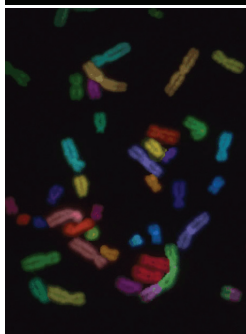
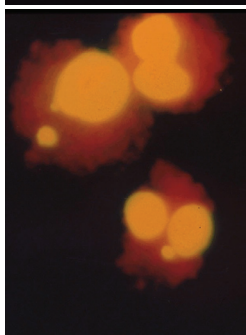


Forty-Fourth Annual Meeting Program



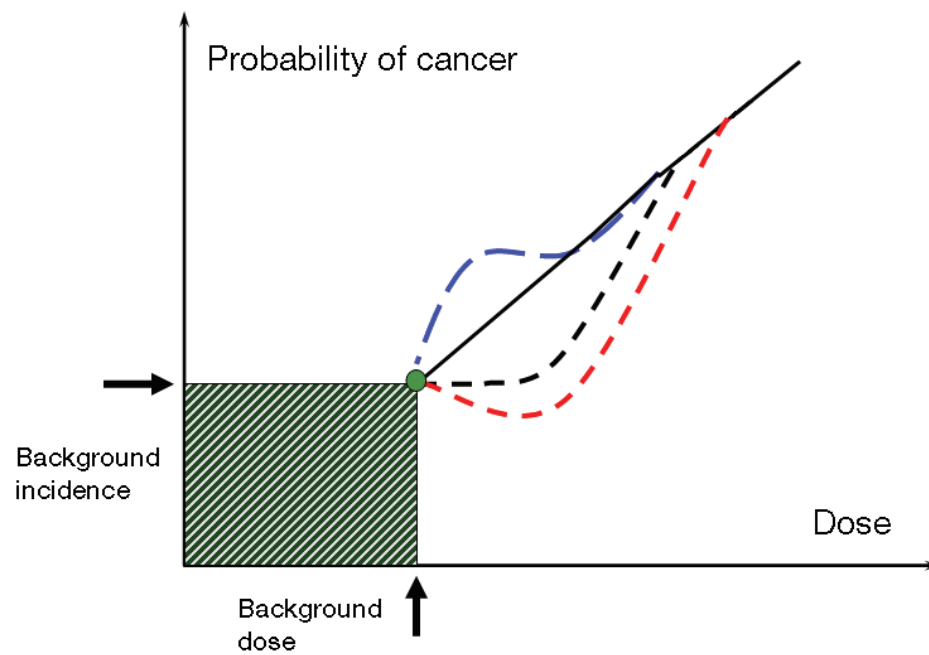
Low Dose and Low Dose-Rate Radiation Effects and Models



April 14–15, 2008

Bethesda North Marriott Hotel
& Conference Center
5701 Marinelli Road
North Bethesda, MD 20852

Dose-Response Relationships



See NCRP Report No. 136 (2001) for a detailed description of the contrasting types of dose-response relationships.

On the cover:

- *top*: Two nuclei have each been “hit” by three alpha particles from a microbeam and show activated γ H2AX foci at the site of the traversal.
- *center*: Chromosome painting technology makes it possible to identify each human chromosome and characterize the number, location and types of aberrations produced by ionizing radiation.
- *bottom*: Measuring the frequency of micronuclei provides a rapid measure of cytogenetic damage, which increases as a function of radiation dose.



Introduction

Low Dose and Low Dose-Rate Radiation Effects and Models

Forty-Fourth Annual Meeting of the National Council on Radiation Protection and Measurements (NCRP)

Potential human health effects of low doses of ionizing radiation such as those experienced in occupational and medical exposures are of great contemporary interest. Considerable debate exists over the applicability of a linear-nonthreshold model for characterizing the biological responses and health effects of exposure to low radiation doses, and alternative models have been proposed. A related subject of interest and debate is the effect of the rate of delivery of radiation doses on the biological and health outcomes of exposure. The primary goal of the 2008 NCRP Annual Meeting will be to bring these issues into the perspective of currently available data and

models of the biological responses and human health impacts of exposure to low doses of radiation. The meeting will feature presentations by international experts on the topics of (1) molecular, cellular, tissue, and laboratory animal studies on the effects of exposure to low dose and low dose-rate radiation, (2) results of epidemiological studies on human health effects of low radiation doses in occupational, medical and other exposure scenarios, (3) potential impacts of these findings on future regulatory guidance and public health policy. The perspectives of research scientists, public health officials, and regulatory agencies will be presented.

Program Summary

Monday, April 14, 2008

Opening Session

- 8:00 am **Welcome**
Thomas S. Tenforde
President
National Council on Radiation Protection and Measurements
- 8:15 am **Fifth Annual Warren K. Sinclair Keynote Address**
Issues in Quantifying the Effects of Low-Level Radiation
Dudley T. Goodhead
Medical Research Council, United Kingdom
- 9:15 am **Overview of Goals of the Meeting**
Antone L. Brooks
Washington State University at Tri-Cities
- 9:35 am **Low-Dose Extrapolation of Radiation-Related Health Risks: Status of Human Studies and State of the Art**
Charles E. Land
National Cancer Institute
- 10:05 am **Break**
- Molecular, Cellular, Tissue and Animal Radiation Responses of Relevance to Radiation Protection**
Gayle E. Woloschak and Amy Kronenberg, *Session Co-Chairs*
- 10:25 am **Molecular Responses: Introductory Remarks**
Amy Kronenberg
Lawrence Berkeley National Laboratory

- 10:30 am **DNA Damage and Repair as a Factor Contributing to Risk from Radiation**
Penny A. Jeggo
University of Sussex, United Kingdom
- 11:00 am **Low-Dose Gene Expression Phenotyping – Molecular Pathways for Radioprotection Against DNA Damage and Chromosomal Abnormalities in Tissues**
Andrew J. Wyrobek
Lawrence Berkeley National Laboratory
- 11:30 am **Radiation Protection and Nontargeted Cellular and Tissue Responses at Low Radiation Doses**
William F. Morgan
University of Maryland School of Medicine
- 12:00 pm **Lunch**
- 1:15 pm **Low-Dose Radiation Responses in Cells, Tissues and Animals: Introductory Remarks**
Gayle E. Woloschak
Northwestern University
- 1:20 pm **Chromosome Aberrations as a Function of Dose, Dose Rate, and Linear Energy Transfer: Implications for Radiation Risk**
Michael N. Cornforth
University of Texas Medical Branch
- 1:50 pm **Factors that Modify Radiation-Induced Carcinogenesis**
Ann R. Kennedy
University of Pennsylvania School of Medicine

Program Summary

- 2:20 pm **Role of Tissue Responses in Modification of Radiation Effects**
Mary Helen Barcellos-Hoff
Lawrence Berkeley National Laboratory
- 2:50 pm Break
- 3:10 pm **Influence of Low Linear Energy Transfer Radiation Dose and Dose Rate on Radiation Risk: Life-Span Dog Studies**
Antone L. Brooks
Washington State University at Tri-Cities
- 3:40 pm **Variations in Radiation Sensitivity Among Individuals—The Potential Impact on Risk Assessment**
Joel S. Bedford
Colorado State University
- 4:10 pm **Biophysical Modelling and Systems Biology Approaches to Understanding Low-Dose Radiation Effects**
Herwig G. Paretzke
GSF-Institut für Strahlenschutz, Germany
- 4:40 pm **Break**
- Thirty-Second Lauriston S. Taylor Lecture on Radiation Protection and Measurements**
- 5:00 pm **Introduction of the Lecturer**
Michael T. Ryan
- Radiation Standards, Dose/Risk Assessments, Public Interactions, and Yucca Mountain: Thinking Outside the Box**
Dade W. Moeller
Dade Moeller & Associates, Inc.
- 6:00 pm **Reception in Honor of the Lecturer**

Tuesday, April 15

8:10 am **NCRP Annual Business Meeting**

9:10 am **Break**

Human Epidemiology Studies

John D. Boice, Jr., *Session Chair*

9:30 am **Human Epidemiology Studies as a Basis for Current Radiation Risk Estimates:**

Introductory Remarks

John D. Boice, Jr.
International Epidemiology Institute

9:35 am **Low-Dose Radiation Epidemiology Studies: Status and Issues**

Roy E. Shore
Radiation Effects Research Foundation, Japan

10:05 am **Impact of Dosimetry Uncertainties on Dose-Response Analyses**

Ethel S. Gilbert
National Cancer Institute

10:35 am **Break**

10:55 am **Debate on the Topic “Does Scientific Evidence Support a Change from the LNT Model for Low-Dose Radiation Risk Extrapolation?”:**
Moderator’s Introductory Remarks

Eric J. Hall
Columbia University

11:00 am **Affirmative Response**

Dietrich Averbeck
Institut Curie, France

11:15 am **Negative Response**

David J. Brenner
Columbia University

Program Summary

11:30 am	Reply to Dietrich Averbeck David J. Brenner	1:55 pm	U.S. Environmental Protection Agency's Perspective on What it Would Take to Promote or Require a Change in Radiation Protection Regulations Juan Reyes
11:35 am	Reply to David J. Brenner Dietrich Averbeck		
11:40 am	Questions and Discussion	2:05 pm	Questions and Discussion Paul A. Locke, <i>Moderator</i>
12:00 pm	Lunch Low-Dose Radiation Effects, Regulatory Policy and Impacts on the Public Susan D. Wiltshire, <i>Session Chair</i>	2:20 pm	Break
1:00 pm	What Would It Take to Promote or Require a Change in Regulations?: Introductory Remarks Jill A. Lipoti <i>New Jersey Department of Environmental Protection</i>	2:40 pm	Public Perception and Policy: Introductory Remarks Susan D. Wiltshire <i>JK Research Associates</i>
1:05 pm	Low-Dose Effects and Modeling in Public Health Decision Making: Examining the Past, Explaining the Present, and Exploring the Future Paul A. Locke <i>Johns Hopkins Bloomberg School of Public Health</i>	2:45 pm	Beliefs About Radiation: Scientists, the Public, and Public Policy Hank C. Jenkins-Smith <i>University of Oklahoma</i>
1:35 pm	Low-Dose Radiation Effects, Regulatory Policy, and Impact on the Public: U.S. Nuclear Regulatory Commission Perspective Martin J. Virgilio	3:15 pm	Federal Programs to Reimburse the Public for Environmental and Occupational Exposures Paul L. Ziemer <i>Purdue University</i>
1:45 pm	U.S. Department of Energy Perspective: Supporting Research to Inform Regulatory Policy Noelle F. Metting	3:45 pm	How Do We Combine Science and Regulations for Decision Making Following Radiological Accidents and Incidents? John W. Poston, Sr. <i>Texas A&M University</i>
		4:15 pm	Closing Remarks Thomas S. Tenforde <i>President, NCRP</i>
		4:25 pm	Adjourn

Abstracts

Monday, April 14, 2008

Opening Session

8:00 am

Welcome

Thomas S. Tenforde

President, National Council on Radiation Protection and Measurements

8:15 am

Fifth Annual Warren K. Sinclair

Keynote Address

Issues in Quantifying the Effects of Low-Level Radiation

Dudley T. Goodhead

Medical Research Council, United Kingdom



Much is known about health risks of ionizing radiation. Quantification of risks is far more advanced than for many other toxins. Acute tissue effects, and then cancers, became apparent remarkably soon after the discovery of x rays and radioactivity, more than 110 y ago. Experimental systems soon showed that heritable genetic risks were also possible, but these have remained elusive in humans. Studies on the survivors of the atomic bombs in Japan shifted the emphasis back to carcinogenic risk; successive follow-ups have tended to yield increased risk factors and reveal direct risks at successively lower doses. Given the inevitable statistical limitations of epidemiology, direct estimates are unobtainable at the low doses of primary relevance in radiation protection. These low-dose estimates must be obtained by purely mathematical extrapolation or with additional guidance. Commonly, the epidemiological data are fitted by applying functions containing only the simple dose dependencies that are statistically justified. Thus crucial assumptions are introduced, such as linear nonthreshold responses. Others are introduced to extend the risk factors to other exposure scenarios.

These approaches have had considerable success in protecting humans. But do they go far enough? Or, conversely, are they overprotective, thereby distorting the allocation of resources and impeding medical and industrial progress? There is a continuing need for quantification, with improved accuracy and confidence. Epidemiology is the essential starting point, but is fundamentally limited in what it can achieve. Support for the current approaches may be sought from basic studies of the critical molecules, cells, tissues, animals and humans. Historical paradigms of radiation carcinogenesis have arisen from such studies and driven the thinking of those who develop protection policy. Notions of single-hit kinetics at low doses, quadratic responses at higher doses due to interacting events, reduction at low dose rates, and increasing effectiveness with increasing ionization density, all stem from basic studies in simpler biological systems. But how robust are they as key features of radiation carcinogenesis in humans, to guide accurate quantification of risk? Advancing studies in biology should lead the way to improved quantification, including replacement of current paradigms if required, and with scope for

Abstracts

extrapolations based on quantitative modelling of key steps in the carcinogenic process.

DNA damage produced by low-level radiation occurs against an extensive background of ongoing damage from natural processes. But the radiation does have special features, enabling the ubiquitous low-energy electrons to act effectively. Are the guiding messages that have been drawn from animal carcinogenesis studies sufficiently consistent for the purpose? How well justified is the reliance that has been placed on analyses of chromosome aberrations? Over the past 15 y or so, a

variety of novel features have emerged in radiation biology, including induced genomic instability, bystander effects, adaptive responses, thresholds, complex and inter-related DNA repair, and signalling pathways in tissues and fundamental differences in responses between low and moderate doses. Yet, to date, these seem not to have altered basic approaches to radiation protection, nor to quantification of risk in most situations. Is this because the historical approaches are so robust and well founded, or is it because available data on the new phenomena are not sufficiently clear or relevant?

9:15 am

Overview of Goals of the Meeting

Antone L. Brooks

Washington State University at Tri-Cities

9:35 am

Low-Dose Extrapolation of Radiation-Related Health Risks: Status of Human Studies and State of the Art

Charles E. Land

National Cancer Institute

Ionizing radiation is a known and well-quantified human cancer risk factor, based on a remarkably consistent body of information from epidemiological studies of exposed populations. Typical examples of risk estimation include use of Japanese atomic-bomb survivor data to estimate future risk from radiation-related cancer among American patients receiving multiple computed tomography scans, persons affected by radioactive fallout, or persons whose livelihoods involve some radiation exposure, such as x-ray technicians, interventional radiologists, or shipyard workers. Our estimates of radiation-related risk are uncertain, reflecting statistical variation and our imperfect understanding of crucial assumptions that must be made if we are to apply existing epidemiological data to particular situations. Fortunately, that uncertainty is also highly quantifiable, and can be presented concisely and transparently.

Radiation protection is ultimately a political process that involves consent by stakeholders, a diverse group that includes people who might be expected to be risk-averse and concerned with plausible upper limits on risk (how bad could it be?), cost-averse and concerned with lower limits on risk (can you prove there is a nontrivial risk at current dose levels?), or combining both points of view. How radiation-related risk is viewed by individuals and population subgroups also depends very much on perception of related benefit, which might be (for example) medical, economic, altruistic or non-existent.

Discussion will focus on implications of quantification and expression of radiation-related cancer risk and its uncertainty, and will draw heavily on NCRP Commentary No. 14 (*A Guide for Uncertainty Analysis in Dose and Risk Assessments Related to*

Environmental Contamination, 1996), NCRP Report No. 126 (*Uncertainties in Fatal Cancer Risk Estimates Used in Radiation Protection*, 1997), the report of the NCI-CDC Working Group to Revise the

1985 NIH Radioepidemiological Tables (2003), and ICRP Publication 99 (*Low-Dose Extrapolation of Radiation-Related Risk*, 2006).

10:05 am

Break

Molecular, Cellular, Tissue and Animal Radiation Responses of Relevance to Radiation Protection

Gayle E. Woloschak and Amy Kronenberg, *Session Co-Chairs*

10:25 am

Molecular Responses: Introductory Remarks

Amy Kronenberg
Lawrence Berkeley National Laboratory

10:30 am

DNA Damage and Repair as a Factor Contributing to Risk from Radiation

Penny A. Jeggo
University of Sussex, United Kingdom

DNA damage responses encompass pathways of DNA repair and signal transduction processes that serve to effect cell cycle checkpoint arrest and apoptosis. For DNA double strand breaks (DSB), the most biologically significant lesion induced by ionizing radiation, the major DSB rejoining process is DNA nonhomologous end-joining and the most significant signaling pathway is dependent upon the kinase, ataxia telangiectasia and Rad3 related ATM. Mammalian DNA is wrapped within chromatin; regions of DNA that are frequently transcribed lie with euchromatic DNA whilst heterochromatin regions, which are likely not transcribed, are more tightly packaged. This packaging makes DNA difficult to repair and hence the repair of even low levels of DSBs can take place over many hours. The DSB signal transduction pathway regulates a process called cell-cycle checkpoint arrest, which arrests cells at critical places in the cell

cycle, to allow additional time for repair before processes such as replication or mitosis. Whilst DSB repair is important for survival postirradiation and cell-cycle checkpoint arrest is important for the maintenance of genomic stability, it is the cooperation between the two processes that is really critical to avoid genomic instability. Surprisingly, however, recent studies have suggested that the cell-cycle checkpoint that regulates entry into mitosis from G2 is not sensitive to a single DSB but rather allows progression of cells with 10 to 20 DSBs to progress into mitosis. Moreover, it does not appear to be activated by very low doses of radiation inducing less than this number of DSBs. This aspect of the damage response exposes a potential window allowing genomic instability to arise even after low doses of radiation. This will be discussed in the context of evaluating the risk from radiation exposure.

11:00 am

Low-Dose Gene Expression Phenotyping – Molecular Pathways for Radioprotection Against DNA Damage and Chromosomal Abnormalities in Tissues

Andrew J. Wyrobek

Lawrence Berkeley National Laboratory

Our research objectives are to characterize the variations in gene expression phenotypes among radiation-sensitive tissues after exposure to low-dose radiation (<100 mGy), to distinguish between pathways that are broadly conserved among tissues and species versus those that are cell-type unique, and to develop molecular models of predicting susceptibility for persistent genetic damage and risks for tissue-specific cancers from low-dose radiation. Using transcriptional profiling of human lymphoblastoid cells from unrelated individuals, we identified low-dose unique genes that were associated with cellular homeostasis, specific signal transduction pathways, and specific subcellular locations. Some genes showed transcriptional modulation at <10 mGy with flat dose-response curves indicative of nonlinearity in the underlying mechanisms. Comparative bioinformatics analyses identified substantial similarities in gene networks and pathways between irradiated human and mouse tissues, suggesting that there are broadly conserved mechanisms of low-dose radiation response. On the other hand, there was also evidence for low-dose responses that were tissue-specific (e.g., low-dose exposure of brain tissue affected pathways that were associated with memory and other neural functions). Furthermore, low-dose exposures are also known to induce radioadaptation in human cells and rodent tissues, but the underlying molecular mechanisms for radioprotection remain poorly understood. We identified a set of genes

associated with protection for chromosomal aberrations in human lymphoblastoid cells, suggesting that the radioadaptive response in these cells is controlled by a multi-gene switch related to TP53 function. We have also shown that whole-body radiation of mice also induced radio-adaptive protection against DNA damage and chromosomal abnormalities in various tissues, including brain and blood. In summary, there is growing evidence that: (1) the response of cells and tissues to low-dose radiation is molecularly complex with nonlinear components, (2) certain pathways appear to be conserved across tissues and species whether irradiated *in vitro* or *in vivo*, and (3) the genomic damage consequences after low-dose radiation depend on the physiological status of cells at the time of radiation as well as on the details of the exposure regimen. Gene-expression phenotyping promises to increase our understanding of how low dose and low dose-rate exposures modulate the molecular susceptibility of cells within tissue microenvironments, and to identify the molecular pathways that control the radioadaptive response and persistence of genomic damage in tissues. Understanding the molecular basis of cellular and tissue responses to low-dose radiation has important implications for assessing long-term tissue injury and cancer risks from environmental exposures to ionizing radiation and from the rapidly increasing usage of low-dose radiation for medical diagnostics.

11:30 am

Radiation Protection and Nontargeted Cellular and Tissue Responses at Low Radiation Doses

William F. Morgan

University of Maryland School of Medicine

Nontargeted responses to ionizing radiation are those cellular and tissue effects observed in cells that were not subject to energy deposition events induced by radiation. These responses can occur in cells that were the progeny of an irradiated cell (radiation-induced genomic instability), and/or they can occur in the nonirradiated neighbors of an irradiated cell (bystander effects) after receiving signals from irradiated cells. Both genomic instability and bystander effects describe responses in nonirradiated, or nontargeted cells and tissues, and the phenotype of these responses is similar to those observed in irradiated (targeted) cells. These responses include changes in gene and protein expression, induction of mutations, chromosomal rearrangements, micronuclei, transformation, and/or apoptosis. Nontargeted effects can be observed at very low radiation doses where the shape of the dose response curve is the matter of considerable

debate. Furthermore, nontargeted effects indicate that responses can be observed outside the radiation field and therefore suggest that the risk for potential radiation effects may well be greater than the volume actually irradiated.

In this presentation the evidence for nontargeted effects will be presented and the experimental systems used to characterize these responses will be described. Subsequent discussion will then debate whether irradiated cells respond differently than naïve nontargeted bystander cells and whether nontargeted effects are beneficial or detrimental to the tissue or organism. The final part of the presentation will focus on whether nontargeted effects are limited to the specific organ irradiated and thus are accounted for under current risk policies, or whether they might be insidious throughout the organism and thus significantly impact current radiation protection standards.

12:00 pm

Lunch

1:15 pm

Low-Dose Radiation Responses in Cells, Tissues and Animals: Introductory Remarks

Gayle E. Woloschak

Northwestern University

1:20 pm

Chromosome Aberrations as a Function of Dose, Dose Rate, and Linear Energy Transfer: Implications for Radiation Risk

Michael N. Cornforth

University of Texas Medical Branch

Most, if not all, important radiobiological phenomena were either discovered, or subsequently verified, using chromosome damage as the experimental endpoint. These include, but are not limited to:

- ionization density (linear energy transfer) and its relationship to relative biological effectiveness;
- aberrations as a principle cause of radiation induced cell killing; and

Abstracts

- the basic shape of dose response relationships following changes in dose and dose rate.

Chromosome aberrations are an exquisitely sensitive indicator of radiation damage, and provide quantitative information on a cell-by-cell basis. For these reasons, cytogenetic data has long been favored by modelers that seek to define through extrapolation, on the basis of biophysical and molecular principles the shape of the dose response following very low doses.

In cases where physical dosimetry is not a feasible option, chromosome damage has become the “gold standard” for use in dose reconstructions. It could be argued

that they hold special status among other biodosimetric approaches, because the end result of processes governing their formation are known, in several instances, to be the cause of certain cancers. That is to say, chromosome aberrations are a sensitive biodosimeter of radiation damage that can be viewed as a surrogate for carcinogenic potential.

Here we discuss briefly the contribution of radiation cytogenetics in establishing and explaining various radiobiological phenomena in connection with radiation risk, particularly that associated with very low doses.

1:50 pm

Factors that Modify Radiation-Induced Carcinogenesis

Ann R. Kennedy

University of Pennsylvania School of Medicine

It is known that numerous factors influence the yields of radiation-induced malignancies in animals; these factors include the specific characteristics of the radiation (radiation type and dose, dose rate, dose fractionation, dose distribution, etc.) as well as many factors that are not specific to the radiation exposure, such as animal genetic characteristics, the envi-

ronment of the animal, dietary factors, and whether specific modifying factors for radiation carcinogenesis have been utilized in the studies. This overview will focus on the modifying factors for radiation carcinogenesis, in both *in vivo* and *in vitro* systems, and will include a discussion of the factors which can increase or decrease radiation carcinogenesis.

2:20 pm

Role of Tissue Responses in Modification of Radiation Effects

Mary Helen Barcellos-Hoff

Lawrence Berkeley National Laboratory

The cell biology of irradiated tissues reveals a coordinated multicellular damage response program in which individual cell contributions are primarily directed towards suppression of carcinogenesis and reestablishment of homeostasis. Previous studies characterized the composition of irradiated mouse tissues, identified transforming growth factor $\beta 1$ (TGF β) as a key growth factor induced by radiation, and developed novel radiation models in both mouse and cultured human cells.

The ability of human mammary epithelial cells to undergo tissue-specific morphogenesis in cell culture shows that radiation disrupts epithelial cell interactions with the microenvironment. A persistently dysfunctional cell-cell and cell-extracellular matrix interaction of irradiated epithelial cells is induced in irradiated tissues. This heritable phenotype is consistent with epithelial to mesenchymal transition. The underlying mechanism of this phenotypic switch is radiation-induced extracellularly regulated kinase activation that is sustained in

the presence of TGF β . As a result, radiation exposure of individual cells leads to the generation of daughter cells with a persistently altered phenotype accompanied by increased invasion and motility that could contribute to malignant progression.

The contribution of irradiated phenotypes in the mouse mammary radiation chimera has been tested. The mammary chimera model takes advantage of the fact that the mammary epithelium can be removed from prepubertal mammary glands and the parenchyma-free stroma can then serve as the recipient of transplanted mammary tissue. To examine the dose dependence of host irradiation on mammary cancer progression, *p53* null mouse mammary fragments were transplanted to wildtype mice irradiated whole body with 100, 500 and 1,000 mGy. Tumor frequency significantly increased and median latency for cancer incidence was decreased in irradiated hosts. To assess the contribution of TGF β , *Tgf β 1* heterozygote hosts were subjected to a similar protocol. The effect of irradiation on *p53* null tumor frequency was absent in *Tgf β 1* heterozygote hosts. Thus, single acute radiation

exposure can act through the host to drive breast cancer progression, which is in large part mediated by TGF β abundance. These data show that high-dose radiation disrupts the interactions of multiple cell types in normal tissues that effectively suppress neoplastic potential.

Together, these studies support the global hypothesis that multicellular responses and extracellular signaling following radiation exposure are integral, rather than secondary in evaluating radiation risks. A systems biology model is needed that emphasizes the irradiated tissue/organ/organism as a system rather than a collection of noninteracting or minimally interacting cells. A key property of a system is that some phenomena emerge as a property of the system rather than the parts. Cancer can thus be considered as an emergent phenomenon of a perturbed system. Given the current research goal to determine the consequences of high versus low radiation exposures, then broadening the scope of radiation studies to include systems biology concepts should benefit risk modeling of radiation carcinogenesis.

2:50 pm

Break

3:10 pm

Influence of Low Linear Energy Transfer Radiation Dose and Dose Rate on Radiation Risk: Life-Span Dog Studies

Antone L. Brooks

Washington State University at Tri-Cities

There is very little human data on the risk from high doses of low linear energy transfer radiation delivered at low dose rates. To help understand this risk, extensive studies were conducted on Beagle dogs exposed to ionizing radiation both from external whole-body ^{60}Co gamma rays and internally deposited beta-gamma emitting radioactive material. This presentation will evaluate this very large data set. The internal emitter studies included

different routes of exposure (ingestion, injection, inhalation), a range of radionuclides with different half-lives (^{90}Y , ^{91}Y , ^{144}Ce , ^{90}Sr , ^{137}Cs) and target organs (lung, liver, bone, whole body). The isotopes were also delivered in different chemical and physical forms which influenced their retention, deposition and distribution. The data defined a high dose rate and total dose for each target organ above which acute deaths occur. Most of the animals

Abstracts

that survived these early acute effects lived for long periods of time and were at increased risk for cancer. When the total data for the internally deposited radioactive material was evaluated at doses to the target organ <10 Gy there was no detectable increase in the cancer frequency. This presentation will compare the dose-response relationships from internally-deposited radioactive materials to that from chronic and acute whole-body exposure and help put dose, dose rate, and dose distribution into a useful framework

for risk estimates. For the same total dose, both cancer frequency and early deaths were markedly decreased when the radiation was delivered at a low dose rate. Nonuniform dose distribution also decreased the effectiveness of the radiation in producing cancers. Such data provide a strong scientific base for predicting the outcome of low dose-rate exposures to large total doses, estimating risk from these exposures, and defining a realistic dose-rate effectiveness factor.

3:40 pm

Variations in Radiation Sensitivity Among Individuals: The Potential Impact on Risk Assessment

Joel S. Bedford
Colorado State University

The possible impact of genetic variation in susceptibility to radiation carcinogenesis has been considered and discussed for many years, especially following the discovery some 40 y ago that certain heritable defects, such as that associated with the autosomal recessive disorder, ataxia telangiectasia, could lead to extreme hypersensitivity to effects of ionizing radiation exposure. For several reasons, including the very low incidence, the limited number of genetic disorders known with hyper-radiosensitive phenotypes, and even the projected numbers of individuals who may be of intermediate radiosensitivities due to heterozygosity for such known genes was sufficiently low that their proportion in the population was not

expected to significantly influence risk estimates. Reports of several studies over the past two decades have increasingly suggested that there may be a much higher proportion of individuals whose cells indicate hypersensitivity phenotypes than previously expected. The levels of hypersensitivity do not reach the extremes seen for cells from *ATM* $-/-$ individuals, or from well known mutants in rodent cell systems, but to the extent that the cellular radiosensitivity phenotypes reflect the proportions of individuals who may be similarly hypersensitive for carcinogenesis this would clearly warrant reevaluation of the possible implications for radiation protection. The data suggesting this conclusion will be presented.

4:10 pm

Biophysical Modeling and Systems Biology Approaches to Understanding Low-Dose Radiation Effects

Herwig G. Paretzke
GSF-Institut für Strahlenschutz, Germany

Radiation affects all three aspects of health as a status of complete physical, mental and social well-being. This is particularly true for real and perceived low-dose radiation effects on human health.

Mathematical quantifications of likelihoods of such health effects in individuals is still not possible. This will never be possible based on epidemiological investigations alone because of statistical reasons

Monday, April 14

and lack of homogeneity in larger populations. The only promising approach is by close cooperation of theorists and experimentalists from various relevant disciplines, and by carefully selected experiments based on well defined, quantitative working hypotheses for important steps of maintaining regular homeostasis and for disturbances (e.g., by irradiation).

Studying such processes at different levels of a complex living system with adaptive responses of its various regulation networks poses high demands on life scientists as well as on mathematicians. This contribution will outline some present approaches to draw general, quantitative conclusions from many types of experimental observations.

4:40 pm

Break

Thirty-Second Lauriston S. Taylor Lecture on Radiation Protection and Measurements

5:00 pm

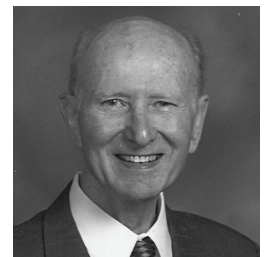
Introduction of the Lecturer

Michael T. Ryan

Radiation Standards, Dose/Risk Assessments, Public Interactions, and Yucca Mountain: Thinking Outside the Box

Dade W. Moeller

Dade Moeller & Associates, Inc.



The information in this presentation is based on studies performed during the past 5 y on various facets of the proposed Yucca Mountain high-level radioactive waste repository. The initial topic pertains to the standards promulgated for this facility by the U.S. Environmental Protection Agency under the restrictions and limitations imposed by the U.S. Congress and the Circuit Courts. This documents that the standards are neither integrated, nor consistent, one example being that the estimated release of a specific radionuclide can comply with one portion of the standards and not another. The second topic includes a summary of the evaluations of the associated dosimetry. These findings revealed that five of the eight

so-called primary radionuclides that will be present in the waste, are of little or no health concern. Equally important is that it was determined that, even if the dose rates could be accurately projected hundreds or thousands of years into the future, it will not be possible to estimate their associated health risks. This, in essence, rules out the application of a risk-based approach to the long-term assessment of the performance of the repository. The third topic pertains to the anticipated events accompanying the processing of the license application. This will involve hearings before the Senate, as well as multiple public hearings in major population centers throughout the United States. If the U.S. Department of Energy



Abstracts

(DOE) is to effectively and successfully complete these hearings, it will be necessary to prepare a set of statements that can be used to respond to the full range of questions that may be raised. One example of such statements would be a review of, and rebuttal to, the multiple myths about radiation that are held by members of the public. In a closing segment, the restrictions in the standards that prohibit DOE from projecting or applying estimates of "changes in ... human biology, or increases or decreases in human knowledge or technology" are evaluated in the light of the fact that a poll of cancer experts showed that the vast majority projected that methods for the prevention and/or cure of most of the cancers affecting humankind today will become a reality within the next 50 to 100 y. Supporting these projections are the development and current application of a vaccine for cervical cancer, and the report of a federal expert cancer group that the annual rate of deaths from colorectal cancer, the second highest contributor to such deaths in the United States, is being reduced at an

annual rate of almost 5 % for men, and 4.5 % for women. It was in anticipation of such developments that the National Council on Radiation Protection and Measurements stated that if "an increased proportion of the adverse health effects of radiation prove to be either preventable or curable by advances in medical science, the estimates of the long-term detriments may need to be revised as the consequences (risks) of doses to future populations ..." are reduced. This confirms the fact that it is time that the radiation protection profession and the regulatory agencies that promulgate the applicable regulations begin "thinking outside the box," as contrasted with adherence to a requirement that DOE must estimate changes "related to the geology, hydrology, and climate" that "could affect the Yucca Mountain disposal system over the next 10,000 years." The latter approach appears neither reasonable nor appropriate in light of the fact that a decrease in the risk of fatal cancer as a disease that threatens the U.S. population will occur within the next 50 to 100 y.

6:00 pm

Reception in Honor of the Lecturer

Sponsored by
LANDAUER[®]



Tuesday, April 15

Tuesday, April 15

8:10 am **NCRP Annual Business Meeting**

9:10 am **Break**

Human Epidemiology Studies

John D. Boice, Jr., *Session Chair*

9:30 am **Human Epidemiology Studies as a Basis for Current Radiation Risk Estimates: Introductory Remarks**

John D. Boice, Jr.
International Epidemiology Institute

9:35 am **Low-Dose Radiation Epidemiology Studies: Status and Issues**

Roy E. Shore
Radiation Effects Research Foundation, Japan

Although the Japanese atomic-bomb study and radiotherapy studies have clearly documented cancer risks from high-dose radiation exposures, the National Council on Radiation Protection and Measurements and other radiation risk assessment groups have long recognized that protracted or low exposures to low linear energy transfer radiations are key radiation protection concerns, because these are far more common than high-exposure scenarios. Epidemiologic studies of human populations with low dose or low dose-rate exposures are one approach to addressing those concerns. A number of large studies of radiation workers (Chernobyl cleanup workers, Mayak workers, United States and Chinese radiologic technologists, and the 15-country worker study) or those exposed to environmental radiation at moderate to low levels (residents near Techa River, Semipalatinsk, Chernobyl, or nuclear facilities) have been conducted. A variety of studies of medical radiation exposures (multiple fluoroscopy, diagnostic ^{131}I , scatter radiation doses from radiotherapy, etc.)

also are of interest. Key results from these studies will be summarized and compared with risk estimates from the Japanese atomic-bomb study.

Ideally, one would like the low dose and low dose-rate studies to guide radiation risk estimation regarding the shape of the dose-response curve, dose and dose-rate effectiveness factor, and risk at low doses. However, the degree to which low-dose studies can do so is subject to various limitations, especially those pertaining to dosimetric uncertainties and limited statistical power.

The identification of individuals who are particularly susceptible to radiation cancer induction is of high interest in terms of occupational and medical radiation protection. Questions also have been raised as to how susceptible individuals in the population may influence the aggregate risk at low doses. Issues pertaining to radiation-related cancer susceptibility studies will be outlined, and several examples of such studies will be discussed.

10:05 am

Impact of Dosimetry Uncertainties on Dose-Response Analyses

Ethel S. Gilbert

National Cancer Institute

Radiation dose estimates used in epidemiological studies are subject to many sources of uncertainty, and the error structure may be a complicated mixture of different types of error. Increasingly, efforts are being made to evaluate dosimetry uncertainties and to take account of them in statistical analyses. The impact of these uncertainties on dose-response analyses depends on the magnitude and type of error as discussed below.

Errors that are independent from subject to subject (random errors) reduce statistical power for detecting a dose-response relationship and increase uncertainties in estimated risk coefficients. However, statistical tests based on uncertain dose estimates are generally valid even without using special statistical methods that account for dose uncertainties. Without improving dose estimates, it is not possible to avoid this loss of power.

Other effects of random errors depend on whether the errors are “classical” or “Berkson.” A measurement error is classical if the error is independent of the true dose, that is, the measured doses vary about the true doses. Classical error can be thought of as error that arises from an imprecise measuring device such as a film badge dosimeter. If data are analyzed without attention to dose uncertainties, the presence of classical error attenuates the dose-response toward the null and may distort the shape of the dose-response.

A measurement error is Berkson if the error is independent of the observed dose. Berkson error occurs when a single dose is used to represent a group so that the true doses of individuals vary about the assigned group dose. An

example is the application of a single factor to convert recorded doses to organ doses for nuclear workers in a given facility and time period even though the correct factor varies among the workers to whom it is applied. In contrast to classical error, the presence of Berkson error does not result in bias in linear risk coefficients. However, non-Berkson error may also be present if the assigned group doses differ from true mean doses for the groups to whom they are assigned.

Uncertainties in quantities that are common to some or all subjects are “shared” uncertainties. For example, in the Japanese atomic-bomb study, uncertainty in the yields of the bombs is a shared uncertainty since it affects doses of all subjects in a given city in a similar manner. Such uncertainties increase the possibility of bias, and accounting for this possibility increases the length of confidence intervals.

The impact of dose uncertainty on the direct evaluation of risks at low doses and dose rates is, in general, as noted above. First, if a significant dose-response relationship is found in a low-dose study, it is unlikely to result from dose uncertainties. Second, the low power inherent in studying small risks may be further reduced by random dosimetry uncertainties. Thus, dosimetry errors are much more likely to mask a true effect than to create a spurious one. In addition, classical errors and shared dosimetry uncertainties increase the potential for bias in estimated risk coefficients, but this potential may already be large due to the extreme vulnerability to confounding in studies involving very small relative risk.

10:35 am

Break

10:55 am

Debate on the Topic “Does Scientific Evidence Support a Change from the LNT Model for Low-Dose Radiation Risk Extrapolation?”:

Moderator’s Introductory Remarks

Eric J. Hall

Columbia University

11:00 am

Affirmative Response

Dietrich Averbeck

Institut Curie, France

Low dose effects of ionizing radiation are usually less evident than high dose effects on living matter. The latter effects are more easily quantifiable and experimentally accessible. Epidemiological studies do not easily detect biological risks at low doses and low dose rates. Thus, knowledge of fundamental mechanisms involved are essential to understand and assess low dose radiation risks. The linear nonthreshold (LNT) model is based on the notion that the physical energy deposition of ionizing radiation lets the carcinogenic risk increase linearly with increasing dose (*i.e.*, the carcinogenic effectiveness remains constant irrespective of dose and dose rate). The model has been taken as a useful basis for regulatory measures in radiation protection. However, recent developments and new findings in radiation and molecular biology strongly challenge the LNT concept. Indeed, as pointed out by the report of the French Academies recent biological results (also quoted in BEIR VII and ICRP reports) are in contradiction with the use of the LNT model for evaluating radiation risks at low and very low-dose exposure levels. In fact, there is evidence against its validity. Several lines of evidence demonstrate that living cells and tissues react differently (quantitatively and qualitatively) to radiation insults from high and low dose exposures. At the cellular level, some protection mechanisms are especially active at low doses. These include protection against reactive oxygen

species (induced by ionizing radiation), cellular signaling activation of DNA repair, and elimination of damaged cells by cell death. In line with this, at very low doses (1 mGy) and dose rates (1.5 mGy min⁻¹) repair of DNA double strand breaks has been shown to be absent due to absence of proper signaling, and damaged cells disappear by cell death. Following the somatic cell mutation theory of carcinogenesis this implies that at those very low exposures there is no initiation of cancer cells. At slightly higher doses and dose rates, DNA repair is fully activated, and at doses >200 mGy repair is increased, probably in order to maintain sufficient cell viability and tissue functions. This repair can be in part error-prone giving rise to chromosomal damage and mutations. If cellular damage is too high, some cells will undergo apoptosis or even necrosis. In accord with this, very low doses induce or repress different types of genes than higher doses. Data from transcriptome and proteome analysis (phosphoproteome) demonstrate that different gene and protein families are induced (or repressed) or activated (or not) at low (20 mGy) and high doses (gray). Thus, different signaling and processing after ionizing irradiation determine the final outcome in terms of mutagenesis and carcinogenesis, and it is illegitimate to extrapolate from high to low doses.

Also, we observed that enzymes involved in DNA damage signaling at low dose-rate

Abstracts

exposures differ from those operating at high dose rate. Cell death, mutation induction, induction of cell transformation *in vitro* and carcinogenicity have been demonstrated to be lower at low than at high dose rate, probably due to more efficient DNA repair at low dose-rate exposures.

In addition, phenomena like low dose hypersensitivity and radioadaptive responses confirm that cellular responses are highly dependent on initial exposure levels. The activation of the nick sensor, poly (ADP-ribosyl) polymerase and induction of apoptosis depend on the dose levels. Bystander effects are known to induce nonlinear responses at low levels due to intercellular communication and signaling, and dose thresholds and protective effects have been reported. Although a low-dose bystander effect giving rise to enhanced mutagenesis has been observed with alpha rays on human cells *in vitro*, epidemiological data on dial painters contaminated with radium or patients contaminated with thorotrast revealed no excess of cancer cases at cumulative doses <1 Gy from alpha ray emitters. Recent work shows that low-dose exposures (alpha and gamma rays) of normal cells, co-cultured with unirradiated preneoplastic cells, exert signaling (including factors such as TGF β) from irradiated normal cells which effectively induce apoptosis in the unirradiated preneoplastic cells (threshold at >2 mGy for gamma rays, and

0.29 mGy for alpha rays). This eliminates premalignant cells at low doses but not at high doses. Effective immunosurveillance is also likely to play an important role in protecting against cancer development after low doses. Indeed, high doses can affect immunological defenses.

Altogether, the above arguments are in favor of lower than expected biological effects (threshold responses) at low doses and low dose rates. Thus, the hitherto plausible biophysical rationale for using LNT for extrapolation from high doses to low doses is overcome by new biological facts concerning low-dose exposures. We are facing higher complexity of the biological response at low dose and low dose rates. This is also true for epidemiological data where the number of possible confounding factors involved appears to be greater at low than at high radiation doses. Thus, risk evaluations at low exposure levels have to take more parameters into account and ask for a different type of modeling. Obviously, the LNT model cannot fulfill this role.

Up to now, radioprotection regulatory measures were conceived as being regularly adjustable to increasing scientific knowledge. Thus, it is scientifically sound, and wise from the practical and economical point of view, to decrease existing uncertainties for low-dose risk evaluation by taking into account the new findings.

11:15 am

Negative Response

David J. Brenner
Columbia University

There is convincing epidemiological evidence that doses of ionizing radiation above about a few tens of milligray cause a small but significant increase in cancer risk. At lower doses, however, even the largest epidemiological studies have insufficient power, and so we have to rely

on "expert opinion" guided, where appropriate, by the best available biology.

Two expert reports have been published recently which give diametrically opposing expert opinions. The BEIR-VII report, from the U.S. National Academy of Sciences, concludes that, at low doses, as the dose

is lowered, the cancer risk simply decreases proportionately—a “linear no threshold” model down to arbitrarily low doses. By contrast, the French Academy of Sciences (FAS) suggested that, at very low doses, the risk per unit dose for ionizing radiation-induced cancer is lower than that established at higher doses, and may well be effectively zero, or even negative.

FAS arguments essentially revolve around the claim that different biological processes dominate radiation damage responses at very low doses (below ~10 mGy), compared with higher doses. For example, the claim is made by FAS that, at these very low doses, essentially all radiation-damaged cells will be eliminated through apoptosis or other mechanisms, while at somewhat higher doses, radiation damage and subsequent misrepair can ultimately result in cancer. It will be argued that (1) there is no plausible evidence for different damage response pathways at very low doses, and (2) even if there were such evidence, which would

by necessity come from *in vitro* studies, we would not be able to predict the consequences in terms of low dose cancer risks in humans.

There is no doubt that the linear (non-threshold) approach for extrapolating risks to low doses (which has been adopted by most national and international organizations) can and should be critically examined. The arguments for a linear nonthreshold model at very low doses are plausible, but rely on assumptions about single cells acting autonomously, which are unlikely to be completely correct. However, at this time we don't know if deviations from the predictions of this linear approach will be large or small, nor even whether they will increase or decrease low-dose cancer risk estimates. We are only just beginning to scratch the surface of our understanding of the impact of intercellular interactions on very low-dose cancer risks, so it is more than premature to be advocating changes in policy or practice.

- | | |
|----------|---|
| 11:30 am | Reply to Dietrich Averbeck
David J. Brenner |
| 11:35 am | Reply to David J. Brenner
Dietrich Averbeck |
| 11:40 am | Questions and Discussion |
| 12:00 pm | Lunch |

Abstracts

Low-Dose Radiation Effects, Regulatory Policy and Impacts on the Public

Susan D. Wiltshire, *Session Chair*

1:00 pm

What Would It Take to Promote or Require a Change in Regulations?: Introductory Remarks

Jill A. Lipoti

New Jersey Department of Environmental Protection

1:05 pm

Low-Dose Effects and Modeling in Public Health Decision Making: Examining the Past, Explaining the Present, and Exploring the Future

Paul A. Locke

Johns Hopkins Bloomberg School of Public Health

The majority of our public health and environmental protection laws, and the federal agencies that administer them, are less than a century old. Public health policy and regulatory decision making at these agencies and in Congress has been transformed substantially, especially during the last 50 y. Scientific methods and understanding about biological processes has evolved during this same time period, particularly in the area of low-dose radiation effects. Policies and practice at federal agencies have sought to keep up with these advancements and Congress has passed laws to respond to this shifting scientific landscape.

During this same period, the scope and character of radiation exposures to the public has changed and public perception about radiation risk has evolved. In setting public policies and promulgating regulations, scientific information about low-dose effects is one of several factors that decision makers weigh. This presentation will examine how issues associated with low-dose radiation exposure were addressed in the past and how they are treated in policy making today. It will also explore the emerging public health protection and policy challenges that are likely to arise as our scientific knowledge expands.

1:35 pm

Low-Dose Radiation Effects, Regulatory Policy, and Impact on the Public: U.S. Nuclear Regulatory Commission Perspective

Martin J. Virgilio

The system of radiological protection implemented by the U.S. Nuclear Regulatory Commission (NRC) aims primarily to provide adequate protection of public health and safety, and to protect the environment. Its health objectives are relatively straightforward: to manage and control exposures to ionizing radiation, so that deterministic effects are prevented, and the risk of stochastic effects is reduced to the extent reasonably achievable.

Analysis of literature reviews by the National Academy of Sciences and the United Nations Scientific Committee on the Effects of Atomic Radiation and the 2007 radiation protection recommendations published by the International Commission on Radiological Protection do not suggest that any significant change to our system of regulatory protection is warranted. However, several issues have been raised that may prompt the NRC to reexamine its radiation protection

standards. These issues include: potential gender and age differences in radiation sensitivity, evidence suggesting that the threshold for cataracts formation may be less than several gray, the ability to identify genetic markers in people who may be abnormally sensitive to radiation exposure, and the possible existence of a real or practical threshold in radiation dose response.

The current system of radiological protection is considered to be adequately protective of both sexes and all ages, especially in view of the considerable uncertainty regarding the induction of adverse biological effects following very low radiation exposures (<10 mSv). Use of the linear nonthreshold (LNT) model is considered to be the best practical approach to managing risk from radiation exposure and remains a prudent basis for radiological protection at low doses and low dose rates. However, additional information is needed, particularly as it pertains to deoxyribonucleic acid (DNA) damage repair, the identification and characterization of radiation sensitive populations, obtaining evidence that supports or refutes the LNT assumption at low dose and low dose-rate exposures, and data to

support the beneficial, or adverse, effects of low-dose radiation exposure.

NRC will continue to monitor basic research programs such as the U.S. Department of Energy's low-dose radiation research program and the European Commission's radiosensitivity and cancer susceptibility research program, and will work with our stakeholders to ensure that our regulations are effective, efficient and realistic, and based on sound scientific information. NRC endorses radiological protection recommendations that (1) provide tangible improvements in the adequate protection of public health and safety and (2) can be implemented by practitioners and regulatory authorities in a practical, timely, and cost effective manner. NRC will continue to review the scientific literature, encourage the scientific community to develop new techniques for better elucidating the biological effects attributable to very low radiation doses, and will work with other federal agencies to develop documents that relate such effects to the needs of radiological protection. NRC supports the development of realistic models that best predict stochastic health effects without incorporating excess conservatism into prediction models.

1:45 pm

U.S. Department of Energy Perspective: Supporting Research to Inform Regulatory Policy

Noelle F. Metting

The U.S. Department of Energy (DOE) is responsible for regulating and managing both the use of radioactive material and the exposure to radiation by its contractors and operations. DOE is committed to ensuring that radiation exposures to its workers and the public and releases of radioactivity to the environment are maintained below regulatory limits, and takes deliberate efforts to further reduce dose where practicable. To meet this objective, DOE establishes and maintains a system

of regulatory policy and guidance reflective of national and international radiation protection standards and recommendations. The incorporation of these recommendations is consistent with federal policies established through interagency coordination that include all of the agencies having radiation protection responsibilities. Coordination is accomplished through such groups as the Interagency Steering Committee on Radiation Standards.

Abstracts

DOE is the primary agency supporting low dose radiation effects research. DOE's Low Dose Radiation Research Program is supporting research to determine health risks from exposures to low levels of radiation. The new scientific information generated by this research is critical input for regulatory agencies who seek to adequately and appropriately protect people from radiation while making the most effective use of our country's national resources.

The Low Dose Program has emphasized research on a number of critical biological phenomena induced by radiation exposure, including adaptive responses, bystander effects, and genomic instability. The research is focusing greater attention on use of more normal tissue systems, moving away from use of artificially isolated cell culture systems and/or tumor cell lines. DOE also partners with the National Aeronautics and Space Administration to fund some of these research projects. To date, the Program has

resulted in publication of over 480 peer-reviewed papers. Future research will be directed towards developing models that incorporate both biological and epidemiological data.

Radiation protection standards are viewed by some as based on overly conservative assumptions that may exaggerate health risks, while others hold equally strong views that the standards should not be changed or may not be conservative enough. Results of this research may help resolve some of the differences between these strongly held views. While our understanding of the biological effects of and responses to low doses of ionizing radiation has increased dramatically as a result of this research program, translating this information into radiation risk models and radiation protection standards remains a significant challenge. This presentation will give a brief review of the status of current low-dose research and our thoughts on how it could impact future regulatory policy.

1:55 pm

U.S. Environmental Protection Agency's Perspective on What it Would Take to Promote or Require a Change in Radiation Protection Regulations

Juan Reyes

The U.S. Environmental Protection Agency (EPA) is committed to using the best available science when writing regulations and establishing policy. As science has moved forward, so have EPA's regulations. Often this means that the regulations we issue today use better science than was available when we issued older regulations. A decision to go back and update a regulation is often determined by whether the old regulation is still adequately protective or not. The rulemaking process, including seemingly minor updates to rules, can be lengthy and costly. When allocating resources to projects during annual budget planning, a decision to update a still adequate regulation may be deferred in favor of more

immediate priorities. That is why EPA has regulations in place dating back to the early 1970s when the International Commission on Radiological Protection's (ICRP) Publication 2 was used to calculate maximum permissible body burdens and critical organ doses. Compared against the newer science, it is often easy to demonstrate that the regulations based on the older dosimetry methods are still protective. Therefore, the first answer to the question posed by this session is that EPA would require a change in a regulation when it can be demonstrated that it is no longer protective of public health and the environment.

A second reason for bringing a regulation in line with current science would be when

doing so would bring substantial regulatory relief, including cost savings, while still maintaining an optimal level of protection for the public and the affected workforce. It is this second reason, the relaxing of overly burdensome regulations, which most people will think of when asking the above question. Many critics of current radiation protection regulations believe that the linear nonthreshold model, which serves as the basis for many standards, is itself overly burdensome and unnecessarily conservative. It is therefore worth considering how EPA would react to scientific evidence of a dose threshold for radiogenic cancer.

First, there would be a need to fully examine the validity and implications of this

finding. Regulatory changes would not likely come fast. If the research was sufficiently compelling, EPA would look to the major radiation advisory bodies for advice. Among these organizations are the National Council on Radiation Protection and Measurements, the National Academy of Sciences, the United Nations Scientific Committee on the Effects of Atomic Radiation, and ICRP. Scientific consensus among these organizations would likely lead EPA to reconsider its radiation protection standards. However, implementing a set of standards based on a threshold, which may vary across members of the population, would pose challenging practical and philosophical problems.

2:05 pm

Questions and Discussion

Paul A. Locke, *Moderator*

2:20 pm

Break

2:40 pm

Public Perception and Policy: Introductory Remarks

Susan D. Wiltshire

JK Research Associates

2:45 pm

Beliefs About Radiation: Scientists, the Public, and Public Policy

Hank C. Jenkins-Smith

University of Oklahoma

Human behavioral responses to potential hazards are mediated by the beliefs people hold about those hazards. This holds whether the “behavior” under consideration is the provision of advice about the hazard, statements of support for policies that address the hazard, or personal behaviors in response to the hazard. This paper focuses on beliefs about radiation and the implications of those beliefs for views about radiation protection by both scientists and members of the U.S. public. Data are used from a large sample of scientists, collected in 2002, and a series of

surveys of the U.S. public collected between 2005 and 2007. Among scientists, the paper focuses on how beliefs about radiation are related to policy prescriptions for radiation protection. Among members of the lay public the focus shifts to the relationship between beliefs about radiation risks and policy preferences for nuclear energy and nuclear waste policy options. The importance of the differences and similarities in the patterns of beliefs of scientists and the lay public are discussed.

Abstracts

3:15 pm

Federal Programs to Reimburse the Public for Environmental and Occupational Exposures

Paul L. Ziemer
Purdue University

Since the mid-1980s there has been growing public concern about possible health effects associated with radiation exposures of veterans and atomic weapons workers. These concerns have led to a series of Congressional actions that have resulted in legislation creating four compensation programs that are intended to compensate individuals whose radiation exposures may be considered a causative agent for specified health effects.

The Veterans Dioxin and Radiation Exposure Compensation Act of 1984 is administered by the U.S. Department of Veterans Affairs (VA) and is directed to veterans exposed while participating in atmospheric nuclear testing or in the occupation of Hiroshima and Nagasaki. The Defense Threat Reduction Agency (DTRA) determines the participation and dose status for some 1,000,000 potential claimants. Eligibility for compensation is based on verification that the individual was in a specified participant group, has medical proof of a qualifying disease, and has a dose estimate for which the probability of causation shows that the disease was "at least as likely as not" caused by the radiation. The program does not provide lump-sum awards, but rather makes use of a somewhat complex award formula.

The Radiation-Exposed Veterans Compensation Act of 1988 is also administered by VA. This program grew out of dissatisfaction of veterans and members of the public on the dose reconstruction processes and payout rates for the 1984 program. In this program, the claimant need only show proof of being in the specified participant group and medical proof of having the eligible disease. Compensation for the 400,000 potential claimants is also based on a complex awards formula.

The Radiation Exposure Compensation Act of 1990 grew out of political pressure by nuclear test-site worker advocates, and civilians who lived downwind from atmospheric test locations. The program is administered by the U.S. Department of Justice, with support from DTRA. Compensation for the 50,000 potential claimants is based on proof that the claimant falls into a defined participant group and medical proof that the claimant has a qualifying disease. Lump-sum compensation is provided for successful claimants in the amount of \$75,000 for on-site atmospheric test participants, \$50,000 for downwinders, and \$100,000 for uranium workers.

The Energy Employees Occupational Illness Compensation Act of 2000 provides for \$150,000 in lump-sum compensation to workers who contracted certain diseases as a result of exposure to beryllium, silica, or radiation while working for the U.S. Department of Energy (DOE), its contractors, or subcontractors in the nuclear weapons industry. The program is administered by the U.S. Department of Labor with support of the U.S. Department of Health and Human Services and DOE. Eligibility is determined by proof that the claimant worked at one of the specified weapons-related sites during an eligible time period, and proof of an eligible disease. Compensation is provided for claimants in cases where the reconstructed dose is shown to result in a probability of causation of 50 % or greater at the 99 % credibility level. In cases where claimants' doses cannot be reconstructed "with sufficient accuracy," the legislation provides a process whereby such individuals may become part of a "special exposure cohort" for which dose reconstruction is not required.

3:45 pm

How Do We Combine Science and Regulations for Decision Making Following Radiological Accidents and Incidents?

John W. Poston, Sr.
Texas A&M University

Approaches to safety regulations—particularly radiation safety regulations—must be founded on the very best science possible. However, radiation safety regulations always lag behind the science for a number of reasons. First, the normal scientific process of peer-review, debate, and confirmation must ensure that the conclusions are indeed correct, the implications of the research are fully understood, and a consensus has been established. Finally, in the United States, there is a well-established, all-inclusive political process that leads to changes in radiation safety regulations. This process can take a very long time, as was demonstrated when the process was initiated to change the Code of Federal Regulations more than 20 y ago in response to Publication 26 from the International Commission on Radiological Protection and other recommendations.

Currently we find ourselves in a situation where the possibility of a radiological accident or attack may occur and where the existing body of regulations provides very little guidance. Many international and national bodies, including several federal agencies, have provided recommendations on the appropriate levels of exposure for first responders and first receivers, as well as for the general public. However,

some agencies provide guidelines based on very conservative dose limits which are not appropriate in situations where there is a substantial chance for the loss of lives and critical infrastructure. It is important that an emergency response is not hampered by overly cautious guidelines or regulations. In a number of exercises the impact of disparate guidelines and training in radiological situations has highlighted the need for clear reasonable limits that maximize the benefit from an emergency response and for any cleanup after the incident.

This presentation will focus first on the federal infrastructure established to respond to radiological accidents and incidents. It will review briefly the major recommendations, both international and national, for responders and will attempt, where possible, to establish the scientific foundation for these guidelines. We will also stress the need to clearly and openly communicate the recommendations to the first responders and the public so that no unnecessary anxiety or associated irrational actions on their part impedes the ability to respond to a disaster. Finally, the use of these guidelines and recommendations by decision makers at all levels will be discussed.

4:15 pm

Closing Remarks

Thomas S. Tenforde
President, NCRP

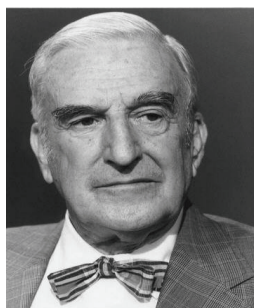
4:25 pm

Adjourn

Mission Statement

The National Council on Radiation Protection and Measurements (NCRP) seeks to formulate and widely disseminate information, guidance and recommendations on radiation protection and measurements which represent the consensus of leading scientific experts. The Council monitors areas in which the development and publication of NCRP materials can make an important contribution to the public interest.

The Council's mission also encompasses the responsibility to facilitate and stimulate cooperation among organizations concerned with the scientific and related aspects of radiation protection and measurements.



Lauriston S. Taylor
1929–1977



Warren K. Sinclair
1977–1991



Charles B. Meinhold
1991–2002



Thomas S. Tenforde
2002–

*Recognized worldwide as an authority on
radiation health protection for over 75 years.*



Program Committee

Antone L. Brooks, <i>Chairman</i>	Ann R. Kennedy
Joel S. Bedford	Amy Kronenberg
Bruce B. Boecker	Charles E. Land
R.J. Michael Fry	Roy E. Shore
Dudley T. Goodhead	Julie E. Timins
Eric J. Hall	Susan D. Wiltshire
Kenneth R. Kase	Gayle E. Woloschak

Registration

Monday, April 14, 2008 7:00 am – 5:00 pm

Tuesday, April 15, 2008 7:00 am – 1:00 pm

(no registration fee)

Register online: <http://registration.ncrponline.org>

2009 Annual Meeting

*Future of Nuclear Power Worldwide: Safety,
Health and Environment*

March 2-3, 2009
Bethesda, Maryland

NCRP Publications

(<http://NCRPpublications.org>)

Low Dose and Low Dose-Rate Radiation Effects and Models

Publication	Title	Price
Report No. 150	Extrapolation of Radiation-Induced Cancer Risks from Nonhuman Experimental Systems to Humans	\$ 65.00
Report No. 136	Evaluation of the Linear-Nonthreshold Dose-Response Model for Ionizing Radiation	50.00
Report No. 126	Uncertainties in Fatal Cancer Risk Estimates Used in Radiation Protection	25.00
Report No. 117	Research Needs for Radiation Protection	30.00
Report No. 116	Limitation of Exposure to Ionizing Radiation	35.00
Report No. 115	Risk Estimates for Radiation Protection	35.00
Report No. 107	Implementation of the Principle of As Low As Reasonably Achievable (ALARA) for Medical and Dental Personnel	35.00
Report No. 104	The Relative Biological Effectiveness of Radiations of Different Quality	45.00
Report No. 96	Comparative Carcinogenicity of Ionizing Radiation and Chemicals	40.00

Excerpts from reviews and correspondence related to NCRP reports:

“The report [NCRP Report No. 150] was authored by an outstanding committee of scientists who have extensive experience in radiation carcinogenesis and mutagenesis in nonhuman systems and in risk assessment.”

“This report will be of considerable interest to anyone concerned with problems in risk assessment and particularly how studies of nonhuman systems can help reduce uncertainties in risks that cannot be addressed in epidemiological studies.”

K.L. Mossman

[published in *Health Physics* **91** (2006) 171]

“Although an exhaustive citation of the vast literature was outside the scope of its report [NCRP Report No. 136], the Committee made a concerted effort to evaluate all data pertinent to the LNT hypothesis, whether pro or con.”

A.C. Upton

[published in *Health Physics* **82** (2002) 256]

Reports and commentaries are available from the NCRP website, <http://NCRPpublications.org>, in both soft- and hardcopy formats. Complete book reviews of recent NCRP publications are also available at this website.



Contracts/Grants/ Contributors/Sponsors

These organizations have supported the work of the National Council on Radiation Protection and Measurements during the period of January 1 to December 31, 2007.

Contracts

- Centers for Disease Control and Prevention
- Defense Threat Reduction Agency
- U.S. Department of Homeland Security
- U.S. Department of Veterans Affairs
- U.S. Navy

Grants

- National Cancer Institute
- U.S. Department of Energy
- U.S. Environmental Protection Agency
- U.S. Nuclear Regulatory Commission

Contributors

- American Academy of Health Physics
- American Academy of Oral and Maxillofacial Radiology
- American Association of Physicists in Medicine
- American College of Medical Physics
- American College of Radiology Foundation
- American Industrial Hygiene Association
- American Nuclear Society
- American Osteopathic College of Radiology
- American Roentgen Ray Society
- American Society for Therapeutic Radiology and Oncology
- American Society of Radiologic Technologists
- Council on Radionuclides and Radiopharmaceuticals
- Health Physics Society
- Landauer, Inc.
- Lillian and Robert Brent Fund
- Radiological Society of North America
- Society of Nuclear Medicine
- Society for Pediatric Radiology
- Xoran Technologies, Inc.

Corporate Sponsors

- 3M
- Duke Energy Corporation
- GE Healthcare
- Global Dosimetry Solutions
- Landauer, Inc.
- Nuclear Energy Institute
