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HEALTH AND ENVIRONMENT REPORT and DOCUMENT REVIEW of the COTTER/LINCOLN PARK SUPERFUND SITE

Prepared for:
Colorado Citizens Against *ToxicWaste*, Inc.
P.O. Box 964
Cañon City, CO 81215

JUNE 2007

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June 28, 2007

Colorado Citizens Against ToxicWaste
Attention: Jeri Fry
P.O. Box 964
Cañon City, CO 81212

Dear Jeri:

IEER's contract with the Colorado Citizens Against ToxicWaste (CCAT) to provide technical assistance to review documents related to the Lincoln Park/Cotter Superfund Site cleanup activities has come to a close. The contract was entered on May 17, 2004, and ended on April 1, 2007.

IEER has produced the following as part of this contract:

1. Comments of Arjun Makhijani, Ph.D., on the *Cotter Canon City Uranium Mill 1988 Remedial Action Program Summary and Status – December 2004*, 14 April 2005
2. A study by David Richardson, Ph.D., addressing the overall aspects of epidemiological studies, a critical review of several cancer studies at the Lincoln Park community and the prospects for further research, 1 March 2007
3. A review by Annie Makhijani and Arjun Makhijani, Ph.D., of a vegetable study produced by Richard Graham, Ph.D., for the EPA, 21 March 2007
4. An overview of the long-term issues regarding Cotter mill tailings
5. A compilation of some observations regarding the BEIR VII report
6. IEER's recommendations for public health protection and information regarding contamination linked to or present on the Cotter mill site.

It has been a pleasure to work with CCAT. Please let me know if you have any questions.

Sincerely yours,

Arjun Makhijani, Ph.D.
President

The following IEER documents are attached:

I. Comments on the *Cotter Canon City Uranium Mill 1988 Remedial Action Program Summary and Status – December 2004*, 14 April 2005
II. *Review of Richard Graham's Report on the Health Risks from the Consumption of Vegetables Irrigated with Contaminated Water*, by Annie Makhijani and Arjun Makhijani, 21 March 2007
III. *Epidemiologic Studies of the Cancer near the Cotter Uranium Processing Facility*, by David Richardson, March 1, 2007
IV. Long-term issues regarding Cotter mill tailings
V. Some observations regarding the BEIR VII report
VI. IEER's Recommendations

Attachment I

Comments on the *Cotter Canon City Uranium Mill 1988 Remedial Action Program Summary and Status – December 2004*, 14 April 2005

Comments of Arjun Makhijani, Ph.D., president, Institute for Energy and Environmental Research, Takoma Park, Maryland, on the *Cotter Canon City Uranium Mill 1988 Remedial Action Program Summary and Status – December 2004*. 14 April 2005

1. **Comments on Section 23, Air Monitoring**

The following represent my comments and recommendations on description of radionuclide air monitoring and related activities by Cotter Mill:

- The thorium-230 dose estimate of 3.5 mrem per year is well over 10 percent of the allowable maximum under the fuel cycle limit. No uncertainty is provided for this estimate or any other estimate of radiation dose via the air pathway. Further, Th-230 concentrations are showing an upward trend at offsite locations. The cause(s) of this upward trend are not described.

  Recommendations:

  1. Cotter should estimate the uncertainties in dose and show the 95 percent upper confidence bound for all radiation doses.
  2. Cotter should determine the causes of the offsite up-trends in Th-230 concentrations and report them as part of its air monitoring section in the RAP Status Reports.

- Cotter operated a high-wind-actuated system to automatically monitor air concentrations of radionuclides for three years. This system showed increases in radionuclide concentrations during high-wind events. Yet, the monitoring has been discontinued. Monitoring during high wind episodes provides confidence that dust control measures are effective enough to protect the public. Without this monitoring, there will be doubt about the effectiveness of these measures for certain periods especially since Cotter does not maintain liquid cover over the all tailings areas. For instance, Cotter has a "performance objective" of maintaining the main impoundment cover to an elevation of only 5580 feet above mean sea level, as opposed to 5598 feet envisioned in the design. Given the requirement of active dust control, and the issues that Cotter faces with the community, the high wind monitoring should not have been discontinued.

  Recommendation

  3. The event actuated, high wind air monitoring system should be restored and enhanced at the points where new housing is being built.

- Cotter has added one air monitor "between the mill property and the golf course, and one near the entrance to the golf course. New houses are being constructed in this area" (p. 21).

  Recommendation

4. The adequacy of this system for detecting accidental releases of radioactivity from Cotter Mill should be evaluated under different assumptions of meteorological conditions during accidents and additional air monitors should be added as needed to be able to detect all plausible accidents with high probability.

2. Comments on Sections 16 through 22

- Cotter proposes to consolidate these sections into one since "all deal with fugitive dust emissions." (p. 18). Such a consolidation is not desirable for several reasons. First, each area of fugitive emissions has source term characteristics that are typical to that source. Consolidation of the sections would tend to obscure these different source term. Second, a lack of clarity in the analysis of each dust source would make it more difficult to determine the effectiveness of proposed mitigation measures. Third, the mitigation measures would be more complex to evaluate. And finally, the process of dust control would be less transparent to the public and to regulators.

  Recommendation

  5. Sections 16 to 22 should be maintained as separate sections.

- Cotter proposes to remove section 19. The removal of this section would remove a discrete source of information about a source of dust that still remains. See above for more details regarding the desirability of maintaining a separate section for each dust control area.

  Recommendation

  6. Section 19 should be maintained as a separate section.

- Cotter inspects dust control areas, including the main impoundment and the old tailings pond area, by frequent visual inspection. Such visual inspections are desirable and should be maintained. There do not appear to be PM-10 or PM-2.5 air monitors to monitor dust loading near the main impoundment or the old tailings ponds. This deficiency is more important in view of the fact that the liquid level in the main impoundment is not maintained at the original design level of 5598 feet above mean sea level.

  Recommendation

  7. Cotter should install PM-10 and PM-2.5 monitors to monitor the air in the area of the main impoundment and the old tailings ponds.

Attachment II

***Review of Richard Graham's Report on the Health Risks from the Consumption of Vegetables Irrigated with Contaminated Water*, by Annie Makhijani and Arjun Makhijani, 21 March 2007,**

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Review of Richard Graham's Report on the Health Risks from the Consumption of Vegetables Irrigated with Contaminated Water

Annie Makhijani and Arjun Makhijani, Ph.D.

Institute for Energy and Environmental Research, Takoma Park, Maryland

Prepared for Colorado Citizens Against ToxicWaste (CCAT), Cañon City, Colorado

21 March 2007

A. Introduction

We have reviewed Dr. Richard Graham's report, hereafter referred to as the *Graham Report*[1], which addresses the health risks arising from the consumption of locally grown vegetables in the Lincoln Park area. These vegetables are being irrigated with uranium- and molybdenum-contaminated water. In an e-mail to IEER, Dr. Graham, who is an Environmental Toxicologist/Radioecologist in the Region 8 office of the Environmental Protection Agency (EPA) has stated that he produced this report as a "White Paper" for his "RPM [Remedial Project Manager] Rebecca Thomas."[2] The *Graham Report* was produced to address the concerns of the Colorado Citizens Against ToxicWaste (CCAT) regarding the health effects that could arise from the consumption of the vegetables. Our review covers only the part of the *Graham Report* relating to uranium contamination.

The *Graham Report*, which was presented at a 16 November 2006 Community Advisory Group (CAG) meeting in Cañon City, relies on a literature review to calculate what the level(s) of contamination in well water would need to be to cause a health concern. The *Graham Report* concludes that the consumption of vegetables even when irrigated with highly contaminated water would not pose a risk to human health. In this review we first present a brief description of the location and the extent of the contamination. We then examine the premises and parameters used in the *Graham Report* to examine the validity of its conclusions.

IEER retained an independent consultant, Dr. Michael C. Thorne to do an independent scientific (rather than regulatory) review of the *Graham Report*, without sending him our own draft. Dr. Thorne, who served as a scientific secretary to the ICRP, came to the similar conclusions. His review is included as Attachment 1 and his Vita is Attachment 4.

Site, wells, and uranium plume contamination location

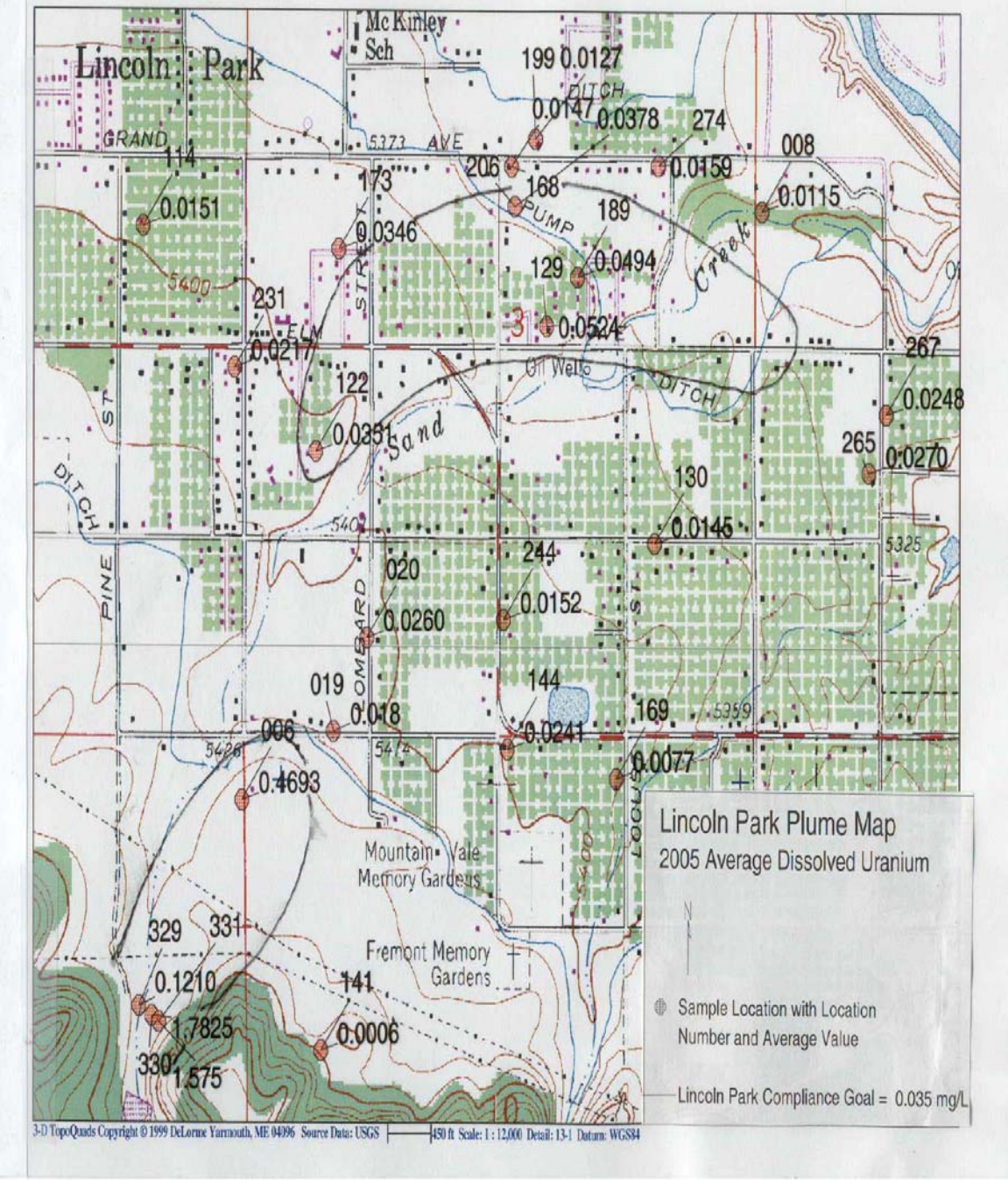
The Lincoln Park area is situated about 1.5 miles north of the Cotter uranium mill.[3] Contaminants from the mill have leached into the groundwater and contaminated wells in the area. Figure-1 shows the uranium plume that lies under Lincoln Park and the wells that are monitored for contamination, along with their average level of contamination found in 2005. It also shows another plume located partly under the site, which includes the most highly contaminated wells. Table-1 lists the same wells and levels of contamination as Figure-1.

The Lincoln Park uranium plume is defined to include all wells that are above the compliance goal of 35 micrograms per liter.[4] The compliance goal is 5 micrograms per liter higher than the 30 micrograms per liter EPA standard for drinking water and Lincoln Park ROD standard.[5] Over

[1] The Graham Report, presented at the CAG meeting, held 16 November 2006, bears no title, author statement, or date. Dr. Graham has confirmed that he is the author of this "white paper." (Graham 2007)
[2] Graham 2007
[3] EPA 2006
[4] Cotter 2006 page 7-1. See also Figure 1.
[5] 40 CFR 141.66 (e) 2006, EPA ROD 2002, and EPA 2002

all, the contamination levels in the monitored wells vary from 0.6 to 1,782.5 micrograms per liter.[6] At least one of these wells, #168, has been used for drinking water. (see Attachment 2).

Figure 1: Plume map for dissolved uranium in groundwater below Lincoln Park



Source: Cotter 2006, Figure 7-26 (page 7-21)
Note: Each well has a pair of numbers associated with it: the monitoring identification and the average uranium concentration, in milligrams per liter.

[6] Cotter 2006 Table 7-3 (page 7-19)

Table 1: 2005 average uranium concentrations for the Lincoln Park groundwater wells

Sample Location Number	Average Uranium micrograms per liter (μg/L)	Remarks
006	469.3	Monitoring well, above the compliance goal
008	11.5	
019	18.0	Compliance well
020	26.0	Compliance well
114	15.1	Background well, only for the third quarter
122	35.1	Monitoring well, above the compliance goal
129	52.4	Above compliance the goal
130	14.5	
141	0.6	
144	24.1	
168	14.7	
169	7.70	
173	34.6	Above the EPA standard
189	49.4	Above the compliance goal
199	12.7	
206	37.8	Above the compliance goal
231	21.7	
244	15.2	
265	27.0	
267	24.8	
274	15.9	
329	121.0	Above the compliance goal
330	1,782.5	Above the compliance goal
331	1575.0	Above the compliance goal

Source: Cotter 2006, adapted from Table 7-3 (page 7-19)

According to Sharyn Cunningham of Colorado Citizens Against ToxicWaste (CCAT) all wells North of 006 are private. Specifically, all wells with 100 and 200 series identification numbers are private.[7] Hence there are currently four wells contaminated above the compliance goal of 35 micrograms per liter.

B. Graham Report Methodology

The *Graham Report* says that, for uranium, the risks from the chemical toxicity exceed those from the radiological toxicity:

> Natural uranium's greatest health risks are derived not from its radiological properties but from its heavy metal chemical toxicity (ATSDR, 1999; BEIR IV). Chemical toxicity exceeds radiological health impacts in importance and concern when the radiological decay half-life of the element is very long (a radionuclide's low specific activity); the element is not effectively bio-

[7] Stephens & Associates 1993 Table 3

> accumulative; and, when radiological emissions are predominately alpha emitters that have low biological impacts (Sheppard et al, 2005). In the case of natural uranium the principal uptake exposure pathway uptake is from ingestion of food and water with the primary organ of concern being the kidney (Wrenn et al., 1985). (page 1)

The report then attempts to substantiate this statement with calculations for a range of concentration levels of uranium in water above which its use for vegetable irrigation would pose a risk to human health. The steps it uses are as follows:

- First, a Food and Drug Administration (FDA) equation is used to calculate the Derived Intervention Level (DIL) for uranium in food. (pages 1 and 2)
- Then, a concentration ratio (which is the ratio of the concentration of uranium in the plant to the concentration of uranium in the soil) is applied to calculate what concentration of uranium in the soil would correspond to the Derived Intervention Level in the food. (page 3)
- Finally several partition coefficients, or K_ds, (concentration of uranium in soil/concentration of uranium in water) including a site specific coefficient are used to calculate what concentration of uranium in the water would correspond to the concentration of uranium in the soil. (page 3 and 4)

The FDA equation for the Derived Intervention Level requires the specification of a maximum allowable dose. The *Graham Report* used a dose limit of 1 millisievert, whole body effective dose equivalent (EDE) and derives a contamination level in food of 16.8 parts per million.

The *Graham Report* then estimates the allowable concentration of uranium in the soil that would result in a concentration of 16.8 ppm of uranium in food. Since plant roots do not take up uranium very efficiently – the factor varies by plant and by soil and moisture conditions – the allowable concentration in soil is much larger than that in food. The *Graham Report* uses a concentration ratio (or transfer ratio) of 0.008 – that is for 1 unit of uranium in soil, the vegetable would have 0.008 units of uranium. Hence the report estimates the maximum concentration in soil as 16.8/0.008 = 2,100 parts per million. The transfer factor of 0.008 is not a site specific factor, but rather a generic factor chosen from a computer model for dose calculations, called RESRAD (for residual radioactivity) developed by Argonne National Laboratory and used by the Department of Energy and the Nuclear Regulatory Commission.

Finally, the *Graham Report* estimates the corresponding maximum water contamination by using a site specific partition coefficient, known as the K_d factor. This factor essentially rolls up into one number the properties of the soil and water that result in water mobilizing a contaminant present in the soil. It is the ratio of soil contamination to water contamination; it can be empirically determined from local conditions. It is important to determine it locally, since the K_d factor varies a great deal from one site to another. In the case of the Lincoln Park Site, the local K_d was estimated by the *Graham Report* as 23 milliliters per gram -- that is, if the concentration of uranium in soil were 23 micrograms per gram, the concentration of uranium in water would be 1 microgram per milliliter.

This distribution coefficient yields a value of water contamination which is about 90 micrograms per cubic centimeter, or 90,000 micrograms per liter. This level is 3,000 times above the

drinking water limit of 30 micrograms per liter and the EPA mandated level for Lincoln Park. According to the *Graham Report*, which considers only vegetable contamination via root uptake, a safe level of water contamination for irrigation would be 90,000 micrograms per liter of natural uranium.

We have a number of scientific, regulatory, and public health criticisms of the calculations and results in the *Graham Report*:

1. 100 millirem EDE is not a "safe radiological dose"
2. The appropriate regulation, 40 CFR 190, should be used to determine the upper limit of dose for the calculation of permissible water contamination, even if only root uptake is considered
3. The report does not consider contamination of vegetables by any other way than root uptake – for example, direct contamination on the leaves and absorption from irrigation spray (see Attachment 1)
4. The report incorrectly compares the calculated concentrations in the well to the measured values (Table-1).
5. The report does not consider all pathways.
6. The report does not consider the health risks of uranium emerging in recent research, such as that being done at the Armed Forces Radiobiology Research Institute.

C. Safe Radiological Dose?

The *Graham Report* speaks of 1 millisievert as "a safe public radiological dose."[8] This is a surprising statement of safety coming from an EPA scientific official. The EPA has taken the position for decades, supported by reports from the National Research Council, that every increment of dose produces a corresponding increment of cancer risk.[9] Therefore, there is *no safe dose of radiation* – that is there is no dose at which the risk reduces to zero. Further, it should be noted that all radiation caused by human activities is on top of a substantial natural background dose. There is no reason to suppose that natural background radiation does not cause some of the cancers that occur as people age, even when they are not exposed to anthropogenic sources of radiation. Indeed, there are very sound reasons to conclude that it does. The reference to 100 millirem as a "safe dose" is in error and contrary to the underlying philosophy of EPA radiation protection standards.

Moreover, 100 millirem per year is the wrong choice of a dose limit since it would allow public health risk grossly in excess of that permitted by present radiation protection regulations.

D. Dose Limits for the Nuclear Fuel Cycle

EPA standards are often geared to a maximum dose to the most exposed organ, which may be the whole body in some cases (like tritium). However, when uranium is ingested in soluble

[8] Graham Report page 2. On the same page, the report states that 1 mSv [millisievert] = 100 mrad." This is incorrect for uranium. 1 mSv = 100 millirem, by definition. However, for alpha emitters, where a quality factor of 20 is applied, a dose of only 5 millirad (mrad) converts 100 millirem, which is the same as 1 mSv.
[9] NAS/NRC 2006

form, the most affected organ is the bone surface, and not the whole body. Therefore the calculation should have been done for this organ.

The specific regulation that applies to nuclear fuel cycle facilities, including uranium mills, is published in the *Code of Federal Regulations* at 40 CFR 190 Subpart B (2006). This rule limits the maximum annual dose equivalent to 75 millirem to the thyroid, 25 millirem to the whole body, and *25 millirem to any other organ*. The organs for which doses can be calculated and the corresponding dose conversion factors (the dose per unit of radioactivity ingested or inhaled) are specified in Federal Guidance Report 13, which is the EPA's most recent publication on these matters. For uranium ingestion the critical organ is the bone surface with a dose coefficient of 7.11×10^{-7} sieverts per becquerel.[10] This dose coefficient is about 16 times greater than that used in the *Graham Report*, which is the dose conversion factor for the whole body. When these two factors are taken into account – a factor of four lower dose and a factor of sixteen larger dose conversion factor, the *Derived Intervention Level (DIL) for uranium in food is 64 times lower than in the Graham Report: 0.27 parts per million, instead of 16.8 parts per million.*

The maximum soil contamination corresponding to 0.27 ppm is about 33 ppm (64 times lower than the value in the *Graham Report*). The maximum allowable water concentration would then be about 1,430 micrograms per liter rather than the 90,000 micrograms per liter calculated by Dr. Graham.

Further, as noted in Attachment 1, leaves and vegetables would have residual water on them and would also absorb some uranium directly from deposited water. Hence, the maximum contaminant level should be significantly lower than the 1,430 micrograms per liter estimated for root uptake alone, even if vegetables are considered as the only pathway of exposure.

E. Comparison to Measured Groundwater Concentrations

In describing the result of maximum water concentration for a "safe" dose from the vegetable pathway, the *Graham Report* states:

> water concentrations containing natural uranium have to **be a million times higher than current sampled values** to pose an undue health concern assuming that the parameters taken from peer review literature represents conditions in the Lincoln Park gardens [page 4, Emphasis added]

This does not describe the result of the calculation in the *Graham Report* correctly, leaving aside all other matters. As noted above, the result was that the maximum allowable concentration would be about 90,000 micrograms per liter. The contamination in the compliance wells in Lincoln Park averaged 22 micrograms per liter in 2005. One million times would be 22,000,000 micrograms per liter, which is 244 times the actual result of about 90,000 micrograms per liter.

Another way to look at it is the statement quoted above implies that the water contamination in Lincoln Park is one million times less than 90,000 micrograms per liter. This would put water concentrations of uranium at about 0.09 micrograms per liter. There is no sampled value within a factor of sixty of this number. Sampled values are typically hundreds of times larger. Even the

[10] FGR 13. The dose conversion factors are published in a CD supplement to FGR 13 issued by the EPA in 2002.

sample taken at the background monitoring well at location 114 contained 15.1 micrograms per liter of uranium, which is almost 170 times the value implied by the "million times higher" statement quoted above.

We also note that the committed dose to the bone surface from drinking this water at a rate of 2 liters per day (the EPA standard assumption) is about 118,000 millirem (118 rem) per year. This is more than 4,700 times the 25 millirem annual dose in the relevant EPA regulation in 40 CFR 190 Subpart B. If we use the drinking water regulation as the reference point, the dose from the estimated 90,000 micrograms per liter would be 3,000 times the allowable maximum. (This is simply the ratio of 90,000 micrograms per liter to the regulatory drinking water limit of 30 micrograms per liter). The dose to infants would be about double that to adults, even taking into account the fact that an infant's water intake is lower.

F. Emerging Health Risks of Uranium

IEER has described elsewhere recent research on non-cancer risks of uranium that are indicated recent research. The research is not definitive; it was done at high doses on animals. However, the picture that emerges is disturbing. It is that the heavy metal toxicity of uranium and the radiotoxicity could produce a variety of adverse health outcomes including

- Neutrotoxicity
- Mutagencity
- Negative effects on reproductive success
- Effects on other organs, including skeleton and brain

Uranium may be like lead in the variety of its effects, but radioactive in addition. Attachment 3 is a reproduction of Chapter 8 from a recent IEER report entitled *Science for the Vulnerable* that summarizes some of the relevant research. It should be noted that the present state of research on these non-cancer effects does not allow extrapolation to low doses.

In assessing maximum concentration of uranium in vegetables and the corresponding soil and water concentrations that were presented to the public as "safe" the *Graham Report* should have at least mentioned these emerging health risks and what the EPA might be doing to address them in the future.

G. Conclusions and Recommendations

The *Graham Report* has a number of fundamental problems ranging from errors in arithmetic (as for instance in the "million times" remark), to wrong choice of regulations for the calculations, to misinterpretation of 100 millirem as a "safe" dose, to bypassing important pathways and ignoring emerging health risks of uranium. The result grossly exaggerates the amount of water contamination that would be allowable even if only vegetable contamination were to be considered. Rather than being protective of public health, the approach of the report and its conclusions show a cavalier disregard for sound science, for careful interpretation of regulations, and for public health.

Given the many ways in which vegetables can be contaminated and the fact that private water wells can be and, in the past, have been used for drinking water, the restriction of the calculation or permissible contamination to the root pathway is impractical, unrealistic, and not protective of public health.

Since most of the wells in Lincoln Park are private, drinking water as well as vegetable contamination should be considered in assessing health risk and maximum permissible contamination. In any case, the maximum allowable concentration should in no case exceed the 30 micrograms per liter limit in drinking water regulations. We recognize that this limit applies only to public water systems, in terms of enforcement. However, it should be remembered that drinking water regulations are set in the framework of protecting individuals. In other words, they specify the maximum individual intakes and do not limit population intakes. This was a deliberate choice made in the mid-1970s. The limitation to public water systems is an economic convenience so that small water systems do not have to go to great lengths to remediate contamination, notably natural contamination. It is reasonable and proper to consider the framework as being applicable to private water wells that have been contaminated by the activities of a third party.

We understand that the goal of the *Graham Report* was to calculate levels of water contamination that would produce vegetables that would not be considered risky according to prevailing radiation and public health criteria. The report fails by every major criterion that it can be evaluated even for this limited goal. Further, we find that the implication in the *Graham Report* that 90,000 micrograms per liter of uranium in water (not to speak of a level one million times the measured values) would be "safe" shows a lamentable disregard of public health and of communication of risks to the public. Water supplies that are used by people should conform to the EPA's Safe Drinking Water Standards, even if the systems are private. The fact that the standards are not enforceable for private systems should not be seen by third parties as a license to pollute such water supplies or for the EPA to condone such pollution.

The *Graham Report* is fundamentally flawed from a scientific, regulatory, and public health point of view. IEER recommends that it should be scrapped and redone.

Our second recommendation, not related to the report, but related to the private wells is that the Cotter compliance goal for Lincoln Park wells should be immediately reduced to 30 micrograms per liter. This corresponds also to the EPA Record of Decision that says:

> On December 7, 2000, EPA promulgated a drinking water standard for uranium (0.030 mg/L) [30 micrograms per liter]. Because this new standard is Relevant and Appropriate to the site cleanup, the Remedial Action Plan will be modified to meet the requirements of the new regulation.[11]

At the time of this writing (March 2007) we have not found any evidence that this has been adopted.

[11] EPA ROD 2002. See also EPA 2002.

H. References

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Attachment 1

Dr. Michael C. Thorne's External Memo:

Health Risks from the Ingestion of Vegetables
12 March 2007

Mike Thorne and Associates Limited

(Director: Dr M C Thorne)

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External Memorandum

Date: 12 March 2007
From: M C Thorne
To: Annie Makhijani
Copies:
Subject: Health Risks from the Ingestion of Vegetables

Annie

I apologise for not having previously sent you comments on this report. I had assumed that because the original deadline was the end of February that such comments would not be helpful, but Arjun tells me that this is not the case. Therefore, comments are provided below. I have concentrated on uranium, as this is of principal interest and illustrates the major problems that exist with this paper.

I agree with the introductory remarks that for natural uranium chemical toxicity is generally considered to be of greater significance than radiological toxicity and that the kidney is the primary target organ. However, this statement applies in an occupational context in which the principal annual dose limit is either 50 mSv or 20 mSv, depending upon the regulatory regime. In an environmental context, the principal annual effective dose limit is 1 mSv and relevant annual effective dose constraints for a single source may be considerably less than this. Bearing in mind that there is thought to be a threshold tissue concentration that determines nephrotoxicity, it is not clear that, in an environmental context, chemical toxicity will be more limiting than the more restrictive dose limits and constraints that apply in such a context. Furthermore, it should be noted that it is somewhat artificial to separate chemical and radiological toxicity, as uranium is a known chemical mutagen, so its chemical and radiological properties may contribute synergistically to its overall toxicity in some contexts.

I agree that, in an environmental context, ingestion is of greater concern than inhalation. However, in view of the relatively limited degree to which uranium is taken up by plants, I think that the ingestion pathways should include food, water and dirt.

The calculation at the top of page 2 estimates a Derived Intervention Level (DIL) for uranium. This calculation is based on an annual effective dose value of 0.001 Sv (1 mSv). This value is described as a safe public radiological dose. I do not agree with this description. The value of 1 mSv is the value recommended by the ICRP (1991) as the dose limit for members of the public. The ICRP describes this value as 'just short of unacceptable for continued exposure as the result of deliberate practices the use of which is a matter of choice' (ICRP, 1991, para. S41). Although the situation in intervention is rather different and it may be decided that intervention is not appropriate unless higher dose levels arise, the description of 1 mSv as a safe annual public radiological dose is clearly misleading.

The calculation at the top of page 2 is inappropriate. In the numerator, it uses the effective dose coefficient of $4.5\ 10^{-8}$ Sv Bq^{-1} for ^{238}U. However, as acknowledged immediately above the expression, natural uranium in soils typically contains its daughter products at or close to equilibrium. These products include ^{226}Ra, which has an effective dose coefficient that is much larger at $2.8\ 10^{-7}$ Sv Bq^{-1}, ^{210}Pb with a value of $6.9\ 10^{-7}$ Sv Bq^{-1} and ^{210}Po with a value of $1.2\ 10^{-6}$ Sv Bq^{-1} (ICRP, 1996). It is common knowledge that most of the internal radiation dose from the naturally occurring uranium series radionuclides comes from ^{226}Ra, ^{210}Pb and ^{210}Po and not from their uranium ancestor (see for example Table 5 of Thorne, 2003). It should also be noted that the calculation is undertaken only for adults and no consideration is given to whether children, infants or the embryo and fetus might be more exposed.

Similarly, while concentration ratios for plants are defined appropriately and relevant literature references are cited for uranium, the key issue is suitable concentration ratios for radium, lead and polonium, taking into account the potential for ingrowth of ^{210}Po from ^{210}Pb taken up into plants.

At page 3, a limiting soil concentration of uranium is deduced (incorrectly for the reasons set out above). This is then used as the basis for deriving a limiting concentration in well water. However, the argument adopted is fallacious. The approach adopted is to use an equilibrium sorption coefficient (K_d value) to calculate the concentration in well water that would result in the limiting concentration in soil. However, this approach assumes that plant concentrations resulting from irrigation occur because the contamination is transferred from the well water to soil and thence to plants via root uptake. However, uranium, radium, lead and polonium are only taken up by roots to a limited degree. In these circumstances, the main way in which plants become contaminated during spray irrigation is through direct foliar contamination. The activity may remain as external contamination on the foliage or may be translocated to internal plant parts. This pathway is not even mentioned, let alone addressed. The situation is further complicated by the consideration that whereas uranium-series radionuclides may be close to secular equilibrium in soils, this is not necessarily the case in well waters, because of the differential sorption of the various chain members and kinetic effects. Without an evaluation of these differential effects, it is unclear how a back calculation from

concentrations of uranium in soil (either alone or in secular equilibrium with its progeny) to concentrations in well water can be undertaken.

The information on ecotoxicity comprises a compilation of no observed effect levels, but does not set them in a regulatory context, in which precautionary safety factors would typically be applied. The relevance of this information to the paper is unclear.

Interestingly, the soil screening level given on page 3 is 2100 ppm. At page 5, the EPA methodology is cited as having been used for calculating the soil screening guidance level for ^{238}U plus its progeny. The value given is 14 ppm. No comment is offered on why this level is a factor of 150 lower than that given on page 3. It is apparently based on a risk of $1\ 10^{-6}$, but it is not stated if this is an annual or a lifetime risk.

Overall, I found this paper to be incoherent and incorrectly argued. In my view, it cannot readily be amended and I consider that a new paper needs to be prepared based on more appropriate arguments.

References

ICRP, 1991, 1990 Recommendations of the International Commission on Radiological Protection, ICRP Publication 60, Annals of the ICRP, **21(1-3)**.

ICRP, 1996, Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 5 – Compilation of Ingestion and Inhalation Dose Coefficients, ICRP Publication 72, Annals of the ICRP, **26(1)**.

Thorne, M C, 2003, Background radiation: natural and man-made, J. Radiol. Prot., **23**, 29-42.

Attachment 2

Sharyn Cunningham's E-mail about Wells
20 March 2007

Subject: Re: question re wells
Date: Tue, 20 Mar 2007 04:43:44 -0600
From: Sharyn Cunningham <sharyn@bresnan.net>
To: Annie Makhijani <annie@ieer.org>
References: <45FEB34E.9050004@ieer.org>

Annie,

1. Well 329, 330 & 331 are inside Cotter's boundary. These wells are just below (north) of the Permeable Reactive Treatment Wall (constructed in 2000 and failed to work at cleaning the water within a few months). The PRTW is just below the SCS Dam. They are not used by residents, but are solely for monitoring the groundwater.

2. Well 006 is relatively new (1998?) and is north of 329,330,331. I'm not sure if it's on Cotter property, but it was installed as a monitoring well only. They then stopped testing #138 that is nearby. All wells north of 006 are private. Well 019 and 020 are on private property, but they are only used as Compliance Wells. Supposedly, when those wells reach the Remedial Action Plan cleanup level, Cotter will be able to declare the water remediated or clean or whatever.

3. Wells numbered 100 and 200 are all private wells in Lincoln Park. The D.B Stephens Assessment (1993), see excerpt attached, shows this. Also, there are many pages of a list of private wells in this report and I counted over 100, but the pages are bad copies and you can barely see the names of owners. The RAP in 1988 required Cotter to do a survey of private well use, but no updated investigation of well use has taken place since then. So, yes, there are many wells that aren't tested, and many people use those wells to irrigate lawns, fields and gardens. CDPHE/Cotter will claim they determined the plume and didn't need to test all wells, and they were allowed to drop wells tested in the 90's because the results were below MCL. However, EPA came back in 2001 or 2002 and required Cotter to add about 10 more private wells to 7 or so they were monitoring.

4. We've been told several times at Superfund meetings by CDPHE's Stoffey (Superfund Site Lead) that 5-6 wells in Lincoln Park are used by residents as their only source of water, but I don't know which wells. My family used Well #168 for drinking and domestic purposes, and Well #206 for irrigating alfalfa and our garden, from 1994 to 2002, when we learned it was contaminated. CDPHE and Stoffey claimed there is no risk to these families as all Ur & Mo results were below MCL, however the WHO would disagree. My wells weren't tested by Cotter or CDPHE from 1991 until 2003, all during the time we were drinking the water. However, our Well #206 used for the garden & alfalfa has been above MCL for Ur & Mo. In 2004 and 2005 it was between 0.056 and 0.040 mg/L-Ur, higher in contaminants than it was in 1988, but it dropped in 2006. Well 206 also was not included in the plume, when it should have been, until they tested it in 2003 and then enlarged the northern edge of the contamination plume. If I hadn't gotten involved in fighting Cotter in 2002, they would never have tested our wells, and they would never have known that the northern edge of the plume was at our property.

5. CDPHE has a program on their website with a water well mapping application. Each well is a "blue dot" and if you click on the toolbar below, the red "i", and then click on a well, a data sheet will pop up with info. If you click on the toolbar "Data Reports" and then on a well, a graphing application will show all sampling results for that well. Here is the link: http://www.cdphe.state.co.us/hm/cotter/CMmaps.htm

6. Another concern of mine is that private wells were tested for Total Ur for a decade or so, and then they stopped testing for Total Ur and started testing for Dissolved Ur. That makes it difficult to look at a long-term trend. Originally the private wells were tested for Total Alpha or Beta and then they stopped

that in the 1980's. I think they should test for Total Alpha, because I've seen Ur below standards in a well while Total Alpha was above the accepted standard for drinking water in the same well.

7. The Record of Decision (ROD) in 2002 stated there would be "no further action" on soil remediation, but that the water in Lincoln Park still has Ur & Mo above the required cleanup level (.035mg/L-Ur; 0.1mg/L-Mo). The ROD also states that the Remedial Action Plan (RAP) will be changed for Ur to the 2000 EPA standard of 0.030mg/L. This has not happened - even though we've requested it several times, and the problem is that Cotter draws the Ur contamination plume in Lincoln Park based on 0.035mg/L. See the map you sent me at the bottom, and you'll see this.

Hope that helps,

Sharyn
(719)275-8294

----- Original Message -----
From: Annie Makhijani
To: Sharyn Cunningham
Cc: Arjun Makhijani
Sent: Monday, March 19, 2007 9:59 AM
Subject: question re wells

Hi Sharyn,

Table 7-3 in the 2005 Cotter Environmental report, Section 7: liquid management lists the wells that are being monitored...

 Stephens Assmt-Well Numbering-1993.pdf

Table 3. Categories of Wells and Surface Water Stations
Cotter Uranium Mill
Canon City, Colorado

Well Location Series	Description
000	Unknown
100	Private wells located in Lincoln Park area
200	Private wells located in Lincoln Park area
300	Observation wells located on Cotter uranium mill property
500	Surface water stations not located on Cotter uranium mill property
600	Unknown
700	Surface water stations located on Cotter uranium mill property
800	Piezometers located in Lincoln Park and Cotter uranium mill areas
900	Unknown
1000	Berm withdrawal and 9/16 hydraulic barrier wells (injection and withdrawal wells and piezometers)
1300-1400	Wells (injection, withdrawal, and observation wells and piezometers) used in the pilot test areas on Cotter uranium mill property

DANIEL B. STEPHENS & ASSOCIATES, INC. 1993. ASSESSMENT OF POTENTIAL SEEPAGE IMPACTS ON GROUND WATER, COTTER URANIUM MILL, CANON CITY, COLORADO, VOLUME I.: Summary of existing hydrological data. Table 3. USEPA Document ID 293646.

Attachment 3

Chapter 8 of IEER's report

Science for the Vulnerable: Setting Radiation and Multiple Exposure Environmental Health Standards to Protect Those Most at Risk

October 19, 2006

On the Web at http://www.ieer.org/campaign/report.pdf

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CHAPTER 8 ONLY

Science for the Vulnerable
Setting Radiation and Multiple Exposure Environmental Health Standards to Protect Those Most at Risk

Arjun Makhijani, Ph.D.
Brice Smith, Ph.D.
Michael C. Thorne, Ph.D.

October 19, 2006

Chapter 8: Case Study -- Depleted Uranium[162]

Except for prolonged exposures to bulk quantities in the workplace or by other means, uranium is primarily dangerous when it gets inside the body through ingestion, inhalation, or through breaks in the skin. Inside the body, uranium creates risks both as a toxic heavy metal and as a radioactive material. There are three naturally occurring isotopes of uranium (U-234, U-235, and U-238) but, in general, only uranium-238 is abundant enough and long-lived enough to cause this dual problem.

The half-life of U-238 is 4.46 billion years. The half-life of uranium-235 is also very long, 704 million years, but it never dominates any given mixture of uranium radiologically. That role belongs either to U-234 (for enriched uranium), U-238 (for depleted uranium), or both U-234 and U-238 (for natural uranium). The dual heavy metal toxicity and radiological damage is particularly a problem with depleted uranium, natural uranium, and low enriched uranium. The damage from uranium that is very highly enriched would tend to be dominated by the radiation aspect, due to the presence of U-234 in much higher concentrations than in natural uranium (about 1 percent compared to about 0.005 percent, respectively).

The vast majority of heavy metal radionuclides, like plutonium-239, neptunium-237 or americium-241, are much more radioactive than U-238 – that is, the radioactivity per unit weight of the material is very high. For comparison, one gram of uranium-238 has an activity of 0.34 *micro*curies whereas one gram of plutonium-239 has an activity of 63 *milli*curies (almost 200,000 times higher) and one gram of americium-241 has an activity of 3.5 curies (over 50 times higher again than plutonium-239). As a result of their high specific activity, radionuclides like plutonium-239 and americium-241 cause a great deal of damage to the body long before an amount of material sufficient to cause heavy metal toxicity has accumulated. By contrast, the specific activity of uranium-238 (radioactivity per unit weight) is low enough that an amount sufficient to cause heavy metal damage can accumulate before the radiation damage overwhelms the cells or organs in question. The balance and interactions between these two types of damage depends on a number of factors, including the solubility of uranium, the organ in question, and the type of adverse health effect under consideration.

Current federal regulations limit the concentration of uranium in drinking water to 30 micrograms per liter based primarily on its chemical toxicity.[163] For natural uranium, this limit translates into 20 picocuries per liter (pCi/L) of radioactivity from uranium. For depleted uranium, the drinking water limit translates into 12 pCi/L of uranium activity. Exposure to uranium in water is regulated for chemical toxicity largely because of uranium's nephrotoxicity (i.e., its chemical toxicity to the kidneys). Despite the importance of this effect, there remain important uncertainties concerning the sensitivity of the human kidney to depleted uranium. This uncertainty is highlighted by the fact that animal studies have shown toxic thresholds that differ by more than an order of magnitude between experiments on rabbits (more sensitive) and rats (less sensitive).[164]

The science surrounding uranium's effects on the body is rapidly expanding due in large part to the concerns that have arisen in the wake of the 1991 Gulf War and the 1999 NATO bombing campaign in the former Yugoslavia. This is particularly true given the gradual recognition of the many health problems that have come to be known as "Gulf War Syndrome." We discuss the emerging picture from this research further below. As an example with particular relevance to the health of children and thus to current drinking water limits, we note that recent experiments in rats implanted with DU metal fragments

[162] This chapter is adapted from Chapter II in *Costs and Risks of Management and Disposal of Depleted Uranium from the National Enrichment Facility Proposed to be Built in Lea County New Mexico by LES*. (Makhijani and Smith 2005 p. 8 to 19)
[163] EPA 2006
[164] Royal Society Part II 2002 p. 15

tentatively concluded that there were effects on the brain that could contribute to "neurological defects" at levels of uranium exposure below those found to cause discernable damage to the kidneys. However, the study recommended further research in view of the lack of similar effects from embedded lead fragments.[165]

In addition to the chemical toxicity of uranium, its radioactivity also creates its own risks. We have already discussed some of these risks at some length in the previous chapters. In addition to discussing the potential impacts of uranium that may have disproportionate impacts on children and the embryo/fetus, the further aim of this chapter is to illustrate the dual risk of uranium's chemical and radiological toxicity, which throws some light on possible synergistic effects between heavy metal poisoning due to uranium and the effects of its radioactive decay in the same location. This may hold some lessons for understanding the interaction between non-radioactive heavy metal poisoning, for instancewith lead, and exposure to radioactivity.

Uranium is generally considered to be a bone seeker in terms of its radiological properties. Its chemical toxicity is generally considered to be most important for the kidneys. However, studies in animals have shown that uranium can concentrate in the liver, testes, and brain,[166] in addition to the skeleton and kidneys. In addition, rats implanted with DU pellets have also been found to have uranium concentrating in the heart, lung, ovaries, and lymph nodes among other tissues.[167] This body of research indicates that exposure to uranium may be mutagenic, cytotoxic, tumorigenic, teratogenic, and neurotoxic, including in a manner analogous to exposure to lead.

The potential synergisms between the chemical and radiological properties of uranium have been explored most closely with respect to its mutagenic and tumorigenic effects. In addition, the growing body of research on uranium's potential neurotoxic effects as well as its effects on skeletal development and reproductive success have raised further questions regarding the adequacy of regulating uranium exposure based on its toxicity to the kidney. In this brief case study we discuss some of these aspects of depleted uranium's potential health effects. We refer the reader to the cited publications for further information.

Before examining these health effects, it is important to note that uranium at all enrichments[168] can be expected to have about the same kinds of effects per unit of radioactivity (when it concerns radiogenic effects) and per unit mass (when it concerns chemical toxicity). When these two effects act together, the effects can generally be expected to be more pronounced as the enrichment of uranium increases, since the total radioactivity of a given mass of uranium increases with its enrichment level. Much of this chapter deals with depleted uranium not because it is more dangerous than either natural or enriched uranium (the contrary is true), but because its use has affected large populations and because the scientific research in recent years has focused on it due to concerns arising over the Gulf War Syndrome. We also have reviewed some research that explored the effect of increasing enrichment on the health effects produced by uranium. It should be noted that much of laboratory research has been conducted at

[165] Pellmar et al. 1999 pp. 790-791
[166] WHO 2001 pp. 65-66
[167] Arfsten, Still, and Ritchie 2001 p. 182
[168] "Enriched" uranium is has a proportion of U-235 higher than natural uranium and the opposite is true for "depleted uranium." Natural uranium has about 0.711 percent U-235, which is the fissile isotope that powers reactors and bombs. The other isotope is U-238 (99.284 percent in natural uranium) and U-234, 0.005 percent in natural uranium. U-234 becomes enriched (higher than 0.005 percent) in enriched uranium. Even though it is present in trace quantities, it dominates the radioactivity of enriched uranium due to its high specific activity. U-238 constitutes most of the radioactivity in depleted uranium. U-235 never constitutes more than 5% of the radioactivity of the mix of uranium isotopes at any enrichment. The effects are slightly different at different enrichments, mainly due to the slightly different decay properties of uranium-234 compared to uranium-238.

relatively high doses of uranium administrated to laboratory animals by injection or it has been done on cells *in vitro*. Extrapolation of this research to human beings, especially at low levels of exposure, cannot be done with any confidence at present for a variety of health effects. The results of this research for human beings are, generally, indicative and provide a reason for caution in exposing people, rather than definitive in the sense of providing reliable health risk estimates.

Section 8.1–Mutagenic and Tumorigenic Effects, the Potential for Synergisms

Depleted uranium is a radioactive material and ionizing radiation is an accepted causative risk factor for many forms of cancer, such as lung cancer, bone cancer, leukemia, and breast cancer. Its effect on the respiratory system and the bone is generally greater than for other organs in terms of cancer risk. In addition, uranium is a heavy metal and many heavy metals (such as nickel) are also known to be carcinogenic in the body due to their ability to cause oxidative damage to the DNA. Some recent research has provided indications that there may be a synergistic effect between the heavy metal aspect of exposure to uranium and its radioactive effects when it comes to the risk of developing cancer. A significant amount of this work is currently being conducted at the Armed Forces Radiobiology Research Institute (AFRRI) under the direction of Dr. Alexandra Miller.

This possibility of synergistic effects for uranium is reinforced by research relating to exposure to non-radioactive toxic metals and external radiation. For example, exposure to cadmium has reportedly indicated a potential synergistic response when exposures were combined with gamma radiation.[169]

Since the late 1990s there has been a growing body of evidence from *in vitro* and *in vivo* studies that indicates that depleted uranium may, in fact, be genotoxic, mutagenic, and carcinogenic.[170] Although they were not able to conclusively identify the biochemical mechanism involved, in 1998 Miller *et al.* demonstrated for the first time that internalized depleted uranium could "result in a significant enhancement of urinary mutagenicity," a common "biomarker of exposure to genotoxic agents."[171] That same year, Miller *et al.* demonstrated for the first time that exposure to DU can transform human cells into the tumorgenic phenotype, and that these transformed cells are capable of producing cancerous tumors in immuno-suppressed mice.[172] Building on this work, in 2000 Miller *et al.* again demonstrated that DU could transform human cells into the tumorigenic phenotype. Significantly, their work also demonstrated that "DU can induce chromosomal aberrations that are distinctly characteristic of radiation exposure suggesting that the alpha particle component of DU exposure may play a role in the transformation and genotoxic process."[173] This is an important distinction to draw given the potential for uranium to also cause genetic damage through its chemical properties as a heavy metal.

The relative role of the radiological and chemical components of the genetic damage caused by depleted uranium is a significant question given that uranium in drinking water is currently regulated with a primary focus on its chemical hazard and thus with an implicit assumption that its radiation hazard can generally be treated as a secondary concern in the environment. In a trio of papers in 2002, Miller *et al.* were able to further clarify the roles of DU's chemical and radiological properties and how they relate to the observed genetic damage. In the first paper, Miller *et al.* reported finding that DU caused a "small but significant increase" in the frequency of dicentric chromosomal aberrations which was not observed in the

[169] Miller et al. 2002b p. 277
[170] Arfsten, Still, and Ritchie 2001 p. 180
[171] Miller et al. 1998 pp. 646-647
[172] Miller et al. 1998b pp. 465, 468-469
[173] Miller et al. 2000 p. 210

case of exposure to non-radioactive toxic heavy metals. The formation of this type of chromosomal defect is known to be correlated with low-dose radiation damage from other types of experiments. [174]

A finding that was especially relevant to considerations of the potential joint metal toxicity and radiation effects of uranium came from experiments with uranium of different isotopic compositions. In this work, the authors found "a specific activity dependent increase in neoplastic transformation frequency" which further suggested "that radiation can play a role in DU-induced biological effects in vitro."[175] Since the amount of radiation dose increases as one goes from depleted to natural to enriched uranium for a given amount of metal, this result suggests that radiation increases the damage caused by the heavy metal aspect of uranium. This raises questions about whether radiation can also increase the heavy metal damage caused by non-radioactive heavy metals, like lead, and *vice versa*.

In discussing these results Miller *et al.* recognized the significant uncertainties that surround this work, but they also highlighted some its more important potential consequences. They noted that:

> Although the data indicate that radiation is involved in DU effects *in vitro*, several questions remain unanswered. The extent to which radiation contributes to the effects exerted by DU is not known nor its mechanism(s) understood. Furthermore, one can only speculate as to whether the radiation- and chemical-effects are synergistic. Limited studies have shown that a non-radioactive metal like cadmium combined with gamma radiation can result in a synergistic response *in vivo*. It is intriguing to ask whether radiation actually play[s] a significant role in DU cellular effects perhaps through nontargeted effects of radiation exposure? Several recent radiation studies have demonstrated the important role that bystander effects have in cellular radiation response by causing damage in unirradiated neighboring cells. In the case of DU, cells not traversed by an alpha particle may be vulnerable to radiation-induced effects as well as chemically-induced effects. [176]

In summary, they concluded that:

> Considering that conventional understanding of potential DU health effects assumes that chemical effects are of greatest concern, these results and similar future results could have a significant impact on DU risk assessments. [177]

The final 2002 paper from Miller *et al.* found that DU was also capable of inducing "oxidative DNA damage in the absence of significant radioactive decay."[178] In light of their other work showing the potential for the radiological aspect of DU to contribute to genotoxic effects *in vitro*, they note that "it is tempting to speculate that DU might exhibit both a tumor 'initiation' and 'promotion' component." [179] This potential dual role could result from the alpha particle radiation causing the cancerous mutation (tumor initiation) followed by a build up of oxidative damage aiding the spread of the cancer (tumor promotion).

A final example of the work being conducted at AFRRI on these issues comes from a 2003 Miller *et al.* publication concerning the potential ability of DU to induce genomic instability in human cells. In this work the authors initially note that:

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[174] Miller et al. 2002 pp. 121-122
[175] Miller et al. 2002b p. 275
[176] Miller et al. 2002b p. 277
[177] Miller et al. 2002b p. 277
[178] Miller et al. 2002c p. 251
[179] Miller et al. 2002c p. 251

> Studies with DU in our laboratory demonstrated neoplastic transformation of human cells under conditions where approximately 14% of the DU-exposed cells were transformed even though less than 5% were traversed by an alpha particle. These findings suggest that factors other than direct or "targeted" damage to the DNA may be involved in the transformations. Chemical effects of DU and "non-targeted" effects of radiation may also play a role. Non-targeted effects can result in damage in cells not traversed by an alpha particle. The overall level of transformation observed may result from contributions by any or all of these factors.[180]

In order to gauge the impact of radiation and heavy metal toxicity separately, the effects of depleted uranium were compared with those of nickel (Ni) and to gamma irradiation. From the results of their experiments, Miller *et al.* concluded that

> In summary, we have presented data showing the production of genomic instability in the progeny of human cells exposed to DU. The findings demonstrate that DU can induce delayed cell death and genetic alterations in the form of micronuclei. Compared to gamma radiation or Ni, DU exposure resulted in a greater manifestation of genomic instability. Although animal studies are needed to address the effect of protracted DU exposure and genomic instability in vivo, results obtained from our in vitro system can play a significant role in determining risk estimates of DU exposure.[181]

While the uncertainties remain significant, the growing body of evidence that is emerging from Miller *et al.*'s laboratory at the Armed Forces Radiobiology Research Institute and from other researchers cannot be ignored. The conclusions from this research are likely to play an important role in shaping future risk assessments of not just uranium but of other joint exposures to heavy metals.

Section 8.2 – Effects of Uranium on Reproduction

In addition to providing an important case study for the potential synergisms between chemical and radiological toxins, uranium also provides a valuable case study for considering non-cancer risks. In this section, we consider the impact of uranium exposure on reproductive success. In the next two sections we turn to considerations of impacts on skeletal development and finally to potential neurotoxic effects.

It is important to note that the non-cancer effects discussed below are indicated by laboratory research, which is often done at elevated levels of exposure. These effects have not been definitively established for human beings in terms of quantitative health risks. Also, some of the experiments we cite were conducted with uranium directly injected into animals or with depleted uranium in metallic form embedded under the skin to simulate injuries from DU weapons, which are pathways significantly different from what would be expected from environmental exposures. Finally, it has not been established whether some of these non-cancer effects have thresholds, in contrast to the well accepted no-threshold hypothesis for cancer risk from ionizing radiation (see Chapter 2).

Investigations of the reproductive effects of uranium exposure were reported as far back as the 1940's, but, these early studies do not appear to have been systematically followed up on by other researchers until many decades later.[182] Even today, there are substantial gaps in our understanding of uranium's effects on human and animal reproduction. In some of the early experiments, it was found that not only continuous feeding, but also that just a single one-time feeding of uranium to rats could detrimentally

[180] Miller et al. 2003 p. 248
[181] Miller et al. 2003 p. 257
[182] The reported experiments were apparently carried out during the Second World War as part of the Manhattan Project although they were not publicly reported until the late 1940s and early 1950s. (Voegtlin and Hodge 1953 p. vi)

affect the animal's reproductive success.[183] The authors concluded that "under the circumstances of this experiment, uranium administration adversely affected the reproductive functions in the absence of a severe derangement of nutrition."[184]

Why these provocative early studies do not appear to have been carried forward or more widely reported is not yet clear. However, the work that has been carried out quite recently on uranium has expanded these early findings, and has resulted in the identification of two distinct areas of concern in regard to the potential impact of uranium on reproductive health. The first area relates to the risks associated with exposures to men whereas the second relates to exposures of women. In regard to the possible effects on men, uranium is found to concentrate in the testes and has been found in the sperm of Gulf War veterans at elevated levels. The U.K. Royal Society's report on veterans concluded that this "raise[d] the possibility of adverse effects on the sperm from either the alpha-particles emanating from DU, chemical effects of uranium on the genetic material or the chemical toxicity of uranium."[185] There may be possible synergistic effects between uranium's ability to damage the sperm's DNA via both chemical oxidative stress and ionizing alpha radiation.[186] In addition, the World Health Organization has noted the observation of "unspecified degenerative changes in the testes" of rats as a result of chronic ingestion of soluble uranium compounds.[187]

Although still very limited, somewhat more work has been done on the reproductive effects of uranium exposure on females. Uranium has been shown to cross the placental barrier and concentrate in fetal tissue.[188] Experiments with animals have demonstrated that exposure to uranium either through ingestion or injection can cause "[d]ecreased fertility, embryo/fetal toxicity including teratogenicity, and reduced growth of the offspring."[189] These findings have been demonstrated in both rats and mice, and provide evidence (at least at the levels of intake examined in these studies) that uranium exposure can adversely affect the reproductive success of females.[190] The one reported experiment to use depleted uranium did not find statistically significant effects on "maternal weight gain, food and water intake, time-to-pregnancy, or the percentage of litters carried to term." However, the researchers did find that higher numbers of DU pellets implanted in the female led to increased concentrations of uranium in the placenta and whole fetus.[191]

While there are still many unknowns as to what the effects of uranium on the reproductive system are, a number of potential mechanisms in addition to the overall radiosensitivity of the embryo/fetus have been proposed to help explain the observed effects. These proposed mechanisms included hormonal or enzymatic disruption.[192]

The potential for uranium to affect the hormonal systems is suggested by research on exposures to lead which shares chemical similarities with uranium in the body.[193] Recent research has shown that both "prenatal and postnatal exposure to lead is associated with growth restriction in laboratory animals and humans" and that exposure to lead can also alter sex hormone production and delay puberty in rats.[194] An

[183] Voegtlin and Hodge 1953 pp. 1255-1256
[184] Voegtlin and Hodge 1953 p. 1258
[185] Royal Society Part II 2002 p. 14
[186] Arfsten, Still, and Ritchie 2001 p. 180 and Domingo 2001 p. 606
[187] WHO 2001 p. 71
[188] Albina et al. 2003 p. 1072 and Royal Society Part II 2002 pp. 14-15
[189] Domingo 2001 p. 603
[190] Albina et al. 2003 pp. 1075-1076, Craft et al. 2004 p. 309, and WHO 2001 p. 71
[191] Arfsten, Still, and Ritchie 2001 p. 185 and Domingo 2001 p. 607
[192] Arfsten, Still, and Ritchie 2001 p. 189 and Domingo 2001 p. 606
[193] Lemercier et al. 2003 p. 243
[194] Selevan et al. 2003 p. 1528

epidemiological study published in 2003 found that even relatively low average levels of lead caused a measurable delay in puberty in African-American and Mexican-American girls, whereas no statistically significant delay in non-Hispanic White girls was found.[195] The observed effect on the girls' sexual development was tentatively attributed, at least in part, to potential "alterations in endocrine function."[196] Many questions as to how lead caused the observed delay and whether or not the children had been exposed to higher levels in the past before the study's screening began remain unanswered. Nonetheless the potential for uranium to play an analogous role in affecting hormonally mediated processes in developing children could add further to its list of health concerns and could also add significant new avenues for potential synergisms with its other chemical and radiological heath effects. This research also raises the question of the combined effects of exposure to uranium and hormonally active compounds. This is an area requiring further study.

Section 8.3 – Effects of Uranium on Skeletal Development

The ICRP notes that many elements of the fetal skeleton "show a complex and thus radiosensitive genesis" and that the other periods when the bones are undergoing rapid development (i.e., in early childhood and during puberty) are also times of heightened sensitivity to the impacts of radiation.[197] In experiments on rats, it has been demonstrated that both acute and chronic intakes of uranium can cause damage to bones. As a result, the Royal Society has stated that, in light of the fact that uranium crosses the placental barrier, "the effects of maternal exposure to DU on skeletal development in the foetus may also need to be considered."[198]

In addition, the World Health Organization and the National Research Council have both recommended studies to determine what effect, if any, uranium integrated into the bone has on the bone marrow, and thus on the production of new blood cells. This research may be of particular importance given the findings from a study in 2004. In this work, the researchers exposed beagle dogs to daily doses of uranyl nitrate from a young age and found "that uranium accumulated in the marrow as much as in the bone, contrary to the results obtained with single, acute doses."[199] If the bone marrow of children concentrates uranium, this would raise concerns over the potential for uranium to contribute to an increased risk of developing leukemia. It also raises concerns about damage to the immune system, which in turn may contribute to a variety of adverse health outcomes. This is because bone marrow-derived stem cells are the initial source of the various types of cells that constitute the immune system.

Section 8.4 – Effects of Uranium on the Brain

Limited evidence raising the possibility of a link between uranium and neurological damage dates back to at least the mid-1980's.[200] These studies, however, have a number of problems that hampered their usefulness in drawing any solid inferences regarding the neurological risks of uranium.[201] One of the major concerns regarding the potential toxic effects of depleted uranium on the brain centers around the fact that uranium's primary chemical form in the body is as the uranyl cation (UO_2^{2+}) which is a toxic

[195] Selevan et al. 2003 p. 1527. The term "non-Hispanic White" is as used by the authors.
[196] Selevan et al. 2003 p. 1535
[197] ICRP 90 p. 30 and 149
[198] Royal Society Part II 2002 p. 67
[199] Arruda-Neto et al. 2004
[200] Pellmar et al. 1999 p. 785 and Craft et al. 2004 p. 307
[201] Fulco, Liverman, and Sox 2000 p. 151 and 153

heavy metal chemically analogous to the lead cation (Pb^{2+}).[202] The tragic history of lead as a neurotoxin is well documented and the potential neurotoxicity of uranium is therefore a particular concern in relation to children's health.

In 1999, Pellmar *et al.* at the Armed Forces Radiobiology Research Institute showed that depleted uranium implanted in rats concentrated in various regions of the brain. In addition, the authors found an increasing concentration of uranium in the brain with increasing exposure. From these results they concluded that "[t]he accumulations in brain, lymph nodes, and testicles suggest the potential for unanticipated physiological consequences of exposure to uranium through this route."[203]

In additional research, Pellmar *et al.* were able to further show that the "exposure to DU fragments caused neurophysiological changes in the hippocampus."[204] The hippocampus was chosen for analysis because it is "a region of the brain involved with memory and learning." Reviews of these AFRRI experiments have concluded that these results provide important evidence of the potential for depleted uranium to display neurotoxic properties.[205]

In addition to the work of Pellmar *et al.*, in 1998 Ozmen and Yurekli showed that following ingestion, uranium concentrated to a large degree in the brains of mice, while in 2003 Lemercier *et al.* demonstrated "that a significant amount of uranium" also concentrated in the brains of rats.[206] Lemercier *et al.* were also able to identify that the uranium in the brain was predominately in the form of uranyl tricarbonate.[207] Finally, in 2005, Briner and Murray found observable behavioral changes in rats after two weeks of exposure to depleted uranium in drinking water.[208]

In addition to tests on animals, specialized "computerized tests designed to assess performance efficiency" have been used to look for potential neurological effects in veterans who were exposed to depleted uranium munitions during the Gulf War.[209] These tests, conducted at the Baltimore Veterans Administration Medical Center, observed a statistically significant correlation between uranium concentration in the veterans' urine and poor performance on the computerized neurocognitive tests.[210] However, no measurable effects were found in this same group using traditional neurocognitive tests.[211] It is important to note, however, that the soldiers were exposed as adults, and that these tests cannot, therefore, provide information on the impacts of exposure during the more sensitive stages of fetal development and early childhood when the brain is undergoing rapid growth.

Finally, we note that radiation is also known to adversely affect the nervous system of the embryo/fetus. From a review of the atomic bomb survivors, the International Commission on Radiological Protection has concluded that:

> There is a clear constellation of effects of prenatal irradiation on the developing central nervous system – mental retardation, decreased intelligence scores and school performance, and seizure disorders.[212]

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[202] Lemercier et al. 2003 p. 243 and Domingo 2001 p. 603
[203] Pellmar et al. 1999b p. 29
[204] Pellmar et al. 1999 p. 790. See also Lemercier et al. 2003 p. 243 and 245
[205] McClain et al. 2001 p. 117-118 and Craft et al. 2004 p. 307-308
[206] Ozmen and Yurekli 1998 p. 111 and Lemercier et al. 2003 p. 245
[207] Lemercier et al. 2003 p. 245
[208] Briner and Murray 2005
[209] Arfsten, Still, and Ritchie 2001 p. 185
[210] Pellmar et al. 1999 p. 791, Royal Society Part II 2002 p. 13, and Craft et al. 2004 p. 307
[211] Craft et al. 2004 p. 307
[212] ICRP 90 p. 118

The ICRP elaborates further on why the prenatal period is of particular concern for radiation damage to the nervous system and why it is so important to consider in assessing risks. The Commission notes that:

> Development of the central nervous system starts during the first weeks of embryonic development and continues through the early postnatal period. Thus development of the central nervous system occurs over a very long period, during which it is especially vulnerable. It has been found that the development of this system is very frequently disturbed by ionising radiation, so special emphasis has to be given to these biological processes.[213]

As with a number of other emerging risks from uranium, there is thus the potential for synergisms between uranium's chemical and radiological effects in relation to its effects on the nervous system that need to be further investigated. Moreover, it is important to note in this context that the radiation dose model adopted by the ICRP for the first eight weeks of pregnancy is not suitable for alpha-emitting radionuclides. The ICRP assumes that the dose to the embryo/fetus in this period is the same as that to the maternal uterine wall.[214] This model is not really relevant to alpha-emitting radionuclides, since alpha particles deposit their energy in a very short range. Uterine dose from such particles may have little or no relation to the dose to the embryo/fetus.

The ICRP's description of the development of the embryo/fetus illustrates the importance of this early period in possible neurological damage as the fetal organs form, develop, and grow:

> Although the morphological appearance of the embryo during the first 3 weeks of development after conception does not seem very structured, the pattern of the basic body plan is already established during this time. Thus, the dorsal ectodermal cells proliferate and differentiate to form the neural plate, which develops into the neural tube, which comprises the nervous system.[215]

Further work on the dose to the embryo/fetus due to alpha-emitters, and especially uranium, is needed to develop a quantitative understanding of the adverse health outcomes, including damage to the neurological system and brain. This conclusion complements the one we arrived at in Chapter 7 in regard to tritium, the dosimetric characteristics of which, during the first eight weeks of pregnancy, are not well characterized by the ICRP's present model, which equates dose to the maternal uterine wall to the dose to the embryo/fetus in the first eight weeks.

Further, it is clear, in light of the existing body of work, some of which is discussed above, that uranium's potential neurotoxicity might be better understood if uranium were considered to be analogous to a kind of radioactive lead, in which the damage from the alpha radiation occurs in conjunction with heavy metal induced damage to produce a variety of health problems at relatively low levels of exposure. This analogy between uranium and lead was made in 2003 by Lemercier *et al.* in reporting their study demonstrating the concentration of uranium in the brains of rats.[216]

Comparing lead to uranium has obvious limitations in regards to understanding the detailed biological mechanisms involved in the damage caused by uranium. But the similar ability of uranium to chemically induce oxidative stress and to cross the blood brain barrier, combined with the high levels of local cellular damage caused by alpha radiation, raises significant warning signs about the potential impact of this material on a child's developing brain.

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[213] ICRP 90 p. 9
[214] ICRP 88 p. 20
[215] ICRP 88 p. 33
[216] Lemercier et al. 2003 p. 243

In light of the analogy of uranium to lead, it should be noted that despite evidence of lead's damaging effect on the brain, dating back more than two millennia, and lead poisoning being first recognized in children as early as the 1890s, action to protect health was slow. Leaded gasoline was introduced in the 1920's despite this history; it was not until 1995 that it was finally taken off the U.S. market.[217] As with the general trend in radiation protection standards, the Centers for Disease Control and Prevention (CDC) has chosen to lower the guideline level it considers to be an indicator of "elevated" levels of lead in the blood of children four times since the late 1960s. The level today is one-sixth of where is stood 35 years ago.[218] In addition, the CDC has adopted the position that there is no safe level of exposure to lead and that any intake will result in some level of harm. Unfortunately, despite significant reductions in exposure since the late-1970s, the current levels of lead in children's blood are still roughly 100 to 1000 times larger than the estimated pre-industrial levels. For the 1999-2002 period, the CDC estimated that nearly 1.6 percent of children in the U.S. still exceeded their guideline for elevated levels of lead in the blood.[219]

Section 8.5 – The Future of Uranium Health Effects Research

There is clearly much that is still not understood about the array of potential deleterious effects that chronic or acute exposures to uranium, including depleted uranium, may cause. For example, a 2003 study by the National Research Council concluded that, "[s]urprisingly there are still substantive gaps in knowledge of the non-radiological health impacts of exposure to uranium and its compounds."[220] As summed up in a recent review by Craft *et al.,* from Duke University:

> Although most of the DU absorbed in the body is metabolized and excreted, enough is distributed throughout the body to raise important toxicological concerns… The long-term effects of DU still have to be definitely resolved, and there is an obvious need for continued studies.[221]

In its 2001 review of depleted uranium, focusing in particular on the impact of military munitions, the World Health Organization concluded that there is inadequate information available concerning the potential impact of uranium in the following areas, and that additional research needs to be undertaken:

> - Neurotoxicity: Other heavy metals, e.g. lead and mercury are known neurotoxins, but only a few inconsistent studies have been conducted on uranium. Focused studies are needed to determine if DU is neurotoxic.
>
> - Reproductive and developmental effects have been reported in single animal studies but no studies have been conducted to determine if they can be confirmed or that they occur in humans.
>
> - Haematological effects: Studies are needed to determine if uptake of DU into the bone has consequences for the bone marrow or blood forming cells.
>
> - Genotoxicity: Some *in vitro* studies suggest genotoxic effects occur via the binding of uranium compounds to DNA. This and other mechanisms causing possible genotoxicity should be further investigated.[222]

[217] Koller et al. 2004 p. 987, Kovarik 2003, and EPA 1995
[218] Rogan and Ware 2003 p. 1515 and Canfield et al. 2003 p. 1518
[219] Koller et al. 2004 p. 993 and CDC 2005
[220] NAS/NRC 2003 p. 63
[221] Craft et al. 2004 p. 315
[222] WHO 2001 pp. 148-149

A 2003 National Research Council report concerning management of the Department of Energy's depleted uranium stockpile formally adopted all four of the WHO recommendations for research.[223] This decision by the NRC strengthened the 2000 recommendation from the Institute of Medicine's Committee on Health Effects Associated with Exposures During the Gulf War that additional animal studies should be conducted to investigate the biological effects of depleted uranium with a particular focus on "studies of cognitive function, neurophysiological responses, brain DU concentrations, and the transport kinetics of DU."[224] In addition, the U.K. Royal Society has also endorsed further research on many areas of DU's effects in the body, including studies concerning its potential neurocognitive and reproductive health effects.[225]

As a final example of importance in the current context of seeking to reorient the regulatory regime to the protection of those most at risk, we note that in 2001 the World Health Organization concluded that:

> Children are not small adults and their exposure may differ from an adult in many ways. Unfortunately, despite their obvious importance little definitive data exists concerning how their uranium exposure differs from that of adults.[226]

In this same vein, a review of depleted uranium health effects by the U.K. Royal Society in 2002 noted that "[a]nimal studies suggest that absorption of uranium from the gut of neonates might be higher than in older children or adults."[227] The World Health Organization, the National Research Council, and the Royal Society have all recognized the need for additional studies to better assess the impacts of uranium exposure on children.[228]

The lessons of lead's tragic history in relation to children's health, including the decades long denial of ever growing evidence of the risks by industries producing lead-based products, as well as the systematic and progressive tightening of health guidelines specifically targeting children once they were finally introduced should be closely examined in relation to the direction in which uranium research is now unfolding. The research summarized in this chapter highlights the strong indications that have already been revealed pointing to the need for tighter interim standards on uranium exposure if pregnant women (including fetal exposures) and children are regarded as the basis for setting standards.

[223] NAS/NRC 2003 pp. 67-68
[224] Fulco, Liverman, and Sox 2000 p. 327
[225] Royal Society Part II 2002 pp. 66-68
[226] WHO 2001 p. 30
[227] Royal Society Part II 2002 p. 17
[228] WHO 2001 p. 148, NAS/NRC 2003 p. 68, and Royal Society Part II 2002 pp. 17, 24

Attachment 4

Dr. Michael C. Thorne's Vita

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Michael Charles Thorne

Qualifications PhD FSRP
Year of birth 1950
Nationality British

PROFESSIONAL ACTIVITIES AND MEMBERSHIP

Visiting Fellow at the Climatic Research Unit, School of Environmental Sciences, University of East Anglia
Fellow of the Society for Radiological Protection and a Past President of the Society
Member of the Editorial Board of the Journal of Radiological Protection
Member of the National Dose Assessment Working Group (NDAWG) and Chairman of the Habits Subgroup
Member of the Eco-ethics International Union
Consultant to the Institute for Energy and Environmental Research, Washington DC.
Quintessa Associate
Director, Mike Thorne and Associates Limited

ACADEMIC RESPONSIBILITIES

Formal supervision of two PhD students at the University of East Anglia:
P Burgess, Future Climatic and Cryospheric Change on Millennial Timescales: An Assessment using Two-dimensional Climate Modelling Studies, PhD awarded 1998.
M Hoar, Reconstructing Climate Gradients across Europe for the Last Glacial-interglacial Cycle, PhD awarded 2004.
Informal supervision of PhD students at the University of Edinburgh (development and retreat of ice sheets) and at Imperial College of Science, Technology and Medicine (radionuclide transport in vegetated soil columns – experimental studies and modelling interpretations).
Teaching on the MSc course on Environmental Radioactivity at the University of Surrey.
Teaching on the MSc course in Environmental Technology at Imperial College of Science, Technology and Medicine.
Supervision of Post-doctoral research activities at the Universities of East Anglia; University of Newcastle and Imperial College of Science, Technology and Medicine on behalf of various commercial clients.

CAREER HISTORY (Selection of Projects)

Mike Thorne and Associates Limited, 2001 onward

Development of Proposals for Setting Radiation Protection Standards based on Consideration of More Sensitive Individuals in a Population
Client – Institute for Energy and Environmental Research, Washington DC

Overall project review and development of techniques for calculating radiation doses to the early embryo from internally incorporated radionuclides.

Review of Impacts of Coastal Erosion at Hunterston
Client – ERM Limited

Evaluation of the potential radiological implications of coastal erosion on the VLLW pits at Hunterston Nuclear Power Station.

Advice on Dose Reconstruction
Client – S A Cohen & Associates for NIOSH

Advice on dose reconstructions for workers at DOE facilities from 1941 onward.

Advice on Effects of Radionuclides on Organisms other than Man
Client – Nuclear Safety Solutions Limited, Canada

Provision of guidance on dosimetry, reference levels and effects relevant to selected protected species.

Participation in Safety Assessment Studies for the Baita Bihor Repository, Romania
Client – Quintessa/for the European Union

Compilation of inventory data, shielding studies and development of both operational and post-closure safety cases.

Review of the Yucca Mountain Project
Client – State of Nevada

Co-ordination of technical activities involved in a review of the proposed License Application by US DOE for disposal of radioactive wastes at Yucca Mountain.

Co-ordination of biosphere research and participation in BIOCLIM
Client – UK Nirex Ltd

Co-ordination of research on climate change, ice-sheet development, near-surface hydrology and radionuclide transport, as well as participation in an international programme on the implications of climate change for radioactive waste disposal. Also includes development of new models for radionuclide transport in the biosphere and for the gas pathway.

Development of a Handbook on Radionuclide Behaviour in the Environment
Client – Serco Assurance

Development of a handbook for Environment Agency staff outlining the behaviour of a wide variety of radionuclides in terrestrial and aquatic environments.

Development of a Simplified Dose Assessment Model
Client – Serco Assurance

Development of a simplified spreadsheet-based dose assessment tool for use by Environment Agency staff in determining Authorisations.

Provision of Biosphere Advice
Client – Ciemat, Spain

Provision of advice on models and data relevant to geological disposal of radioactive wastes

Provision of Advice on Safety
Client – NNC Ltd/Defra

Provision of expert advice to the UK Committee on Radioactive Waste Management (CoRWM).

Effects of Radiation on Organisms Other Than Man
Client – AEA Technology/Serco Assurance

Study for ANDRA to identify appropriate indicator organisms and develop appropriate dosimetry and effects models for those organisms.

Member of the Site Investigation Expert Review Group (SIERG)
Client – SKB

Oversight reviews of site investigation activities and the associated research and assessment programmes.

Advice on the Short-, Medium- and Long-term Effects of Climate Change on Nuclear Licensed Sites
Client – BNFL and Nexia Solutions

Interpretation of results from the international BIOCLIM project in relation to decommissioning and solid radioactive waste management, with particular emphasis on the potential significance of sea-level changes. Review of information on coastal vulnerabilities at NDA sites.

Advice on Submarine Reactor Accidents and the Development of Detailed Emergency Planning Zones
Client – Electrowatt-Ekono

Assistance to MoD in revising emergency planning criteria in the light of recent changes of views on Emergency Reference Levels and other technical developments.

Review of Continuing Operational Safety Cases
Client – Electrowatt-Ekono

Review of COSRs developed by BNFL for contaminated land.

Development of a New Soil-Plant Model for use in Radiological Assessments
Client – Food Standards Agency/Quintessa

Development of the specification for a new soil-plant model (PRISM) to replace that implemented in the SPADE suite of codes (implementation of the model has been by Quintessa) and extension of that work to new models for ^{3}H and ^{14}C.

Review of Probabilistic Safety Assessment and Criticality Issues relating to a Proposed Surface Storage Facility for Spent Nuclear Fuel
Client – State of Utah

Review of the potential for criticality in breached storage casks and of the probability of breaching by aircraft impacts. Also, supervision of various criticality and radiation shielding calculations.

Development of Models for Radionuclide Transfers to Sewage Sludge and for Evaluating the Radiological Impact of Sludge applied to Agricultural Land
Client – Food Standards Agency

Includes a review of literature and the development and implementation of probabilistic models for such transfers.

Development of Biokinetic Models for Radionuclides in Animals
Client – Serco Assurance

Development of updated biokinetic models for use by the Food Standards Agency in their SPADE and PRISM modelling systems.

Review Studies for the Proposed Australian National Radioactive Waste Repository
Client – RWE NUKEM

Reviews of reports on animal transfer factors and of the potential effects of climate change on the repository plus development of a model for the biokinetics of the ^{226}Ra decay chain in grazing animals.

Development and Application of a Model for Assessing the Radiological Impacts of ^{3}H and ^{14}C in Sewage Sludge
Client – NNC Ltd

Development of a model based on physical, chemical and biochemical principles for the uptake of ^{3}H and ^{14}C into sewage sludge and their subsequent distribution and transport after application of the sludge to agricultural land.

Support for development of the Drigg Post-closure Radiological Safety Assessment
Client - BNFL

Support in the areas of FEP analysis, biosphere characterisation, human intrusion assessment and the effects of natural disruptive events. In addition, provision of advice of future research initiatives that should be pursued by BNFL.

Review of Parameter Values
Client – AEA Technology/Serco Assurance

Review of biosphere parameter values for use in the ANDRA assessment model AQUABIOS.

Development of a Database related to Emergency Planning
Client – AEA Technology (Rail)

Identification of relevant international, overseas and national legislation, regulations and guidance, and production of brief summaries of the documents.

Dose Reconstruction for Workers on a Uranium Plant
Client - McMurry and Talbot

Dose reconstruction for the plaintiffs in a case relating to the Paducah Gaseous Diffusion Plant.

Dose Reconstruction for a Worker Exposed to Pu and Am
Client – Pattinson and Brewer

Dose reconstruction for a worker exposed by a puncture wound in the finger while working at a glove box.

AEA Technology, 1998-2001

Revision of Exemption Orders Made Under the Radioactive Substances Act
Client – DETR

Review of requirements for revision and preparation of a draft text for the purposes of consultation.

Assessment of Remediation Options for Uranium Liabilities in Eastern Europe
Client - European Commission

Studies of remediation requirements relating to mines, waste heaps and hydrometallurgical plant in Bulgaria, Slovakia and Albania.

Evaluation of Unusual Pathways for Radionuclide Transport from Nuclear Installations
Client – Environment Agency

Review of literature and conduct of formal elicitation meetings to determine potential pathways and evaluate their radiological significance.

Support Studies on the Drigg Post-closure Performance Assessment
Client - BNFL

Support in the areas of FEP analysis, biosphere characterisation, human intrusion assessment and the effects of natural disruptive events. In addition, provision of advice of future research initiatives that should be pursued by BNFL.

Development of Models for the Biokinetics of H-3, C-14 and S-35 in Farm Animals
Client - FSA

Review of relevant literature, development of appropriate biokinetic models and implementation in stand-alone software.

Integration of Aerial and Ground-based Monitoring in the Event of a Nuclear Accident
Client - FSA

Desk-based review and simulation study designed to determine optimum monitoring strategies for different types of accidents.

Elicitation of Parameter Values for use in Radiological Impact Assessment Models
Client - FSA

Expert elicitation study to provide distributions of parameter values for use in the suite of assessment models currently used by the FSA for routine and accidental releases.

Biosphere Research Co-ordination and Assessment Studies
Client - United Kingdom Nirex Ltd

Continuation of a programme of work originally undertaken at Electrowatt Engineering (UK) Ltd

Site Investigation and Risk Assessment - Hilsea Lines
Client - Portsmouth City Council

Radiological assessment of a radium-contaminated site.

Electrowatt Engineering (UK) Ltd, 1987-1998

Development of a Siting Policy for Nuclear Installations: Harbinger Project and Follow-up Study
Client - HSE/NSD

Review of existing policy and development of alternatives as a precursor to application to a wide range of installations, not restricted to commercial reactors.

Support to the Rock Characterisation Facility Public Enquiry
Client - UK Nirex Ltd

Preparation of position papers and rebuttals of evidence.

Rongelap Resettlement Project
Client - Marshall Islands Government

Participation in an oversight committee evaluating the radiological safety of Rongelap in the context of resettlement by its evacuated community.

Evaluation of Inhalation Doses from Uranium
Client - Baron & Budd

Provision of expert witness support in a class action relating to environmental exposure from a uranium plant.

Biosphere Studies Relating to Drigg
Client - BNFL

Provision of advice on time-dependent biosphere modelling for the Drigg low-level radioactive waste disposal facility.

Radiation Doses to an Individual as a Consequence of Working on the San Onofre Nuclear Power Plant
Client - Howarth & Smith

Interpretation of personal and area monitoring data for legal purposes.

Interpretation of Uranium in Urine Data for the Fernald, Ohio Feed Materials Processing Center
Client - Institute for Energy and Environmental Research

Interpretation of urinalysis and lung counting data, and appearance as an expert witness in the associated trial.

Determination of Failure Probabilities for use in PRA
Client - Nuclear Installations Inspectorate

Development of new approaches to the use of Bayes Theorem in defining component failure probabilities for use in PRA when statistics on actual failures are limited.

Review of Inventory Information
Client - UK Nirex Ltd

Review of uncertainties in inventories of individual radionuclides.

ALARP Study of Options for the Treatment, Packaging, Transport and Disposal of Plutonium Contaminated Material
Client - UK Nirex Ltd

Use of multi-attribute utility analysis to establish which option is preferred.

Expert Judgement Estimation of Intrusion Model Parameters
Client - British Nuclear Fuels plc

Project Manager of a study assessing the risks of human intrusion into Drigg radioactive disposal site using expert judgement techniques.

Brainstorming Study of Risks Associated with Building Structures
Client - Building Research Establishment

Participation in a classification study of the health risks associated with buildings including both injuries and disease.

Radiological Consequences of Deferred Decommissioning of Hunterston A
Client - Scottish Nuclear Ltd

Project Manager of a study of the radiological impacts of groundwater transport of radionuclides, releases to atmosphere and intrusion.

Reviews of Safety Documentation
Client - UK Nirex Ltd

Review of safety related documentation for Packaging and Transport Branch.

The Sheltering Effectiveness of Buildings in Hong Kong
Client - Ove Arup & Partners

Project Manager of a study evaluating the shielding effectiveness of all types of building in Hong Kong for volume sources of photons in air and surface deposition sources.

Assessment of the Radiological Impact of Releases of Radionuclides from Premises other than Licensed Nuclear Sites
Client - Ministry of Agriculture, Fisheries and Food

Project Manager of a study to identify representative premises, obtain data on their releases of radionuclides and assess radiological impacts using a new methodology developed for the project.

Assessment of the Radiological Implications of Uranium and its Radioactive Daughters in Foodstuffs
Client - Ministry of Agriculture, Fisheries and Food

Project Manager of a review study of concentrations of uranium and its daughters in foodstuffs, taking local and regional variations in uranium concentrations in soils, sediments and waters into account.

Radionuclides in Sewage
Client - Her Majesty's Inspectorate of Pollution

Project Manager of a study including a desk review on alternative methods of disposal of sewage sludges, interpretation of monitoring data relating to radionuclide discharges from Amersham International to the public sewer system, development of a model for radionuclide transport in sewers, and collection and analysis of effluent, foul water, sediment, sludge and other samples suitable for use in model validation studies.

Accident Consequence Calculations
Client - Nuclear Installations Inspectorate

Project Manager of a study to assess the radiological consequences of various atmospheric releases using the MARC code.

Definition of Threshold Recording Levels for Drums of ILW
Client - UK Nirex Ltd

Project Manager of a study of the implications of post-closure radiological impacts of radioactive waste disposal in defining Threshold Recording Levels for radionuclides in individual waste drums.

Definition of Expert Judgment Exercises Relating to Nuclear Safety
Client - Commission of the European Communities

Project Manager for a study defining expert judgment exercises relating to conceptualisation, representation and input data specification. Included a comprehensive review of available formal expert judgment procedures, and mathematical and behavioural aggregation techniques.

Definition of Research Requirements Relating to the Use of Expert Judgment in Parameter Value Elicitation for Reactor Safety Studies in a UK Context
Client - Nuclear Safety Research Management Unit, HSE

Development of proposals for using combined behavioural and mathematical aggregation procedures in formal elicitations of expert judgment.

Development Priorities for the Drigg Technical Development Programme
Client - British Nuclear Fuels plc

Provision of detailed advice to BNFL on future design options, and research and development priorities, in relation to radioactive waste disposal at Drigg.

Channel Tunnel Safety Studies
Client - Channel Tunnel Safety Authority

Provision of advice and guidance on safety criteria appropriate to the Fixed Link, on the classes of Dangerous Goods that may properly be carried and on the overall characteristics of the proposed Safety Case.

Development of Societal Risk Criteria
Client - Marathon Oil

Interpretation of F-N curves in the context of the offshore oil/gas industry, taking risk aversion into account.

Impacts of Salt Dispersal on Plant Communities
Client - Sir William Halcrow

Evaluation of salt dispersal from a major road in winter in relation to adjacent Sites of Special Scientific Interest.

Offsite Consequence Assessments
Client - Nuclear Electric

Studies of the offsite radiological impacts of atmospheric and liquid releases of radioactive materials from Magnox stations.

Dry Run 3
Client - Her Majesty's Inspectorate of Pollution

Uncertainty and bias studies involving formal expert judgment procedures to develop a conceptual model of those factors and interrelationships which are of significance in determining the post-closure radiological impact of a deep geological repository for radioactive wastes. This project also included advice on data and models to be used for post-closure radiological assessments.

Radiological Assessments of Drigg
Client - British Nuclear Fuels plc

Project Manager for post-closure radiological impact assessments of the Drigg LLW disposal site. Also included specification and development of computer codes relating to the radiological impact of fires, releases of radioactive gases produced by microbial action and metal corrosion, and human intrusion.

Biosphere Co-ordination
Client - UK Nirex Ltd

Co-ordination of the UK Nirex Ltd Biosphere Research Programme from its inception, including requirements definition, technical management of all projects and QA surveillance as the Client's Representative.

Biosphere Support for the Nirex Disposal Safety Assessment Team
Client - AEA Technology

Development of approaches for assessing the radiological impact of releases of radionuclides to the biosphere, plus advice on radiological protection criteria, definition of individual risk, implications of conventionally toxic chemicals in wastes and a variety of other matters.

Evaluation and Radiological Assessment of Liquid Effluent Releases from Various Premises
Client - Her Majesty's Inspectorate of Pollution

Reviews of monitoring data and evaluations of radiological impact, primarily related to Harwell, Aldermaston, Capenhurst and Amersham International.

Evaluation of the Radiological Impact of Overseas Nuclear Accidents
Client - Her Majesty's Inspectorate of Pollution

Studies of the impact of potential overseas nuclear accidents on the UK, with emphasis on survey and monitoring requirements, and the selection of appropriate radiation detection equipment for monitoring.

Bilsthorpe Power Station
Client - British Coal/East Midlands Electricity

Preparation of an Environmental Statement with emphasis on atmospheric dispersion of SO_2 and NO_x.

Gas Generation in Radioactive Waste Disposal Facilities
Client - AEA Technology

Development of a coupled microbial degradation and corrosion model for gas generation in repositories for LLW and ILW.

Effects of Chernobyl on Drinking Water Supplies
Client - Her Majesty's Inspectorate of Pollution

Evaluation of the radiological implications of enhanced concentrations of radionuclides in water supplies in England and Wales subsequent to the Chernobyl accident.

Sea Disposal of Radioactive Wastes
Client - UK Nirex Ltd

Participation in an Environmental Impact Assessment of the proposed resumption of sea-dumping of radioactive wastes.

UK Research Related to Radioactive Waste Management
Client - Her Majesty's Inspectorate of Pollution

Identification of gaps in the UK national research effort related to radioactive waste management.

Research Requirements for Repository Design and Site Investigations
Client - UK Nirex Ltd

Review of research requirements for repository design and site investigations in relation to LLW and ILW disposal in near-surface and deep repositories.

International Commission on Radiological Protection, Sutton, Surrey, England, 1985-1986

Scientific Secretary responsible for arranging and minuting meetings, administrative arrangements, technical review of reports, editing of the Commission's journal, liaison with other international organisations and public relations.

ANS Consultants Ltd, Epsom, Surrey, England, 1979-1985

Reviews of data on the distribution at transport of radionuclides in terrestrial and aquatic ecosystems (see publications list).

Development of a dynamic model for radionuclide transport in agricultural ecosystems and implementation of the model on various microcomputer systems.

Photon and neutron shielding studies of radiochemical plant, together with area classification and ALARA studies.

A review of UK use of the criticality code MONK and other approaches to criticality safety assessment.

Radiological and conventional safety aspects of Magnox reactor decommissioning.

Development of metabolic models for inclusion in ICRP Publication 30.

Development of pharmacodynamic models for toxic chemicals.

Review of neutron activation analysis in studies of radionuclide transport in soils and plants.

Experimental studies on radionuclide transport in soils and plants using various photon-emitting radionuclides.

Support for DoE work on probabilistic risk assessment of LLW and ILW disposal.

Review of UK research requirements for HLW disposal.

Post-closure radiological impact assessment of the proposed LLW and ILW facility at Elstow, Bedfordshire.

Development of a generalised biosphere model for use in probabilistic risk assessments of solid radioactive waste disposal.

Initial development of a mathematical model for use in assessing the radiological impact of contaminated groundwater.

Development, computer implementation and comprehensive documentation of a model to calculate the radiological impact of intrusion into radioactive waste repositories.

Development of a general-purpose computer code for solving first-order differential equations using a hybrid Predictor-Corrector/Runge-Kutta method.

Studies on the potential radiological consequences of Magnox reactor accidents.

Medical Research Council Radiobiology Unit, Chilton, Didcot, Oxon, England, 1974-1979

Development of dosimetric and metabolic models for use in ICRP Publication 30.

Studies on the metabolism of plutonium in bone and relationships to blood flow.

Theoretical studies on radionuclide metabolism and dosimetry.

Development of techniques in neutron-induced autoradiography and alpha imaging.

Image analysis studies of plutonium in bone, uranium in lungs, lysosomal inclusions in cells and heterochromatin.

Studies on the clearance of inhaled UO_2.

Alpha spectroscopy in support of toxicity studies with Ra-224.

Data analysis in connection with experimental animal studies on the potential efficacy of neutron therapy using 42 MeV neutrons.

University of Sheffield, 1971-1974

Experimental studies on the reaction $\gamma + p \rightarrow \pi^o + p$ at photon energies between 1 and 3 GeV, using a linearly polarised photon beam.

SELECTION OF PUBLICATIONS

A measurement of the beam asymmetry parameter for neutral pion photoproduction in the energy range 1.2 - 2.8 GeV. P.J.Bussey, C. Raine, J.G. Rutherglen, P.S.L. Booth, L. Carroll, G.R. Court, A.W. Edwards, R. Gamet, C.J. Hardwick, P.J. Hayman, J.R. Holt, J.N. Jackson, J. Norem, W.H. Range, F.H. Combley, W. Galbraith, V.H. Rajaratnam, C. Sutton and M.C. Thorne. London Conference (1974) Abstract 997.

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Attachment III

***Epidemiologic Studies of the Cancer near the Cotter Uranium Processing Facility*, by David Richardson, March 1, 2007**

Epidemiologic Studies of the Cancer near the Cotter Uranium Processing Facility

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1. Introduction

Epidemiology is the study of health and disease in populations. Its methods have been used to address a range of topics including evaluation of the health effects of exposure to ionizing radiation. This review begins with a brief discussion of the methods used by epidemiologists to study radiation health effects, followed by a discussion of the Life Span Study of atomic bomb survivors and some studies of the effects of environmental exposures to ionizing radiation. The latter part of this report provides a critical review of several epidemiological studies of cancer in Lincoln Park, Colorado and discusses the prospects for additional research in this community.

2. Epidemiologic Methods

All epidemiologic studies must address concerns about the accuracy of measurements of exposure and disease, and the appropriateness of comparisons between groups of people.

2.1 Measuring exposure

People have to be accurately classified with respect to their exposures if an association between radiation exposure and disease is to be accurately assessed. If people have not been accurately classified into exposure groups then evidence of any adverse effect of radiation exposure may be obscured.

It is often presumed that studies of occupational exposure to radiation may have advantages relative to studies of environmental radiation exposures, since the magnitude of occupational exposures is typically greater than the magnitude of environmental radiation exposures and a researcher may have better information about the doses received by workers in occupational settings than about the doses received by people in environmental settings. For example, workers at nuclear facilities have often been issued personal dosimeters to monitor penetrating radiation exposures, a seemingly ideal measurement situation. While records of individual dose estimates are clearly a valuable resource, changes over time in who was monitored, the sensitivity of dosimeters, and the frequency of reading dosimeters could affect the reliability of

recorded doses. In addition, some workers might have removed their badges before performing tasks which entailed high exposures in order to ensure that their recorded external exposures were below standards which would otherwise require them to stop work.

In environmental and occupational settings, internal exposure to radionuclides typically is more difficult to assess than external radiation exposure. Ingested or inhaled alpha- and beta-emitting radionuclides have greater density of ionization than gamma or x-rays, but they have little penetration, so the relevant dose is delivered to a particular organ or cells within the organ. Knowledge about retention of radionuclides has been used to estimate exposures, and information from excreta samples (urine/fecal analysis) or field data (nose swipes, air samples, skin and clothing contamination estimates) are traditionally employed to estimate body burdens of workers. Estimates can also be made using whole-body counters that detect the penetrating radiation emitted by the internally deposited particles. However, most epidemiological studies have used crude indicators to identify those people likely to have been exposed to internal radionuclide contamination. In occupational settings, exposure categories might be based on job titles, area monitoring data, or history of monitoring for internal radionuclides, while in environmental settings, exposure categories might be determined by geographical location and patterns of environmental contamination.

2.1.1 Timing of exposures

The proper classification of people in a study by level of exposure requires not only good measurement of exposure, but also correct decisions about which periods of exposure are etiologically relevant. Sometimes only the doses received several years in the past are considered in forming exposure groups based on the assumption that cancers take time to develop and that recent exposures are not relevant to disease (so-called "lag" or "latency" analyses). Chronic exposures might have a greater opportunity to impact an organism during especially susceptible states; or it may be that only high dose rate exposures are relevant to the onset of later disease. So, a further difficulty in interpreting radiation-cancer associations, beyond the measurement process itself, is that mechanisms of radiocarcinogenesis are not

sufficiently well understood to provide a sound theoretical basis for knowing in advance what should be measured.

2.2 Measuring disease

The ability to accurately quantify the effects of radiation exposures also depends on accurate measurement of the outcome. Long-term studies of cancer typically rely on cancer mortality, rather than cancer incidence. In the United States, death certificates are the only universally collected health data, consequently mortality is an endpoint that can be readily determined in nationwide follow-up studies. There are important limitations, however, to mortality studies. The sensitivity and specificity of cancer death certificate diagnoses is often not very good. Incident cancers that are in remission, unrelated to the primary cause of death, or undetected at the time of death from other causes, may not be counted. Furthermore, non-fatal health effects cannot be assessed in mortality studies. While studies of cancer incidence overcome these obstacles, for many US states comprehensive cancer registry data began to be collected relatively recently and there is no national registry that may be used to easily ascertain cases when people move across state boundaries.

2.3 Comparing Groups of People

Most epidemiological studies are observational. They attempt to imitate a controlled experiment by making exposed and unexposed groups as similar as possible in every way other than exposure itself. The method of the discipline is to observe whether disease occurs more or less commonly among individuals who received an exposure than among those who did not. Comparability of groups with different levels of exposure is important in order to be able to attribute differences or similarities in disease rates between groups to radiation *per se*. This is accomplished both through the design of the study and through statistical analysis of the data.

One widely-used method involves comparison of disease rates in the study population to that of the general population using Standardized Incidence Ratios or Standardized Mortality Ratios (SIRs or SMRs, respectively). This method of analysis is useful in settings in which there is little or no ability to accurately discriminate between people in a study population with respect to

exposure level (either because exposure estimates are unreliable or because historical exposures were of similar magnitude for most people in the study). Such analyses permit an investigator to contrast cause-specific disease rates for the people in a study population, to age-, sex-, race-, and calendar-period appropriate rates for an external referent population. The study group and the referent group that are being compared are therefore balanced by means of adjustment for differences in age, sex, race, and birth cohort. However, these groups are not necessarily balanced with respect to other factors that may influence disease rates.

Interpretation of the SIR or SMR as an effect measure that represents the independent effect of exposures requires comparability (or 'exchangeability') of the study population and the external referent population. This condition may not be met if, for example, there are socioeconomic or other differences between people in the study group and the referent group that impact disease rates.(1-3)

As noted above, SMRs or SIRs are often calculated when an investigator has little or no ability to accurately classify people in a study population with respect to exposure. The contrast drawn (people in the study area to those in a referent area) implies that the data analyst is treating all people in the study population as though they have equal and identical exposures. If, in fact, the study population is composed of people some of whom received relatively high exposures (during an etiologically-relevant time period) and some of whom did not then collapsing these groups together for the purposes of calculating standardized rates will obscure important heterogeneity in disease rates.

2.4 Summary

Issues of exposure measurement, disease classification, and comparability of study subjects (between exposure groups and between the study population and the more general population) are issues for concern in reviewing the following studies.

The effects of measurement errors, selection bias, over-reliance on mortality data, and limited periods of follow-up tend to bias studies towards finding no radiation-cancer association. Given

these impediments, it is very difficult to detect associations between low level ionizing radiation and cancer. Findings which have been reported may be expected to be biased downwards. It should also be noted that epidemiology is used for different purposes in different circumstances. Often, epidemiological studies are conducted in order to identify a potentially hazardous agent. Such studies may use relatively crude indicators of exposure, and yet evaluate whether an agent is associated with a disease.

3. Radiation risk estimates

The risk estimates used to inform assessments of environmental radiation exposures are derived primarily from studies of atomic bomb survivors.

3.1 Atomic bomb survivor studies

Recent reports of the US. National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation, and the International Commission for Radiological Protection focus on the Life Span Study (LSS) of survivors of the atomic bombings of Hiroshima and Nagasaki as the primary source of information for understanding radiation health effects. These reports, subsequently, serve as a primary resource for a number of other recent reviews on radiation health effects. Most reports of results of LSS analyses concern radiation-related changes in cancer mortality rates among the survivors, often reported as the estimated excess relative risk of cancer mortality per Sievert (Sv) radiation dose (ERR_{1Sv}).

In the Life Span Study of atomic bomb survivors, estimation of the exposures received by thousands of survivors has occupied researchers for more than forty years. The level of radiation exposure received by an individual from the bombing was affected not only by where the person was situated geographically, but also their body position at the time of the explosion, whether they were shielded from the explosion, the type of shielding material, atmospheric humidity, patterns of movement and activities immediately after the explosion (due to concerns about residual radiation), ingestion of radioactive material, and bomb design. Exposure misclassification, which may result from incomplete information, and from inaccurate survey data elicited from a highly traumatized population, is likely to produce errors in dose estimates and in radiation dose-response estimates.

Overall, increases in solid cancers and leukemias have been observed with increasing doses; leukemia mortality has shown larger associations than solid cancers, with the largest excess risks among survivors who were under 20 years old ATB. The range of health effects from exposure to ionizing radiation may extend beyond cancer, to non-cancer diseases (recent evidence suggests that cardiovascular disease risk is associated with radiation dose in the LSS) and heritable genetic effects.

Summary

In section 2, we outlined some concerns that are common to all epidemiologic studies, which related to comparability of populations at risk and to exposure and disease measurement. Exposure measurement in studies of a-bomb survivors relied on questionnaire data collected over a period of many years after the bombing. Patterns of exposure were extremely complex due to shielding by buildings and terrain, and little attention has been given to the contribution of delayed, residual or induced radiation to the doses received by survivors presumed to have little or no radiation exposure. Evaluation of death certificate records also suggest problems of disease misclassification. It has been noted that the overall percentage agreement between death certificate and autopsy diagnoses in the LSS data was only 52.5%, with 25% of cancers diagnosed at autopsy missed on death certificates(4). These are issues which affect the internal validity of a study. Validity may also be evaluated with respect to the appropriateness of using results from one study population to make conclusions about people in other situations--this is called external validity. Some researchers have questioned whether it is appropriate to use results drawn from the LSS, a study population of five-year survivors of an atomic bomb detonation, to form conclusions about the effects of radiation in contemporary populations exposed to environmental exposures. One reason to question the validity of such conclusions relates to the difference in exposure patterns. The pattern of exposure from an atomic bomb blast is significantly different from exposure patterns in occupational and environmental settings. In contrast to studies of atomic bomb survivors, concerns about occupational and environmental exposures are related to the effects of long term exposure to low level radiation, at low dose rates. Others have raised concerns about selective survival of people after the atomic bombing. Premature deaths of people who were sensitive to the acute effects of radiation may have led to

the selective removal of those who were more sensitive to the later effects of radiation as well. Consequently, when follow-up began five years after the bombing only a select population of less radiosensitive persons may have been left(5-7). A recent survey of mortality in Nagasaki during the period 1945-1950 has also suggested potential selective survival among A-bomb survivors(8). This situation raises questions about the applicability of estimates of radiation-cancer associations among A-bomb survivors to other populations.

One alternative source of quantitative radiation risk estimates come from large pooled analyses of workers in the nuclear industry. The largest study to-date is the International Collaborative Study of nuclear workers. While occupational cohort studies also suffer problems of exposure misclassification (e.g., problems of misclassification with respect to internal doses) and problems of outcome misclassification (due to reliance on death certificate data), as well as problems of bias due to confounding (i.e., non-comparability of workers in exposure groups with respect to unmeasured factors such as smoking), these studies provide a potentially important source of information on risks associated with protracted radiation exposures.

4. Epidemiological Studies of Effects around Nuclear Facilities

4.1 Introduction

While environmental releases of radiation are of wide concern, epidemiologic analyses of the effects of these releases suffer from lack of available data on magnitudes of exposure, pathways for exposure, and time-patterns of exposure. Furthermore, in a general population, complicated patterns of migration limit the ability to follow-up people to assess disease status. Environmental epidemiological studies generally rely on correlations between geographical patterns of exposure and disease incidence. A reasonable concern in such studies is the potential for confounding factors to lead to spurious observed associations (due to differences in the geographical distribution of other cancer risk factors); critics less often note that confounding, in addition to migration and errors in dose estimates, could lead to masking or underestimation of exposure effects.

Typically, these studies compare death or disease rates among populations presumed to have different levels of exposure. Environmental exposures are generally assumed to be low, consequently, differences in disease rates between populations are presumed to reflect very small differences in exposure magnitude. For example, excesses of childhood leukemia were reported in the area around Sellafield in the 1980s. An investigation of this cluster of leukemia was conducted by Gardner et al. Using a case-control study method, information was collected about all known cases of leukemia and lymphoma among children in the area health authority between 1950-1985 and compared to information about local controls selected from the birth registry. A number of potential risk factors were examined, and father's employment at the Sellafield nuclear facility was identified as an important risk factor (9-11). Furthermore, fathers of cases who had worked at Sellafield had larger cumulative preconception doses than fathers of controls who had worked at Sellafield. A subsequent study examined followup through 1991, attempting to avoid criticisms which were directed at previous analyses by specifying, a priori, the outcomes of interest and geographic areas defining the study population(12). Excess leukemia incidence was noted in the area, which the authors suggest might reflect occupational or environmental exposures.

In Scotland, excess childhood leukemia and non-Hodgkins lymphoma has been reported in the area around the Dounreay nuclear facility. The excess first reported in the 1980s has persisted with more recent follow-up through 1991 (13); a case-control study identified use of the local beaches as associated with childhood leukemia (14). An analysis of leukemia and lymphoma incidence around seven nuclear sites in Scotland found a significant excess only around Dounreay (15). Similarly, a case-control study of childhood leukemia near the La Hague plutonium reprocessing facility in France found evidence that environmental radiation exposure from recreational activities on beaches and from shellfish consumption could be associated with increased childhood leukemia among area residents (16, 17).

Cancer around the Rocky Flats nuclear weapons facility has been the subject of a more detailed investigation which used environmental exposure estimates. Johnson evaluated cancer incidence patterns for the period between 1969-1971 in areas with varied estimated levels of contamination from plutonium and other radionuclides emitted by the Rocky Flats plant near Denver, Colorado

(18). He compared the cancer incidence rates of four geographic regions around Rocky Flats that were determined using isopleths from an area-wide survey by the AEC in 1970. There was a 24 % higher cancer incidence in males in Area I (highest exposure) vs. Area IV (lowest exposure), and a 15 % higher cancer incidence in Area II vs. Area IV. For females, there was a 10 % increase in cancer incidence in Area I, and 10 % increase in Area II. Johnson concluded that exposure of general populations to Pu and other radionuclides may have an effect on cancer incidence rates and that further study is warranted to investigate the poorly understood dose response relationship between Pu exposure and cancer in populations living near nuclear facilities. In a re-analysis, Crump obtained similar results for 1969-71 and extended the analysis to 1979-81 (19, 20). Positive findings were diminished by adjustment for distance from the State Capitol. Crump argued that distance from the State Capitol was a measure of socioeconomic factors related to cancer incidence, however, he does not present findings for conventional measures of socioeconomic status, and provides no quantitative evidence for this assertion.

Reports of excess adult leukemia incidence around the Pilgrim power plant in Massachusetts led to an investigation of association between proximity to the facility during years of 'high emission' and leukemia incidence; a positive dose-response association was observed. In other analyses innovative applications of geographical information systems data were used in conjunction with cancer registry data to explore spatial and temporal distributions of cases.

An analysis of leukemia and lymphoma incidence was conducted in Northern Germany following reports of a cluster of childhood leukemia cases near a nuclear facility (21). A highly-detailed population-based case-control study of leukemia and lymphoma was subsequently conducted which assessed potential exposure to ionizing radiation from the routine operation of nuclear power reactors, as well as pesticide exposures, electromagnetic field exposures, illustrating an approach for a comprehensive assessment of known or suspected risk factors.

4.2 Conclusions

Epidemiological techniques are well suited to documenting strong risk factors that show little or minor variation in impact in various population subgroups, such as regular cigarette smoking or

high dose ionizing radiation. However, due to the importance of environmental contamination and the potentially large population receiving exposure, radiation epidemiology must now focus on weaker relationships at lower exposure levels, where poor measurement and the presence of unmeasured differences between exposure groups become major potential problems. Relatively small differences in disease occurrence, such as those that are suspected in the case of many environmental radiation exposures, are difficult to detect. But small increments in disease incidence can have a great population impact when many people are exposed.

Environmental releases of radioactive material may be of particular concern because the effects of radionuclide exposures are believed to be modified by many substances. For example, gastric absorption of plutonium tends to be very low in occupational settings; however, in the presence of fluoride, chlorine, or carbonate ions, the gastric absorption of plutonium rises to near 100% absorption. Consequently, environmental releases of radionuclides that contaminate drinking water, which is often chlorinated and may contain fluoride and carbonate ions, may lead to high levels of internal contamination. Through the food chain, radionuclides may be incorporated and uptake increased as well.

The effect of low level radiation exposure on cancer incidence in populations is difficult to quantify with epidemiological methods. Epidemiological studies tend to suffer from poor measurement of exposures. Furthermore, movement of people across local and national borders makes long term follow-up (which must span decades to study cancer effects, or generations to study genetic effects) difficult and nearly always incomplete. These problems affect studies of atomic bomb survivors as much as studies of environmental contamination; and, the tendency of these problems is to bias studies towards an underestimate of the true consequences of radiation exposure (22). While radiation risk estimates from studies of atomic bomb survivors are often used, in conjunction with environmental exposure estimates, for the purposes of risk assessments it is important to note these common limitations of epidemiologic research.

5. Critical Review of Epidemiological Studies of Cancer in Lincoln Park

Several studies have been conducted of cancer among people living near the Lincoln Park Superfund site. The studies that have been conducted to-date involve comparisons of the observed numbers of cases of specific cancers to expectations based upon cancer rates in other parts of Colorado. In other words, these studies address the question "Are there more cancer cases in the Lincoln Park area than in other parts of Colorado?" It is reasonable to ask whether this is the question initially posed by the citizens of Lincoln Park. Suppose that the question of actual concern to members of the community is, "Have exposures to radiation or other hazards from the Superfund site increased my risk of developing cancer?" The comparisons of observed cancer cases in Lincoln Park to expectations based upon rates in other parts of Colorado do not necessarily have bearing on the latter question. The studies that have been done, therefore, may have given the right answer to the wrong question.

5.1 Limitations of study design

It is useful to consider some elements of study design that might be employed to provide a more direct investigation of whether exposures from the Lincoln Park Superfund site affected cancer rates of residents of Lincoln Park. To address such concerns one might enumerate a roster of people who have lived in the area during a specified period (e.g., anyone resident in the area during a period encompassing mill operations, 1958-1989). Those who lived in the area during the study period would then be followed up to determine their exposure status (e.g., based upon their residential and occupational history) and their disease status (e.g., based upon cancer diagnoses or causes of death). The relationship between estimated exposures from environmental contamination and subsequent disease could be assessed.

Such an approach differs in several important ways from the study design employed in the 1991, 1993, and 1998 analyses of cancer in this community. Rather than a fixed roster (i.e., cohort) followed over time, people entered and exited the study depending upon whether they moved into or out of the study area. As noted in section 2, follow-up of a roster of people in the US to determine cancer incidence is relatively difficult.

Also as noted in section 2, the adverse effect of an exposure may be obscured by misclassification of people with respect to exposure. If a person residing in the study area was diagnosed with cancer they were classified as an exposed case, regardless of how long they had lived in Lincoln Park. Similarly, if a person moved out of the area and was diagnosed with cancer they did not contribute to the case count for those residing in the exposed area, regardless of how long they had lived in Lincoln Park. Induction and latency periods were not considered. People were classified as exposed based upon residence at time of diagnosis; however, the exposure status of interest is presumably environmental exposures associated with residence years or decades prior to diagnosis. Temporal changes in the magnitude of exposure have not been considered in the design of the study or the interpretation of results. Concerns about environmental exposures from the uranium mill date back to 1968 and the mill stopped operations in the 1980s. As follow-up of this cohort continues, therefore, it may not be reasonable to expect that the exposure-related excess of disease will persist at the same magnitude indefinitely. The magnitude of the exposure may have changed; and, given that the magnitude of exposure does not remain constant, even if the rate of in-migration and out-migration is steady people will be increasingly misclassified with respect to exposure.

At the outset of a study it is useful to define outcomes of interest. The exposures of primary concern in Lincoln Park are uranium and its decay products. In this setting, lung cancer is one obvious outcome of concern. Ascertainment of lung cancer cases should be relatively complete and diagnoses should be relatively accurate, which supports consideration of this outcome.

5.2 Limitations in the interpretation of results

In addition to considerations of the determinants of validity of a study (e.g., exposure and outcome classification and appropriate comparisons) a critical review should consider the use of statistical methods and the interpretation of results derived from such methods.

Statistical modeling of epidemiological data is useful primarily as a means of data summarization and pattern detection. Statistical models should not be confused with biological

or etiological models of disease processes. This seems to be a recurrent problem in the interpretation of evidence in the 1991, 1993 and 1998 reports. The proposition in these reports that chance is a causal explanation is wrong. Chance as a cause of health effects is only valid in scenarios where chance is introduced by the investigator (e.g., random assignment to exposure groups). In an observational setting, diseases are caused by biological and physical processes. We analyze these data using models in which we may choose to model observations as though they were random variables (e.g., conforming to Poisson or Bernoulli processes) however this is for purposes of data summarization and pattern recognition not biological inference. If more cases of disease occur in an area than expected then a scientist should not posit that this occurred because of chance. Reasonable explanations would be confounding, selection bias, or measurement error.

Similarly, interpretation of confidence intervals in these settings are only appropriate in quantifying what results might be observed if we assume that there is a super-population from which we could redraw observations, and we assume that the processes generating these data persistently conform to our statistical model, and we assume that there is no confounding, selection bias, or measurement error. Over a large number of replications, 95% of the confidence intervals constructed using this formula would include the true population parameter (in this case the ratio measure). However, this is a long list of assumptions. The point estimate of the standardized incidence ratio is the most likely value for the population parameter given the observed data at hand and the posited statistical model, values closer to the bounds of the confidence interval tend to be much less likely than the point estimate.

In the 1991, 1993, and 1998 reports, the authors give excessive attention to the lower bound of the 95% confidence interval while giving minimal attention to the point estimate which is the most likely estimate of the population parameter. During the period 1979-1990 the observed number of cases of lung cancer among men (41) is about forty percent greater than the expected number. With further follow-up through 1995 this has diminished to about 12% excess, although there is no reason to expect an excess to persist indefinitely nearly two decades after the facility has closed. Potential explanations for this observed excess relate to comparability of the study area and the referent area; perhaps the investigators made a poor choice of referent populations

(i.e., confounding away from the null). On the other hand, possible explanations for attenuation include poor choice of referent (i.e., confounding toward the null) and also include exposure misclassification (e.g., due to migration patterns) and misspecification of the etiologically-relevant time window of exposure. In other words, there are plausible reasons to expect that the study design employed could have attenuated or obscured any true effect of exposure.

In contrast to statistical models are biological models of carcinogenesis. Cancer is typically viewed as a disease that does not have a single cause, even for a single person's case of cancer. Rather cancers are widely viewed as arising from a step-by-step process of induction that involves early and late stages of action, with agents that may act as initiators or promoters, including factors that spur clonal expansion or proliferation and factors that influence the environment of pre-malignant cells. Just as the statistical models employed for data summarization should be interpreted as such, this biological framework is important to keep in mind when reading assertions such as "smoking is generally believed to be the cause of 85% of lung cancers" and "90% of the Lincoln Park lung cancer cases for 1988-1999 had a history of smoking." Smoking is clearly a factor that influences the risk of lung cancer and plays a role in the multistage process leading to lung cancer. That does not imply, however, that radiation exposure has not also played a role in the induction of the very same cases of lung cancer. The fact that a lung cancer case has arisen among a smoker does not mean that this person would have developed cancer regardless of their radiation exposure (or, that the person's cancer would have developed at the same age).

6. Prospects for Further Epidemiological Studies of Cancer in Lincoln Park

The prospects for meaningful additional research addressing the health effects of exposures from the Lincoln Park Superfund site largely depend upon the ability to address the fundamental requirements for epidemiological research: exposures assessment, disease ascertainment, and valid contrasts between groups under study. The methods employed previously for analyses of standardized incidence ratios provide some useful information, although these methods clearly fall short on each of the requirements for valid inferences (i.e., exposure misclassification is substantial; disease ascertainment only occurs during the period when residents reside in the

state; and contrasts adjust for basic demographic confounders but may well be unbalanced with respect to other risk factors of concern). While additional research could be done, if it fails to move beyond the limitations of the prior research then little will be added in terms of value in understanding these relationships.

That said, studies are done for a variety of reasons and in some circumstances the conduct of the study itself may be of benefit; for example, community-initiated research can serve as a catalyst for great democratic participation, self-organization, and community dialogue. However, in terms of etiological research, the fundamental elements of study design necessary for valid inferences must hold.

6.1 Case-control studies

As opposed to the idealized cohort study outlined in section 5.1, the case-control study design has been employed in a number of settings to investigate hypotheses about environmental factors influencing cancer risk. Some investigators have conducted studies that focus on identifying newly diagnosed cases in the community (for whom information might be obtained via interviews) while others have conducted studies that include retrospectively ascertained cases (many of whom will be deceased). Often more accurate and detailed information can be collected from living cases than from next-of-kin or acquaintances, although a larger number of cases might be identified retrospectively. If a study is focused on characterizing any exposure-induced excess risk of disease then retrospective ascertainment of cases may also be important if the magnitude of environmental exposures has tended to diminish in more recent years.

Dr. Jarvis has offered his assessment of the feasibility of a case-control study, with his first concern being limitations of statistical power. He noted that given: a study that included 55 lung cancer cases, standard assumptions about type I and II error, and a binary exposure that was of intermediate prevalence among the controls, the study would have power to detect an odds ratio of 3 (i.e., on average result in an effect estimate with 95% confidence intervals that excluded unity). On the one hand, such power estimates are unduly conservative as a guide for decision making for contemporary residents of Lincoln Park. In follow-up through 1995, 74 lung cancer

cases had been ascertained (furthermore, there is no reason that citizens of Lincoln Park would be limited to conducting a case-control study only of lung cancer, cases could be enumerated for all cancers or even broader categories of disease). Also, assuming that a contemporary study could derive a categorical or continuous exposure classification, as opposed to the binary classification assumed by Dr. Jarvis, a statistical analysis based on trend tests would tend to have greater power than the test of a binary classification of subjects. While unstated, it appears that Dr. Jarvis' calculation assumes a 1:1 matched case-control design; such a design has approximately 50% of the statistical efficiency of a full cohort analysis. A more powerful case-control design would be obtained via a 4:1 or 5:1 control to case ratio; the latter would allow a study to have power to detect effects of smaller magnitude. Lastly, the presumption that power calculations should be premised on the construction of 95% confidence intervals (i.e., alpha=0.05 for type I error) is arbitrary and, in radiation epidemiology, often replaced by consideration of 90% intervals.

This is not to discount the fact that small magnitude effects associated with low level environmental exposures are very difficult to detect via epidemiological methods. However, the obstacles to such studies often follow more from measurement error problems than they do from the random (i.e., sampling) errors quantified by the statistical power calculations described above. I would argue that the fundamental question about the prospects for any epidemiological study in this community is "Is there a method for accurately estimating historical exposures among Lincoln Park residents?" As noted above, epidemiology is often a relatively crude tool well suited to detection of differences in disease rates between sizable groups of people who have markedly different exposure patterns (resulting in markedly different disease patterns). When the populations are small, the exposure patterns are poorly characterized, and/or the differences between groups in disease rates are not large, epidemiological methods may be inadequate to address community concerns.

Summary

Prior studies of cancer in Lincoln Park have suggested greater than expected numbers of lung cancer cases (as well as excesses of several other types of cancer). If there is no ability to accurately estimate historical exposures then there is very little basis for conduct of additional

epidemiological research. If exposure estimates can be derived then one option is to use these in conjunction with previously reported radiation risk coefficients to derive estimates of the potential risks from these exposures. Alternatively, historical exposure estimates could be used with a cohort or case-control study design. Such studies often require substantial time and resources, and may suffer from low participation rates (in case-control studies), loss to follow-up, and measurement error. If the number of exposure-induced excess cases is small, it is easy for the adverse effects of exposure to be masked by the errors in the study data. Therefore, it is important to be circumspect about the potential of epidemiological research to detect the effects of environmental hazards, appreciate the limitations of such research, and recognize that when such studies are conducted the results seldom provide evidence that is unambiguous. In this context, I would view the limitations for studying the effects of environmental contamination in the Lincoln Park community not as primarily following from statistical power; rather the utility and validity of such a study will depend upon the accuracy and completeness of information on exposures, disease, and potential confounding factors that could be derived for the population and time period of concern.

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Attachment IV

Long-term issues regarding Cotter mill tailings

Long-term issues regarding Cotter mill tailings

Like all uranium mill tailings sites, Cotter mill tailings contain high concentrations of radium-226 and its parent radionuclide, thorium-230. The latter has a half-life of over 75,000 years. Hence significant concentrations of both thorium-230 and radium-226 (with a half life of 1,600 years) will remain in the tailings for hundreds of thousands of years. This is far longer than any reasonable expectation of the life of the liner or of institutional control. Further, the heavy metal pollutants in the tailings, notably molybdenum, being stable elements, are literally forever. Neither the State of Colorado nor the federal government has addressed the long-term disposition of mill tailings beyond the kinds of remediation measures now in place, notably the liner (which will not endure anywhere near the requisite time) and the pumping back of groundwater from the Soil Conservation Service flood control dam/Permeable Reactive Treatment Wall[1] to the tailings pond, which requires both funds and institutional control. A failure of the federal government to address issues does not, in our view, relieve the State of Colorado from the responsibility to initiate a dialog with the community about its long-term protection. Such a dialog is necessary before any permission is granted to Cotter to bring in new materials for processing.

Further, there is evidence that the liner under the tailings is torn as stated in the 1986 Remedial Investigation report:

> A continuing history of breaches (tears, rips, holes, separations, fabric failures) in the impoundment's Hypalon liner has been documented at the Cotter site. Since October 1980, at least thirty-two instances have been reported in Cotter Corporation memoranda documenting over 70 breaches of the Hypalon liner. …. This history was compiled from observations where the Hypalon is exposed and observable; most of the Hypalon is submerged beneath raffinate and is neither observable nor repairable. While many of the breaches are due to operation and maintenance of the impoundment, others are related to poor construction practices, affecting all areas of the impoundment.[2]

The 70 breaches that were "exposed and observable" were repaired,[3] according to the letter from Sentinel Consulting Services which goes on to say that:

> Multiple lines of evidence contained in design, construction, and post-construction documentation strongly suggest that the current effectiveness of the Hypalon liner is severely limited…[4]

The integrity of the liner is of the utmost importance because

> … the hydrogeology immediately beneath the impoundments is not well understood. … [C]onditions suggest that pathways exist for migration of impoundment seepage into both shallow and deep aquifers.[5]

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[1] Cotter 2003 pages 54 and 55
[2] Geotrans, et al. 1986 pages 3-27 to 2-28
[3] Sentinel 2004 page 9
[4] Sentinel 2004 page 15
[5] Sentinel 2004 page 14

There is at present no plan in place to address that problem or the possible acceleration of liner deterioration that the tears may cause. Nor has there been a comprehensive effort to address the long-term implications of a deterioration of the liner for groundwater protection. For instance, such deterioration could originate in the weakened or compromised locations.

IEER understands that the history of the liner, including the problems with it and the attempts to address them, is long and complex. But the uncontroverted facts point to the need for a public dialog and also the development of a specific remediation plan (along with funding for the plan) before a permit is given for new wastes to be put into the tailings ponds.

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Attachment V

Some observations regarding the BEIR VII report

Some observations regarding the BEIR VII report

In 2006, the Nation Research Council of the National Academies published its findings updating prior work on the risks of low-level radiation. This report, commonly called the BEIR VII report,[1] reaffirmed a key prior finding, but also added new findings that are important for public health protection. The most important finding of the BEIR VII report in relation to its prior report was that it reaffirmed the linear no-threshold (LNT) hypothesis for cancer risk as the one which best fits the available scientific data overall. This means that public health standards and risk assessments will continue to be based on this cancer risk model – every increment of radiation, no matter how small, creates a corresponding increase in cancer risk. It also means that small doses to individuals can be added up to calculate a population dose and corresponding number of expected cancers in that population.

Every official scientific assessment for decades has come to the same conclusion about low-level radiation risk and reaffirmed the LNL hypothesis. There is a tendency among some to disregard this central finding (which is qualitatively the same as that in the BEIR V report).[2] This is inappropriate for the licensees of the Nuclear Regulatory Commission and their consultants, notably those who are charged with preparation of environmental documents and health risk assessments for workers and the public.

The BEIR VII report also contains new analysis that was not part of the earlier BEIR V report. Specifically it contains

- Data on risk of cancer incidence, in addition to cancer mortality, per unit of radiation exposure.
- Data on cancer risk by gender and age.

The incidence data are important, since they are a more fundamental indicator of risk. Cancer deaths are a function of incidence and the state of treatment and health care systems. Incidence is more a reflection of underlying cancer risk factors. Similarly, the BEIR VII analysis shows that, overall, women are at about 50 percent greater risk of getting cancer than men as a result of a given amount of radiation exposure. Similarly, children are generally at greater risk than adults. Public health assessments should take this into account.

The BEIR VII findings are similar to those of Federal Guidance Report No. 13, which has been published by the EPA.[3] Cotter Corporation as well as the Colorado Department of Public Health and Environment should either use the BEIR VII report or, alternatively, FGR 13 in their estimation of public health risk. Cancer incidence should be the focus of the risk assessments, and risks should be estimated by gender and age.

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[1] NAS/NRC 2006
[2] NAS/NRC 1990
[3] FGR 13

The Environmental Protection Agency, which requested that the National Academies produce the BEIR VII report, is engaged in a process of evaluating it and incorporating it in its standards. In the meantime, it is prudent for corporations and their consultants, to use the BEIR VII results to estimate risk when communicating with the public. At the very least, the dose conversion factors provided by the EPA in FGR 13 and the differential risks between men and women that are discussed there should be reflected in corporate risk assessments.

References

FGR 13 Keith F. Eckerman, Richard W. Leggett, Christopher B. Nelson, Jerome S. Puskin, and Allan C.B. Richardson. *Cancer risk coefficients for environmental exposure to radionuclides*. EPA 402-R-99-001. Federal Guidance Report No. 13, CD Supplement. Rev. 1. Oak Ridge, TN: Oak Ridge National Laboratory; Washington, DC: Office of Radiation and Indoor Air, U.S. Environmental Protection Agency, April 2002. Includes original 1999 FGR no. 13, which is on the Web at http://www.epa.gov/radiation/docs/federal/402-r-99-001.pdf.

NAS/NRC 1990 Arthur C. Upton (Chair) et al. *Health Effects of Exposures to Low Levels of Ionizing Radiation, BEIR V*. Committee on the Biological Effects of Ionizing Radiations, Board on Radiation Effects Research, Commission on Life Sciences, National Research Council. Washington, D.C.: National Academy Press, 1990.

NAS/NRC 2006 Richard R. Monson (Chair) et al. *Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII – Phase 2*. Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation, Board on Radiation Effects Research, National Research Council of the National Academies. Washington, DC: National Academies Press, 2006. On the Web at http://darwin.nap.edu/books/030909156X/html.

Attachment VI

IEER's Recommendations

IEER'S recommendations are summarized below:

1. The recommendations made regarding the air monitoring system have, to our knowledge, not been implemented. They should be implemented. They are
   (i) Cotter should estimate the uncertainties in dose and show the 95 percent upper confidence bound for all radiation doses.
   (ii) Cotter should determine the causes of the offsite up-trends in Th-230 concentrations and report them as part of its air monitoring section in the RAP Status Reports.
   (iii) The event actuated, high wind air monitoring system should be restored and enhanced at the points where new housing is being built.
   (iv) The adequacy of this system for detecting accidental releases of radioactivity from Cotter Mill should be evaluated under different assumptions of meteorological conditions during accidents and additional air monitors should be added as needed to be able to detect all plausible accidents with high probability.
2. The Agency for Toxic Substances and Disease Registry (ATSDR) should pay attention to Dr. Richardson's report, in particular section "5. Critical Review of Epidemiological Studies of Cancer in Lincoln Park," for the production of its Public Health Assessments. In particular it should find the people who lived in Lincoln Park at the time of operation of the old mill, from the late 1950s to the late 70s, and define the source term for the old mill. An exposure assessment should be done on this basis. This is essential to a credible and scientifically defensible study. Without it, any study of the health effects of Cotter operations may well lead to the conclusion that there were no adverse effects even if there were some. A study done without the scientific prerequisites for a sound analysis would be at best a waste of public funds.
3. The Graham report is fundamentally flawed and needs to be scrapped and redone.
4. The 35 micrograms of uranium per liter compliance goal for Lincoln Park should be changed to 30 micrograms per liter, the EPA standard for drinking water.
5. An experiment should be conducted with a control plot irrigated with water from the river and with water from the contaminated wells. The type of vegetables to be grown should also be determined. Appropriate attention should be given to water chemistry, to the independence of the study, and to the quality control at the laboratory where the results are analyzed.
6. A community water use survey should be done in Lincoln Park by the Colorado Department of Public Health and Environment. This is a critical issue; the survey should be thorough and done as soon as possible.
7. A long-term management plan for Cotter Mill tailings should be developed that takes into account (i) the need for continued pumping back of contaminated groundwater above the Soil Conservation Service flood control dam back into the tailings ponds, (ii) the fact that at over 75,000 years the half-life of thorium-230, a dominant radionuclide in the tailings, is much longer than the life of even an intact liner, (iii) that long-term site control is unlikely. The plan should be developed in a public process with community involvement. Due consideration should be given to the fact that institutional controls are generally not expected to

last for more than a century. There is at present no long-term plan in such a circumstance to protect water supplies after the lapse of institutional controls (for instance, in the event that Cotter leaves the community or runs into financial difficulties. Before new wastes are authorized to be put into the tailings, a long-term plan to protect groundwater and surface water resources should be developed.

8. Cotter Corporation and its consultants should be required to use the cancer risk results of the BEIR VII report consistently, in all its public presentations, including in its environmental reports, until the EPA publishes a new federal guidance report on radiation risk. Cotter's reports should be required to include cancer incidence risks by age and gender.