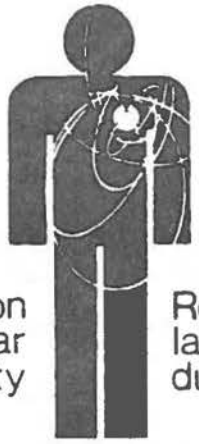

Canadian Coalition for Nuclear Responsibility

Regroupement pour la surveillance du nucléaire

ESTIMATING LUNG CANCERS

OR,

"IT'S PERFECTLY SAFE, BUT DON'T BREATHE TOO DEEPLY"

A summary of testimony presented by

Gordon Edwards

to the Elliot Lake Environmental Assessment Board

dealing with

the problem of radon gas in new homes

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Canadian Coalition for Nuclear Responsibility
Regroupement pour la Surveillance du Nucléaire
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INTRODUCTION (1985)

In March, 1978, there were three days of hearings by the Elliot Lake Environmental Assessment Board into the question of how much radon gas should be considered acceptable in new housing.

At the invitation of Homer Séguin of the Steelworkers , I attended the hearings and testified as a mathematician on the health risks of radon gas. Using the Ontario Government's own published mortality figures, I pointed out that continuous exposure to the officially proposed level (for the maximum permissible dose of radon in new homes) would result in a 30 percent increase in lung cancer deaths among the exposed population.

At present, 54 out of every 1000 males in Ontario eventually die of lung cancer. A 30 percent increase in this mortality rate means an additional 16 lung cancer deaths per 1000, for a total of 70 per 1000: a shocking increase in the incidence of an already shocking disease.

At the time, I had no way of knowing whether my conclusions would stand the test of time. I had simply accepted the government's figures and used basic arithmetic to estimate the increase in lung cancer mortality. Nevertheless, the evidence I gave convinced the Assessment Board that a re-evaluation of the radon standard should be undertaken. A recommendation was made to that effect. It was ignored by both provincial and federal authorities.

In 1980, the British Columbia Medical Association published a 300-page book entitled "The Health Dangers of Uranium Mining and Jurisdictional Questions", written by two medical doctors: Eric Young and Robert Woollard. The authors, who carefully reviewed all available evidence from the leading medical authorities on the subject of radon hazards, fully confirmed my 1978 estimates. In fact, they estimated a 40 to 50 percent increase in lung cancer rates resulting from continuous exposure at the so-called "acceptable" level of radon exposures in homes.

The Canadian nuclear establishment, which had been assuring people for years that low levels of exposure are perfectly harmless, was understandably upset by these unpleasant predictions. The Atomic Energy Control Board (AECB) refused to credit the BCMA risk estimates, and yet made no attempt whatsoever to discuss the medical evidence with the authors of the Report. Atomic Energy of Canada Limited (AECL) published an angry retort, and tried, without success, to get the BCMA to disassociate itself from the Report. Today, the BCMA still stands behind the integrity of the Young/Woollard Report.

Meanwhile, independent American scientific organizations -- notably the National Academy of Sciences (NAS) and the National Institute of Occupational Safety and Health (NIOSH) -- were arriving at conclusions very similar to those reached by the BCMA.

The Academy's 1980 BEIR-III Report reported risk estimates for lung cancer mortality caused by low levels of radon exposure which were fully consonant with the BCMA findings. That same year, a NIOSH Report called for a tightening of the standards governing permissible levels of radon exposure, citing the results of numerous studies which indicate that low levels of radon exposure may be much more harmful than was previously thought.

Against this background, the AECB's Advisory Committee on Radiological Protection commissioned a study by Duncan Thomas of McGill University to review the existing medical evidence on radon hazards. Dr. Thomas, a trained epidemiologist, based his study on the numbers of recorded deaths from lung cancer among workers exposed to various levels of radon gas in Sweden, Czechoslovakia, Colorado, and Canada. His report, the most careful study of its kind ever done in Canada, was published by the AECB in 1982. It estimates that continuous exposure to the maximum permissible level of radon in homes will likely result in a 37 percent increase in lung cancer deaths. (The report also estimates that continuous exposure of underground miners to the much larger maximum permissible level of radon in the mines will likely cause a three or four hundred percent increase in lung cancer deaths!)

Although the AECB published Dr. Thomas' report, it has chosen to ignore it. In a slim thirteen page document published in 1982, the same Advisory Committee which commissioned Dr. Thomas' report dismisses it with scarcely a word of explanation. No reason is given, except that it doesn't "jibe" with the findings of various pro-nuclear bodies (such as the International Commission on Radiological Protection) which the AECB prefers to place its trust in. Neither the Advisory Committee nor the AECB has prepared any critique of Dr. Thomas' work, nor have they identified any mistakes in his methodology. They just don't like his findings.

The reason for their dislike became clear when, in November of 1983, AECB announced plans to completely change the existing regulations governing radiation standards. Under the new proposed régime, various vital organs in both atomic workers and members of the Canadian public could be exposed to considerably larger doses of radiation than are currently permitted. In particular, the maximum permissible exposure to radon gas would be increased by about 20 percent.

Because of an unprecedented storm of opposition from all of the major unions representing Canada's 100,000 atomic workers -- including uranium miners, reactor operators, and those who handle radioisotopes -- AECB has temporarily withdrawn its proposed new regulations on radiation standards. As of August 15 1985, no further effort has been made by AECB to relax the existing radiation standards. But the situation may change at any time.

Radioactive pollution should be a matter of concern for all Canadians. Although this report deals only with lung cancer caused by radon gas I hope it is of some use in awakening other Canadians to the dangers we all face in an increasingly radioactive world.

Gordon Edwards
August 15 1985

INTRODUCTION (1978)

In the Spring of 1978, Ontario Hydro signed contracts with Denison Mines and Preston Mines to supply uranium for those nuclear reactors already operating, under construction, or firmly committed in the Province of Ontario. The value of these contracts may exceed $7 billion.

As a result the town of Elliot Lake is undergoing phenomenal expansion. Uranium production is expected to increase by a factor of about five over the next few years. Whole new subdivisions are springing up to accomodate the workers and their families. Unfortunately, many of these new homes are showing high levels of radon gas in their basements -- presumably because of the natural radioactivity of the soil. This situation raises important questions of public health policy since radon gas is an extremely potent cancer-causing agent.

Mechanical aids have been incorporated into the architecture of the Elliot Lake homes in order to alleviate the problem. In some cases, pipes have been laid under the basement floor, and fans have been installed to blow most of the radon gas outside the house. In other cases, the basement floors and walls have been coated with a special sealant designed to prevent radon gas from getting into the house. However, the problem cannot be eliminated altogether, nor can it be controlled in a maintenance-free manner unless the homes are built without basements or in an altogether different location.

Recognizing the problem, the Province of Ontario has proposed a standard for an "acceptable level" of radon gas in newly built homes, following recommendations laid down by the Atomic Energy Control Board. In March of 1978, the Elliot Lake Environmental Assessment Board (which was established by an Order in Council to investigate the environmental implications of the proposed expansion plans) scheduled three days of special hearings in Elliot Lake to consider the question of radon gas in homes and the adequacy of the proposed government standards. The present paper is a summary of the evidence which I presented to the Board on March 10 and 13 on behalf of the United Steelworkers of America. Using data supplied by the Ontario Ministry of Housing, I argued that a 31% increase in the male lung cancer rate could result if the presently proposed standard for radon gas in homes is adopted. On the basis of this testimony, the Board recommended that the Province re-evaluate the radon gas standard which they are proposing. However, there is no indication that such a re-evaluation is taking place. The expansion is proceeding at an undiminished rate, and new homes are being built according to the very standard which is under question. Apparently, public health has to take a back seat to economic expediency.

I was personally shocked to discover that the Elliot Lake Environmental Assessment Board had no funds to call independent medical experts to testify on the biomedical effects of radon inhalation. Instead, the Board had to rely on those experts brought in by the mining companies and by the United Steelworkers of America at their own expense. This is not conducive to a balanced perspective on an important public health matter, since the financial resources are heavily biased in favour of the industry.*

The industry witnesses and the government witnesses all seemed to downplay the hazard to a remarkable extent. One witness who presented himself as an expert in cost-benefit analysis argued that the 156 extra lung cancer deaths which one might expect over the next 30 years as a result of radon exposure in Elliot Lake were of no great consequence, because

a) those people would have died anyway, from some other cause, if they hadn't died of cancer;

b) they would each die only one day sooner than they would have otherwise died, on the average (!!), and therefore,

c) the cost of the lost person-days, for these 156 cancer deaths, calculated at a rate of $30,000 per year, would only be about $12,000.

The stupidity of this calculation did not escape the Board. However, it is a shame that better testimony was not available.

This little story, which can be found in the transcript of the Elliot Lake Hearings for March 13, provides a grim illustration of biopolitics at work. Biopolitics is the dubious art of justifying whatever economic decisions have been made by arguing that the biological effects will be negligible.

It is my belief that the public health will only be protected when society is prepared to err on the side of safety rather than on the side of expediency.

Gordon Edwards
May 1978

* Note: In 1978, after this Introduction was written, the Elliot Lake Environmental Assessment Board did bring in Dr. Karl Morgan and Dr. Victor Archer to testify on radon hazards.

SUMMARY

What is the issue?

The Ontario Ministry of Housing, in cooperation with the Atomic Energy Control Board, is proposing a standard for an "acceptable" level of radon gas in homes and other buildings. According to data published by the Ministry, this proposal could result in a 30% increase in the incidence of lung cancer among the male occupants of such buildings. (Data on female risk figures is not available.)

What is radon and what does it do?

Radon is a naturally occurring radioactive gas. It is produced as an inevitable byproduct of the radioactive disintegration of uranium. Since uranium is found in small amounts almost everywhere on earth, radon gas is also found, in small amounts, almost everywhere. However, in places where uranium or its radioactive daughters are present in higher-than-usual concentrations, radon gas also occurs at higher concentrations -- for example, wherever there is uranium or phosphate ore, or the "tailings" left over from mining and processing such ores.

Radon gas has a half-life of 3.8 days, whereupon it produces other radioactive substances (which are solids) known as "radon daughters". These latter substances, the radon daughters, are mainly responsible for the high incidence of fatal lung cancer in uranium and other hard-rock miners. The radon daughters attach themselves to microscopic dust particles, which are then inhaled down into the deepest parts of the lung. Radon is much more harmful in a confined area such as a mine shaft or basement, since the radon daughters then have a chance to build up to higher concentrations, and thus deliver a higher dose of radiation to the lungs when inhaled.

How does radon get into buildings?

In 1967, in Grand Junction (Colorado), it was discovered that thousands of homes and other buildings had been built on uranium tailings, leading to high radon gas levels in homes, schools, and workplaces. In 1975, many homes in Port Hope (Ontario) were found to be constructed with radioactively contaminated material, creating similar problems. In 1976, hundreds of homes in Elliot Lake (Ontario) were found to have high radon levels indoors, presumably because of the higher-than-usual amounts of uranium in the soil. In 1977, some homes in Newfoundland were found to have been constructed using radioactive slag from a phosphate plant, leading to excess levels of radon gas indoors. Moreover, recent surveys have turned up "pockets" of homes having excess radon levels in almost every city in Canada.

In all these cases, the inhabitants of such buildings are being exposed to radon levels which are much higher than average. The question is: since radon cannot be totally eliminated, what is an "acceptable level" in terms of public health policy?

What are the proposed standards?

The Ontario Ministry of Housing, following criteria established by the Atomic Energy Control Board of Canada, is suggesting the following standards:

Radon Levels Inside Buildings (in working levels, WL)

Prompt Remedial Action :	over 0.15 WL
Acceptable:	under 0.02 WL
Requiring Investigation:	over 0.01 WL

Gamma Radiation Inside Buildings (in millirems per hour)

Prompt Remedial Action:	over 0.10 mr/hr
Acceptable:	under 0.05 mr/hr

(measured 1 metre above floor, centre of room)

The proposed acceptable limit for radon gas in buildings, 0.02 working levels, is expected to cause some additional cases of lung cancer over and above the natural incidence of this usually fatal disease. The present paper deals with the question of how much additional lung cancer can be expected if a sizeable population is exposed to such levels of radon in their homes, schools, and workplaces.

What are the expected public health consequences?

The Ministry of Housing does not expect that exposure to 0.02 WL of radon over a lifetime will cause more than a 5% increase in lung cancer among males. This conclusion is based on studies of uranium miners, most of whom suffered much higher exposures than those to be expected from radon gas in buildings.

However, recent scientific evidence from many countries indicates that at lower dose rates, radon is much more effective in causing cancer (per unit dose) than at higher dose rates. If this is so, then the official estimates of the health effects of living in a radon atmosphere of 0.02 WL are grossly understated, and we have the potential for a major public health tragedy. Some of this evidence is summarized in the ensuing paper, and the implications for public health are clearly explained.

Using only the data supplied by the Ontario Ministry of Housing to the Elliot Lake Environmental Assessment Board, it is shown that continuous exposure to 0.02 WL for 12 hours per day could lead to a whopping 31% increase in the incidence of lung cancer for males. It is therefore concluded that the housing standards have to be tightened up considerably.

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LIST OF EXHIBITS

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OR,

"IT'S PERFECTLY SAFE, BUT DON'T BREATHE TOO DEEPLY"

by Gordon Edwards

MATHEMATICAL MODELS

A mathematical model is a description of reality using mathematical language. Such a description or model can be simple (like a graph) or complicated (like a computer simulation). It is quite possible for such a description to be wrong, in that it does not give correct results when applied to reality, even though the internal mathematical logic is impeccably correct.

A very simple illustration of this principle can be provided using a familiar geometric example. It was once thought that the earth was flat -- not an unreasonable assumption, since the earth looks flat. "Geo-metry" literally means "earth measurement", and in ancient days it was believed that the results of elementary geometry accurately indicate what happens on the surface of the earth.

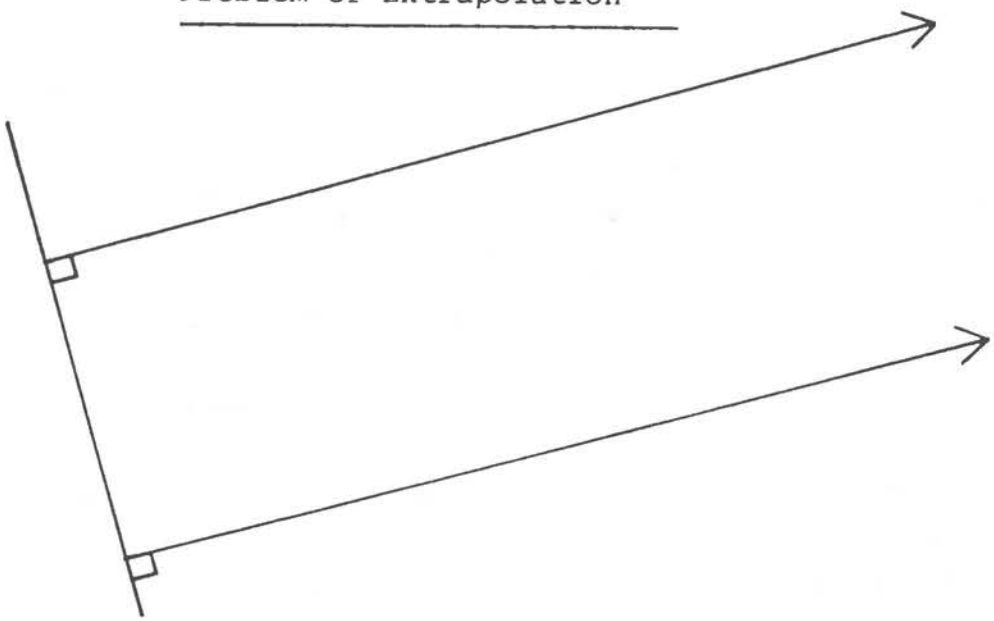
On a flat surface, two perpendiculars drawn from the same line will never intersect, no matter how far they may be extended (see Figure 1). But of course we now know that the earth is round, not flat, and so this conclusion about perpendiculars is wrong if it is applied to the surface of the earth. In fact, two perpendiculars drawn from the equator will intersect at the North Pole![1]

The fault is not in the mathematics; the fault is not in reality; the fault is in the mathematical model -- in other words the mathematical description of reality is not entirely correct.

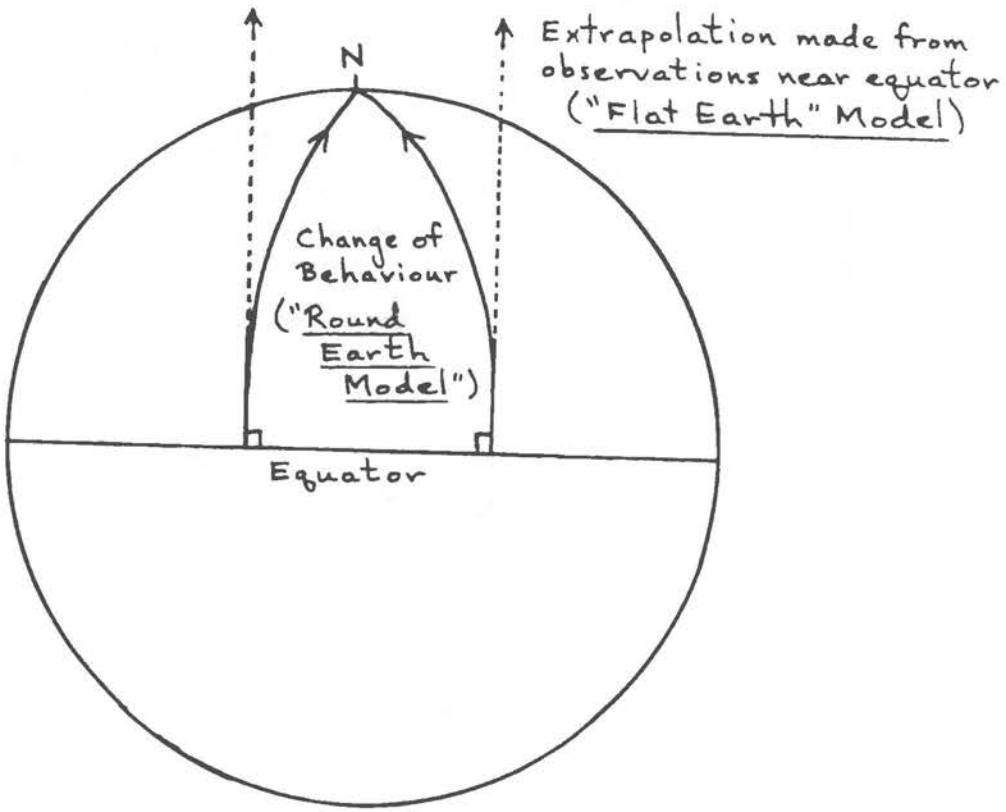
.../3

FIGURE 1

Geometric Illustration --
Problem of Extrapolation



On a flat surface, two perpendiculars drawn from the same line will never meet ...



... but on the surface of the earth, two perpendiculars drawn from the equator will meet at the North Pole.

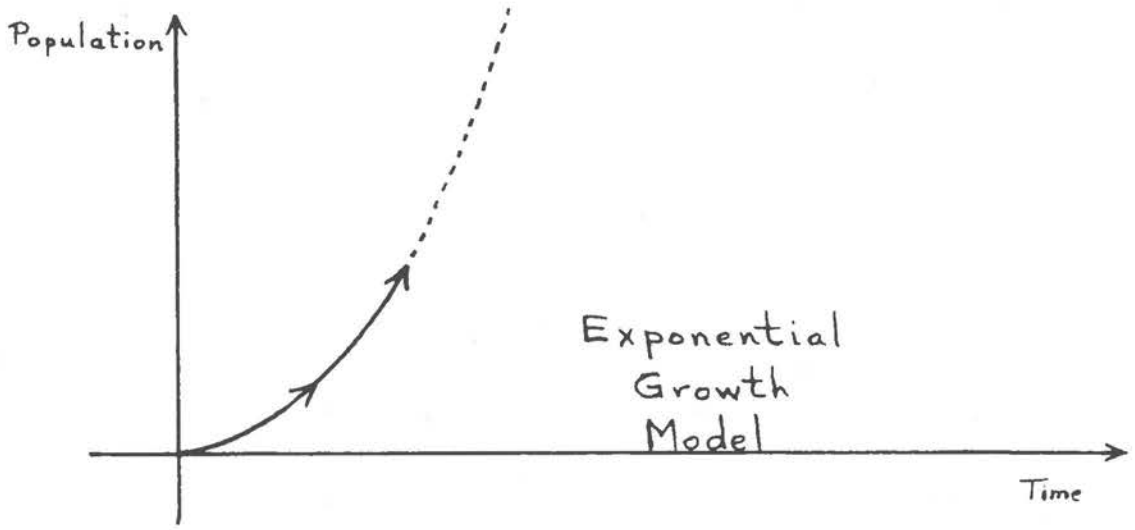
THE PROBLEM OF EXTRAPOLATION

The previous example illustrates a general problem in mathematical modelling, which is the problem of extrapolating from known results to unknown regions. The ancients who developed the principles of elementary geometry lived in a limited region of the earth (near the Mediterranean), and in that region the geometrical principle seemed to be true that two perpendiculars drawn from the same line do not intersect. The ancient thinkers had no way of knowing that the behaviour of the two lines would change thousands of miles away from where they started, and that the two lines would eventually intersect (if drawn on the surface of the earth) some 6,000 miles later!

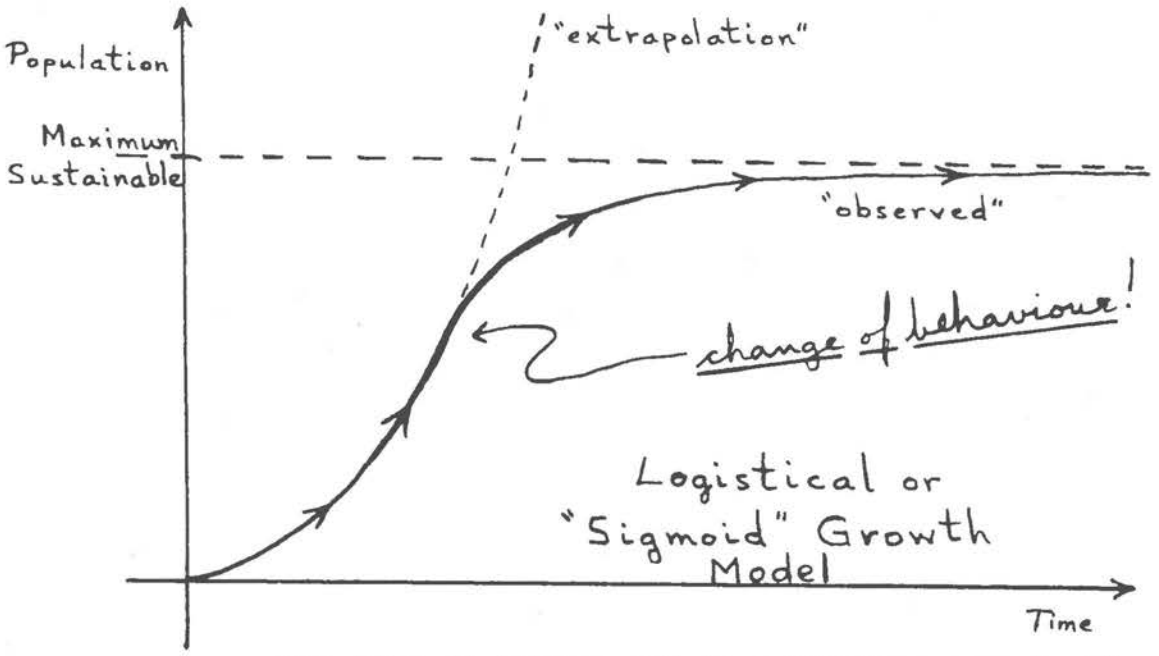
Another simple example, drawn this time from the field of biology, may clarify the problem of extrapolation still further. If a new species is introduced into an ecosystem, it spreads very rapidly, following an "exponential growth law" (see Figure 2). But this kind of rapid, accelerating growth cannot continue forever, and eventually, as the population grows, a levelling-off takes place as a result of new, previously unimportant factors -- competition for food, competition for nesting sites, increase of predators -- causing a marked change of behaviour. This change of behaviour would not be predicted by extrapolating from the initial observations made while the new species was still "young".[2] (The same does not apply to the growth of a single organism. It is true that if we all continued to grow as rapidly as children grow, we would be gargantuan in size by the time we become middle-aged. However, in that case the diminishing growth rate is observed even at the earliest stages, unlike the population model used here.)

.../5

FIGURE 2: Biological Illustration -- Problem of Extrapolation



When a species is "young", it grows very fast in numbers, following an "exponential growth" curve . . .



. . . but then it levels off to a maximum sustainable population which depends on the carrying capacity of the environment.

The two examples just given reveal how, if the mathematical model is wrong, there can be a very pronounced divergence between the expected results (results predicted by extrapolating from the model) and observed results (what actually happens in reality).

LIVING SYSTEMS VERSUS NON-LIVING SYSTEMS

Living systems are more complicated in their behaviour than non-living systems, and consequently they are harder to describe. For this reason, mathematical models have been much less successful in the biological and social sciences than they have been in the physical sciences.

In carefully engineered systems, a great many predictive mathematical models have been developed to a very high degree of precision and sophistication and there is a high degree of reliability in the accuracy of most of those models (even though they are sometimes found to be wrong).

In the biological and social sciences, this is by no means the case. Due to the complexity of living systems, the capacity for error is enormous, and the mathematical models which are used are wrong more often than not.[3]

This point was brought home to me very strongly when I worked for the Science Council of Canada as the Assistant Director of a nationwide study of the role of the Mathematical Sciences in Canada. The study examined the uses of mathematical modelling techniques in science, business, government, and industry, and concluded when living systems are involved that mathematical methods are very frequently misused, that mathematical models are very frequently in error, and that an undue reliance is placed in these models due to a lack of understanding of the problem of extrapolation.

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Ample evidence to this effect can be found in a volume entitled "MATHEMATICS IN TODAY'S WORLD" (reference 11) edited by myself and published by the Science Council of Canada, which contains the Proceedings of three one-day seminars held in Ottawa in 1974 dealing with:

- Mathematics and Policy Planning (March 4)
- Mathematics, Statistics, and the Environment (March 5)
- Mathematics, Science, and Technology (March 6)

Copies of these proceedings have been deposited in all university libraries in Canada.[4]

The use of mathematical models to describe living systems is still in its infancy, and there is much to be learned. In the meantime, extreme caution must be used in applying such models to real life situations.

THE LINEAR HYPOTHESIS AND ALPHA RADIATION

There is no doubt that exposure to radon gas and radon daughters causes lung cancer, at least for sufficiently high exposures.[5] This has been well established by studying the incidence of lung cancer in uranium miners and other hardrock miners. However, the precise nature of the relationship between lung cancer and radon exposure is extremely complicated, due to problems of measurement, lack of knowledge of the precise mix of radon daughters, the aggravating effect of dust, numerous biological factors affecting the latency period, synergistic effects with smoking and diesel fumes, and statistical uncertainties due to spontaneous fluctuations in the incidence of lung cancer. These complications are all well recognized (see for example the Ham Commission Report, reference 1).

In the early days, it was hoped that there was a "safe threshold" -- that is, a level of exposure to radon gas and its daughters below which no harm would be done and no extra lung cancers would be expected. However, experience has not supported the existence of such a safe threshold

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and no regulatory body in the world assumes that there is such a threshold. (Incidentally, this same "no-threshold" principle seems to apply also to cancer-causing agents other than radioactivity).[6]

There has been a great deal of good scientific evidence brought forward over the years to support the so-called "linear hypothesis" as a generally conservative principle for estimating the number of cancers produced by a given dose of radiation; this "linear hypothesis" has been adopted by Canadian regulatory bodies as a basis for estimating such health effects. The linear hypothesis states that the number of excess cancers per unit exposure is always the same, no matter what the total dose of radiation is and no matter whether the dose is delivered slowly or quickly. In other words, the number of excess cancers is proportional to the sum total of all the doses administered to the population.[7]

This linear hypothesis is the mathematical model which has been used in the MOH Report to estimate the number of excess lung cancers that might be expected as a result of radon exposure in homes in Elliot Lake. It is also the model which has been espoused by the Atomic Energy Control Board as a (hopefully) "conservative" model -- which means that the model (hopefully) overestimates rather than underestimates the actual numbers of lung cancers that would be expected at low doses.

In the case of X-rays, gamma rays, and beta rays, there is a wealth of experimental evidence published in the scientific literature which supports the contention that the linear hypothesis is conservative when extrapolations are made from high doses to low doses. To my knowledge, however, there is no such evidence published relating to low doses of alpha radiation,

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especially in the case of alpha radiation to the lungs.[8] Rather, as we shall see, there is much evidence pointing to the opposite conclusion.

There are basically only two reasons for assuming that the linear hypothesis is conservative in estimating lung cancers at low doses of radon exposure:

1) at high exposures the epidemiological evidence from miners exposed to radon is reasonably consistent with the linear hypothesis;

2) for other types of radiation (other than alpha radiation) and various types of cancer (including lung cancer) the linear hypothesis seems to be conservative at low exposures.

But, in extrapolating from high doses to low doses of alpha radiation we encounter the classical problem of extrapolation of mathematical models from known results into unknown regions.

MATHEMATICAL MODELS OF RADIATION CARCINOGENESIS

On May 4 1976, at a Congressional Seminar on Low Level Ionizing Radiation held in Washington, D.C., the Chairman, Dr. Karl Morgan[9] stated that the number of cancers (R = response) resulting from a given exposure to radiation (D = dose) seems to follow the mathematical relationship

$R = cD^n$ (where c and n are constants which depend on the type of radiation and the organ exposed)

Exhibits 1 and 2 on the following pages provide some additional information about the Congressional Seminar.

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EXHIBIT 1

CONGRESSIONAL SEMINAR
ON LOW LEVEL IONIZING RADIATION
May 4, 1976
EXCERPT FROM PROCEEDINGS (reference 2)

For definition of terms, see glossary on page 45; for explanation of $R = cD^N$, see Figure 3 on page 12; for more information, see "Suggested Reduction of Permissible Exposure to Plutonium & Other Transuranium Elements", American Industrial Hygiene Assoc. Journal, v. 36, Aug 1975, 567-575, by Karl Z. Morgan

Chapter IV

Dose-Response Relationships, Linear or Non-Linear?

Dr. Morgan: Let us go on to the next question.

Question 3: Do these effects that we are talking about increase linearly with increasing dose?

I believe data suggests that the cancer risk can in a simple way be expressed by an equation such as, R, the risks, equal a constant, C, times the accumulated dose, D to some power, N in other words:

$$R = CD^N$$

In the case of low LET radiation, for example, X, gamma and beta radiation, the accumulated dose, D, must be corrected for repair of damage over time, as pointed out by Dr. Bond in which it does appear in most cases that N is equal to or greater than 1, suggesting the greater efficiency of multiple hits.

In the case of high LET radiation, however, such as alpha and fast neutrons, there seems to be little or no repair and best fit curves are obtained when N is less than 1, indicating the damage per rem is greater at lower doses.

Dr. Baum (J. Baum, Health Physics Society, Houston, Texas, 1974) and many others have shown that in the case of human exposure to radium the best curve fit for cancer induction is when N is equal to ½. Thus, for high LET radiations, such as those from plutonium 239, the linear hypothesis underestimates the risk.

In a recent paper, I gave five reasons why the linear hypothesis as now applied is nonconservative. I might summarize as follows:

1. Extrapolations are often made to zero of effects on animals and man, and they are sometimes extrapolated from the high dose descending portion of the parabolic curve where there would be overkill.

2. Estimates are made from exposures to animals of short life spans, and for a man (as pointed out in the BEIR Report), out to only about twenty years. Of course, other data over the remainder of man's life would have to increase the slopes of these curves or the risks per rem.

3. A uniform population is usually assumed taking little account of the age distribution and the disease patterns, as Dr. Bross has pointed out.

4. There is cell sterilization at the higher doses and so it is somewhat risky to extrapolate from these doses because you would underestimate the risk at low doses.

5. I think quite important is the fact that the recent data from Drs. C.W. Mays and H. Spiess on radium 224, a bone seeker like plutonium and other actinide elements, indicate that the cancer risk increases with protraction of the dose. This is just the opposite of what we have observed from low LET radiation.

In summary, I would state that it is my opinion that the linear hypothesis is always nonconservative for high LET radiations. Usually it is nonconservative for in utero exposure of children to low or high LET radiations, but in some cases of adult exposure, it is probably conservative for low LET radiation. *

I am sure I have provoked a lot of discussion.

Dr. Morgan: Dr. Sternglass.

Dr. Sternglass: I would like to say that at the recent hearings by the EPA on radiation standards for the nuclear fuel cycles I presented evidence obtained by many people in the literature that at the very low dose rates that we are talking about, we are dealing predominantly with a different biological mechanism than we are dealing with at the high dose rates.

The recent data by Dr. Petkau show that as the dose rate decreases, it takes less and less dose to break a cell membrane. This evidence was not available at the time of the BEIR Report.

What it means is simply this, that for somatic, not genetic situations, we are now faced with a whole new problem, namely the fact that when cell membranes are injured as a result of indirect chemical effects the data of Dr. Petkau both for free membranes and his new data on micro-organisms and mice, show clearly that the lower the dose rate is, the less it takes to break a membrane.

As a result, one is led to a non-linear effect at low doses, which is opposite to what we had expected in the past. In fact, the curve of response versus dose goes up much more rapidly at the origin to the degree that this leads to an under-estimate of biological effects of very low doses using a linear extrapolation of something like a few hundred, possibly as much as a thousand-fold.

Now recent studies have shown that membranes are involved in the functioning of the immune system of the body. One of the most important things about the immune system is that it not only defends the body against viruses and bacteria, but we now know from recent evidence published in the last few years, that the immune system also detects and controls cancer cells.

Thus, we are now faced with the evidence that cell membrane damage is possibly the controlling one in cancer induction at low dose rates, while at high dose rates, the controlling process seems to be direct damage to the DNA. *

This means that we now have a mechanism that we did not have before that can explain not only the very large increases in infant mortality, but also the changes in heart disease and cancer all over the world following the period of nuclear testing.

EXHIBIT 2

Congressional Seminar on Low Level Ionizing Radiation - May 4 1976

List of Participants

Karl Z. Morgan, Professor of Health Physics, Georgia Institute of Technology, CHAIRPERSON.

John T. Edsall, Professor Emeritus of Biochemistry, Harvard University.

Irwin Bross, Director of Biostatistics, Roswell Park Memorial Institute for Cancer Research, Buffalo.

Rosalie Bertell, Research Associate, Roswell Park Memorial Institute for Cancer Research, Buffalo.

Victor Archer, Medical Director of U.S. Public Health Services, National Institute for Occupational Safety and Health, Salt Lake City, Utah.

Seymour Jablon, Associate Director, Medical Follow-up Agency of the National Research Council; Staff Officer, Radiation Effects Research Foundation; formerly Chief of Epidemiology, Atomic Bomb Casualty Commission.

Edward Martell, National Centre for Atmospheric Research, Boulder, Colorado; formerly Advisor on High Altitude Fallout, Strategic Air Command.

Victor Bond, Associate Director in Life Sciences, Brookhaven National Laboratory; formerly on the National Academy of Sciences Advisory Committee on the Biolgical Effects of Ionizing Radiation.

Ernest Sternglass, Professor of Radiology, University of Pittsburgh.

Charles Richmond, Associate Director, Biomedical and Environmental Sciences, Oak Ridge National Laboratory.

William Ellett, Criteria and Standards Division, Environmental Protection Agency, Office of Radiation Programs.

Bernard Shleien, Office of Medical Affairs of the Bureau of Radiological Health, Food and Drug Administration.

Mark Barnett, Associate Director, Division of Training and Medical Applications, Bureau of Radiological Health.

Roger Mattson, Director, Division of Siting, Health, and Safeguards Standards, Nuclear Regulatory Commission.

Helen Caldicott, Researcher in Cystic Fibrosis, Boston Clinic; formerly Director of Cystic Fibrosis Research, Adelaide Children's Hospital, Adelaide, Australia.

In addition, there were several representatives from the trade union movement; among the invited participants who could not attend were John Gofman, Professor Emeritus of Medical Physics, University of California at Berkeley; and Bernard Cohen, Director, Nuclear Physics Laboratory, University of Pittsburgh.

The conference met at 9:35 a.m. on May 4 1976 in room 1202, Dirksen Senate Office Building, with Senator Gary Hart presiding.

I have studied the literature quite extensively and can testify that most mathematical models proposed to explain the carcinogenic nature of radiation at low doses do fall into the category described above -- except those theories which assume a "safe threshold", and which are quite unfashionable at the present time (as stated by Dr. Howard Newcombe, an eminent radiation biologist employed by AECL, during cross-examination at the Porter Commission on Electric Power Planning on January 19, 1978).[10]

THREE SITUATIONS DESCRIBED BY THE EQUATION $R = CD^n$

(see Figure 3)

If n equals 1, the above equation produces a straight-line graph which corresponds to the linear hypothesis.

If n is greater than 1, the corresponding graph "scoops upward". In this case the linear hypothesis is conservative — it overestimates the actual harm at low doses.

If n is smaller than 1 the graph "scoops downward" and the linear hypothesis is non-conservative: it tends to underestimate the actual number of cancer deaths which will result from low cumulative doses of radiation.

For the sake of completeness, I should point out that some of the mathematical models proposed to explain radiation carcinogenesis are composites of two of these three cases. For example, some have suggested that for external irradiation, n = 2 might be appropriate for low doses and n = 1 might be appropriate for high doses, yielding a parabolic upward-bending curve with a "linear tail" (see Figure 4). On the other hand, if n = ½ were appropriate for low doses and n = 1 for high doses, you would have a downward-bending parabola with a linear tail (see Figure 4). The first composite would make the linear hypothesis conservative, while the second composite would make the linear hypothesis non-conservative at low doses.

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FIGURE 3: Models of Radiation Carcinogenesis at Low Doses

The General Formula is $R = cD^n$, where R = number of tumors, c = constant of proportionality, D = accumulated dose of radiation, and n = constant exponent. (Proceedings, page 20).

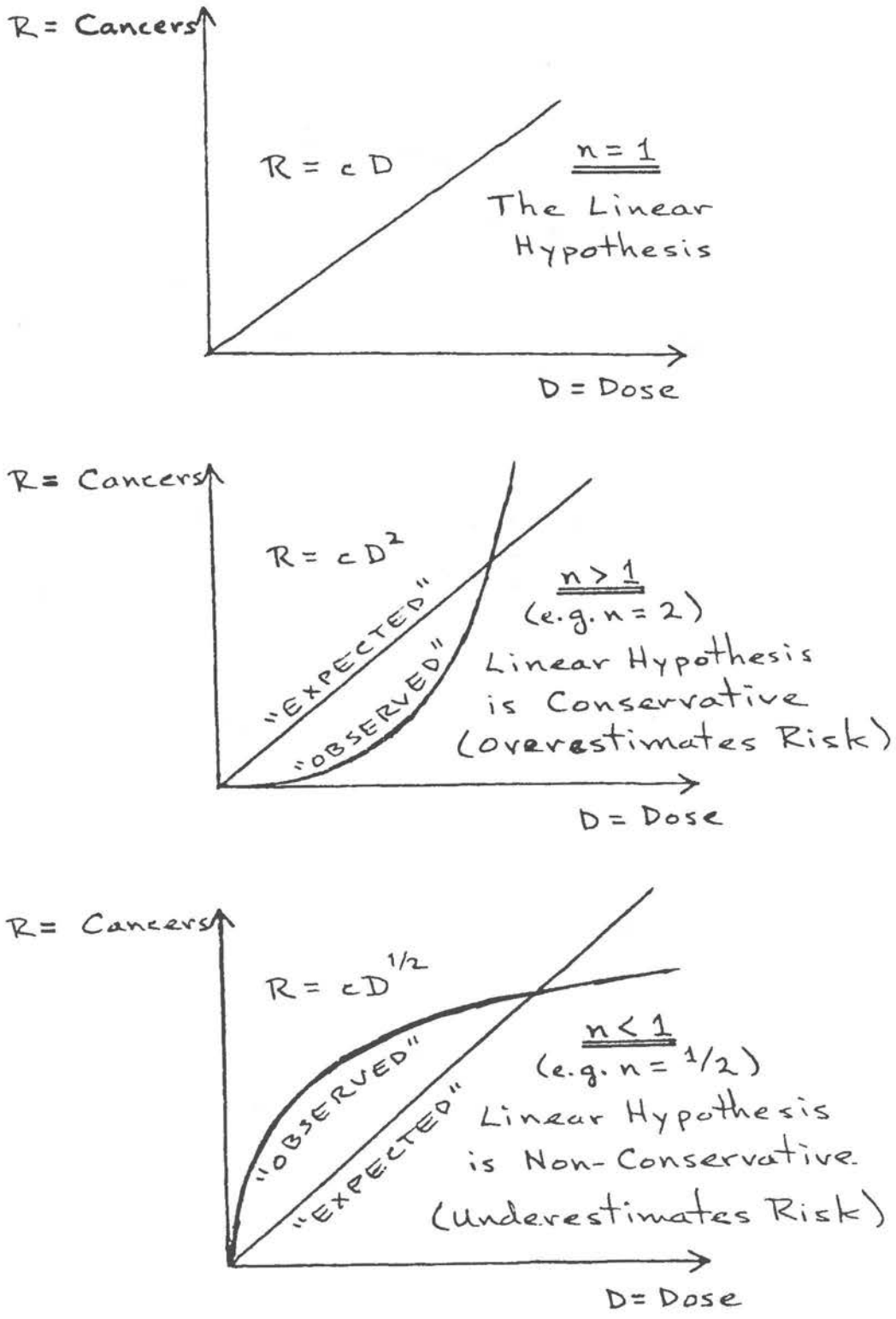
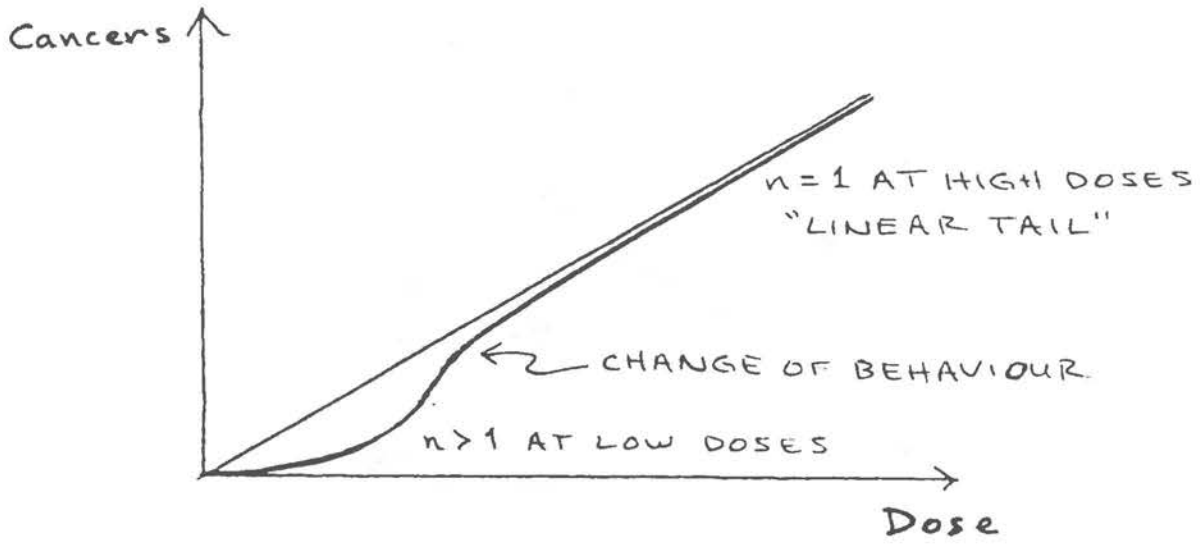
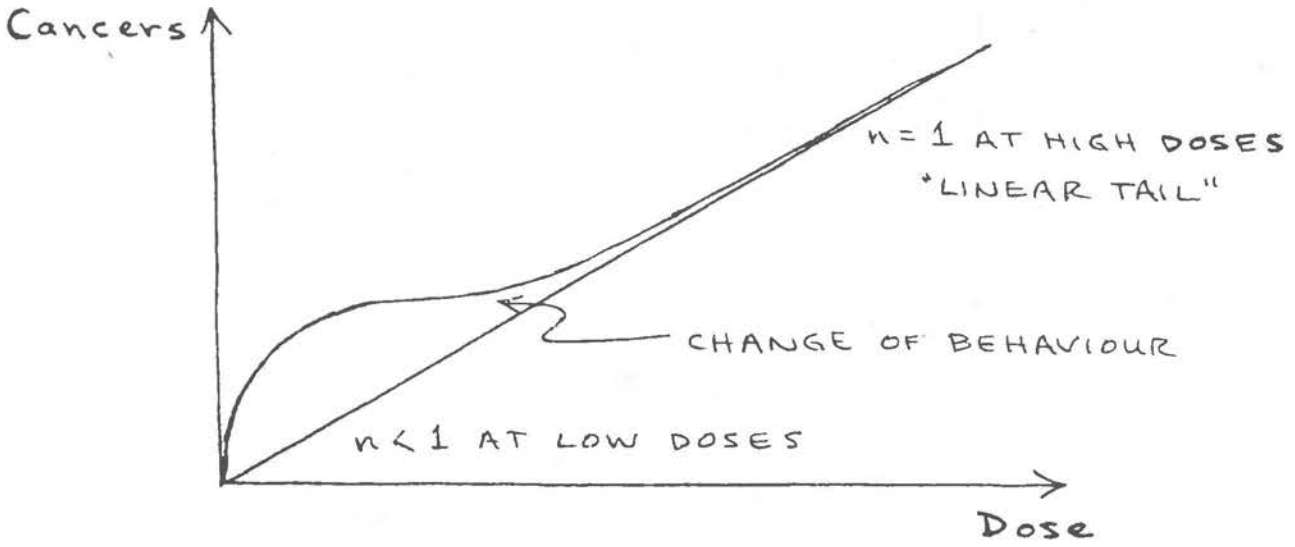


FIGURE 4: Composite Models of Radiation Carcinogenesis



Linear hypothesis agrees with observed risk at high doses, but overestimates risk at low doses.



Linear hypothesis agrees with observed risk at high doses, but underestimates risk at low doses.

ALPHA RADIATION AT LOW DOSES: EXPERIMENTAL RESULTS

In order to test the linear hypothesis at low doses of alpha radiation, additional data is needed to see if extrapolation from high doses is appropriate or not. It has long been recognized that alpha radiation poses a different kind of radiation hazard than X-rays, gamma rays, or beta rays.

a) For one thing, alpha radiation has very little penetrating power (it cannot penetrate a sheet of paper) and so it is not a hazard unless alpha-emitting substances are ingested or inhaled into the body, and then the exact distribution of such substances within the body is not completely known.[11]

b) Moreover, low doses of alpha radiation are usually delivered slowly over a period of time. This fact makes it very difficult to measure the exact accumulated dose of alpha radiation that is delivered to living tissues, especially when the dose is small.[12]

c) It is also well known that alpha radiation is extraordinarily effective in causing cancer -- so much so that a given amount of alpha radiation is about 20 times as effective as the same amount of X-radiation, gamma radiation, or beta radiation in causing cancer. That is why Dr. Muller uses a "quality factor" of 20 for alpha radiation (p. 5, line 1, Appendix MOH Report) and a "quality factor" of 1 for gamma radiation (p.6, bottom line, Appendix MOH Report).[13]

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Until recent years, there has been almost no data published in the scientific journals dealing explicitly with the cancer-causing ability of alpha radiation at low doses. (As indicated on p.5 of the MOH Report, with an erroneous conclusion that this lack of evidence establishes conservatism).[19] In the last five years, however, numerous papers have appeared which indicate that at low doses, the linear hypothesis may seriously underestimate the cancer risk from alpha radiation.[14] These results are discussed in the Proceedings of a Congressional Seminar on Low Level Ionizing Radiation (reference 2; for a sample of the text, see exhibit 1 on page 9). As already noted, the Seminar was held on May 4, 1976 under the chairmanship of Dr. Karl Morgan, a very prominent and well respected figure in the field of Health Physics. The other participants in the seminar are listed in exhibit 2, p. 10. (For background information on Dr. Morgan, Dr. Archer, Dr. Gofman, and other scientists referred to in this paper, see the Biographical Notes on page 38.)

According to the Introduction to the Proceedings:

> "The meeting was aimed at informing Congressional members and their staff of recent evidence indicating greater than expected health effects from low dose rates of ionizing radiation. . . . Central to the discussion was the contention that the established method of calculating dose effects from ionizing radiation is non-conservative."[15]

The evidence presented on low level alpha radiation is summarized graphically in Figure 5. It clearly suggests that at low doses, the linear hypothesis is non-conservative for alpha radiation.

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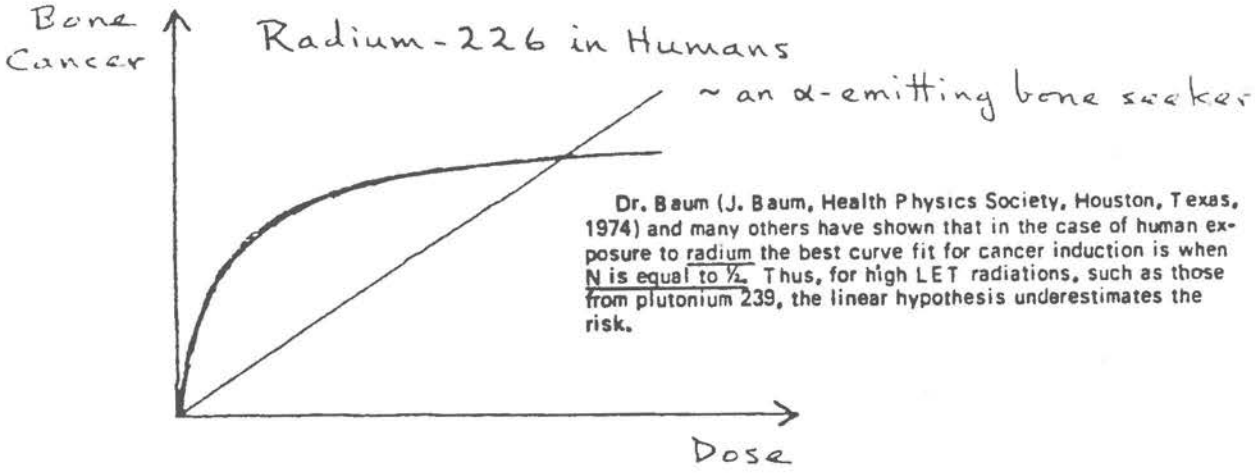
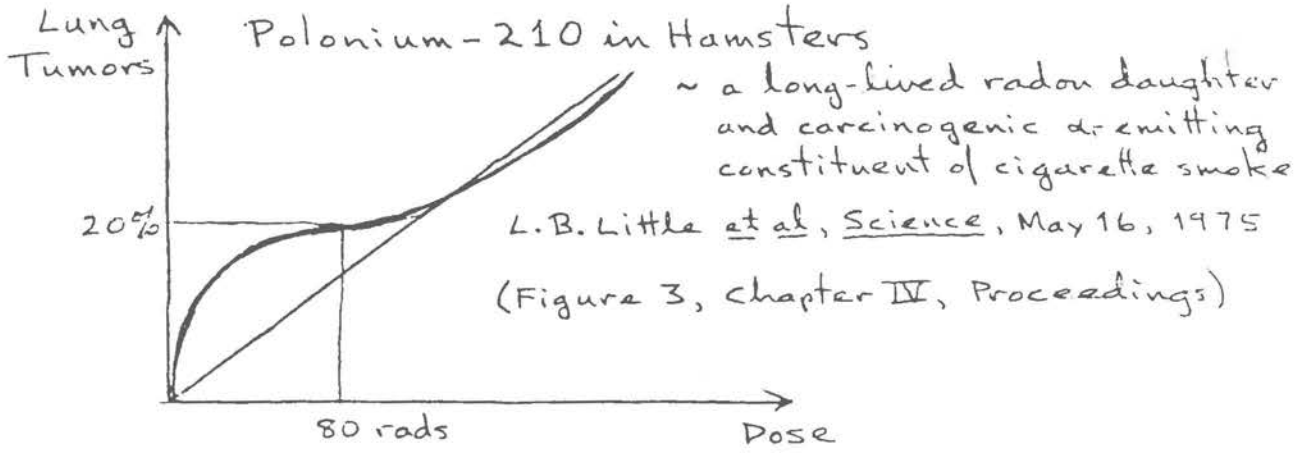
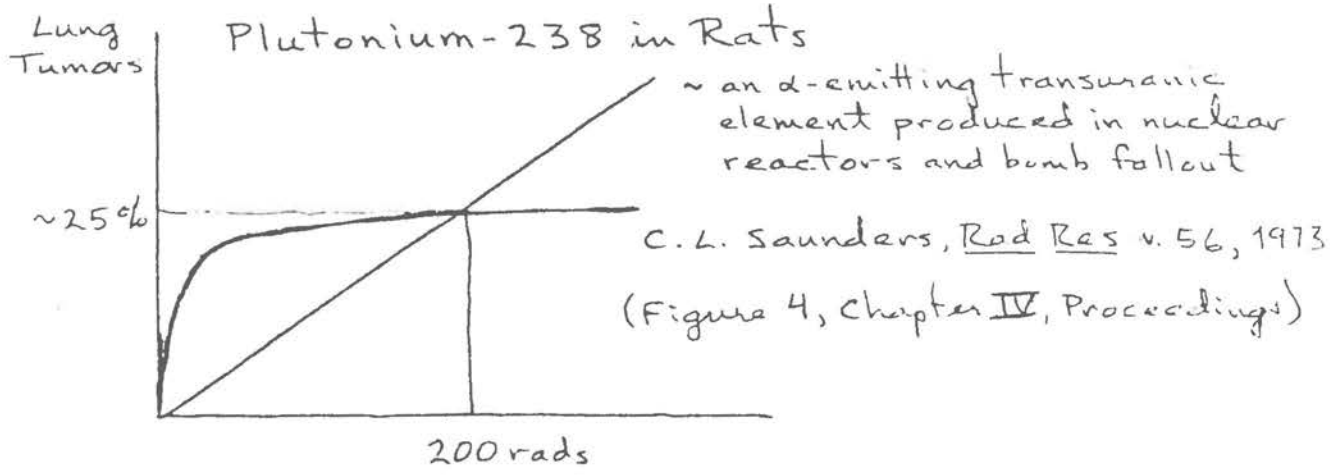
FIGURE 5: Evidence on Alpha-Induced Tumors at Low Doses

The evidence given here indicates that alpha radiation is more effective in producing cancer (per unit dose) at low doses than it is at high doses -- unlike X-rays, gamma rays, and beta rays.







There may be published evidence in the scientific literature dealing with low level alpha radiation which would suggest a different conclusion. I am unaware of any such evidence, however, although I have been searching for such evidence for the last six months. For example, on January 19, 1978 I cross-examined Dr. Howard Newcombe on this subject at the Royal Commission on Electric Power Planning in Toronto, and he was unable to cite any evidence of a contrary nature.[16] Dr. Newcombe is one of the most esteemed radiation biologists in Canada. He is currently a member of the International Commission on Radiological Protection and has served on the Advisory Committee on the Biological Effects of Ionizing Radiation for the U.S. National Academy of Sciences.

RADON EXPOSURE AT LOW DOSES: EPIDEMIOLOGICAL RESULTS

All epidemiological evidence dealing with lung cancers resulting from radon exposure points away from the existence of a "safe threshold", and towards the conclusion that the linear hypothesis is non-conservative at low doses. Regarding the concept of a safe threshold, the Ham Commission concluded:

> "Since the Commission's Study of data based on the Ontario Uranium Nominal Roll provides no evidence supporting the hypothesis of a threshold of exposure below which there is not significant excess risk, the concept of a maximum safe exposure is not tenable on the basis of these data." (p.95, reference 1)

Dr. Victor Archer, M.D., Medical Director at the U.S. National Institute for Occupational Safety and Health, has recently reviewed the epidemiological

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EXHIBIT 3

ABSTRACT

Archer, V.E., Radford, E.P., and Axelson, O. Radon Daughter Cancer in Man: Factors in Exposure-response Relationships. Radiat. Res.

Lung cancer among fifteen different mining groups exposed to radiation from radon daughters was analyzed to determine what factors influence incidence and induction-latent period. As the exposure rate decreases, cancers per unit of radiation increases. The induction-latent period is shortened by increased age start of mining, by cigaret smoking, and by high exposure rates. For followup periods of 20-25 years, the incidence increases with age at start of mining, with magnitude of exposure, and with amount of cigaret smoking. For very long followup periods, the incidence among nonsmokers sometimes exceeds that among smokers. Both lung cancers/yr/WLM and relative risk were found to vary greatly with exposure rate, age of cohort at start of mining and with length of followup period. Lifetime risk/WLM, adjusted for exposure rate, was proposed as the best statistic for use in predicting lung cancers among other groups exposed to radon daughters. These findings are consistent with the theory of radiation carcinogenesis which postulates that cancer is caused by a series of changes in chromosomal proteins (some of which occur with increasing age) followed by a promoting factor.

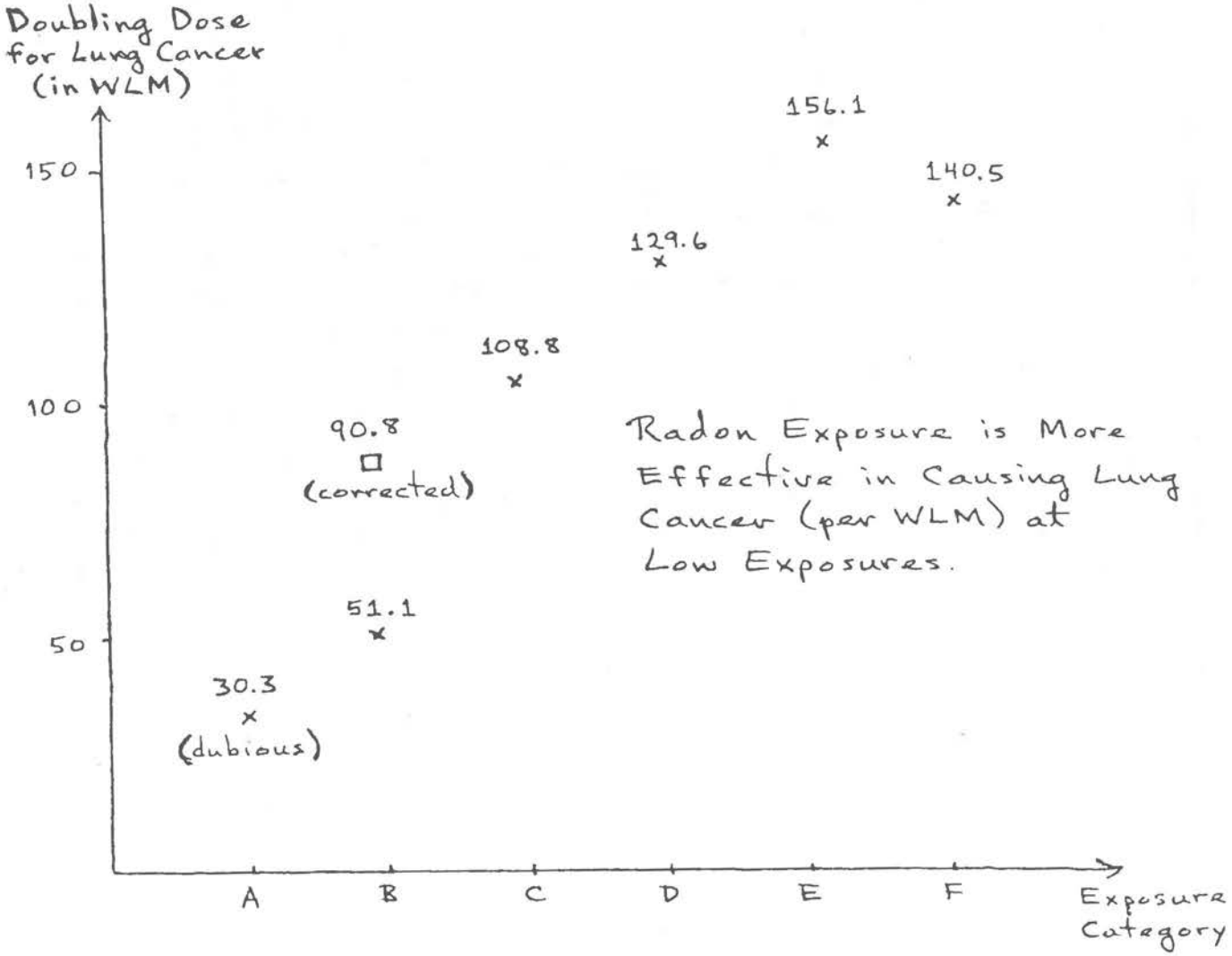
Key words: Radiation, radon daughters, miners, lung cancer, carcinogenesis.

FIGURE 6: Graphical Summary of Gofman's Calculations (reference 4)

For Uranium Miners on the Colorado Plateau, the natural incidence of lung cancers is doubled with fewer accumulated WLM at low exposures than at higher exposures to radon. Exposure categories are:

F: total population of 1981 miners with 49 lung cancers
E: miners exposed to less than 3719 WLM (37 cancers)
D: miners exposed to less than 1799 WLM (27 cancers)
C: miners exposed to less than 839 WLM (16 cancers)
B: miners exposed to less than 359 WLM (11 cancers)
A: miners exposed to less than 120 WLM (4 cancers)

Category A is of dubious significance because of so few cancers.
Category B was corrected for possible additional radon exposure due to previous hardrock mining experience.



evidence for fifteen different groups of uranium miners, and has concluded that the linear hypothesis seriously underestimates the risk of cancer at low doses in every single case. In other words, the existing epidemiological evidence on uranium miners fully supports the evidence mentioned earlier about alpha-induced cancer at low doses. Dr. Archer has only reached this conclusion in the last two or three years, although he has twenty years experience in the field of lung cancer epidemiology for uranium miners.[17] (Archer et al,ref 3; see exhibit 3, page 18)

In fact the epidemiological evidence has always been there, but until recently it was ignored because it did not conform to the linear hypothesis. In 1970, for example, Gofman & Tamplin published a paper reviewing the evidence of lung cancer incidence among uranium and hardrock miners who began working on the Colorado Plateau before 1955. Dr. Gofman's arithmetic, using data provided by the U.S. Federal Radiation Council, clearly demonstrated the increasing effectiveness of radon exposure at low doses in causing lung cancer. His calculations are graphically presented in Figure 6 on page 19 (based on reference 4).[18]

MINISTRY OF HOUSING DATA

Let us now turn to the data supplied by Dr. Muller in reference 5. An examination of Dr. Muller's first four tables -- reproduced on the following pages -- confirms Dr. Archer's observations: in each case, the greatest risk occurs at the lowest exposures. In each table, the first and last columns are the important ones to look at; the first column gives the degree of exposure to radiation and the last column gives the number of radiation-caused cancers expected per unit dose at that exposure level. In Table 2 (exhibit 5 on page 22), dealing with the Colorado Plateau data, a marked increase in excess cancers per WLM is observed at exposures below 359 WLM (Gofman's categories A and B).

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EXHIBIT 4

This table is based on data from the Colorado Plateau - c.f. figure 6, page 19.

MOH TABLE 1

ABSOLUTE RISK FACTORS FIVE AND MORE YEARS AFTER START OF URANIUM MINING

Exposure Group (WLM)	Mean Exposure of Group (WLM)	Pulmonary Cancer O*	Pulmonary Cancer E*	O-E*	Person-years at Risk (PYR)	$\frac{O-E^*}{PYR}$	$WLM_i \times PYR$	Absolute Risk Factor (Excess cases per WLM per million men per year).
< 120	60	5	1.84	3.16	5530.11	5.71×10^{-4}	3.32×10^{5}	9.5
120- 359	240	9	1.99	7.01	6225.32	1.13×10^{-3}	1.49×10^{6}	4.7
360- 839	600	13	2.52	10.48	7006.03	1.50×10^{-3}	4.20×10^{6}	2.5
840-1799	1,320	11	2.26	8.74	5730.88	1.53×10^{-3}	7.56×10^{6}	1.2
1800-3719	2,760	20	1.27	18.73	3131.09	5.98×10^{-3}	8.64×10^{6}	2.2
>3720	4,000	10	0.41	9.59	901.38	1.06×10^{-2}	3.61×10^{6}	2.7
Total	900	68	10.29	57.71	28524.81	2.02×10^{-3}	2.58×10^{7}	2.2

average 2.15

Notes

1) The data is taken from Radon Daughter Exposure and Respiratory Cancer, Quantitative and Temporal Aspects, Joint Monograph No. 1 (1971), Nat'l Institute for Occupational Safety & Health and Nat'l Institute of Environmental Health Sciences.

*2) In the table, O = observed cancers and E = expected cancers; because of the small number of cancers observed in the lowest category (below 120 WLM), the risk factor is more dubious than other entries in the table. With longer followup, however, it can only get worse, not better.

3) The Absolute Risk Model, used here, compares the excess cancers with the entire population exposed, on the assumption that radiation causes proportional increases in the absolute cancer rate.

4) The first column and the last column are the important ones to study; notice that the overall average of 2.2 excess cancers per WLM per million men tends to ignore the low-exposure data.

EXHIBIT 5

MOH TABLE 2

RELATIVE RISK FACTORS FIVE AND MORE YEARS AFTER THE START OF URANIUM MINING

Exposure Group (WLM)	Mean Exposure (WLM)	Pulmonary Cancer O*	Pulmonary Cancer E*	Mean Exposure x E	O-E* / E	Relative Risk factor (Excess lung cancer deaths per WLM per 1,000 lung cancer deaths expected.)
< 120	60	5	1.84	1.1×10^2	1.72	29
120 - 359	240	9	1.99	4.8×10^2	3.52	15
360 - 839	600	13	2.52	1.5×10^3	4.16	7
840 -1799	1320	11	2.26	3.0×10^3	3.87	3
1800 -3719	2760	20	1.27	3.5×10^3	14.75	5
>3720	4000	10	0.41	1.6×10^3	23.39	6
Total	990	68	10.29	1.0×10^4	5.61	5.7

average 5.25

Notes

1) The raw data for this table is exactly the same as the data for table 1, dealing with the Colorado Plateau miners.

* 2) See note 2 from table 1; the same observation applies here.

3) The Relative Risk Model used here compares the excess cancers with the "normal" (or expected) incidence of cancer, on the assumption that radiation causes proportional increases in the relative cancer rate. The excess, O-E, is compared with the expected, E, rather than the total population PYR, as in table 1. The Relative Risk Model is more often used than the Absolute Risk Model -- see tables 3 and 4 on pages 23 and 24, for example.

4) The first column and the last column are the important ones to study; note that the overall average of 5.7 excess cancers per WLM per 1000 cancers expected tends to ignore the low-exposure data.

EXHIBIT 6

MOH TABLE 3

RELATIVE RISK FACTORS DERIVED FROM URANIUM MINES IN CZECHOSLOVAKIA

WLM Category	WLM per Miner	Frequency of Lung Cancer per 1,000 miners. E	O	O-E / E	Relative Risk (excess cases per 1,000 expected lung cancer cases per WLM).
< 50	39	16.6	33.2	1.0	26
50 - 99	80	13.2	21.2	0.6	8
100 - 149	124	13.8	34.0	1.5	12
150 - 199	174	15.2	69.8	3.5	21
200 - 299	242	15.7	76.3	3.9	16
300 - 399	343	17.4	102.3	4.9	14
400 - 599	488	16.5	117.9	6.2	13
> 600	716	17.2	138.9	7.1	10
Total	309	15.1	65.6	3.3	11

average 13.4

Notes

1) The data for this table is taken from "Lung Cancer in Uranium Miners and Long-Term Exposure to Radon Daughter Products" by J. Sevc and E. Kunz, Health Physics v. 30, 433-437, 1976.

2) Notice that these miners received much less of an accumulated dose than the Colorado Plateau miners; most of the entries in this table fit into the first three categories in tables 1 and 2.

3) In this table, the overall average of 11 excess cancers per WLM per 1000 cancers expected is almost twice the overall average risk factor from table 2, reflecting the higher risk per WLM among lower exposure groups.

4) However, the average risk factor of 11 given here does not reflect the low-exposure data given in the table (below 50 WLM).

5) In the last column, the factor of 2 difference between the first entry and the average of the other entries in the same column should be compared with the factor of 2 difference between the first two entries in table 2.

EXHIBIT 7

MOH TABLE 4

RELATIVE RISK FACTORS DERIVED FROM PERSONS EXPOSED IN HIROSHIMA

Mean Dose Equivalent (Q) per person (rem.)	O	E	Q x E	Relative Risk Factor (Excess lung cancer deaths per rem per 1000 lung cancer deaths expected)
61.3	49	34.4	2108.72	average 4.5 { 7
190.2	13	9.0	1712.70	2
413.0	10	5.8	2395.40	average 1.5 { 2 } average 1.7
1213.1	8	4.4	5337.64	1
215.6	80	53.6	11554.46	2.3

Notes

1) The data for this table is taken from Sources and Effects of Ionizing Radiation, United Nations Scientific Committee on the Effects of Ionizing Radiation, 1977 Report to the U.N. General Assembly, with Annexes.

2) Using 1 WLM = 4 rem (a very conservative conversion factor), we see that the first four categories here correspond to 15 WLM, 48 WLM, 103 WLM, and 303 WLM.

   Using 1 WLM = 5 rem (the factor suggested by the U.S. National Academy of Sciences), we see that the first four categories here correspond to 12 WLM, 38 WLM, 83 WLM, and 243 WLM. These are certainly low exposures, in the context of uranium mining.

3) There is a tripling between the last two risk factors and the first two risk factors, and there is a quadrupling between the last three risk factors and the first one. It seems that the extra risk per WLM becomes ever more pronounced as the exposure gets progressively lower.

4) To convert the risk factors in the last column to risk factors per WLM, multiply each entry by the appropriate conversion factor (e.g. if 1 WLM = 4 rem, then multiply by 4; if 1 WLM = 5 rem, multiply by 5). The Ham Commission Report states that typical conversion factors are 1 WLM = 5-6 rems (reference 1, page 116).

The last column of Table 2 tells the story: the average of the last four entries in this column is 5.25 excess cancers per WLM, but the second entry shows that the number of excess cancers per WLM is almost three times larger for exposures between 120 and 359 WLM, and the first entry shows that the cancer risk is almost six times larger for exposures between 0 and 120 WLM! The overall average risk of 5.7 excess lung cancers per WLM (given at the bottom of Table 2) greatly underestimates the risk for those exposed to less than 120 WLM.

Similar observations can be made about Table 3 (exhibit 6 on page 23) dealing with Czechoslovakian data. Notice first of all that the exposures in this table are in the range from 0 to 600 WLM for the most part, corresponding to only the first two or three entries in Table 2. In other words, the Czechoslovakian miners received considerably less exposure to radiation than the American miners. And, sure enough, the average risk of 11 excess lung cancers per WLM in Table 3 is twice the average of 5.7 from Table 2, thus confirming once more that lower exposures correspond to larger risks per unit dose.

Moreover, within Table 3 itself, the number of excess cancers for exposures below 50 WLM is twice the average number of excess cancers from 50 WLM to 600 WLM, in full agreement with the doubling indicated between the first two entries of Table 2. Once again, in Table 3, the overall average of 11 excess cancers per WLM seriously underestimates the risk for those with low exposures (in this case, those with less than 50 WLM).

The same relationships hold in Table 4 (exhibit 7 on page 24) which is based on data from Hiroshima. Using the correspondence 1 WLM = 4 rem to the lungs (slightly more conservative than Dr. Muller's 4.42 rem given on page 5 of Appendix 1, MOH Report), we see that the first two entries in Table 4 fall in the "below 50 rem" range, while the second two entries lie between 50 and 300 WLM equivalent exposure. In this table, the average of the first two entries (4.5 excess cancers/rem) is triple the average of the last two entries (1.5 excess lung cancers/rem). Moreover, the first entry (7 excess cancers/rem) is four times as large as the average of the other three entries (1.7 excess cancers/rem).

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Thus, the Ministry of Housing data, assembled by Dr. Muller, is entirely consistent with the evidence cited earlier which suggests that the linear hypothesis seriously underestimates the risk of lung cancer at low exposures to radon. In fact, the relative risk seems to get consistently worse as the exposures get progressively smaller. All of this evidence points away from a safe threshold and away from the linear hypothesis, contrary to what is stated on page 5 of the MOH Report.[19]

INTERPRETATION OF M.O.H. ESTIMATES

The Ministry of Housing is recommending a standard of 0.02 WL of radon in buildings. If one were to spend one's lifetime in such a building, what would be the risk of getting lung cancer as a result of this radon exposure? Table 6 from the MOH Report, reproduced on the next page, summarizes the Ontario government's risk estimates for a lifetime exposure at 0.02 WL of radon at the rate of one hour's exposure per day. These are based on the average cancer risk values presented in Tables 1, 2, 3, and 4, with some additional assumptions. As the MOH Report explains, "increasing or decreasing the hours of exposure per day will increase or decrease the risk [as given in Table 6] by the same factor." (Appendix, page 6)

We will limit ourselves to the male risk figures in Table 6, since almost all of the epidemiological evidence is based on male populations, and it is not clear how the female figures are arrived at.[20] As the MOH Report refers to "the fact that people spend no more than half their time outdoors during the course of a year" (accompanying the Summary of Clean-up Criteria in the Appendix), let us assume a minimum of 12 hours per day exposure indoors. We then arrive at the following risk figures for males (making use of Table 6 and the natural incidence of lung cancer in Ontario males of 54 per 1000, given in Table 8 of the MOH Report as reproduced on page 27 of this text.)

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EXHIBIT 8

MOH TABLE 6

EFFECTS OF EXPOSURE TO 0.02 WL FOR ONE HOUR PER DAY OVER A LIFETIME

Study Population.	Model Used	Number of Radiation Induced Cancers per 100,000 persons.		Mean Loss of Life Expectancy at birth per person (Days)	
		Male	Female	Male	Female
Colorado Plateau Uranium Miners	Absolute risk model	12	15	1.4	2.0
	Relative risk model	28	7	1.3	0.45
Uranium Miners in Czechoslovakia.	Relative risk model	53	13	2.4	0.85
Hiroshima	Relative risk model	50	12	2.3	0.80
ICRP		25	31	3.1	4.1

ICRP assumes that 1 rem to the lungs will cause 2 radiation induced cancers in 100,000 persons over their lifetimes.

Assuming all lung cancers appear within a 20 year period, the risk from 1 rem to lungs is 1 case per million persons per year.

Note

The female figures in table 6 are calculated in an obscure way; they certainly seem low, even in comparison with ICRP figures, and they do not seem entirely compatible with the male figures, despite the fact that they are supposedly calculated from the same data given in tables 1,2,3,4. Also, the government's loss-of-life figures are inexplicably lower than the ICRP estimates.

MOH TABLE 8

ONTARIO POPULATION DATA

	Male	Female
Probability at birth of dying of lung cancer over the total life span	5.4%	1.2%
Probability at birth of dying of cancer over the total life span	18%	14%
Life expectancy at birth (years)	69.55	76.76

ESTIMATES OF MALE CANCER RISK FROM LIFETIME EXPOSURE TO 0.02 WL

Source of Information	1 hour/day	12 hours/day	
	Extra Cancers per 100,000	Extra Cancers per 1,000	Increase in Cancer Rate
Minimum Risk from Table 6	12	$\frac{12 \times 12}{100} = 1.4$	2.6%
Muller's Estimate (Appendix, p.9)	20	$\frac{20 \times 12}{100} = 2.4$	4.4%
I.C.R.P. from Table 6	25	$\frac{25 \times 12}{100} = 3.0$	5.6%
Overall Average from Table 6	33.6	$\frac{33.6 \times 12}{100} = 4.0$	7.4%
Relative Risk Data (Averaged)	47	$\frac{47 \times 12}{100} = 5.6$	10.4%
Maximum Risk from Table 6	53	$\frac{53 \times 12}{100} = 6.4$	11.8%

A glance at the right hand column shows that there is a very wide spread in the risk estimates that one might make on the basis of the MOH data, and that Dr. Muller's estimate is toward the low end of this spectrum. If radon exposure is more effective in producing cancer at lower doses, as the evidence indicates, then one would be tempted to rely more heavily on the low-exposure populations of Czechoslovakia and Hiroshima -- thereby arriving at a risk estimate two-and-a-half times larger than Dr. Muller's estimate.

But even this does not fully reflect the risk at low exposures, because table 6 is based on average risk values and does not use the low-exposure data from tables 1,2,3, and 4. What happens if we take this low-exposure data into account?

CORRECTIONS TO THE M.O.H. ESTIMATES

By definition, 1 WLM is the accumulated exposure of and average male individual spending 170 hours in a radon environment of 1 WL. Exposure to 0.02 WL for one hour per day over a lifetime of 70 years leads to an accumulated exposure of $\frac{0.02 \times 365 \times 70}{170}$ = 3 WLM; over a lifetime of

.../29

50 years, the accumulated exposure would be only 2 WLM. So, for 12 hours per day exposure, the accumulated dose would be 36 WLM for a 70-year lifespan and 24 WLM for a 50-year lifespan. The only purpose of this little calculation is to demonstrate that the persons at risk in homes with a 0.02 WL radon environment will be in the lowest exposure, highest risk categories previously identified in the text.

The risk estimates in table 6 are based on the average risk figures from tables 1,2,3, and 4; but those averages systematically underestimate the actual risk to the low exposure groups in each case. If we make the appropriate adjustment to account for the low-dose risk data in the tables, we arrive at the following corrected estimates:

		ADJUSTMENT			1 hr/day exposure Extra Cancer/100,000	
	Sources	Average Figures	Low Dose Figures	Correction Factor	Table 6 Estimate	Corrected Estimate
absolute	Table 1	2.2	9.5	9.5/2.2	12	52
relative	Table 2	5.7	29	29/5.7	28	142
relative	Table 3	11	26	26/11	53	125
relative	Table 4	2.3	7	7/2.3	50	152

Notice that this adjustment brings the three relative risk figures into much closer agreement. (The first entry, based on a different model known as the "absolute risk model", is not really comparable with the relative risk figures since it is calculated in a different fashion -- see note 1 for both tables 1 and 2.)

The average number of excess lung cancers per 100,000 given by the relative risk model is therefore 140 -- exactly seven times larger than the risk figure cited by Dr. Muller. But this is for only one hour per day exposure; multiplying by 12 and dividing by 100, we get 16.8 excess lung cancer cases per thousand for 12 hours per day exposure. This represents a 31% increase over the normal lung cancer rate for Ontario males as given in table 8 of the MOH Report (see page 27).

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COMMENTS ON THE CALCULATIONS

The risk figures calculated on page 28 from table 6 (and subsequently reflected in my corrected estimates on page 29) may be wrong for a number of reasons. The method of calculation has both conservative and non-conservative factors built into it. A brief summary of these is given below.

a) Non-Conservative Factors

1. The number of excess cancers per WLM may be even greater than indicated here at the low doses and low dose rates which are actually involved. This possibility is suggested by both experimental and epidemiological evidence on alpha-emitters. If we had used the appropriate table from reference 3 as the basis for our calculation (see exhibit 9, page 31), we would have arrived at something like a 45% increase in lung cancer as a result of 0.02 WL at 12 hours per day, assuming only a 50-year lifetime. It may be that the MOH data is just too coarse to reveal the true hazard at very low dose rates.

2. Dr. Muller assumes that all lung cancers will appear within a 20 year period following a single exposure (see his comment, reproduced under table 6 on page 27). There is no epidemiological evidence presented to support this assumption. In fact, no less than 11 of the Colorado Plateau miners studies in reference 4 developed cancer more than 20 years after initial exposure -- and this number, 11, is almost double the expected number of lung cancers for the entire population of 1981 miners (using U.S. data on lung cancer incidence in those age groups.) As reference 3 points out, "It is not clear how long after start of ex-

.../32

EXHIBIT 9

ESTIMATION OF LUNG CANCER RISKS FROM RADON DAUGHTERS AT DIFFERENT EXPOSURE RATES (TABLE III from reference 3)

	Mean Exposure Rate (in Working Levels)				
	Up to 0.01	0.01-0.36	0.36-1.09	1.09-2.5	2.6 or more
Cumulative exposure in WLM	Up to 3.0	3.1-100	101-300	301-700	701 or more
Attributable lung cancers per million per year/WLM	39	34	26	14	4.5
Attributable cancer for a lifetime per million/WLM	1170	1020	780	420	135
Average WLMM required to produce one lung cancer	855	980	1280	2380	7410
Relative risk per million per WLM	3.1	2.8	2.2	1.2	0.4

1) At 12 hrs/day exposure, 0.01 WL yields a lifetime dose (over 50 years) of about 12 WLM. Excess cancers (using this table) would then be 12 x 1170 = 14,040 cases per million, or 14 extra cases per thousand -- a 26% increase in the Ontario male lung cancer rate.

2) At 12 hrs/day exposure, 0.02 WL yields a lifetime dose (over 50 years) of about 24 WLM. Excess cancers (using this table) would then be at least 24 x 1020 = 24,480 cases per million, or 24.5 extra cases per thousand -- representing a 45% increase in the Ontario male lung cancer rate.

posure the incidence of lung cancer continues to increase; certainly no one has yet observed a decrease with increasing time, as has been observed for radiation-induced leukemia" (page 5). The gradual build-up of long-lived radon daughters in the lung, such as lead-210 with its 21-year half-life, makes it highly unlikely that extra cancers would stop appearing after 20 years.[21] Lead-210 gives rise to polonium-210 as a daughter product. The carcinogenic properties of polonium-210 are well documented (see page 16 for examples). In addition, epidemiological evidence reveals that non-smokers who start mining at an early age only begin to show dramatic increases in lung cancer some 40 or 50 years after initial exposure (reference 3, page 21).

3. Children are known to be more radiosensitive than adults. In the late 1960's, Dr. Alice Stewart showed that a single diagnostic x-ray to the abdomen of a pregnant woman in the first six weeks of pregnancy leads to a 50% increase in childhood cancer and leukemia among the offspring[22] -- a risk factor which is in turn higher than the relative risk for children up to nine years of age, which is in turn greater than the relative risk for adults (see reference 6, especially table 1 therein). This extra sensitivity of children to radiation-induced cancers may be compounded by heavy juvenile exposures to radon, as a result of (1) children crawling or playing on the floor or close to the walls, where the radon concentrations are often higher than elsewhere in the house; (2) children spending more than 12 hours per day inside the house and/or spending more time in the basement; (3) children playing outside close to the outer walls of the house, where the radon gas rises from under the house.

.../33

4. Mothers and invalids may spend much more time indoors than able-bodied men and older children, thus giving rise to proportionately greater doses.

5. Mechanical problems or structural deterioration may incapacitate protective systems (such as fans or sealants) within the buildings, resulting in indoor radon levels above 0.02 WL.[23]

6. Atmospheric radon gas from uranium tailings in the Elliot Lake area will contribute an outdoor component of radon exposure which is by no means insignificant and which should also be evaluated (see reference 7, which is based on data from the U.S. Environmental Protection Agency).[24]

b) Conservative Factors

1. Not all buildings will aproach the 0.02 WL limit. Nevertheless, I have been informed that 50 out of 58 new homes recently tested in Elliot Lake showed levels in excess of 0.02 WL before fans were installed to provide extra ventilation. Of a total of 1900 older homes tested in Elliot Lake since 1976, about 325 were found to be over the 0.02 limit. This fraction (1/6) is not very reassuring -- if 1/6 of the planned population of 30,000 were exposed to 0.02 WL, we could have over 80 radon-induced lung cancer deaths just from breathing radon gas at home.

2. For uranium miners, the additional radon exposure in the home will be a relatively small augmentation to the exposure which they receive in the mines. However, the risk is additive, and the ICRP recommends that all unnecessary exposure to radon be avoided.

3. Most people will not spend their entire lives in Elliot Lake; there will be a considerable population turnover. Such a turnover of population will not reduce the total number of expected cancers however

.../34

(even according to the linear hypothesis -- see pages 103-105 in reference 1). The cancers will just be diluted in a larger population -- the human tragedy will be undiminished, but the statistical percentage will look smaller. According to the non-linear hypothesis described in this paper, a turnover in population may actually increase the number of cancers by decreasing the individual exposures without diminishing the total dose to the entire population -- thereby bringing about an increased risk per WLM because of the lower individual exposures.

VIOLATION OF CONTROL BOARD GUIDELINES?

The Atomic Energy Control Board has laid down annual dose limitations for whole-body exposure, and for various organs of the body.

For whole-body exposure to penetrating radiation, AECB limits are 5 rems per year for atomic workers and 500 millirems per year for members of the general public; however, AECB policy is to aim for no more than 1% of the Maximum Permissible Dose of 500 mr/year as an official guideline -- in other words, members of the public should not be exposed to more than 5 mr/year.

For the lungs, AECB exposure limits are set at 15 rems for atomic workers and 1.5 rems for members of the general public.

Let us deal with the lungs first. Using Dr. Muller's equivalence of 1 WLM = 4.42 rems (page 5, Appendix, MOH Report), it is easily seen that one year's accumulated dose at 0.02 WL for 12 hrs/day is almost 2.28 rems, which is far in excess of the 1.5 rem limit set by the AECB. Even if we use 1 WLM = 4 rems, the annual accumulated exposure at 0.02 WL for 12 hrs/day is just over 2 rems, which is 33% higher than the maximum permissible exposure for members of the public. (As the Ham Commission Report notes, typical conversion factors are 1 WLM =

.../35

5-6 rems, which makes the situation even worse: see reference 1, page 116).

Whole-body exposure results primarily from gamma radiation. The MOH Report advocates a standard of 0.05 mr/hr (gamma) at a height of one metre above the centre of the floor (where your gonads might be when you stand up). With 12 hr/day exposure, this will produce an accumulated annual dose of 219 mr, which is more than 40 times larger than the AECB Guideline of 5 mr/yr. Recent standards laid down by the U.S. Environmental Protection Agency limit the exposure of any member of the general public from any U.S. nuclear facility to an absolute maximum of 25 mr/yr. Thus, on a 12 hr/day basis, the proposed housing standard of 0.05 mr/hr will lead to an annual accumulated dose which is 8.76 times higher than the Maximum Permissible Dose from a nuclear facility in the United States.

ESTIMATING THE RISK FROM GAMMA RADIATION

The health risk from exposure to low level gamma radiation includes not only cancers and genetic defects, but also possible increases in such diseases as diabetes milletus, cardiovascular disease, mental retardation, stroke, hypertension, and a great many infectious diseases. These somatic risks are discussed in some detail in the Proceedings (reference 2, Chapter III); they should definitely be included in any risk assessment associated with setting housing standards for gamma radiation.

There are many well-qualified and well-respected people in the field of health physics or radiation biology who believe that current risk estimates are understated by about a factor of ten.[25] As Dr. Morgan says on page 84 of the Proceedings, "the somatic risks and in particular the risk of radiation-induced cancer of almost every type are more --

.../36

to an order of magnitude [i.e. ten times greater] -- than we considered them to be some time back." A more detailed discussion of the controversy is given in reference 9.

There is also some evidence which seems to indicate that low dose rates may be more harmful than high dose rates in producing cancer, even in the case of gamma radiation; but the evidence is quite confused on this subject and I am not able to form a professional judgment as to what the correct risk factor might be (see reference 2, Chapter IV). When it is a matter of life and death, however, I believe that the standards must be made as stringent as possible. It is far better to overestimate the risks than to underestimate them -- standards can always be relaxed later on, but dead people cannot be resurrected so easily. Moreover, if the housing standards are tightened up at some future date, it will be very difficult and costly to do the remedial work needed to bring older buildings into conformity with the new standard.

CONCLUSION AND RECOMMENDATIONS

Radon is a very potent carcinogen, mainly because of the radon daughters which inevitably accompany it. Even if we use the linear hypothesis, it has been estimated that about 8% of all spontaneous lung cancers in the United States are due to naturally-occurring radon gas, and that is at an average level of exposure (0.001 WL) which is only 5% of the proposed housing standard.[26] Allowing a twenty-fold increase in public exposure to such a potent carcinogen seems a very questionable policy. The U.S. Environmental Protection Agency has calculated that outdoor exposure to radon gas emitted by a typical tailings pond, even with five metres of earth covering it, would cause from 60 to 200 extra deaths in the surrounding population per century, due to radon-caused lung cancer (see reference 7 for details.)

.../37

In this paper, I have argued that (1) there is good scientific evidence that alpha radiation is more effective in causing cancer at low dose rates than at high dose rates; (2) using data provided by the Ministry of Housing, one can reasonably estimate a 31% increase in the incidence of lung cancer among people who spend a lifetime in buildings having a 0.02 WL radon environment.

Two recommendations suggest themselves. The first is that people should be told that there is a very real risk of excess lung cancer from radon exposure in homes, and that the proposed housing standard could, under the worst conditions, lead to a substantial increase in lung cancer rates. This may not be a pleasant thing to do, but it must be done. People deserve to know the worst, since they are the ones who will be taking the risks -- they certainly deserve more than soothing reassurances which make the problem seem to be non-existent. The second recommendation which I would like to make is that every effort should be made to prevent excess radon in Elliot Lake buildings, if necessary by building them above ground without basements, elevated by means of cinder blocks or other props under the foundations. If all else fails, serious consideration should be given to having workers live away from Elliot Lake and commute to work.

When there is conflicting testimony on the nature of a public health hazard with a high degree of credibility on both sides, it seems to me that the standards should be set on the assumption that the more pessimistic estimate may in fact be the true one. Certainly my training as a mathematician tells me that when this kind of conflicting evidence exists, it can be dangerously misleading to rely on one simplistic mathematical model which incorporates only one narrow view or version of the truth. As Fred Knelman has said, when human life is at stake, the "magic numbers" provided by a calculational model can turn out to be "tragic numbers" for the people involved.

Finis.

EXHIBIT 10

BIOGRAPHICAL NOTES

ADDENDUM (1985)
Dr. Archer is now retired. Dr. Gofman has authored the book Radiation and Human Health - a definitive medical text book.

Victor Archer

Now Medical Director at the U.S. National Institute for Occupational Safety and Health, Dr. Archer (MD) has been engaged in studying lung cancer among uranium miners for over twenty years. He worked very closely with J. K. Waggoner (author of the famous Waggoner Report on Uranium Miners in the United States, 1967, which led to a drastic reduction in the maximum permissible radon exposure for U.S. miners in 1971 -- from 12 WLM to 4 WLM annually. The Canadian standard of 4 WLM was not adopted until four years later.)

Dr. Archer has played a major role in the field of radon carcinogenesis epidemiology. The Ham Commission Report (reference 1) cites six papers co-authored by Dr. Archer out of a total of about twenty papers on the subject.

John Gofman and Arthur Tamplin

In 1963, the U.S. Atomic Energy Commission appointed Dr. Gofman as Assistant Director of the Lawrence Radiation Laboratory in Livermore, California. His mission was to head up a team of experts to investigate the biological effects of radiation on man. After seven years of intensive study of all existing experimental and epidemiological evidence on the subject, Dr. Gofman and his colleague Dr. Tamplin published results which claimed that the health effects of radiation were very much higher than official estimates indicated. The research program of Drs. Gofman and Tamplin was terminated not long afterwards, to the mutual dissatisfaction of all parties.

Dr. Gofman is an M.D. and a Ph.D. in nuclear physical chemistry. He is co-discoverer of U-232, U-233, Pa-232, and Pa-233. He is Professor Emeritus in Medical Physics at the Berkeley Campus of the University of California, and Lecturer in Medicine at the San Francisco Campus of the same university. His medical researches are well known; for example, in 1972 he won the Stouffer Prize (one of the most prestigious awards in the field of heart research, carrying a $50,000 cash award) for his work on the role of lipoproteins in arteriosclerosis.

Dr. Tamplin is a Ph.D. in biophysics; he served as a group leader under Dr. Gofman in the Biomedical Division of the Lawrence Radiation Laboratory from 1963 to 1969, when funds for the project were terminated. He is currently a staff scientist at the Natural Resources Defence Council, 917 15th Street NW, Washington DC, 20005.

Karl Z. Morgan

A world-renowned pioneer in the field of Health Physics, Dr. Morgan was Director of the Division of Health Physics at the Oak Ridge National Laboratory for over 30 years. He was one of the original members of the International Commission on Radiological Protection, and was editor of the professional journal Health Physics until quite recently.

In 1971, Dr. Morgan was prevented by his superiors at Oak Ridge from delivering a paper on the health hazards of plutonium (an alpha-emitting transuranic element -- see reference 10). That was only one of several instances of suppression of scientific results at Oak Ridge (referred to by Dr. Morgan in reference 8 reprinted on the next two pages). Dr. Morgan left Oak Ridge in 1972 and is now Professor of Health Physics in the School of Nuclear Engineering at the Georgia Institute of Technology.

Acronyms and abbreviations appearing in Dr. Morgan's letter are explained below:

Pu = plutonium
U = uranium
Th = thorium
NO_x = nitrogen oxides
SO_x = sulphur oxides
CO_x = carbon oxides

ORNL = Oak Ridge National Laboratory
ORAU = Oak Ridge Associated University
OSHA = Occupational Safety and Health Agency
ERDA = Energy Research and Development Administration
LMFBR = Liquid Metal Fast Breeder Reactor

Alice Stewart

In the 1960's, Dr. Stewart (MD) did an epidemiological study of childhood cancers and leukemias caused by obstetric x-rays in England. Her work showed that a single x-ray to the abdomen of a pregnant woman during the first six weeks of pregnancy would result in a 50% increase in childhood cancer and leukemia among the offspring. She also verified the linear hypothesis for x-rays down to very low doses in the range from 0 to 1.5 rads (low doses, but high dose rates).

When her results were greeted with scepticism, she and her statistician colleague George Kneale undertook a far more ambitious study which took in the entire British Isles. The results of this second study, the largest ever done in the field of radiation carcinogenesis epidemiology up to that time, were printed in Lancet (the British Medical Journal) in 1970. They fully confirmed her earlier findings. A similar study was done by Dr. Brian McMahon of Harvard University using U.S. data, and it gave additional confirmation to Dr. Stewart's results.

ADDENDUM (1985)

Dr. Morgan now works as an independent consultant on the health effects of radiation; most of his clients are radiation victims.

EXHIBIT 11
Letter from Karl Morgan to James Schlesinger

Atlanta, Georgia 30332 (404) 894-3720

May 25, 1977

ORNL = Oak Ridge Nuclear Laboratories
LMFBR = Liquid Metal Fast Breeder Reactor
Pu = Plutonium
U = Uranium
Th = Thorium
AEC = Atomic Energy Commission
ERDA = Energy Research & Development Agency
ORAU = Oak Ridge Associated University
OSHA = Occupational Safety & Health Agency
NRC = Nuclear Regulatory Commission
NOx = Nitrogen Oxides
SOx = Sulfur Oxides
COx = Carbon Oxides

Mr. James Schlesinger
Executive Office of The President
Energy Policy and Planning
Washington, D.C. 20500

Dear Mr. Schlesinger:

As a followup of my letters of March 30, 1977 and May 23, 1977, and following a lecture I gave recently at the University of Tennessee, Knoxville, Tennessee, several persons at ORNL have contacted me suggesting that perhaps I would be willing to lend my support to a current ORNL proposal that the LMFBR-CRBR program be continued by replacing the Pu fuel with ^{233}U and the ^{238}U with ^{232}Th. I indicated to them that were I to approve such a program, it would be only with a number of qualifications and with assurance of many program changes. We certainly need information on the Th-^{233}U cycle, but I'm not sure this is the cheapest and best way to get the information needed. One of the greatest causes of my trepidation relates to the ORNL management and its past record of blind support of the LMFBR in spite of knowledge of its very serious shortcomings. ORNL management should have been objective and should have insisted on following the best course — not the politically expedient one. Instead, it only did those things that would please the AEC (and later the ERDA). It did not display any vision or desire to be successful — rather, it wanted to preserve status quo, to keep the money rolling, and everyone on the payroll. Any ideas in Oak Ridge contrary to the Washington approved course (prior to my leaving ORNL in 1972) were suppressed. Even studies relating to such important questions as brittle fracture of the reactor containment vessel, common mode failure and emergency core cooling were supressed, the findings depreciated and not published.

Perhaps management at the various Oak Ridge operations can change this poor record, but I'm not sure it can or that recent events in Oak Ridge would justify our encouragement. For example, when the Mancuso Program (to which I am a consultant) indicated there was an increase of statistical significance in four types of cancer (myeloid neoplasm, breast cancer, pancreatic tumors and lung cancer), Mancuso was informed shortly afterwards that his program would no longer be funded by ERDA, and we learned that Jim Liverman plans to reincarnate this program in Oak Ridge (probably under the supervision of ORAU) to be conducted by Drs. C.C. Lushbaugh and Edith Tompkins. This change would be at a very great cost and would represent a serious discontinuity of scientific effort. One can only suppose that the new Oak Ridge team must get the <u>right</u> answer (i.e. prove there is <u>NO</u> radiation risk to Hanford and Oak Ridge workers) if it cares to have a continuation of funding. I believe Dr. Lushbaugh would try to be objective, but I have good reason to question if this would be true of Dr. Tompkins.

Mr. James Schlesinger
May 25, 1977

Page 2

Perhaps at this stage there is something you can do to prevent this transition. My suggestion would be to ask OSHA to take over the support of this Mancuso program and, hopefully, it would see the wisdom of asking Dr. Mancuso to continue his studies and continue the services of the two British scientists, Drs. Alice Stewart and George Kneale, that have contributed so much to the success, scientific stature, and independent, unbiased evaluation of these data from Hanford and Oak Ridge.

Actually, the cancer risk at Hanford, as reported by Mancuso, Stewart, and Kneale, in comparison with other occupational risks is rather small. The only problem is that many of the early ORNL, Hanford, and AEC employees have been saying repeatedly in public (and contrary to my cautionary warning) that there are NO radiation risks from work at these facilities. The word NO is such a small number that true scientists refrain from using it. Surely, it is a conflict of interest for this program to be conducted in Oak Ridge under contract with either ERDA or NRC.

I am a strong supporter of nuclear energy, but not at any cost. Many people agree with me that the AEC, NRC, ERDA, ORNL, etc. are often their worst enemies and get in the way of those of us who believe we can build and operate a nuclear energy industry that is acceptably safe and presents occupational and environmental risks that are far less than those of a well conducted fossil fuel power program. After all, the risks from NO_x, SO_x, CO_x, hydrocarbons and particulates in terms of lung carcinoma, chronic bronchitis and emphysema are very real and some of us are very concerned about the effects of CO_2 on the climate. I am all for Jimmy Carter's emphasis on conservation, solar and fossil fuel energy for our power, but I believe with your help our country can take the lead in placing nuclear power in its proper place.

In closing, and in contrast with the faults at ORNL and Oak Ridge which I mentioned above, I would like to close with the reminder that some of our countries best scientists have worked at ORNL and a few of them are still there.

Best personal regards.

Sincerely,

Karl Z. Morgan
Neely Professor

KZM:rs

cc John F. Ahearne

EXHIBIT 12
Verbatim Excerpts from the BCMA Report

BRITISH COLUMBIA MEDICAL ASSOCIATION

THE HEALTH DANGERS OF URANIUM MINING

AND JURISDICTIONAL QUESTIONS

A SUMMARY OF MATERIAL BEFORE THE

BRITISH COLUMBIA ROYAL COMMISSION OF INQUIRY

HEALTH AND ENVIRONMENTAL PROTECTION - URANIUM MINING

PRESENTED: AUGUST 1980

E.R. YOUNG, BSc, MD
R.F. WOOLLARD, MD

ENVIRONMENTAL HEALTH COMMITTEE
BRITISH COLUMBIA MEDICAL ASSOCIATION

LUNG CANCER AMONG UNDERGROUND MINERS

Dr. Wagoner well described the discovery of the relationship between lung cancer and radon daughters:

> The real nature of this pulmonary disorder among miners of the Schneeberg (Germany) area was not identified until 1879 when Harting and Hesse first diagnosed it.
>
> In 1913, Ainstein reported that of 665 Schneeberg miners dying during 1875-1912, 40 percent (or 276) died of lung cancer. Pircham and Sikl, in 1932, reported that of 17 deaths observed during 1929-1930 among miners of uranium-bearing ores in Joachimsthal (Czechoslovakia), 53 percent (or 9) were due to cancer of the lung.
>
> These same investigators ... concluded that the most probable cause of these tumors was radiation in the air of the mines. These investigators also made note "the miners themselves state that discovery of a rich uranium vein is always followed some years later by a strongly increased mortality among them".

Hollywood, in his article on "The Epidemiology of Lung Cancer Among Workers Exposed to Radon and Radon Daughters" in May, 1979, noted:

> By 1940, then, excess deaths from lung cancer among two groups of European miners had been associated with relatively high concentrations of radon in the mine atmosphere. In that same year ... conclusions were drawn that prolonged breathing of air containing a high concentration of radon, may have caused what was estimated at that time to be a 30-fold increase in the incidence of lung cancer.
>
> The percentage of miners developing carcinoma of the lungs in Schneeberg was 63 percent, in Joachimsthal 42 percent, and in St. Lawrence [Newfoundland] 36 percent.

Studies in the U.S. were undertaken in the 1950's on uranium miners in the Colorado plateau area. These results began to appear in the early 60's, and they showed an increase in lung cancer with an increase in exposure to radon daughters. Dr. Wagoner noted that these studies had to be extended and refined to rule out any possible other agent:

> First there was a basic denial that there was such a problem. Then there was a position that it had to be due to smoking. Then it was on the basis -- well, it had to be due to hard rock mining. There were sequential analyses undertaken to address all of these, what in statistical terms I would call confounding factors, but in public health terms I would call delaying [factors].
>
> In 1967, Lundin demonstrated that during the period 1950 through June 1965, white underground uranium miners experienced 37 deaths due to lung cancer whereas only 7.3 would have been expected [and] through September 1967, 62 deaths due to lung cancer as contrasted to only 10.02 expected.

EXHIBIT 12

LUNG CANCER AMONG UNDERGROUND MINERS (continued)

[As noted by Dr. Wagoner, referring to the Colorado data], observed versus expected carcinoma of the lung cases in 1978 was 205 versus 40, with an attributable risk of 164 men

> who have died due to lung cancer over and above what I would expect in that population if they had not been subjected to those exposures. I would consider that as epidemic.

With the long latent period of carcinoma induction by low level radiation, these numbers will increase further over the next 20 years.

The submission of Dr. Wigle relating to the St. Lawrence (Newfoundland) fluorspar miners who were exposed to elevated levels of radon daughters demonstrated an observed incidence of lung cancer of 65 versus an expected 6.41, with an average ratio of observed to expected of 10.1 . Dr. Radford noted that the ongoing studies, such as the one of the Newfoundland fluorspar miners,

> clearly indicate the seriousness of this problem, still with us fifty years after the risk was originally identified in the Bohemian miners of central Europe.

The collection of the Canadian [uranium mining] data began in 1974. The Royal Commission on Health and Safety in Mines in Ontario [the Ham Commission] commissioned an epidemiological study of the uranium miners in the Elliot Lake area; this was conducted from 1975 to 1976. Dr. Muller noted that

> The Ontario uranium mining population is characterized by relatively low exposures and relatively short periods of exposure. There is, therefore, less extrapolation involved from high to low doses and dose rates, ... relatively short periods of exposure in most men, ... and nearly 20 years of observation time.

The Ham Commission analyzed the data [81 observed lung cancer deaths versus 45.08 expected] in order to determine whether radon daughters were the agent:

> The lung cancer cases tended to accumulate more in the higher exposure groups, which indicates that lung cancer risk was greater in the higher exposure groups than in the lower ones.

In his analysis of the Ontario data, Ellett stated:

> From the occupational health point of view, it is certain that exposure to radon daughters leads to an increased risk of lung cancer for the working force as a whole, and that this risk extends to levels of exposure that are below current occupational guidelines.

According to the United Steelworkers of America, the number of lung cancer cases should now read well in excess of 100 at Elliot Lake and are "climbing steadily".

MORE EVIDENCE ON LUNG CANCER AND RADON GAS

Dr. Axelson, in his submission on Swedish Miner Lung Carcinoma, stated:

> Several studies have shown an increased lung cancer mortality among Swedish metal [zinc-lead-iron] miners as probably caused by the exposure to radon and radon daughters in the mine atmosphere. In a nation-wide survey, as yet unpublished, the average lung cancer mortality among Swedish miners was found to be about fivefold the normal.
>
> These Swedish studies deal with a life-time follow-up of miners, whereas most other mining populations have been studied by means of cohorts with a follow-up time of not less than about 25-30 years or more.

Wagoner noted that

> In 1942, Campbell reported the induction of lung tumors in 20.3 percent of mice exposed by inhaling dust from the Joachimsthal mines, whereas only 2.1 percent was found in the unexposed controls.

The most detailed and conclusive evidence showing the carcinogenic effect of radon daughters has been done by Dr. Lafuma of the Radiation Protection Department of the Atomic Energy Commission of France:

> Studies have been carried out by two teams from the Commission of Atomic Energy in France.... Throughout the ten years of research, close to 10,000 rats were used of which 3,000 were used for radon studies. In these 3,000 rats, more than 600 pulmonary cancers were observed.

Dr. Lafuma's research indicates a higher risk [per unit of exposure] at lower cumulative working level months (WLM).

==========

It seems that the controversy over low level radiation which is now taking place is following a similar pattern to that of the health hazards of cigarettes that began 30 years ago when epidemiological studies were met with flat denials that cigarettes could possibly cause cancer of the lung.

One of the serious consequences of down-playing the effects of low-level radiation will be to deny those who have developed various carcinomas adequate compensation which may be their due. With the abundant information on the effects of low-level ionizing radiation, the humane course of action would be to give the worker, or in most cases the deceased worker's family, the benefit of the doubt as to whether his or her particular carcinoma was a product of radiation, and compensate accordingly.

Society and industry must be willing to shoulder this burden if we wish to continue with the production of nuclear power and nuclear weapons.

==========

RISK ESTIMATES: COMPARISON WITH A SAFE INDUSTRY

Mr. Bush, Manager of the Radiation Protection Division of the AECB, described mining as an industry with high risk:

> "one [accidental] death per year for every thousand workers"

According to Mr. Bush,

> Workers in the safest occupations -- manufacturing, for example -- are subject to an annual risk of accidental death of about one in ten thousand.

When asked whether the mining industry in Canada was an industry with a high standard of safety, Mr. Bush replied: "No". [In particular] he knew of no industry that exceeds the combined risk of uranium mining.

According to the AECB,

> The risk of lung cancer associated with an exposure of 4 WLM per year over a normal working life is considered to be acceptably small, compared to the risk of [accidental] death associated with other ["safe"] industries.

Mr. Bush re-iterated this in cross-examination:

> The risk of working with the present dose limits is no greater than the occupational risk of the safer industries.

Of course, what he clearly means is that the risk is no greater than adding the occupational risk of a safer industry on top of the occupational risk of an industry which does not have a high standard of safety.

[In any event], the risk of accidental death in a "safe industry" can be approximated at 100 deaths per million workers per year. Several authors have produced estimates of lung cancer cases per million people exposed to one working level month (WLM). According to Mr. Bush,

> Dr. Gordon Stuart, formerly of Chalk River, reviewed the American and Czechoslovakian data and he concluded that ... you get about 14 to 20 lung cancer cases per million people exposed to one WLM.
>
> A year or two ago, the [European] Nuclear Energy Agency concluded that a reasonable risk estimate, for purposes of radiation protection, would be about 100 cases of lung cancer per million people per WLM.

Sevc, in his calculations of the [Czechoslovakian] data in 1976, found

> 0.23 ± 0.04 lung cancer cases per thousand workers per WLM [230 lung cancer cases per million workers per WLM] as an estimate of average radiation risk for the total group.

As can be seen, even using the Nuclear Energy Agency's calculations, the [cancer] risk to miners would be four times as great at present radiation standards [4 WLM/y] than the accident risk in safe industries.

RISK ESTIMATES: COMPARISON WITH A SAFE INDUSTRY (continued)

Using Sevc's calculations, [the cancer risk] would be 9.2 times as great -- approximately 10 times as great -- which would then be in a category of industries with a high degree of risk [one accidental death per thousand workers per year].

Moreover, there is a very important flaw in the AECB's comparison of accidental risks per year with lung carcinomas [per year], which makes direct comparison meaningless:

- Risk of accidental occupational death is a relatively instantaneous risk, which exists (by definition) only during the period of employment and ends upon termination of employment.
- Risk of lung cancer from radiation, although beginning after several years of employment, continues many years past termination of employment; thus a gradually flowering crop of cancers grows larger each year.

[Indeed] Archer & Lundin in 1967 concluded that an exposure of 120 WLM

> appears to double the lung cancer incidence characteristic of the general [unexposed] population.

Summary of doubling dose estimates for lung cancer in uranium miners:

Archer	(1967)		120 WLM
Hewitt	(1980)	- Ontario	40-50 WLM
		- Newfoundland	50 WLM
Sevc	(1976)		~50 WLM
US EPA	(1980)		~40 WLM
Ellett	(1980)		40 WLM
BEIR-II	(1972)		34 WLM
BCMA	(1980)	- NIOSH & Sevc	19-20 WLM
BEIR-III	(1980)		12-17 WLM
Axelson	(1980)		2 WLM

The lifetime incidence of lung cancer in males can be calculated to equal 52.5 per thousand, equivalent to approximately a five percent lifetime risk for lung cancer development in males. It would appear that the doubling dose from exposure to radon daughters would be 40 WLM or less, in the exposure ranges experienced by today's miners.

Thus, at a lifetime dose of 40 WLM, a miner would have approximately a 10 percent rather than a 5 percent risk of developing carcinoma of the lung; that is a risk of 1250 lung cancer cases per million workers per WLM. The risk [per million workers] would be four times as high at today's maximum permissible exposure of 4 WLM per year. Compare this value with the risk of accidental death in safe industries of 100 accidental deaths per million workers per year!

Because of the long latent period of lung cancer, and its variability with age and smoking, Archer has calculated the attributable cancer for lifetime per million [workers] per WLM, which is certainly the value most significant to the mining population. Using the exposure rates present in today's mines and mills, the attributable cancer per lifetime per WLM is approximately 1000 [per million workers].

RISK ESTIMATES: COMPARISON WITH A SAFE INDUSTRY

Mr. Bush, Manager of the Radiation Protection Division of the AECB, described mining as an industry with high risk:

> "one [accidental] death per year for every thousand workers"

According to Mr. Bush,

> Workers in the safest occupations -- manufacturing, for example -- are subject to an annual risk of accidental death of about one in ten thousand.

When asked whether the mining industry in Canada was an industry with a high standard of safety, Mr. Bush replied: "No". [In particular] he knew of no industry that exceeds the combined risk of uranium mining.

According to the AECB,

> The risk of lung cancer associated with an exposure of 4 WLM per year over a normal working life is considered to be acceptably small, compared to the risk of [accidental] death associated with other ["safe"] industries.

Mr. Bush re-iterated this in cross-examination:

> The risk of working with the present dose limits is no greater than the occupational risk of the safer industries.

Of course, what he clearly means is that the risk is no greater than adding the occupational risk of a safer industry on top of the occupational risk of an industry which does not have a high standard of safety.

[In any event], the risk of accidental death in a "safe industry" can be approximated at 100 deaths per million workers per year. Several authors have produced estimates of lung cancer cases per million people exposed to one working level month (WLM). According to Mr. Bush,

> Dr. Gordon Stuart, formerly of Chalk River, reviewed the American and Czechoslovakian data and he concluded that ... you get about 14 to 20 lung cancer cases per million people exposed to one WLM.
>
> A year or two ago, the [European] Nuclear Energy Agency concluded that a reasonable risk estimate, for purposes of radiation protection, would be about 100 cases of lung cancer per million people per WLM.

Sevc, in his calculations of the [Czechoslovakian] data in 1976, found

> 0.23 ± 0.04 lung cancer cases per thousand workers per WLM [230 lung cancer cases per million workers per WLM] as an estimate of average radiation risk for the total group.

As can be seen, even using the Nuclear Energy Agency's calculations, the [cancer] risk to miners would be four times as great at present radiation standards [4 WLM/y] than the accident risk in safe industries.

RISK ESTIMATES: COMPARISON WITH A SAFE INDUSTRY (continued)

Using Sevc's calculations, [the cancer risk] would be 9.2 times as great -- approximately 10 times as great -- which would then be in a category of industries with a high degree of risk [one accidental death per thousand workers per year].

Moreover, there is a very important flaw in the AECB's comparison of accidental risks per year with lung carcinomas [per year], which makes direct comparison meaningless:

- Risk of accidental occupational death is a relatively instantaneous risk, which exists (by definition) only during the period of employment and ends upon termination of employment.
- Risk of lung cancer from radiation, although beginning after several years of employment, continues many years past termination of employment; thus a gradually flowering crop of cancers grows larger each year.

[Indeed] Archer & Lundin in 1967 concluded that an exposure of 120 WLM

> appears to double the lung cancer incidence characteristic of the general [unexposed] population.

Summary of doubling dose estimates for lung cancer in uranium miners:

Archer	(1967)		120 WLM
Hewitt	(1980)	- Ontario	40-50 WLM
		- Newfoundland	50 WLM
Sevc	(1976)		~50 WLM
US EPA	(1980)		~40 WLM
Ellett	(1980)		40 WLM
BEIR-II	(1972)		34 WLM
BCMA	(1980)	- NIOSH & Sevc	19-20 WLM
BEIR-III	(1980)		12-17 WLM
Axelson	(1980)		2 WLM

The lifetime incidence of lung cancer in males can be calculated to equal 52.5 per thousand, equivalent to approximately a five percent lifetime risk for lung cancer development in males. It would appear that the doubling dose from exposure to radon daughters would be 40 WLM or less, in the exposure ranges experienced by today's miners.

Thus, at a lifetime dose of 40 WLM, a miner would have approximately a 10 percent rather than a 5 percent risk of developing carcinoma of the lung; that is a risk of 1250 lung cancer cases per million workers per WLM. The risk [per million workers] would be four times as high at today's maximum permissible exposure of 4 WLM per year. Compare this value with the risk of accidental death in safe industries of 100 accidental deaths per million workers per year!

Because of the long latent period of lung cancer, and its variability with age and smoking, Archer has calculated the attributable cancer for lifetime per million [workers] per WLM, which is certainly the value most significant to the mining population. Using the exposure rates present in today's mines and mills, the attributable cancer per lifetime per WLM is approximately 1000 [per million workers].

ATOMIC ENERGY CONTROL BOARD: UNFIT TO REGULATE

The AECB policy regarding a lifetime exposure limit for uranium miners [February 1978] is based on one study [published in 1969], which is not only 11 years out of date, but which has been revised several times by the authors. The AECB notes in passing that in Ontario,

> only 20 of the 81 lung cancer victims who had worked in uranium mines had accumulated as much as 120 WLM (the exposures of the other 61 victims being 0 to 99 WLM, or 35 WLM on average).

Ignoring this and using the 1969 study (which seems to be the extent of their literature review as no other references are cited) AECB states:

> If one had to choose a WLM value that had some special significance, 840 WLM would be a more logical choice [than 120 WLM] because it marks the level above which lung cancer incidence appears to increase with increasing exposure; (i.e. although an excess of lung cancer is evident in each of the exposure categories, the excess appears to be independent of exposure below 840 WLM.)

Such a policy statement, based on antiquated data and inadequate literature review, would be irresponsible coming from the nuclear industry, let alone the regulatory agency of that industry. However, as will become clear, it is difficult to ascertain where one ends and the other begins.

The Manager of the Radiation Protection Division of the AECB is Mr. Bush, who has a degree in Chemical Engineering (1955). He worked for Atomic Energy of Canada Limited (AECL) in Chalk River from 1957 to 1969, and subsequently with the AECB from 1969 to the present. One notes that Mr. Bush is responsible for developing radiation protection guidelines and regulations.

Mr. Bush admitted,

> I'm not a medical doctor. I'm not an epidemiologist.

(This is evident as well from the Board's paltry data analysis upon which their statements of risk are made.) The AECB

> is currently considering how the latest recommendations [ICRP 1977] might be incorporated into AECB regulations. AECB is being assisted in its review of the ICRP recommendations by its Advisory Committee on Radiologic Protection [ACRP], which it established early in 1979. The Advisory Committee was set up to provide the Board with independent advice ... no Board staff member is [on it].

Mr. Bush pointed out the difficulty the U.S. Nuclear Regulatory Commission may have in adopting these new ICRP higher dose limits:

> they would be difficult to implement under the climate of nuclear controversy currently existing in the U.S.A. For example, the new ICRP system of dose limitation implies higher dose limits for irradiation of some individual organs ...

ATOMIC ENERGY CONTROL BOARD: UNFIT TO REGULATE (continued)

The Chairman of the new Advisory Committee, Dr. G. C. Butler, listed members of this Committee.

It includes himself, who has been an employee of AECL at Chalk River from 1957 to 1965, a member of the ICRP Committee from 1963 to 1973 and again from 1973 to 1977, and worked from 1945 to 1947 with the National Research Council (Ottawa) in the Atomic Energy Project; he has been with the National Research Council since 1965.

It also includes Dr. Marko of AECL [Director of Health Physics at Chalk River] and Dr. Hollywood from Newfoundland, who wrote a section in the AECB Elliot Lake Uranium Mine Inspector's Training Course Manual. The [1979 Elliot Lake] manual contains the following:

> The AECB has seen no convincing evidence for a limitation on cumulative lifetime exposure, provided the average exposure received during a working life does not exceed 4 WLM per year....
>
> Radiation damage is observed only at doses higher than about 100 rads; and although effects have generally not been observed at lower doses, it is assumed for radiation protection purposes that the effect is proportional to the dose right down to zero exposure.

Not only is the last sentence grossly in error, any trainee inspector who is using the graph [showing "observed" cancers at low doses to be less than "expected" cancers obtained by linear extrapolation] would be led to the incorrect conclusion that for all radiation, the linear hypothesis will over-estimate the effects.

Other members [of ACRP] include Dr. Jan Muller from the Department of Labour, Ontario, [who is of the opinion] that there is no serious risk at current standards of 4 WLM per year of radon exposure, despite mounting evidence to the contrary. No follow-up study on the Ontario uranium miners has been completed because the information is still being processed by Dr. Muller. It is unfortunate, because of the crucial nature of the Ontario studies, that there has been such a long delay since 1976. It is hoped that this data will be available to the scientific community soon.

Dr. Butler also noted that his Committee did have Dr. Stuart from AECL, but that he had now retired. [ACRP now includes both Dr. Myers and Dr. Newcombe, both of AECL.]

Dr. Butler agreed that his Committee had not asked any independent bodies, such as the Canadian Medical Association, the Royal College of Physicians and Surgeons, or the Royal Society, to place a member of its own choice on the Committee.

The "independence" of this Committee must be seriously questioned. This lack of "independence" is characteristic of the AECB. As Dr. Bates noted about the previous Standing Committee on Safety,

> There appeared to be only one M.D. on it, and he had worked at Chalk River for all of his life before that.

RADON GAS IN HOMES: AN INDUSTRIALLY-INDUCED EPIDEMIC?

> The Atomic Energy Control Board has announced adoption of radiation criteria for use in the investigation and clean-up of communities contaminated by radiation.

The Government of British Columbia has adopted the AECB exposure limits [for public exposure to radon daughters]:

> The WLM unit is not appropriate for exposures in the home or in other non-occupational situations. In such situations the maximum permissible annual average concentration of radon daughters (attributable to the operation of a nuclear facility) shall be 0.02 WL.

[Outdoor] levels higher than 0.02 WL may be produced locally by uranium mines. Higher outdoor concentrations would obviously produce higher indoor concentrations of radon. [According to Dr. Wagoner:]

> On the basis of additional data, the EPA has estimated that 110 to 230 extra lung cancer deaths would occur among 100,000 population with a lifetime residency at ambient levels of radon daughter exposure (i.e. 0.004 WL). In contrast, 2000 to 3000 extra lung cancer deaths per 100,000 population were estimated to occur over a lifetime indoor radon daughter exposure to 0.02 WL.

In light of the present state of knowledge, one could well view the allowable exposure to the public from nuclear facilities as tantamount to allowing an industrially-induced epidemic of cancer.

Dr. Radford in his submission to the Commission stated that

> epidemiological and experimental evidence indicates that alpha radiation is more effective (per unit dose) in producing cancer when exposure is at low dose rates over long periods of time, than when the equivalent dose is given at a high rate for short periods of time.

Dr. Archer observed that

> Alpha radiation appears to be approximately eight times as efficient at 100 WLM as at 1000 WLM. This data makes it highly likely that radon daughter levels in residences are responsible for some lung cancers.

In 1971, the joint monograph by NIOSH and NIEHS also noted:

> The risk of respiratory cancer per unit of exposure appeared to be greater in the lower cumulative radiation groups than in the higher ones -- i.e. an assumption of linearity appears not to be conservative [does not overestimate the actual risks].

Nevertheless, the AECB assumes that this [linear hypothesis]

> is a cautious assumption; i.e. the number of cancer cases will probably be overestimated.

EXHIBIT 12

13 January 1984

TO WHOM IT MAY CONCERN:

As there appears to be some confusion among representatives of industry and government with respect to the British Columbia Medical Association's efforts as a major participant in the British Columbia Royal Commission of Inquiry, Health and Environmental Protection - Uranium Mining, we wish to make the following comments:

1) Dr Eric R Young and Dr Robert F Woollard participated as intervenors at the Inquiry as representatives of this Association.

2) Dr Young is presently the chairman of the environmental health committee of the BCMA and Dr Woollard is past-chairman.

3) During the Inquiry the BCMA was privileged to present statements of evidence of internationally-recognized authorities on various aspects of this issue.

4) The report entitled "The Health Dangers of Uranium Mining and Jurisdictional Questions" authored by Drs Young and Woollard is the summary argument of the BCMA presented in 1980 to the Royal Commission in response to its call for final arguments from participants in the Inquiry. As such it has been supported by the BCMA Executive and Board of Directors.

5) This report has had significant peer review and there has been ample opportunity for public comment.

6) The substance of the report is reflective of BCMA's policies in the area of environmental health as established over several years by consideration and debate at the general assembly and Board of Directors and, as confirmation of this, the BCMA holds copyright on both printings of this BCMA publication.

Extensive feedback has confirmed the report's value as an aid in promoting public participation in this important area of environmental health and has vindicated the medical association's expressed interest to raise the level of debate on this issue.

Yours sincerely

G D McPherson, MD
President

M/ERY/jh

BRITISH COLUMBIA MEDICAL ASSOCIATION

ACADEMY OF MEDICINE BUILDING 1807 West 10th Avenue, Vancouver, B.C. V6J 2A9 Telephone (604) 736-5551

EXHIBIT 13

Verbatim Excerpts from the BEIR-III Report

The Effects on Populations of Exposure to Low Levels of Ionizing Radiation: 1980

COMMITTEE ON THE BIOLOGICAL EFFECTS OF IONIZING RADIATIONS
Division of Medical Sciences
Assembly of Life Sciences
National Research Council

National Academy Press

The National Academy Press was created by the National Academy of Sciences to publish the reports issued by the Academy and by the National Academy of Engineering, the Institute of Medicine, and the National Research Council, all operating under the charter granted to the National Academy of Sciences by the Congress of the United States.

NATIONAL ACADEMY PRESS
Washington, D.C. 1980

THE LUNG

Lung cancer - or more properly, bronchial cancer - was the first internal cancer of which exposure to ionizing radiation was implicated as a cause (in Bohemian miners). As follow-up investigations of radiation-exposed groups have been extended, bronchial cancer has emerged as one of the most important radiation-induced cancers. Since the 1972 BEIR report, our understanding of radiation induction of bronchial cancer in man and lung tumors in animals has advanced considerably.

Czechoslovakian Uranium Miners

The exposure in the Czechoslovakian mines was relatively slight: if the underground work experience was 20 years or more and the average cumulative exposure was about 300 WLM, then the concentrations of radon daughters were about one working level - much lower than in the US uranium mines before 1960.

The lung-cancer risk estimates were given by the [Czech] authors simply as excess cases per 1000 miners.... Precise correction of the published relative-risk estimates to eliminate the latent-period years is not possible; but ... an approximate value is obtained of 1.8 percent excess lung-cancer risk per WLM over the period under study. This value indicates a doubling dose of about 56 WLM [cumulative].

A substantial excess of lung cancer has already begun to occur in the nonsmokers among these miners.... Because the latent period for lung-cancer induction in non-smokers is longer than that for smokers, with further follow-up the relative risk would be expected to rise more rapidly for nonsmokers than for smokers.

United States Uranium Miners

The US uranium miners had exposures to high concentrations of radon daughters; at least before 1960, the radon-daughter concentrations ranged generally from 10 to 100 or more working levels. This explains the fact that the average cumulative exposure, 1180 WLM, is well above that of most of the other mining populations studied.... The lower exposure groups have risk estimates 2 or 3 times those for the highest dose groups.

If we consider only the data for [US] miners exposed to less than 360 WLM ... the relative risk is 0.8 percent per WLM. These values indicate a risk well below the results for the Czecho-slovakian miners with comparable total cumulative doses. This difference cannot be explained by smoking experience, and the American miners have had about the same follow-up as the Czecho-slovakian miners. A possible explanation for the lower risk in the US miners is the high dose rate at which exposure occurred. An increased bone-cancer effect from a reduced dose-rate of alpha-radiation exposure from radium-224 has also been observed.

Canadian Uranium Miners

Although exposures were below one working level, except in a few mines, a significant excess risk of lung cancer has been observed.... The relative risk of 81/45.1 [85 lung cancer cases observed compared with 45.1 expected] or 1.8, is undoubtedly an underestimate, because of incomplete ascertainment of cases and because of the inclusion of years at low risk during the latent period in calculating the expected deaths.

A plot of lung-cancer deaths, as an estimated proportion of the population born before 1933, versus cumulative exposure in WLM, gives a reasonably linear relationship, the slope being such that the crude doubling dose is about 12 WLM. This latter figure is not an accurate indication of the relative risk, [but] a more complete analysis may well show this group of miners to be at high risk.

The lowest cumulative dose category in this analysis was 1-30 WLM, in which 29 lung cancer deaths were recorded.... The doubling dose for this low-dose group would be 17 WLM, in reasonable agreement with the analysis discussed above. Although this assessment is tentative, the data suggest an excess risk for these miners at this very low cumulative-dose range. The importance of an adequate epidemiologic follow-up of this mining population is obvious.

Newfoundland Fluorspar Miners

Estimates of radon-daughter concentrations varied from 2 to 8 working levels, according to the type of work, during the period up to 1960, when with improved ventilation they decreased to below 0.5 working levels. Exposures in these mines were therefore substantially lower than in the US uranium mines, but somewhat higher than in the Czechoslovakian or Canadian uranium mines.

Sixty-five deaths from lung cancer have occurred among the underground miners (lung cancer was the cause of 27 percent of all deaths up to 1971) and six among the surface workers (4 percent of all deaths).... For the entire group of underground miners during the years under study, the expected number of [cancer] deaths was 3.76.... In this group, the relative risk was 8.0 percent per WLM, which yields a doubling dose of 12.5 WLM.

Swedish Metal Miners

Several reports of lung-cancer excess among Swedish metal miners have been published. A number of these reports are preliminary and include incomplete follow-up or material on only active miners. Therefore, it is not possible to determine risk estimates from them.

Axelson and Sundell have recently published data on a group of zinc miners studied for the period 1956-1976.... Radon concentrations have been extensively measured in the shafts since

1969 and ... have been found to be equivalent to 0.3 to 1.0 working levels..... Twenty lung-cancer deaths have been observed, compared with 2.32 expected.... The mean cumulative exposure is estimated at 270 WLM.... This group of miners has had very long follow-up into retirement.... These data would indicate little difference in radiation risk between smokers and non-smokers....

Forty-five lung-cancer deaths have been observed between 1953 and 1976 in Swedish iron mines at Malmberget - a larger group than the zinc miners, but also with very long follow-up.... The study is not yet completed, but [the data] indicate that the excess risk for smokers may not be markedly greater than that for nonsmokers. The very long follow-up of these Swedish groups is an important factor in determining risk estimates for nonsmokers, because of the long[er] latent period that may be observed in these cases.

Summary of Risk Estimates

The Newfoundland fluorspar miners and Czechoslovakian uranium miners have risk estimates very comparable with those for the entire population [of underground miners]; the Swedish zinc miners have higher estimates ... apparently because they have been followed to a greater age.

The US uranium miners have risk estimates well below those of all the other groups. Only two explanations seem reasonable to account for this difference: either the radon-daughter measurements in the US mines have overestimated exposures by as much as a factor of three (not likely, in view of the great efforts made to obtain this information), or the much higher dose rate (working levels in the mines) has led to less risk per unit of cumulative exposure than the lower working levels in the other mines.

The most likely risk estimates, at an exposure of about one working level and with characteristic smoking experience, are

about 10 cases per million person-years per WLM
for the age group 35-49,

20 cases per million person-years per WLM
for the age group 50-65,

and about 50 cases per million person-years per WLM
for those over 65.

The following tables, compiled by Dr. Duncan Thomas of McGill University at the request of Dr. Gordon Edwards, utilize these BEIR risk estimates to calculate the expected lung cancer mortality in a population of men exposed
(1) at the maximum permissible occupational level and
(2) at the maximum permissible level for radon gas in homes.

(Retyped from a computer printout supplied by Dr. Duncan Thomas)

TABLE 1. LIFETIME RISK OF LUNG CANCER RESULTING FROM 50 YEARS EXPOSURE TO RADON AND ITS DAUGHTERS AT TODAY'S OCCUPATIONAL LIMITS

Occupational Exposure to 4 WLM per year from age 15 to 64.

Method of Projection: Absolute Risks per Unit Dose Varying with Age (according to estimates in BEIR, page 325)

⇒ Initial Population: 100,000 men

AGE GROUP	TOTAL RADON DOSE	CANCER RISK FACTOR	DEATH RATES LUNG CANCER	DEATH RATES OTHER DEATHS	OVERALL PROB OF DYING	NUMBER ALIVE AT START	LUNG CANCER DEATHS	PERSON-YEARS
15-19	10	.00000	.000000	.00168	0.00835	100,000	0	497,908
20-24	30	.00000	.000001	.00206	0.01028	99,165	0	493,272
25-29	50	.00000	.000003	.00151	0.00755	98,145	1	488,876
30-34	70	.00000	.000007	.00158	0.00791	97,405	3	485,096
35-39	90	.00001	.000947	.00228	0.01603	96,634	454	479,289
40-44	110	.00001	.001265	.00339	0.02301	95,085	594	469,936
45-49	130	.00001	.001671	.00539	0.03469	92,897	763	456,382
50-54	150	.00002	.003828	.00834	0.05905	89,674	1665	434,999
55-59	170	.00002	.004722	.01369	0.08795	84,379	1903	403,057
60-64	190	.00002	.005992	.02084	0.12555	76,958	2158	360,094
65-69	200	.00005	.012751	.03193	0.20021	67,296	3845	301,543
70-74	200	.00005	.013821	.04814	0.26641	53,823	3198	231,417
75-79	200	.00005	.013850	.07570	0.36094	39,484	2204	159,141
80-84	200	.00005	.013948	.11794	0.48286	25,233	1289	92,380
85-99	200	.00005	.012197	.21587	1.00000	13,049	698	57,215
TOTALS							18,776	5,410,595

Expected Lung Cancer Deaths (in the General Population) = 5,342.

⇒ Excess Lung Cancer Deaths (Due to Occupational Exposure) = 13,434.

Notes (by Gordon Edwards, after consultation with Dr. Thomas)

1. At 4 WLM per year over 50 years, the incidence of lung cancer is almost quadrupled over the "normal" incidence of lung cancer.
2. To calculate excess lung cancers due to a lower dose rate, divide the excess lung cancer deaths cited above by the appropriate factor. (For example, at 1 WLM per year for 50 years, only one-quarter as many excess lung cancer deaths would be observed: 13,434/4 = 3,358. This is probably an underestimate, since lower dose rates seem to result in higher risk factors.)
3. The risk factors are taken from BEIR, p.325, and refer to lung cancer.
4. Death rates are annual death rates; the lung cancer rate includes the radon risk factor derived from BEIR.
5. Probability of dying refers to the entire five-year interval. It is slightly different than five times the total annual death rate, because of population changes during the five-year interval.
6. During the first and last five-year interval, only 10 WLM are credited to the total radon dose; this is because the average radon exposure is of most significance during each interval.

(Retyped from a computer printout supplied by Dr. Duncan Thomas)

TABLE 2. LIFETIME RISK OF LUNG CANCER RESULTING FROM HOUSEHOLD EXPOSURE TO RADON AND ITS DAUGHTERS AT TODAY'S ACCEPTABLE LIMITS

Household Exposure to .02 WL for 17 Hours per Day from Birth to Death

Method of Projection: Absolute Risks per Unit Dose Varying with Age (according to the risk estimates appearing in BEIR, page 325)

AGE GROUP	TOTAL RADON DOSE	CANCER RISK FACTOR	DEATH RATES: LUNG CANCER	DEATH RATES: OTHER DEATHS	OVERALL PROB OF DYING	NUMBER ALIVE AT START	LUNG CANCER DEATHS	PERSON-YEARS AT RISK
00-19	6.387	.00000	.000000	.00168	0.00835	100,000	0	497,908
20-24	8.212	.00000	.000001	.00206	0.01028	99,165	0	493,272
25-29	10.037	.00000	.000003	.00151	0.00755	98,145	1	488,876
30-34	11.863	.00000	.000007	.00158	0.00791	97,405	3	485,096
35-39	13.688	.00001	.000184	.00228	0.01227	96,634	88	480,203
40-44	15.512	.00001	.000320	.00339	0.01838	95,449	151	472,844
45-49	17.337	.00001	.000544	.00539	0.02924	93,694	251	461,587
50-54	19.162	.00002	.001211	.00834	0.04666	90,954	538	444,077
55-59	20.987	.00002	.001742	.01369	0.07426	86,710	727	417,248
60-64	22.813	.00002	.002648	.02084	0.11081	80,272	1003	378,686
65-69	24.637	.00005	.003983	.03193	0.16437	71,377	1301	326,677
70-74	26.462	.00005	.005144	.04814	0.23388	59,645	1347	261,803
75-79	28.287	.00005	.005264	.07570	0.33290	45,695	989	187,886
80-84	30.112	.00005	.005454	.11794	0.46042	30,483	620	113,742
85-99	31.938	.00005	.003794	.21587	1.00000	16,448	284	74,878
TOTALS							7305	5,584,776

Expected Lung Cancer Deaths (in the General Population) = 5,342

⇒ Excess Lung Cancer Deaths (Due to Household Exposure) = 1,963

At .02 WL exposure to radon, the lifetime risk of lung cancer is increased by almost 37 percent, according to the BEIR estimates.

REMARK

These calculations are based on the BEST ESTIMATES of risk given in the lung cancer section of BEIR-III (1980). At occupational levels of exposure, the lifetime risk (per WLM) is about 6.7 extra lung cancer cases per 10,000 people exposed. However, at lower rates of exposure - for example, in radon-contaminated homes - the lifetime risk per WLM is likely to be greater:

"The US uranium miners have risk estimates well below those of all other groups. Only two explanations seem reasonable to account for this latter difference: either the radon-daughter measurements in the US mines have over-estimated exposures by a factor of three (not likely, in view of the great efforts made to obtain this information) or the much higher dose rate (working levels in the mines) has led to less risk per unit of cumulative exposure than the lower working levels in the other mines.... An increased bone-cancer effect from a reduced dose rate of alpha-radiation from radium-224 has also been observed." (See Figure 7, next page.)

FIGURE 7
Effect of Dose-Rate on Bone-Cancer Incidence

==========

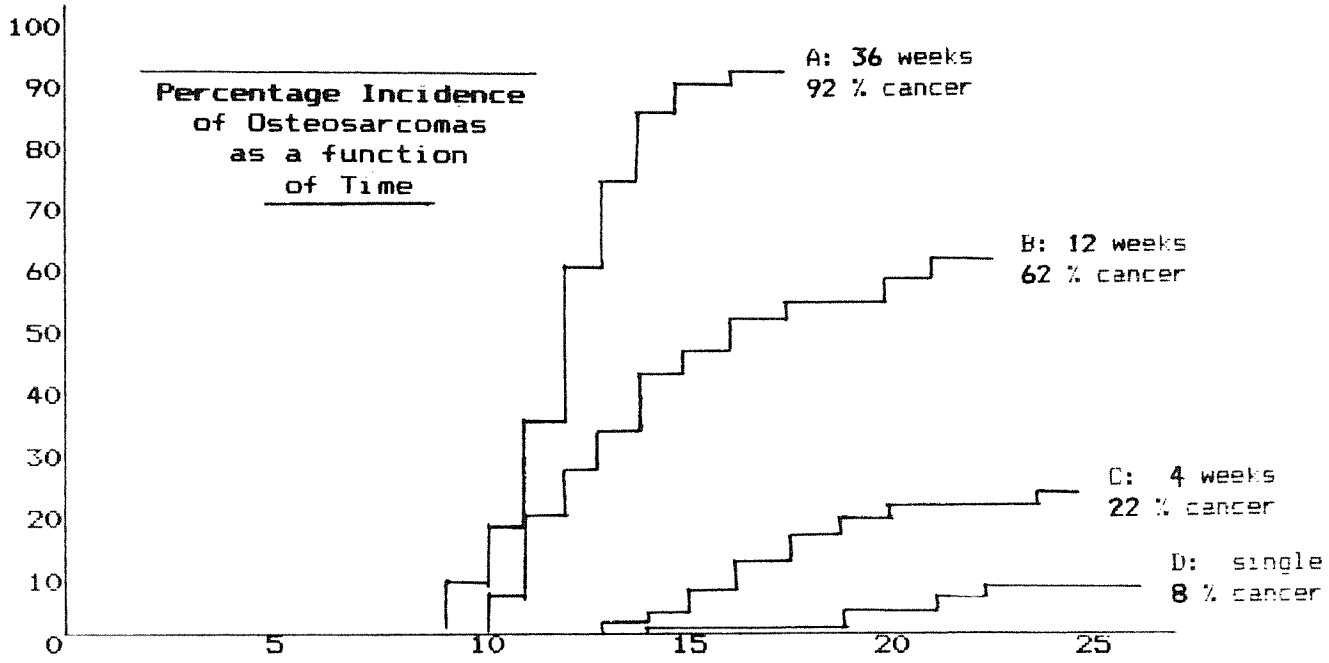
In this experiment, female mice divided into four populations were injected with equal doses of radium-224 (36 microcuries per kg body weight). The only difference between the four populations was the rate at which the same total alpha radiation dose was administered.

==========

Populations (According to Dose-Rates)

A: 0.5 microcuries per kg, injected twice a week, for 36 weeks
B: 1.5 microcuries per kg, injected twice a week, for 12 weeks
C: 4.5 microcuries per kg, injected twice a week, for 4 weeks
D: 36 microcuries per kg, one single injection at the outset

(In each case, the mean skeletal dose was about 1000 rads.)



The vertical axis measures the percentage of osteosarcomas (bone cancers) observed in each of the four populations.

The horizontal axis indicates the number of months which have elapsed since the beginning of the experiment.

The results are striking: 92 percent of group A developed bone cancer, while only 62 percent of group B, 22 percent of group C & 8 percent of group D contracted the disease - with equal doses!

Source: Figure 7 in "Late Effects After Incorporation of the Short-Lived Alpha Emitters Radium-224 and Thorium-227 in Mice," by W. A. Mueller, W. Goessner, O. Hug and A. Luz in Health Physics, vol. 35, pp. 33-75 (July 1978).

EXHIBIT 14
Verbatim Excerpt from the NIOSH Report

THE RISK OF LUNG CANCER AMONG UNDERGROUND MINERS OF URANIUM-BEARING ORES

NIOSH STUDY-GROUP REPORT - JUNE 30 1980

Published by the

U.S. National Institute of Occupational Safety & Health

SUMMARY AND CONCLUSIONS:

(excerpts)

The earlier predictions of excess lung cancer among miners of uranium-bearing ores in the United States and in other countries have been documented and repeatedly confirmed. Recent studies of uranium and non-uranium underground miners have raised the concern that an increased risk of lung cancer mortality may persist even if miners are exposed only to radiation within the radon daughter exposure limit defined by the present standard [4 WLM per year: see note]. The question addressed in this report is how well the miners are being protected by keeping exposures within that limit.

A critique of the relevant literature has been completed and bears directly on the health hazards associated with contemporary underground mining of uranium-bearing ores. There is a clear indication that cumulative exposure to radon daughters is associated with increased risk of lung cancer for workers in underground mines generally and uranium mines specifically. There is also strong evidence that a substantial risk extends down to and below 120 WLM of exposure [see note].

The exact magnitude of the risk cannot be precisely quantified. However, studies of underground miners occupationally exposed to radon daughters in several countries lead to the conclusion that at these levels of exposure (below 120 WLM) an excess risk of lung cancer mortality is evident (greater than two-fold) and of sufficient magnitude to be of major public health concern. This appears to be true for both high and low exposure rates.

When the present standard (4 WLM per year) is evaluated in terms of the magnitude of the dose delivered and its predicted biological effect, a sense of the relative degree of protection provided by the standard can be made. Estimates of the risk per WLM are at least 2 to 4 times greater now than the estimates that were made 10 years ago. This leads to the conclusion that there is no margin of safety associated with the present standard.

NOTE

A "working level month" (WLM) is a crude measure of the amount of radon and its daughters inhaled into the lungs. Thus the present standard of 4 WLM per year is essentially an annual intake limit. Over a 30-year working lifetime, a miner exposed at this level would accumulate 120 WLM. The AECB wants to increase the maximum permissible intake to 4.7 WLM per year -- a 17 percent increase.

EXHIBIT 15
Verbatim Excerpts from the Thomas/McNeill Report

Atomic Energy Control Board
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Commission de contrôle de l'énergie atomique
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INFO-0081

RISK ESTIMATES FOR THE HEALTH EFFECTS OF ALPHA RADIATION

by

Duncan C. Thomas and
K.G. McNeill

A report prepared for the
Atomic Energy Control Board
Ottawa, Canada

RESEARCH REPORT

September 1982

ABSTRACT

This report provides risk estimates for various health effects of alpha radiation. Human and animal data have been used to characterize the shapes of dose-response relations and the effects of various modifying factors, but quantitative risk estimates are based solely on human data: for lung cancer, on miners in the Colorado plateau, Czechoslovakia, Sweden, Ontario and Newfoundland; for bone and head cancers, on radium dial painters and radium-injected patients.

Slopes of dose-response relations for lung cancer show a tendency to decrease with increasing dose. Our best estimate of curvilinearity is given by raising dose to the power 0.92 ± 0.07, but the improvement in fit beyond simple linearity is not significant. On the other hand, the addition of a cell-killing term significantly improves the fit of the linear model. In any event, linear extrapolation is unlikely to underestimate the excess risk at low doses by more than a factor of 1.5. However, these inferences about curvilinearity are highly subject to error from the choice of reference populations, dosimetry, and latency. Under the linear cell-killing model, our best estimate of excess relative risk is 2.28 ± 0.35 per 100 WLM (a doubling dose of 44 WLM). Attributable risks in these five studies range from 3.4 to 17.8 per 10^6PY-WLM.

Risks from radon daughters appear to interact with age and smoking in a form intermediate between additive and multiplicative, though on balance, closer to multiplicative. We therefore favor the "relative risk model" for projecting lifetime risks, but have carried out life-table projections under a wide variety of assumptions. Our best estimate of the effect of a 50-year occupational exposure to 4 WLM/yr is 130 excess lung cancer deaths per 1000 persons (0.65 per 1000 person-WLM), with a range from 60 to 250 per 1000. Similar calculations for lifetime exposure to an additional 0.01 WL beyond normal background produces an estimate of 10 excess lung cancers per 1000 persons. *

Our risk estimate for radium are 2.4 bone sarcomas and 2.0 head carcinomas per 10^5Py-μCi. The lifetime risk from radium in drinking water at the Canadian MAC is about 0.4 per 1000 persons, compared with a natural risk of 1.0 per 1000.

No major health effects of plutonium have yet been demonstrated in human populations, probably because of the small number of persons exposed to significant doses, though animal studies clearly show its carcinogenic potential. Other effects of alpha emitters which have been reported include gastrointestinal, skin, and liver tumors, leukemia, liver cirrhosis, and chromosomal abnormalities, but these require further study before their risks can be adequately described.

μCi = microcurie = 10^{-6} curies

MAC = maximum acceptable concentration

PY = person-years

WLM = working-level month

CHAPTER 9.1: CONCLUSIONS

(excerpts)

1. The lung is a major radiosensitive site, and for short-lived or insoluble inhaled alpha emitters [including radon gas] it is the primary concern.

2. Our best estimate for the excess relative risk [associated with radon] is 2.28 ± 0.35 [times the normal incidence of lung cancer] per hundred WLM, which gives a doubling dose of 44 WLM.

3. Considering the differences in populations and methods, and comparing these results with those of other epidemiological studies (such as those on asbestos), there is a remarkable degree of agreement between the various studies [Colorado, Czechoslovakia, Newfoundland, Ontario and Sweden]. Only the Colorado plateau data stand out as giving very much lower risk estimates than the other studies of miners.

6. There is no justification for assuming that linearity would be a conservative basis for radiological protection for high-LET effects. [In other words, it cannot be assumed that the linear hypothesis will over-estimate the biological damage from alpha radiation; it may in fact under-estimate the damage.]

7. On the basis of a linear dose-response model with a constant relative risk of 2.28 per 100 WLM, the 4 WLM per year standard for occupational groups could increase the lung cancer risk of an individual working all his life at the maximum by a factor of from 2.4 to 6.2 (best estimate 3.8).

8. This is of course an estimate of the maximum risk obtained at 50 years of constant exposure to the maximum level of 4 WLM per year. It should be recognized that average exposures and hence average risks would be very much lower. [If average exposures are only one-tenth of the maximum], the 4 WLM per year standard would on average over the entire industry produce a 10 to 20 percent increase in the risk of lung cancer.

10. On a similar basis, a 0.02 WL maximum [radon exposure] for homes could increase the lifetime lung cancer risk [of people living their lives in such homes] by about 40 percent. However, this is the predicted increase for a lifetime of additional 0.02 WL exposure beyond normal background levels (which vary widely but might reasonably be as much as one third of 0.02 WL).

16. Protraction of dose [that is, delivering the same dose over a longer period of time] appears to increase risks of lung cancer (and other effects). For this reason, epidemiological studies of populations exposed for relatively short periods may underestimate the risks of life-long exposure.

.../

CHAPTER 9.2: RECOMMENDATIONS

1. In view of [analytical uncertainties] these conclusions should be considered to be highly tentative. However, as the necessary data already exist for several mining populations, many of these limitations could be overcome relatively quickly by reanalyses. We therefore recommend that linearity be treated only as a temporary ad hoc basis for interim standards, which should incorporate a "safety factor" to allow for the possibility of convexity [whereby actual risks are greater than those anticipated by the linear hypothesis]; this safety factor need not be more than two-fold. The conclusions should be reviewed after better analyses have been carried out on existing or improved data.

2. The Ontario and Newfoundland mining populations should continue to receive the best possible follow-up; in particular, efforts should be made to obtain smoking histories, to evaluate the contribution of radiation to the reported risks of gastrointestinal and skin cancer, and to use the best available statistical methods.

5. Consideration should be given to initiating a cohort study of family members of Ontario uranium miners during the 1950-1960 boom, and a case-controlled study of lung cancer in relation to radon daughter levels in homes. Of the two, the case-control study would probably be the more feasible starting point. We estimate that a sample size of about 500 cases and 500 controls would be sufficient to demonstrate a significant association with lung cancer risk. Should it prove feasible to identify and trace family members of Ontario uranium miners, similar sample size calculations could be easily done.

7. The criteria for awarding compensation for lung cancer in radiation workers should be re-examined in the light of the evidence contained herein. We suggest that on the average, 50 percent of the lung cancers with an accumulated exposure of about 44 WLM (the "doubling dose") would have been radiogenic in origin. The onus of establishing causation should not be placed on the individual concerned.

9. In our view, the present 4 WLM per year maximum does not provide sufficient protection to an individual who works at that level for an entire lifetime, though for an entire group of uranium workers the average risk resulting from the use of this individual maximum might be considered acceptable. We recommend that regulations setting permissible annual exposures be supplemented by additional explicit requirements that average exposures to all relevant (i.e. potentially exposed) workers in a company be very much lower.

   It is also desirable that few workers ever accumulate total lifetime exposures approaching the maximum possible under present regulations (approximately 200 WLM). We therefore recommend that lower maximum individual doses be encouraged by education of the workers themselves, maintenance of low average doses, and non-discriminatory policies of job rotation.

Uranium miners facing higher risk of lung cancer death, study says

By BARRY KLIFF
of The Gazette

The Atomic Energy Control Board (AECB) says there is no substantiation to a study it commissioned, which shows uranium miners face a greater risk of dying from lung cancer than the general public.

The $15,000 report commissioned by the board says Canada's 4,500 uranium miners are four times more susceptible to lung cancer death.

It says the board should consider tougher controls on radioactive gases, especially radon, emitted during mining which is an apparent cancer cause.

But the board wants to ease controls on radon, said William Bush, manager of the board's radiation protection division.

Bush said workers are adequately protected. He said international studies have shown it is possible to raise the amount of radon gas a miner is exposed to while decreasing his over-all exposure to radioactive gases.

"There is no substantiation in the authors' report for the conclusions they have drawn," Bush said. "I don't think anything would be gained from studying the matter any more."

One of the report's co-authors, Duncan Thomas, associate professor of epidemiology at McGill University, said other international studies show uranium miners aren't adequately protected against radon gas.

To reach his conclusion, Thomas compared results of five studies involving 18,000 miners in Canada, Czechoslovakia, Sweden and the United States. It was not original research.

"If the AECB responds by raising the limit, I feel our work has been in vain," Thomas said

Each year among the general public, Thomas said, five per cent of all deaths are due to lung cancer. But under today's standards for miners, Thomas said, the risk could reach 20 per cent.

In two studies, involving a total of 5,500 miners in Newfoundland and the U.S., Thomas said, researchers expected to find 35 cases of death caused by lung cancer but found 224.

Bush said various AECB committees are studying Thomas' report now and a final report is expected early next year.

Page 12, The Citizen, Ottawa, Wednesday, October 20, 1982

Canada

AECB disregards report saying radon-gas standards inadequate

By Mitchell Beer
For Southam News

The Atomic Energy Control Board has chosen to disregard a research report it recently commissioned that concludes current standards for exposure to radon gas are inadequate.

The report found uranium miners exposed to the radioactive gas over a full working life could run a 20- to 30-per-cent risk of dying of lung cancer, compared to about five per cent for the general population.

"The conclusion we arrive at ... is that the standard does not provide adequate protection to an individual, and the only justification for such a high standard can be that the average exposure to the entire work force is very much lower," said Duncan Thomas, associate professor of epidemiology at McGill University and co-author of the report.

The report also found maximum standards for radon exposure in homes would give residents a 40-per-cent higher than average lung cancer risk.

William Bush, manager of AECB's radiation protection division, said a board advisory committee has reviewed the available literature on radon gas, including the research report. "Their conclusion is that there is no basis for reducing the limit."

Thomas said the average risk a group of miners would face might appear low, but "the maximum risk an individual can theoretically run with today's standards is not acceptable or negligible. It's certainly a measurable increase in risk."

Based on his findings, Thomas said the increased risk of lung cancer for miners and occupants of radon-exposed homes "are substantial enough that they ought not be dismissed, and serious consideration should be given to the possibility of lowering the standards." But Bush said AECB is now considering a 20-per-cent increase in allowable radon exposure, in line with changes in international standards.

Because the increase is part of an integration of standards for various types of radiation, the net effect is a tougher overall limit, Bush said. "In effect you're reducing it, because people are never exposed to radon alone."

But Gordon Edwards, head of the Montreal-based Canadian Coalition for Nuclear Responsibility, said AECB staff are on record as saying radon exposure up to 100 times the current annual limit would be safe. Edwards said medical doctors have told him they know of no other carcinogen with such high permissible exposure levels.

"The AECB has for years been reassuring people that the standards are set so stringently that there's no significant risk," he said, "but according to the Thomas-McNeill findings, the risk is very significant."

EXHIBIT 16
Atomic Energy Control Board Miscalculation of the Radon Risk

Atomic Energy Control Board
Commission de contrôle de l'énergie atomique

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INFO-0090

AECB used this 13-page report to dismiss the 300-page Thomas/McNeill Report.

ACRP Report

ACRP-1

RISK ESTIMATES FOR EXPOSURE TO ALPHA EMITTERS

by the

Advisory Committee on Radiological Protection

A report to the
Atomic Energy Control Board
Ottawa, Ontario

This report MISQUOTES the risk estimates given in the Thomas/McNeill Report, and MISCALCULATES the risk estimates for uranium mining from the BEIR-III Report.

ADVISORY COMMITTEE REPORT

July 1982

A report submitted to the Atomic Energy Control Board by the Advisory Committee on Radiological Protection.

ABSTRACT

The primary scope of this report is to evaluate the risk of lung cancer from occupational exposure to short-lived daughters of radon and thoron. The Subcommittee on Risk Estimates (SCRE) considers that inhalation of radon and thoron daughters is the major radiation hazard from alpha radiation in uranium mining.

The secondary scope of this report is the consideration of the applicability of the risk estimates derived from miners to the general public.

The risk to members of the public from radium-226 in drinking water is also condidered.

Some research requirements are suggested.

Preface

See BCMA comments page 49.

Since the 1950's the Atomic Energy Control Board has made use of advisory committees of independent experts to assist it in its decision-making process. In 1979 the Board restructured the organization of these consultative groups resulting in the creation of two senior level scientific committees charged with providing the Board with independent advice on principles, standards and general practices related to radiation protection and the safety of nuclear facilities. The two committees are the Advisory Committee on Radiological Protection (ACRP), which held its first meeting in May, 1979, and the Advisory Committee on Nuclear Safety (ACNS), which was established a year later.

The records of meetings are filed in the AECB Library, and reports are catalogued and published as part of the Board's public document collection. Reports carry both a committee-designed reference number, e.g. ACRP-1, or ACNS-1, and an AECB reference number in the "INFO"-series.

Acknowledgements

The following report was prepared by the Standing Subcommittee on Risk Estimates (SCRE) of the Advisory Committee on Radiological Protection (ACRP) and endorsed by the ACRP at its May 1982 meeting.

The members of the Subcommittee on Risk Estimates at the time of preparation of this report were:

Dr. J. Muller, Chairman
Dr. T.W. Anderson
Dr. G.W. Gibbs
Dr. D.K. Myers } AECL
Dr. H.B. Newcombe }

Dr. V. Elaguppillai, Secretary

See Myers' Testimony pp. 71-73

See pp. 61-65

The Subcommittee made use of the report on, "Risk Estimates for the Health Effects of Alpha Radiation", prepared by D.C. Thomas and K.G. McNeill under a contract with the Atomic Energy Control Board,

Note

EXHIBIT 16

EXECUTIVE SUMMARY

Based on the consideration of practical needs to protect workers and the public from undue exposure to alpha radiation, the Subcommittee on Risk Estimates decided to give the evaluation of the health effects of inhalation of radon-222 and its short lived daughters the highest priority. The effect of radon-220 (thoron) and its short lived daughters was also considered because of the presence of these nuclides in some Canadian uranium mines. The Subcommittee also considered the health effects of ingestion of two naturally-occurring radium isotopes, radium 226 and radium 228.

The Subcommittee reviewed current research and considered future research requirements in Canada that might help to improve risk estimates for exposure to alpha emitters.

Type of data considered:

The effects of exposure to alpha radiation can be studied using simple biological models, experimental animals, observations on human populations, and microdosimetric techniques.

The Subcommittee found that each of the above approaches has its advantages and shortcomings. However, for the purpose of deriving quantitative risks estimates, it is most desirable to use human observations whenever possible, and resort to other approaches, mostly microdosimetry, whenever no useful human data are available.

NOTE!

Risk estimates for miners derived from epidemiological data:

A number of epidemiological studies, including studies of uranium miners in the U.S.A., Czechoslovakia and Ontario, fluorspar miners in Newfoundland and metal miners in Sweden, were reviewed by the Subcommittee.

The studies on uranium miners in the U.S.A. and Czechoslovakia were considered best suited for the purpose of risk evaluation.

] NOT Canada or Sweden!?!

Having examined the available data and their limitations, the Subcommittee concluded that the exact shape of the exposure-response relationship cannot be established with certainty. However, the best estimates of lifetime risk, as based on a linear relationship are not substantially different from the corresponding estimates obtained from a supralinear (convex) relationship. Differences between these estimates are of no practical significance.

The Subcommittee noted limitations in the various epidemiological studies on which risk estimates for miners exposed to radon daughters are based. In all cases, assumptions have to be made to extrapolate

from observed risks over a limited period of observation to lifetime risk. The use of the relative risk model for extrapolation beyond the period of observation yields higher risk estimates, in terms of predicted life time excess cancer cases per unit exposure, than does the absolute risk model. The predicated loss of life expectancy calculated by the two approaches is not greatly different.

In risk estimates given by various agencies, different weights were assigned to various epidemiological studies. None of the agencies, however, had the benefit of the use of raw data for analysis. Since all agencies considered the same published data, it is not surprising that they arrived at similar ranges of risk estimates; minor differences are mainly due to the various weights given to the different studies and to the various models used to extrapolate to lifetime risk.

A summary of the various risk estimates for exposure to radon daughters are given in the following table:

Excess Cancers (Lifetime Risk) for 1000 men at 4WLM for 50yr	Authors	Lifetime risk per WLM	
40-90	UNSCEAR 1977	$2 - 4.5 \times 10^{-4}$	(1)
14	UNSCEAR 1977 (Colorado data)	0.7×10^{-4}	(2)
40-120	BEIR 1980	$2 - 6 \times 10^{-4}$	(3) Miscalculated
30-90	ICRP 1981	$1.5 - 4.5 \times 10^{-4}$	(4)
10-240	Thomas & McNeill 1982	$0.5 - 12 \times 10^{-4}$	Misquoted
20-120	SCRE 1982	$1 - 6 \times 10^{-4}$	(5)

For 50 years exposure at 4WLM, just multiply by 200.

The Subcommittee considers that the risk estimates by national and international agencies, as given above, are compatible with published epidemiological information, and that the lifetime risk of lung cancer incidence for miners is probably in the range of 1 to 6 x 10^{-4} per WLM. (For Miners)

Risk estimates for members of the public

The risk estimates based on miners' data should not be applied dirctly to the general population. No reliable epidemiological data are available at present that would allow risk estimates to be made directly for the general population. Since the absolute risk for the general population is likely to be lower than that for miners, the Subcommittee recommends that, based on the present evidence, for practical purposes, a risk estimate in the region of 1 x 10^{-4} per WLM may be applied to the general population.

TRUE.

Totally Unjustified! See p.57 "REMARK"

1) based on Czechoslovakian and Swedish data
2) based on Colorado data only
3) derived indirectly from the data given in BEIR 1980 report
4) obtained from ICRP publication 32
5) recommended by the Subcommittee on Risk Estimates of ACRP.

See Myers' NWT Testimony (next!) pp. 71-73

COMMENTS ON AECB/ACRP MISCALCULATION OF RADON HAZARDS

1. As the BC Medical Association has observed, "The independence of this committee [ACRP] must be seriously questioned" (p 49).

2. AECB and ACRP have rejected the findings of the Thomas/McNeill Report, with no errors discovered in its methodology or in its calculations, and no discussions held with its authors (p 86).

3. ACRP misquotes the Thomas/McNeill risk estimates (p 61):

   Thomas/McNeill ("best estimate") ..... 6.5×10^{-4} per WLM

   Thomas/McNeill (range of values) ..... $3.0\text{-}12.5 \times 10^{-4}$/ WLM

   ACRP misquote of Thomas/McNeill ..... $0.5 - 12 \times 10^{-4}$/ WLM

4. ACRP also miscalculates the risk from BEIR-1980 (pp 55-57):

   BEIR-1980 ("best estimate") ..... 6.7×10^{-4}/ WLM

   ACRP miscalculation ..... $2\text{-}6 \times 10^{-4}$/ WLM

   The ACRP figure, "derived indirectly from data given in BEIR-1980", is apparently based on data from Hiroshima & Nagasaki having no bearing on human exposures to radon (pp 71-77).

5. Deleting the antiquated UNSCEAR figures and correcting those from BEIR and from the Thomas/McNeill Report, we obtain:

   LIFETIME RISK ESTIMATES PER WLM

   BEIR ("best estimate") ..... 6.7×10^{-4}

   T/M ("best estimate") ..... 6.5×10^{-4}

   T/M (range of values) ... $3.0\text{-}12.5 \times 10^{-4}$

   ICRP ..... $1.5\text{-}4.5 \times 10^{-4}$

   ACRP ..... $1\text{-}6 \times 10^{-4}$

   The ACRP upper limit of risk is LOWER than the "best estimate" of both BEIR and T/M. Moreover, the ACRP lower limit of risk is LOWER than any figure in the table! (The 0.7×10^{-4} UNSCEAR figure cited on page 69 is based on antiquated Colorado data.)

6. ACRP recommends as a lifetime risk estimate: 1-6 cancers per 10,000 persons for each WLM of occupational exposure, and 1 cancer per 10,000 persons for each WLM of household exposure.

   There is no scientific basis for using a lower risk estimate for household exposures, where the dose rate is so much lower.

   In fact BEIR-1980, Thomas/McNeill, the BCMA and NIOSH all agree, based on human evidence and confirmed by animal experiments (p 58), risk per WLM seems greater for lower dose-rates.

7. ACRP and ICRP appear to be either incompetent, or guided by priorities unrelated to health and safety considerations.

Testimony of David Myers
(Atomic Energy of Canada Limited)

to NWT Legislative Assembly

(excerpts)

[from Hansard, Thursday, February 26, 1981]

"My name is Dave Myers and I work for Atomic Energy of Canada Limited at Chalk River. Unlike most of the other people who will be talking to you, I am a research scientist who has been working on the biological effects of radiation for about 22 years.

"In general, one finds that people who have been working in nuclear power reactors for some years are healthier than the average person in Canada. These studies have been carried out in Canada as well as in the United States and in the United Kingdom.

"Radiation is a natural life force. It can be used for harmful purposes; it can also be used for our own good. What we are concerned with in the health sciences is that people are not exposed to amounts of radiation which would have harmful effects, either on their health or upon the health of their children. One might make an analogy with fire. Fire, as you know, in one form or another is considered essential to life by most people. It can also be very destructive if it is not properly controlled. The situation with uranium is very similar.

"I might point out, one of the beneficial aspects of natural radioactivity is that it helps to keep our earth warm. This is evidenced in the hot springs, such as radium hot springs.

"All of the food that we eat, all of the water that we drink, all of the air that we breathe contains radioactive materials. It has always contained them, ever since the world was created. So, what we are trying to do is to relate the results of our own activities in the nuclear power area to the natural levels of radiation -- which usually range between 80 and 120 millirems [0.08 - 0.12 rems per year].

"I do not wish to comment on the legislation that is involved. What I would like to point out is the purpose of these regulations, which is to bring any increase in radiation exposures down to a small fraction of natural background levels -- those natural levels to which we are all exposed, inevitably, every day of our lives.

"The question of hazards from radon has been raised. I might point out that one of the reasons that people are now aware of these hazards is because of the nuclear power industry. There are two small villages in Germany where miners had been digging up gold originally. Later they were after silver, cobalt, various other elements that people wanted to us. It has been known since the year 1500 approximately, that is somewhat over 400 years ago, that these miners died of a chest disease. In 1951 it was noted, or it was pointed out, that the cause of this chest disease was primarily radon daughters.

David Myers is a member of the AECB Advisory Committee on Radiological Protection which misquoted and rejected the Thomas/MacNeill risk estimates for radon exposure.

"In Newfoundland we have another tragic story. There were people mining fluorspar for some years in the 1930's. Again, many of the people developed lung cancer. This was a combination of the radon daughters to which they were exposed and the cigarette smoking. It is known that there is an excess of lung cancer in certain iron mines, certain cobalt mines, various other mines of this type around the world. If the uranium concentrations in the rock nearby happen to be higher than normal, you will have higher concentrations [of radon] and you will have unfortunate and frequently fatal results.

"Since the cause of these lung cancers was identified, the exposures in all mines in Canada have been carefully monitored & kept to extremely low levels. In 1959, the ICRP did make a recommendation on the maximum permissible levels of radon daughters in mines [4 WLM per year]. This was not immediately adopted in the United States, and because it cost money to ventilate mines -- I think this is the primary reason, that is my personal opinion -- it was not adopted in Canada either. The level [in Canada] was set at three times the recommendation of the ICRP [that is, 12 WLM per year. Canada finally adopted the 4 WLM per year standard 16 years later, in 1975.]

"The problem arises when one does not have strict regulations to protect people and when these regulations are not enforced. This is what happened in the very early stages of uranium mining, both in Canada, the United States, and in Europe."

[from Hansard, Friday, May 22, 1981]

"Most miners in Canada are currently exposed to much lower levels, in the region now of 0.1 (that is, one-tenth) of a working level. This is very much lower than the values of 50 to 100 to which miners were exposed shortly after World War II, and which tragically resulted in a number of cases of lung cancer in these miners. It is still anticipated that a hazard exists, but the hazard from radiation from radon daughters is thought to be relatively low, and in the same region as the hazards to which persons working in other industries in Canada are exposed.

"If a miner is exposed to one-tenth of a working level in the mines for 12 months of the year, his accumulated exposure over the year is one-tenth times 12, or approximately one WLM per year. As mentioned by Dr. Chambers, 55 percent of the uranium miners in Canada accumulate less than one WLM per year at present. The other 45 percent are more than this. The average for all miners is about one WLM per year.

"A person who worked in a uranium mine for 50 years, under current operating standards, would accumulate a total of 50 WLM over that time. This person would have one to two chances in 100 of dying from lung cancer at some later date as a result of radon exposures in the mine. This number -- that is, one to two chances in 100 after 50 years of work -- this number is approximately the same as the risk of a fatal accident to persons who work for 50 years in government or in the transportation and communications industries in Canada. These are the best numbers available. I leave it to you as a legislative body, and to the miners themselves, to decide whether that is an acceptable risk."

David Myers was also the principal liaison with Dr. Duncan Thomas (on behalf of the ACRP) when the Thomas/MacNeill Report was being prepared, edited and finalized.

Letter from David Myers

to the Speaker of the Legislative Assembly of the Northwest Territories September 21 1981

(excerpt)

"In my comments to the Legislative Assembly on 1981 May 22, it was noted that a person exposed to 50 working level months (WLM) would have a one to two percent risk of dying of [a radiation-induced] lung cancer, using as a basis the United Nations report of 1977, which suggests a lifetime risk of about 2 to 4 fatal lung cancers per 10,000 miners exposed to one WLM each. The arithmetic is straightforward:

50 WLM x (2-4 cases per 10,000) = 100-200 cases per 10,000
= 1-2 cases per 100

"A doubling of [the normal rate of] 5.4 lung cancer deaths per hundred, by 120 WLM, corresponds to a lifetime risk of [at least] 4.5 fatal [radiation-induced] lung cancers per 10,000 persons per WLM. This is at the upper limit of the United Nations risk estimate [it is actually beyond] and is a little lower than the upper limit of the lifetime risk estimates derived from the 1980 BEIR Report of the U.S. National Academy of Sciences, namely 6.4 per 10,000 persons per WLM (as derived from Table V-20 of this report, for persons age 20-65 at time of exposure; this latter value is the same one that was quoted in round numbers in my statement of May 22).

"The NIOSH Study Group Report does not attempt to give a quantitative value for the risk of lung cancer after inhalation of radon daughters, but suggests (in agreement with the 1980 BEIR Report) that the "doubling dose" is probably below 120 WLM. There is thus no major disagreement between the risk estimate in the 1980 BEIR Report & the more indefinite statement on risks in the NIOSH Study Group Report."

SEE SUBSEQUENT CORRESPONDENCE RE MYERS' USE OF TABLE V-20 FROM BEIR-III

Ignoring the data in the Lung Cancer section of BEIR-III Report, Myers (and ACRP) uses Table V-20 to estimate the risks of radon.

EXHIBIT 16

Basis for David Myers' calculation

Somatic Effects: Cancer From BEIR 1980

TABLE V-20 Estimated Excess Fatal Cancers Other than Leukemia and Bone Cancer from Low-LET Radiation Dose (D): L-L Model[a]

This category EXCLUDES alpha radiation! (and therefore radon)

Estimated dose-response relationship [b] Excess risk = $3.470D$

Estimated age- and sex-specific regression coefficients for D

Sex	Age at Exposure, yr					
	0-9	10-19	20-34	35-49	50+	All[c]
M	1.920	1.457	4.327	5.291	8.808	5.087
F	2.576	1.955	5.807	7.102	11.823	7.254

Life-table estimates of excess cases per million persons

	Absolute-Risk Projection Model		Relative-Risk Projection Model	
	M	F	M	F
Single exposure to 10 rads				
Normal expectation	170,400	139,400	170,400	139,400
Excess deaths: number	919	1,473	4,226	4,852
% of normal	0.54	1.1	2.5	3.5
Continuous exposure to 1 rad/yr, lifetime				
Normal expectation	165,700	149,200	165,700	149,200
Excess deaths: number	5,827	10,400	22,080	29,030
% of normal	3.5	7.0	13.3	19.5
Continuous exposure to 1 rad/yr, ages 20-65				
Normal expectation	171,600	152,800	171,600	152,800
Excess deaths: number	4,324	7,745	8,916	14,100
% of normal	2.5	5.1	5.2	9.2
Continuous exposure to 1 rad/yr, ages 35-65				
Normal expectation	175,700	153,300	175,700	153,300
Excess deaths: number	2,420	4,603	2,905	5,685
% of normal	1.4	3.0	1.7	3.7
Continuous exposure to 1 rad/yr, ages 50-65				
Normal expectation	178,000	147,300	178,000	147,300
Excess deaths: number	1,046	2,153	1,069	2,265
% of normal	0.59	1.5	0.60	1.5

[a] D is in rads; coefficients for D are per million persons per year.
[b] Based on Hiroshima and Nagasaki mss data, 1955-1974.
[c] Weighted average for U.S. life-table population, 1969-1971.

Does NOT include ANY uranium mining data!

But Table V-20 is based on Hiroshima and Nagasaki data. It has nothing to do with radon or any other kind of alpha radiation.

EXHIBIT 17
Correspondence on Radon Exposure Standards

Gordon Edwards
1300 Raimbault
St Laurent Que
H4L 4R9 Canada
November 8 1981

Dr. Duncan Thomas
Assistant Professor
Department of Epidemiology
McGill University

Dear Dr. Thomas:

Following our recent telephone conversation, I would appreciate very much a response to the following questions:

1. What is the basis of your knowledge about lung cancer risks associated with radon exposure in human beings?

Basis of Myers' and ACRP's risk figure from BEIR-III

2. In your opinion, is it reasonable from a scientific point of view to use Table V-20 in the BEIR-III Report to calculate lung cancer risks resulting from radon exposure?

3. What is the best basis for estimating lung cancer risks resulting from radon exposure, in your opinion, if one wishes to use the BEIR-III Report for this purpose?

4. If the method outlined in answer to question #3 is applied to a worker who accumulates 50 working level months (WLM) at the rate of 1 WLM per year over a period of 50 years, what would his excess risk of lung cancer be (and how does that compare with the lung cancer risk for the general population)?

5. Based on the results of the BEIR-III Report, does the current standard for permissible radon exposure in homes (.02 WL) preclude the possibility of a significant increase in lung cancer fatalities among people inhabiting such homes?

As I told you on the telephone, I am trying to provide information to the legislative assembly of the Northwest Territories on the subject of radon-related health risks, and I would like permission to send a copy of your letter to the clerk of the assembly for distribution. Many thanks for your assistance.

Yours very truly,
Gordon Edwards
Gordon Edwards.

EXHIBIT 17

McGill University

Department of Epidemiology and Health

11 November 1981

Dr Gordon Edwards
1300 Rainbault
St Laurent PQ
H4L 4R9

Dear Dr Edwards,

Thank you for your interest in my work on the health effects of alpha radiation. Though trained as a statistician, I have been working in epidemiology for about ten years. I have generally been involved in the development of methods of risk analysis for epidemiologic studies, and am particularly interested in environmental health and cancer epidemiology. Since September 1980, I have been working in collaboration with Dr Kenneth McNeill of the Department of Physics, University of Toronto, to prepare a comprehensive review and synthesis of available data on the health effects of alpha radiation for the Atomic Energy Control Board. Our draft report was submitted in May 1981, but we are not yet free to release our findings until the final report is accepted, probably around January 1982. Nevertheless, I hope you will find the following personal opinions helpful.

What's wrong with Myers' use of Table V-20 of BEIR See pp 73-74

With regard to Table V-20 of the BEIR III report, I would like to emphasize that this table clearly refers to low-LET radiation and thus may well not be applicable to the effects of alpha emitters like radon daughters for several reasons. First, there is good reason to believe that the shape of the dose-response relation would differ between low- and high-LET radiation. Second, for radon daughters, the main target organ is the lung whereas for low-LET (penetrating) radiations, many organs might be affected; Table V-20 provides risk estimates for all cancers except leukemia and bone cancer. Third, these estimates are expressed in units of rads, which would require conversion to WLMs for radon daughters, and there is considerable controversy about the appropriate conversion factor to use. Finally, the estimates in Table V-20 are based solely on data from the Hiroshima and Nagasaki survivors, about which doubts have recently been raised concerning the validity of the dosimetry, and they make no use of the several sets of data on miners exposed to radon daughters.

/2

Postal address 3775 University Street, Montreal, PQ, Canada H3A 2B4

Dr Gordon Edwards 11 November 1981

Where BEIR does give risk estimates for radon-induced lung cancer.

These mining data (summarized on pp 380-390 of the typescript edition of the BEIR III report), I feel, provide a much more suitable basis for estimating the risks of exposure to radon daughters. Dr McNeill's and my synthesis of these data are contained in our draft report, but though they cannot yet be released, I feel it is safe to say we are in general agreement with the summary provided on p 390 of the typescript edition of BEIR III, namely 10 lung cancer cases per 10^6 PY-WLM between ages 35-49, 20 per 10^6PY-WLM between 50 and 64, and 50 per 10^6 PY-WLM over age 65.

Life table methods, similar to those used in constructing table V-20 of BEIR III, can then be used to predict the lifetime risk of lung cancer for various exposure scenarios and assumptions about how risks will continue to accumulate in the future. For purposes of illustration, I enclose one page of a computer printout which is being incorporated into our own report on risk estimates. This table indicates that 4 WLM per year of exposure to radon daughters from age 15 to 64 would lead to an expected increase of 13,434 lung cancers per 100,000 persons exposed at that level. The effect of 1 WLM per year over the same period would be about ¼, or 3.4 additional lung cancers per 100 persons. However, I must emphasize that this estimate is but one of a large number we have considered and turns out to be one of the larger. Nevertheless, if we were to base our conclusions solely on the BEIR III report, I would consider this to be the best estimate.

See pp 56-57

I would certainly not say that the .02 WL standard for household exposures *precludes* a significant risk of lung cancer. You can see this for yourself by scaling down the occupational risk estimate above proportionally. However, the uncertainties in this calculation are substantially greater than those in the occupational risk calculation, and I would rather not commit myself to a particular figure until our report (with all its qualifications) is released.

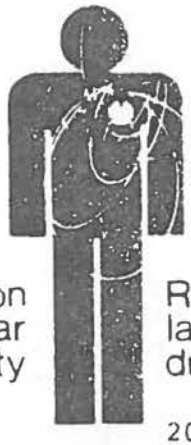
I hope your find all this helpful. You certainly have my permission to send this letter to the Legislative Assembly of the Northwest Territories. I will send you a copy of our complete report as soon as it becomes available.

Sincerely

Duncan C Thomas Ph D
Assistant professor

DCT/md

EXHIBIT 17



Canadian Coalition for Nuclear Responsibility

Regroupement pour la surveillance du nucleaire

2010 MacKay
Montréal, Québec
H3G 2J1

August 8, 1980

Maurice Foster, MP
House of Commons
Ottawa, Ontario

Dear Mr. Foster:

It was a pleasure to meet with you again in Roy MacLaren's office recently.

As I pointed out during our discussion, there is a very real danger of a lung cancer epidemic some twenty or thirty years hence among the residents of radioactively contaminated homes in Elliot Lake. The fact that the contamination is "natural" does not lessen the problem.

We know that radon gas is a killer. We know from the Ham Commission Report of 1976 that there is no evidence to support the concept of a "safe dose".[1] In Appendix C, the Ham Report points out that significant increases in lung cancer occurred among uranium miners even at the lowest recorded exposure levels. "To be at all plausible in relation to the Ontario experience, a postulated threshold [safe dose] would have to be lower than 10 WLM." (p.323)

The standard adopted by the AECB (and hence by the Ontario Government) for radon gas in homes is 0.02 WL. At this level of exposure, residents of such homes will rather quickly accumulate a dangerous dose:

ACCUMULATED RADIATION DOSE AT 0.02 WL

	5 years	10 years	15 years	20 years
12 hours/day:	2.6 WLM	5.2 WLM	7.7 WLM	10.3 WLM
17 hours/day:	3.65 WLM	7.3 WLM	10.95 WLM	14.6 WLM

.../2

Since children and mothers are generally indoors (home, school, friends) more than 17 hours a day on the average, these calculated exposures tend to underestimate the exposures that will be received.

How much extra lung cancers might one expect among people who pass their lives inside such contaminated buildings? According to calculations performed by Dr. Jan Muller of the Ontario Ministry of Labour, assuming 12 hours/day exposure, there could be as much as an 11.8% increase in the lung cancer rate.[2] This means that, in addition to the 54 "expected" lung cancers per thousand population which presently occur in Ontario, there would be an extra 6 lung cancer deaths per thousand.

As you know, the projected population of Elliot Lake after the boom is between 30,000 and 40,000 people. Even if only half the buildings are contaminated, we are facing the prospect of up to 120 extra lung cancer deaths in Elliot Lake due to excess radon gas in homes and schools.

Of course, all this would be merely academic if most of the new buildings in Elliot Lake are well below the 0.02 WL standard. Such is not the case. In neighbourhood 3C, for example, only half a dozen homes out of about three hundred were found to be habitable according to the 0.02 WL criterion. The other homes will have electric fans installed under the floorboards to blow the toxic gas out of the house or to dilute it with fresh air drawn in from outside. However, past experience has shown that one fan is often not enough to meet the 0.02 WL standard, and that some people are turning off these fans in order to obtain some relief from the constant noise. (Consequently, they greatly increase their exposure to the deadly radon daughters.)

I must add that Dr. Muller's analysis has been criticized as being biased and unscientific in that it seriously underestimates the health hazard associated with low levels of radon exposure. Dr. Muller's own data shows that, at these low levels, the cancer-causing effect of radon is very much higher than one would expect from the high exposure data.

By using Dr. Muller's own low-exposure data and duplicating his calculations, I found that 12 hours/day exposure at 0.02 WL would likely cause an extra 17 lung cancer deaths per thousand population - almost three times larger than Dr. Muller's highest estimate. This evidence was presented to the Elliot Lake Environmental Assessment Board in 1978, who subsequently recommended that the Province of Ontario undertake a reassessment of the 0.02 WL standard for radon gas in homes.[4] As you know, no such reassessment has occurred.

.../3

Later, in 1979, my revised estimate of 17 extra lung cancers per thousand was given added impetus by Dr. Victor Archer (M.D.), Medical Director of the U.S. National Institute for Occupational Safety and Health in Salt Lake City, Utah, and one of the world's outstanding experts in the field of lung cancer caused by radon gas.[5] Using his own independent data, Dr. Archer calculated between 18 and 42 extra lung cancer deaths per thousand[6] population, assuming 17 hours/day occupancy and a 0.02 WL standard.

Whether you accept Dr. Muller's tables, or my reanalysis of Dr. Muller's data, or Dr. Archer's independent analysis, it is obvious to me that we are flirting with the possibility of a public health disaster and a major political scandal if the present situation is not corrected.

I would be happy to consider any countervailing evidence which you may have to show that my apprehensions are unfounded. It would be, indeed, a great relief.

At the very least, I believe that a careful epidemiological study should be begun, starting now, to monitor the subsequent history of lung cancer among the people who will be living in these contaminated homes. In view of the evidence, anything less would be totally irresponsible in my opinion. Ideally, however, I would favour correcting the situation <u>now</u> so we don't have to count corpses later.[7]

May I hear from you before long on this matter?

Yours very truly,

Gordon Edwards

Gordon Edwards

P.S. I am enclosing a speech by Dr. David Bates (M.D.) which points out the rather alarming number of recent epidemiological studies which indicate that the medical effects of low level radiation exposure may be far worse than we thought just five years ago.[8] As you know, Dr. Bates was the chairman of the B.C. Royal Commission of Inquiry into Uranium Mining, and author of the Science Council of Canada's study on <u>Poisons and Policies</u>. Toward the end of his speech, Dr. Bates warns against placing too much trust in the soothing reassurances of the nuclear advocates:

> "It is rather as if one was a parent of a six-year-old boy whom one knew, on past occasions, had not told the truth, and considering entrusting him with a box of matches. One might do this; but one should recognize that there is a great deal of difference between giving

.../4

him a box of matches, and giving him a stick of dynamite or a loaded revolver. It is this aspect of the public debate that many people have most difficulty understanding.

The proponents of nuclear energy are paying a heavy price for the secrecy in which their profession was initially shrouded, and for the tradition of lack of openness which they inherited. It seems to me that we have to say to them that until we can see that this tradition and these policies have been completely changed, no sensible member of the public can give full support to the maximal possible development of the nuclear industry."

GE

NOTES

1. Report of the Royal Commission on the Health and Safety of Workers in Mines, Government of Ontario, 1976. James Ham, the author of the report, is now President of the University of Toronto.

2. Elliot Lake Protection from Radiation in New Housing, Report to the Environmental Assessment Board by the Ontario Ministry of Housing, with an Appendix by Jan Muller (M.D.) and R. Kusiak of the Ministry of Labour, February 1978. For the 11.8% figure, see page 28 of reference 3. Dr. Muller expressed his preference for a 4.4% figure, but it is clear from his own table 6 that the calculated increase in lung cancer, using his assumptions and methodology, could range from a low of 2.6% to a high of 11.8%.

3. Estimating Lung Cancers, CCNR-78-3, May 1978. See pp. 20-29.

4. Interim Report, April 6 1978, and Final Report, May 9 1978, Criteria and Approval Procedures - Naturally Occurring Radiation in New Construction, Elliot Lake Environmental Assessment Board (reproduced as Appendices 8 and 9 in The Expansion of the Uranium Mines in the Elliot Lake Area, Final Report of the E.A.B., May 1979).

5. The Ham Commission Report (reference 1) cites 11 papers co-authored by Dr. Archer out of a total of about 20 papers on the subject - no other researcher is cited more often. In the B.C. Medical Association's Annotated Bibliography, entitled Health Dangers of the Nuclear Fuel Chain, Dr. Archer's work is cited 15 times - and again, no other researcher is cited more often.

6. See letters from Dr. Archer to Dr. Gordon Edwards (May 30 1979) and to Mr. Frank Palmay (February 2 1979), copy to follow.

7. In addition to the suggestions contained in reference 3, see point #7 in my letter to Don McDonald of Blind River (July 21 1980), copy to follow.

8. "Occupational and Environmental Health Considerations", address to the Nuclear Policy Conference, Carleton University (Nov 9 1978), copy to follow.

cc. Roy MacLaren, Marc Lalonde, David Bates, James Ham.

EXHIBIT 17

(letterhead)

DEPARTMENT OF HEALTH EDUCATION AND WELFARE

P U B L I C H E A L T H S E R V I C E

Health Services & Mental Health Administration

COPY

National Institute for Occupational
Safety and Health
Room 133 U.S.P.O. and Courthouse
350 South Main Salt Lake City Utah
84101

February 2, 1979

Mr. Frank Palmay,
Lang, Michener, Cranston,
Farquarson, and Wright,
P.O. Box 10, First Canadian Place,
Toronto, Canada, M5X 1A2

Dear Mr. Palmay:

You will recall that in my recent testimony before the Environmental Assessment Board, I gave some risk estimates, comparing my risk estimate for persons exposed in homes at a level of .02 WL with that given by Dr. Mueller. Those calculations were made rather hurriedly the night before the testimony. I regret that there were errors in those calculations.

I have now reviewed those calculations and obtain the following:

Dr. Mueller's calculations:

He states (page 9 of his risk estimate calculations) that .02 WL for 1 hour per day from birth to death will cause about 20 cases of lung cancer per 100,000 persons over their lifetime. If one assumes an exposure of 17 hours/day, then his figure yields 340 cases/100,000, or 3.4/1000 deaths.

Dr. Archer's calculations are as follows:

0.02 WL x 17 x 7 ÷ 40 x 12 x 50 x 1000 = 35,700 WLMM/1000 persons living their lifetimes at .02 WL.

Dividing 37,500 by 855 or by 2000 gives a range of 18 to 42 lung cancer deaths/1000.

17 represents hours per day of exposure; 7 is days per week of exposure.

40 is hours per week of exposure by uranium miners; 12 is months per year.

50 is the years of effective exposure per lifetime.

WLMM is Working-Level-Man-Months, that is, the cumulative exposure per person multiplied by the number of exposed persons.

2000 is about the average population exposure in WLMM required to produce 1 lung cancer among uranium miners (from Exhibit 399).

855 WLMM is the extrapolated value for WLMM required to produce 1 lung cancer among a population exposed at very low levels of exposure (from Exhibit 399).

Letter to Frank Palmay from Victor Archer, February 2 1979

We therefore have the following estimated number of deaths for lifetime exposure of populations at 0.02 WL in their homes:

Mueller - 3.4/1000 persons

Archer - 18/1000 persons as low estimate
42/1000 persons as high estimate

These numbers may be compared to the approximate number of lung cancer deaths among nonsmokers (which might be due to background levels of radon daughters): 10/1000 persons.

The level of radon daughters in the average home is between .004 and .008 WL. Since .02 is 3 or 4 times the average level, then one might predict that living at a level of .02 WL would increase the lung cancer risk among nonsmokers by a factor of 3 or 4 (to 30 or 40/1000), and that a comparable increase would occur among smokers (assuming no synergism).

It is apparent from these numbers that my calculations are not as close to Mueller's as I had thought, and that my risk estimates are more compatible with a postulated effect from background radiation than are Dr. Mueller's.

I would suggest that you present this letter to the Environmental Assessment Board so as to set the record straight.

Yours truly,

Victor E. Archer, M.D.,
Medical Director

Both Dr. Archer and Dr. Mueller are now retired. Dr. Archer's paper on "Factors in Exposure-Response Relationships of Radon Daughter Injury" appears in the Proceedings of a Conference/Workshop on Lung Cancer Epidemiology and Industrial Applications of Sputum Cytology, held November 14-16 1978 at the Colorado School of Mines, Golden, Colorado -- printed January 1979 by Colorado School of Mines Press. On the basis of Dr. Archer's work, based on epidemiological studies in Canada, Sweden, Czechoslovakia, and the United States, the National Institute for Occupational Safety and Health has issued a report asking the U.S. Department of Labour to reduce significantly the permitted exposure limit for uranium miners. The report states that the full effects of radon exposure may have been underestimated and that "there appears to be no margin of safety associated with the present standard" (4 Working Level Months - WLM - per year). It is suggested that the limit be reduced from 4 WLM to 0.7 WLM per year.

(Source: Nature, vol. 286, 28 August, 1980)

Gordon Edwards

EXHIBIT 17

United Steelworkers of America

AFL - CIO
IN CANADA AFFILIATED TO THE CANADIAN LABOUR CONGRESS

Dave Patterson
DIRECTOR DISTRICT 6

20 ALBERTA ROAD
ELLIOT LAKE, ONTARIO, P5A 1Z6
PHONE (705) 848-2773
848-2226

November 2, 1981

Dr. Gordon Edwards
1300 Raimbault
Ville St. Laurent, Quebec
H4L 4R9

Dear Gordon:

As requested, I have now had an opportunity to review the Company supplied radon daughter exposures for the year 1980. At the outset, I would bring a couple of important factors to your attention. The Atomic Energy Control Board has informed us that they are currently attempting to achieve 50% accuracy within 95% confidence on the measurement of radon daughters in uranium operations. This lack of efficiency has all kinds of error potential. In addition, we have reason to further suspect the accuracy of the figures provided by the Company for various reasons. The current procedure of estimating radon daughter exposures is usually based on two samples in each work place per month and the exposure given the employee is the average of these readings. It goes without saying that if you have high levels for most of the month and then engineer additional ventilation to a specific work place, the average of the two figures will be considerably on the low side.

Secondly, the Company is allowed to reduce employee exposures by a factor of 50% by designating certain work areas as mandatory respirator areas. In other words, they assume the employee wears the respirator for eight hours each day and reduce his exposure by 50%. Our members continually complain that it is impossible in mining to wear the airstream respirators for prolonged periods of time and, therefore, do not do so. In spite of this, credits are taken in mandatory respirator areas. In reviewing the Company supplied (Denison Mines) yearly statistics, I note that there are some employees recorded as receiving exposures just under 4 W.L.M. and if we were to add the respirator credit (which has been subtracted from their actual exposures), they in fact would have exceeded the 4 W.L.M. yearly dose. In addition, the Company practices a system of rotating workers from high to lower exposure areas, thereby attempting to prevent 4 W.L.M. readings. In other words, if the worker were to remain in his assigned work place, he would exceed the radiation levels. I would also caution you that both the Company and the regulatory agencies like to use average

.../2

figures and this is deceiving. For example, there is a high turnover of employees and, therefore, the master yearly list may indicate numerous employees with low exposure levels, however, a check reveals many employees or former employees with very short employment time. Hires late in the year are also part of this master list and obviously show low exposure levels. In spite of the foregoing, the average of the mine frequently used as so-called sound statistics, utilizes these short term type workers and, in my opinion, falsifies the true potential for year round employment exposure. The figures I have been reviewing were taken for the year 1980 in the underground operations of Denison Mines Limited. A quick review of the figures (with the above concerns kept in mind) indicate that almost 300 workers received exposure to radon daughters in excess of 1.5 W.L. with some very close to 4 W.L.M. and about 120 of these received exposures between the range of 2 W.L.M. and 3 W.L.M. The above figures are those after subtracting the respirator credits referred to above and if the subtraction did not take place, the W.L.M. readings and the numbers would be higher.

It should also be understood that we have now proven (acknowledged by the regulatory agencies) that thoron daughters, total uranium uptake, and gamma radiation are present in our uranium mines/mills as additional radioactive hazards and, if integrated with radon, considerable numbers of workers would have received a total integrated dose well in excess of the 4 W.L.M. criteria. A government study indicates that underground uranium workers may be receiving gamma doses as high as 3 rems per year in addition to the other radioactive hazards. It should also be understood that Denison Mines Limited through the urgings of the Union has substantially increased its ventilation to a point where it provides more air for ventilation purposes underground than any other mine in Canada. Despite this, we continue to encounter radiation levels which, we believe, are unacceptable.

I am enclosing a copy of a brief the writer prepared and presented under oath to the Royal Commission on Uranium Development in British Columbia. I might add that it withstood all cross-examination. The brief is put together basically from government reports and, in some areas, with Union information accumulated over the years.

We will be obtaining the year end W.L.M. figures for 1981 in January, however, I believe, they are comparable to the 1980 figures.

If I can be of further assistance, feel free to contact me.

Kindest personal regards.

Sincerely yours,

UNITED STEELWORKERS OF AMERICA

Homer Seguin
Staff Representative

/ts

EXHIBIT 17

Duncan Thomas Ph.D.
UNIVERSITY OF SOUTHERN CALIFORNIA
SCHOOL OF MEDICINE
Department of Family & Preventive Medicine
Parkview Medical Building
2025 Zonal Avenue
Los Angeles, California 90033

February 9, 1984

Regulations Development Section
Atomic Energy Control Board
P. O. Box 1046
Ottowa Ont K1P 5F9
Canada

Re: Consultative Document C-78

Gentlemen:

I recently received excerpts of your Proposed Regulatory Guide from colleagues at McGill. As one of the principal consultants to the Subcommittee for Risk Estimates, I was disappointed never to have been sent either the report of the Advisory Committee on Radiological Protection (document ACRP-1 (1981) INFO-0090) or these proposed regulations, both of which appear to be based in part on our work (INFO-0081).

My comments will be restricted to section 4.4 on intake limits for radon and thoron daughters. I am disturbed that the AECB has chosen to give less weight to the epidemiological evidence than to dosimetric calculations. We reviewed the attempts to establish an "effective dose equivalent" for radon and thoron daughters in considerable depth in Appendix E of our report and concluded that

> "Due to the complexity of the interactions, any figure derived in this way can only be a gross approximation; a reasonable range of values that have been proposed is 1-20 rem/WLM" (p. 16).

These uncertainties are far greater than those from the epidemiological data, despite the apparent variation in the latter as quoted in your Table 1 (page 20). For example, the range of 0.5 to 12 lung cancers per 10^4 person-WLM quoted from our report is apparently derived from the single smallest and single largest "crude" risk estimate from our Table 8.2 and ignores our suggested reasonable range of adjusted risk estimates quoted in our Table 8.3 and in our abstract of 3.2 to 12.3. Recent data from the Ontario uranium miners (Muller et al, Ontario Ministry of Labor), as well as a new cohort of Swedish metal miners (Radford, Banbury Report 1981; 9:151-163) both lead to point estimates close to the upper limits we quoted.

.../

The consultative document dismisses these epidemiological data as probably "conservative" (i.e., overestimates) primarily on the grounds that miners would have been exposed to gamma radiation, radon, and other carcinogens in addition to radon daughters and that actual doses of the lung cancer cases may have been underestimated. These and other potential biases were discussed in considerable depth in our report, notably sections 4.2.1.1, 7.4, and 8.1 and are common to epidemiologic studies of many other agents, for which epidemiologic evidence is used as the basis for standards.

The fact that coal and other mining groups do not show excesses of lung cancer argues that the presence of other carcinogens in mining operations is unlikely to be the explanation for the excess as among miners exposed to radon daughters. These other factors have been well analyzed in the new Swedish study (Radford and Axelson, submitted to New England Journal of Medicine) and the authors have concluded that they are unlikely to have made a significant contribution.

Underestimation of doses for lung cancer cases would tend to cause risk coefficients to be underestimated unless doses were equally underestimated for the cohort at risk. While this may have been the case for the Ontario cohort (cf Muller's attempts to reevaluate the doses), the reverse appears to be true for the US miners. Furthermore, nonsystematic error, which is certainly present in all studies, will cause risk coefficients to be underestimated.

The higher proportion of gamma radiation in today's well-ventilated mines supports the proposed policy of including gamma radiation in calculating limits. However, its impact on risk coefficients for radon daughters derived from epidemiologic studies is likely to be negligible. This is certainly true, for example, in the new Swedish study, for which gamma ray measurements ranged from 60 to 150 mrem/working year.

The net result of all the potential biases in epidemiologic studies is impossible to quantify, but it seems unlikely that risk coefficients could have been systematically overestimated by more than the range of uncertainty we quoted. Our "best estimate" of 130 excess lung cancers per 1000 persons continuously exposed to the 4 WLM limit for 50 years is clearly unacceptable. While not all miners work continuously at the limit or work for 50 years, the results from the Swedish cohort (which has been exposed to concentrations close to or below the 4 WLM/yr limit) clearly demonstrates the inadequacy of that standard: for the cohort as a whole, the relative risk for lung cancer was 3.4.

In our view, as expressed in recommendation 9 of our report, the 4 WLM/yr standard does not provide adequate protection for individuals. If the Board feels that it is more important to maintain an acceptable average risk, then we recommended that it should do so by explicit limitations on average levels rather than expecting the limitation on maximum levels to accomplish the purpose. I am skeptical that the relationship between average and maximum doses used by the ICRP for the non-mining nuclear industry would apply to mining, where workers would tend to spend most of the time where the ore is, i.e., where levels are the highest.

.../

Whether average risks are "acceptable" or not, we strongly recommended that maximum risks to individuals be reduced. The only way to accomplish this, short of much tighter annual limits, is to impose additional limits on the total dose. So far, Sweden appears to be the only country to have done this: their limit on lifetime exposure is 35 WLM. Obviously, the implementation of such a policy in a non-discriminatory fashion could be quite difficult.

In any event, considering the inadequacy of the present standard, I find it very difficult to comprehend the present proposal to raise the annual limit to the equivalent of about 4.7 WLM/yr without imposing additional limits on either average levels or lifetime doses. I strongly recommend that the Board reconsider this proposal rather than simply adapt the recommendations of the ICRP.

If you wish to clarity my views on this matter, please do not hesitate to contact me. My new office phone is (213) 224-7434.

Sincerely,

Duncan C. Thomas, Ph.D.
Associate Professor

DCT:bw
cc: Dr. Ken McNeill
Mr. Mark Goldberg

FIGURE 8

HOW RADON GAS IS CREATED

When a radioactive substance decays, it gives off alpha, beta, or gamma radiation and changes into another substance. In many cases that new substance is also radioactive and so a third substance is created when it decays. In this way, naturally-occurring U-238 gives rise to a whole family of radioactive substances called the "uranium daughters". Even after the uranium has been removed from the ore for use as a nuclear fuel or as a nuclear explosive, most of the uranium daughters remain behind in the wastes. These waste materials ("tailings") continue to produce radon gas (radon-222) at an undiminished rate for many thousands of years.

NAME OF ISOTOPE	TYPE OF RADIATION	HALF-LIFE
URANIUM-238		
↓α	alpha	4.5 billion years
THORIUM-234		
↓β	beta	24 days
PROTACTINIUM-234		
↓β	beta	1 minute
URANIUM-234		
↓α	alpha	248 thousand years
THORIUM-230		
↓α	alpha	78 thousand years
RADIUM-226		
↓α	alpha	16 hundred years
RADON-222		
↓α	alpha	3.8 days
POLONIUM-218		
↓α	alpha	3 minutes
LEAD-214		
↓β,γ	beta,gamma	27 minutes
BISMUTH-214		
↓β,γ	beta,gamma	20 minutes
POLONIUM-214		
↓α	alpha	160 microseconds
LEAD-210		
↓β	beta	21 years
BISMUTH-210		
↓β	beta	5 days
POLONIUM-210		
↓α	alpha	138 days
LEAD-206	- none -	- not radioactive -

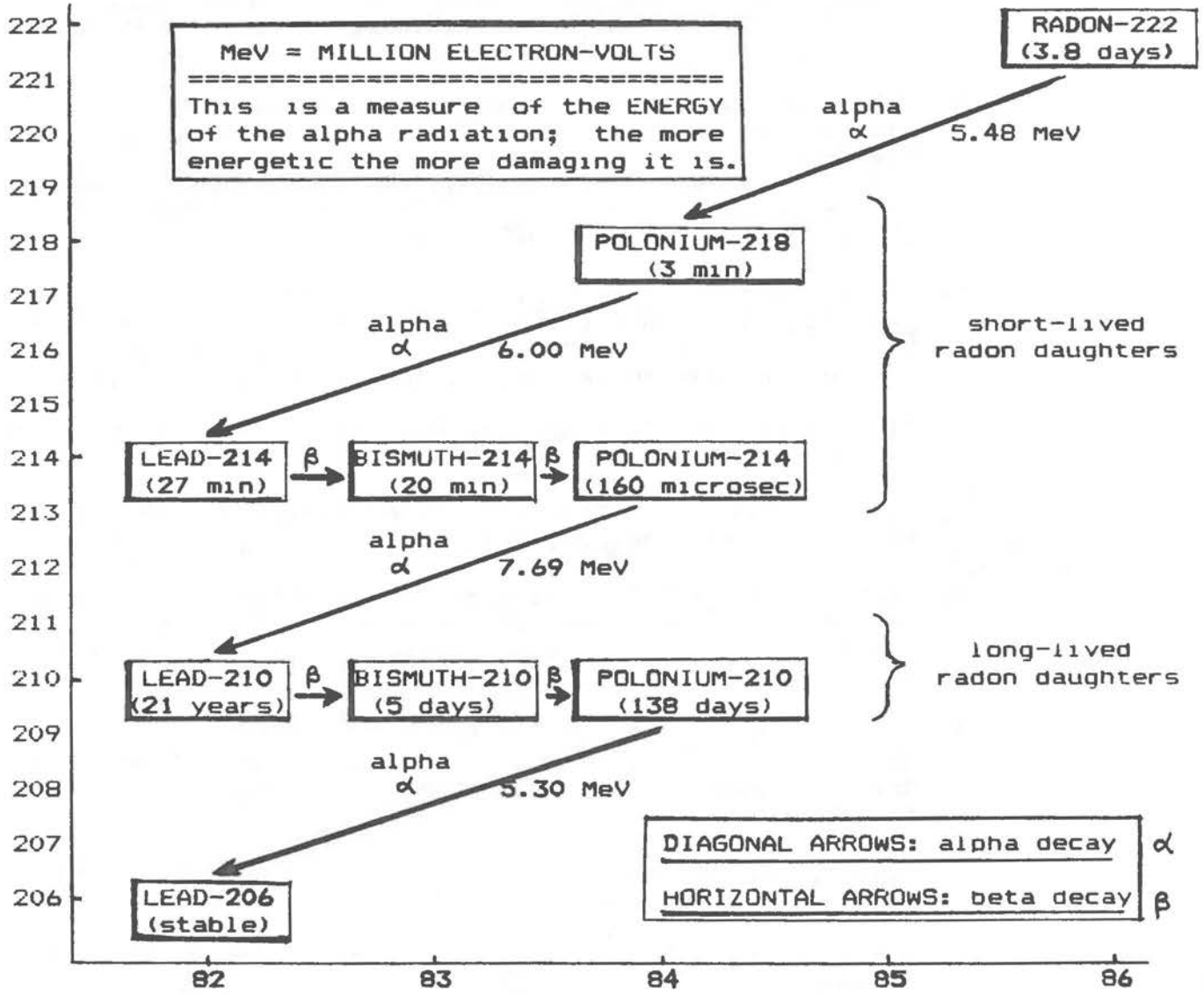
FIGURE 9

THE DAUGHTERS OF RADON

Radon is an inert gas which does not form chemical compounds. It is inhaled easily into the lungs, but it is exhaled just as easily.

The daughters of radon are solids at normal temperatures. Created by the decay of radon atoms, they attach themselves to microscopic dust particles. When these are inhaled, the radon daughters tend to lodge in the lungs, where they can deliver large doses of alpha (and beta) radiation to the sensitive living tissue lining the lungs.

If radon gas is confined in an enclosed space, the concentration of radon daughters increases with time. Under such circumstances - in mines or in radon-contaminated homes, for example - fully eighty-five percent of the dose to the lungs is due to radon daughters.



The vertical axis measures the "mass number" - the number of protons and neutrons contained in the nucleus of each atom. The horizontal axis measures the "atomic number" - the number of protons in the nucleus - which determines the chemical nature of the substance. The mass number is not affected by beta radiation, but both numbers are altered when alpha radiation occurs.

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8. Letter from Karl Z. Morgan to James Schlesinger (dealing in part with the epidemiological study of cancers in Hanford Workers by Thomas Mancuso, Alice Stewart, and George Kneale) May 25, 1977 (see Exhibit 11, page 40-41).

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19. Findings on Uranium Tailings and Nuclear Waste Disposal: Direct Quotations and Parenthetic Summaries from Seventeen Basic Documents, ed. Gordon Edwards. CCNR: Montreal (1980).

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GLOSSARY

Although it is not necessary to understand all of the technical jargon in order to understand the problem of radon gas in buildings, here is a set of explanations which you can refer to if you wish. Don't use it unless you feel you have to.

Radioactivity is the property of certain atoms (which are not stable) to spontaneously disintegrate by emitting either energetic particles or rays of pure energy (or both) from the nucleus or centre of the atom.

The half-life of a radioactive substance is the time required for half of its atoms to disintegrate.

The daughters of a radioactive substance are the other substances which are created as byproducts in the process of radioactive disintegration; in many cases, the daughters of a radioactive substance are also radioactive.

Ionizing Radiation is the term used to describe the various energy forms which can be emitted by the disintegration of radioactive atoms; these include

energetic particles -- alpha, beta, and neutrons
rays of pure energy -- gamma rays and x-rays

Exposure to even low levels of ionizing radiation can cause cancer and/or genetically defective children in the exposed population. These effects are caused by submicroscopic damage to the cells of the body which causes some of them to reproduce in an abnormal fashion.

Gamma Radiation (γ) is the most penetrating of all forms of ionizing radiation, capable of penetrating thick layers of metal; it is given off by the radioactive disintegration of such substances as radium-226, and is similar in nature to x-rays. (Radium-226 is primarily an alpha-emitter, however.)

Beta Radiation (β) is the next most penetrating form of ionizing radiation after x-rays and gamma rays; it actually consists of high velocity particles called beta particles or electrons.

Alpha Radiation (α) is the least penetrating form of ionizing radiation, unable to penetrate through a sheet of paper; it consists of high velocity particles (called alpha particles) which are more than 7000 times heavier than electrons.

Linear Energy Transfer (LET) measures the relative amount of damage done by a particular type of radiation per unit distance travelled. It is inversely related to the penetrating power, but not in a simple way. The most penetrating types of radiation (gamma, x, beta) are referred to as "low LET radiation", while the least penetrating types (alpha, neutrons) are called "high LET radiation". High LET radiation is far more damaging per unit dose than low LET radiation.

GLOSSARY (continued)

A curie (Ci) is a measure of radioactivity in disintegrations per second; one curie corresponds to the radioactivity in a gram of pure radium

A picocurie (pCi) is a trillionth (i.e. a millionth of a millionth) of a curie.

Radon is an alpha-emitting radioactive gas with a half-life of 3.8 days. It is a daughter of radium-226, and it gives rise to other radioactive substances known as radon daughters, most of which are also alpha-emitting substances.

One working level (WL) designates a concentration of 100 picocuries of radon daughters per litre of air (abbreviated as 100 pCi/l)

A working level month (WLM) is a measure of human exposure to radon daughters. One WLM is equal to the concentration of radon daughters, measured in working levels, times the number of hours of exposure, divided by 170. Thus a man exposed to 1 WL for 170 hours (approximately one month's exposure at 40 hours per week) will accumulate an exposure of 1 WLM.

A rad ("radiation absorbed dose") is a measure of how much energy is absorbed by tissue when exposed to a certain source of ionizing radiation. Technically, 1 rad corresponds to 100 ergs of energy being absorbed in each gram of tissue exposed to ionizing radiation.

A rem ("radiation equivalent man")is a measure of the ability of a given dose of radiation to do harm to living cells (thereby causing a predictable increase in cancer, or in genetic defects to the children of people whose gonads have been exposed to ionizing radiation). For low LET radiation, 1 rem corresponds almost exactly with 1 rad of exposure; but for high LET radiation, each rad of exposure corresponds to 10 or 20 rems, because of the much greater relative damage which is done to living cells by high LET radiation.

A millirem (mr) is a thousandth of a rem. The natural background radiation to which we are all exposed as a result of cosmic radiation from outer space and naturally occurring radioactive substances is about 100 mr/year, or about 0.01 mr/hr.

The quality factor associated with a given type of radiation is the factor which must be used to convert a radiation dose measured in rads to the equivalent number of rems. For gamma radiation, the quality factor is 1 (1 rad = 1 rem), but for alpha radiation, the quality factor is about 20 (1 rad = 20 rems).

The linear hypothesis states that the extra cancers and genetic defects that will occur in a given population as a result of exposure to ionizing radiation is directly proportional to the sum total of all of the individual doses received by each member of the population.

NOTES

1. The shortest distance between two points on a sphere is not a straight line, but a "great circle" -- that is, a circle which has the same radius as the sphere itself. However, if a "line" is defined as the shortest distance between two points, then these great circles are in fact "lines" on the surface of the sphere, since they do represent the shortest possible paths joining points on a sphere. The technical term for such a curved "line" is "geodesic". By definition, a geodesic is any path on a curved surface which provides the shortest distance between any two neighbouring points along the path.

2. Other changes of behaviour are, of course, possible. A culture of bacteria will often grow exponentially until it exhausts its food supply, whereupon it will suffer a sudden catastrophic collapse. In other cases, where there are predator-prey relationships at work, a cyclic rise and fall of animal populations is frequently observed. However, when a species of bird or mammal is free from serious competition or predation, the logistical growth model seems to offer a good description of what happens in a natural setting.

3. Consider the following words by Dr. Donald Miller, Head of Biomathematics at the Canadian National Research Council, addressing a senior seminar of applied mathematicians in Ottawa on March 5 1974: (Reference 11, pages 160-162)

> "Are ... people satisfied with the results of mathematical modelling? ... I think generally that they are not satisfied when the problems involve very complicated systems -- as they invariably do in ecological studies, in regional planning, and in studies of pollution or energy supply. I recently heard the former director of the Marion Lake Project, one of Canada's contributions to the International Biological Programme, make the comment that he was not convinced that mathematical modelling was any help at all in the study of ecological systems....
>
> "In many such projects, not enough care is devoted to the formulation and testing of the mathematical description. In its most fundamental terms, this means that we, the mathematical community, might have forgotten something that we should have learned many years ago, under the heading of the scientific method. We all know how that goes; one looks at a system and inductively frames a hypothesis, deductively works out the consequences of this hypothesis in a form that can be tested, experimentally tries to verify or disprove the hypothesis, and returns to frame a new one on an improved basis. This seems simple enough, and most people in this audience, I'm sure, are feeling

a bit insulted. But the fact is that we do not seem to be doing it. We are not following the basic philosophy of science." (Emphasis added)

4. These Proceedings are 360 (plus xxv) pages long. The bulk of the text is actually an anthology of about forty short papers which were specially prepared by the participants. Each of these papers deals in considerable detail with specific applications of mathematical methods to real-life problems arising in Federal Government Departments in Ottawa, using an absolute minimum of technical jargon and no intimidating mathematical symbolism. The Proceedings have also been translated into German and distributed by the West German Government.

5. There is an impressive list of references provided by the Ham Commission Report (our reference 1), but they are limited to the study of uranium miners. It is perhaps worth noting, in another context, that the two most potent carcinogens in tobacco smoke are now known to be benzopyrene (a cancer-causing aromatic hydrocarbon which is also present in automobile exhaust) and polonium-210 (one of the more persistent radon daughters).

   As already remarked in the summary (page iii), phosphate ore is relatively rich in uranium. As a result, radon gas is slowly released from the phosphate feritlizer which is used on most tobacco crops. Being heavy, the gas accumulates somewhat before dissipating, and the short-lived radon daughters (which carry an electrical charge) promptly attach themselves to microscopic dust particles. These dust particles, in turn, adhere to the sticky, resinous hairs which grow on the underside of the tobacco leaves. These short-lived daughters will all disintegrate within a few hours after being formed, leaving a deposit of the radioactive substance lead-210 (with a half-life of 21 years) in the tobacco leaves.

   When the tobacco leaves are harvested, cured, shredded, rolled into cigarettes, and sold in the stores, they still carry a burden of lead-210 with them. Polonium-210 is a radioactive daughter of lead-210, and, like its parent, it is a solid at normal temperatures. However, when a smoker draws on his or her cigarette, the intense localized heat at the burning tip of the cigarette is enough to volatilize both substances. Thus the chronic smoker ends up with a deposit of lead-210 and polonium-210 in his or her lungs.

   For evidence on the carcinogenicity of polonium-210, see the reference cited in Figure 5 on page 16. For more information on this topic, and for further references, see "Tobacco Radioactivity and Cancer in Smokers" by Edward A. Martell, in American Scientist, volume 63, July-August 1975, pp. 404-412. Dr. Martell has been a staff member at the

National Centre for Atmospheric Research in Boulder, Colorado for many years, and has written widely on the subject of radioecology.

6. As far as medical science can tell, carcinogens act directly on the nucleus of the cell, causing random damage to the chromosomes and DNA molecules contained therein. Most of the cells so damaged are either killed or sterilized. However, in a very few cases, one of these damaged cells may survive the injury and still be capable of reproducing. Such a cell may become a cancerous cell, if it begins to proliferate in an undifferentiated or "cancerous" manner. On the other hand, if a reproductive cell is damaged in this way, it can lead to genetic deficiencies in the offspring -- and if an embryonic cell is so affected, the normal development of the fetus can be disrupted. For this reason, it is recognized that substances having a carcinogenic effect will also have a genetic and a teratogenic effect. It is also widely believed that since these effects take place in a random manner at the cellular level, there is no such thing as a "harmless" dose. Any dose, however small, will produce gross malignancies and deformities if it is administered to a sufficiently large population.

   Consider the following quotations from the Proceedings of a Public Forum on Policies and Poisons held in Toronto, on November 15 1977, under the auspices of the Science Council of Canada and the Canadian Public Health Association:

   > "... there is good circumstantial evidence that 80 percent of human cancers are environmental in origin...." (page 11)
   >
   > "There are occasions when it is known that there are severe risks attendant upon exposure to certain substances and yet no action to control them is undertaken. This appears to be a sort of 'paralysis by analysis'. For example, the risks associated with both asbestos and radiation were well-known to the medical profession in the 1930's, and yet no preventive or regulatory action appears to have been taken." (page 15)
   >
   > "The National Institute of Occupational Safety and Health's position ... is that 'excessive cancer risks have been demonstrated in all fiber concentration studies to date. Evaluation of all available human data provides no evidence for a threshold or for a safe level of asbestos exposure'." (page 21)
   >
   > "It is necessary that we should strive for as near zero risk in the workplace as is technologically possible to achieve. For known carcinogens the level of exposure should be zero. For non-carcinogenic agents the level of permissible exposure should be revised downwards from that point at which there are gross effects on society." (pages 17-19)

7. The sum total of all the doses administered to the population is also called the "integrated dose". According to the linear hypothesis, this "integrated dose" is proportional to the total number of damaged cells, of which a certain fraction will become cancerous. Thus the number of cancers can be predicted once the integrated dose is known.

8. This straight-line relationship between integrated dose and cancers is called a linear relationship; hence the name for the linear hypothesis. (see note 16 please!)

9. Dr. Morgan is an esteemed member of the Health Physics community. He is one of the founding members of the International Commission on Radiological Protection, and is today the only emeritus member of that Commission: see also the biographical sketch on page 39.

10. Dr. Morgan has written an excellent article entitled "Cancer and Low Level Ionizing Radiation" in the September 1978 issue of the Bulletin of the Atomic Scientists (pp. 30-41). In this article, Dr. Morgan reviews recent medical evidence which shows not only that the threshold theory is probably wrong, but also that "the cancer risk from exposure to ionizing radiation is much greater than was thought to be the case some years ago." He then gives six documented arguments to show why even the linear hypothesis may consistently underestimate the carcinogenic powers of radiation at low levels. Immediately following the Morgan article is another entitled "The Risks for Radiation Workers", written by Joseph Rotblat. It is also well worth reading, and much to the same effect.

11. Plutonium-239 is one of the most well-known examples of an alpha emitter. Since the radiation from plutonium has little penetrating power, plutonium can be stolen and transported with relative ease. However, when inhaled into the lungs, it is extraordinarily toxic. A speck of plutonium weighing only one thousandth of a gram can, if inhaled into the lungs, cause death within hours by massive fibrosis of the lungs. A speck of plutonium only one thousandth of one thousandth of a gram (in other words, a microgram), if inhaled into the lungs, may cause a fatal lung cancer to develop many years or even many decades after exposure, but with almost 100 percent certainty. See the article by Dr. John Edsall, Professor Emeritus of Biochemistry at Harvard University, entitled "Toxicity of Plutonium and Some Other Actinides" -- Bulletin of the Atomic Scientists, September, 1976.

12. Once alpha emitters are inside the body, they cannot be detected by any external instruments. The degree of internal contamination can only be inferred by such things as urine analysis and sputum analysis, which give only crude results.

13. Neutron radiation, like alpha radiation, is also more effective in causing cancer than either beta or gamma radiation. Although the same amount of energy will be delivered to the tissues by a given dose of radiation, no matter whether it is made up of neutrons, alpha particles, beta particles, or gamma rays, it is known that a higher density of ionization is caused by alpha particles and neutrons than by the other types of ionizing radiation. Higher ionization means that more chemical bonds can be broken, and therefore greater biological damage can be done, per gram of tissue exposed. See "linear energy transfer", "rad", "rem", and "quality factor" in the glossary (pages 43-44).

14. Several hypotheses have been advanced to explain why this should be so, but none of them has been thoroughly tested. According to one theory, there is overkill at high doses (cells which would have developed into cancer cells are instead killed by the high dosage) and therefore, at low doses, more cancer is observed per unit dose. Another theory is that the cell membrane is more effectively damaged at low dose rates than at high dose rates, thereby allowing other carcinogens (such as chemical carcinogens) easier access to the nucleus. (If this theory is correct, then not only alpha radiation but all forms of ionizing radiation should be more effective in causing cancer at low dose rates.) Still other theories deal with the distribution of alpha emitters inside the body; if a "warm particle" or a "hot particle" is lodged in the lung, it is believed by some that such a particle may be more effective in causing cancer than if the same total dose were evenly distributed throughout the lung. But all of these theories are conjectural, and so we will limit ourselves to discussing the experimental and epidemiological evidence which indicated that more cancer is observed per unit dose at low dose rates of alpha exposure, whatever the reason for that might be.

15. See reference 2, as well as exhibits 1 and 2 on pages 9 and 10.

16. Since this was written, I have received a list of 12 references from Bob Wilson, Director of the Health and Safety Division of Ontario Hydro, which are supposed to provide evidence indicating that the linear hypothesis is conservative for low level alpha radiation. Although I have not yet had time to do a thorough review of all of these papers, it is clear that some of them do not suggest a different conclusion from that stated in the text.

    For example, the very first reference given by Mr. Wilson is the famous paper by Ševc, Kunz, and Plaček, which appeared in Health Physics in June of 1976, entitled "Lung Cancer in Uranium Miners and Long Term Exposure

to Radon Daughter Products". In the concluding paragraph of that paper, the authors state that "the estimate of risk of low doses, obtained by means of linear extrapolation of the relationship between higher doses and effect in a heterogeneous population, need not under all conditions represent the maximum possible risk." In other words, the linear hypothesis may not be conservative at low doses.

Mr. Wilson also cites "Sources and Effects of Ionizing Radiation", the 1977 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Annex G of the UNSCEAR Report entitled "Radiation Carcinogenesis in Man" lists three studies which are supposed to confirm the conservatism of the linear hypothesis for low level alpha radiation. The first study is the paper by Ševc, Kunz, and Plaček just referred to. The second is co-authored by Dr. Victor Archer, who has since changed his mind about the linear hypothesis as a result of more detailed analusis of all the existing evidence. The third study is my reference 1, the Ham Commission Report, which states: "This analysis is most emphatically not offered as the basis for any estimate of risk per unit dose. . . . [which would be a trivial task if the linear hypothesis were true]. It should also be possible ... to accomodate the idea of a response more than proportional to cumulative dose." (from Appendix C, "Radiogenic Lung Cancer in Uranium Miners 1955-74"). Thus none of the three UNSCEAR references indicates an unqualified confirmation of the linear hypothesis, and indeed at least two of them explicitly include the possibility of non-linearity at low doses.

17. For more information about Dr. Archer, see the biographical notes on page 38.

18. For more information about Dr. Gofman, see the biographical notes on page 38. The data on which Dr. Gofman bases his calculations are the same as those used in the Ontario Ministry of Housing's Table 1 and Table 2, reprinted on pages 21 and 22.

19. "It is generally assumed that the risk of radiation induced cancer is proportional to the exposure and that there is no absolutely safe threshold below which the risk is zero. It should be borne in mind, however, that no direct proof of carcinogenic effect, at extremely low doses, exists at this time so the assumption of "no threshold" is conservative." (M.O.H. Report, reference 5, page 5)

    It is indeed strange that the Ministry of Housing should consider that no evidence provides a proof of conservatism! Especially since, in Appendix C of the Ham Commission Report (reference 1) the threshold hypothesis is tested and

"easily discredited" on solid statistical grounds. The report then goes on to say: "The possibility of a 'safe' threshold dose cannot be excluded by these, or any other finite amount of data. However, further analyses, to be reported in full elsewhere, have shown that, to be at all plausible in relation to the Ontario experience, a postulated threshold would have to be lower than 10 WLM." This is not very encouraging to those who still believe in a safe threshold!

20. According to Dr. Muller's analysis (in reference 5), the volume of air inhaled daily by men and women was obtained from ICRP Publication 26, as was pertinent data on the mass of the male and female lungs at different ages. It was also assumed that the retained fraction of radon daughters in the lungs was 70%, that there is no biological effect during the first five years of exposure to radiation, and that all radiation-induced deaths occur within 25 years of initial exposure. Nevertheless, the details of his calculation are very fuzzy, and his results are extraordinarily low. For example, the female mortality figures calculated by Dr. Muller are less than half as large as the ICRP estimates, and the loss of life expectancy for females is also far lower (less than a quarter, in most cases) than the ICRP estimates, as can be seen from M.O.H. Table 6, reproduced on page 27. Why should there be such a wide discrepancy ?

21. See note 5 regarding lead-210 and polonium-210 in tobacco smoke.

22. For more information on Dr. Stewart, see the biographical notes on page 39. Her completed work was published as "Radiation Dose Effects in Relation to Obstetric X-Rays and Childhood Cancer" in Lancet 1185 (June 5, 1970). The findings of her very ambitious epidemiological studies have been confirmed by other studies done by Dr. Brian McMahon of Harvard University ("X-Ray Exposure and Malignancy", Journal of the American Medical Association, v. 183, 1963) and Dr. Irwin Bross of the Roswell Park Memorial Institute for Cancer Research in Buffalo ("Leukemia from Low-Level Radiation", New England Journal of Medicine, v. 287, 1972). Dr. Bross' results come from a study of 13 million human beings in three states; an updated account of his findings can be found in reference 2.

More recently, Dr. Stewart and her statistician colleague George Kneale have assisted Dr. Thomas Mancuso in studying the cancer incidence among workers at the Hanford Plutonium Works in Washington State. The results of this study have appeared as "Radiation Exposures of Hanford Workers Dying from Cancer and Other Causes" in Health Physics, 33, 1977. Using statistics on over 24,000 ex-employees at the Hanford nuclear facility, the authors (Mancuso, Stewart, and Kneale) have shown that as small a dose as 12.2 rads accumulated radiation exposure could lead to a doubling of the normal incidence of most cancers. According to the study, for cancers of the pancreas or lung, the "doubling dose" may be as low as 6.1 rads, and for cancers of bone marrow, the

"doubling dose" is less than 2.5 rads. These latter cancers, it is worth noting, are often induced by alpha emitters such as plutonium, or radon daughters, or radium.

It is a sad commentary that both Dr. Bross' funding and Dr. Mancuso's funding have been terminated, so that these men are unable to complete the investigations which they have begun. In a similar way, the ground-breaking work of Gofman and Tamplin was terminated back in the late 1960's with much acrimony. I have included as an exhibit a revealing letter, written by Dr. Karl Morgan to James Schlesinger on May 25 1977, concerning the "biopolitics" which seems to be at work in suppressing scientific researches which do not conform to the official dogma that radiation is relatively harmless at low doses. Dr. Morgan's letter appears as Exhibit 11 on page 40.

23. Testimony given at the Elliot Lake Environmental Assessment Hearings indicated that any gouges in the sealant (caused perhaps by children playing roughly, or by men sliding heavy furniture over the floor) would allow almost as much radon gas into the basement as if there were no sealant whatsoever. The situation is made even worse by the fact that radiation is not perceptible to any of our senses, and most homeowners cannot make measurements of the radon gas levels in their own homes. Thus there could be serious deterioration of the protective systems, which could go undetected for a very long time.

24. In 1973, the U.S. Environmental Protection Agency published a substantial report entitled "Environmental Analysis of the Uranium Fuel Cycle", EPA-520/9-73-003-B. According to the Report, about 200 extra lung cancer deaths per century could be expected to occur among members of the general population as a result of the radon gas emissions from a typical uranium tailing pile (assuming that only 5% of the radon gas produced actually escapes into the atmosphere, and assuming that population does not grow at all). Dr. Pohl's article simply takes the E.P.A. figures and converts them into a figure which represents the number of extra deaths that one could expect in the long run per 1000 megawatt-years of nuclear electricity produced. The number he comes up with is 396 extra deaths per gigawatt year of nuclear electricity, which is far in excess of the number of deaths usually attributed to an equivalent coal-burning plant. It is worth noting that both the E.P.A. figures and Dr. Pohl's figures are based on the linear hypothesis. If the linear hypothesis underestimates the actual risk by a factor of ten or thereabouts, as argued in this paper, then the actual health effects of uranium tailings may be far worse than anyone has yet reckoned.

25. See the articles by Morgan and Rotblat mentioned in note 10. See also the Proceedings of a Congressional Seminar on Low Level Ionizing Radiation, reference 1.

26. Reference 7, page 350.

"NO SAFE DOSE OF RADIATION"
-- NUCLEAR AUTHORITIES (1982)

In November 1981, two atomic workers at Chalk River, Ontario, were granted full pensions because of cancers which they had contracted as a result of radiation exposure on the job. "We acknowledge that it was probable that their cancers were caused by working here," said a statement issued by Chalk River Nuclear Laboratories, despite the fact that neither of the men had ever been over-exposed to radiation.

Thomas Arnold was awarded a pension of $1335 a month by the Ontario Workman's Compensation Board (WCB), on the advice of Atomic Energy of Canada Limited (AECL). Arnold credits AECL with doing all the work to get him the pension. He developed lymph cancer during his 28 years of work as a reactor maintenance man at Chalk River.

The other case involves a 31-year veteran of Chalk River who died of leukemia shortly before the WCB granted him compensation. His widow was awarded $490 a month for life, the maximum permitted under WCB rules. A spokesman for the WCB said there is a third claim pending from Chalk River over a case of skin cancer. Meanwhile, a 50-year old Pembroke man has also filed a claim with the WCB. Raymond Paplinskie, who has lost an eye and most of the skin on one side of his face, says that he got cancer of the sinuses from doing nuclear cleanup work following a 1958 reactor accident at Chalk River.

AECL spokesman Hal Tracy explained that the nuclear industry in Canada accepts the theory that there is no safe threshold limit for radiation exposure; hence, it must also be accepted that any dose at all has the potential for harm, and that eventually there will be some evidence of this harm. "Possibly there will be more cancers among our workers," said Mr. Tracy. "These first cases weren't a total surprise. Deaths due to radiation exposure had been predicted. We've always believed there was an increased risk."

Robert Potvin, a spokesman for the Atomic Energy Control Board (AECB), which regulates the Canadian nuclear industry, said that the two cases of compensation have "no implications" from the safety standpoint. They "simply confirm the long-standing expectation" that nuclear workers run a higher-than-usual risk of cancer due to years of exposure to low-level radiation, he said. "Our limits admit that any dose can increase the risk and, on that premise, cancer deaths are not unexpected." He added that "studies say the average risk under these limits is comparable to the risk in an industry with a high safety standard -- for example, manufacturing shoes."

A spokesman for Ontario Hydro, Richard Furness, said in an interview with the Toronto Star that "no one has ever died or suffered lost-time injuries due to radiation at a Hydro nuclear plant -- or any other Canadian nuclear facility." When told about the AECL acknowledgement of two cases at Chalk River, Furness remarked: "Oh. Well, there goes that record."

Ontario Hydro's Health and Safety Director Bob Wilson said it was time the public recognized the facts. For every hundred million hours of work done under radiation exposure (at no more than the permissible limits) about 2 to 4 otherwise unexpected cancer cases will develop, Wilson said. "We have never said a radiation worker is without risk," he insisted, but added that radiation workers are 10 to 100 times less likely to die from work than such people as fishermen, forestry workers, miners or even Hydro linemen.

But a well-informed AECL worker told the Toronto Star that "this is going to open an intense debate about safety. What can we expect from all the other live or dead cancer victims who have long-term low-level radiation exposure at AECL or Ontario Hydro? It could mean that the whole system of predictions that five rems of radiation was an acceptable dose for workers is dead wrong."

Critics of the nuclear industry have argued that the industry's predictions could prove fatally wrong for many more workers than anticipated. It can take 20 years or more for cancers to develop from low-level long-term radiation exposure, and at least 250 Hydro workers and about the same number at AECL are coming up for the 20-year turning point.

In fact, a special report on the medical effects of alpha radiation published by the AECB in September 1982 indicates that the present permissible exposure limits could result in a quadrupling of the risk of lung cancer deaths among uranium miners, whether they smoke or not. This conclusion is based on actual mortality figures among uranium miners from Colorado, Sweden, Czechoslovakia, Canada, and elsewhere.

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