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Written submission from Antone L. Brooks

Mémoire de Antone L. Brooks

In the Matter of

Bruce Power Inc. – Bruce A and B Nuclear Generating Station

Request for a ten-year renewal of its Nuclear Power Reactor Operating Licence for the Bruce A and B Nuclear Generating Station

Commission Public Hearing – Part 2

May 28-31, 2018

À l'égard de

Bruce Power Inc. - Centrale nucléaire de Bruce A et Bruce B

Demande de renouvellement, pour une période de dix ans, de son permis d'exploitation d'un réacteur nucléaire de puissance à la centrale nucléaire de Bruce A et Bruce B

Audience publique de la Commission – Partie 2

28-31 mai 2018

Canada

Testimony Dr. Antone L. Brooks,

Bruce Power May 30-31

Introduction

We were out in the cool early morning of Utah's Dixie hoeing and watering the new corn before the heat of a day set into the desert. I enjoyed watching the water trickle down the rows bringing life to this hot desert climate in St. George Utah where little life was possible without irrigation. Very early morning we did much of the hard work, a tradition passed on for generations. For a short second, the sky lit up to the brightness of mid-day then returned to the pale light of a new day. "Quick", dad would say "count and you can tell how far away the atomic bomb was on Jackass or French Man Flats since we know the speed of light and how fast the shock and sound wave travels". The earth quivered as the shock wave rolled across the sleepy Southern Utah town. I was really into such exercises and was pleased to announce that the bomb was less than 100 miles away. We had just experienced another of the one hundred and three above ground Nuclear Weapons tests at the Nevada test site. This was an exciting experience for me and produced a memory that was engraved into my young brain. It also triggered questions. Just how wonderful is radiation? Were these A-bomb tests wonderful or deadly? I have lived and worked through some very interesting times in radiation biology.

Early research: How does radiation move through the food chain?

I attended Dixie Jr. College in St. George Utah then transferred to the University of Utah where I got my BS and MS degrees. In 1962 I got my MS degree in the field of Radiation Ecology. For my research I followed the transfer of radioactive fallout through the food chain with final evaluation of the levels in humans. During this research it was shown that the radioactive materials moved through the food chain and ended up in the human population. One of the prime radionuclides of concern then, and now because of Fukushima, was ^{137}Cs. We counted a small number of farmers to determine the level of ^{137}Cs present in the population. These results were published (Brooks et al. 2016). The highest-exposed individual who was a 19 year-old male, he had highest daily milk consumption (3.0 l/day) and had of total body burden of 1,398 Bq, and the lowest-exposed individual was a 26 year-old female with a burden of 211 Bq. Her daily milk consumption was 0.5 l/day. This illustrates that there was about a factor of 6 difference in the

body burden of ^{137}Cs in this small population of exposed individuals, which was primarily driven by milk consumption. In addition, I was counted once (726 Bq) to look at ^{137}Cs in non-farmers. In this case, the high total body burden could be related to the large amount of venison and milk in my diet, which contained high levels of ^{137}Cs.

What health effects are caused by different radionuclides? What happens with changes to the route of exposure and the dose?

The radioactive materials were in everything and on everything. My question then became would the fallout result in health effects? I devoted my scientific life trying to address this question. After completing my PhD at Cornell University where I studied the effects of radiation on chromosomes of both somatic and genetic cells I got my first job in Albuquerque New Mexico at the Lovelace Inhalation Toxicology Research Institute (ITRI). This provided me with the opportunity to study both the genetic and cancer risks from internally deposited radioactive materials.

While at Lovelace I was involved in research to address several very important radiobiology questions.

1. **Is ^{239}Pu the most hazardous substance known to man? Does a single particle of ^{239}Pu represent a very high cancer risk?** This "hot particle" hypothesis was raised since the alpha particles have a short range in tissue and deposit a lot of energy in a few cells. This results in a high cellular dose to cells close to the particle while the tissue dose could be small. This high dose was thought to result in a high risk for the induction of cancer.
2. **Was internally deposited radioactive material that deposited non-uniformly in the body more hazardous than uniform radiation exposure?** This was especially a concern for ^{90}Sr which was deposited and retained for long periods of time in the bone.
3. **Are there threshold doses for internally deposited radioactive materials below which no detectable change in life span or cancer can be observed?**

1. Is ^{239}Pu the most hazardous substance known to man? Does a single inhaled particle of ^{239}Pu result in a very high cancer risk?

To address the first question, which was widespread in the literature at this time, "Is ^{239}Pu was the most hazardous substance known to man?" My research was focused on the induction of

chromosome aberrations in the liver following the deposition of different radionuclides including ^{239}Pu. The radioactive material was retained in the liver for long periods of time, resulting in large cumulative doses delivered at low dose rates. The cells of the liver divide very slowly and accumulate radiation dose and damage over long periods of time. After accumulating the dose and damage the liver cells were stimulated to divide by partial-hepatectomy, the removal of part of the liver. This made it possible to study the cells in the metaphase phase of cell division and measure the frequency of chromosome aberrations.

Our experimental design was to inject the radioactive materials into the animals, let the radiation dose and chromosome damage accumulate with time, do a partial hepatectomy and score the frequency of metaphase chromosome aberrations induced by each of the radionuclides as a function of total dose and dose rate. As positive controls we exposed the animals whole body to ^{60}Co given as either a single acute exposure, over a matter of minutes, or to a protracted exposure, that was of similar length of time used for the exposure to the internally deposited materials. In these studies the alpha emitting radionuclides ^{239}Pu, ^{241}Am, and ^{252}Cf were used. ^{252}Cf also produced spontaneous fission fragments which impacted the cells. We compared the effectiveness of the alpha emitters in producing chromosome aberrations to internally deposited ^{144}Ce-^{144}Pr a beta gamma emitter and to the positive control, whole body ^{60}Co gamma ray exposures. This resulted in an important paper (Brooks 1975) which demonstrated that ^{239}Pu was no more effective in producing chromosome damage than any of the other alpha emitters, that the alpha emitters were about 20 times as effective in producing chromosome aberrations per unit dose than the beta gamma emitters and that external exposure to gamma rays had the same effectiveness as the internally deposited material if the time of exposure was the same. Acute exposure was much more effective than protracted exposure in producing chromosome damage. The results of this study are shown in Figure 1. There was no unique chromosome damage associated with ^{239}Pu.

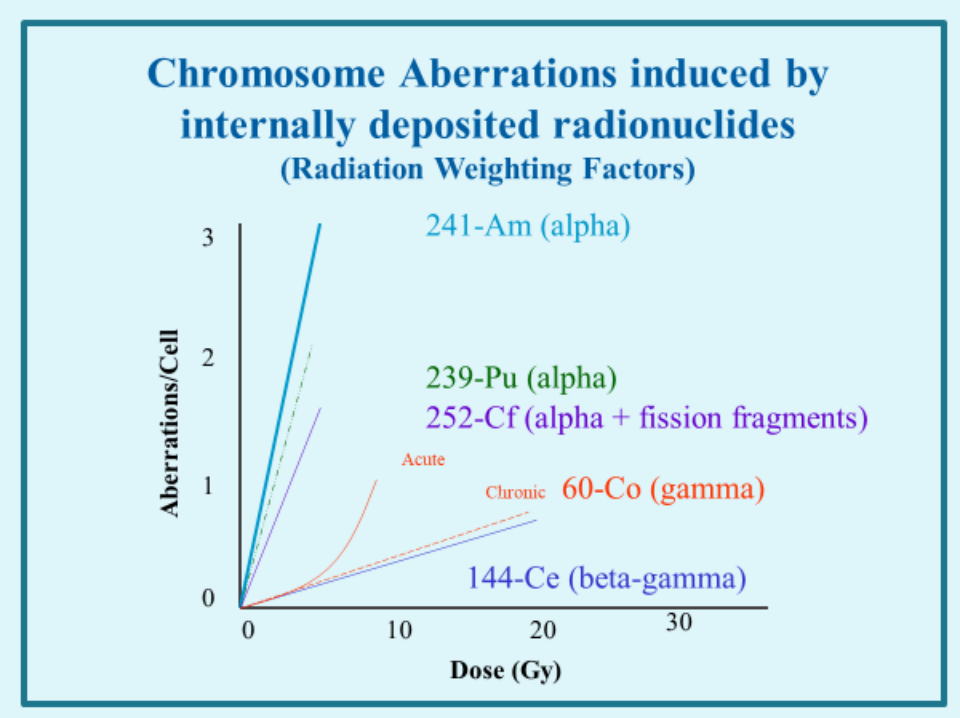

Figure 1.

2. Was internally deposited radioactive material that deposited non-uniformly in the body more hazardous than uniform radiation exposure?

There were important life span studies using the Beagle dog on the effects of internally deposited radioactive materials being conducted at ITRI as well as at a number of different national laboratories to address question 2. Was internally deposited radioactive material that deposited non-uniformly in the body more hazardous than uniform radiation exposure? Each of the Laboratories had dogs with a common genetic background that were exposed to radioactive materials by different routes of exposure, injection, ingestion and inhalation. The University of California at Davis had dogs that were fed different concentrations of ^{90}Sr over their life-times. The dogs were studied carefully for the induction of cancer with special emphasis on bone cancer, since the ^{90}Sr was deposited in the bone (Raabe 2010).

The data from these studies supported the observations in humans for bone cancer induced in the radium dial painters (Rowland 1995). Raabe 2010 demonstrated that for the internal emitter ^{90}Sr which delivered it dose to the bone at a low dose rate, that large doses and long times were required before any bone cancers are observed. This results in a very non-linear dose-response with large doses and times where no cancers were observed. This represents a threshold dose of

about 10 Gy below which no cancer was observed. The research also further documented the fact that the bone is a very radiation resistant organ which results in very small tissue weighting factors.

With funding from Bruce Power, additional further analysis of the dog data for animals that inhaled a very insoluble form of beta-gamma emitting radioactive materials was conducted (Puukila et al. 2017). In these animals the radionuclides were in fused clay particles so they stayed in the lungs and tracheal bronchial lymph nodes. The radionuclides were selected to represent a wide range of physical half-lives which would result in a highly variable dose rate patterns. The physical half-lives, effective half-life in the lungs and the time required for the radionuclide to deliver 90% of the total dose are shown in Table 1.

Table 1. The physical and effective half-lives and the length of time required for deposition of 90% of the total dose for these radionuclides in insoluble fused clay particles.

Radionuclide	Physical half-life	Effective half-life in lung (d)	Time to deliver 90% of dose
^{90}Sr	29 years	600 days	5.5 years
^{144}Ce	285 days	175 days	1.6 years
^{91}Y	59 days	50 days	0.5 years
^{90}Y	2.6 days	2.5 days	8 days

Early analysis of these data illustrated that when the total doses to the lung was less than 20 Gy there was no change in lifespan or cancer incidence (Brooks et al. 2009). Further analysis of the data was conducted working closely with Dr. Doug Boreham and Bruce Power (Puukila et al. 2017).

3. Are there threshold doses for internally deposited radioactive materials below which no detectable change in life span or cancer can be observed?

This research demonstrated that very high doses rates and dose per cell turnover resulted in early death (survival less than 200 days) from acute lung injury. If the dose per cell turnover is high many cells die resulting in chronic inflammatory disease, acute lung injury, pneumonitis and fibrosis which results in death of the animal. As the dose rates decreased survival increased to from 1000-4000 days with less acute lung injury allowing time for the dogs to develop cancer. At these early times and high dose rates many of the dogs died of lung cancer. As the dose rate and dose per cell turnover further decreased, to the point that there was no observed chronic inflammatory disease or tissue disorganization the lung was able to maintain its normal function.

At these doses per cell turnover or dose rate there was no significant increase in lung cancer or decrease in life span. Such data suggest that inflammation plays an important role in the development of radiation induced lung cancer. Thus, there was a large plateau where large total doses delivered at low dose rates did not increase risk. Such results support a non-linear dose response relationship with large thresholds for the induction of lung cancer. This shows that the lung is a very radiation resistant when the dose is localized in the lung and delivered at a low dose rate. These studies further demonstrate that for non-uniform distribution of radioactive material in the body the risk is lower than whole body uniform exposure. In fact, in all the studies on internally deposited radioactive materials conducted by multiple institutions there was no case, that I am aware of, where the non-uniform distribution of the radionuclide in any organ from internally deposited material was more hazardous than external whole-body exposure. This may be related to the sparing of the bodies protective mechanisms many of which are not in the non-uniform radiation field and would continue to function normally.

What are the health effects of alpha emitters such Radon?

My research conducted at the Pacific Northwest National laboratory was focused on alpha emitters including ^{239}Pu and Radon. Radon and its daughter products are the environmental radionuclides that contributes the majority of the natural background radiation exposure. Our research was focused on the health effects of Radon. As the result of this research I was selected to be a member of the BEIR VI committee that working through the National Academy of Science wrote a document on health effects of Radon (NAS/NRC 1998). The bottom line from this report, which was written after the committee had completed its work was, “Next to cigarette smoke Radon is the second leading cause of lung cancer.” This caused some serious disagreement among the members of the committee. This disagreement was reviewed at the National Council on Radiation Protection (NCRP) Taylor Lecture (Brooks 2013). The slide used in that lecture is shown in Figure 2.

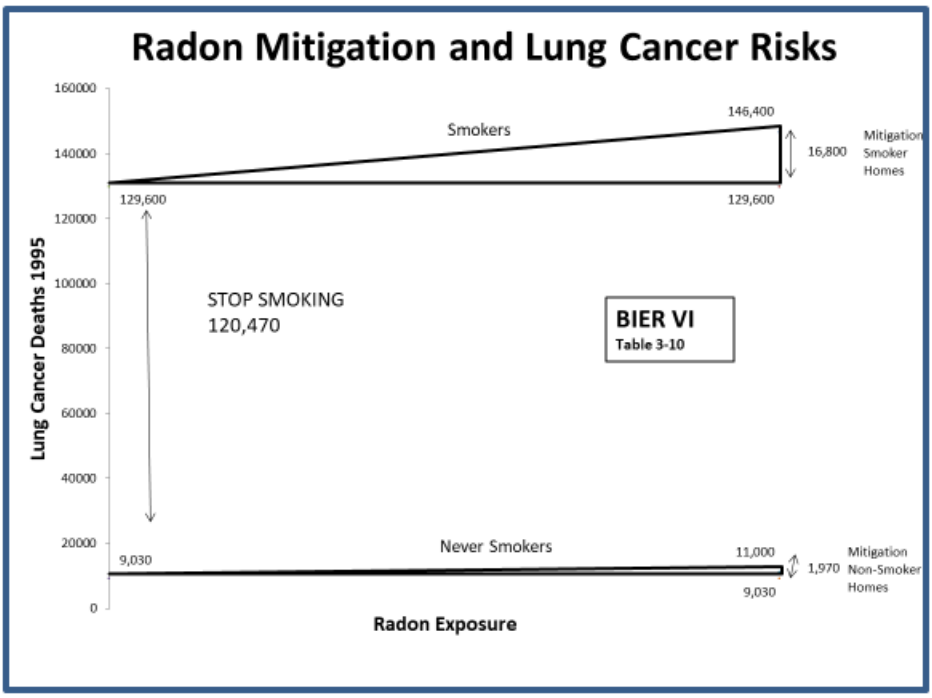

Figure 2. Lung cancer deaths in the Unites States in 1995, by smoking status and increasing radon exposure.

The data for this slide came directly out of the BEIR VI document and illustrates that radon is not the second leading cause of lung cancer. The illustration shows the changes in number of lung cancers as a function of either radon remediation or when one stops smoking. The y-axis shows the lung cancer death and the x-axis is the cumulative radon exposure taken from the homes in the United States. Radon alone produces a very small increase in lung cancer frequency. Cigarette smoking is responsible for most lung cancer. When cigarette smoking is combined with radon then the frequency of lung cancer is increased to a higher than additive level. The take home message from BEIR VI should have been, "Exposure to radon, when combined with cigarette smoking, may be the second leading cause of cancer". The BEIR VI also demonstrated that most of the collective dose used to calculate risk from radon comes from homes that are below the EPA recommended levels for remediation. Thus, remediation will not change the population risk but could influence the individual risk to lung cancer. Thus, remediation of homes of non-smokers has very little impact on lung cancer risk. If a person is concerned about radon induced lung cancer they should stop smoking cigarettes, radon is no longer a problem at low environmental levels.

What are the biological effects of low doses of radiation?

The health effects and risk for radiation induced cancer from high doses of radiation are very well documented and accepted by the scientific community. The major unknown associated with radiation exposure is the response and risk from low doses of radiation delivered at either high or low dose rates to large populations. Since we live in a sea of natural radiation the concern is what happens when additional low doses are added to the normal background level. Do these small additions to the existing highly variable background radiation increase radiation risk? People do not recognize the large amount of radiation that they can encounter in their everyday lives. Dr. Noelle Metting of the US Department of energy published a chart that shows the wide range of doses (over 10,000 times the regulatory limits are delivered during cancer therapy) that we encounter in our daily lives which helps to put low doses into a useable framework. I have copies of this graphic which I can give to any of you that want to have this as a useful reference. With the wide spread use of radiation in medicine especially CT scans and cancer therapy, the total population dose from medical radiation now exceeds the population dose from natural background. The other thing that this graph points out is that we are regulating environmental radiation exposures to levels that are lower than background by two orders of magnitude. Natural background is highly variable around the world and many large populations live in environments that are more than 100 times as high as found in the United States or Canada.

To help understand the biological responses induced by low doses of radiation the US Department of Energy initiated a research program in 1998 to fund research in the low dose range. For this program, a low dose was defined as 0.1 Sv or 100 mSv. Extensive research was conducted in this program using modern biology and advances in technology. These new techniques and equipment made it possible to measure many changes not observable in the past. Scientists from around the world were funded under this Program if they had techniques or methods developed that were unique. Several projects were funded in Canada, one at McMaster University and showed that low dose exposures from medical radiation did not increase but actually decreased cancer risk in a mouse. I was the Chief Scientist on the program for many years. My job was to maintain a web site on the publications produced, help communicate the results to the scientific community and the public, help set up annual meetings to monitor research progress and to provide advice to the Department of Energy on research needs and direction. I did not play a role in funding decisions, these decisions were made by the Department of Energy. As

the result of my involvement in the Program I was able to publish a book, “Low Dose Radiation, The History of the U.S. Department of Energy Research Program” published by Washington State University Press in May of 2018. I will be happy to provide copies of the book to anyone on this commission that is interested in it.

The research from the Program resulted in a huge data base on the cell and molecular changes induced by low doses of radiation. Even though the Program was shut down in the United States, the research has continued in several different countries around the world, including Canada. It is not possible in the short space I have here to summarize the results of the research program. It is enough to relate a few small examples. It was determined that the cell and molecular responses to low doses of radiation were very different from those observed following high doses. Thus, the mechanisms of action are different depending on the dose, making a linear extrapolation of cancer or other adverse outcomes scientifically untenable. In addition many observations did not support the current paradigms used in radiation biology. These new data made it necessary to completely rethink how radiation produces diseases, especially mutations and cancer.

A few of the observations that require the paradigm changes are listed for your consideration.

- Bystander effects. Using microbeams it was possible to demonstrate that cells that were “hit” or had energy deposited in them communicate with non-hit cells and produce a large range of biological responses in the non-hit cells. This makes it essential to re-evaluate the “hit theory” as a basic paradigm in radiation biology. This theory was used to describe radiation responses where the cells with energy deposited in them were the only cells responding to the exposure and suggested that “hit” cells were responsible for the biological changes. With the new research and the recognition of processes like cell/cell and cell/tissue communication it became obvious that the target for radiation induction of cancer was much larger than a single cell.
- Adaptive responses. It was observed that if a low dose of radiation (tickle dose) was followed by a high dose (challenge dose) the response to the challenge dose was greatly reduced. This was called the adaptive response. Extensive research on this observation was conducted by scientists in the Program on a wide range of

biological endpoints. A second type of adaptive response was observed. This is where a small dose of radiation decreases the frequency of the measured response to levels below that of the natural background. This suggests the potential for a decrease in risk at small doses. A process of selective apoptosis (programmed cell death) in transformed cells was induced by low doses of radiation again suggesting a decrease in risk below the normal background risk of cancer. It has been established that adaptive responses are observed at all levels of biological organization. The primary paradigm challenged by the adaptive response is the Linear-No-Threshold model, widely used to calculate cancer risk to large populations exposure to low doses of radiation. The data from the Program suggested the potential need to include a negative term in models of risk to represent a decrease in risk.

- Genomic Instability. An additional observation was that of radiation induced genomic instability. It was determined in some systems that a dose of radiation can result in the loss of genomic stability which is an important process during cancer development. This observation challenged the mutation theory of cancer where DNA, mutations and cancer all increased linearly with dose. The induction of DNA damage increases linearly with radiation dose over a wide dose range. However, the processing of that damage is very dose dependent. Thus, many pathways and processes involved in the response to low doses of radiation suggested that the Linear-No-Threshold models of cancer may not be scientifically accurate. It is of interest to note that it was not possible to detect radiation induced genomic instability in irradiated human populations or in normal human cells.
- Dose rate. Extensive research was also conducted at the cell and molecular level to determine the role of dose rate on basic biological processes. Key molecular events on the critical pathway as cells change from normal to cancer were carefully studied to determine how dose-rate influenced these key changes (Brooks et al. 2016). Dose rate had a marked effect on the induction of these cell and molecular responses. The scientific evidence suggested that low dose rate exposure would be much less effective than a single acute high dose rate in making changes thought to be essential in the many steps required to convert normal cells to cancer. Such

research also questions the scientific basis for the Linear-No-Threshold theory for the induction of cancer.

Research demonstrated that all of these observed responses were highly dependent on the genetic background of the animals, cells or molecular systems being used to measure the changes. Thus, genetic background is of prime importance in understanding radiation risk for cancer and other biological endpoints like the induction of cataracts.

Recent work: Radiation, Cataracts and the Lens of the Eye

After retiring, I resigned from my scientific duties as Chief Scientist and webmaster for the DOE Low Dose Radiation Research Program, Board of Directors at the National Council on Radiation Protection, Editor-In-Chief, Radiation and Environmental Biophysics, US Environmental Protection Agency Science Advisory Board, Radiation Advisory Committee and my position as a Professor at Washington State University and went on a mission for my Church. The Web for the DOE Research Program was then given to the Pacific Northwest National Laboratory under the direction of Dr. William Morgan. While I was on my mission I wrote the book, Low Dose Radiation: The History of the DOE Low Dose Research Program. When I returned from the mission Dr. Morgan agreed to have me work on a contract with Pacific Northwest National Laboratory and run the web site for them. This provided me with office space and access to the resources of the National Laboratory. During this time I worked on a number of projects for the Electric Power Research Institute (EPRI). Of recent interest was a project that resulted in an EPRI Publication 2014, "Technical Report, Epidemiology and Mechanistic Effects of Radiation on the Lens of the Eye" with P. Tran as project manager and team members, Dauer LT, Blakely EA, Brooks AL, and Hoel D. (EPRI 2014). For me the take home messages from all the literature reviewed for this publication are:

- Cataracts have a very high spontaneous frequency which increases as a function of age which makes it difficult to determine the change in frequency following radiation exposure.
- There are several different types of cataracts only some of which are increased following rather high acute doses of radiation.

- There seems to be a threshold for the acute radiation dose required to induce cataracts which is very difficult to determine without careful studies of exposed populations using the best techniques available.
- There is limited data on the induction of cataracts following protracted or low levels of radiation exposure such as observed for most industrial and environmental exposures. The data suggests a higher threshold for low dose rate exposure.

My input to this report was more on the cell and molecular changes induced by cataracts. This report and several subsequent publications have resulted in recommendations by the International Council on Radiation Protection (ICRP) with follow on studies by the National Council on Radiation Protection (NCRP). The action of the ICRP to lower the limits for the exposure of the eye are still somewhat controversial with which have resulted in a number of papers some of which support the change and others suggesting that the action is not well based on science (Thome et al. 2018, Dauer et al. 2016). Much of this work has been supported by Bruce Power.

Summary

I have invested my life studying the health effects of ionizing radiation at all levels of biological organization. This life time of research has been directed toward and helped me draw several important conclusions regarding the hazards and health effects of ionizing radiation.

- The fallout in Southern Utah did not produce a measurable increase in cancer frequency. The cancer frequency in Utah is the lowest in the United States. The cancer frequency in Washington County, where I lived and the fallout was the highest, is the second lowest cancer frequency in the State. Still through political action I get $50,000.00 if I develop a cancer shown to be associated with radiation.
- Plutonium-239 is not the most hazardous substance known to man. There are no unique effects from exposure to this radionuclide when compared to other alpha emitting radionuclides. The frequency of chromosome aberrations in the liver of the Chinese hamster are 20 times higher following exposure to alpha emitters than observed following either internal or external exposure to beta and gamma irradiation. Cancer frequency in experimental animals following exposure to ^{239}Pu is between 6 and 10 times as high as that from beta or gamma irradiation. This suggests a need for the reevaluation of the radiation

weighting factor of 20 used in standard setting for cancer produced by high LET radiation. Plutonium particles are less hazardous than uniformly distributed Plutonium in causing cancer.

- There was not a single example of non-uniformly distributed internally deposited radionuclides being more hazardous than external radiation. Many organs are very radiation resistant when exposed to internally deposited radioactive materials. These include bone, lung, liver and adult thyroid. Such data demonstrates a large threshold in the dose response relationship below which no detectable change in cancer frequency can be detected. It also points out the need to reevaluate the tissue weighting factors used in radiation regulations since these organs are all very radiation resistant to low dose rate ionizing radiation exposure. Finally, using cell and molecular biology it was demonstrated that the dose rate effectiveness factor for internally deposited radioactive material needs to be set at a higher value than 1.5-2.0 currently used in regulations.
- Radon is not the second leading cause of lung cancer. Without interaction with cigarette smoke there is little increase in lung cancer following exposure to environmental levels of radon. Remediation of homes for radon has little impact on the predicted lung cancer frequency since most of the collective dose is from radon in homes below the EPA action level. For anyone concerned about radon in the home the first and only useful action for low levels of radon is to stop smoking which will result in a very marked decrease in lung cancer frequency and risk.
- Extensive research on the risk from low doses of radiation has been conducted and has demonstrated that the biological response at the molecular and cellular level are different for low doses and dose rates than observed for single high doses of radiation. Using key events in the critical pathways from normal cells to cancer cells it was demonstrated that dose rate has a marked influence on these changes. This supports the use of a dose rate effectiveness factor that is much greater than two. These observations resulted in the need for basic paradigm shifts in the field of radiation biology. These include changing “hit theory” for a whole organ response to radiation. The Linear-No-Threshold theory has been challenged by observations of adaptive responses at all levels of biological organization. Cancer has been shown to be a very complex disease and the single DNA damage, single mutation and linear extrapolation to linear cancer frequency needs to take this into

consideration. Genomic instability provides another mechanism for radiation induced cancer that requires additional research.

- The high spontaneous frequency of cataracts and the marked increase in the frequency as a function of age make it very difficult to establish a threshold for radiation induced cataracts. There is very little data on radiation induced cataracts following low dose and low dose rate exposure which is the case for most environmental and occupational exposures. These two facts suggest that changing the threshold for the induction of cataracts and regulating at new lower levels is not founded on solid science

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Role of Animal, Cellular and Molecular Data in Cancer Risk Assessment

Bruce Power Hearing

Drs. Antone L. Brooks

Retired Professor WSU

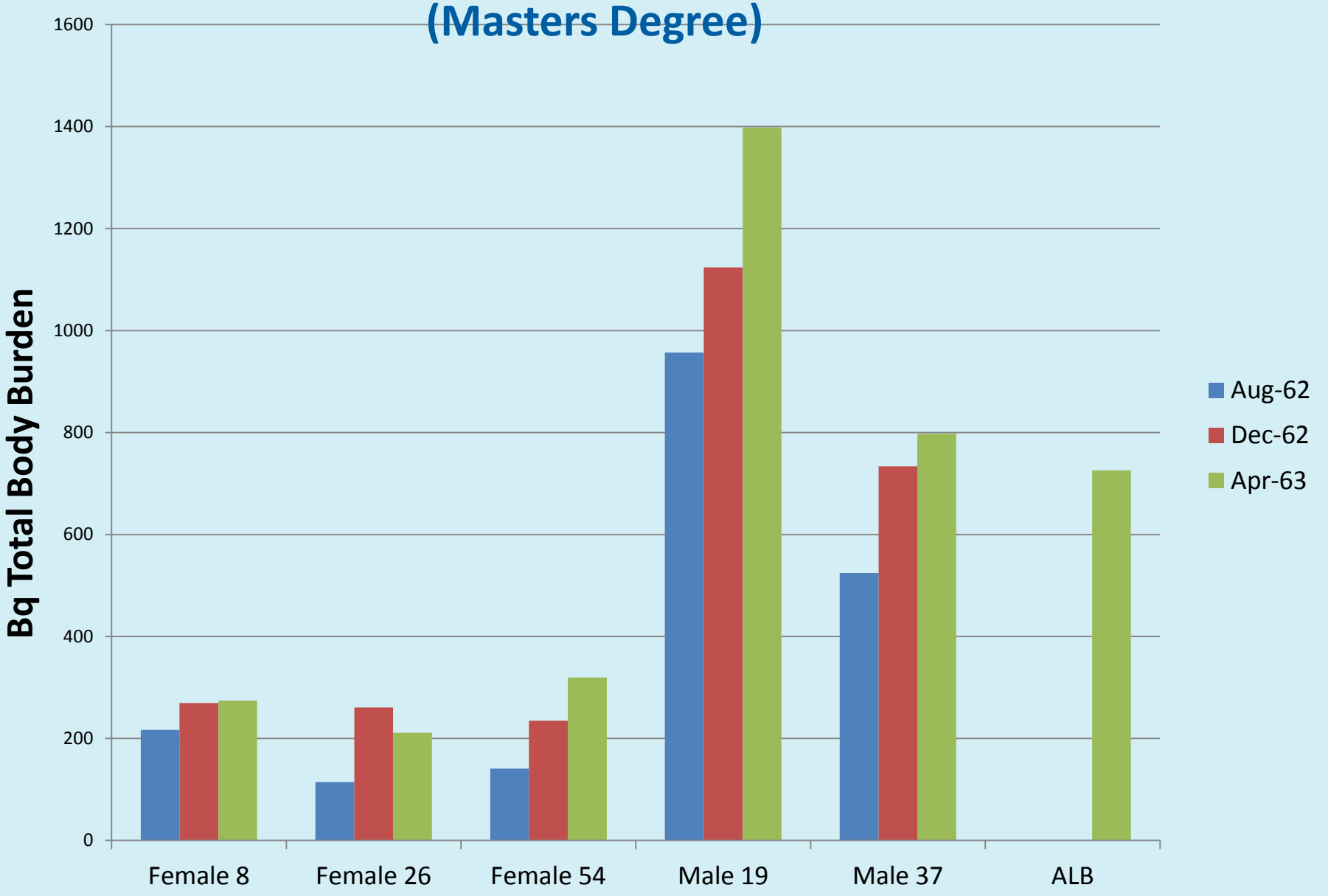
Nuclear weapons were part of my early life

St. George, Utah
1955

My Scientific Training and Background

- BS and MS at University of Utah (Chasing fallout through the environment).
- PhD Cornell University (Chromosome damage)
- Lovelace Inhalation Toxicology Laboratory (Cancer and genetic effects from Internally deposited radioactive material)
- Pacific Northwest National Laboratory (Radon studies)
- Washington State University (Chief Scientist DOE Low Dose Radiation Research Program)
- EPRI (Studies on dose rate effects and cataracts)
- Bruce Power (Post-doctoral fellows, Studies on mechanisms of action and internally deposited radioactive materials).

^{137}Cs in Humans From Fallout Utah (1962) (Masters Degree)

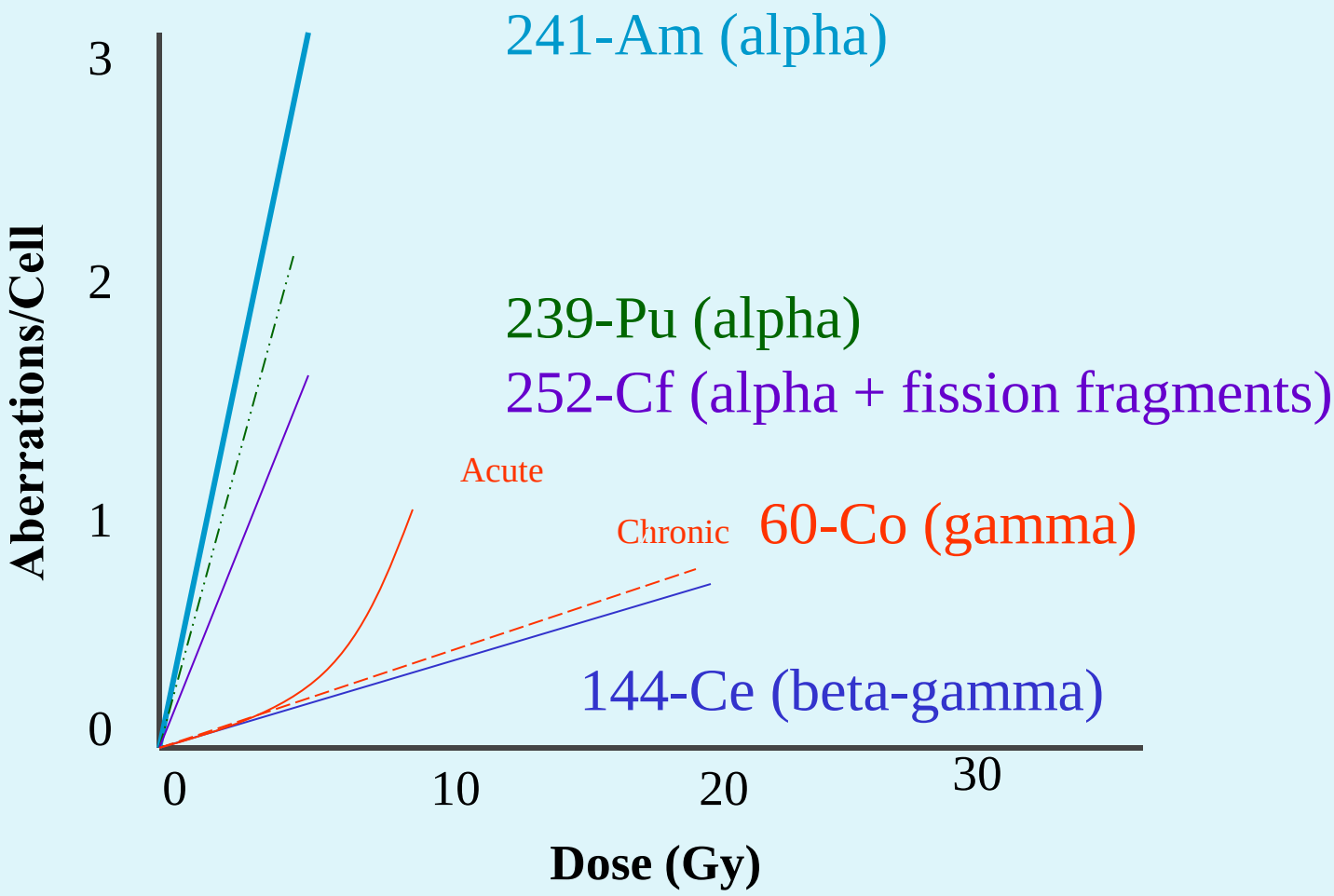


Internal Emitters

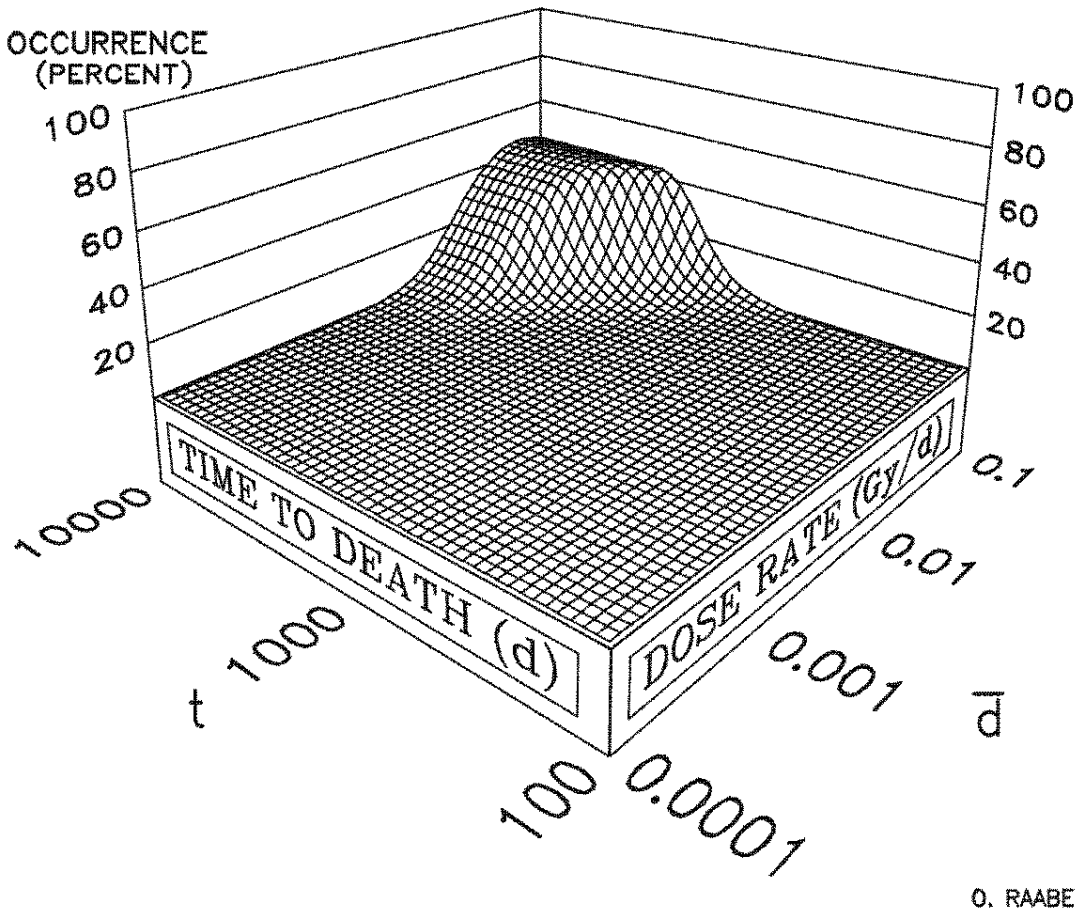
- Most research before the 1960's was focused on single acute exposure
- Very little information was available on the biological changes induced by internally deposited radioactive material
- My research at ITRI focused on genetic and carcinogenic effects of internally deposited radioactive materials

Chromosome Aberrations induced by internally deposited radionuclides

(Radiation Weighting Factors)



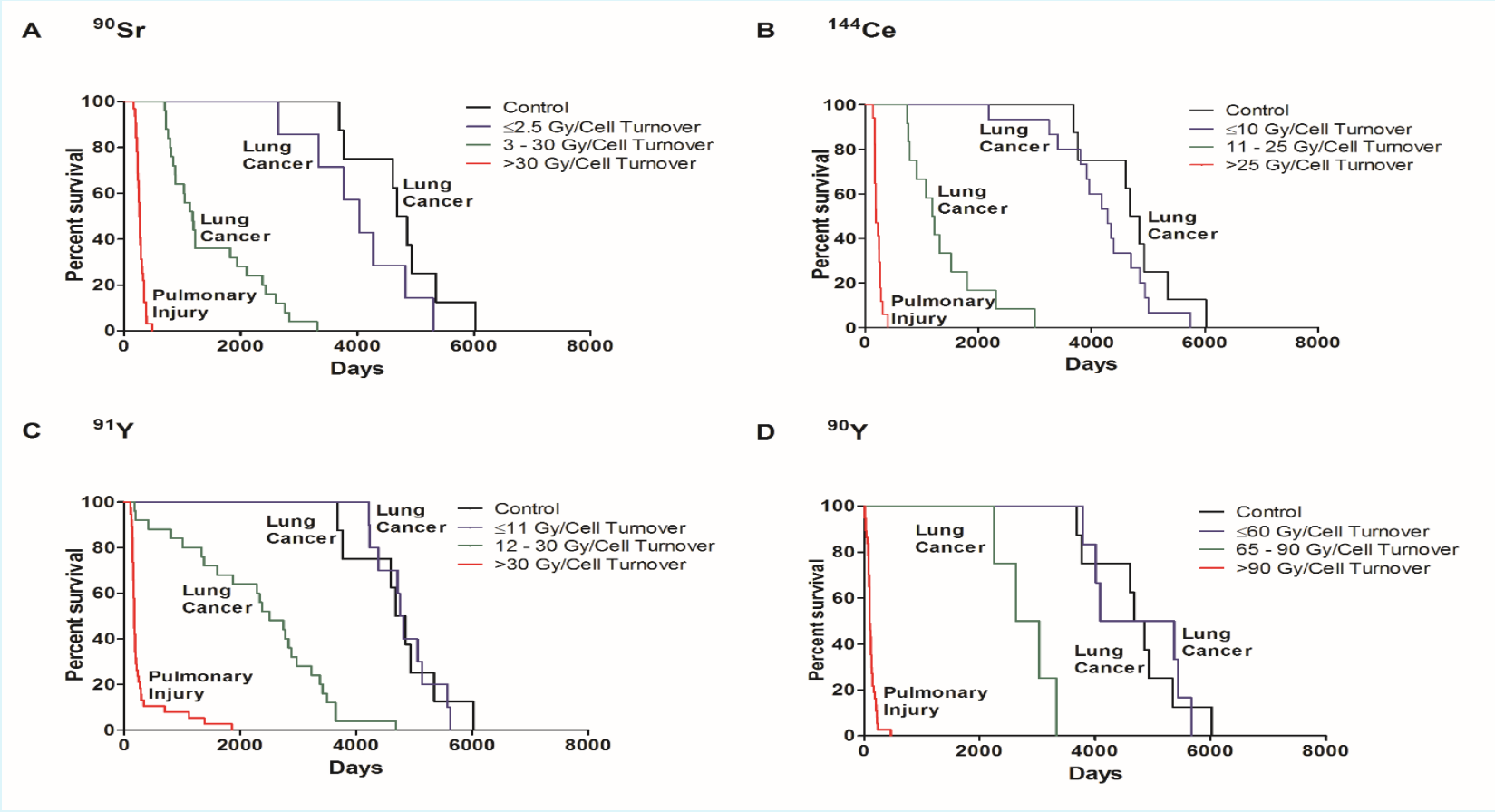
OCCURRENCE OF DEATHS FROM BONE CANCER FOR BEAGLES FED ^{90}Sr AT DAVIS



TIME AFTER BIRTH & AVERAGE BETA DOSE RATE TO SKELETON (LOG SCALES)

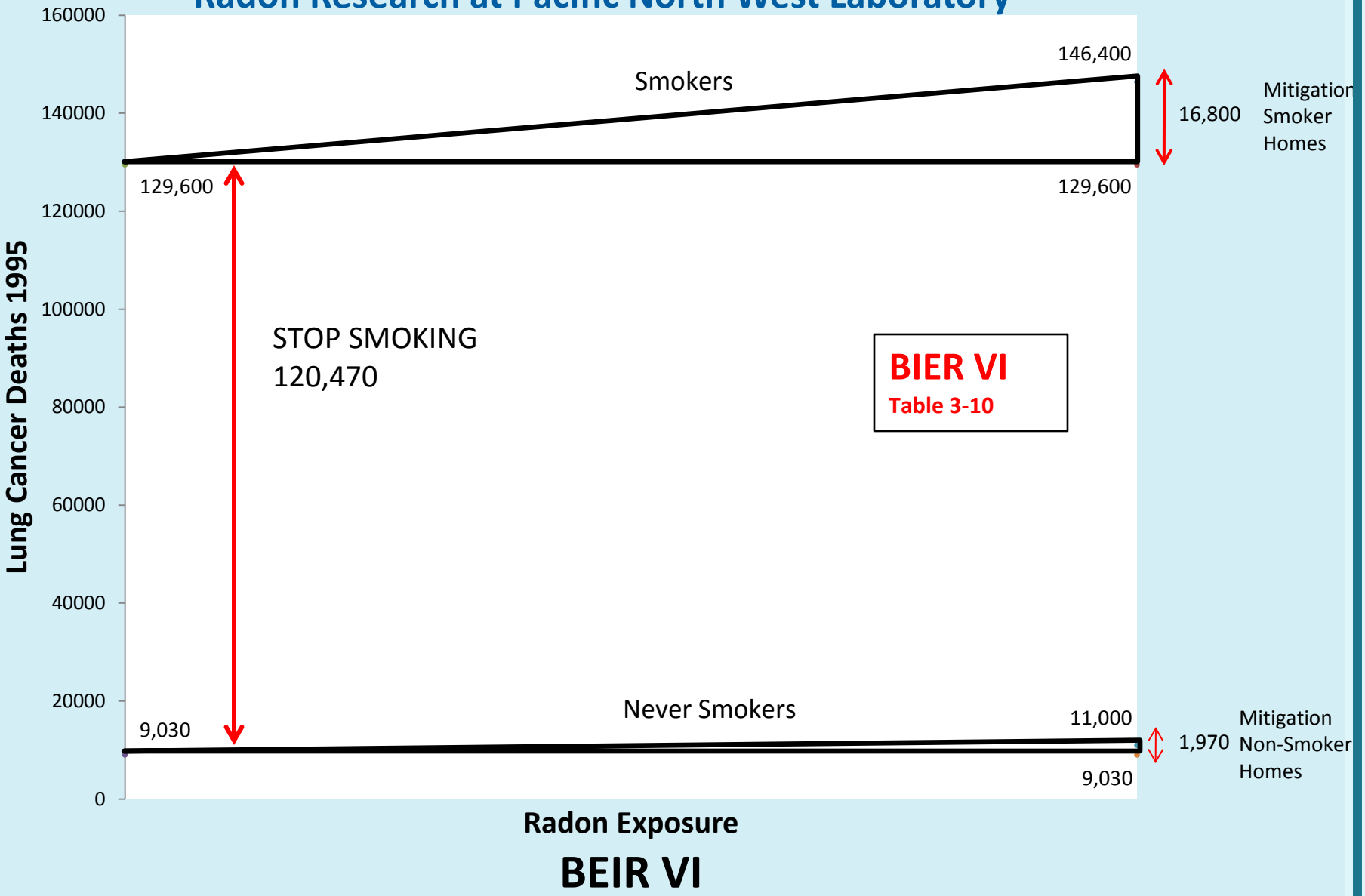
Dose per cell cycle and injury on Lung Cancer

(Puukila et al. 2017) Funded by Bruce Power



Radon Mitigation and Lung Cancer Risks

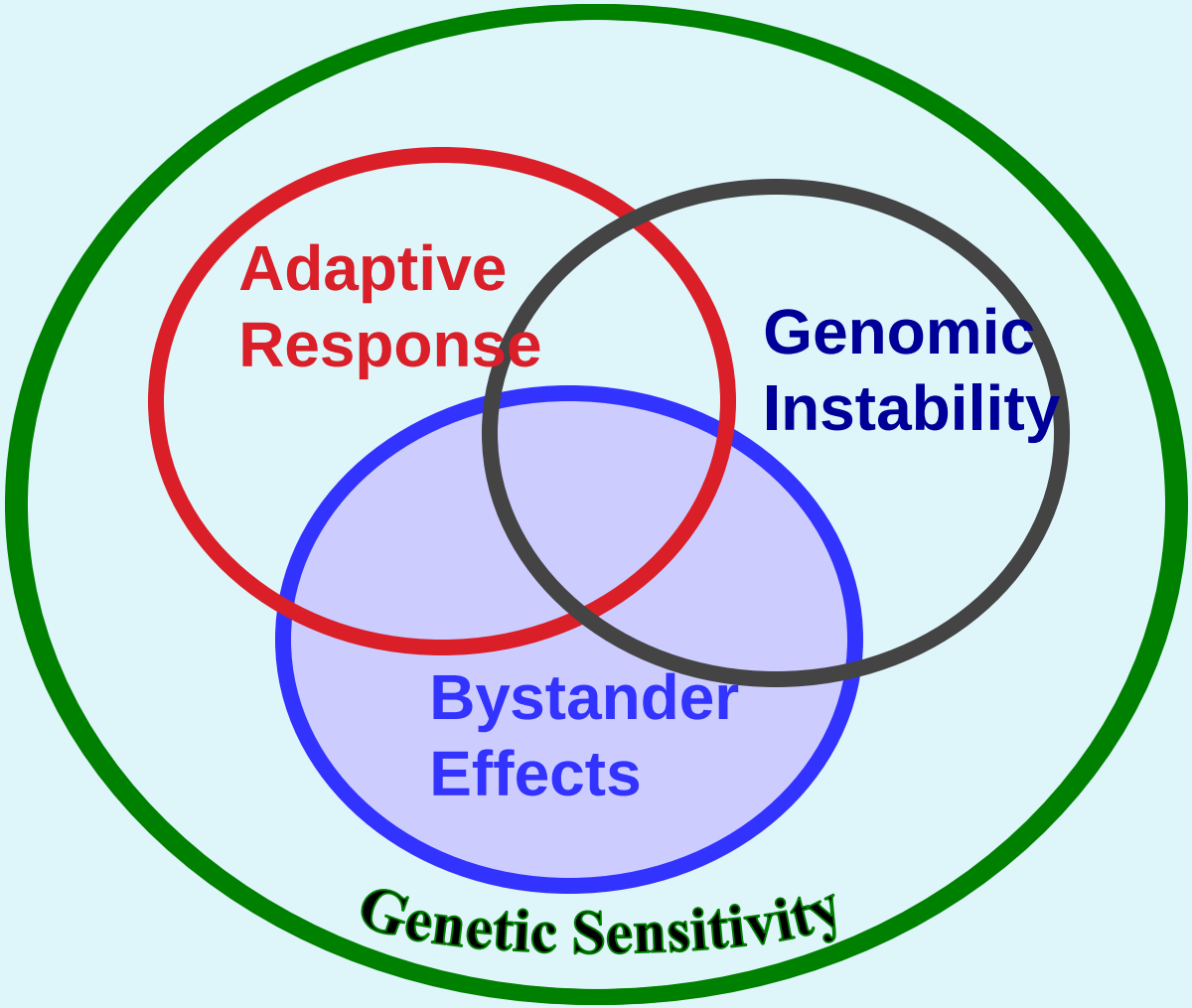
Radon Research at Pacific North West Laboratory



Chief Scientist (US Department of Energy Low Dose Radiation Research Program)

- Are the mechanisms of action the same for low and high doses of radiation?
- Do we need to change current paradigms in radiation biology?
- Is the LNTH an accurate scientific description for the dose-response relationship for cancer in the low dose region?

Biological Responses Induced by Low Doses of Radiation



Need for Paradigm Sifts

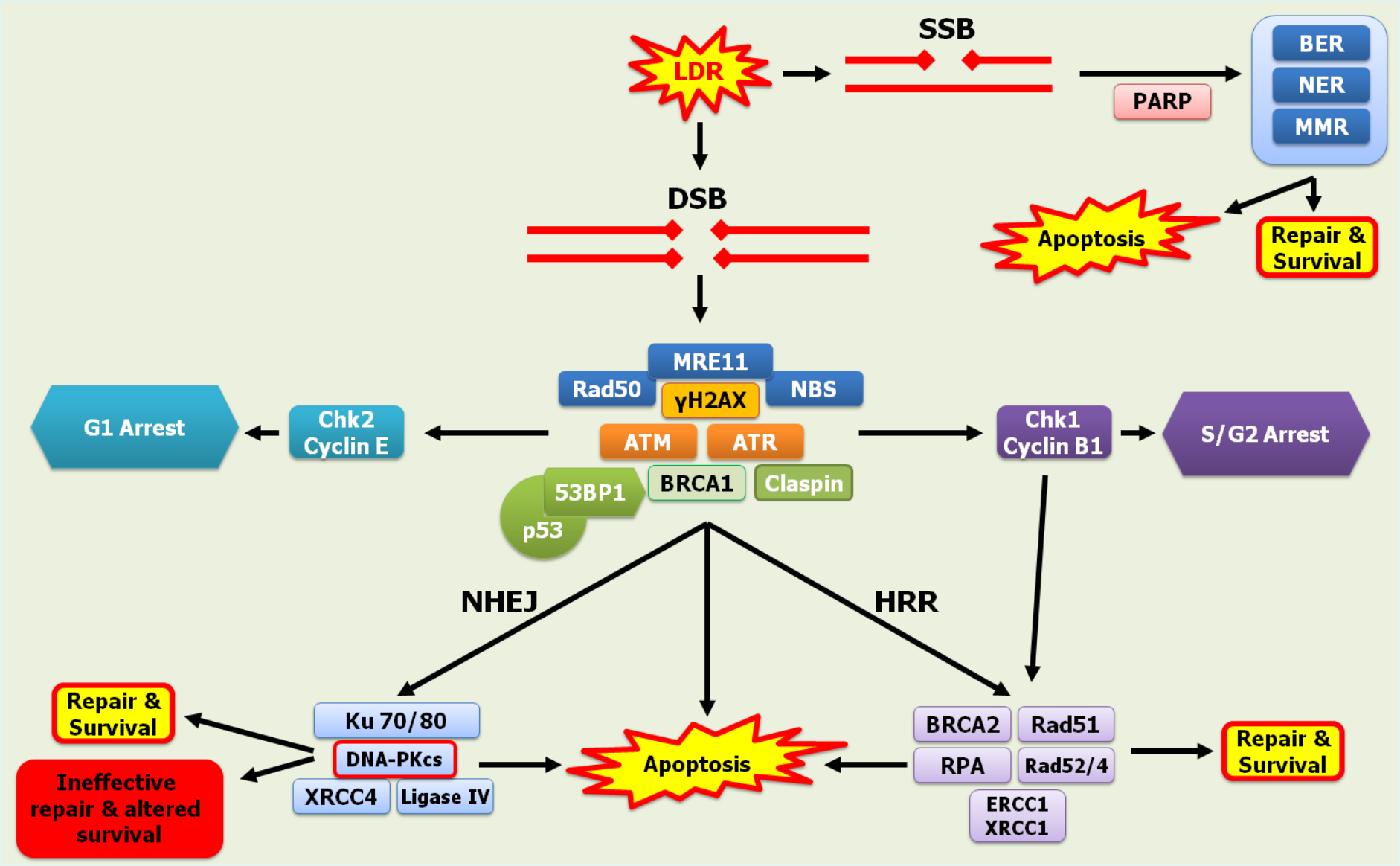
- "Hit Theory" vs Bystander Effects
  - Cells without energy deposited in them respond
  - Target size is much bigger than a cell
- LNTH vs Adaptive Protection
  - Low doses of radiation protect against subsequent high doses.
  - Low doses of radiation reduce background response.
- Mutation theory vs Complex Cancer Pathways
  - Cancer is a complex disease a single mutation may not be adequate to produce cancer.

Modern Molecular Biology and Mechanisms of Action

- Using newer equipment and molecular tools, many mechanisms of action for low doses radiation response are identified.
  - DNA damage and repair
  - Radiation induced apoptosis
  - Adaptive responses to low dose radiation
  - Bystander effects
  - Epigenetic effects following low dose radiation
- All these mechanism impact the key events on the critical pathways to cancer and reduce the effectiveness of low doses and dose rates relative to that predicted by the Linear No Threshold Hypothesis. (Brooks et al. 2016)

LDR & DNA Damage

Funded by Bruce Power (Sujeenthar Tharamalingam et al. in preparation)



Summary

- The scientific community knows more about the health effects of radiation than any other environmental factor.
- In all cases internally deposited radioactive material was less hazardous than predicted from external exposure.
- Large threshold doses for cancers induced by internally deposited radioactive materials (Lung, bone, liver, thyroid).
- Animal, Cell and molecular biology show that low dose rates are much less hazardous than high dose rate exposures.
- Radon risk is minimal without cigarette smoking.
- Basic biology suggest that the mechanism of action are different for high and low dose and dose rate radiation exposures.
- Mechanistic studies demonstrate large thresholds for low dose and dose rate radiation exposures.