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FEED MATERIALS PRODUCTION CENTER (US DOE) [a.k.a. FERNALD ENVIRONMENTAL MANAGEMENT PROJECT] HAMILTON AND BUTLER COUNTIES, OHIO

HEALTH OUTCOME DATA

In general terms, health outcome data are morbidity and mortality data for the Fernald community. ATSDR evaluates these data to determine the overall health status of the community and to identify specific adverse health effects that may be occurring in the community as a result of exposure to chemicals and radioactive materials from the Fernald facility.

ATSDR evaluated the following sources of health outcome data for this public health assessment: (1) CDC's Fernald Risk Assessment Project (FRAP) ([CDC 1998, 1999](#)); (2) preliminary results of cancer analyses among participants in the Fernald Medical Monitoring Program (FMMP) ([Pinney 1999b](#)); and (3) a Master of Science thesis on β -2-microglobulin levels in urine of potentially exposed persons living near the Fernald site ([Kammer 1998](#)).

The CDC's National Institute for Occupational Safety and Health (NIOSH) has conducted and initiated several investigations involving Fernald workers. A brief description of these projects is provided (below). These studies are not reviewed as part of this public health assessment, because they pertain to Fernald workers and not the community as a whole.

Lastly, the community group Fernald Residents for Environmental Safety and Health, Inc. (FRESH) has conducted an extensive survey of adverse health effects reported by residents of the Fernald community. The survey indicates that Fernald residents are concerned about various types of cancer and a variety of other non-cancer effects. A compilation of the survey findings is provided in the ACommunity Concerns[®] section of this report.

Fernald Risk Assessment Project

The CDC's National Center for Environmental Health (NCEH) conducted the FRAP to provide a comprehensive summary of the potential health effects of the Fernald site on the surrounding community ([CDC 1998, 1999](#)). The risk assessments used information in the Fernald Dosimetry Reconstruction Project (FDRP) ([Voilleque et al. 1995](#); [Shleien et al. 1995](#); [Kilough et al. 1998a, 1998b](#)), and demographic information about the population around the Fernald site, to produce community-level estimates of cancer risk. (The Fernald Dosimetry Reconstruction Project is discussed in [Appendix D](#) of this report.) Cancer risk was estimated for people exposed to radioactive materials released from the Fernald site during its years of operation - specifically, for persons who resided within the assessment domain (i.e., within 10 kilometers of the Fernald facility boundary) for any length of time between 1951 and 1988. There have been two phases of the FRAP to date.

Phase I

Because the results of the FDRP indicated that lung cancer was the most likely adverse health outcome associated with exposure to radionuclides produced while Fernald was operating, Phase I focused on potential lung cancer risk in the community ([CDC 1998](#)). Phase I's goal was to produce a realistic (not worst-case) estimate of the number of lung cancer deaths associated with Fernald-related radiation exposure in the assessment population. The assessment used realistic (not maximum) assumptions about factors affecting exposure.

To estimate risk for the entire community living in the assessment domain, NCEH researchers divided the assessment domain into 160 cells and obtained information about the number, age, and gender of people living in those cells during the years of plant operations. The researchers used software produced by FDRP to generate estimates of radiation exposure and accompanying risk of lung cancer for different subgroups within this population. Life-table methodology, which models mortality in a population over time, was used in conjunction with lung cancer risk estimates to estimate possible lung cancer deaths over time. Estimates were projected through 2088, the year in which someone first exposed in 1988 (the last year of plant operations) would turn 100. The number of background[®] lung cancer deaths that would normally be expected to occur in this population was also estimated.

The Phase I report estimated that between 40,000 and 53,000 people lived in the assessment domain for some period of time between 1951 and 1988. Lung cancer deaths in this population were predicted to be between 1% and 12% greater than the number expected if that population had not been exposed to radiation from Fernald. (This range was the 90% credibility interval; the median estimate was 3%.) This translates to a range of 25 to 309 lung cancer deaths, with a median of 85 deaths. The majority of these deaths were estimated to occur among smokers (65 deaths) rather than among people who have never smoked (20 deaths). Fernald-related lung cancer mortality was highest closest to and east of the site, with declining rates farther from and west of the site. Because the installation of containment measures in the K-65 silos in 1979 greatly reduced radon emissions, mortality in people first exposed between 1951 and 1979 was compared to mortality in people first exposed in 1980 and later. It was found that almost all of the estimated increase in lung cancer deaths occurred among those first exposed before 1980.

Phase II

Phase I of the FRAP focused on lung cancer mortality, primarily resulting from exposure to radon and radon decay products, because radon and radon decay products were estimated by the FDRP to comprise the majority of the radiation exposure dose, and radon and its decay products primarily affect the lung ([CDC 1998](#)). However, the FDRP also found that uranium and other radionuclides accounted for part of the radiation exposure dose. These radionuclides primarily affect body organs other than the lung. Therefore, Phase II focused on potential health effects resulting from exposure to radionuclides other than radon. The health outcomes addressed were kidney cancer, female breast cancer, bone cancer, and leukemia ([CDC 1999](#)). These cancers were selected based on scientific information and community concerns.

Unlike the Phase I evaluation, which was intended to provide a realistic estimate of increased lung-cancer mortality, the Phase II evaluation was intended to provide a screening-level estimate of the increased incidence of selected cancers. The Phase II report used estimates of the maximum Fernald-related radiation dose that members of the assessment population may have received to provide an upper-bound, or worst-case, estimate of the number of resulting cancers. It was assumed that all members of this population were breathing contaminated air, receiving external radiation exposure, and using contaminated irrigation water. In addition, it was assumed that all of the milk, eggs, fish, meat, and vegetables eaten by this population were contaminated by radiation. As in the Phase I report, the researchers used the software produced by the FDRP to generate estimates of radiation exposure based on these assumptions.

The Phase II report analyzed the same assessment population (people living within the assessment domain for any period of time between 1951 and 1988) as the Phase I report. This domain was divided into 12 geographical areas, for which risk estimates were produced; in addition, a risk assessment was produced for a hypothetical individual who received the maximum exposure previously described and drank contaminated well water. The FDRP report indicated that at least three off-site wells south of Fernald were likely contaminated with radionuclides by the mid-1960s. Of these, a well designated as Well 15 was found to have the highest concentrations of radionuclides. Thus, the contamination levels estimated over time for this well by the FDRP were used to derive estimates of maximum dose for an individual using contaminated well water for drinking and irrigation. The number of persons who were exposed to contaminated well water is likely very small. However, this exposure pathway is of particular interest, because the radiation dose to the organs considered in the Phase II report may be substantially greater among persons who drank, and irrigated their gardens with, water from contaminated wells.

To translate the estimated doses into cancer risks, the report used risk values recommended by the International Council on Radiation Protection (ICRP), the National Council on Radiation Protection (NCRP), and the U.S. Environmental Protection Agency (EPA). These values are based on the cancer experience of human populations exposed to ionizing radiation, primarily atomic-bomb survivors and people exposed to radiation for medical reasons. Based on these risk values, researchers produced upper-bound estimates of the number of cases of certain types of cancer that might occur in the assessment population as a result of exposure to radiation released from Fernald during its years of operation. This estimate was produced first for the hypothetical population that did not use contaminated well water. A new estimate was then produced using the assumption that all persons residing in the two areas 1 to 4 kilometers southeast and southwest of the site used contaminated well water. (This is a conservative assumption that likely greatly overestimates the number of people who used contaminated well water.) Estimates for Fernald-related incidences of kidney cancer, breast cancer, and bone cancer in the assessment population as a whole did not change when it was assumed that contaminated well water was used. The leukemia incidence estimate did increase from a range of 1 to 18 additional cases to a range of 3 to 23.

Including the assumption that a segment of the population used contaminated well water, the report estimated that exposure to Fernald-related radiation in the entire assessment population resulted in 23 or fewer additional cases of leukemia, 4 or fewer additional cases of kidney cancer, 4 or fewer additional cases of bone cancer, and 3 or fewer additional cases of female breast cancer over what would be expected in the assessment population in the absence of exposure to radiation from Fernald.

Individual risks to the small segment of the population that used contaminated well water were also estimated using a hypothetical individual exposure scenario. The median estimates of the percentage increase in the lifetime risk of cancer for this hypothetical individual were as follows: 0.7% for kidney cancer, 0.03% for breast cancer, 6% for bone cancer, and 6% for leukemia. Judging from the results of the Phase II report, CDC did not recommend a more detailed analysis of the potential risk for kidney, female breast, or bone cancer resulting from radiation released from the site.

Further Work

As a follow-up to the Phase I and Phase II studies, NCEH conducted a Feasibility Assessment for a Community-Based Epidemiologic Study of Lung Cancer and Radiation Exposures near the Former Feed Materials Production Center ([Garbe 1999](#)). Based on their assessment, they concluded that an in-depth epidemiological study of Fernald-related radiation exposures and lung cancer is not feasible at this time. Such a study would not address community concerns about cancer related to the site, primarily because the availability and quality of local records does not appear to be adequate for systematic, unbiased, and complete identification of past residents ([Garbe 1999](#)).

The Fernald Medical Monitoring Program (FMMP) and the Fernald Workers Medical Monitoring Program (FWMMP)

In January 1985, Fernald area residents filed a class action lawsuit against National Lead of Ohio (the Fernald site manager from 1954 to 1985) and the DOE. These legal actions resulted in the establishment of a Settlement Fund and two programs: the Fernald Medical Monitoring Program (FMMP) and the Fernald Workers Medical Monitoring Program (FWMMP).

The objectives of the FMMP are to (1) provide a complete medical evaluation of the current health status of eligible persons, (2) provide a comprehensive evaluation of risk factors for illnesses or diseases of participants, (3) provide education to participants on how to modify risk factors for illness or disease, and (4) establish a good baseline database which may be useful for subsequent epidemiological research (Pinney 1999a).

Persons who lived or worked within 5 miles of the former Feed Materials Production Center for any 2-year continuous period, between January 1, 1952, and December 18, 1984, are eligible to participate in the residents' program (FMMP). Persons who worked at the production center as employees of National Lead of Ohio or National Lead Industries are excluded from participating. Participation is on a volunteer basis. Participants initially complete questionnaires on health risk, health status, lifestyle, and possible exposure. A physician administers a history, a physical exam, and medical tests. The first set of formal examinations was initiated in December 1990. From 1990 to November 1998, medical examinations were offered every 3 years. As of December 1998, medical examinations are offered every 2 years. The FMMP is slated to operate for 25 years in total. Confidentiality of medical records is maintained, and data on participants are stored in a computerized database. Participants receive medical advice based on the results of their examinations and tests. In addition, each woman over 40 years old receives an annual mammogram.

As of June 1999, a total of 8,520 adult participants (persons 18 years or older at the time of their first examinations) have enrolled in the FMMP and had their first medical examinations. As of December 1, 1998, the age range of participants is 19 to 95, and slightly more than half (55%) of the participants are women, almost all (99%) are Caucasian, most (73%) are married, and most (84%) have education beyond high school (Pinney 1999b).

Medical examinations, questionnaires, follow-up documentation, and death certificates are sources of disease information collected for the FMMP. Information collected by questionnaire include new medical problems, hospitalizations, surgeries, smoking and alcohol consumption, and medications used. When needed, follow-up documentation (e.g., outside medical records such as death certificates, pathology reports, medical test reports, and operative and discharge summaries) of diseases are obtained for the participants (Pinney 1999b).

Two research questions are being addressed by the FMMP. The first question addresses whether the number of newly diagnosed cancer cases among participants of the FMMP, for the first 4 years in the program (first medical examination plus 48 months of follow-up), is greater than what would be expected among a similar population. The analysis includes FMMP participants (7,937 persons in FMMP) who had their first medical examinations before December 1, 1993, and who were diagnosed at the first medical exam plus 48 months (i.e., each participant contributes about 1 to 4 person-years). This analysis has been funded by FMMP which arose out of a class action lawsuit and settlement.

Analyses were performed for 16 cancer systems and for all cancer sites combined. Four different comparison populations are used in the analysis: (1) the National Cancer Institute's Surveillance, Epidemiology, End Result (SEER) data for all of the United States; (2) the SEER data for Ohio; (3) the Ohio Cancer Surveillance (for three counties, Butler, Warren, and Clermont); and (4) the Ohio Cancer Surveillance (for Ohio as a whole). The *a priori* best comparisons are the SEER data for Ohio and the tri-county Ohio Cancer Surveillance data.

The findings indicate that in the FMMP population, the number of new cancer cases for three types of cancer (urinary system and kidney/renal pelvis, melanoma of the skin, and prostate) was greater than expected. The incidence of urinary system cancer in the FMMP population was statistically significant with all four comparisons. Within the urinary system, the incidence for kidney/renal pelvis cancer was significant compared only to the tri-county Ohio area (2.50, 1.14 - 4.75). The incidence of melanomas of the skin and prostate cancer were significant compared only to the SEER Ohio data; e.g., melanomas (2.22, 1.11 - 3.97) and prostate cancer (1.53, 1.12 - 2.70). The researchers acknowledge that the greater than expected incidence of prostate cancer was possible due to the introduction of a new diagnostic test (i.e., PSA) that improved the identification of existing cases, rather than an actual increase in the number of new cases. Although not statistically significant, the expected number of new lung cancer cases among FMMP participants increased from 1% to 12% over the expected number of cases. This is consistent with the predictions made in the CDC's Community-Based Lung Cancer Risk Assessment (Pinney 1999b).

The researchers state that the FMMP volunteer population is representative of the general population, although they acknowledge possible sources of bias due to a 'healthy volunteer screening effect' and because a volunteer study population, rather than a representative sample of the entire Fernald community, was used. These results are considered to be screening-level, because the analysis only addressed whether there is an excess of a specific type of cancer in Fernald residents. No data analyses were performed to determine if this excess is related to historical radiation or chemical exposures from the Fernald site (Pinney 1999b).

The second research question being addressed is whether the rate of certain chronic medical conditions, reported by participants in the FMMP at the time of their first medical examination, is greater than the rate reported from national health databases - the National Health and Nutrition Examination Survey (NHANES) and National Health Interview Survey (NHIS). The chronic conditions being analyzed were selected by a review of the scientific literature, interviews with the medical community, and input from Fernald residents (Pinney 1999b). This phase of analysis of FMMP data is being funded by the ATSDR's Division of Health Studies. The findings are currently being reviewed by ATSDR prior to release to the public.

The FWMMP (for former workers) is similar to the FMMP (for residents) in many ways, although there are some important differences. It involves Fernald workers, and information collected from participants focuses on occupational histories. Participants are re-examined annually. The FWMMP has about 3,000 participants. Because the focus of this public health assessment is on community residents, rather than workers, this report does not discuss the FWMMP in depth.

Effects of Uranium-Contaminated Drinking Water on Urinary β -2-Microglobulin Concentration

A retrospective study, conducted by a Master of Science student from the University of Cincinnati College of Medicine, evaluated renal (kidney) effects from drinking water contaminated with uranium by residents living near the Fernald site (Kammer 1998). Contaminated wells were identified using water concentration measurements with results equal to or greater than 20 μ g uranium per liter of water. These measurements were made by contractors for DOE, ODH, and OEPA. The exposure group (25 people) was defined as participants in the FMMP who drank water from contaminated wells within the area of the South Plume, as characterized in 1991. The control group (569 people) consisted of Fernald area residents who resided within 4 to 5 miles of the Fernald site and who drank water from wells. The age and gender distribution of South Plume residents and control residents are similar (Kammer 1998).

The biological marker β -2-microglobulin was used to measure the effect of uranium on kidney function. This marker is not specific for uranium-induced renal toxicity, because there are numerous other diseases and chemicals (e.g., chronic active and viral hepatitis, predampsis, rheumatoid arthritis) that cause alterations in urinary β -2-microglobulin concentrations (Kammer 1998). Concentration measurements for urinary β -2-microglobulin were not available for all South Plume residents or for the entire control group. (They were available for 24 South Plume residents and for 499 people in the control group.) Likewise, concentrations of urinary β -2-microglobulin, standardized for creatinine, were only available for 22 South Plume residents and 496 control residents. Mean urine β -2-microglobulin concentrations in the South Plume and control groups were not statistically different, and mean urine β -2-microglobulin concentrations, standardized for creatinine, were also not statistically different.

Although the findings indicate that South Plume residents did not have increased urine β -2-microglobulin concentrations compared to the control group of residents, several issues must be considered when interpreting these results (Kammer 1998). The most important of these is the relatively imprecise estimation of uranium exposure and the use of urinary β -2-microglobulin as a biological marker of effect.

For exposure to uranium, water measurements were used to define the boundaries of the South Plume in 1991. Actual measurements of uranium concentrations at residences within the plume (and outside the plume) would provide more precise estimates of exposure concentrations, and changes in concentrations, over the period of exposure. This is important because uranium concentrations varied over time, and maximum concentrations were presumably present in the 1960s, not the 1990s. In addition, such data would help minimize misclassification bias (based on exposure).

The use of urinary β -2-microglobulin concentrations as a marker of effect, and the length of time between exposure and measurement of β -2-microglobulin concentration, may have hampered the ability of this study to detect positive associations if they were present. As mentioned previously, concentrations of uranium in the South Plume varied over time. The maximum period of exposure was presumably the 1960s; concentrations decreased substantially from the 1960s to the 1990s. Many residents were provided bottled water to drink in 1984. Measurements of urinary β -2-microglobulin concentrations were made in the late 1990s, thirty years or more after maximum exposure occurred. The kidneys of South Plume residents may have recovered from any toxicity by the time urinary β -2-microglobulin concentrations were measured. Few studies have examined the chronic effects of uranium on the kidney and its ability to repair itself once exposure to uranium has ended (Kammer 1998). While urinary β -2-microglobulin is a valid marker for acute toxicity, it may not be appropriate for use in chronic exposure conditions.

NIOSH Activities

NIOSH has conducted various investigations involving past and current workers at the Fernald site. At present, they are conducting an exposure assessment of hazardous waste, decontamination and decommissioning, and clean-up workers and a retrospective exposure assessment for workers at the Fernald plant. These and other NIOSH activities are not discussed in detail in this report, which addresses health issues related to the surrounding community. Further information about NIOSH's activities at the Fernald site can be obtained by calling or writing the NIOSH contact person listed in the AFor Additional Information@ section of this report.

COMMUNITY CONCERNS

Background

ATSDR representatives first met with members of the Fernald community in May 1992, during the initial visit to the Fernald area. Many times since then, ATSDR representatives have traveled to the Fernald area and met with various members of the community, both in public and private meetings. The purpose of the visits was to learn more about the Fernald site and to hear from community members about their health concerns. The most concerted efforts to compile community concerns in the Fernald community were public availability sessions (or open-house meetings) sponsored by ATSDR on December 6, 7, and 8, 1993. Four open-house meetings were held, two each in Crosby and Ross, at which concerned citizens met individually, in pairs, or in small groups with ATSDR representatives. The public availability sessions were advertised widely in local and area newspapers. The advertisements stated that the purpose of the availability sessions was to hear the community's health concerns related to the Fernald Environmental Management Project and the former Feed Materials Production Center. ATSDR representatives met with approximately 110 people and recorded their concerns, questions, and comments. Personnel from Boston University's School of Public Health, the U.S. Environmental Protection Agency's National Air and Radiation Environmental Laboratory (NAREL), and the National Center for Environmental Health (NCEH) assisted ATSDR staff at the public availability sessions.

The public availability sessions attracted a reasonable cross-section of the community, including residents affiliated with local government, representatives of local community organizations, and former workers at the site. Many of the people who attended the public availability sessions gave their names, but some spoke anonymously. ATSDR made every effort to maintain confidentiality for the residents who came to the sessions; representatives of the news media were not allowed to sit in on conversations or record them.

In addition to the public availability sessions, ATSDR representatives have attended meetings sponsored by DOE, Fernald Residents for Environmental Safety and Health, Inc. (FRESH), NCEH, and the National Institute for Occupational Safety and Health. ATSDR representatives have attended meetings

of the Fernald Citizens Advisory Board, formerly the Fernald Citizens Task Force, since March 1994. Upon receiving an invitation from the board (or task force), an ATSDR representative has occupied an *ex-officio* chair at the meetings since December 1995. Representatives also have attended meetings of the Fernald Health Effects Subcommittee (FHES) since its inception and have reviewed and compiled public comments sent to the FHES by mail (see below). ATSDR scientists have also reviewed information collected by the community group, FRESH, concerning health problems reported by local residents. Many Fernald residents have spoken with ATSDR personnel on the telephone, anonymously. ATSDR staff have also visited with community members in their homes or their automobiles.

With the assistance of personnel from NAREL, ATSDR conducted an environmental sampling program in the Fernald area. Our visits to the area to collect environmental samples, including setting up radon detection canisters in residents' homes, gave us ample opportunity to talk with many residents of the area, particularly those who live close to the facility.

Fernald Community Concerns

Throughout all of the meetings and activities sponsored and attended by ATSDR representatives, an ongoing list of community concerns related to the Fernald site has been kept. In many cases, ATSDR representatives do not know where some of the respondents live or lived, because this information was not always provided.

We have grouped the community concerns under the following headings:

HEALTH CONCERNS
Cancers
Non-Cancer Effects
ENVIRONMENTAL EXPOSURES
Air
Soil
Surface Water
Groundwater
Biota
SPECIFIC POPULATIONS
PROCEDURAL CONCERNS
Remediation
Lack of Trust
Emergency Response
Monitoring or Sampling
General
Recommendations by the Public

Of the concerns that the representatives heard, ATSDR did not record any that were addressed during the public availability sessions or other meetings, or any that had already been reported and recorded. A summary of the concerns, and ATSDR's responses, is presented in Appendix C of this report. Also included in Appendix C is a summary of concerns submitted to the FHES and compiled by FRESH.

CONCLUSIONS

ATSDR scientists evaluated chemicals and radioactive materials in completed and potential exposure pathways for the Fernald site. ATSDR scientists reached the following conclusions:

- One exposure pathway evaluated by ATSDR, ingestion of uranium in water from privately owned off-site wells in the South Plume, poses a *public health hazard* under *past* conditions at the site. The pathway poses a health hazard because available information indicates that people were exposed to contaminants at levels that could result in adverse health effects. Also, one pathway evaluated by CDC, inhalation of radon and radon decay products, poses a *public health hazard* under *past* conditions at the site. This pathway poses a public health hazard because available information and the estimation of exposures from modeling this pathway indicate that people were potentially exposed to contaminants at levels that could result in adverse health effects. Although human exposure to chemicals and radionuclides may have occurred via other exposure pathways, the levels and conditions of exposure (e.g., duration, frequency, route of exposure) were not sufficient to cause adverse health effects, even for the most sensitive individuals. Other exposure pathways contribute minimally to the total exposure to chemicals and radioactive materials by Fernald area residents under past conditions at the site.
- According to the information reviewed by ATSDR, *there are no exposure pathways that pose a public health hazard to the surrounding community under current conditions at the site.* However, ATSDR has only limited information about one current exposure pathway, ingestion of chemicals and radioactive materials in water from off-site privately owned wells. Therefore, ATSDR concludes that this pathway poses an *indefinite public health hazard* under current conditions at the site. Additional information about exposure (e.g., concentrations of chemicals and radioactive materials in these wells, number and location of wells with contamination) is needed to assess the level of health hazard for this pathway.
- *Future exposures* to chemicals and radioactive materials from the Fernald site pose *no apparent public health hazard* to surrounding communities under normal operating conditions.

RECOMMENDATIONS

Based on the information reviewed, ATSDR recommends the following:

Environmental Issues

- An in-depth assessment of *past* exposure to chemical and radioactive contaminants in privately owned residential wells near the Fernald facility should be conducted using available environmental sampling data and appropriate modeling techniques (e.g., spatial analysis, transport and fate modeling).
- DOE should continue monitoring groundwater in the South Plume. This monitoring should include analyses for contaminants that may be drawn into the South Plume (from sources other than the Fernald facility) due to groundwater remediation activities.
- An in-depth assessment should be conducted of *current* usage of, and potential exposure to chemical and radioactive contaminants from, privately owned residential wells that are most likely to be impacted by contaminants from the site. This assessment should use existing and new environmental sampling data. Analysis of samples should include a full spectrum of chemicals and radionuclides, from the Fernald site and other sources near it, that are being introduced into the South Plume by remediation activities at the Fernald site. If contamination of these wells is found at levels that pose a health hazard, additional activities should be considered to determine the source of contamination and the need for further public health actions.
- More frequent analyses should be conducted for uranium/thorium decay products, especially radium 226 and radium 228, in all biota samples collected off site of the Fernald facility. There should be routine quality control reviews of these analyses.
- Monitoring for radon and radon daughters should be continued in the Fernald area during remediation activities at the site, particularly those involving the K-65 silos. Consideration should be given to using alpha-track detectors in addition to continuous monitors in case of power supply failures, interference from drastic temperature changes, etc. Additional sampling locations should be added (especially on the west fence-line) during remediation of the silos.

Additional Considerations

- Further evaluation of possible risk factors for adverse health effects among participants in the Fernald Medical Monitoring Program should be considered.
- Additional health education activities, including workshops for health care providers in the Cincinnati area, should be considered.

PUBLIC ACTION HEALTH PLAN

The Public Health Action Plan (PHAP) for the Fernald Environmental Management Project (formerly the Feed Material Production Center) contains a description of the public health actions taken and those planned to be taken by ATSDR, DOE, and/or responsible state agencies at, and in the vicinity of the site based on the recommendations of this public health assessment. The purpose of the PHAP is to ensure that this public health assessment not only identifies public health hazards but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. The public health actions that are completed, being implemented, or planned are as follows:

Public Health Actions Taken:

- DOE is monitoring and plans to continue monitoring for radon and radon decay products during remediation activities at the site.
- ATSDR's Division of Health Education and Promotion (DHEP) has co-sponsored, with the University of Cincinnati College of Medicine and Mercy Hope Partners, two educational programs for health care professionals in the Cincinnati area.

Public Health Actions Planned:

- ATSDR's Division of Health Education and Promotion (DHEP) will continue to develop health care provider training programs for health care professionals in the Fernald community. Primary care providers will be given information about studies and assessments being conducted by ATSDR, NCEH, and NIOSH and important findings from the Fernald Medical Monitoring Program. In addition, they will be provided information needed to diagnose, treat, and counsel persons concerned about the potential health impact of the Fernald site.
- ATSDR's DHEP will also work with members of the Fernald Health Effects Subcommittee to develop and implement community health education programs as requested by the community.

ATSDR is discussing the implementation of the additional recommendations contained in this public health assessment with DOE and the responsible state agencies. The resulting actions planned or agreed to by these agencies will be reported in the final release of this public health assessment.

FOR ADDITIONAL INFORMATION

ATSDR's Division of Health Assessment and Consultation (Public Health Assessment)

Carol Connell

Health Physicist, Energy Section

Federal Facilities Assessment Branch

Division of Health Assessment and Consultation

Agency for Toxic Substances and Disease Registry

1600 Clifton Road, NE, Mailstop E-56

Atlanta, GA 30333

(404) 639-6060

ATSDR's Division of Health Education and Promotion (Health Care Providers Workshops)

Joe Maloney

Division of Health Education and Promotion

Agency for Toxic Substances and Disease Registry

1600 Clifton Road, NE, Mailstop E-42

Atlanta, GA 30333

(404) 639-6350

CDC's National Center for Environmental Health (NCEH)

(Fernald Dosimetry Reconstruction Project, Fernald Risk Assessment Project, and Fernald Health Effects Subcommittee)

Mike Donnelly

Deputy Chief, Radiation Studies Branch

National Center for Environmental Health

Centers for Disease Control and Prevention

1600 Clifton Road, NE, Mailstop E-39

Atlanta, GA 30333

(404) 639-2550

CDC's National Institute for Occupational Safety and Health (NIOSH)

Dave Pedersen, Ph.D.

CDC-NIOSH

Mailstop P03/R19

Cincinnati, OH

(513) 841-4223

Ohio Environmental Protection Agency

Tom Ortko

Ohio Environmental Protection Agency

Southwest District Office

401 East Fifth Street

Dayton, OH 45402-2911

(937) 285-6073

Ohio Department of Public Health

Jim Colleli

Ohio Department of Health

Bureau of Radiation Protection

246 North High Street

Columbus, OH 43226

(614) 644-2727

Environmental Protection Agency, Region V

Gene Jablonowski

U.S. Environmental Protection Agency

Region V, SRF-5J

Chicago, IL 60604-3590

(312) 886-4591

Fax: (312) 353-8426

U.S. Department of Energy

Johnny W. Reising

Fernald Remedial Action Project Manager

DOE Fernald Area Office

P. O. Box 538705

Cincinnati, OH 45253-8705

(513) 648-3155

Fernald Citizens Advisory Board (FCAB)

James C. Bierer, Chair

Fernald Citizens Advisory Board

Mailstop 76, P. O. Box 538704

Cincinnati, OH 45253-8704

(513) 648-6478

Fernald Medical Monitoring Program and Fernald Workers Medical Monitoring Program

Susan Pinney, Ph.D.

Associate Professor

Department of Environmental Health

College of Medicine

University of Cincinnati

Holmes Hospital, 1st Fl, Rm. 1001

Eden and Bethesda Avenues

Cincinnati, OH 45268-0684

(513) 568-0684

Fernald Residents for Environmental Safety and Health, Inc. (FRESH)

Edwa Yocum
9860 Hamilton Cleves Pike
Harrison, OH 45030
(513) 738-1659

PREPARERS OF REPORT

Brenda K. Weis, M.S., Ph.D.
Health Scientist
Federal Facilities Assessment Branch
Division of Health Assessment and Consultation

Carol Connell
Health Physicist
Federal Facilities Assessment Branch
Division of Health Assessment and Consultation

Contributors

ATSDR acknowledges the contribution to this public health assessment made by Ms. Jerri Anderson and Mr. Kevin Liske, GIS Analysts, Electronic Data Systems, Inc.

ATSDR acknowledges the contribution to this public health assessment made by Dr. Scott Telofski from U.S. EPA's National Air and Radiation Environmental Laboratory, Montgomery, AL.

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Reviewers of Report

Burt J. Cooper, M.S.
Chief, Energy Section
Federal Facilities Assessment Branch
Division of Health Assessment and Consultation
ATSDR

Sandy Isaacs
Chief, Federal Facilities Assessment Branch
Division of Health Assessment and Consultation

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APPENDIX A - SELECTION OF CONTAMINANTS

ATSDR scientists used several criteria for selecting the chemical and radioactive contaminants for the exposure pathways identified at the Fernald site. These criteria include (1) environmental levels exceeding the media-specific comparison value, (2) noted community health concerns, and (3) the quality and extent of sampling data with which to evaluate potential exposure and human health hazard. For inorganic compounds (metals) and radionuclides, background values may also be considered, since some of these substances occur naturally.

For chemicals, the highest environmental concentration detected in off-site samples is compared with the media-specific comparison values to determine if it is high enough to warrant further evaluation. Generally, if the contaminant concentration exceeds one or more media-specific comparison values, then the contaminant is evaluated further in the *Exposure Pathways Analyses* and *Public Health Implications* sections of the public health assessment.

Media-specific comparison values are chemical contaminant concentrations in specific environmental media (e.g., soil, water, air) that are considered to be *Asafe* under default assumptions about exposure. Comparison values are *not* thresholds of toxicity. While concentrations at or below comparison values may be considered safe, it does not automatically follow that any environmental concentration that exceeds a comparison value would produce adverse health effects. To reiterate, if a chemical concentration exceeds a comparison value, this does not mean that a public health concern exists; rather, it indicates the need to consider the contaminant further in the *Exposure Pathways Analyses* and *Public Health Implications* sections of the report.

The media-specific chemical comparison values used in this public health assessment include ATSDR's environmental media evaluation guides (EMEGs), cancer risk evaluation guides (CREGs), and reference dose media evaluation guides (RMEGs) (ATSDR 1992). Similar values developed by the U.S. Environmental Protection Agency (EPA) and used in this health assessment are maximum contaminant levels (MCLs) and Region III Risk-Based Concentrations (EPA 1989).

EMEGs are media-specific chemical comparison values that are developed for soil, water, and air. EMEGs are derived from ATSDR's minimal risk levels (MRLs), which are presented in ATSDR's Toxicological Profiles. An MRL is a health-based comparison value representing an estimate of daily human

exposure to a chemical that is not likely to pose appreciable risk of adverse non-cancer effects over a specified duration of exposure. MRLs are developed for acute (less than 14 days), intermediate (15 to 364 days), and chronic (365 days or more) exposure durations.

RMEGs are media-specific chemical comparison values derived from EPA's reference doses (RfDs). RfDs are health-based guidelines for non-cancer effects. An RMEG is used when an EMEG is not available for a chemical. An RfD is an estimate of daily exposure to a contaminant below which adverse non-cancer effects are not likely to occur over a lifetime (EPA 1989).

CREGs are estimated chemical contaminant concentrations in a specific medium that are anticipated to result in one excess cancer in one million persons exposed over a lifetime. CREGs are calculated from EPA's cancer slope factors (CSFs), also known as cancer potency factors (CPFs). CPFs are cancer potency estimates for chemicals shown to be carcinogenic in either animals or humans (EPA 1989).

MCLs are contaminant concentrations in water derived by EPA to be protective of public health (considering the availability and economics of water treatment technology) over a lifetime at an ingestion rate of 2 liters of water per day. MCLs are enforceable regulatory values.

APPENDIX A - SELECTION OF CONTAMINANTS

(for Completed Exposure Pathways)

Groundwater Pathway (Privately owned Wells)

ATSDR scientists used sampling data collected from 1981 to the present to select contaminants for groundwater pathways. A summary of sampling programs and activities that include privately owned off-site wells is in Table A-1. A brief description of these programs and activities follows.

Uranium in Privately Owned Wells

In 1981, the Department of Energy (DOE) first detected uranium at concentrations above background levels in privately owned drinking water wells south of the facility. Because of the elevated uranium concentrations, DOE began routine monitoring of the private wells near the Fernald site in 1982. In 1984, the monitoring program was formally established as the Radiological Environmental Monitoring Program. Sampling results were reported in the annual Site Environmental Reports (DOE 1972 - 1999). In addition, property owners could request sampling of residential well water for uranium, in which case the one-time results would be reported to the owners. If any of the samples showed above-background concentrations of uranium, the owners had the option to participate in the routine monitoring program (DOE 1972 - 1999).

Of the wells routinely sampled by the facility, only four wells (numbers 12, 13, 15, and 17) located south of the Fernald site showed uranium concentrations above the proposed drinking water standard of 20 µg/L (Volleque et al. 1995; DOE 1972 - 1999). Because maximum uranium concentrations in these wells exceeded both proposed drinking water standards and ATSDR's media-specific comparison value for ingestion of chemical uranium, we selected total uranium as a chemical contaminant of concern for groundwater pathways. Uranium concentrations were not measured in private wells before 1981, but were estimated using existing sources of environmental data and information (Volleque et al. 1995). Table A-2 presents maximum yearly concentrations of uranium in private wells 12, 13, 15, and 17, during the period from 1982 to 1997.

From 1986 to 1988, groundwater monitoring was also being conducted at the Fernald facility under the Resource Conservation Recovery Act (RCRA). The RCRA Groundwater Monitoring Program included four private wells off site of the facility. In 1989, DOE began consolidating groundwater monitoring efforts at the Fernald site to avoid duplication of sampling efforts under the various site programs (e.g., Remedial Investigation/Feasibility Study, Routine Monitoring Program, RCRA). In 1990, DOE contractors managed all long-term, environmental monitoring efforts at the site under the Comprehensive Groundwater Monitoring Program. In 1996, this program became part of the Integrated Environmental Monitoring Program (DOE 1997).

Table A-1. Summary of sampling programs and activities that include privately owned wells off site of the Fernald facility

Date of Sampling	Program or Activity	Parameters Analyzed in Well Water	Wells Sampled and Comments
1981 to 1996	Routine Monitoring: Radiological Environmental Monitoring Program and Comprehensive Groundwater Monitoring Program; special sampling (owner=s request); and State Route 128 (SR 128) study, in 1990 (DOE 1972B1999)	Monthly, quarterly, or annual analyses for total uranium and 16 metals and elements (Primary and Secondary Drinking Water Standards); monthly analyses for nitrate-nitrogen (from 1983 to 1985)	The Fernald facility conducted routine monitoring of up to 37 off-site private wells; special sampling was conducted on a one-time basis at the owner=s request; and the SR 128 study sampled 18 wells and some cisterns along a 2-mile stretch of SR 128, south of the Fernald site, and analyzed water for total uranium. The facility conducted monthly analyses for nitrate-nitrogen (1983 to 1985) for up to 26 off-site private wells.
1978 to 1982	Sampling by International Technology, Inc. (IT 1986)	Sampling of off-site groundwater and analyses for total uranium	Seventeen off-site groundwater samples, five of which had uranium concentrations above background (0.8 µg/L). Maximum concentration was 268 µg/L. No information about whether any wells were private.
1985 to 1988	Sampling conducted by the Ohio Department of Health (ODH 1988)	Sampling of off-site private wells and cisterns, mostly from 1985 to 1986	Ohio Department of Health (ODH) sampled more than 200 private wells, selected on a voluntary basis at the owner=s request. Three wells had uranium concentrations above background and the proposed water standard of 20 µg/L. Two of these wells were used by local industries; only one of them was used for drinking water (maximum uranium concentration in this well was 370 ± 10 µg/L). DOE reported a 30% higher uranium concentration than

			ODH in split samples from this well.
1986 to 1988	RCRA Program monitoring (DOE 1972B1999)	Quarterly analyses for metals, elements, total phenol, and nitrates	Fernald facility sampled water from off-site private wells 8, 12, 15, 17, and 26 only
1996 to present	Integrated Environmental Monitoring Program (DOE 1972B1999)	Quarterly analyses for total uranium	Fernald facility sampled water from off-site private wells 12, 13, and 14 only

Table A-2. Maximum yearly concentrations of uranium (in µg/L or ppb) in private wells 12, 13, 15, and 17 off site of the Fernald facility from 1982 to 1995

Year	Well 12	Well 13	Well 15	Well 17	Comments
1960s	918 to 4,144*	NA	918 to 4,144*	918 to 4,144*	Concentration range represents the highest <i>estimated</i> yearly median concentration (lower bound) to the highest <i>estimated</i> yearly 95 th percentile (upper bound) concentration based on uranium measurements in the storm sewer outfall ditch (SSOD) and Paddy-s Run and known releases to the SSOD (Voilleque et al. 1995).
1982	310	NA	554	99	Fernald facility began sampling private wells.
1983	306	NA	578	68	
1984	270	NA	365	68	
1985	243	NA	360	55	
1986	332	1	378	61	
1987	410	1	330	170	Wells 12, 15, and 17 used for monitoring purposes only
1988	300	2	310	73	Wells 12, 15, and 17 used for monitoring purposes only
1989	350	1	320	54	Wells 12, 15, and 17 used for monitoring purposes only
1990	210	2	330	56	Wells 12, 15, and 17 used for monitoring purposes only
1991	190	14	310	54	Wells 12, 13, 15, and 17 used for monitoring purposes only

1992	307	30	260	50	Wells 12, 13, 15, and 17 used for monitoring purposes only
1993	176	78	264	NS	Wells 12, 13, and 15 used for monitoring purposes only; well 17 not sampled after September 1992
1994	162	99	219	NS	Wells 12, 13, and 15 used for monitoring purposes only; well 17 not sampled after September 1992
1995	177	93	177	NS	Wells 12, 13, and 15 used for monitoring purposes only; well 17 not sampled after September 1992
1996	41	120*	NS	NS	Maximum uranium concentrations were detected in well 13 in 1996; concentrations in well 12 were reported in SED, and concentrations in well 13 were estimated from trend analysis (IEMP 1998).
1997	145*	60*	NS	NS	Concentrations in wells 12 and 13 were estimated from trend analysis (IEMP 1998). Wells 15 and 17 were not included in routine groundwater sampling in 1997.
<p>Key * - estimated concentrations NA = data not available NS = not sampled SED = Site-Wide Environmental Database µg/L = micrograms of uranium per liter of water ppb = parts per billion</p> <p>Sources: DOE 1972B1999; Voilleque et al. 1995</p>					

Other Contaminants in Privately Owned Wells

As part of routine environmental monitoring at the Fernald facility, private well water samples were analyzed for metals from 1986 to 1993 (DOE 1972 - 1999). The maximum concentrations of metals detected in the samples are presented in Table A-3. Iron and manganese were frequently detected in private well water at concentrations above secondary federal drinking water standards, although high concentrations of iron and manganese are typical for groundwater in this area (DOE 1972 - 1999). Manganese and manganese compounds were not routinely used in operations at Fernald. Manganese was a minor impurity (< 1%) in uranium ores and ore concentrates (DOE 1994). Potential sources of manganese at the Fernald site are the local soils, the waste pit area, flyash piles, South field area, solid waste landfill, Plant 1 area, Plant 2/3 area, Plant 8 area, and laboratory area.

Maximum concentrations of iron and manganese in all the wells were below ATSDR's media-specific comparison values. Therefore, they were not selected as contaminants of concern for groundwater. Several other metals (e.g., arsenic, cadmium, chromium, lead, selenium, and zinc) were found at concentrations above federal drinking water standards in a very few private well water samples collected from 1986 to 1995. Because these contaminants were detected so infrequently, we could not identify any patterns of contamination in the wells, nor could we identify any individual wells that were consistently found to be contaminated. Therefore, we did not select any of these metals as contaminants of concern for groundwater pathways.

Table A-3. Maximum concentrations of metals found in samples from private wells off site of the Fernald facility from 1986 to 1993

Year	Iron Concentration (in mg/L or ppm) and (number of wells above standard of 0.3 ppm/ number of wells sampled)	Manganese Concentration (in mg/L or ppm) and (number of wells above standard of 0.05 ppm/ number of wells sampled)	Other Metals Detected and Comments
1986	4.43 (13/24)	0.393 (13/24)	No other metal concentrations exceeded standards.

1987	2.95 (11/25)	0.45 (17/25)	No other metal concentrations exceeded standards.
1988	6.09 (15/26)	0.399 (14/26)	No other metal concentrations exceeded standards.
1989	3.6 (14/25)	0.56 (15/25)	Concentrations of arsenic and selenium in one well (well 19) exceeded standards, and concentrations of selenium in 12 other wells exceeded standard. Maximum concentrations of arsenic and selenium found in all wells were 0.071 and 0.034 ppm, respectively.
1990	17 (16/32)	1.8 (15/32)	No other metal concentrations exceeded standards.
1991	16 (15/34)	0.49 (17/34)	Concentrations of cadmium and zinc exceeded standards in one well each (wells 11 and 15), with concentrations of 0.02 ppm and 32 ppm, respectively.
1992	12 (18/37)	0.39 (17/37)	No other metal concentrations exceeded standards.
1993	4.0 (16/36)	0.36 (18/36)	Concentrations of lead exceeded standards in five wells (wells 11, 12, 19, 22, and 29), with a maximum concentration of 0.043 ppm. The drinking water standard changed from 0.05 ppm to 0.15 ppm in 1992.
1994	3.57 (12/31)	0.298 (13/31)	Concentrations of arsenic exceeded standards in one well (well 19), with a concentration of 0.063 ppm.
1995	31.37 (13/32)	0.43 (18/32)	Concentrations of arsenic, cadmium, chromium, and lead exceeded standards, each in one of four wells, with maximum concentrations of 13.1 ppm (well 3), 0.012 ppm (well 12), 0.0231 ppm (well 34), and 0.0196 ppm (well 12), respectively.
<p>Key mg/L = milligram per liter ppm = parts per million</p> <p>Sources: DOE 1972B1999; Voilleque et al. 1995</p>			

APPENDIX A - SELECTION OF CONTAMINANTS

(for Potential Exposure Pathways)

Soil Pathway

ATSDR scientists used soil sampling data collected from 1971 to the present to select contaminants for soil pathways. These data are summarized in Table A-4 (below). Uranium was detected in off-site samples at concentrations that exceed media-specific comparison values. Maximum uranium concentrations were found in samples collected along the northeastern and eastern facility boundary and along the outfall line to the Great Miami River. Uranium was selected as both a *chemical* and *radioactive* contaminant of concern for soil pathways.

Off-site soil samples were analyzed for organic compounds and metals on a few occasions from 1991 to 1993. Samples were collected at five to six different locations east and northeast of the facility boundary. A greater number of samples were collected and analyzed for metal contaminants. These samples were considered representative of background concentrations because they were collected upwind of air pathways and upstream of surface water and groundwater pathways from the Fernald site. Overall, there is a limited amount of information on levels of organic compounds and metals in off-site soils. Analyses for these contaminants were not routinely conducted because these chemicals were not generated or used in significant quantities during routine operations at the facility.

One organic compound, benzo(a)pyrene, was positively detected and quantified in one (sample SS-58) of six off-site soil samples. Although the level of benzo(a)pyrene in this sample was slightly above ATSDR's media-specific comparison value, benzo(a)pyrene was not selected as a contaminant for soil pathways because this location represents the only potential point of human exposure, and any exposure to this concentration is expected to occur with a limited frequency and duration.

Several metals (e.g., arsenic, barium, beryllium, boron, cadmium, chromium, lead, manganese, and thallium) were found at levels above media-specific comparison values in soils off site of the Fernald facility. However, none of these metals was present at concentrations that exceeded background concentrations. Of the metals in surface soil, beryllium, cadmium, and thallium were not selected as contaminants for the following reasons: (1) they were detected infrequently in surface soil samples, (2) maximum concentrations in surface soil samples were just above the lower limit of analytical detection, and (3) maximum concentrations were similar to (or lower than) background soil concentrations.

Table A-4. Summary of analytical data used for selecting chemical contaminants in soil pathways

Contaminant*	Sample with Maximum	Maximum	Mean	Sample	Range of	Freq. of	Media-Specific Comparison
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	Concentration	Concentration (mg/kg or ppm)	Concentration ^H (mg/kg or ppm)	Year	Contamination	Detection	Value (mg/kg or ppm)
UraniumC Current Exposure Past Exposure^I	C-2 (just off site, east, excavated)	87	NA	1991	ND(11)B87	NA	6 (ATSDR EMEG)
	2393 (off site, south)	18	NA	1990		NA	
	BS-3 (eastern facility boundary)	137	NA	1973	2.9B136.5	NA	
PAHC Benzo(a)pyrene	SS-58 (just off site, NE)	0.110	NA	1993	NDB0.110	1/6	0.088 (benzo(a)pyrene) (EPA III)
Arsenic	SS-58 (just off site, NE)	5.3	5 (n=31)	1993	1.9B5.3	5/5	0.37 (EPA III)
	1873 (NW of site)	9.2		1992	ND(2.9)B9.2	22/26	
Barium	SS-56 (just off site, east)	237	77 (n=35)	1993	14.3B237	5/5	100 (ATSDR EMEG)
	1873 (NW of site)	331		1992	31B331	30/30	
Beryllium	SS-55 (just east of site)	1.3	NA	1993	ND(0.11)B1.3	1/5	
	1873 (NW of site)	0.6	1	1992	ND(0.47)B0.6	1/30	
Boron	No samples immediately off site	NA	NA	NA	NA	NA	200 (ATSDR EMEG)
	1831 (NW of site)	1,140	97 (n=14)	1992	ND(12.2)B1,140	14/30	
Cadmium	No positive detections off site	NA	NA	NA	NA	0/5	
	1762 (NW of site)	0.95	0.7 (n=30)	1992	ND(0.47)B0.95	7/35	
Chromium	SS-56 (just off site, east)	12.1	11 (n=35)	1993	5.6B12.1	5/5	10 (hexavalent) (ATSDR EMEG)
	1758 (NW of site)	18.1		1992	6.7B18.1	30/30	
Lead	SS-58 (just off site, NE)	28.5	18 (n=32)	1993	8.3B28.5	5/5	NA
	1875 (NW of site)	40.3		1992	ND(15.6)B40.3	27/29	
Magnesium	C-7 (off site, east; excavated)	24,800 ^I	2,421 (n=35)	1991	1,540B24,800	5/5	300 (ATSDR EMEG)
	SS-58 (just off site, NE	[3,060]		[1993]	1,020B3,590	30/30	
	1758 (NW of site)	3,590		1992			
Manganese	SS-56 (just off site, east)	3,420	921 (n=35)	1993	400B3,420	5/5	300 (ATSDR EMEG)
	1873 (NW of site)	4,850		1992	189B4,850	30/30	
Thallium	SS-58 (just off site, NE)	0.42	0.4 (n=35)	1993	ND(0.24)B0.42	2/5	
	1827 (NW of site)	0.58		1992	ND(0.48)B0.58	1/30	

Key

mg/kg = milligrams of substance per kilogram of soil

ppm = parts per million

PAH = polyaromatic hydrocarbon

NW = northwest

NE = northeast

Range of Contamination = analytical range of contaminant concentration

ND = not detected

Estimated Exposure Dose	=	Calculated using the equation provided above (in mg/kg/day)
ABS	=	Absorption factor, or fraction of uranium in soil that is absorbed from the gastrointestinal tract into the blood (unitless) = Distribution factor, or fraction of the absorbed dose that is distributed to the kidney (unitless)
DC	=	Distribution factor, or fraction of the absorbed dose that is distributed to the kidney (unitless)
BW	=	Body weight, in kilograms (kg)
CF	=	Conversion factor, or 10 ³ micrograms per gram
Kidney WT	=	Average weight of two kidneys, in grams (e.g., 120 g per kidney for a child, and 310 g per kidney for an adult)

The assumptions ATSDR used to calculate exposure doses for chemicals in completed or potential exposure pathways are provided in the discussion section for each pathway (i.e., groundwater, soil, air, surface water, biota). ATSDR used the estimated exposure doses in the Exposure Pathways Analyses section of this report as screening-level analyses of public health hazard. ATSDR then compared the estimated exposure doses with health-based guidelines for the chemical, route of exposure, and exposure duration (i.e., acute, intermediate, or chronic). When an estimated exposure dose for a chemical exceeded its corresponding health-based guideline, ATSDR then evaluated the chemical further (see the Public Health Implications section of the report), using a more in-depth, weight-of-evidence approach.

Health guidelines include ATSDR's minimal risk levels (MRLs) and EPA's reference doses (RfDs) for non-cancer effects, and cancer risk ranges (1 in 10,000 to 1 in 1,000,000 excess cancer risk) for cancer effects. MRLs and RfDs are conservative values because they are based on levels of exposure reported in the scientific literature, which represent no-observed-adverse-effect-levels (NOAELs) and lowest-observed-adverse-effect-levels (LOAELs) for the most sensitive outcome for a given route of exposure (e.g., ingestion or inhalation). In addition, uncertainty factors are applied to NOAELs and LOAELs to account for variation in the human population and uncertainty in extrapolating from animals to humans, and for added protection of the most sensitive individuals. Therefore, MRLs and RfDs may have uncertainties spanning an order of magnitude or more.

When evaluating the potential for cancer to occur, ATSDR scientists often use cancer risk ranges based on EPA's cancer potency factors (CPFs). CPFs define the relationship between exposure doses and the likelihood of increased risk of developing cancer over a lifetime. CPFs are developed using data from animal or human health studies and often require extrapolation from high exposure doses administered in animal studies to the lower exposure levels typical of exposure to environmental contaminants. CPFs represent the upper-bound estimates of the probability of developing cancer at a defined level of exposure; therefore, they tend to be conservative and may even overestimate the actual risk, in order to account for uncertainties in the data used in the extrapolation.

ATSDR scientists may also use cancer effect levels (CELs) reported in the scientific literature to determine possible cancer effects from exposure at the exposure doses estimated for the contaminant of concern and pathway. The CELs are similar to LOAELs for non-cancer effects, but they represent minimal levels of effect for cancer effects. The CELs are derived from animal and human health studies and represent the lowest dose of a chemical in a study, or group of studies, that produces a significant increase in the occurrence of tumors in the exposed population as compared to an unexposed group.

Radiation

The following general equation is used to estimate human exposure dose (committed effective or equivalent doses) for radioactive contaminants:

$$\text{Committed Effective or Equivalent Dose (lifetime dose in sieverts from specified intake)} = C \times IR \times EF \times ED \times IDC$$

Where:

Committed Equivalent Dose	=	Equivalent dose rate received in a particular tissue or organ over a person's lifetime following the intake of radioactive materials into the body
Committed Effective Dose	=	Sum of the committed tissue or organ equivalent doses and the appropriate organ or tissue weighting factor integrated over the person's lifetime
C	=	Contaminant concentration, in becquerels per gram (Bq/g), or becquerels per liter (Bq/L)
IR	=	Intake rate for ingestion or inhalation
EF	=	Exposure frequency, or number of exposures per unit of time of exposure
ED	=	Exposure duration, or the duration over which exposure occurs
IDC	=	Age-dependent ingestion or inhalation dose coefficients, in sieverts per becquerels (Sv/Bq)

The committed effective and equivalent doses for radioactive contaminants in completed or potential exposure pathways are presented in the discussion section for each pathway (i.e., groundwater, soil, air, surface water, biota). At the screening-level analysis phase in the exposure pathways analyses, ATSDR used the maximum concentrations to estimate committed effective or equivalent doses (to the target organ). In assessing public health implications, we determined the likelihood of developing a fatal cancer (or organ-specific cancer) over a person's lifetime based on the committed effective or equivalent doses.

APPENDIX B - EXPOSURE DOSES AND HEALTH-BASED GUIDELINES (for Potential Exposure Pathways)

(Soil Pathway)

ATSDR's estimated ingestion doses and health-based guidelines for metals in soil pathways are presented in Table B-1. We estimated these doses assuming the same hypothetical exposure scenarios described in the Exposure Pathways Analyses section of this report under Soil Pathway. Under this scenario, we assumed exposure to a child, 1 to 6 years old and weighing 13 kilograms, who plays in off-site areas and exhibits Apica-like behavior. We assumed this child ingests 200 milligrams (mg) of maximally contaminated soil per day, on 35 days a year, for 4 consecutive years. Additional assumptions that we used to estimate exposure doses for metals selected as contaminants for soil pathways (i.e., arsenic, barium, boron, chromium, lead, and manganese) are presented below. We have also provided information about health-based guidelines for these contaminants. As Table B-1 shows, none of ATSDR's estimated exposure doses for metals in off-site soils exceed the health-based guidelines.

Arsenic

The maximum concentration of arsenic detected in off-site surface soil is 9.2 milligrams per kilogram of soil (mg/kg) in a sample (sample 1873) collected northwest of the site. Because this area is upwind of any airborne transport and upstream of any water transport pathways from the Fernald site, it is considered representative of background conditions, as it is unlikely to have been affected by site contaminants.

The maximum concentration of arsenic detected in surface samples near the site is 5.3 mg/kg in a sample (sample SS-58) collected just outside the eastern facility boundary in 1993. This area has been affected by contaminants released from the site, as other chemicals and radionuclides that have also been detected on site have been identified in soil there.

Arsenic concentrations are lower in areas that may have been affected by the Fernald site. Nonetheless, to estimate exposure doses, ATSDR scientists used the maximum concentration of arsenic in all off-site samples (9.2 mg/kg), as they did not know whether human exposure to this level is possible.

Table B-1. Estimated exposure doses and health-based guidelines for chemical (metal) contaminants in soil pathways

Contaminant	Estimated Ingestion Dose for a Child (mg/kg/day)*	Health-Based Guidelines ¹
Uranium	Estimated doses are presented in the AExposure Pathways Analyses, @ ASoil Pathway@ section.	2 x 10 ⁻³ mg/kg/day (ATSDR int/chr oral MRL) 0.1 to 1 µg/g (dose to the kidney) (Morris and Meinhold 1995)
Arsenic	8 x 10 ⁻⁶	3 x 10 ⁻³ mg/kg/day (ATSDR chr oral MRL) 1.5 (mg/kg/day) ⁻¹ (EPA oral CSF) 0.0043 (per µg/m ³) (EPA inhal unit cancer risk)
Barium	5 x 10 ⁻⁴	0.07 mg/kg/day (EPA oral RfD)
Boron	2 x 10 ⁻³	0.01 mg/kg/day (ATSDR int oral MRL) 0.09 mg/kg/day (EPA oral RfD)
Chromium (VI)	3 x 10 ⁻⁵	5 x 10 ⁻³ mg/kg/day (EPA oral RfD) 0.2 mg/day (ATSDR interim oral intake)
Lead	5.9 µg/dLI	10 µg/dL (CDC/ATSDR blood screening level, children)
Manganese	7 x 10 ⁻³	0.07 mg/kg/day (ATSDR interim oral guideline)
<p>Key</p> <p>MRL = Minimal Risk Level int = intermediate chr = chronic int/chr = intermediate/chronic RfD = Reference Dose CSF = EPA=s Cancer Slope Factor CDC = Centers for Disease Control and Prevention EPA = U.S. Environmental Protection Agency µg/dL = micrograms of lead per deciliter of blood µg/g = milligrams of lead per gram of kidney mg/kg/day = milligrams of substance per kilogram of body weight per day mg/day = milligrams of chromium per day</p> <p>* Equations used to calculate exposure doses are described in the introduction to this appendix.¹ Health-Based Guidelines are discussed in the introduction to this appendix.</p> <p>¹ Dose presented as micrograms of lead per deciliter of blood (see text for discussion).</p>		

Arsenic is present in the environment in both inorganic and organic forms. (Note: the term *Aorganic*® refers to compounds containing carbon and hydrogen.) Inorganic forms of arsenic predominate in soils and are more toxic than the organic forms (NEPI 1998). When humans and other animals are exposed to inorganic arsenic, they metabolize it to the much less toxic, methylated organic form, which is readily excreted from the body in urine. This methylation is effective as long as the inorganic arsenic intake remains below a level of 0.2 to 1 mg of arsenic per day (ATSDR 1998a), indicating that people can tolerate a certain level of arsenic exposure without experiencing adverse effects. At higher exposure levels, the body's capacity to detoxify arsenic may be exceeded or saturated, leading to increased blood levels of arsenic and possible adverse effects.

Saturation of the body's detoxification mechanism may explain why both non-cancer and cancer effects of arsenic appear to have a threshold, or minimum effective dose. Adverse health effects may result when exposure levels exceed the threshold. In addition, a growing body of scientific evidence suggests that arsenic carcinogenicity may result from mechanisms other than direct attack on genetic material, which supports the belief that there is a threshold for arsenic (ATSDR 1998a).

The amount of arsenic taken into the body following exposure depends to a large extent on the solubility of the arsenic compound. Soluble arsenic compounds are almost completely absorbed into the blood from the gastrointestinal tract (stomach and small intestine) while less soluble compounds have a lower absorption rate. Inorganic compounds vary widely, from 10% to 95%, in their solubility (NEPI 1998). ATSDR scientists do not have specific information about the types (and solubilities) of arsenic compounds present in surface soils; therefore, they made very conservative assumptions that *all* arsenic was present as *inorganic* compounds that are *completely* soluble (i.e., 100% absorbed from the gastrointestinal tract into the blood).

ATSDR's estimated exposure dose for ingestion of arsenic-contaminated soil is presented in Table B-1. This dose is equivalent to 0.0001 mg arsenic per day, which is many times lower than the levels required to saturate the body's arsenic detoxification mechanisms.

The lowest levels at which toxicity, including skin and gastrointestinal effects, has been reported in humans are 0.014 to 0.05 mg/kg/day. These findings are based on a study of Taiwanese persons who drank arsenic-contaminated water for 45 years (Tseng et al. 1968; Tseng 1977). EPA derived a health guideline of 3×10^{-4} mg/kg/day for adverse effects on the skin (e.g., hyperpigmentation, keratosis) and a cancer slope factor of 1.5 (mg/kg/day)⁻¹ for skin cancer, based on the Taiwanese study (IRIS 1998). Although ATSDR's estimated doses are lower than levels shown to cause adverse effects in the Taiwanese people, the study has important limitations that must be considered when public health hazard is evaluated.

First, the study reported an association between arsenic in drinking water and skin cancer but failed to account for a number of potential confounding factors, including exposure to other nonwater sources of arsenic, genetic susceptibility to arsenic, and poor nutritional status of the exposed population. Therefore, arsenic exposure may have been underestimated in the study, possibly leading to an overestimation of cancer effects associated with exposure levels. Second, the cancer slope factor for arsenic is based on the conservative assumption that no threshold exists for cancer. As discussed previously, arsenic carcinogenicity appears to have a threshold. Lastly, the adverse effects observed in the Taiwanese people were due to absorbed arsenic. ATSDR scientists assumed that *all* arsenic off site of the Fernald facility was present as completely soluble, inorganic arsenic compounds. These assumptions resulted in a dose estimate that was not realistic and most likely overestimated the actual absorbed dose.

Considering these limitations, ATSDR scientists conclude that adverse effects are not likely from ingestion of arsenic-contaminated soil. This is supported by the fact that ATSDR's estimated doses were many times lower than levels required to saturate arsenic detoxification mechanisms in the body. It is important to note that the maximum arsenic concentrations that were used to estimate dose were similar to background levels, indicating regional soil conditions.

Barium

The maximum concentration of barium detected in off-site surface soil is 331 mg/kg in a sample (number 1873) collected northwest of the site. This area is considered representative of background conditions because it is unlikely that site contaminants would have affected the area. The highest level of barium detected in surface samples near the site, and likely to have been affected by contaminants released from the site, is 237 mg/kg in a sample (SS-56) collected just outside the eastern facility boundary in 1993. ATSDR scientists used the maximum concentration of barium detected in off-site surface soil to estimate exposure doses. For purposes of estimating exposure dose, we assumed that 100% of the ingested barium was absorbed from the gastrointestinal tract into the blood.

The health guideline for ingestion of barium was derived by EPA and takes into account recent findings from both human (epidemiologic) studies of adult males and from various chronic animal (rodent) studies of barium in drinking water (IRIS 1998). The guideline is derived from a no-observed-adverse-effect-level (NOAEL) of 0.07 mg/kg/day, which is the dose that does not increase blood pressure when barium is ingested over a chronic duration. Our estimated dose for ingestion of barium-contaminated soil is many times lower than this NOAEL. Therefore, adverse human health effects are not likely to result from exposure to barium at the maximum concentrations found in soil off site of the Fernald facility.

Boron

No boron measurements have been made within close proximity to the Fernald facility boundary. Several samples have been collected northwest of the Fernald facility in an area that is considered representative of background conditions because it is unlikely to have been affected by contaminants released from the site.

The maximum concentration of boron in off-site surface soil is 1,140 mg/kg. This value is considered a statistical outlier, and not likely to represent actual environmental concentrations, because it is almost 100 times higher than other boron measurements made in the same area. Of a total of 30 samples collected in off-site surface soil, 14 have positive detections for boron. Of these 14 samples, the next highest boron concentration is 27.6 mg/kg, detected in sample 1758 from the same area. The average boron concentration in the 14 samples is 97 mg/kg when the value of 1,140 mg/kg is included in the averaging, but the average is only 16.8 mg/kg when this extreme value is excluded. Although it is unlikely that contaminants from the Fernald site have affected this area northwest of the site, ATSDR scientists estimated exposure dose using the maximum concentration of 1,140 mg/kg, because they do not know whether it represents an actual environmental concentration, and whether human exposure at this level is possible.

No human studies were identified in the literature for ingestion of boron or boron compounds. Health guidelines for boron ingestion are based on reproductive (testicular) effects in dogs exposed to borax and boric acid in their diet for intervals ranging from 90 days to 2 years (Weir and Fisher 1972; ATSDR 1992a; IRIS 1998). A no-observed-adverse-effect-level (NOAEL) of 8.8 mg/kg/day was reported for the 2-year study. An additional study of rats fed borax and boric acid in their diet for 2 years reported a lowest-observed-adverse-effect-level (LOAEL) of 58.5 mg/kg/day based on adverse effects on the testes (Weir and Fisher 1972). Of these animal species, the dog is considered more sensitive than the rat to boron toxicity (IRIS 1998). ATSDR's estimated dose for the Fernald site is many times lower than the NOAEL and LOAEL reported in these studies.

Chromium

The maximum concentration of chromium in off-site surface soil is 18.1 mg/kg in a sample (number 1758) collected northwest of the site. This area is considered representative of background conditions, because it is unlikely that site contaminants would have affected it. The maximum concentration of chromium in surface samples near the site is 12.1 mg/kg in a sample (SS-56) collected just outside the eastern facility boundary in 1993. ATSDR scientists used the maximum concentration of chromium detected in all off-site samples, or 18.1 mg/kg, to estimate exposure doses.

Chromium occurs in the environment in several forms, depending on its valence state, e.g., trivalent (III) chromium or hexavalent (VI) chromium. Chromium in the environment (e.g., soil, water) and in the body is more commonly found as chromium III than as chromium VI (ATSDR 1998b). Chromium VI is considerably more toxic to humans than the chromium III. ATSDR scientists do not have specific information about the form of chromium present in surface soils off site of the Fernald facility. Therefore, we made a conservative assumption that *all* chromium found in off-site soil is in the more toxic, hexavalent form.

ATSDR has not established a health guideline for ingestion of chromium because the available data are insufficient or too contradictory to establish minimum levels of effect (e.g., LOAELs). Because chromium is an essential nutrient in the body, the National Research Council has established a range of Aestimated safe and adequate daily dietary intakes® (ESADDIs) for chromium; the upper end of the range is 0.2 mg/day (NRC 1989). This value has been adopted by ATSDR as an interim guideline for oral exposure to chromium VI and chromium III compounds (ATSDR 1998b).

ATSDR's interim guideline is similar to the health guideline established by EPA for chronic ingestion of chromium VI. EPA's guideline is 0.005 mg/kg/day and is based on studies in animals (IRIS 1998). EPA's guideline is equivalent to an adult exposure of 0.35 mg/day and a child exposure of 0.065 mg/day. The estimated exposure dose for this pathway is many times lower than the upper bound ESADDI for chromium. Therefore, adverse effects from ingestion of chromium-contaminated soil off site of the Fernald facility are not likely.

Lead

The maximum concentration of lead in off-site surface soil is 40.3 mg/kg in a sample (number 1875) collected northwest of the site. This area is considered representative of background soil conditions. The highest level of lead detected in surface samples near the site is 28.5 mg/kg in a sample (SS-58) collected northeast of the site in 1993. ATSDR scientists used the maximum concentration of lead in all off-site samples, or 40.3 mg/kg, to estimate exposure doses, because they do not know whether human exposure at this level is possible.

Health guidelines for lead are based on blood lead concentrations rather than exposure doses. A strong positive correlation exists between exposure to lead-contaminated soils and human blood lead levels. Generally, blood lead levels rise 3 to 7 micrograms per deciliter (µg/dL) of blood for every 1,000 mg/kg increase in soil or dust lead levels (ATSDR 1992b). The relationship between lead in environmental media and lead in blood has been described mathematically by the following equation (where *ln* is the natural logarithm):

$$\ln(\text{blood lead conc}) = 0.879 + 0.241 \ln(\text{environmental lead conc.})$$

The increase in blood lead concentration as a function of soil lead concentration is not linear. At high lead concentrations in soil (many times higher than maximum concentrations in soil off site of Fernald) the rate of increase in blood lead tapers off. The equation is considered appropriate when exposure to lead occurs at relatively low levels (i.e., levels similar to maximum concentrations found in off-site surface soil). The equation is used to calculate blood lead concentrations assuming exposure to lead in soil via multiple routes of exposure (e.g., ingestion, inhalation).

The Centers for Disease Control and Prevention (CDC) and ATSDR consider a blood lead level of 10 µg/dL for children a health-based screening level (ATSDR 1992b; CDC 1991). When estimated or measured blood lead levels are 10 µg/dL or higher, then further evaluation of potential health hazard is warranted. Blood lead levels above 10 µg/dL in children may be associated with neurological and behavioral problems, such as impaired learning ability and reduced intelligence quotient (ATSDR 1992b, 1997a; CDC 1991).

Bioavailability of lead refers to the ability of humans and animals to absorb lead into their bodies following exposure by ingestion or inhalation. Bioavailability of lead can vary considerably depending on various factors, such as the size and chemical composition of the ingested or inhaled particles. In the absence of information about the bioavailability of the lead from soil off site of Fernald, ATSDR scientists made the conservative assumption that *all* lead present off site was *completely* (100%) bioavailable for uptake by humans.

Using the above equation, which correlates dietary intake of lead to blood lead level, ATSDR's estimated blood lead level for people exposed to contaminated soil off site of the Fernald facility is 5.9 µg/dL. This level is considerably lower than the screening level of 10 µg/dL. This difference is not surprising, however, because the maximum environmental lead concentrations off site are lower than levels shown to be correlated with elevated blood lead levels.

Manganese

The maximum concentration of manganese detected in off-site surface soil is 4,850 mg/kg in a sample (number 1873) collected northwest of the site. This area is considered representative of background conditions because it is unlikely that site contaminants would have affected the area. The maximum concentration of manganese in surface samples near the site is 3,420 mg/kg in a sample (SS-56) collected just outside the eastern facility boundary in 1993. ATSDR used the maximum concentration of manganese in all soil samples, or 4,850 mg/kg, to estimate exposure doses.

Manganese is a naturally occurring element that is essential for normal functioning of the human body. Toxicity in humans has been associated with both deficiencies and excess intake of manganese. Manganese deficiencies have not been observed in the general population, because manganese is found in a variety of foods, including whole grains, nuts, leafy vegetables, and tea. Suboptimal manganese intake may be more of a concern than excess intake: several diseases, including multiple sclerosis, cataracts, osteoporosis, and epilepsy, may be associated with low levels of manganese in the body (ATSDR 1997b). Two cases of manganese deficiency have been reported for persons consuming from 0.11 to 0.34 mg of manganese per day (Doisy 1973; Friedman et al. 1987).

It is difficult to define safe and adequate daily intakes of manganese because several factors, both environmental and biological, greatly influence an individual's response to manganese (ATSDR 1997b). Because manganese is essential in the human diet, the National Research Council has established a range of *Estimated safe and adequate daily dietary intakes*® (ESADDIs) for manganese. The ESADDIs are 0.3 to 2.0 mg/day for children under age 6 and 2 to 5 mg/day for persons over age 11 (NRC 1989). The World Health Organization estimates that the average consumption of manganese in the adult diet ranges from 2 to 9 mg/day, and that an intake of 8 to 9 mg/day is *A*perfectly safe.® ATSDR has established an interim health guideline for manganese ingestion of 0.07 mg/kg per day (ATSDR 1997b). This guideline is equivalent to a daily intake of 4.9 mg for a 70-kg adult and 0.91 mg for a 13-kg child.

ATSDR's estimated ingestion dose for manganese (Table B-1) is equivalent to a daily intake of 0.49 mg for adults (assuming a body weight of 70 kilograms) and 0.091 mg for children (assuming a body weight of 13 kilograms). Therefore, ATSDR's estimated ingestion doses are lower than the ATSDR interim health guideline for ingestion of manganese and lower than *A*safe and adequate® daily intakes established by the National Research Council and World Health Organization. Manganese deficiency is not likely for Fernald area residents, however, because additional sources of manganese are contributed by the diet and possibly the environment (manganese is naturally high in groundwater in the Fernald area). Considering these additional sources of manganese exposure, estimated daily intakes for Fernald area residents are likely to be within *A*safe and adequate® ranges and are not likely to result in adverse health effects.

EXPOSURE DOSES AND HEALTH BASED GUIDELINES

(for Potential Exposure Pathways)

Air Pathway

ATSDR's estimated inhalation doses and health-based guidelines for *metals* in air pathways are presented in Table B-2. We estimated these doses assuming the same two hypothetical exposure scenarios described in the *A*Exposure Pathways Analyses® section of this report, under *A*Air Pathway.®

Under scenario #1, we assume exposure to a child, 1 to 6 years old and weighing 13 kilograms, who inhales airborne contaminants while playing near the site. The airborne contaminants are from resuspended soil. We assumed this child inhales 5 cubic meters of maximally contaminated air per day, on 351 days a year, for 6 consecutive years. Under scenario #2, we assume exposure to an adult farmer who weighs 70 kilograms and inhales airborne contaminants while performing heavy work near the site. We assumed this farmer inhales 51 cubic meters of maximally contaminated air per day, on 351 days a year, for 10 consecutive years (or the period from 1989 to 1998) (EPA 1999; ATSDR 1993; Killough et al. 1998).

For both scenarios, we assume that soil contaminated with metals becomes resuspended in air and is a source of potential exposure to off-site residents via air pathways. Because there are no measurements of metal concentrations in air at the site, we estimated the airborne concentrations using *measured* concentrations of metals in surface soil and *measured* concentrations of total suspended particulates in air off site of the facility.

For both scenarios, we also assume that 100% of the airborne concentration of metal is deposited into the respiratory tract and is completely available for absorption into the blood. These are conservative assumptions considering that the chemical form of each metal in off-site surface soil is not known for the Fernald site, the bioavailability of metals varies considerably by compound, and resuspended soils are likely to be large (10 microns in diameter or more) and, therefore, deposited in the upper regions of the respiratory tract where absorption is minimal (NEPI 1998). Additional information about maximum concentrations of metals in off-site surface soil is provided in the *A*Exposure Pathways Analyses® section of this report, under *A*Soil Pathway.® Additional information about the methods ATSDR used to estimate airborne concentrations of metals in air is provided in the *A*Exposure Pathways Analyses® section of this report under *A*Air Pathway.®

In the following section, we have provided additional information about health-based guidelines for the metal contaminants in air pathways. As Table B-2 shows, none of ATSDR's estimated exposure doses for metals in off-site air exceed health-based guidelines.

Table B-2. Estimated exposure doses (for a child and an adult farmer) and health-based guidelines for chemical (metal) contaminants in air pathways

8 x 10⁻³ mg/m³ (ATSDR int inhal MRL-insoluble uranium) 3.4 x 10⁻⁴ mg/m³ (ATSDR chr inhal MRL-soluble uranium)

Contaminant	Estimated Airborne Concentration (mg/m ³)	Estimated Inhalation Doses (mg/kg/day) for a Child and an Adult Farmer*		Health-Based Guidelines*
		Child	Adult	
Uranium	Estimated airborne concentrations and exposure doses are presented in the <i>A</i> Exposure Pathways Analyses, @ <i>A</i> Air Pathway® section of this report.			8 x 10 ⁻³ mg/m ³ (ATSDR int inhal MRL-insoluble uranium) 3.4 x 10 ⁻⁴ mg/m ³ (ATSDR chr inhal MRL-soluble uranium)
Arsenic	2 x 10 ⁻⁶	8 x 10 ⁻⁷	1 x 10 ⁻⁶	0.0043 (per µg/m ³) (EPA inhal unit cancer risk)
Boron	5 x 10 ⁻⁴	2 x 10 ⁻⁴	3 x 10 ⁻⁴	10 mg/m ³ (OSHA PEL) 0.01 mg/kg/day (ATSDR int oral MRL) 0.09 mg/kg/day (EPA oral RfD)
Chromium (VI)	7 x 10 ⁻⁶	3 x 10 ⁻⁶	5 x 10 ⁻⁶	5 x 10 ⁻⁴ mg/m ³ (ATSDR int inhal MRL) 0.012 (per µg/m ³) (EPA inhal unit cancer risk) 3 x 10 ⁻³ mg/kg/day (EPA oral RfD) 0.2 mg/day (ATSDR interim oral intake)
Manganese	2 x 10 ⁻⁶	7 x 10 ⁻⁴	1 x 10 ⁻⁴	0.04 mg/m ³ (ATSDR chr inhal MRL) 0.07 mg/kg/day (ATSDR interim oral guideline)

Key

MRL = Minimum Risk Level

inhal = inhalation

int = intermediate

chr = chronic

int/chr = intermediate/chronic

EPA = U.S. Environmental Protection Agency

OSHA = Occupational Safety and Health Administration

PEL = permissible exposure limit

* Equations used to estimate exposure doses and the Health-Based Guidelines are discussed in the introduction to Appendix BC Estimation of Exposure Doses and Health-Based Guidelines.

Arsenic

When humans and other animals are exposed to inorganic arsenic, that metabolize it to the much less toxic, methylated organic form, which is readily excreted from the body in urine. This methylation is effective as long as the inorganic arsenic intake remains below a level of 0.2 to 1 mg of arsenic per day (ATSDR 1998a), indicating that people can tolerate a certain level of arsenic exposure without experiencing adverse effects. At higher exposure levels, the body's capacity to detoxify arsenic may be exceeded or saturated, leading to increased blood levels of arsenic and possible adverse effects.

ATSDR's estimated dose for incidental inhalation is presented in Table B-2 of this appendix. The dose is equivalent to 5×10^{-8} (or 0.00000005) mg/day, which is many times lower than levels required to saturate the body's arsenic detoxification mechanisms. The dose is based on an estimated arsenic particulate concentration of 2×10^{-4} $\mu\text{g}/\text{m}^3$ that we calculated using the methods described previously in this appendix.

EPA has derived a unit cancer risk for inhalation of inorganic arsenic of 0.0043 per $\mu\text{g}/\text{m}^3$ arsenic in air, based on the incidence of lung cancer among male smelter workers (IRIS 1998). The unit risk can be interpreted to mean that there are 0.0043 additional lung cancer cases expected for every $\mu\text{g}/\text{m}^3$ increase in arsenic concentration. Based on this unit cancer risk and our estimated airborne arsenic concentration, there is no expected increased risk of cancer from inhalation of arsenic-contaminated soil off site of the Fernald facility. Again, this is supported by the fact that our estimated exposure dose for arsenic is many times lower than levels required to saturate detoxification mechanisms for arsenic in the body.

Boron

A study of workers in the borax industry who were employed an average of 11 years reported that average exposures of 4.1 mg/m³ are associated with dryness of the mouth, nose, and throat; sore throat; and productive cough (Garabrant et al. 1984). Borates are considered mild irritants at concentrations exceeding the Occupational Safety and Health Association's (OSHA) Permissible Exposure Level (PEL) of 10 mg/m³ in the workplace (ATSDR 1992a). Our estimated boron airborne concentration is many times lower than the OSHA PEL for boron, indicating that adverse health effects from incidental inhalation of boron-contaminated soil are not likely.

Chromium

ATSDR's health guideline for intermediate (less than 1 year) inhalation of particulate chromium VI compounds is 5×10^{-4} mg/m³. This guideline is based on respiratory effects, such as slightly decreased lung function among workers exposed to chromium for an average of 2.2 years (Lindberg and Hedenstierna 1983; ATSDR 1998b). The no-observed-adverse-effect-level (NOAEL) reported for these effects is 0.001 mg/m³ (or 1 $\mu\text{g}/\text{m}^3$).

ATSDR's estimated exposure dose for incidental inhalation is 1×10^{-8} (or 0.00000001) mg/kg/day. This dose is based on a chromium air particulate concentration of 6×10^{-7} mg/m³ that we estimated using methods described previously in this appendix. Our estimated chromium airborne concentration is many times lower than the health-based guideline. The actual particulate concentration of chromium VI from airborne soils off site of the Fernald facility is probably even lower than estimated (above) because ATSDR scientists assumed that all chromium was present as chromium VI, and it is more likely that the soils contained both chromium VI and less toxic chromium III compounds.

EPA has established a unit cancer risk of 0.012 per $\mu\text{g}/\text{m}^3$ chromium in air, based on a lung cancer mortality rate in chromate-exposed male workers compared to the U.S. white, male population (Mancuso 1975; IRIS 1998). The unit risk can be interpreted to mean that there are 0.012 additional lung cancer deaths expected for every $\mu\text{g}/\text{m}^3$ increase in chromium exposure concentration. The worker studies involved exposure to both chromium III and chromium VI, although it was assumed that only chromium VI contributed to lung cancer deaths, and that no less than one-seventh of total chromium exposure involved chromium VI. Other scientists have argued that the assumption that the ratio of chromium III to chromium VI is 6 to 1 may lead to a seven-fold underestimation of cancer risk. Because the smoking habits of chromate workers were assumed to be similar to those of the general U.S. white male population, it has also been argued that this assumption may lead to an overestimation of cancer risk, because it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population (IRIS 1998).

Based on this unit cancer risk and our estimated airborne concentrations, there is no expected increased risk of cancer from inhalation of chromium-contaminated soil off site of the Fernald facility. However, when considered together with the possible contribution from radionuclide exposure to lung cancer occurrence in Fernald area residents (CDC 1998), any addition to lung cancer risk may be important.

Manganese

ATSDR's health guideline for chronic (more than 1 year) inhalation of manganese is 4×10^{-6} mg/m³ (ATSDR 1997b). The guideline is based on neurological effects in foundry workers exposed to manganese dust, at concentrations ranging from 0.2 to 1.4 mg/m³ (median = 0.14 mg/m³) for 1 to 35 years (regren 1990). Exposed workers had below-average scores on neurobehavioral tests such as reaction time and finger tapping. A lowest-observed-adverse-effect-level (LOAEL) of 0.14 mg/m³ was reported for this study. The health-based guideline is considerably lower than the reported LOAEL, because of uncertainty in extrapolating from occupational to chronic (continuous) exposure to account for effects from continuous exposure and because of differences in the toxicity of different forms of manganese exposure in the available studies (ATSDR 1997b). The findings of this study are supported by other studies involving occupational exposure to manganese among battery and alloy factory workers (Roels et al. 1987, 1992; Mergler et al. 1994). Workers in these later studies had decreased performance in neurobehavioral tests and other neurological deficits from chronic exposure to various manganese compounds in dusts.

Our estimated airborne concentration of manganese is 0.002 mg/m³ for air pathways at the Fernald site. Although our estimated airborne concentration is higher than the health-based guideline, there is considerable margin of uncertainty in the derivation of the guideline from the reported LOAEL (ATSDR 1997b). Our estimated air concentration is several times lower than the reported LOAEL for neurotoxic effects in workers (ATSDR 1997b).

Because manganese is essential in the human diet, the National Research Council has established a range of estimated safe and adequate daily dietary intakes (ESADDIs) for manganese. The ESADDIs are 0.3 to 2.0 mg/day for children less than under age 6 and 2 to 5 mg/day for persons over age 11 (NRC 1989). The World Health Organization estimates that the average daily consumption of manganese in the adult diet ranges from 2 to 9 mg/day, and that an intake of 8 to 9 mg/day is perfectly safe. ATSDR has established an interim health guideline for manganese ingestion of 0.07 milligrams (mg) manganese per kilogram (kg) body weight per day (ATSDR 1997b). This guideline is equivalent to a daily intake of 4.9 mg for a 70-kg adult and 0.91 mg for a 13-kg child.

ATSDR's estimated manganese inhalation doses for air pathways (Table B-2) are equivalent to a daily intake of 0.02 mg/day for adults (assuming a body weight of 70 kilograms) and 0.009 mg/day for a child (assuming a body weight of 13 kilograms). Therefore, our estimated exposure doses are lower than ATSDR's interim health guideline for ingestion of manganese and lower than safe and adequate daily intakes established by the National Research Council and World Health Organization. Manganese deficiency is not likely for Fernald area residents, however, because additional sources of manganese are contributed by the diet and possibly the environment (manganese is naturally high in groundwater in the Fernald area). Considering these additional sources of manganese exposure, estimated daily intakes for Fernald area residents are likely to be within safe and adequate ranges and are not likely to result in adverse health effects.

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APPENDIX C - COMMUNITY CONCERNS

As stated previously in this report, ATSDR has been compiling community concerns expressed by Fernald residents since 1993. We have grouped the community concerns on the following pages under the following headings:

HEALTH CONCERNS

- Cancer
- Non-Cancer Effects

ENVIRONMENTAL EXPOSURES

- Air
- Soil
- Surface Water
- Groundwater
- Biota

SPECIFIC POPULATIONS

PROCEDURAL CONCERNS

- Remediation
- Lack of Trust
- Emergency Response
- Monitoring or Sampling
- General
- Citizens Recommendations

This structure for presenting the comments and concerns is somewhat arbitrary; for example, some concerns that we present as statements under one heading appear again as questions under another heading. Although we have not intentionally reported individual concerns under more than one heading, some of the concerns were voiced by more than one person and, depending on how the concerns were expressed, may appear under more than one heading. We adopted this structure to show how the concerns were expressed and to emphasize the recurring topics that we heard. We have paraphrased the comments, and we have presented most of them in the first person to emphasize the personal nature of the conversations that took place during the private meetings.

At the end of this section, we have summarized concerns gathered by the Fernald Health Effects Subcommittee and by the community group, Fernald Residents for Environmental Safety and Health, Inc. (FRESH).

Health Concerns

Cancer

Cancer is classified as a group of diseases that arise from normal cells that become abnormal and begin to grow uncontrollably. These abnormal cells may invade surrounding normal tissue and spread to distant parts of the body (metastasize). Although cancer can originate in different parts of the body, different types of cancer have some common characteristics. Substances that cause cancer are known as carcinogens. Carcinogens can be chemical, physical (e.g., radiation), or biological (e.g., viral). Most scientists agree that the process that causes cancer is complex and involves the interaction of environment, genes, and lifestyle factors. Other factors that may play a role are socioeconomic status and access to health care. The overall risk for cancer increases with age, as does the risk of cancer mortality (Laszlo 1988).

Although many different risk factors are associated with the different types of cancers that people develop, many people who get the disease do not have a readily identifiable risk factor (ACS 1999). Although many studies have examined how environmental exposure may increase a person's risk of developing cancer, more studies are needed on the interaction of environmental, genetic, and lifestyle factors. Many studies have examined populations, not individual risk. If you are concerned about your own or a family member's risk of developing cancer, you should consult your personal physician. The local chapter of the American Cancer Society also has information on risk factors and prevention.

Skin Cancer

I am concerned about skin and lung cancer.
I am concerned about skin lesions and lip cancer.
My husband had facial cancer. He did not work at Fernald.
A child's father had skin cancer and fungus on his foot.

Around December 18 - 25, 1984, Fernald had its last big air release. At the time, I had an outdoor wedding. After that my husband developed skin cancer and my son developed a bad skin rash and a spot on his lungs. My husband used to eat vegetables grown in our garden and fish from the Great Miami River, my son is a non-smoker.

There have been at least six cancers in my old neighborhood (Thrush Road), including lung cancer in a non-smoker. My grandmother had skin cancer. What health effects could uranium in the groundwater have caused?

Skin cancer is one of the most common types of cancers in the United States. There are two main types of skin cancers, non-melanoma and melanoma. Non-melanoma is the most common type of skin cancer. Most non-melanoma cancers are either basal cell carcinoma or squamous cell carcinoma. Basal cell carcinoma - the most prevalent type of skin cancer - is usually found on the head and neck. Squamous cell carcinoma is the second most common type of skin cancer. It usually occurs on the face, neck, ear, lips, and back of the hand. Some of the known risk factors for non-melanoma skin cancer are exposure to ultraviolet (UV) radiation or sunlight, fair skin, chronic exposure to certain chemicals (such as arsenic and industrial tar), and exposure to radiation (ACS 1999).

Melanoma is a more serious form of skin cancer that occurs in the melanocytes, which are cells that produce melanin, the substance that gives skin its color. Although melanoma tends to appear on the trunk of fair-skinned men and the lower legs of fair-skinned women, other types of people and other body parts can be affected. Melanoma accounts for approximately 4% of all skin cancer diagnoses, but 79% of all skin cancer deaths. During the past 20 years the incidence of melanoma has doubled in the United States. The risk factors for melanoma include an atypical mole, fair skin, family history of the disease, and treatment with immune suppression agents (ACS 1999).

Some studies suggest that dermal exposure to polycyclic aromatic hydrocarbons (PAHs) in the workplace may increase a person's risk for skin cancer (Soll-Johanning et al. 1998; Boffetta et al. 1997; Moran 1992; Nelson 1987). However, similar studies involving environmental exposures do not provide evidence for an association with skin cancer occurrence (Purde and Rahu 1979; Hussain et al. 1998). This may be due to the fact that PAHs are present in the environment at generally lower concentrations than generally present under occupational conditions. For example, the concentrations of PAHs in surface soils off site of the Fernald facility are many times lower than the levels shown to cause adverse health effects in occupational studies. In addition, PAHs are present in the environment as mixtures of both carcinogenic and essentially non-carcinogenic individual PAHs. The individual PAHs comprising occupational mixtures may differ from mixtures present in the environment. Therefore, occupational studies may not always be relevant for assessing health hazard from exposure to environmental mixtures of PAHs.

One study showed an increased risk for non-melanoma skin cancer in a population potentially exposed to the radionuclide radium 226 (Black et al. 1994). However, this finding was of borderline statistical significance, and the overall study was hampered by many confounding factors, making interpretation of the finding difficult.

Preliminary analyses of data from the Fernald Medical Monitoring Program (FMMP) suggest that the number of new cases of melanoma may be greater than expected among Fernald residents, as compared to the Surveillance Epidemiology and End Result (SEER) data for Ohio (Pinney 1999). However, the findings of these initial analyses are being evaluated further to determine whether the observed increases warrant further investigation.

Based on available information, ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for skin cancer in the surrounding population.

Respiratory (Lung & Bronchus) Cancer

I am concerned about skin and lung cancer.
I've had breast cancer and lung cancer and currently have brain cancer.
Three of my brothers died of cancer; one died of lung cancer.
My husband worked at the plant and died of lung cancer in 1982.
My church congregation (in Crosby) has a large number of cancer illnesses; many women have had lung and breast cancer.

There have been at least six cancers in my old neighborhood (Thrush Road), including lung cancer in a non-smoker. My grandmother had skin cancer. What health effects could uranium in the groundwater have caused?

Lung cancer can begin in any part of the lung and can spread (metastasize) to other parts of the body. There are two major types of lung cancer: Small Cell Lung Cancer (SCLC), which makes up about 10% of lung cancers, and Non-Small Cell Lung Cancer (NSCLC), which makes up approximately 80% of lung cancers. NSCLC can be further divided into squamous cell carcinoma, adenocarcinoma, and large cell undifferentiated carcinoma. Some lung cancers can have the characteristics of both SCLC and NSCLC (ACS 1999).

Lung cancer is the leading cause of cancer deaths in males and females in the United States. The average age at diagnosis is 60 years old. Risk factors for lung cancer includes smoking tobacco, chronic exposure to environmental tobacco smoke (second hand smoke), recurring lung infection with tuberculosis (TB) and some types of pneumonia, and a family history of lung cancer. Some studies show that exposure to air pollution in some urban areas can cause a slight increase in the risk for lung cancer in those populations. There is also some suggestive evidence that smoking marijuana can increase the risk for lung cancer (ACS 1999).

Exposure to radon and radon daughters is also a risk factor for lung cancer (Lubin et al. 1997; Samet et al. 1990). Although it is mostly a concern for people who live in certain areas of the country where radon gas tends to accumulate in their homes, it has also been recognized as a public health concern for the general population. Testing of indoor air and proper ventilation in homes can reduce the risk for lung cancer due to indoor exposure to radon.

The CDC's Fernald Risk Assessment Project predicted the number of lung cancer deaths that are likely to occur among residents (residing within 10 kilometers of the facility), through 2088, as a result of exposure to radiation from the Fernald facility during its period of operation (Kilgough et al. 1998). They predicted a 1% to 12% greater than expected number of lung cancer deaths in the Fernald community, compared to a community without exposure to radiation from the Fernald site. The increase in lung cancer mortality was presumed to result primarily from exposure to radon and radon daughters from the K-65 silos on the Fernald site. Almost all of the increased lung cancer deaths occurred among persons first exposed to radon emissions from the Fernald site before 1980, when emissions were highest. The results of the Fernald Risk Assessment Project are considered predictive, as they have not been confirmed in an analytical epidemiologic study. Additional information about the CDC's Risk Assessment Project is provided in the A-Health Outcome Data[®] section of this report.

Epidemiologic studies of workers occupationally exposed to uranium in mines, mills, and processing facilities provide evidence, although not conclusive, for an association between exposure to uranium in air and lung cancer (ATSDR 1999; Hornung et al. 1998). Because workers were exposed to toxic and cancer-causing substances in addition to uranium, such as silica dust, radon and radon daughters, tobacco smoke, phosgene gas, heavy metals and solvents, the studies are considered inconclusive. These other exposures are likely to have contributed to the lung cancers observed. For example, miners who smoked cigarettes were at a much greater risk of developing lung cancer than those who did not smoke cigarettes. People who work with asbestos are also at an increased risk for lung cancer (Ahrens et al. 1998). As in most industrial exposures, correct use of the proper protective equipment or environmental controls can significantly reduce risk.

The heavy metals arsenic and chromium, found in surface soils off site of the Fernald facility, have been shown to cause lung cancer incidence and death in workers exposed daily to these metals in air. (This is discussed in detail in Appendix B - Exposure Doses and Health-Based Guidelines [for Potential Exposure Pathways]). Because there are no measurements of arsenic and chromium in air on site or off site of the Fernald facility, ATSDR used very conservative assumptions to estimate the concentration of arsenic and chromium (VI) that would result if surface soils became re-suspended in air and were a source of human exposure to Fernald residents. Our estimated airborne concentrations were considerably lower than the levels shown to produce lung cancer in occupationally exposed workers.

Finally, there is also some evidence for a genetic predisposition in some people that make them more susceptible to respiratory cancer after exposure to a carcinogen (Ghardirian et al. 1997; El-Zien et al. 1997).

Gastrointestinal Cancer

My wife died of cancer of the stomach, intestines, and pancreas 9 years ago.
My father died of stomach cancer.
One uncle had colon cancer.
I've had cancer three times (breast and colon).
I worked at the plant from 1952 through 1984 and was involved in all the plant operations. I was diagnosed with colon cancer in 1989, and I had surgery.
I was diagnosed with esophageal cancer in 1992; I quit smoking 25 years ago.

Cancer of the esophagus, stomach, and colon can all be categorized as diseases of the gastrointestinal tract. Cancer of the esophagus is three times more likely to occur in men than women, and affects African-Americans about three times as often as whites. There are two main types of esophageal cancer, squamous cell carcinoma and adenocarcinoma. Squamous cell carcinoma accounts for about 50% of the disease and is more likely to affect African-Americans than whites and other racial and ethnic groups. Adenocarcinoma occurs more often in whites than other racial and ethnic groups. Both types of esophageal cancer are more often diagnosed in people 45 years of age or older (ACS 1999).

The risk factors for esophageal cancer include gender, race, alcohol abuse, and tobacco use (e.g., cigar, cigarettes, chewing tobacco). Other risk factors are chronic heartburn or acid reflux, a diet low in fruits and vegetables, Barrett's esophagus, and other rare diseases of the esophagus.

Stomach cancer, also known as gastric cancer, can develop in any of the five sections of the stomach. If left untreated, it can spread to other areas of the body. Most Americans diagnosed with stomach cancer are 50 years of age or older.

Stomach cancer is relatively rare in the U.S. compared with other areas of the world, possibly because dietary habits are important risk factors for this disease. A diet high in salted or smoked foods, as well as one that is high in starch and low in fiber, may contribute to the occurrence of stomach cancer. Other risk factors for this disease include tobacco and alcohol abuse, stomach polyps, vitamin B12 deficiency, *Helicobacter pylori* infection, previous stomach surgery, and rare genetic conditions. There is also evidence that people with blood type A may be at slightly higher risk for the disease (ACS 1999).

Colon cancer, referred to medically as colorectal cancer, can begin in any of the four areas of the colon or rectum and spread to other areas of the body if left untreated. As with stomach cancer, colorectal cancer is mostly diagnosed in people 50 years of age or older. Risk factors for this disease include colon polyps, chronic inflammatory bowel disease, a diet high in animal fat, a low level of physical activity, a family history of colon cancer, and certain genetic conditions (ACS 1999).

There is limited evidence from animal and human (occupational) studies for an association between exposure to the polycyclic aromatic hydrocarbon (PAH) benzo(a)pyrene and cancer of the gastrointestinal tract (Xu et al. 1996; Thyssen et al. 1981). One occupational study reported that sufficient exposure to benzo(a)pyrene in air resulted in an increased occurrence of stomach cancer (Xu et al. 1996). Benzo(a)pyrene was found in a few surface soil samples off site of the Fernald facility. However, ATSDR's estimated exposure dose to residents who may have accidentally ingested contaminated soil while playing near the site (i.e., children) was many times lower than the levels shown to cause stomach cancer in this study. One study in hamsters reported an excess number of tumors of the esophagus and forestomach following chronic exposure to benzo(a)pyrene at considerably higher concentrations than predicted in air off site of the Fernald facility. Other studies examining the association between exposure to environmental contaminants and colorectal cancer have been suggestive, but the studies could not take into account individual risk factors for the disease (Gulis et al. 1998; Riberio et al. 1996).

ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for esophageal, stomach, or colorectal cancer in the surrounding community.

Breast Cancer

I've had breast cancer and lung cancer and currently have brain cancer.

I've had cancer three times (breast and colon).

I live half a mile from the site. I had a brain tumor, breast cancer, kidney stones, gallbladder problems, stomach problems, and a herniated bowel. My doctor says the brain tumor is probably due to an increase in radiation dose from living near Fernald. I had a hysterectomy, and the doctors said I had many, many fibroid tumors rather than a single large tumor, which is more typical.

My church congregation (in Crosby) has a large number of cancer illnesses; many women have had lung and breast cancer.

My sister was diagnosed with breast cancer.

Although most breast cancers are diagnosed in women, breast cancer does occur very rarely in men. However, the following discussion pertains only to female breast cancer, as there is limited information on risk factors for male breast cancer.

Breast cancer is the second most common cancer in women, the second leading cause of cancer death in women, and the leading cause of death in women between 40 and 55 years of age. The number of newly diagnosed breast cancer cases rose by 4% per year during the 1980s, but has since leveled off. Although a variety of factors have been associated with an increased risk for breast cancer, it is important to note that some people who get breast cancer do not have any of these risk factors (ACS 1999).

Gender can be considered a risk factor, since women are 100 times more likely to develop breast cancer than men. The risk of developing breast cancer increases with age. Whites tend to develop breast cancer at a higher rate than African-Americans, but African-American women are more likely to die from the disease. Asian and Hispanic women have lower rates of this disease than do African-American women. Other risk factors are a family history of breast cancer, therapeutic irradiation of the chest area, and alcohol abuse. Factors associated with a slightly increased risk are early age of first menstruation or late age of menopause, use of oral contraceptives, late age (more than 30 years of age) of first childbirth or not having children, and estrogen replacement therapy. Genetics is thought to play a role in an estimated 5% to 10% of breast cancer cases (ACS 1999). Numerous inconclusive studies have evaluated the association between breast cancer and various other risk factors, such as having a child but not breast-feeding, consuming a diet high in animal fat, obesity, and a low level of physical activity.

There is much controversy in the scientific community as to whether there is an association between exposure to certain environmental contaminants, called endocrine disruptors, and breast cancer. These chemicals are so named because of their ability to behave like hormones and other substances that occur naturally in the body and in some foods. Once taken into the body, these chemicals alter the function (e.g., by increasing or decreasing the response) of the endocrine system, and may cause adverse effects on an organism or its offspring (NIOSH 1998).

Within the category of endocrine disruptors, much attention has been given to two widely spread contaminants, polychlorinated biphenyls (PCBs), and the banned insecticide DDT and its metabolite, DDE. Some scientists believe that these chemicals contribute to the development of breast cancer in humans, because they have been found to mimic the activity of the hormone estrogen in laboratory experiments (Feigelson et al. 1996; Rudel 1997). In these experiments, the contaminants bind to the estrogen receptors of breast cells grown *in vitro* (in a test tube), causing the cells to divide and grow continuously, a common feature of cancerous cells. Other scientists point to evidence of endocrine disruption in certain wildlife populations exposed to these chemicals in the environment (Solo 1998). Still other scientists, not convinced that these chemicals contribute significantly to breast cancer development in humans, point out that human health studies have failed to show a definite association between occupational or environmental exposure to endocrine disruptors and an increase in the risk of developing breast cancer (Davidson 1998; Datson et al. 1997; Safe 1997). However, almost all scientists agree that more studies are needed on how diet (Schieldkraut et al. 1999; Verma et al. 1998), genetics (Moysich et al. 1998), and environmental exposures may together affect an individual's risk for this disease.

EPA's Office of Prevention, Pesticides and Toxic Substances chairs an Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) that focuses on chemical substances in drinking water and food that may be endocrine disruptors. Additional information about chemicals that may be classified as endocrine disruptors, and methods for screening and testing for these substances in water and food can be obtained by accessing EPA's home page (<http://www.epa.gov>) (EPA 2000).

ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for breast cancer in the Fernald community. Additional information about endocrine disruptors, and the possible effects on breast cancer development, can be obtained by contacting the National Institute for Occupational Safety and Health or EPA.

Central Nervous System (CNS) Cancer

Eleven years ago my child (then aged 7) was diagnosed with a brain tumor. Hospital staff asked if the child was exposed to radiation.

I've had breast cancer and lung cancer and currently have brain cancer.

My wife has a brain tumor (frontal lobe meningioma), my next door neighbor had two brain tumors within the same year, and a neighbor down the street died of a brain tumor.

Three of my brothers died of cancer. One died of brain cancer.

There is a high incidence of brain tumors in the area. Is it safe to eat produce grown in the area? I live half a mile from the site. I had a brain tumor, breast cancer, kidney stones, gallbladder problems, stomach problems, and a herniated bowel. My doctor says the brain tumor is probably due to an increase in radiation dose from living near Fernald. I had a hysterectomy, and the doctors said I had many, many fibroid tumors rather than a single large tumor, which is more typical.

My son was diagnosed with neuroblastoma. There are 12 houses on our street and 6 cancers there. Is neuroblastoma related to site activities?

The central nervous system (CNS) is composed of the brain and spinal cord. There are many different cell types in the CNS, and cancer can occur in any of them. Metastatic brain cancer, which originates in other organs and then spreads to the brain, is the most common type of brain cancer in adults. Primary brain cancer originates in the brain itself and is relatively rare in adults. Brain cancer is the second most common type of cancer in children. Neuroblastoma, the third most common type of brain cancer diagnosed in children, is very rare in adults (ACS 1999).

The risk factors for CNS cancers differ for adults and children. In adults, therapeutic radiation to the head area (as treatment for other cancers) is a risk factor for brain cancer. Some studies suggest an association between ingesting the sugar substitute aspartame and an increased risk for brain cancer in adults. Other studies of the relationship between exposure to low-level electromagnetic fields (EMF) and the risk for brain cancer are inconclusive and controversial. For children, exposure to ionizing radiation (as a treatment for other cancers) is a known risk factor for brain cancer. There is some evidence of genetic susceptibility to brain cancer in families with very specific genetic disorders (ACS 1999; Preston-Martin 1996).

Many studies conducted in the United States and abroad have examined the association between occupational or environmental exposure to ionizing radiation and the risk for brain cancer. Most of the occupational studies have been done in groups of workers (called cohorts) employed in the nuclear energy or nuclear weapons industry. The majority of these studies have investigated cancer mortality rates among workers who died from a variety of causes, including brain cancer, rather than cancer incidence among exposed workers (Alexander 1991). This is because it is often easier to obtain information on causes of death than on disease status during the work history of an employee, especially if a worker died before the beginning of the study. In addition, many of these studies do not include a measure of individual exposure to radiation, because the studies were conducted years after the exposure occurred. Overall, there is some disagreement within the scientific community about the role of occupational exposure to ionizing radiation and cancer mortality in the studies' cohorts.

Studies of the association between environmental exposure to ionizing radiation and occurrence of brain cancer in communities surrounding nuclear energy or nuclear weapons facilities suffer from methodological issues related to exposure and case ascertainment (Forman 1987; Wakefield and Berry 1996). For example, there is rarely information about individuals' exposure levels. In most of these studies, exposure is estimated using surrogates such as residence relative to the nuclear facility. Also, individuals in the study population who have been diagnosed with brain cancer may move or die before the study begins and may not be included in the study. This creates a selection bias if more highly exposed individuals tend to move away from a community more often than less exposed individuals. Overall, the results from these studies do not provide conclusive evidence for an increased risk for brain cancer and living in the vicinity of a nuclear facility. However, because many of these studies have been conducted or funded by agencies and corporations that are responsible for the contamination, members of the public are often skeptical of their results.

ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for CNS cancers in the Fernald community. There are no chemicals or radioactive materials known to have been released from the site at levels that have been shown to cause CNS cancers in humans or laboratory animals.

Hematopoietic Cancer

There are three cases of leukemia and one case of cervical cancer on my street.
My father-in-law died of leukemia.

Leukemia is a form of cancer that begins in the blood-forming cells of the body. Although these cells are located in the bone marrow, the disease can spread to other parts of the body. Leukemia is divided into four main types, each of which is further divided into subtypes. The four main types of leukemia are acute myelogenous leukemia (AML), acute lymphocytic leukemia (ALL), chronic myelogenous leukemia (CML), and chronic lymphocytic leukemia (CLL) (ACS 1999). Acute leukemia occurs when the affected cells do not mature properly and continuously divide and propagate. In chronic leukemia, the cells mature but are abnormal. The terms *myelogenous* and *lymphocytic* refer to the types of cells that are involved.

Acute leukemia is the most common type of cancer in children. Chronic leukemia is rare in children and occurs mostly in adults. Diet, smoking, alcohol abuse, exposure to benzene, and certain infections are risk factors for leukemia in adults. Both adults and children who have been exposed to high doses of

ionizing radiation (e.g., atomic-bomb survivors, Chernobyl survivors) are at increased risk of developing leukemia. There is an increased risk for leukemia in children with Down's syndrome or certain rare genetic conditions. Other potential risk factors for childhood leukemia are maternal alcohol abuse, maternal cigarette smoking, paternal occupational exposure to certain chemicals and solvents, and exposure to contaminated groundwater (ACS 1999).

Studies have been conducted to evaluate the association between leukemia and exposure to low-frequency electromagnetic radiation (EMF); the results of these studies are inconclusive and somewhat controversial. Several studies have been conducted to examine the significance of geographical clustering of leukemia cases around some urban areas or contaminated sites (Alexander 1998, Preitidou et al. 1997). The findings of these studies are suggestive at best because they generally lack specific information about individual exposures and potential risk factors.

ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for leukemia in the Fernald community.

Lymphoplastic Cancer

An 18-year-old boy next door died of lymphoma in 1986.

Both Hodgkin's lymphoma (HL) and Non-Hodgkin's lymphoma (NHL) are cancers that develop in lymphatic tissue, which is part of the immune and blood-forming cell systems. Because lymphatic tissue is present in many areas of the body, cancers can develop virtually anywhere and spread throughout the body. NHL consists of many different subtypes that are based on clinical classifications. The majority (95%) of NHL cases occur in adults. Men are more affected by these cancers than women, and whites are more affected than African-Americans or Asians. Among children in the United States, boys are three times more likely to develop cancer than girls, and white children are twice as likely to develop cancer as African-Americans (ACS 1999).

Risk factors for adult Hodgkin's lymphoma include having Acquired Immune Deficiency Syndrome (AIDS) and being an organ transplant recipient, both of which involve decreased function of the immune system. Risk factors for NHL include a reduced level of immunity and exposure to benzene, insecticides, or herbicides. Exposure to high levels of ionizing radiation, such as experienced by atomic-bomb survivors or Chernobyl survivors, has been shown to produce slight increases in the risk of developing NHL. Radiation therapy to treat other cancers has also been shown to produce slight increases in the risk for NHL. In children, the risk factors also include a congenital immune deficiency that makes them more susceptible to infectious disease (ACS 1999).

ATSDR has no evidence that past or current exposure to contaminants from the Fernald site has contributed to an increased risk for either Hodgkin's or non-Hodgkin's lymphoma in the surrounding community.

Prostate Cancer

My father had prostate cancer. He was an organ donor, but upon his death, the hospital would not accept his organs due to their contamination.

My father had prostate cancer.

Prostate cancer develops from the prostate gland and can spread to different parts of the body. It is the second most common cancer in men after skin cancer, and the second leading cause of death in men after lung cancer. The primary risk factor for prostate cancer is age; it is most often diagnosed in men over 50 years of age. African-American men are twice as likely as white men to develop prostate cancer. Other potential risk factors for prostate cancer are a diet high in animal fat, being overweight, and a low level of physical activity. There is some evidence that prostate cancer may run in families. Studies of the relationship between prostate cancer and vasectomy have been equivocal (ACS 1999).

There is no evidence that prostate cancer is linked to exposure to environmental contaminants. Preliminary findings of the Fernald Medical Monitoring Program (FMMP) suggest that the number of new cases of prostate cancer among FMMP participants is greater than expected compared to persons in the Surveillance Epidemiology End Result (SEER) program for Ohio. However, the observed increase may not be an actual increase in the number of new cases, but may have resulted from the use of a new diagnostic test that improved the identification of existing cases (Pinney 1999).

Reproductive System Cancers

I am concerned about breast and testicular cancer.

There are three cases of leukemia and one case of cervical cancer on my street.

Are cervical cancer and thyroid disorders related to site activities?

My wife and I drank well water for 3 2 years and cistern water for 7 years. She died of vaginal cancer.

I have an enlarged thyroid and have had cervical cancer. Are cervical cancer and thyroid disorders related to the site?

I've lived near the plant since I was 9. My 19-year-old daughter was diagnosed 1 year ago with an ovarian tumor; one ovary was removed. I have had uterine cancer and tumors on my back.

Cancers of the reproductive system are associated with a variety of risk factors. Some occupational exposures have been associated with an increased risk for testicular cancer. Other risk factors are undescended testicles and injury to the testicles. Infection with Human Papilloma Virus (HPV) is a risk factor for both cervical and vaginal cancer. Human immunodeficiency virus (HIV) infection, smoking, low socioeconomic status, and a diet low in fruits and vegetables are all risk factors for cervical cancer. Some women with vaginal cancer developed the disease because their mothers took the drug diethylstilbestrol (DES) during pregnancy. Women over 50 years of age are at greater risk for ovarian and vaginal cancer. Family history of ovarian cancer or breast cancer, early age at first menstruation or late age of menopause, late age (over 30 years) of first childbirth, or not having children are also associated with an increased risk for ovarian cancer. Finally, both infertility and the use of a specific type of fertility drug have both been associated with a slight increased risk for ovarian cancer (ACS 1999).

ATSDR has no evidence from human health studies, medical case reports, and animal laboratory studies that exposure to the types and levels of contaminants in environmental media off site of the Fernald facility are risk factors for reproductive system cancers.

Other (and Unspecified) Cancers

A 15-year-old neighbor died of cancer 8 or 9 years ago.

A neighbor woman died of cancer 6 or 7 years ago.

I feel that there is an excess of cancer in the community.

There are two other cases of cancer in people who grew up playing in the creek.

My wife died of cancer. Two of my neighbors died of cancer.

My husband died of cancer. Also, two neighbors had cancer.

One citizen offered a list of people on Buell Road with cancer and thyroid problems, past and present.

In my neighborhood, eight people have had cancer.

My mother and I have had cancer.

A boy with a large tumor in his leg had the leg amputated.

Both my parents died of cancer in the 1970s.

My husband worked at Cintas Corporation in a heating job that used formaldehyde. He had Guillain-Barré syndrome, Graves' disease, and a collapsed lung. He had his gallbladder and bladder removed and died of cancer.

My husband ate meat and produce from our farm and drank well water; he died of cancer. Is eating meat and produce raised in the area and drinking well water hazardous to the community's health?

My mother-in-law and father-in-law both died of cancer.

There are many cancers on Buell Road; there are also many animal sicknesses and deaths there.

My church congregation (in Crosby) has a large number of cancer illnesses; many women have had lung and breast cancer.

I live 2 or 3 miles from the site. I am concerned that many others in the nearby area have cancer.

My son was diagnosed with neuroblastoma. There are 12 houses on our street and 6 cancers there. Is neuroblastoma related to site activities?

I have tumors all over my body for no reason, including benign tumors on my neck and breast. I've had female disorders and a partial hysterectomy; soon after that tumors appeared again. My father-in-law died of leukemia; a great aunt died of cancer. I have a great deal of stress from living in this area. Are my tumors due to the site's activities?

There have been at least six cancers in my old neighborhood (Thrush Road), including lung cancer in a non-smoker. My grandmother had skin cancer. What health effects could uranium in the groundwater have caused?

I have a list of deaths from cancers and other causes in the community.

My daughter died of small-cell cancer at age 35; her physician said he had never seen this type of cancer in anyone under age 65. She was a smoker but the tumor was in the middle of her chest, and there was no lung involvement. Her son had hair loss his junior year in high school. Her daughter was born with deformities of the left fingers and toes. Could these health problems be related to the site?

As stated above, many different risk factors are associated with different types of cancers, but many people who get the disease do not have a readily identifiable risk factor. Although many studies examined how environmental exposures may increase a person's risk of developing cancer, more studies need to be conducted on the interaction between a person's lifestyle, genetics, and environmental exposures. The studies that have been done examined populations, as opposed to individual risk. If you are concerned about your own risk or that of someone in your family, you should consult your personal physician. The local chapter of the American Cancer Society also has information on risk factors and prevention.

Non-Cancer Effects

Dermatological Effects

Around December 18 - 25, 1984, Fernald had its last big air release. At the time, I had an outdoor wedding. After that my husband developed skin cancer, and my son developed a bad skin rash and a spot on his lungs. My husband used to eat vegetables grown in our garden and fish from the Great Miami River; my son is a non-smoker.

I am concerned about my children. They have warts, cysts, and moles. One has chicken pox scars and the 10-year-old has sores in his mouth.

Back in the 1950s an occurrence of a mist covered [my husband and me] one evening. After that, I developed skin problems - little spots on my face.

Occupational exposure to high levels of some organic chemicals (e.g., PCBs and dioxins) has been associated with various dermatologic effects, such as chloracne (Klaassen et al. 1996). If you are concerned about your exposure and risk for dermatological problems, contact your health care provider. At present, ATSDR has no evidence that exposure to chemicals and radioactive materials from the Fernald site would result in any dermatologic effects in persons in the surrounding community.

Endocrine

I have a cousin with thyroid problems and memory loss. I have something in my lung and difficulty breathing; also, I have a deteriorating disk in my back.

One citizen offered a list of people on Buell Road with cancer or thyroid problems, past and present.

I have diabetes, and there is no history of diabetes in my family.

Are cervical cancer and thyroid disorders related to site activities?

I have thyroid and gallbladder problems. Are these related to the site? My son lived all his life near the site, and now I have grandkids living there. The kids have kidney problems and sugar in their blood.

I have an enlarged thyroid and have had cervical cancer. I know that studies have shown that female thyroid disorders can be related to the site's activities. Are cervical cancer and thyroid disorders related to the site?

I have Graves' disease (thyroid disorders) and the highest metabolism ever recorded. I've had three radioiodine treatments, and I've built up immunity to the treatments. I have two knots on my leg, which my physician cannot explain. My daughter is very sick. The site causes us lots of anxiety. What are the illnesses in the community, and how severe are they?

Of the many risk factors for thyroid disorders and diabetes, most are associated with genetics and lifestyle (e.g., smoking, diet, exercise). There is some evidence from human health studies that thyroid disorders and diabetes may result from exposure to certain types of environmental contaminants. For example, some studies indicate that thyroid disorders can occur in populations exposed to high levels of radioactive iodine (Edwards 1995). Radioactive iodine has not been found in environmental samples collected on site or off site of the Fernald facility. Diabetes has been shown to result from exposure to high levels of inorganic arsenic in drinking water (Shih-Meng et al. 1999). Arsenic was found in surface soil off site of the Fernald facility; however, ATSDR's estimated doses to Fernald residents from exposure (ingestion) of arsenic-contaminated soil are considerably lower than the exposure doses shown to be related to the development of diabetes in this study.

Developmental and Reproductive Effects

There are a large number of birth defects among my daughter's classmates.

I had a grandson born deformed 3 years ago, who died at 4 months.

My nephew has a birth defect of double toes. My son was born with a cleft palate and missing femurs.

My older sister's grandson has a gene defect - Hurler's syndrome.

[My daughter] has a deformity and can't see from her right eye. Is there a problem with birth defects or some other type of genetic problem?

[My son] has another child with multiple bones in his toe. My daughter, born in 1957, has a child with Pierre Robin syndrome. Will these birth defects carry to future generations?

My wife was diagnosed with Guillain-Barré syndrome in 1973.

My husband worked at Citras Corporation in a heating job that used formaldehyde. He had Guillain-Barré syndrome, Graves' disease, and a collapsed lung. He had his gallbladder and bladder removed and died of cancer.

Are there any genetic mutations in children from things at FEMP?

My daughter was born with trisomy 18. She was diagnosed at birth and died 5 weeks later. A home nurse who cared for her before she died reported that eight babies with genetic defects lived within a 10-mile radius of the site. Could my daughter's defect be related to environmental exposure from the site? Anecdotal information indicated that an 18-month-old has Hoopers syndrome, and friends have had multiple miscarriages and infertility.

I am infertile. I have had various tests and have low or no sperm.

One of my sons was born in 1953. He has low sperm counts and has adopted all his children.

My daughter had two miscarriages within 3 years.

There is evidence from human health and laboratory animal studies that maternal exposure to certain environmental chemicals, such as solvents, PCBs, dioxins, and heavy metals is related to an increased occurrence of developmental and reproductive effects in offspring (ATSDR 1997; Fredriksson et al. 1993; MDPH 1997; Weir and Fisher 1972). Some of the effects observed in these studies are abnormalities or delays in cognitive and physiological development, neurobehavioral effects, and childhood leukemia. Boron is the only one of these chemicals found off site of the Fernald facility. However, the maximum concentrations of boron found in these surface soils, and predicted in air, are considerably lower than levels shown to cause adverse effects in the laboratory animals studied (Weir and Fisher 1972).

Many of the conditions listed above are not believed to be linked to exposure to environmental contaminants. Pierre Robin syndrome has no known risk factors and is most likely solely genetic in nature (Widesmiles 1999). Guillain-Barré syndrome is a neurological condition that may be an autoimmune disease. (An autoimmune disease is a condition in which the body forms an immune response against its own tissues.) There are no known risk factors or causes for this syndrome (GBS 1999). Cleft palate - the fourth most common birth defect among children born in the United States - is believed to be caused by a combination of genetic, nutritional, and environmental factors (IIBD 1999). Hurler's syndrome is a metabolic disease that is genetic in nature (NORD 1999).

Although exposure to chemical and radioactive contaminants can theoretically cause cellular mutations, ATSDR has no evidence that exposure to any of the contaminants from the Fernald site would result in an increase in birth defects in the surrounding community. Residents who are concerned about their own or their children's risk of passing on genetic disorders to offspring should consult their physician or a geneticist who can assess an individual's risk for these disorders.

Musculoskeletal Effects

I have severe arthritis that severely limits my mobility.

I have a deteriorating disk in my back.

Could my rheumatoid arthritis and muscular problems be related to the fact that my mother worked at Fernald as a clerk during her pregnancy?

Arthritis is a general term for many different diseases affecting the joints and connective tissues in the body. Both disk deterioration and rheumatoid arthritis are autoimmune diseases. Currently, none of these conditions are believed to be caused by exposure to environmental contaminants (AF 1999).

Respiratory Effects

I have kidney stones, gallbladder problems, and upper respiratory problems.

Several of the contaminants (e.g., uranium, chromium, and boron) present in surface soils off site of the Fernald facility are capable of causing respiratory toxicity when exposure occurs at high enough levels. But with the exception of uranium, there are no on-site or off-site air measurements of these chemicals; therefore, ATSDR used conservative methods in estimating the maximum concentrations that may be present in ambient air off site of the Fernald facility. We also made the conservative assumption that children and farmers were exposed to these predicted maximum concentrations during most of each day, for several consecutive years. Our predicted maximum airborne concentrations, and human exposure doses for all of these contaminants (including uranium), are not likely to cause respiratory effects in Fernald residents.

Non-Specific Effects

Our son, born in 1955, has a child with a low white blood count and another child with multiple bones in his toe.

My family suffers from boils two to three at a time over the last 2 - 3 years. One son has blood in his urine. I have elevated liver enzymes and blood in my stool.

I have ringing in my ears, balance and dizziness problems, concentration difficulties, a terrible memory, and prostate problems.

My 10-year-old limps for no apparent reason and is a slow learner.

My entire family has chronic fatigue. My sister had seizures between ages 8 and 10; she has a weak immune system and suffers hair loss.

I am concerned about what may be in the soil and the frequency of different types of illnesses.

I raised six children on a farm near the Fernald plant, and I am worried about adverse health outcomes that might result from living close to Fernald. One son committed suicide, and I think his depression was caused by some chemical exposure. This son also had asthma. One daughter has asthma and sinus problems. Another daughter has food allergies and boils. This daughter and her husband have been experiencing memory losses.

I have a lung nodule and suspect asbestos exposure. I also have an ulcer, colon problems, and dizzy spells. I realize that these may not be related to Fernald, but I would very much like to be put on Butler County water.

I live half a mile from the site. I had a brain tumor, breast cancer, kidney stones, gallbladder problems, stomach problems, and a herniated bowel. My doctor says the brain tumor is probably due to an increase in radiation dose from living near Fernald. I had a hysterectomy, and the doctors said I had many, many fibroid tumors rather than a single large tumor, which is more typical.

This list includes many different health concerns, each of which is associated with a variety of known and unknown risk factors. Based on the information reviewed for this public health assessment, ATSDR has no evidence that exposure to chemical and radioactive contaminants from the Fernald site is associated with an increased risk for any of these health conditions, except kidney effects, in the population surrounding the site. (A detailed discussion of the evidence for kidney effects from exposure to uranium from the Fernald site is provided in the [APublic Health Implications](#) section of this report.) However, a physician or health care provider can better assess an individual's risk for these conditions.

Environmental Exposures

ATSDR grouped the list of concerns about environmental exposures by exposure medium, i.e., air, soil, surface water, groundwater, and biota. ATSDR did not provide specific responses to these concerns, because most of them are addressed in the [AExposure Pathways Analyses](#) section of this report.

Air

When Fernald was operating there were odors from the site.

I have tested my house for radon, but I think the radon testing kits are unreliable. Could it still be contaminated because of previous absorption?

A tornado came through here in the late 1960s and went toward Indiana. Could it have carried contaminated soil, air, or water?

Soil

A tornado came through here in the late 1960s and went toward Indiana. Could it have carried contaminated soil, air or water?

I am concerned about what may be in the soil and the frequency of different types of illnesses.

Surface Water

A tornado came through here in the late 1960s and went toward Indiana. Could it have carried contaminated soil, air, or water?

People used to swim in the gravel pits and in Paddy's Run Creek. Are the gravel/sand pits contaminated?

Are the food and water in the area contaminated?

The Miami River was very clear in 1967; now it appears very cloudy and dirty.

Local gravel pits fill with rain water, and there are no outlets for the runoff.

Groundwater

Fernald representatives sample my well annually, tests indicate some contamination.

The groundwater is not monitored on the west side of Paddy's Run Creek.

My husband and I used to drink well water. We still use cistern water for non-potable uses. Is the cistern water safe?

I drank well water at home and at the plant. What about exposures to employees drinking well water on the job (at Fernald)?

[My spouse and I] have used well water and grown our own food and eaten wild game from this area.

I drank water from a well from 1979 through 1991. What are the effects of using contaminated groundwater for potable and non-potable purposes?

My husband ate meat and produce from our farm and drank well water; he died of cancer. Is eating meat and produce raised in the area and drinking well water hazardous to the community's health?

I drank well water for 9 years and then found out it is contaminated. Is it safe to wash with the well water?

Biota

When Fernald was operating, there were odors from the site and a silver sheen on plants around the area.

I've heard that birds in the area were tested at Fernald and that they were extremely contaminated. If the birds were dying, we must have been exposed to something very dangerous.

[My spouse and I] have used well water and grown our own food and eaten wild game from this area.

Are the food and water in the area contaminated?

Have fish in the river been checked for uranium?

Why is produce from this area still being sold? Is the milk safe?

[My nephew] ate vegetables grown in the yard and drank water from a cistern. He is now in his late 40s and has no known health problems other than a growth on his gum.

I have heard rumors of blind fish in the river. Could this be a result of contaminants in the river?

Is there a health problem associated with consumption of wild game? I have killed deer and small game with tumors on them and that appeared to be no good to eat.

My husband ate meat and produce from our farm and drank well water; he died of cancer. Is

eating meat and produce raised in the area and drinking well water hazardous to the community's health?

There is a high incidence of brain tumors in the area. Is it safe to eat produce grown in the area?

ATSDR determined that there are no pathways of exposure that pose a human health hazard under *current* conditions at the Fernald site. ATSDR found that only one current exposure pathway - ingestion of water from privately owned wells near the site - poses an *indeterminate* health hazard. ATSDR used the term *indeterminate* because we do not have enough information about potential exposure (i.e., location and usage of wells and cisterns, and potential contaminant concentrations) to make a definitive assessment of the level of public health hazard. Based on the available data we reviewed, we do not expect that residents who are currently using well water will experience adverse health effects. If you are concerned about your well water, please contact the Ohio Department of Health to obtain information about how to get your well tested.

In addition, ATSDR has conducted three Health Consultations that specifically address public health hazards from environmental exposure: (1) consumption of milk from farms near the Fernald site, (2) consumption of produce (fruits and vegetables) grown near the Fernald site, and (3) use of groundwater for non-potable uses (ATSDR 1995a, 1996a, 1996b). We determined that none of these pathways of exposure poses a human health hazard, and that wild game caught in the area should also be safe for consumption. Additional information on the health of wildlife in the area can be obtained from the local Fish and Wildlife office.

Specific Populations

Children

Is it safe for me and my children to stay here (continue living in this area)?

I am concerned about exposures to children.

A Girl Scout camp north of the Fernald Environmental Management Project (FEMP) has been closed. Is there any danger in trying to use it now?

I am concerned about radiation leakage from the site and its effect on the health of my wife and children.

Elderly

Both sets of my grandparents lived within one-half mile of the site.

ATSDR has no information indicating that children and elderly persons are at increased risk of adverse health effects from exposure to chemicals and radioactive materials from the Fernald site. Therefore, it is not necessary for anyone to move away from the area. In addition, ATSDR completed a Health Consultation addressing the public health hazard from exposure to radon emissions from the K-65 silos at the Fernald site (ATSDR 1995b). We determined that exposure from the K-65 silos does not pose a public health hazard. However, if you are concerned about the safety of using the former Girl Scout camp site, you should speak with the environmental officials at the site.

Workers

I am concerned about take-home exposure; I used the same locker for street clothes and work clothes. I worked in all areas of the plant.

Did the plant physician die of causes related to radiation exposure?

The focus of this public health assessment is chemicals and radioactive materials from the Fernald site that have migrated off site and are a source of exposure to persons in the surrounding community. Additional information about Fernald workers may be obtained by contacting the representatives from the Department of Energy or the CDC's National Institute of Occupational Safety and Health (NIOSH) listed in the AFor Additional Information@ section of this report.

Procedural Concerns

The focus of this public health assessment is chemicals and radioactive materials from the Fernald site that have migrated off site and are a source of exposure to persons in the surrounding community. Therefore, we did not specifically address many of the following procedural concerns. Some concerns are addressed in the main body of this report. Additional information about other issues may be obtained by contacting the representatives from the Department of Energy, CDC's National Center for Environmental Health, U.S. Environmental Protection Agency - Region VII, Ohio Environmental Protection Agency, Ohio Department of Health, and the Fernald Medical Monitoring Program listed in the AFor Additional Information@ section of this report.

Remediation

I am concerned about releases to the environment from the remediation process.

What are the health effects of exposure from the 1950s?

Will FEMP be receiving wastes from other plants?

Lack of Trust

In the late 1960s and early 1970s in the mornings a white film covered Aeverything.@ This was during the time when Fernald representatives told residents they would stop burning materials on site; instead they began burning things late at night and early on Sunday mornings.

I am upset because I was told it was too late to get into the Fernald Medical Monitoring Program even though I sent in my paperwork at the start of the program.

The Fernald Medical Monitoring Program is not as good as it is made out to be. We need better, continued health monitoring.

Communications from the Fernald fund management (trustees) are not good. Forms were not sent out to all residents; forms were left at stores, banks, and other places to be picked up.

I heard that money distributed from the fund was based on how well a person filled out the forms and not on actual conditions. This should be checked.

I have not received answers from the Fernald Medical Monitoring Program to any of my questions.

I believe that dose reconstruction will not account for the buildup of white substance that occurred in the night.

I feel the public was lied to through the years and kept in the dark. The Purina sign is an example of this.

When the site was first built the Aolder ones@ said it would cause a lot of illnesses; see what has happened.

A major concern or desire I have is to finally get accurate and true information.

The University of Cincinnati collected blood samples from people who live close to the site to compare the results with others further away from the site, but no one has received the results of the blood analyses or the study results.

Citizen is concerned about the adequacy with which medical monitoring results are disseminated.

Emergency Response

Might natural disasters such as tornadoes move drums off site and cause public exposure?

Monitoring or Sampling

Where can we have our water tested for mercury, lead and uranium: Should it be tested for contaminants other than these?

FEMP sampled the riverbanks near my house, and I haven't seen any results.

Someone sampled my well but I have not been informed of the results.

How do I get on the mailing lists and get my water tested?

Citizen would like to obtain results of water samples collected from the trailer park system.

General

Citizen provided names of doctors in the area who may have information useful to ATSDR and CDC.

Citizen is willing to release her medical records to ATSDR.

Citizens are Ameeting-ed to death.@

Well water will not sour in a cistern, but city water will.

The government took the best location over the aquifer for itself, though it could have taken an out-of-the-way place.

Property values are going down.

Were former residents included in the CDC Dosimetry Reconstruction study?

Do occupational medical records reveal whether radiation testing was done?

Can citizens get the radiation records of deceased family members?

Citizens' Recommendations

Document the groundwater flow and direction.

Educate the community about health issues.

ATSDR should attend FRESH meetings

Advertise in the *Cincinnati Enquirer*, the *Venice Cornerstone*, and the *Harrison Press*.

ATSDR should conduct public forum-type meetings and be available for question and answer sessions.

Try to locate our soil samples and use them in the public health assessment if they are useful.

A couple wishes to be on the ATSDR mailing list.

Investigate the large number of cancers in the members of our church.

I would like ATSDR to track birth defects and provide information on these to the community.

ATSDR should enlist volunteers from the community to go door to door to collect information. I will volunteer to do so.

Address the incidence of cancer.

Discuss concentration in soils.

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Concerns Compiled by the Fernald Health Effects Subcommittee

The following are concerns that were compiled by the Fernald Health Effects Subcommittee (FHES). FHES has already addressed many of these individual concerns. If you are interested in finding out more information about the activities of the FHES please contact the FHES representative identified in the AFor Additional Information[®] section of this report.

- I worked at Fernald for 32 years. It's imperative that the working conditions which existed when operating be exposed. The exposure to radon and thorium gases were unnoticed until 1986 when Westinghouse came on board. The health conditions in plants 2 and 3 should be exposed.
- I have no confidence in CDC or any of the associations it may have with Fernald or its contractors since CDC's involvement with political issues outside of its jurisdiction, specifically gun control. I've watched the waste at Fernald of which CDC is merely a part. Don't waste my time or tax money!
- Please keep on it to better our area. I am sorry I haven't been able to attend any meetings due to health problems, but I do try every chance I get to talk to Lisa Crawford - not often though.
- What good is all of this? It's like a pacifier put in your mouth. Originally not knowing what this Fernald crap was about, I settled for an amazingly low amount and now the health problems are escalating. So what good are they?
- I believe supervision and management are poor although maybe made more difficult because of union rules. Money is wasted (new materials thrown away, sloppy cleanup - spills, etc.). I question the safety of the whole cleanup. I question water being pumped into the big Miami. I really resent the fact that we are 1/4 mile from Fernald and cannot get on the public water line - why can't we pay a prorated share and have the line extended? A private water line for Delta Steel and another firm was laid from Crosby/Wiley Road past our property and over to Delta Steel. All that cost and work for nothing because the public water line is over there now. This money could have been spent on other things - I know it was a deal with the government - lawsuit with Delta - that is just another stupid, wasteful use of tax money.
- My husband died of cancer last November. Cancer destroyed a wonderful person, and he suffered the torture of the damned his last few months on this earth. Also, I had to be operated on for skin cancer on my face and upper arm. What is the point of coming to any more meetings? The government will never admit that they are to blame, and there isn't enough compensation in the entire world to make up for the loss of my dear husband.
- It would be really useful to have a single list (and thumbnail description) of the numerous health studies of the Fernald workers and residents.
- It seems very clear that the whole Fernald Plant's activities should be closed down as soon as possible after removal of all contaminated materials off-site!!! The concrete findings of your current research should be published in the Cincinnati newspapers when completed!!! We also feel any and all irresponsible actions of those in charge of removing and cleaning up waste and contaminants need to be prosecuted and punished to fullest extent of the law to offset the ridiculously high costs of having this work done at the Fernald plant!!! Thank you!
- Given the difficulty in conducting a valuable, meaningful, epidemiological study with the small number of people in the affected area, wouldn't it be better to spend the time and resources on establishing a medical monitoring program for area residents?
- My real concern is that everyone is aware that a problem or problems exist at Fernald. For a decade now there had been a lot of posturing over this, by the private sector and the U.S. Government, for all of which I volunteered 4 years of my life to protect. Now the same people are risking my health and my family's life because no one is willing to step up and take responsibility. If there is a problem and we have been and are still at risk, then do something about it. If it becomes evident that I develop problems due to neglect at Fernald you can be certain I will take whatever action to protect myself and my family. If we are at future risk, I think we should be informed, and told to what extent the dangers are present. I am meeting with a realtor Sunday to discuss leaving this area after 12 years, and this is one of my concerns. I have had open heart surgery and have developed tendonitis in both shoulders and arthritic conditions in my neck and hands since living here. My age is 50 and it is not abnormal for someone to have these problems, but I am concerned as to whether living in the vicinity of Fernald may have caused or added to my discomfort. Bottom line, if it is broke, fix it! At least come clean and tell it and what other risks we may face. Ten years is a long time to hold your breath. I do not see any reason to continue the masquerade.
- Just how safe is the well water outside of the southern 3-mile area around Fernald? My wife and I live about 5 miles south of AF. @ just short of the water line built by the DOE.
- Is the subcommittee considering the files of information and health concerns ATSDR gathered 2 or 3 years ago from Appee who live near the Fernald site[®] in Oxford, Ohio - the Alba Craft site - which was a satellite machine shop for Fernald and less than 15 miles away as the crow flies? You should. And you should show more interest in such populations.
- He had deterioration of the brain. He began having this in 1987 or before. He was 56 years old when he started noticing this.
- I appreciate the concern for our health and also the environment. Keep up the good work.
- How much longer will this cleanup up take to complete??? Why aren't more reports of hard information put in the Cincinnati newspaper regarding the health effects of the plant's operation???? How effectively are the huge costs of doing this site cleanup being checked????
- Can the administrative costs of this entire operation be trimmed down at all??? After all we're talking about taxpayers' money!!! We need to hear more hard responses from our state senators about this whole situation. Thank you for working on these concerns!!!
- Please re-check our water supply! It has been a year or two.
- I have no interest in this project. Where was the federal government oversight when all this contamination was going on for 30 years???? I think it's just a big boondoggle now to waste taxpayers' money and send all this literature out to citizens who aren't involved and don't want it cluttering up their mail. It is another example of government waste.
- Please continue to keep us informed.
- Thank you for sending the FHES Meeting Announcement for the November 5 - 6, 1997 meeting, including the copy of the AExecutive Summary[®] report covering the sixth meeting of the Health Effects Subcommittee. Though not specifically stated in the agenda of the upcoming meeting, I hope that you will include the effects of thorium 232 in the study, since this element carries a rather high energy decay level.
- Why is no one addressing the fact that several have already said that they saw or were part of the burning of dangerous chemicals at night? This looks more like a cover-up than a real effort to find out just what the persons from this area were really exposed to. I would like to see this looked into, not just brushed aside. If you all really are what you say you are, you will find out about this because I know it to be true, after hearing it from many, many workers and neighbors.
- I am also concerned about the Potential health impacts[®] on my vegetable garden. Please supply information.
- My parents bought 60 acres in 1941. We had milk cows, pigs, and chickens and raised most of our vegetables and some fruit. In 1949 my husband and I built a house on the farm. Most of the farm is now Interstate 74. My dad died in 1971 of leukemia. In 1953 I had a baby boy born with a hole in his heart. At that time there was nothing they could do for him so in 1954 he died. We all lived there from -41 to -66. I'm 76 years old, and in October 1996 I had to have colon cancer operations. I never thought about reporting any of this to Fernald Health Effects Subcommittee until I got sick. Now things haven't been going too well for me and I was wondering if I could get any financial help.
- Please send me all the information you have available concerning Fernald.
- I'm sorry I didn't get to the meeting of May 7, 1998. I would like to have heard about bone cancers and kidneys. If you have any thoughts on those subjects, would you please send them to me? I have been laid up since June 18, 1996, when my leg went out just walking across the floor. They put a total knee in August 28, 1996. I had so much pain they took part of it out and put a new one in on April 8, 1997. I have so much pain they had a brace made special for my leg. They want me to wear it to see if it will help; if it doesn't I will go through my third operation. If you have any information on thyroid I would like that also, as my husband and I both have thyroid conditions. We have lived out here since 1952.
- The AHealth Care Providers Working Group[®] has a major task to develop educational material for local health care providers. I would like to follow their progress. Maybe attend meetings, see agendas, and get any mailings they produce. I work around the Mound Nuclear Weapons facility and we have no Health Effects Subcommittee. Local health care providers are greatly in need of education about contamination coming from these sites. Please respond with ways I could interact with this working group.
- Start conducting these meetings when AWorking[®] people may attend (weekends) and legal action will not be taken.
- We live 6 miles from the plant. My husband was diagnosed with colon cancer in September 1995 at age 51, then liver and brain cancer in December 1997. He died in February 1998. Please add his name and cancer type to your records for our area. Is colon cancer more prevalent in our geographic area? If so, what preventable measures do you suggest for the rest of us?
- My husband died in November 1994 of colon cancer. He had cancer in both lungs and a malignant rectal tumor for which he had undergone surgery to have a colostomy in the summer of 1996. He had 30 radiation treatments for the rectal cancer and more than 50 chemotherapy treatments for the lung cancer. All to no avail. He also underwent surgery on his right arm and shoulder due to a malignant tumor that weakened the bone and caused it to break - he had an entire right shoulder prosthesis and in the upper arm. He had more radiation treatments, since the tumor was too extensive and was into the muscle also and could not be removed entirely during surgery. He had some physical therapy, but never regained the use of his arm. Before he died, he had several more breaks in other parts of his right arm due to more malignant tumors and underwent more radiation treatments. My husband was practically helpless the last 2 months of his life. I had to bathe him, dress him, and undress him. I had a hospital bed for him and oxygen. Finally, I called hospice for help in the middle of October. They helped me a few times per week. My husband was in so much pain. In November the hospice had him taken to the hospital, where his pain could be controlled better. I spent most days and nights with him at the hospital until he died 6 days later.
- I want to know what is being done as far as exposure and birth defects when Fernald was releasing gases. There has got to be a way of getting data on children born in the 1970s and -80s and where their mothers lived or worked. I work for the NW district and there are too many children in the DH, MH, and SBH classes that were born in -85to-87 and whose mothers lived or worked in the area of Fernald. The article in the *Enquirer* was right. You are going to wait so long that the affected children will be dead and you'll be off the hook and we'll never know for sure whether the fact that 3 children were born on the same street, downwind, and in the 6.2-mile radius of Fernald and had birth defects is a coincidence, or there is a link. Contact the surrounding school districts and I think you would be amazed at the number of kids in special classes that may have a link to Fernald. I hope to hear from you, but I will not let this drop. I am going to follow this letter up with a phone call to Senator Dewine and Congressman Chabot. I feel very strongly that this should be looked into.
- I am a resident at the Branch Hill trailer court and I am worried about my health and my family's health. The *Cincinnati Enquirer* stated that they estimate deaths until the year 2088 from Fernald. I moved 3 years ago and I became pregnant, and my daughter who is almost 2 years old was born with a cleft lip, and she was diagnosed with having gross motor delay. Children's Hospital neurology department did an MRI on her to rule out cerebral palsy. I have two other older daughters with no birth defects. I would like a physical every few years to help detect any problems associated with Fernald.
- Thank you for the complete physicals you are now offering every 2 years. I have a nephew who lives in California and was never contacted by anyone from Fernald (never received any compensation - nothing) even though he was there almost every day - in and out of different areas delivering things for Federal Express. I feel the very least they could do is offer him something. He has just been overlooked by everyone since he transferred to California about the time this all came about.
- I want to know why I am not qualified for the medical monitoring program. My whole family is in it. I have lived in Ross for 55 years since 1943. I was told everyone was to be in it, but when I called to get papers to sign up I never received them. This has been going on for one year or more if you start when they first started, but I just tried again to get into it a year ago. There is never any explanation why I never receive anything. Also, I am the only one in the family that receives this information about meetings.
- My family has been living in the area since 1985. We missed the chance for the medical monitoring program. I recently learned that until 1987 radon releases from the K-65 silos were very high. Has the committee ever considered recommending medical monitoring for residents such as my family? I would really like it, especially for my children who have lived here most of their lives.

- I am interested in knowing if my husband's work at the Herring-Hall-Marvin Company had any connection with the Fernald company. He machined uranium slugs from rolled rods in the 1940s to the early 1950s. He mentioned to me at different times that whenever the metal chips fell to the floor they would smoke or catch on fire, so he knew it was something that was secret for the government.

Community Study of Health Outcomes in the Fernald Area

(Conducted by Fernald Residents for Environmental Safety and Health, Inc.)

Sometimes, a group of residents in a community affected by a hazardous waste site take the initiative to conduct a health study on their own, in order to assess the amount of and different types of illnesses in their community. These are typically not analytical epidemiologic studies and, therefore, cannot link specific types of environmental exposure to adverse health outcomes. Because the information about cases is volunteered by residents, it is not possible to determine if all cases have been identified, nor is it possible to verify the exact diagnosis. Nonetheless, when this information is viewed together with other scientific studies of residents in the area, it helps illuminate the overall health status of the community and focus community-based health activities.

A community health study is currently being carried out by members of Fernald Residents for Environmental Safety and Health, Inc. (FRESH). FRESH is gathering information directly from the residents of the Fernald area about health outcomes that have occurred in the vicinity of the Fernald site. The information in the study is not obtained from a cancer registry, hospital database, or other official source. The purpose of this study is to map disease occurrence in order to discern disease patterns in the area. Members of FRESH provided the information to ATSDR. The residents' comments up to January 1999 are listed below. The total number of cases identified is 426. Some people reported having more than one illness.

The group of adults between the ages of 30 through 40 have low sperm counts, multiple miscarriage, brain tumors, pituitary tumors, thyroid diseases, and liver cancer.

There is a large cluster located in Ross, Ohio, northeast/southeast of the Fernald site. This is the direction of the prevailing winds. As you look toward Morgan Township (southwest) this area is rural, but one road has been greatly affected. The wind blows in a southwesterly direction. The families were possibly affected by radon emission from the K-65 silos along with thoron gas from thorium storage.

As you move south, the next cluster is Branch Hill (Trailer Court). If you continue to move south you notice a tiny cluster. This is the location of some of the contaminated wells. Fernald's effluent line is north of the private wells. Many young adults played in Paddy's Run Creek, which empties into the Great Miami River.

FRESH community study of health outcomes

Health Outcomes	# of Cases	Health Outcomes	# of Cases
Unspecified cancer	253	Female cancers	7
Childhood leukemia	7	Bladder cancer	2
Adult leukemia	1 (worker at site)	Bone cancer	9
Malignant tumors	6	Pancreatic cancer	5
Breast cancer	16	Multiple miscarriages	5
Brain tumors	4	Birth defects	5
Testicular cancer	4	Learning disability	7
Colon cancer	18	Guillain-Barré Syndrome	1
Liver cancer	9	Pituitary tumors	4
Stomach cancer	3	Kidneys	10
Lymphoblastic lymphoma in infants	2	Turned greenCworker	1
Thorium/U-235/U-238	2	Adrenal cancer	1
Lung cancer	18	Babies born with 18 chromosomes	1
Respiratory disease	3	Throat tumors	3
Adenoid cystic carcinoma	1	Rare heart defects/stillborn	2
Melanoma	5	Soft tissue disease	3
Hodgkin=s disease	1	Reproductive problems (male)	3

Prostate cancer	1	Idiopathic bilateral fibrosis	1
Thyroid diseases	13	Spina bifida	1
Hepatitis	2	Chronic fatigue syndrome	1
Blood disorders	3	Chronic depression	3
Blood clotting disorders	1	Diabetes	1

APPENDIX D - SUMMARY OF THE FERNALD DOSIMETRY RECONSTRUCTION PROJECT

In response to community concerns, the Centers for Disease Control's (CDC's) National Center for Environmental Health (NCEH) contracted the Radiological Assessments Corporation (RAC) to perform an extensive assessment of the amount of radioactive materials released from Fernald during its years of operation (1951 to 1988) and to estimate radiation doses received by people living near Fernald during its years of operation. The project, called the Fernald Dosimetry Reconstruction Project, was conducted as a series of tasks; the final report, Task 6, was released in September 1998 (Kilough et al. 1998).

The RAC contractors used historical records from the facility and conducted interviews with former and current Fernald employees and area residents to reconstruct routine plant operations, document accidents, and evaluate unmonitored emission sources. They estimated the quantities of radioactive materials released to air, surface water, and groundwater, then used computer models to predict the transport and fate of releases in the environment. They evaluated available environmental monitoring data and compared them with model predictions to verify that the estimates of releases and transport were reasonable. Finally, they estimated radiation exposure doses that resulted from the estimated releases and the potential risks associated with those doses.

To estimate doses, RAC developed nine exposure scenarios representing hypothetical residents of the area. Each of the scenarios used different assumptions about lifestyle, diet, and locations of home, school, and work, all of which are variables that affected the amount of radiation exposure. For example, all scenarios included inhalation exposure; however, the amount of inhalation exposure varied according to many factors such as proximity of the residence to Fernald, activity level, and the amount of time spent outdoors. The nine scenarios were developed to represent a range of typical area residents. The contractors felt that people living near the Fernald site would find that one of the scenarios paralleled their own experiences. The key assumptions used for each of the nine scenarios are shown in Table D-1. All the scenarios considered only exposure that occurred between 1951 and 1988 within 10 kilometers (6.2 miles) of the site, the area designated by the study as the Assessment domain.®

Table D-1. The Nine Exposure Scenarios in the CDC's Fernald Dose Reconstruction Project (CDC 1998)

Scenario Number	Gender	Date of Birth	Years of Exposure	Key Feature	Home	School	Work	Percentage of Diet From Local Sources
1	F	1-1-46	38	Received inhalation exposure close to site	Family farm within 1 mile of site center (Northeast sector)	Elda Elementary, Ross Middle and High Schools	Family farm	Vegetables, fish, beef and poultry: 50% Eggs and milk: 100%
2	M	1-1-51	38	Lived close to K-65 silos	Family farm within 1.2 mile of site center (West sector)	Elda Elementary, Ross Middle and High Schools	Hamilton	Vegetables and poultry: 0% Eggs and milk: 100%
3	M	1-1-51	38	Drank well water	Family farm within 1.2 miles of site center (South sector)	Elda Elementary, Ross Middle and High Schools	Family dairy farm	Vegetables and poultry: 50% Eggs and milk: 100%
4	F	7-15-60	18	Received typical inhalation exposure from 1960 to 1978	2.4 miles from site center in Ross (East-northeast sector)	Elda Elementary, Ross Middle and High Schools	Moved away	Vegetables, eggs, and milk: 10%
5	M	1-1-51	38	Worked outside the area	Near Layhigh, 5 miles north of site center (North sector)	Morgan Elementary, Ross Middle and High Schools	Hamilton	Consumed no food produced or grown locally
6	F	1-1-46	38	Irrigated using water near the site	Family farm 1.9 miles from site center (East-southeast sector)	Elda Elementary, Ross Middle and High Schools	Family farm	Vegetables, fish, beef, and poultry: 50% Eggs and milk: 100%
7	M	1-1-51	38	Irrigated using water farther from the site	6.2 miles from site center in Miamitown (South sector)	Elda Elementary, Ross Middle and High Schools	Miamitown	Vegetables, fish, and poultry: 50% Eggs and milk: 100%

8	M	1-1-70	13	Received exposure as a child in Ross	2.5 miles from site center in Ross (East-northeast sector)	Elda Elementary, Ross Middle and High Schools	Family farm	Vegetables, eggs, milk: 10%
9	M	1-1-51	18	Attended school in Ross and then left area	Near Route 128 6.2 miles from site center (Northeast sector)	Elda Elementary, Ross Middle and High Schools	Moved away	Consumed no food produced or grown locally

Using mathematical models, the RAC contractors estimated the radiation dose that each of these hypothetical residents would have received. The models incorporated a variety of uncertainties associated with the estimates of levels of radiation in the environment. Therefore, the estimated doses are presented as median values with a range between the 5th and 95th percentile of the median values. The range indicates the range of uncertainty surrounding the median estimate.

The results of the FDRP indicate that the majority of the estimated radiation doses for each scenario resulted from breathing radon and radon decay products. For each scenario, the lung was the organ that received the highest radiation dose, and radon and its decay products accounted for 85% to 95% of the dose to the lung, depending on the scenario. The most important factors affecting the radiation dose for each scenario were the duration of residence in the assessment domain and the location of the residence relative to the site. Because the predominant winds in the area are from the southwest, exposures were predicted to be highest to the northeast of the site.

The RAC contractors used studies of underground miners, who were occupationally exposed to radon, to estimate the risk of lung cancer mortality that would result from the estimated exposures to radon and its decay products. The median excess lifetime risk of lung cancer mortality associated with exposure to Fernald-related radiation ranged from 0.1% (1 chance in 1000) for scenario #8 to 1.3% (about 1 chance in 100) for scenario #1. Scenario #1 was designed to represent the realistic maximum inhalation exposure to Fernald-related radiation, because the hypothetically exposed person was assumed to reside less than 1 mile northeast of Fernald. For scenario #1, the 5th percentile value was 0.24%, and the 95th percentile value was 9.6%. This means that the hypothetical person in scenario #1 may have had a risk of lung cancer mortality as low as 0.24%, or as high as 9.6%. The median risk of lung cancer for this scenario, 1.3%, is considered the best estimate of the lung cancer risk for this person.

Reference

Killough, G.G., et al., 1998. Task 6: Radiation doses and risk to residents from FMPC operations from 1951 - 1988. Volumes I and II (final report). Neeses, SC: Radiologic Assessments Corporation (RAC). September, 1998.

APPENDIX E - AIR PARTICULATE RELEASES AND ICRP LUNG MODELS

Air Particulate Releases - Scrubbers and Dust Collectors

The most important sources of air particulate emissions from the Fernald facility are from the dust collector and scrubber systems installed on stacks in the on-site processing plants. Additional releases have occurred from monitored and unmonitored sources (e.g., cooling towers, waste incinerators, and waste pits) and from spills during processing operations (Killough et al. 1998; Volleque et al. 1995).

Scrubbers and dust collectors are two types of treatment system used by the Fernald facility to control air emissions of hazardous and radioactive materials from the production areas. Scrubbers used either acid or caustic solutions to scavenge particles from emissions being discharged to the atmosphere. In the typical wet scrubber system, particles were scavenged by mist droplets, collected by special devices, and recycled to a liquid reservoir called scrub liquor. Often, however, mist droplets would combine to form larger droplets, become re-entrained in air, and escape from the scrubber system. Scrubber systems were installed in Plant 2/3 and Plant 8. Liquid droplets (scrub liquor) emitted from these scrubber systems contained various concentrations of uranium (Killough et al. 1998).

Releases of uranium from Plant 2/3 scrubbers were not sampled until 1988. Samples collected in 1988 indicated that releases from Plant 2/3 scrubbers contributed about 30% of total air releases from the facility (Volleque et al. 1995). Releases before 1988 were estimated using 1988 data and information about plant operations (Killough et al. 1998). The most substantial releases were estimated to have occurred from 1957 to 1961 and from 1974 to 1976. About 40% of releases were estimated to have been small particles of uranium trioxide, with a median diameter of 0.5 microns, that passed through the scrubber into the atmosphere. The remaining 60% of releases were large particles of uranyl nitrate hexahydrate with a mean diameter of 25 to 62 microns.

Plant 8 contained air scrubber systems of various types, which cleansed exhaust air by contact with droplets of caustic liquid. Emissions from these systems were not sampled on a regular basis. Some measurements were made in the 1960s, when uranium releases were presumably highest; some measurements were also made in the 1980s, when Plant 8 production was lower. There are no reported measurements of the particle or liquid droplet sizes for Plant 8 scrubber emissions. It is estimated that 30% of total uranium emitted from Plant 8 scrubbers were small, solid particles of triuranium octoxide less than 10 microns in diameter. The remainder of releases were estimated to have been large droplets, 80 to 180 microns in diameter, of reentrained scrub liquor (Volleque et al. 1995).

Dust collectors were installed on many exhaust stacks to reduce uranium loss to the atmosphere and to minimize worker exposures to uranium in the process area. The majority of uranium emissions were from dust collectors in Plants 4, 5, 7, and 8; the other five facilities (Plants 1, 2/3, 6, 9, and the Pilot Plant) made minor contributions to overall uranium dust emissions (Volleque et al. 1995).

Sampling systems were installed in the dust collector stacks to monitor uranium concentrations. The facility began periodic sampling of some stacks as early as 1953. By 1956, routine monitoring of stack effluent was conducted on a weekly, bi-weekly, or monthly basis in all plants (Killough et al. 1998). From the 1960s to 1980s, the frequency of sampling varied over time and type of processing plant, but generally was conducted monthly.

A review of dust emissions data revealed that release estimates were incomplete or not available for several periods while the facility was operating. This is because dust samplers were not always operating or releases were too low to be detected. In addition, routine dust monitoring was not conducted until 1956, although the facility began operating in 1951. Therefore, estimates of uranium releases were made for these periods using other sources of environmental data and information about sampling and analysis procedures (Killough et al. 1998).

The only measurements of particle sizes for stack emissions were made in 1985. Measurements were made at inlet and outlet ducts for the major uranium stacks with dust collectors. Measured and estimated median particle sizes and distributions for the major uranium compounds released from uranium processing buildings during past operations at the Fernald facility are presented in Table E-1 (below).

For dust collector emissions, the median particle size ranges from 5.1 to 11 microns and the range covering the 25th to 75th percentile size distribution is 2.1 to 14 microns. This means that there is a 50% chance that the estimated particle size lies between the 25th and 75th percentile values. For scrubber emissions, the median particle size range is much wider, from 0.5 to 62 microns.

Lung Dosimetry Models

The International Commission in Radiological Protection (ICRP) has developed several lung dosimetry models to predict deposition of airborne particulates in various regions of the human respiratory tract (ICRP 1979, 1994, 1995). The models assume that particulates with an activity median aerodynamic diameter (AMAD) of 1 micron are deposited in three regions of the respiratory system, including (1) the most exterior region or nasal passage (N-P); (2) the middle region, or trachea and bronchial tree (T-B); and (3) deepest region, or pulmonary parenchyma (P).

ATSDR scientists made the conservative assumption that uranium emissions from processing areas at the Fernald site were 5 microns in diameter. This value represents the lower bound of the range of median particle size for dust collectors, and is near the lower end of that range for scrubbers. It is also near the lower bound of the range defined by the 25th to 75th percentile particle size distributions for dust collectors.

Table E-1. Particle size distributions for measured and estimated uranium air releases from dust collector outlets and scrubbers at the Fernald facility processing plants

Release Source	Uranium Compound	25 th to 75 th Percentile Particle Size Distribution (in microns)	Median Particle Size (in microns)
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Dust Collectors			
Composite dust collectors	UF ₄ (uranium hexafluoride)	4.2B14	NA
Composite dust collectors	U ₃ O ₈ (triuranium octaoxide)	2.8B9.6	NA
Plant 1 (ore handling)	U ₃ O ₈ , UO ₃ , UO ₂	2.1B14 (U ₃ O ₈)	7 (U ₃ O ₈)
Plant 2/3 (ore handling)	U ₃ O ₈ , UO ₃ , UO ₂	Refer to composite for U ₃ O ₈	7 (U ₃ O ₈)
Plant 4 (hydrofluorination)	UF ₄	Refer to composite	8.1
Plant 5 (foundry operations)	U ₃ O ₈	Refer to composite	6.0
Plant 6 (machining)	U ₃ O ₈	2.4B11	5.1
Plant 7 (UF ₆ reduction to UF ₄)	UF ₄	Refer to composite	9B11
Plant 8 (scrap recovery)	U ₃ O ₈	Refer to composite	NA
Plant 9 (machining)	U ₃ O ₈	Refer to composite	5.1
Pilot Plant (UF ₆ reduction to UF ₄ , and U ₃ O ₈ discharges)	UF ₄ , U ₃ O ₈	Refer to composite for UF ₄ and U ₃ O ₈	9B11 (UF ₄) NA (U ₃ O ₈)
Scrubbers			
Plant 2/3	UO ₃ (uranium trioxide)	NA	0.5
	UNH (uranyl nitrate hexahydrate)	NA	25B62
Plant 8	U ₃ O ₈	NA	Less than 10
Key NA = data not available Source: Voilleque et al. 1995			

ATSDR scientists used information developed by the International Commission on Radiological Protection (ICRP 1979, 1994, 1995), which describes the relationship between particulate size and deposition in the respiratory tract, to estimate the deposition of particles 5 microns in diameter. As particle size

increases, more particles are deposited in the upper regions of the respiratory tract and fewer are deposited in the deep regions of the respiratory tract. The estimated percent deposition of particles 5 microns in diameter (or with an AMAD of 5 microns) is 73% in the N-P region, 8% in the T-B region, and 8% in the P region. ATSDR scientists used these deposition fractions to estimate exposure doses for inhalation of uranium emissions (releases) and re-entrained uranium-contaminated soils.

The dosimetry models also account for clearance of material from the respiratory tract by absorption into the bloodstream. Absorption of particulates from the lungs depends primarily on the particle size and the solubility of the compound. In general, smaller particles (AMAD less than 2 microns) of more water soluble compounds are more rapidly absorbed into blood than larger, less soluble compounds (NEA/OECD 1988). Dosimetry models estimate that the fraction of small (AMAD of 1 micron), soluble particles removed from each respiratory region by absorption into the blood is 50% for the N-P region, 95% for the T-B region, and 80% for the P region (ICRP 1979, 1994, 1995; ATSDR 1999).

Larger-sized particles, such as the 5-micron particles assumed by ATSDR scientists for air releases from the Fernald facility, would not be absorbed as completely as the small particles assumed in these models. However, as a conservative measure, ATSDR scientists assumed that absorption of 5-micron particles would be the same as absorption of 1-micron particles used in the ICRP models.

Uranium particles deposited in the trachea and bronchi may be transported upwards toward the throat by mucociliary action, then swallowed. According to the dosimetry models for deposition and absorption, 5% of particulates deposited in T-B region are not absorbed into blood and may be swallowed. For purposes of estimating exposure doses for inhalation, ATSDR scientists assumed that 5% of swallowed particulates are absorbed into blood from the gastrointestinal tract (ICRP 1979, 1994, 1995; ATSDR 1999).

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APPENDIX F - COMPARISON OF ATSDR AND NAREL AND DOE RADON MONITORING PROGRAM DATA

DOE's Fernald Environmental Management Project (FEMP) operates an ambient radon monitoring program. In the past, the program had two separate monitoring systems: (1) a real-time monitoring system using Pylon detectors that operate continuously and provide radon concentration data for set time intervals, such as hours, and (2) a long-term monitoring system using alpha-track etch detectors (radon cups) that measure total radon concentration over longer intervals. In more recent years, FEMP used a 6-month monitoring period for the alpha-track detectors, which provided an estimate of long-term radon concentrations but was not extremely sensitive to fluctuations in radon concentration (i.e., they were not very sensitive to individual radon releases). The Pylon detectors are more effective at detecting short-term fluctuations in radon concentrations, such as those expected from a release.

In 1992, ATSDR entered into an interagency agreement with the U.S. Environmental Protection Agency's National Air and Radiation Environmental Laboratory (NAREL) to monitor environmental radon in the vicinity of the Fernald site. Data collected by NAREL in 1993 and 1994 were presented in ATSDR's Health Consultation for the K-65 sites, issued in May 1995. NAREL has continued to monitor for radon near the site since that time. The information provided in this appendix compares the NAREL data for 1995 and 1996 with the radon monitoring results reported by FEMP for these years.

The alpha-track detectors used by FEMP are very similar to the detectors used by NAREL. NAREL uses three alpha track detectors at each location. In 1995 and 1996, FEMP used two or three alpha track detectors at each location (DOE 1972 - 1999). The biggest difference between the two programs is that the majority of the FEMP's monitors were located on site or at the fence line, while NAREL's monitors were located at selected residences surrounding the site. However, a comparison between the radon monitoring programs can be conducted for similar time frames to determine if the programs are finding similar results.

FEMP radon monitoring data from 1995 were obtained from the Table 25 of the 1995 Site Environmental Report (DOE 1972 - 1999). Results for FEMP's 1996 radon monitoring programs were obtained from the 1997 Integrated Site Environmental Report (DOE 1972 - 1999). The 1997 data were also available in this report, but were altered, reportedly to correct the results for bias of the detectors. Since this was not done in previous monitoring periods, the FEMP data for 1997 were not considered comparable to previous years. Therefore, the comparison period consists of two years, 1995 and 1996.

To compare the results of the two monitoring programs, the data for each were plotted for similar time periods. A plot of the average results for 1995 and 1996 is shown in Figure F-1. Figure F-1 shows that over the 2-year period, the differences between the two data sets are not statistically significant.

Table F-1 summarizes the results for each monitoring system for 1995 and 1996, and shows a bias toward higher readings for NAREL in 1995 and FEMP in 1996. The table also shows an agreement in the results when averaged over the 2-year period. These biases could be caused by using different manufacturers' devices or by the different time periods monitored. The exposure time for FEMP monitors was 6 months, whereas the exposure period for NAREL monitors was approximately 4 months. Data from 27 locations were averaged from the FEMP program, and data from 10 locations were averaged from the NAREL program.

Table F-1. Summary of comparison between FEMP and NAREL radon monitoring (values in pCi/L)

Program	Statistic	1995 Results	1996 Results	1995/1996 Results
FEMP	Range	0.5B1.1	0.6B1.0	0.5B1.1
	Average ± Standard Deviation	0.75 ± 0.14	0.78 ± 0.10	0.77 ± 0.12
NAREL	Range	0.8B1.2	0.4B0.7	0.4B1.2
	Average ± Standard Deviation	1.01 ± 0.13	0.54 ± 0.10	0.78 ± 0.27
Key pCi/L = picocuries per liter				

According to the 2 years of radon monitoring around the FEMP site, there does not appear to be a difference in long-term concentrations measured by the FEMP program and the NAREL program. Therefore, FEMP data were used for 1989 through 1998 to determine current potential exposure off site to radon and radon decay products.

Reference

DOE, 1972 - 1999. Environmental monitoring annual reports for 1972 - 1995, and the integrated environmental monitoring program for 1997 - 1999. U.S. Department of Energy, Fernald Environmental Management Project (and Feed Materials Production Center).

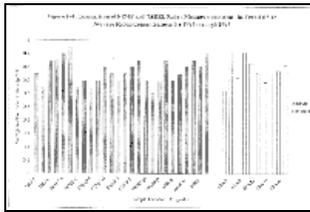


Figure F-1. Comparison of FEMP and NAREL Radon Measurements near the Fernald Site

APPENDIX G - ATSDR'S GLOSSARY OF TERMS

Absorption The process of taking in, as when a sponge takes up water. Chemicals can be absorbed through the skin into the bloodstream and then transported to other organs. Chemicals can also be absorbed into the bloodstream after breathing or swallowing.

Activity (Radioactivity) The number of nuclear transformations occurring in a given quantity of material per unit of time.

Acute Occurring over a short time, usually a few minutes or hours. An acute exposure can result in short-term or long-term health effects. An acute effect happens a short time (up to 1 year) after exposure.

Ambient Surrounding. Ambient air is usually outdoor air (as opposed to indoor air).

Analyte A chemical component of a sample to be determined or measured. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Background Level A typical or average level of a chemical in the environment. Background often refers to naturally occurring or uncontaminated levels.

Background Radiation Radiation resulting from cosmic rays and naturally occurring radioactive material. Background radiation is always present, and its level can change with altitude and the amount of radioactive material present in soil and building materials.

Becquerel (Bq) A unit of measure for a quantity of radioactive material; one becquerel is that quantity of radioactive material in which one atom decays in one second (1 Bq = 1 dps = 27 pCi).

Biological Indicator of Exposure Biomedical testing or the measurement of a chemical

(analyte), its metabolite, or another marker of exposure in human body fluids or tissues in order to validate human exposure to a hazardous substance.

Biological Monitoring Measuring chemicals in biological materials (blood, urine, breath, etc.) to determine whether chemical exposure in humans, animals, or plants has occurred.

Biological Uptake The transfer of hazardous substances from the environment to plants, animals, and humans. This may be evaluated through environmental measurements, such as measurement of the amount of the substance in an organ known to be susceptible to that substance. More commonly, biological dose measurements are used to determine whether exposure has occurred. The presence of a contaminant or its metabolite in human biologic specimens, such as blood, hair, or urine, is used to confirm exposure and can be an independent variable in evaluating the relationship between the exposure and any observed adverse health effects.

Body Burden The total amount of a chemical in the body. Some chemicals build up in the body because they are stored in fat or bone or are eliminated very slowly.

Carcinogen Any substance that may produce cancer.

Carcinoma Any malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case Study The medical or epidemiologic evaluation of a single person or a small number of individuals to determine descriptive information about their health status or potential for exposure through interview or biomedical testing.

CERCLA The Comprehensive Environmental Response, Compensation, and Liability Act of 1980, also known as Superfund. This is the legislation that created ATSDR.

Chronic Occurring over a long period of time (more than 1 year).

Committed Effective Dose The International Commission for Radiological Protection (ICRP) term for the sum of the products of the weighting factors applicable to each body organ or tissue that is irradiated and the committed equivalent dose to the organs or tissues. The committed effective dose is used in radiation safety because it implicitly includes the relative carcinogenic sensitivity of the various tissues.

Committed Equivalent Dose The equivalent dose to organs and tissues of reference that will be received from an intake of radioactive material by an individual over the 50-year period following the intake.

Comparison Values Estimated contaminant concentrations in specific media that are not likely to cause adverse health effects, given a standard daily ingestion rate and standard body weight. The comparison values are calculated from the available scientific literature on exposure and health effects.

Concentration The amount of one substance dissolved or contained in a given amount of another. For example, sea water contains a higher concentration of salt than fresh water.

Contaminant Any substance or material that enters a system (the environment, human body, food, etc.) where it is not normally found.

Curie (Ci) The quantity of radioactive material in which 37 billion transformations occur per second, which is approximately the activity of 1 gram of radium.

Decay Product (Daughter Product, Progeny) Radioisotopes that are formed by the radioactive transformation of some other radioisotope.

Decay, Radioactive Transformation of the nucleus of an unstable nuclide by spontaneous emission of charged particles and/or photons.

Depleted Uranium Uranium in which the percentage of uranium 235 to total uranium of all isotopes is decreased from 0.72% to a lower value.

Dermal Referring to the skin. Dermal absorption means absorption through the skin.

Dose The amount of a substance to which a person is exposed. For chemicals, dose often takes body weight into account. For radioactive materials or radiation, dose denotes the quantity of radiation or energy absorbed and is a generic term for absorbed dose, dose equivalent, effective dose equivalent, committed dose equivalent, committed effective dose equivalent, or total effective dose.

Enriched Uranium Uranium in which the percentage of uranium 235 to total uranium of all isotopes is increased from 0.72% to a higher value.

Environmental Contamination The presence of hazardous substances in the environment. From the public health perspective, environmental contamination is addressed when it potentially affects the health and quality of life of people living and working near the contamination.

Epidemiology The study of the occurrence and causes of health effects in human populations. An epidemiological study often compares two groups of people who are alike except for one factor, such as exposure to a chemical or the presence of a health effect. The investigators try to determine if any factor is associated with the health effect.

Exposure Contact with a chemical by swallowing, by breathing, or by direct contact (such as through the skin or eyes). Exposure can be short-term (acute) or long-term (chronic).

Exposure Registry A system for collecting and maintaining in a structured record, information on persons with documented environmental exposure(s). The exposure registry evolved from the need for fundamental information concerning the potential impact on human health of long-term exposure to low and moderate levels of hazardous substances.

Geographic Information System (GIS) A computer hardware and software system designed to collect, manipulate, analyze, and display spatially referenced data for solving complex resource, environmental, and social problems.

Gray (Gy) The international (SI) unit of absorbed radiation dose. One gray equals the absorption of one joule of energy per kilogram of absorber. One Gy equals 100 rad.

Hazard A source of risk only if an exposure pathway exists, and if exposures create the possibility of adverse consequences.

Health Consultation An ATSDR response to a specific question or request for information pertaining to a hazardous substance or facility (which includes waste sites). It often contains a time-critical element that necessitates a rapid response; therefore, it is a more limited response than an assessment.

Health Education A program of activities to promote health and provide information and training about hazardous substances in the environment that will result in the reduction of exposure, illness, or disease. This program - both national and site-specific in focus - includes diagnosis and treatment information for health care providers and activities in communities to enable them to prevent or mitigate the health effects from exposure to hazardous substances at hazardous waste sites.

Health Outcome Data A major source of data for public health assessments. The identification, review, and evaluation of health outcome parameters are interactive processes involving the health assessors, data source generators, and the local community. *Health outcome data* are community specific and may be derived from databases at the local, state, and national levels, as well as from data collected by private health care organizations and professional institutions and associations. Databases to be considered include morbidity and mortality data, birth statistics, medical records, tumor and disease registries, surveillance data, and previously conducted health studies.

Indeterminate Public Health Hazard A category assigned to sites or pathways for which no conclusions about public health hazard can be made because data are lacking.

Ingestion Swallowing (such as eating or drinking). Chemicals can get into or on food, drink, utensils, cigarettes, or hands where they can be *ingested*. After *ingestion*, chemicals can be absorbed into the blood and distributed throughout the body.

Inhalation Breathing. Exposure can occur from *inhaling* contaminants, because they can be deposited in the lungs, taken into the blood, or both.

Isotopes Any nuclides of the same element having the same number of protons in their nuclei (same atomic number), but differing in the number of neutrons (different mass number or atomic weight).

Media Soil, water, air, plants, animals, or any other parts of the environment that can contain contaminants.

Metabolism All the chemical reactions that enable the body to work. For example, food is *metabolized* (chemically changed) to supply the body with energy. Chemicals can be *metabolized* and made either more or less harmful by the body.

Metabolite Any product of metabolism.

Microcurie One-millionth of a curie, symbolized as μCi .

MilliCurie One-thousandth of a curie, symbolized as mCi .

Minimal Risk Level (MRL) An estimate of daily human exposure to a dose of radiation or a chemical that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity Illness or disease. *Morbidity rate* is the number of illnesses or cases of disease in a population.

National Priorities List (NPL) The Environmental Protection Agency's (EPA) list of sites that have undergone preliminary assessment and site inspection to determine which locations pose immediate threat to persons living or working near the release. These sites are most in need of cleanup.

No Apparent Public Health Hazard A category assigned to sites or pathways where human exposure to contaminated media is occurring or has occurred in the past, but below a level of health hazard.

No Public Health Hazard A category assigned to sites for which data indicate no current or past exposure and no potential for exposure in the future and, therefore, no health hazard.

Picocurie One-trillionth of a curie, symbolized as pCi ($1 \text{ pCi} = 0.037 \text{ Bq} = 0.037 \text{ dps}$).

Plume An area of chemicals or radioactive materials in a particular medium, such as air or groundwater, moving away from its source in a long band or column. A *plume* can be a column of smoke from a chimney or contaminants moving with groundwater.

Public Health Hazard A category assigned to sites or pathways that pose a *public health hazard* as a result of long-term exposures to hazardous substances.

Public Availability Session An informal, drop-by-meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public Comment An opportunity for the general public to comment on Agency findings or proposed activities. The public health assessment process, for example, includes the opportunity for public comment as the last step in the draft phase. The purposes of this activity are to (1) provide the public, particularly the community associated with a site, the opportunity to comment on the public health findings contained in the public health assessment, (2) evaluate whether the community health concerns have been adequately addressed, and (3) provide ATSDR with additional information.

Radiation The emission and propagation of energy through space or through media in the form of waves. The term, when unqualified, usually refers to electromagnetic radiation, such as infrared, visible light, ultraviolet, x-ray, or gamma ray. It can also refer to corpuscular emissions, such as alpha and beta radiation.

Radioactivity The property of certain nuclides to spontaneously transform into another element by emitting alpha or beta particles.

Rem A unit of radiation dose equivalent. The dose equivalent in *rem* is numerically equal to the absorbed dose in rad multiplied by a quality factor.

Risk In risk assessment, the probability that something will cause injury, combined with the potential severity of that injury.

Route of Exposure The way in which a person may contact a chemical substance. For example, drinking (ingestion) and bathing (skin contact) are two different *routes of exposure* to contaminants that may be found in water.

Sievert An international standard unit of radiation dose equivalent. One sievert equals 100 rem.

Specific Activity The total radioactivity of a given nuclide per gram of an element - a measure of the concentration of radioactivity, which may be expressed as $\mu\text{Ci}/\text{gram}$, Bq/L , etc.

Superfund Another name for the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), which created ATSDR.

Toxicological Profiles Documents in which ATSDR scientists interpret all known information on a specific substance and specify the levels at which people may be harmed if exposed. It also identifies significant data gaps in knowledge on substances and serves to initiate further research, when needed.

Urgent Public Health Hazard A category assigned to sites or pathways that pose a serious risk to public health as the result of short-term exposures to hazardous substances.

Volatile Organic Compounds (VOCs) Substances containing carbon and different proportions of other elements such as hydrogen, oxygen, fluorine, chlorine, bromine, sulfur, or nitrogen; these substances easily become vapors or gases. A significant number of the VOCs are commonly used as solvents (paint thinners, lacquer thinner, degreasers, and dry cleaning fluids).

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