given an estimate for a possible value of the total cell population in the 1950-1954 period, n_{i1950}, the next step in developing the lung cancer estimates was to subdivide the population into males and females and age classes. The number of persons in each age and gender group was estimated using n_{i1950} and the age and gender distributions observed in Butler County in the 1950 Census. The age and gender distributions for Butler County for each of the time periods covered in the life table estimation are shown in Table B6.

Table B6. Percent distribution of the Butler County population by year, age group, and gender. 1950-1990.

Years	Age group	Distribution (%)	
		Males	Females
1950-1954	0-4	11.5	10.8
	5-9	8.9	8.6
	10-14	7.6	7.3
	15-19	7.9	8.2
	20-24	9.0	9.2
	25-29	8.3	8.4
	30-34	7.1	7.2
	35-39	6.8	7.0
	40-44	6.4	6.5
	45-49	5.9	5.7
	50-54	5.2	5.1
	55-59	4.2	4.4
	60-64	3.4	3.6
	65-69	2.9	3.0
	70-74	2.1	2.2
	75-79	1.1	1.3
	80-84	1.0	1.2
	85+	0.3	0.4

Table B6, continued

Years	Age group	Distribution (%)	
		Males	Females
1955-1959	0-4	11.4	10.5
	5-9	9.6	9.1
	10-14	9.0	8.4
	15-19	8.0	8.1
	20-24	6.9	7.5
	25-29	6.6	6.8
	30-34	6.6	6.8
	35-39	6.6	6.9
	40-44	6.5	6.6
	45-49	6.0	5.8
	50-54	5.3	5.2
	55-59	4.5	4.7
	60-64	3.9	3.9
	65-69	3.4	3.4
	70-74	2.6	2.7
	75-79	1.6	1.7
	80-84	1.0	1.2
	85+	0.5	0.5
1960-1964	0-4	10.3	9.4
	5-9	10.3	9.5
	10-14	10.6	9.8
	15-19	9.3	9.2
	20-24	6.7	7.4
	25-29	5.6	5.8
	30-34	5.9	6.1
	35-39	6.2	6.4
	40-44	6.4	6.5
	45-49	6.0	5.8
	50-54	5.1	5.2
	55-59	4.5	4.7
	60-64	3.9	4.0
	65-69	3.3	3.5
	70-74	2.6	2.9
	75-79	1.7	2.0
	80-84	0.9	1.1
	85+	0.5	0.7

Table B6, continued

Years	Age group	Distribution (%)	
		Males	Females
1965-1969	0-4	9.3	8.4
	5-9	10.3	9.5
	10-14	10.9	9.9
	15-19	10.6	10.4
	20-24	8.7	9.2
	25-29	6.3	6.3
	30-34	5.7	5.9
	35-39	5.9	5.9
	40-44	6.3	6.2
	45-49	6.0	5.8
	50-54	5.0	5.0
	55-59	4.2	4.4
	60-64	3.5	3.7
	65-69	2.7	3.1
	70-74	2.1	2.6
	75-79	1.3	1.9
	80-84	0.7	1.1
	85+	0.4	0.7
1970-1974	0-4	8.7	7.9
	5-9	9.5	8.7
	10-14	10.7	9.7
	15-19	11.2	11.0
	20-24	10.1	10.2
	25-29	7.0	6.7
	30-34	5.9	6.0
	35-39	5.5	5.6
	40-44	6.0	5.8
	45-49	5.8	5.8
	50-54	5.2	5.1
	55-59	4.2	4.3
	60-64	3.4	3.6
	65-69	2.6	3.1
	70-74	1.9	2.5
	75-79	1.2	1.9
	80-84	0.7	1.2
	85+	0.4	0.8

Table B6, continued

Years	Age group	Distribution (%)	
		Males	Females
1975-1979	0-4	7.8	7.0
	5-9	8.6	7.7
	10-14	9.3	8.4
	15-19	11.2	11.0
	20-24	11.2	10.9
	25-29	8.0	7.7
	30-34	7.0	6.9
	35-39	5.7	5.8
	40-44	5.4	5.3
	45-49	5.4	5.5
	50-54	5.4	5.3
	55-59	4.5	4.5
	60-64	3.4	3.7
	65-69	2.6	3.2
	70-74	1.9	2.7
	75-79	1.3	2.0
	80-84	0.8	1.3
	85+	0.4	1.0
1980-1984	0-4	8.0	7.1
	5-9	7.9	7.0
	10-14	8.3	7.5
	15-19	9.8	9.5
	20-24	10.9	10.7
	25-29	8.7	8.3
	30-34	8.1	8.0
	35-39	6.7	6.6
	40-44	5.6	5.5
	45-49	5.0	5.0
	50-54	5.0	4.8
	55-59	4.7	4.8
	60-64	3.8	4.0
	65-69	2.8	3.4
	70-74	2.0	2.7
	75-79	1.3	2.1
	80-84	0.7	1.4
	85+	0.4	1.2

Table B6, continued

Years	Age group	Distribution (%)	
		Males	Females
1985-1989	0-4	7.9	7.0
	5-9	8.1	7.1
	10-14	7.5	6.7
	15-19	9.0	8.6
	20-24	9.6	9.7
	25-29	8.6	8.4
	30-34	8.6	8.4
	35-39	7.9	7.7
	40-44	6.5	6.3
	45-49	5.2	5.2
	50-54	4.6	4.5
	55-59	4.5	4.5
	60-64	4.1	4.3
	65-69	3.2	3.7
	70-74	2.1	2.8
	75-79	1.4	2.3
	80-84	0.7	1.6
	85+	0.5	1.4
1990	0-4	8.0	7.0
	5-9	8.1	7.1
	10-14	7.5	6.7
	15-19	8.5	8.1
	20-24	9.0	9.1
	25-29	8.1	8.0
	30-34	8.7	8.6
	35-39	8.1	7.9
	40-44	7.3	7.1
	45-49	5.4	5.4
	50-54	3.8	3.8
	55-59	5.2	5.2
	60-64	4.1	4.2
	65-69	3.3	3.8
	70-74	2.2	2.8
	75-79	1.4	2.3
	80-84	0.8	1.6
	85+	0.5	1.4

To illustrate how we proceeded in the life table estimation from this point, we now define n_{ij1950} as the estimated number of individuals of a specified gender who resided in cell *i* and were in age class *j* during the time period 1950-1954. Because assumptions about the latency period between exposure to radioactive material released from the FMPC and the initiation of time at risk resulting from that exposure, we estimate no FMPC-related lung cancer death among this group during the first time interval. However, this cohort did experience background lung cancer mortality risk during this period. Using equations B20 and B21, we estimate the number of background lung cancer deaths among ever smokers in this group as

$$LC^{0}_{ij1950(S)} = n_{ij1950(S)} * P^{0}_{ij1950(S)} \qquad \text{(B26)}$$

and among never smokers using

$$LC^{0}_{ij1950(NS)} = n_{ij1950(NS)} * P^{0}_{ij1950(NS)} \qquad \text{(B27)}$$

where $n_{ij1950(S)}$ and $n_{ij1950(NS)}$ are the estimated number of ever and never smokers among this population. The number of individuals in the smoking categories is derived by multiplying n_{ij1950} by the appropriate ever smoker prevalence from Table B4. In addition to risk of background lung cancer, the cohort is also at risk for death due to causes other than lung cancer during the interval. The estimated number of deaths due to causes other than lung cancer is derived for ever and never smokers using

$$D^{*}_{ij1950(S)} = n_{ij1950(S)} * P^{*}_{ij1950(S)} \qquad \text{(B28)}$$

and

$$D^{*}_{ij1950(NS)} = n_{ij1950(NS)} * P^{*}_{ij1950(NS)} \qquad \text{(B29)}$$

respectively. Therefore the number of ever smokers predicted to survive into the 1955-1959 time period is given by

$$n^{1955}_{i(j+5)1950(S)} = n_{ij1950(S)} - LC^{0}_{ij1950(S)} - D^{*}_{ij1950(S)} \quad . \qquad \text{(B30)}$$

Similarly, the number of surviving never smokers is estimated by

$$n^{1955}_{i(j+5)1950(NS)} = n_{ij1950(NS)} - LC^{0}_{ij1950(NS)} - D^{*}_{ij1950(NS)} \quad . \qquad \text{(B31)}$$

Under our latency assumptions, during the 1955-1959 time interval the cohort is considered to be at risk for lung cancer death resulting from exposure to radon released from the FMPC site during the 1950-1954 time interval. Using equations B18 and B19, the estimated number of FMPC-related lung cancer deaths in the 1955-1959 period is modeled as

$$LC^{F}_{i(j+5)1950(S)} = n^{1955}_{i(j+5)1950(S)} * P^{F}_{i(j+5)1950(S)} \qquad \text{(B32)}$$

for ever smokers and

$$LC^{F}_{i(j+5)1950(NS)} = n^{1955}_{i(j+5)1950(NS)} * P^{F}_{i(j+5)1950(NS)} \qquad \text{(B33)}$$

among those who never smoked. Estimates for the number of deaths due to background lung cancer and to causes other than lung cancer are estimated as in the previous interval. The predicted number of individuals who survive until the 1960-1964 period given by

$$n^{1960}_{i(j+10)1950(S)} = n^{1955}_{i(j+5)1950(S)} - LC^{F}_{i(j+5)1950(S)} - LC^{0}_{i(j+5)1950(S)} - D^{*}_{i(j+5)1950(S)}$$

for ever smokers and

$$n^{1960}_{i(j+10)1950(NS)} = n^{1955}_{i(j+5)1950(NS)} - LC^{F}_{i(j+5)1950(NS)} - LC^{0}_{i(j+5)1950(NS)} - D^{*}_{i(j+5)1950(NS)}$$

for never smokers.

By repeating this process of estimating the time and age-specific probabilities of FMPC and background lung cancer death, and that of death due to causes other than lung cancer, the number of survivors in each successive time period is modeled for this cohort until the cohort attains an age of 100 years. Note that, under the assumed 10-year latency period between

exposure and risk assumed for uranium exposures, in the next time period, 1960-1964, the cohort experiences the first lung cancer mortality risk due to FMPC-related uranium exposures resulting from uranium lung doses incurred in the 1950-1954 time period. The sum of the collection of FMPC-related lung cancer deaths that results from repeating this estimation process across all time periods for this cohort provides an estimate of the total number of lung cancer deaths related to exposure from the site among those of the specified gender who were in age class *j* in 1950-1954 and resided in cell *i*. The result of summing such estimates across both gender groups and age classes of the 1950-1954 cell *i* population is an estimate for the total number of FMPC-related lung cancer deaths among those individuals first exposed in the cell in the 1950-1954 period. Similarly, the sum of these values across cells provides an estimate of the total number of FMPC-related lung cancers among the estimated population residing within the assessment domain in the 1950-1954 time period. To reflect the uncertainty associated with this estimate, several possible values of this sum were developed in the Monte Carlo algorithm based on repeated sampling of the uncertain values of age- and time-specific cumulative lung dose and the resultant probability of lung cancer death.

Given an estimate for the total number of lung cancer deaths resulting from FMPC-exposure to radioactive material among the population first exposed in 1950-1954, we now consider developing a similar estimate among those first exposed in the 1955-1959 period. We consider this group to be entirely composed of births to those individuals present in the 1950-1954 time period who survived until 1955. In other words, if b_{i1955} is the annual population-based birth rate for cell *i* in the time period 1955-1959, then the number of individuals first exposed to radioactive material from FMPC in this period is modeled as

$$n_{i1955} = n_{i1950}^{1955} * (b_{i1955} * 5) \qquad \text{(B34)}$$

where n_{i1950}^{1955} is the total number of persons, present in the cell in 1950-1954 who are estimated to survive until 1955. The value used for b_{i1955} is the Census estimate of the annual population-based birth rate for Butler County during this time period. This value and the birth rates used for subsequent time periods are shown in Table B7.

Table B7. Average annual birth rates[1] for Butler County. 1950-1990.

Years	Birth rate
1950-1954	32.5
1955-1959	49.5
1960-1964	42.0
1965-1969	21.3
1970-1974	16.9
1975-1979	15.0
1980-1984	15.4
1985-1989	14.7
1990	15.2

[1] Rates are expressed per 1000 residents

Because we considered birth to be the only means of being first exposed in the 1955-1959 time period, there is only one age class present in this population. The number of FMPC-related lung cancer deaths in this group can be estimated, as was done for the cohorts first exposed in 1950-1954 by repeated estimation of age- and time-specific risk and modeling of survival. By modeling this cohort through age 100 years and summing across gender groups, an estimate of the total number of lung cancer deaths resulting from FMPC exposure can be derived for those first exposed in 1955-1959. Again, several realizations of the estimate were developed in the Monte Carlo process reflecting the uncertain components of the risk estimation algorithm.

Because 1960 was a census year and our approach to estimating migration patterns depended on census data, it is for those first exposed in the 1960-1964 time period that we first attempt to model immigration into and emigration out of the assessment domain. To model migration into and out of the cells, we first estimate the number of births in the 1960-1964 period for each cell by multiplying the total number of cell residents estimated to be alive at the end of 1959, which we will designate by n_i^{1959} for cell *i*, by five times the appropriate population-based birth rate from Table B7. The sum of n_i^{1959} and the estimated number of births in the 1960-1964 time period, b_{i1960}, is the total number of persons estimated to be present in the cell during the

interval. We will designate this life table-based estimate of the cell *i* population in the 1960-1964 interval as $n_{i(table)}^{1960}$. For comparison, we develop an alternative estimator likely to be more reflective of meaningful immigration or emigration trends. Recall from the discussion of the map-based approach used to make the initial estimates of population size that the number of structures within each cell was illustrated on the USGS maps for years close to 1950 and 1980. Using the methodology of Rogers and Killough (1997), we assumed a constant exponential growth rate in the number of structures over time to interpolate estimates for the number of structures per cell in the years 1960 and 1970 based on the 1950 and 1980 endpoint values. Thus, an alternative estimator for the cell-specific population size, which we will call $n_{i(map)}^{1960}$, can be derived as

$$\hat{n}_{i(map)}^{1960} = \hat{S}_{i1960} * \hat{O}_{1960}$$

where $\hat{S}_{i,1960}$ is the interpolated structure count in cell *i*, and $\hat{O}_{1960}$ is the 1960 Census average occupancy rate for Butler County. We felt that $\hat{n}_{i(map)}^{1960}$ was likely to be more reflective of changes in cell population size due to factors other than births and deaths than would be $\hat{n}_{i(table)}^{1960}$. This belief is based on the idea that a dramatic change in cell population due to immigration or emigration would likely be reflected in a meaningful change in the number of structures between 1950 and 1980. Recall, however, that the actual value for the population of the cell in 1950 used in each realization of the Monte Carlo process was generated from the uncertainty distribution given in equation B24. It is reasonable to assume that the uncertainty in the true number of residents relative to the map-based estimate for 1960 is correlated with the uncertainty of the true number relative to the 1950 map-based estimate. To maintain this correlation, a possible alternative value for the true number of residents in cell *i* in the 1960-1964 period was generated by

$$n_{i(map)}^{1960} = \hat{n}_{i(map)}^{1960} * e_{i,1950}$$

where

$$e_i = \frac{n_i^{1950}}{\hat{n}_i^{1950}} \quad .$$

In other words, the percentage change between the map-based population estimate and the generated value for population size used in each run of the Monte Carlo process is considered to be constant across all time periods. Once a value for $n_{i(map)}^{1960}$ has been generated, it is compared to $n_{i(table)}^{1960}$. If $(n_{i(map)}^{1960} - n_{i(table)}^{1960})$ is greater than or equal to 20, then the realization for the cell *i* population size is increased by that difference. This increase in population size is considered to be an estimate of immigration into the cell. Twenty is used as the cut-off to require approximately one immigrant into each of the age classes. The collection of immigrants is assumed to have an age distribution equal to the 1960 age distribution of Butler County residents as shown in Table B6. If $(n_{i(table)}^{1960} - n_{i(map)}^{1960})$ is greater than or equal to twenty, then the number of births in cell *i* is reduced to reflect estimated emigration. If this reduction results in a negative population size estimate, then population size is set to zero.

Once the size of the population first exposed in the 1960-1964 time period is estimated, the lung cancer mortality experience of this group is modeled in the same fashion as for groups with first years of exposure between 1950 and 1959.

Cell specific population sizes for those first exposed in the 1965-1969, 1975-1979, and 1985-1989 time periods were estimated in the same manner as were the estimates for the 1955-1959 period. The adjustments for immigration and emigration made to cell-specific estimates of the number of individuals first exposed in the 1970-74 and 1980-1984 periods were derived as described for those first exposed in 1960-1964. Once these estimates were obtained, estimates of the number of FMPC-related lung cancer deaths were derived by applying the life table methods described previously to each of the year of first exposure groups. By summing these estimates across year of first exposure groups, we obtained an estimate for the total number of lung cancer deaths related to exposure to radioactive material from the FMPC site for those exposed during some time interval between 1951 and 1988. Replication of the entire modeling procedure in a Monte Carlo process using randomly sampled values for the uncertain components of dose, risk per unit dose and population size produced a collection of possible values for this total number of lung cancer deaths.

E.

ESTIMATION OF THE PERCENTAGE INCREASE IN LUNG CANCER DEATHS THAT MAY BE DUE TO FMPC-RELATED EXPOSURES

In addition to estimating the number of lung cancer deaths that may have occurred among individuals who resided in the assessment domain potentially due to FMPC-related exposure to radioactive material, we also estimated the percentage increase in lung cancer deaths over the number that would be expected in the absence of FMPC exposures. The background number of lung cancer deaths expected in the population was estimated by repeating the Monte Carlo process described above with the exception that all FMPC-related exposures were set to zero. The resulting collection of possible values for the number of background lung cancer deaths in the assessment population provides a measure by which to interpret the relative potential effects of FMPC exposures. These background estimates are provided Tables 3 and 5 of this report. The percentage increase, PI_n, in the number of lung cancer deaths that may have resulted from exposure to radioactive material released from the FMPC site was estimated for the *n*th realization of the Monte Carlo process as

$$PI_n = \frac{LC_n^F}{median(LC^0)} * 100$$

where LC_n^F is the estimated total number of FMPC-related lung cancer deaths among the assessment population generated in the *n*th realization and $median(LC^0)$ is the median of the uncertainty distribution for the estimated number of background lung cancer deaths. Note that the resulting uncertainty in the estimated percentage increase in lung cancer deaths will not reflect the uncertainty in the estimated background number of lung cancer deaths. The uncertainty in the estimated background number, however, was small relative to the uncertainty range in the estimated number of FMPC-related lung cancer deaths. For example, the ratio of the median of the uncertainty distribution for the estimated total number of background lung cancer deaths to the upper and lower bounds of the 90% credibility interval was about 1.12 (see Table 3 in the main text). The corresponding ratio for the estimated total number of FMPC-related lung cancer deaths was approximately 3.5. Thus, the uncertainty in the estimated percentage increase will likely be dominated by the uncertainty associated with the estimated

number of FMPC-related lung cancer deaths and ignoring the estimated uncertainty in the background number of lung cancer deaths should have little impact on the width of the estimated 90% credibility interval for the percentage increase.

F. ESTIMATION OF THE AVERAGE LUNG CANCER MORTALITY RATE AND THE AVERAGE LIFETIME RISK OF LUNG CANCER DEATH THAT MAY RESULT FROM FMPC-RELATED EXPOSURE TO RADIOACTIVE MATERIAL

Estimates for the total person-years at risk for FMPC-related lung cancer death in the assessment population were enumerated in each Monte Carlo run of the life table algorithm. Combining these estimates, which we will call PY_n for the *nth* Monte Carlo realization, with LC_n^F enables us to estimate the average annual lung cancer mortality rate resulting from FMPC-exposures. The resulting realization for this rate, which we will designate as mr_n^F, was calculated as

$$mr_n^F = \frac{LC_n^F}{PY_n} \quad .$$

A realization for the estimated average lifetime probability of dying from lung cancer resulting from exposure to radioactive materials released from FMPC was produced for each Monte Carlo run using the model

$$LP_n^F = \frac{LC_n^F}{N_n}$$

where N_n is the realization for the total number of individuals who resided within the assessment domain for some period of time from 1951 through 1988.

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APPENDIX C

Evaluation of the Impact of the Dose Estimation Assumptions.

I. Evaluation of the Impact of the Dose Estimation Assumptions

Radiation exposure to specific body organs occurs through inhalation and ingestion of radionuclides and direct external exposure to radiation fields. Estimation of organ dose ultimately depends on numerous factors including information on the radionuclide and its physical and chemical form, mode of exposure, concentration of the radionuclide in the exposure medium, target tissue and numerous other exposure and physiologic parameters. The dosimetric methods incorporated for use in estimating dose to residents around the Fernald site are discussed in detail in Appendix I, Volume II, of the Fernald Dosimetry Reconstruction Project final report. (RAC, 1998)

A. ASSUMPTIONS

In order to apply the dose algorithm developed in the Fernald Dosimetry Reconstruction Project to our estimation of lung cancer deaths in the Fernald Risk Assessment Project, a number of assumptions about factors that impact dose must be specified. These factors include the following: date of birth, gender, physical locations with regard to home, work and school, time period and duration of time spent within these locations, diet, and other lifestyle factors such as percent of time spent swimming in contaminated water (Table C1). The impact of these factors on dose depends on the organ being specified and the radionuclide being considered. For example, for uranium and thorium isotopes, percent of time spent swimming in contaminated water should have little effect on lung dose. However, factors that impact estimates of internal exposure via inhalation may significantly affect the magnitude of the estimated lung dose. These may include following: (1) age at exposure - because dose conversion factors are age-dependent; (2) level of physical activity - because this determines a subject's breathing rate; and (3) primary and secondary locations of that individual (e.g. home, school and work locations), including amount of time spent at those locations and time spent indoors and outdoors - because these determine the concentration of the radionuclide in the air and the amount taken into the body.

Table C1: Factors specified for dose estimation algorithm

gender	source of contaminated irrigation water
date of birth	irrigation volume
location of home	% of intake of contaminated vegetables
location of school	% of intake of contaminated milk
location of workplace	% of intake of contaminated beef
% of time indoors	% of intake of contaminated poultry
indoor activity index	% of intake of contaminated eggs
% of time outdoors	% of intake of contaminated fish
outdoor activity index	contaminated water source for fish
indoor particulate factor	% of time spent swimming
air turnover rate	contaminated water source for swimming
source of contaminated drinking water	amount of ingested soil

Exposure scenarios were utilized in the Fernald Dosimetry Reconstruction Project to provide dose and risk estimates for hypothetical residents of the communities surrounding the FMPC. The specifications for these nine exposure scenarios and the resulting dose and risk estimates are provided in Appendix A. In our current lung cancer risk assessment, we are estimating doses and lung cancer mortality risk for the entire population of the FMPC assessment domain. Doing so requires that a consistent set of assumptions for many of the factors shown in Table C1 be defined for the domain population. This standard set of assumptions will be utilized in the dose estimation algorithm. The assumptions specified are listed in Table C2. The values for these assumptions were based on a review of those used in the nine exposure scenarios used in the Fernald Dosimetry Reconstruction Project and the supporting documentation for the scenario assumptions provided in the final Fernald Dosimetry Reconstruction Report. Some factors, such as amount of time spent indoors and outdoors, were selected to maximize the potential for exposure and the resulting impact on the risk of lung cancer death.

1. Sensitivity Analysis

We used the nine exposure scenarios from the Fernald Dosimetry Reconstruction Project to evaluate the impact of our standard set of dose assumptions on estimates of lung dose. The 5^{th}, 50^{th}, and 95^{th} percentiles of the radon and uranium doses for the nine exposure scenarios estimated using our assumptions were compared to those estimated using the original assumptions specified in the final Fernald Dosimetry Reconstruction Report to determine if our standardization of these assumptions significantly influenced lung dose. A significant change in lung dose could considerably affect lung risk. (See Appendix A, Tables A1-A9). With the

exception of gender and date of birth, the impacts of changes in the assumptions were evaluated separately and all together. The results are provided in Tables C3 and C4. As expected, changing factors that impact exposure via an ingestion route (e.g. % intake of contaminated vegetables) or by direct external exposure had essentially no effect on lung dose estimates. These dose estimates were most influenced by leaving the "individual" in one location for the entire exposure period (i.e. not allowing changes in location due to school attendance or work outside the home), by setting the indoor activity index to 1.0 and by setting time spent outdoors to 67 percent. Of these, increasing the amount of time spent outdoors from about 19 percent, on average, in the original scenario specifications to 67 percent had the greatest single impact on the magnitude of the dose estimate. (Figures C1-C2). This is to be expected since the concentration of radioactive materials from FMPC would have been higher outdoors. Eliminating location changes had slightly less effect, despite the fact that location, in terms of distance and direction from the FMPC site, has a major influence on radiation dose. This is because the original scenario specifications included only 25 percent or less of a subject's time being spent at a different school or work location. (Figures C3 – C4). Overall, however, changing the scenario assumptions resulted only in slightly increased median values of dose and slightly wider credibility intervals when applied separately and all together. (Figures C5 -C6).

B. CONCLUSION

Changing scenario assumptions to be consistent with those selected for use in our risk analysis had little effect on resulting radon and uranium lung doses for the nine exposure scenarios. In general, applying all assumptions resulted in negligible increases in the median values of the scenario dose estimates and small increase in the range of uncertainty. This effect appears to be reasonable given the inherent uncertainties in the dose estimation assumptions.

Table C2: Values of dosimetric factors used for estimating population dose and risk

Factor	*Assumed Value*
Time Period	Maximum: 1951 – 1989
Gender	Varies with distribution of cell population
Date of birth	Varies with distribution of cell population
Location of home	Centroid of cell
Location of school	Centroid of cell of residence
Location of workplace	Centroid of cell of residence
Time indoors (%)	33
Indoor activity index[a]	1
Time outdoors (%)	67
Outdoor activity index[a, b]	1.5
Indoor particulate factor[b]	0.7
Air turnover (hour^{-1})[b]	0.4
Contaminated drinking water source	none
Contaminated irrigation water source	Great Miami River
Irrigation volume (Lm^{-2} day^{-1})	0.5
Contaminated vegetables (% of intake)	50
Contaminated milk (% of intake)	10
Contaminated beef (% of intake)	10
Contaminated poultry (% of intake)	10
Contaminated eggs (% of intake)	10
Contaminated fish (% of intake)	50
Contaminated water source for fish	Great Miami River
Swimming (% of time)	2
Contaminated water source for swimming	Great Miami River
Ingested soil (g day^{-1})	0.5

[a] light activity
[b] using same value as used in the nine exposure scenarios

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