June 26, 1995

EPA-SAB-RAC-95-011

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, DC 20460

Subject: Recommendations for Radon Research

Dear Ms. Browner:

Since 1984 the Science Advisory Board has reviewed a number of radon-related scientific and technical issues, usually in the context of a request by the Agency for a review of specific topics or issues. This report, undertaken upon the initiative of the Radiation Advisory Committee, is the first Science Advisory Board report to look at the entire field of radon research and the contribution additional scientific understanding can make on the Agency's policies. This examination approached radon research from the joint perspective of using risk and risk reduction as an element for setting priorities, as articulated in the SAB's *Reducing Risk*, and recognizing the contribution further examination of uncertainties can make to informed debate on science and policy issues. The goal was to identify research needs that would help the Agency address uncertainties in radon risk evaluation and reduction. This report assesses these needs not only in the context of programs within the Agency, but also identifies programs and activities elsewhere whose results would be of importance to the Agency.

The Radon Science Initiative Subcommittee, established by the Radiation Advisory Committee, had three broad objectives for this effort:

a) What are the remaining important areas of scientific uncertainty that affect: 1) the estimates of exposure and risk associated with radon; and 2) risk-reduction strategies?

b) Broadly, what scientific efforts are currently underway, both within the Agency and outside, that would address these areas?

c) What are the short- and long-term research needs for the Agency's own programs, and what are the priorities for these efforts?

A broad range of research topics was identified, based on the assembled expertise of the Subcommittee members, and through discussions with colleagues and staff members of the EPA and other federal agencies. These topics fell into five areas:

a) Factors affecting the bases for radon risk estimates.
b) Factors affecting concentrations, exposures and exposure assessment.
c) Ingestion of drinking water.
d) Exposure/dose/risk reduction methods.
e) Risk communication.

The report provides discussions of the present status of the research in each of these areas and the potential of studies to improve the scientific basis for evaluating and reducing radon risks to the public.

Upon further examination of these topics, the Subcommittee established several priority research areas, as discussed in more detail in the report. An overarching research priority identified by the Subcommittee is that of improving risk communication with the public to enable the public to participate effectively in risk management. Priorities for both short-term and long-term technical research were also identified: high-priority, short-term research (e.g., durability and performance of active and passive mitigation systems); high-priority, long-term research: (e.g., synergism between smoking and radon and implications for radon effects in non-smokers; radon risks at low cumulative doses and dose rates); medium-priority, short-term research: (e.g., development of methods to identify high radon areas; improvements in the accuracy and precision of radon measurement methods and protocols; better characterization of mine exposure comparison to home exposure stressing differences as well as similarities); and medium-priority, long term research: (e.g., development of potential physical or biological markers of exposure or dose).

The Subcommittee hopes you will find this report useful and is ready to answer any questions you may have about the details. The Subcommittee looks forward to a written response from the Agency regarding this report.

Sincerely,

Genevieve M. Matanoski

Dr. Genevieve M. Matanoski, Chair
Science Advisory Board

James E Watson

Dr. James E. Watson, Chair
Radiation Advisory Committee

Richard G. Sextro

Dr. Richard G. Sextro, Chair
Radon Science Initiative
Subcommittee

NOTICE

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ABSTRACT

Radon, which is the single largest source of ionizing radiation exposure to the general public, is at an important conjunction between science, public policy and the communication of science and of risk. Although the scientific basis for understanding radon exposures and risks is reasonably solid, a number of uncertainties remain that affect the policies and programs aimed at evaluating and reducing risks to the public associated with radon. For these reasons, the Radon Science Initiative Subcommittee of the Science Advisory Board's Radiation Advisory Committee has prepared this report on radon research from the perspective of the potential contribution that improved scientific understanding may make to policy through uncertainty reduction. The Subcommittee reached consensus on research recommendations in five areas: a) factors affecting the bases for radon risk estimates, b) factors affecting concentrations, exposures, and exposure assessment, c) ingestion of drinking water containing radon, d) exposure/dose/risk reduction methods, and e) risk communication.

An overarching area of research identified by the Subcommittee is that of improving risk communication with the public as a means of better linking public policy and public response. Priorities for both short-term and long-term technical research were also identified: high-priority, short-term research: (e.g., durability and performance of active and passive mitigation systems); high-priority, long-term research: (e.g., synergism between smoking and radon and implications for radon effects in non-smokers; radon risks at low cumulative doses and dose rates); medium-priority, short-term research: (e.g., development of methods to identify high radon areas; improvements in the accuracy and precision of radon measurement methods and protocols; better characterization of mine exposure comparison to home exposure stressing differences as well as similarities); and medium-priority, long term research: (e.g., development of potential physical or biological markers of exposure or dose).

KEYWORDS: Radon, Radon Decay Products, Research, Uncertainty, Exposure, Risk, Risk Reduction, Risk Communication

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1. EXECUTIVE SUMMARY

Radon is the largest single source of ionizing radiation exposure for members of the general public. The scientific basis for understanding radon exposures and risks is reasonably solid, based on health risks observed in humans - albeit with much higher exposures than are found among the general population. There are, however, a number of uncertainties with regard to the assessment of these risks for the general public and the methods of communicating and reducing these risks to which further research should be directed.

The Science Advisory Board (SAB) has reviewed a number of radon-related scientific and technical issues for the United States Environmental Protection Agency (EPA) since 1984 (see listing in Appendix A). Following the principles articulated in the SAB's *Reducing Risk* regarding the use of risk and risk reduction as an element in identifying priorities, and utilizing the premise that further examination of the uncertainties can assist in providing an informed debate, the SAB's Radiation Advisory Committee formed a Subcommittee to advise the Agency about radon research in a more comprehensive and integrated manner.

The goal of the effort undertaken by the Subcommittee was to identify research areas and needs that would help the Agency identify and address the uncertainties that exist in the current understanding of radon science. This review assesses these needs not only in the context of programs within the Agency, but also identifies programs and activities elsewhere whose results would be of importance to the Agency in continuing to develop and refine public policies directed toward reducing the risks associated with radon exposure. Radon research is a dynamic field spanning multiple disciplines. Indeed the deliberations reported here benefitted greatly from the disciplinary mix of the Subcommittee members.

The Subcommittee had three broad objectives in undertaking this effort:

a) What are the remaining important areas of scientific uncertainty that affect: 1) the estimates of exposure and risk associated with radon and 2) risk-reduction strategies?

b) Broadly, what scientific efforts are currently underway, both within the Agency and outside, that would address these areas?

c) What are the short-and longer-term research needs for the Agency's own programs, and what are the priorities for these efforts?

A broad range of research topics was identified, based on the assembled expertise of the Subcommittee members, and through discussions with colleagues, members of the public and through presentations and comments by staff from the Environmental Protection Agency, the Department of Energy, the Department of Housing and Urban Development, the National Institute of Science and Technology, the National Cancer Institute and from staff of the National Academy of Science/National Research Council associated with the Committee on the Biological Effects of Ionizing Radiation VI (BEIR VI). These topics fell into five broad areas:

a) Factors affecting the bases for radon risk estimates.
b) Factors affecting concentrations, exposures and exposure assessment.
c) Ingestion of drinking water.
d) Exposure/Dose/Risk reduction methods.
e) Risk Communication.

In undertaking this effort, the Subcommittee was aware that in some cases research on some of these topics is already underway, sponsored by several federal agencies and in some cases, the states themselves. The Subcommittee did not want to prejudge the outcome of these efforts, but by including issues already a focus of an ongoing study, the Subcommittee recognized the importance of the research and wanted to ensure that it received the Agency's attention and critical scrutiny.

Through further Subcommittee discussion, a consensus list of research recommendations was developed within these areas and a summary list is shown in Table I. The report provides discussions of the present status of the research

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Table I. Summary List of Consensus Topics for Radon Research (Section Number) (This table is also in Section 3.1)

1. Factors Affecting the Bases for Radon Risk Estimates (3.3)

Epidemiology (3.3.1)
-Epidemiological studies of lung cancer and residential radon exposures
-Further evaluation of the epidemiologic studies of miners
-Interaction between smoking and radon

Table I. Summary List of Consensus Topics for Radon Research (Section Number) (Continued)

Mechanistic Studies (3.3.2)

-*In vitro* studies of cellular response to alpha particles
-Biomarkers of prior exposure to radon decay products
-Molecular studies and genetic susceptibility

Dosimetric Studies (3.3.2)

-Biological markers or biological measures of dose (biodosimeters)
-Critical cells at risk to lung cancer induction by alpha-particle irradiation
-Skeletal lead-210 in miners as an estimator of cumulative exposure to radon decay products
-Polonium-210 on surfaces as an integrating exposure monitor
-Dosimetric models

General Issues In Radiation Biology Pertinent To Radon (3.3.4)

-Extrapolation to low doses - use of the linear/no threshold model
-Effects of exposure rate on radon risk estimates
-The alpha-particle quality factor

2. Factors Affecting Concentrations, Exposures, and Exposure Assessment (3.4)

High Radon Regions (3.4.1)

-Identification of regions with the greatest potential for elevated indoor radon concentrations

Measurement and Interpretation Methods (3.4.2)

-Accuracy and precision of radon measurement devices and protocols.
-Adequacy of sampling methods for radon in drinking water

Factors Affecting Total Exposure and Dose (3.4.3)

-Exposure to radon progeny in mine and indoor atmospheres
-Contribution of radon-220 to radiation exposures indoors

3. Ingestion of Radon in Drinking Water (3.5)

Gastrointestinal and Non-gastrointestinal Cancer (3.5.1)

Stomach dosimetry
Epidemiological evidence for stomach and other gastrointestinal cancers

4. Exposure/Dose/Risk Reduction Methods (3.6)

Source Control for Retrofit and New Applications (3.6.1)

-Durability, long-term performance, and adverse effects of active mitigation systems
-Passive radon mitigation methods

-Radon-resistant new construction
-Control of Radon-220

Radon and/or Radon Decay Product Reduction Methods (3.6.2)
-Radon concentration control
-Airborne radon decay product removal
Reduction in Water-borne Radon Concentrations
-Removal of radon from domestic water

5. Risk Communication and Evaluation (3.7)

Communicating Technical Information and Scientific Uncertainty (3.7.1)
-Communication of technical information
-Communication of scientific uncertainty
Motivating Public Action: Goals for Risk Communication (3.7.2)
-Education vs persuasion as risk communication approaches
-Sequential decision-making
Public Participation (3.7.3)
-Incorporation of public participation in decision-making at EPA

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in each of these areas and the potential of such studies to improve the scientific basis for evaluating and reducing of radon risks to the public.

These areas were then examined further and the Subcommittee endeavored to prioritize the research and to select high- and medium-priority topics. The Subcommittee recognized that the time frame for the research is an important element in comparing research proposals and in setting priorities. In discussing priorities, the Subcommittee used an approximate 3 year time horizon as the distinction between short- and long-term research, recognizing that in some cases, certain research areas would not fall easily into either category.

The recommendations that follow represent the Subcommittee's combined expert judgment as to the areas in which a significant reduction in either the uncertainty of current risk estimates, or in domestic radon risk itself, can potentially be obtained. However, the Subcommittee believes that a more formal quantitative analysis could be undertaken, in which estimates are made of the potential reduction in risk and risk uncertainties, as a function of research dollars invested. Some preliminary work of this type has been performed, and the Subcommittee suggests that such a formal analysis should be considered as a research topic in its own right.

An overarching priority for research is the combined topic of continued research on the communication of technical information along with the uncertainties associated with that

information. This topic is an extremely important part of the link between policies to reduce radiation exposures and obtaining public action. Studies to improve the communication of information about radon should contribute to the overall goal of reducing radon risk while generally improving knowledge and awareness of the issue. The Subcommittee felt that the goals of improving the technical content of public information and improving the communication process with the public are not incompatible and that research in these areas is vital to providing a better basis for decision making by members of the public.

With respect to technical areas, two long-term research areas were considered by the Subcommittee to be high priority research topics. The first is the question of the interaction between risks associated with smoking and those associated with radon progeny exposure.

The nature of the synergism between smoking and radon has important implications for the assessment and discussion of the risks and the attendant uncertainties among the various segments of the population (never-, former- and current smokers). The second high-priority topic is research on risks at low cumulative doses and dose rates. Though the Subcommittee is not suggesting that the present risk assessment paradigm - extrapolation of effects observed in miners with high doses and dose rates to the lower total doses and dose rates experienced in residential settings - is incorrect, it should be recognized that the uncertainties inherent in this process are not insignificant. Several recently published residential epidemiological studies or analyses have yielded results that are consistent with the risk extrapolations based on the miner studies. However, the statistical significance of these results is weak and they are not sufficient to address the uncertainties quantitatively.

In addition to research priorities designed to reduce the scientific uncertainties surrounding assessment of the risks associated with radon exposure, the Subcommittee also selected a high-priority short-term research topic that addresses risk reduction - the continued research into techniques and systems to reduce radon concentrations. These investigations should focus on the durability and performance of radon mitigation systems, including both active (fan-powered) and passive systems, in retrofit as well as application to new buildings.

With regard to medium priority topics, the Subcommittee felt that the development of physical or biological markers of exposure or dose from radon progeny is an important long-term research area. In part, the need for research in this area arises out of the common concern that radon progeny exposure estimates are often complicated by uncertainties in the temporal variability of radon and/or radon progeny concentrations, "unattached" fractions, radon and radon progeny equilibrium and other confounding issues. The use of polonium-210 in glass has received some attention as a means of estimating the cumulative radon exposure. In the area of biological monitors, two areas of focus currently exist, skeletal lead-210 as in integrating

exposure monitor and damage to the p53 tumor suppressor gene. This latter indicator could also be found to play a role as a biomarker of effect. These types of biomarkers could also prove extremely valuable in both mechanistic and epidemiological studies.

There are three short-term research topics assigned a medium priority by the Subcommittee. These topics have a potential effect on the assessment of concentrations and/or exposures - a) development of methods to identify regions with the greatest radon potential; b) improvements in the accuracy of radon measurement methods (devices and protocols); and c) better characterization of 22 exposure conditions in mines and homes, especially with respect to the differences between them.

2. INTRODUCTION

2.1 Background

Radon, because it is the single largest source of ionizing radiation exposure to the general public and is largely unregulated, is at an important conjunction between science, public policy and the communication of science and of risk. Public policy should be informed by the best science. Although the scientific basis for understanding radon exposures and risks is reasonably solid, and has improved considerably over the past decade, a number of uncertainties remain that affect the policies and programs aimed at evaluating and reducing risks to the public associated with radon. Examples of these areas of uncertainty include factors that affect exposures and the resultant dose, measurement methods and strategies, the effects and/or significance of co-carcinogens (including environmental tobacco smoke (ETS) contaminants), and risk reduction strategies and technological approaches for existing and new buildings.

Over the past several years, the SAB has provided reviews and input in several of these areas. These reviews and SAB reports are listed in Appendix A. While these results have covered a number of important areas, often these considerations were constrained to relatively narrow topics both by the time pressure for review as well as by the framework in which the review was requested and conducted.

In 1991 the Radiation Advisory Committee of the SAB began to consider whether a broader perspective in providing the Agency with information and advice regarding the expanding field of radon research might be a constructive undertaking. In 1992, the Committee approved such an initiative, but deferred its start to allow the Committee resources to undertake a time-critical review of the release of carbon-14 from the disposal of high-level radioactive wastes.

In 1993, the Committee established the ten-member Radon Science Initiative Subcommittee which prepared this report. Most Subcommittee members conduct research on radon and came to the Subcommittee with distinct and varied views about radon research. Their expertise includes radon risk communication, dosimetry, epidemiology, health physics, measurement, mitigation, radiation biology, and state radon programs. The goal of the effort undertaken by the Subcommittee was to take a broad view, and to identify research areas and needs that would help the Agency identify and address the uncertainties. This review would assess these needs not only in the context of programs within the Agency, but would also identify programs and activities elsewhere whose results would be of importance to the Agency.

To prepare this document, the Subcommittee held public meetings on February 16-17, May 20-21, September 7-8, 1993 and by conference call on October 21, 1993. The Subcommittee also scheduled several conference call writing and editing sessions.

2.2 Approach

This effort was undertaken with the goal of developing a comprehensive and integrated view of radon research being undertaken within or for the Agency and, to the extent possible, of research efforts underway or sponsored by other agencies. This appraisal sought to determine what further research is needed toward improving the scientific basis for understanding the occurrence, behavior, health effects and exposure reduction methods for radon and its radioactive decay products.

Consistent with the goal of providing a broad perspective on radon research, the Subcommittee considered research that would lead to reduced radon risks and/or reduced uncertainty about radon risks to be important to EPA's mission of protecting public health and the environment.

The Subcommittee agrees with the principle of reducing risk as articulated in the Science Advisory Board's 1989 report, Reducing Risk (U.S. EPA 1990). Among the ten recommendations highlighted in that report are:

> EPA should target its environmental protection efforts on the basis of opportunities for the greatest risk reduction.
>
> EPA should reflect risk-based priorities in its strategic-planning process.
>
> EPA should reflect risk-based priorities in its budget process.

Radon research associated with risk reduction includes radon-resistant construction of new homes, identification of homes with higher radon levels, mitigation of existing homes with elevated concentrations, and risk communication with the public.

The Subcommittee, like the Radiation Advisory Committee and the Science Advisory Board, believes quantitative uncertainty analysis (and disclosure of that analysis) is increasingly important to scientists and is increasingly the norm for those making decisions based, at least in part, on science. More specifically, the Subcommittee believes that the honest debate in the scientific community about radon risks may influence public perceptions of the risk and in this way affect risk reduction. The debate also relates to the relative importance (and cost) of

reducing radon risk instead of some other human health risk. Uncertainty analysis and research on those parameters of greatest uncertainty are ways of informing the debate. Radon research topics that address uncertainty reduction include radon measurement, exposure and dose assessment, dose rate effects, risk assessment, and factors such as smoking or genetic susceptibility that would affect an individual's likelihood of developing cancer.

Prior Science Advisory Board reviews have made recommendations for priorities based upon the expert judgment of the assembled reviewers. The Radon Science Initiative Subcommittee discussed the merits of setting research priorities based on formal quantitative uncertainty analysis and research cost information. While this approach appears to be an interesting and perhaps useful methodology, it was not entirely clear to the Subcommittee that the approach could be extended quantitatively across the spectrum of research issues considered. In this report, the Subcommittee relied on its expert judgment to reach consensus on certain research issues and research priorities that lead to risk or uncertainty reduction and, within those categories, to identify the issues which would produce results of most importance to the Agency's programs. A more formal quantitative uncertainty analysis could be undertaken, at least for some technical topics, that would yield a measure of the reduction in uncertainty vs. the effort required. Such a formal analysis should be considered as a research topic in its own right.

3. CONSENSUS RECOMMENDATIONS AND PRIORITIES FOR RADON RESEARCH

In its discussions about radon research, the Subcommittee considered a broad scale of topics and ideas. In this process, the Subcommittee reached consensus regarding the importance or utility of a number of these research topics. These research areas are listed below in Table 1 and are discussed in more detail in sections 3.3 through 3.7. These issues were then examined further and the Subcommittee endeavored to prioritize the research and to select high- and medium-priority research topics . Finally, there were several research areas discussed and considered for which the Subcommittee did not reach consensus. In order that these not be "lost" from future consideration by the Agency or by others these topics are listed and briefly discussed in Section 4.

3.1 Consensus Research Topics

The Subcommittee found that the research topics fell into five general categories. These are shown in Table 1, along with a listing of the research topics for each category. Additional discussion and details are given in Sections 3.3 to 3.7 that follow.

TABLE 1 Summary List of Consensus Topics for Radon Research (Section Number)

1. Factors Affecting the Bases for Radon Risk Estimates (3.3)

Epidemiology (3.3.1)

-Epidemiological studies of lung cancer and residential radon exposures

-Further evaluation of the epidemiologic studies of miners

-Interaction between smoking and radon

Mechanistic Studies (3.3.2)

-*In vitro* studies of cellular response to alpha particles

-Biomarkers of prior exposure to radon decay products

-Molecular studies and genetic susceptibility

TABLE 1 Summary List of Consensus Topics for Radon Research (Section Number) (Continued)

Dosimetric Studies (3.3.2)

-Biological markers or biological measures of dose (biodosimeters)
-Critical cells at risk to lung cancer induction by alpha-particle irradiation
-Skeletal lead-210 in miners as an estimator of cumulative exposure to radon decay products
-Polonium-210 on surfaces as an integrating exposure monitor
-Dosimetric models

General Issues In Radiation Biology Pertinent To Radon (3.3.4)

-Extrapolation to low doses - use of the linear/no threshold model
-Effects of exposure rate on radon risk estimates
-The alpha-particle quality factor

2. Factors Affecting Concentrations, Exposures, and Exposure Assessment (3.4)

High Radon Regions (3.4.1)

-Identification of regions with the greatest potential for elevated indoor radon concentrations

Measurement and Interpretation Methods (3.4.2)

-Accuracy and precision of radon measurement devices and protocols.
-Adequacy of sampling methods for radon in drinking water

Factors Affecting Total Exposure and Dose (3.4.3)

-Exposure to radon progeny in mine and indoor atmospheres
-Contribution of radon-220 to radiation exposures indoors

3. Ingestion of Radon in Drinking Water (3.5)

Gastrointestinal and Non-gastrointestinal Cancer (3.5.1)

-Stomach dosimetry
-Epidemiological evidence for stomach and other gastrointestinal cancers

TABLE 1 Summary List of Consensus Topics for Radon Research (Section Number) (Continued)

4. Exposure/Dose/Risk Reduction Methods (3.6)

Source Control for Retrofit and New Applications (3.6.1)
- -Durability, long-term performance, and adverse effects of active mitigation systems
- -Passive radon mitigation methods
- -Radon-resistant new construction
- -Control of Radon-220

Radon and/or Radon Decay Product Reduction Methods (3.6.2)
- -Radon concentration control
- -Airborne radon decay product removal

Reduction in Water-borne Radon Concentrations
- -Removal of radon from domestic water

5. Risk Communication and Evaluation (3.7)

Communicating Technical Information and Scientific Uncertainty (3.7.1)
- -Communication of technical information
- -Communication of scientific uncertainty

Motivating Public Action: Goals for Risk Communication (3.7.2)
- -Education vs persuasion as risk communication approaches
- -Sequential decision-making

Public Participation (3.7.3)
- -Incorporation of public participation in decision-making at EPA

3.2 Priorities for Radon Research

In assembling both the individual research recommendations and a broader priority perspective, the Subcommittee is aware that in some cases research on some of these topics is already underway. In some situations, the results of those projects may substantially address the issues raised here, in which case further research may not carry the same priority. This would depend greatly upon the nature of the issue and how well the present research activity(ies)

addressed the topic. The Subcommittee did not want to prejudge the outcome of these research efforts, but in recognizing the importance of various research areas, the Subcommittee wanted to ensure that these areas received the Agency's attention and critical scrutiny.

The Subcommittee recognized that the research time frame is an important element in comparing research proposals and in setting priorities. In discussing priorities, the Subcommittee used an approximate 3-year time horizon to distinguish between short-and long-term research, recognizing that in some cases, certain research areas would not fall easily into either category, depending upon the level of funding available, whether breakthroughs in the research might accelerate the conclusions or applications of the work, or conversely, whether the research area turns out to be more complicated than first thought (a not uncommon occurrence).

An overarching priority for research is the combined topic of continued research on the communication of technical information along with the uncertainties associated with that information. This topic area is an extremely important part of the link between policies to reduce radiation exposures and obtaining public action. Studies to improve the communication of information about radon should contribute to the overall goal of reducing radon risk while generally improving knowledge and awareness of the issue. The Subcommittee felt that the goals of improving the technical content of public information and improving the communication process with the public are not incompatible and that research in these areas is vital to providing a better basis for decision making by members of the public. This view echoes the recommendations made previously by the SAB in *Future Risk* (U.S. EPA, 1988) where risk communication was listed as a candidate core research area for risk reduction.

3.2.1 High Priority Technical Research Areas

Two long-term research areas were considered by the Subcommittee to be high priority research topics. The first is the question of the interaction between risks associated with smoking and those associated with radon progeny exposure. The nature of the synergism between smoking and radon has important implications for the assessment and discussion of the risks and the attendant uncertainties among the various segments of the population (never-, former- and current smokers). Although the Subcommittee was not able to suggest a definitive study for this issue, it recognized that further well-defined animal or mechanistic studies could be useful in understanding the issue. The Subcommittee did not believe that, based on present knowledge, new residential epidemiological studies focusing on this topic would have sufficient power to significantly resolve the current uncertainties in this area. However, the topic is of sufficient importance that solicitation and potential funding of high-quality, peer-reviewed research proposals in this area should be a high priority for the EPA or other federal agencies.

The second high priority topic is research on risks at low cumulative doses and dose rates. Although the Subcommittee is not suggesting that the present risk assessment paradigm - extrapolation of effects observed in miners with high doses and dose rates to the lower total doses and dose rates experienced in residential settings - is incorrect, it should be recognized that the uncertainties inherent in this process are not insignificant. Several recently published residential epidemiological studies or analyses have yielded results that are consistent with the risk extrapolations based on the miner studies. However, these results are not sufficient to address the uncertainties quantitatively. While the Subcommittee does not believe that new epidemiological studies will, by themselves, have the power to address the uncertainties, the Subcommittee did not identify a definitive course of research in this area. As with the issue of smoking, the Subcommittee believes that this topic is of sufficient importance that the Agency and/or other federal agencies should attach high priority to soliciting and funding proposals in this area. This general topic cuts across a broad set of radiation protection issues, including both low-and high-LET radiation exposures.

In addition to research priorities designed to reduce the scientific uncertainties surrounding the risk of radon exposure, the Subcommittee also identified a high priority short-term research topic that addresses risk reduction. Research into techniques and systems to reduce radon concentrations should continue, including further investigations on the durability and performance of radon mitigation systems, including both active (fan-powered) and passive systems, in retrofit as well as new applications. This view is consistent with the recommendation made to the Agency by the SAB in 1993 (U.S. EPA, 1993b) that research funding for radon mitigation research conducted by the Agency should not be further curtailed; rather, consistent with risk-based prioritization, it should receive additional attention.

3.2.2 Medium Priority Technical Research Areas

A long term research area that the Subcommittee felt had medium priority is the development of biological or physical markers of prior exposure to radon progeny. The need for such research is primarily motivated by the considerable uncertainty inherent in reconstructing prior exposure to radon progeny, and by the potential for ultimately identifying a biomarker that is predictive of radon-induced lung cancer. An example of a physical marker is polonium-210 in household glass objects, such as picture covers, which might have stayed in the possession of an individual through the various residences that person occupied. Possible radon-specific biomarkers that are currently under investigation include skeletal lead-210 as an integrating exposure monitor and unusual mutations in the p53 gene chromosomal aberration yields and/or expression of p53 protein, though this field is still very much in its infancy. The p53 gene could also be found to play a role as a biomarker of effect. These types of physical or biological markers could prove extremely valuable in both mechanistic and epidemiological studies.

Three short-term research topics were assigned a medium priority by the Subcommittee. These topics have a potential effect on the assessment of concentrations and/or exposures: 1) development of methods to identify regions with the greatest radon potential; 2) improvements in the accuracy of radon measurement methods (devices and protocols); and 3) better characterization of exposure conditions in mines and homes, especially with respect to the differences between them (extrapolation from mines to homes depends upon accounting for these differences).

3.3. Factors Affecting the Bases for Radon Risk Estimates

Based on data from epidemiological studies of uranium and other hard rock miner populations, little doubt exists that chronic exposure to relatively high levels of radon progeny causes lung cancer, and does so in a dose-dependent manner. However, whether lifetime exposure to low levels of radon progeny in indoor environments also carries a significant risk for lung cancer is much less certain. In addition, whether such risk can be predicted based on results obtained from the miner studies is complicated by uncertainties in accounting for the differences between the populations and differences in their exposure environments.

Miners, particularly uranium miners, were exposed to high levels of radon progeny for a relatively modest proportion of their lifetimes (i.e. years to tens of years during the second third of their life span). Cumulative exposures were mostly in the hundreds to thousands of working level months (WLM; the SI equivalent for exposure is mJ h/m^3, see Appendix B). In comparison, for the general population, total exposure to radon progeny is smaller and occurs at much lower exposure rates. Cumulative exposures over 70 years at an average radon concentration of 1.25 pCi/L (45 Bq/m^3) are approximately 17 WLM (60 mJ h/m^3). Some miner studies have cohorts with total exposures below 50 WLM (200 mJ h/m^3) - equivalent to 70 year lifetime residential exposures at $\approx$4 pCi/L (150 Bq/m^3) - for which the relative risks are greater than one, but the uncertainties in the relative risk estimates do not provide unequivocal evidence for risk at those exposures (see summary in Lubin et al., 1994a). Thus the data that exist from epidemiological studies of miners and from experimental animal studies are not adequate to determine directly whether the risk models can be extrapolated to the relevant cumulative exposures and low exposure rates characteristic of home indoor environments.

There are additional uncertainties in extrapolating results of the miner studies to the general population due to the many differences between the two populations. These differences relate to anatomical and physiological characteristics of the exposed populations, including gender, aerosol properties, coexposure to other potential carcinogens and promoters, and preexisting disease. The present lack of understanding of the absolute and relative importance of the various exposure-dose and dose-response modifying factors comprises the major portion of the

uncertainty in extrapolating risk factors derived from the results of the miner studies to the general population. However, several scientific committees (NRC, 1988; NCRP, 1984; and ICRP 1987, 1993) have estimated risks by extrapolating miner data to residential exposures.

The broad question addressed in this section is what further research can be (or is being) conducted to improve estimates of, or to reduce the significant uncertainties in, the average risk of radon-induced lung cancer as a function of dose, dose rate, and co-carcinogens, from nominal domestic exposure levels up to occupational (miner) levels. In the view of the Subcommittee, it is unlikely that these problems can be resolved solely by new epidemiological studies focusing directly on domestic radon exposure. In an analysis of design issues for epidemiological studies of domestic indoor exposure to radon, Lubin et al. (1990) concluded that "realistically such studies may never be able to answer many of the subtle questions about risk patterns that burden current risk assessments with uncertainty".

Therefore, new epidemiological studies should focus on comparative risks related to specific patterns of radon exposure or exposure to other factors which may differ between residential and occupational (mining) settings. A review of available data from both mechanistic and epidemiological studies should be undertaken to quantify dose-effect models before specific new studies are undertaken.

3.3.1 Epidemiology

Several uncertainties in using epidemiological data from miner studies create limitations for the risk estimates when extrapolated to residential situations. The average radon-progeny exposure in the miner data is much higher than the average lifetime domestic radon exposure (several hundred to a few thousand WLM for many miners compared with 10 to 20 WLM in an average residence). In addition the exposure rates are quite different; typical residential exposure rates are approximately two orders of magnitude lower than average miner exposure rates. The effects of these exposure rate differences on risk estimates are not understood. Other complicating features include the effects of smoking and uncertainty in the miner dosimetry. These uncertainties have recently been reviewed by the ICRP (1993).

3.3.1.1 Epidemiological Studies of Lung Cancer and Residential Radon Exposures

Given the uncertainties in the miner studies and their applicability to residential radon exposures, there are several issues for which epidemiological studies based on residential exposures are important. These include:

a) the risk associated with radon exposure in females;
b) the compatibility of the dose-effect estimates from the miners and from residential exposures at comparable cumulative doses;
c) the risk associated with radon in populations of non-smokers (many of whom are exposed to ETS);
d) the risk when the exposure pattern does not vary significantly over the exposure lifetime, compared with the mining population, where most of the exposure was accumulated during adulthood and then during work hours; and
e) the risk in populations who are exposed to other environmental contaminants that might alter the risk radon presents.

Such studies are very difficult to conduct, particularly when one objective is to reduce the uncertainties in our present understanding. In order to ensure sufficient power to show an effect, sample size and exposure magnitude are key issues, as has been discussed by Lubin et al. (1990). No single study is likely to have sufficient numbers of cases to establish unequivocally whether there is a risk from radon in residential exposure or whether that risk follows the same model predicted from the high dose miner data. Combining studies (pooling) can provide large numbers of cases over all age groups and both genders in addition to offering some information on the range of values which occur based on different samples. However, the use of combined studies can create additional problems which do not exist in the single study. For example, there may be differences in lung dose based on anatomy or changes in susceptibility based on population characteristics. Thus, a new source of error or variation is introduced by joint analysis of pooled data. Such problems must be taken into account when selecting very disparate populations for combined analysis, though such issues do not rule out the usefulness of the procedure to add statistical power.

There are also problems associated with the low total exposures encountered in residential studies. Based on the time since exposure model of BEIR IV (NRC, 1988) exposure at an average concentration of 1.2 pCi/L (44 Bq/m^3), which yields an annual exposure rate of 0.2 WLM/yr (0.9 mJ h/m^3), would result in a lifetime risk ratio of lung cancer of only 1.1 in both men and women. Relative risks of this magnitude are very difficult to detect in epidemiologic studies. It is thus important to seek study populations for which average exposures are likely to be higher than the U.S. average. The recently published Swedish residential epidemiological study (Pershagen et al., 1994) had average exposures equivalent to an average lifetime radon concentration of ≈2.9 pCi/L (100 Bq/m^3), which is almost 2.5 times the U.S. national average of 1.25 pCi/L (46 Bq/m^3) (Marcinowski et al., 1994). The recent Canadian residential epidemiological study reported average bedroom concentrations of ≈3.2 pCi/L (120 Bq/m^3) (Letourneau et al., 1994). The first of these two studies found, overall, an increase in the relative risk of lung cancer with increasing average radon concentration (exposure) that was statistically

significant for the two highest exposure cohorts (with equivalent concentrations categories from 3.8 to 10.8 pCi/L (140 to 400 Bq/m^3) and greater than 10.8 pCi/L (400 Bq/m^3). The second study, on the other hand, showed no statistically significant increase in relative risk for any of the exposure categories. While these results do not conflict statistically, the contrasting conclusions are indicative of the difficulties associated with low total exposure epidemiological studies.

Estimating total exposure is difficult - especially retrospectively - when multiple residences are involved, because it is unlikely that the total will be dominated by the exposure received in any one residence. Also, the high lung cancer risk from smoking and the synergism between smoking and radon make it difficult to isolate the small excess risk due to radon alone when the exposures are low. The risk from radon exposure alone would be easier to find among non-smokers but they comprise only a small proportion of lung cancer cases in a population. In many cases, these non-smokers will have been exposed to ETS, which will further complicate the results because ETS has been judged to be carcinogenic also (U.S. EPA, 1992c).

It is, nevertheless, important to use residential studies to investigate issues for which studies of the miner populations are ill-suited. One such pooled analysis of residential data has been reported recently (Lubin et al., 1994b). Several residential epidemiological studies which are already completed or are in progress might be additional candidates for pooling to determine whether there is any risk from radon exposure at low doses that would fit the risks calculated from the miner models. The feasibility of conducting new studies in regions where population exposures to radon are higher should be investigated, accompanied by a thorough analysis of the uncertainties that affect the statistical power of such studies. As noted in the discussion in Sections 3.3.2 and 3.3.3 below, better estimates of exposure, through development of biological or physical methods, could substantially improve the power of residential epidemiological studies.

3.3.1.2 Further Evaluation of the Epidemiologic Studies of Miners

The risk associated with radon progeny exposure in underground mining, especially uranium mining, is the major source of data for risk estimates regarding radon. The appropriateness of using that data for projecting risks in homes has been questioned, as has been discussed above. Many of the issues derive from the comparability of exposures in the home versus the mines. The mining environment includes exposure to other substances such as dust, diesel fuels, and chemicals which are not present in the home. The miners may also differ from the general population in other regards, such as breathing patterns and lung physiology.

A recently published joint analysis (Lubin et al., 1994a) builds upon and extends the earlier combined analysis conducted as part of BEIR IV (NRC, 1988). This study combines data from eleven underground miner studies from North America, China, Australia, and Europe. The study

presents the individual data from each of the miner studies and a pooled analysis and appears to be a comprehensive and uniform analysis of these data. This analysis also notes that the problem of errors in the key variables affecting exposures have not yet been addressed quantitatively. Additional discussion of the analysis of combined data is given in ICRP (1993).

The following recommendations regarding further mining studies would help reduce the uncertainty in risk estimates or illuminate some of the differences observed across the different studies:

a) because the risk per unit (estimated) dose varies by 2 to 4-fold or more for comparable doses, the populations should be examined more closely to determine the possible reasons for the differences.

b) current mining populations are a source of additional data, with regard to information on low total exposures. Further follow-up will also increase the reliability of estimates of risk for advanced age groups. Phosphate miners and other mining populations who work in radon exposed areas of industry but have very low total exposures might be an appropriate group to examine for the effects of low exposure levels.

c) estimates of the errors (uncertainties) in the exposure estimates and their consequences are of central importance to the evaluation of epidemiological analyses of miner groups and to the development of exposure-response relationships. Research into the likely nature of these errors and their implications would benefit our understanding of the exposure-response relationship for radon progeny.

3.3.1.3 Interaction Between Smoking and Radon

The miner data studies have generally had very sparse data on smoking rates in the cohorts. The recent joint analysis of miner data (Lubin et al., 1994a) has indicated that while the joint association of radon progeny exposure and smoking is consistent with a multiplicative model, the most likely relationship from that analysis is intermediate between multiplicative and additive. This joint analysis estimates that for radon-related lung cancer in the general population, smokers comprise about 2/3 of the total estimate of $\approx$15,000 deaths while non-smokers make up about the remaining 1/3. The Swedish residential study (Pershagen et al., 1994), on the other hand, suggests that smokers have relative risks of lung cancer associated with radon exposure that range from about 6 to almost 30 times the relative risk for non-smokers, depending upon both the smoking rate as well as the radon concentration category.

The discrepancy is partly due to the small number of non-smokers who have lung cancer, while the greater incidence for smokers makes estimates of the rates for smokers comparatively more stable. Animal data have suggested that smoking has a synergistic effect only when the smoking exposure follows radon exposure, suggesting that smoking has a promotional effect. In general, the miners were already smokers at the time of exposure to the elevated radon concentrations in the mines. With current changes in smoking habits of men and women in the general population it may be possible to examine this issue in households when it has not been possible to do so in the mining populations in the past.

Examination of risks of radon and smoking are especially important in residential exposures where the smoking characteristics may have more variability than in the mines. Elderly women who develop lung cancer today still may never have been smokers. Men in the general population may have had a lower rate of smoking than the miners. The miner smoking rate in the Colorado group was about 75%. The rate in the general population of males in the past was about 60 to 70%. However, population characteristics are changing rapidly and recently only about 23% of both men and women in the U.S. were found to smoke. (Siegel et al., 1991).

Due to the importance of this issue in assessing the health risks in non-smokers associated with radon, additional studies are warranted. Although the Pershagen et al. (1994) study is provocative with respect to the differences between smoker and non-smoker risks due to radon, this study had both high average radon concentrations and a large number of cases and controls (1360 and 2847, respectively). Even then, there were so few cases of lung cancer among the non-smokers that the confidence intervals for the relative risk of lung cancer in each of the radon exposure categories overlapped a relative risk of 1 (i.e., no increase in risk).

The results of Thomas et al. (1994a) as modified by a recently published *erratum* (Thomas et al., 1994b), suggest that, among the Colorado Plateau miners, the radon-smoking interaction is essentially additive if exposure to significant concentrations of radon (i.e. in the mines) is followed by smoking, while smoking followed by radon exposure produced a more-than-multiplicative effect. If these findings on exposure sequences apply to residential environments, where radon exposures will usually precede direct smoking exposures (but not necessarily exposure to environmental tobacco smoke), the estimated smoking-radon interaction might be less significant than would be estimated when the exposure sequence is reversed, as in the case of most miners.

Additional miner data should be collected to add information on more mining cohorts, particularly among contemporary miners where better estimates of actual smoking exposures might be possible. Even if data are not complete for all individuals, smoking in relation to job might be collected on a sample of the population and an effort made to get data on all lung cancer

cases. This nested case control type of study is efficient and has been used to examine several cohorts.

The model proposed in BEIR IV (NRC, 1988) has assumed that females have slightly higher risk ratios than males, especially among smokers. As the smoking habits of women begin to resemble those of men, and as their lung cancer rates increase, the models could incorporate trends to get a better value for risks for radon with smoking. Additional data on female risks from residential studies would also improve the model for women.

3.3.2 Mechanistic Studies

Based on many of the uncertainties discussed earlier, epidemiological data alone are not capable of defining an appropriate model for extrapolation from the miner exposure situation (or from the Japanese atomic bomb data) to environmental radon risks. Input from mechanistically-based *in vitro* model systems has the potential to provide additional insight for such extrapolations.

Four key areas where such mechanistic studies can be useful are:

a) extrapolation from high to low exposures
b) extrapolation from high to low exposure rates
c) elucidation of the interaction mechanisms between tobacco smoke (and other co-carcinogen) -induced damage and alpha-particle induced damage
d) estimation of an appropriate alpha-particle quality factor

3.3.2.1 *In Vitro* Studies of Cellular Response to Alpha Particles

Until recently, *in vitro* radon research has been hampered by the lack of realistic cellular models for human cancer. Much of the relevant research has been carried out using non-epithelial rodent cells in which oncogenic transformation rates can be quantified. However the use of *in vitro* human epithelial cell lines that can be transformed by alpha-particles is a key recent development in the field, and allows the investigation of dose-response relations at very low doses (i.e. where an average of less than one alpha-particle hits each cell). This capability may provide a semi-mechanistic approach for producing more credible model systems that can be used to explore dose/dose-rate/co-carcinogen effects. Such systems may, as an example, be a useful way to examine the radon - smoking interaction. Eventually, given a more realistic model and functions relating these different parameters to effects, these results could be used to guide the analysis of epidemiological data. (Brenner, 1994; Moolgavkar et al., 1993)

3.3.2.2 Biomarkers of Prior Exposure to Radon Decay Products

Because of the great difficulty in directly measuring the alpha radiation dose to the respiratory tract epithelium, the development of biological markers that can be related to radon-progeny dose (biodosimeters) should be undertaken. Such research is currently being done in several laboratories, and the preliminary results appear to be both suggestive as well as contradictory.

Vahakangas et al. (1992) studied 19 lung tumors from uranium miners and reported p53 point mutations in 7 (37%). These point mutations were unusual in that a) they were clustered around codons 146-161 and 195-208 and b) none were G:C -> T:A transversion, which are the most common inversions associated with tobacco-related lung cancer. This observation was widely considered as the first "fingerprint" of radon-induced biological damage.

Subsequently, Taylor et al. (1994) examined a larger sample of 52 lung tumors from uranium miners. They found 29 (56%) contained p53 point mutations, and, of these, 16 had G->T transversion at codon 249. This observation was again taken to be suggestive of a radon-related biomarker. In contrast, however, Vahakangas et al (1992) found zero G->T transversion, and also zero mutations at codon 249.

More recently, Hei et al. (1994) presented data suggesting that point mutations are unlikely to be an important component of radon-induced damage, in agreement with earlier theoretical arguments by Rossi (1991). Further supportive data for this suggestion were published by Venitt and Biggs (1994), who demonstrated that p53 point mutations in miners were likely to be caused by a mold (mycotoxin) present in uranium mines. Hei et al. (1994) pointed out that larger scale damage (deletions, exchange-type chromosomal aberrations) are more likely to be characteristic of radon-induced damage, and a recent paper suggests that the ratio of intra- to inter-chromosomal aberrations may represent a practical characteristic biomarker for radon damage (Brenner and Sachs, 1994).

The search for radon-specific biomarkers is a key activity in support of any future molecular-epidemiological studies of radon. Point mutations now appear unlikely to provide such a "fingerprint", but other approaches may well be feasible. Probes for specific radon-induced events (e.g., gene deletion(s), oncogeny activation(s), or specific chromosomal aberration(s)) would be useful regardless of whether or not they can be correlated with radon-induced oncogenesis. The development of such "fingerprints" would significantly affect future epidemiological studies investigating the relationship between radon exposure and cancer. "Molecular epidemiology" in which individual cancers are directly linked with a particular causative agent through detection of biological adducts or "fingerprints" is a powerful tool for chemical risk assessment, and the same techniques, if successful, could be applied to epidemiological studies of radon effects.

3.3.2.3 Molecular Studies and Genetic Susceptibility

As a long-term objective, it is desirable to use information about the molecular genetics of cancer to guide the construction of dose-effect relationships. Currently, remarkably little is known about the molecular basis of radiation-induced oncogenesis in mammalian cells, and even less about densely-ionizing radiation-induced oncogenesis. Currently, no genes associated with radiation-induced cancer have been identified, either with regard to induction or with regard to repair. If such genes are identified, the functional significance of changes in these genes may be identified, as well as the effects of dose, dose rate, and radiation quality on their activation or inactivation.

An application of identifying such radiation damage relates to the question of individual susceptibility. Little is currently known about the susceptibility of individuals to radiation-induced cancer - although there have been suggestions that A-T heterozygotes represent such a susceptible group. There is therefore a need to identify specific genes that might control radiation susceptibility, if such genes exist.

3.3.3 Dosimetric Studies

Although substantial research has been done to characterize the radiation dose to the respiratory tract from inhaled radon and radon progeny, consensus scientific committees have long recognized the considerable uncertainties that exist in determining these radiation doses. For example, the BEIR IV Committee (NRC, 1988) stated:

"Further studies of dosimetric modeling in the indoor environment and in mines are necessary to determine the comparability of risk per WLM (working level month) in domestic environments and underground mines."

A follow-up National Research Council Committee (NRC, 1991) attempted to define some of the outstanding uncertainties in comparing dosimetries for mine and home exposures by using a common dosimetry modeling approach and incorporating specific characteristics for mines and homes. Other committees and researchers have also addressed this issue (NCRP, 1984; ICRP, 1987, 1993; James, 1988). Despite these efforts, there still exists considerable uncertainty in determining the radiation dose patterns in the various regions of the respiratory tract from exposure to airborne radon and radon-progeny-containing aerosols. Recognizing that these uncertainties in determining appropriate exposure-dose relationships are probably of lesser magnitude than those associated with the dose-response relationships, they nevertheless merit research attention. Several of the major uncertainties are therefore highlighted here.

3.3.3.1 Biological Markers or Biological Measures of Dose (Biodosimeters)

It is extremely difficult to measure the alpha-radiation dose to respiratory tract epithelial tissue, due mainly to the short half-lives of the radon-progeny and to the lack of techniques to measure the radon-progeny deposition sites with sufficient resolution commensurate with the needs of the theoretical models. The development of biological markers or measures of dose (biodosimeters), in which dose distribution is determined by measuring the biological response at the cellular level both *in vivo* and *in vitro,* should be undertaken. Some studies are currently in progress at several laboratories (e.g. Inhalation Toxicology Research Institute, Pacific Northwest Laboratory, AEA Technologies, in the U.K., and CEA, in France) and some preliminary results published (Thomassen et al., 1991), but much more needs to be done. These data can provide the necessary data base, particularly across different species, for validating the existing and future theoretical dosimetry models.

3.3.3.2 Critical Cells at Risk to Lung Cancer Induction by Alpha-Particle Irradiation

Identification of the critical cells at risk to lung cancer induction by alpha-particles from the radioactive decay of deposited radon progeny is important for dosimetric modeling and for elucidating the mechanisms of radiation-induced lung cancer. Although an active area of study and debate in the lung biology community, research has not identified whether the basal or secretory cells (or both) in the lung epithelial tissue are the cells at risk, having the potential of transforming to neoplastic cells. The dosimetric significance of this question relates to the fact that, on average, basal cells are located some tens of micrometers deeper in the bronchial epithelium than secretory cells. For radon progeny remaining in the mucous layer, basal cells are exposed to a lower dose and to lower-energy alpha-particles (with potentially different biological effectiveness) compared to secretory cells. For those progeny taken up by the epithelium before decay, the doses and alpha-particle energies are approximately the same for basal and secretory cells. The degree to which the progeny migrate into the epithelium is uncertain (NRC, 1991).

3.3.3.3 Skeletal Lead-210 in Miners as an Estimator of Cumulative Exposure to Radon Decay Products

Lead-210 is a long-lived (22.3 year radioactive half life) beta-emitting radionuclide that is produced as a result of the *in vivo* decay of radon progeny. A portion of the lead-210 deposits in bone, where it remains for very long times (the effective retention half-life is about 16 years; Cohen et al., 1992), long enough to serve as a measurable indicator of cumulative exposure to radon-progeny. Techniques for measuring lead-210 in human skulls *in vivo* exist (Eisenbud et al., 1969), and should be employed to obtain better individual estimates of radon-progeny exposure,

particularly for miners. Although better exposure-dose estimates may be most useful for prospective analyses, they would also have value for retrospective dose reconstruction, primarily because of the substantial lack of information on the characteristics of the mine atmospheres (radon and radon progeny concentration, particle size, equilibrium factor, variability in different areas of the mine, variability in different mines or even the miners' homes in some cases). Some effort has been made to measure skeletal lead-210 levels in miners *in vivo* but more work is needed. Although there are uncertainties in relating measured lead-210 levels to integrated exposure, there is no doubt that there is a proportionality between the two (Laurer et al., 1993), and such measurements should reduce the interindividual uncertainties in exposure estimates.

3.3.3.4 Polonium-210 on Surfaces as an Integrating Exposure Monitor

A major difficulty in residential epidemiological studies of radon exposure and effects is reconstruction of the exposure history of the individual over the previous 25 to 40 years. With a highly mobile population, finding and measuring the current radon in all the homes that an individual has inhabited may not be possible. The house may no longer exist, it may have been substantially modified, it may have been mitigated, or the current occupants may not permit measurements to be made.

Even if a current measurement could be made, there can be variability in the annual average radon concentration in the same house over several years (Lively and Steck, 1991; Martz et al., 1991). Thus, the currently measured radon concentration may not be an accurate representation of the average value over prior years.

Studies by Dr. R. Lively of the Minnesota Geological Survey (Lively and Ney, 1987) and Dr. C. Samuelsson of Lund University, Sweden (Samuelsson, 1988) showed a relationship between measured polonium-210 embedded in glass and ambient radon concentrations. When lead-214 or bismuth-214 are deposited on a surface in a room, they will adsorb to the surface. When the resulting polonium-214 nuclei decay by emitting alpha-particles, about half of the recoiling lead-210 nuclei will be embedded in the glass, with most penetrating deep enough that subsequent cleaning of the glass surface will not remove them. However, these nuclei are close enough to the surface that the 5.3 MeV alpha from the subsequent polonium-210 can be observed. Because objects such as pictures are transported with the occupants from house to house and because the 22 year half-life of lead-210 decay permits integration of the exposure over a period of up to 60 years, measurement of the polonium-210 in the surface of household artifacts such as the glass covering pictures or documents hanging in a home may provide a good surrogate for assessing the integrated exposure to radon over an extended period of time.

All of the previous work on estimating the average radon concentration from embedded polonium-210 activities has been done using existing glass surfaces in houses and current integrated radon measurements over a period of a year or so (Samuelsson, 1992; Lively and Steck, 1993). Some laboratory and modeling studies have examined the range of possible indoor conditions and cleaning activities that might affect the relationship between airborne radon and embedded polonium-210 (Cornelis et al., 1992). However, there are still uncertainties in the calibration factor because of the variability in average radon concentrations and because homeowners are not always certain when a particular piece of glass was placed into its current environment. Research is currently in progress in the U.S. and in Europe to obtain better estimates of the variability in the calibration constant depending on the indoor aerosol conditions in homes so that it may be possible in the near future to use measurements of polonium-210 in solid surfaces as an integrating monitor for total radon exposure over the period of time that the object has been in use. At least one epidemiological study in the U.S. and one in Europe are making polonium-210 in glass measurements part of their exposure assessment protocol. A feasibility study conducted as part of this U.S. study has found that measurements of the alpha activities in the surface of selected household artifacts correlates with time-weighted radon concentrations ($R^2 = 0.48$) (Mahaffey et al., 1993). Additional studies are needed to better define when such measurements can or cannot be used to assess the exposure of household occupants to radon with sufficient accuracy to be of assistance in improving the risk and uncertainty assessment of radon indoors.

3.3.3.5 Dosimetric Models

Alpha-radiation doses to respiratory tract tissues are currently obtained strictly through the use of theoretical mathematical models. However there are several issues involving their use and incorporation of new experimental data.

a) Discrepancies in dosimetric model predictions should be resolved to enable better, more robust dosimetry.

The three different models in current use (NRC, 1991; NCRP, 1984; ICRP, 1987) predict different doses per unit exposure for different exposure scenarios. Thus, there currently is no consensus as to the appropriate model that best predicts these doses. Resolution of the discrepancies in the model predictions may lie either in improving the parameter estimates that go into the models, in reaching agreement on the appropriate deposition theory (deposition controls dosimetry for radon progeny), or in experimental validation of the radiation dose patterns incurred by respiratory tract tissues following inhalation of radon-progeny-containing aerosols.

b) Detailed studies of airway deposition using combined bronchial and head airway replica models are needed.

Much new knowledge has been obtained on the importance of the nasal and oral airways in filtering out ultrafine particles (unattached and attached fractions) containing radon progeny. Thus far, this has been accomplished using deposition data obtained from head airway replica models (Cheng et al., 1991, Swift et al., 1991), and by confirmatory exposure of small numbers of human volunteers (Strong et al., unpublished, Cheng et al., unpublished). Additionally, some studies of deposition patterns in bronchial airways models have been done. However, detailed deposition studies using bronchial models together with head airway models (including pharynx and larynx) need to be done, as it is likely that deposition patterns in the thoracic conducting airways will be strongly influenced by the inclusion of the nasal and oral passages in the models.

c) More complete data are needed on respiratory tract anatomy and breathing patterns and on cell histology and morphometry.

More complete data are needed on age-dependent respiratory tract anatomy and breathing patterns for different activity levels to allow dosimetry modeling throughout the lifetime. Additionally, it is well known that diseases such as bronchitis and emphysema alter the structure of lung tissue, and that both obstructive and restrictive lung disease alter breathing patterns significantly. Both types of changes will affect the radiation dose patterns associated with inhaled radon progeny aerosols. In addition, the limited data on cell types and dimensions that are currently available lead to uncertainties in the dosimetry. Better information on the range of cell characteristics would improve the model estimates.

3.3.4 General Issues In Radiation Biology Pertinent To Radon

With few exceptions, most of our current understanding of the carcinogenic effects of exposures to either low- or high-LET ionizing radiation has come from observations at high doses and high dose rates. Most direct human evidence has come from the atomic bomb survivors for low-LET radiation and from underground miner populations (mainly uranium miners) for alpha (high LET) radiation (NRC, 1990). Because there are few data on effects from radiation exposures at lower total doses and dose rates, estimates of the health consequences of these exposures are based on extrapolation from data at higher doses and dose rates. Three issues arise in using this approach for which further research is important: extrapolation to low doses, effect of exposure rates, and the quality factor for alpha particles.

3.3.4.1 Extrapolation to Low Doses: Use of the Linear/No Threshold Model

Because radon risk estimates are based for the most part on higher cumulative exposures (e.g., from miners) than that which accumulates in residential environments, dose-response relations need to be assumed in order to permit extrapolation to lower exposures. This has almost universally been accomplished by assuming that the dose-response relationship at low exposures is linear and does not exhibit an effect threshold, i.e., zero effect is predicted to occur only at zero dose. Animal data show no threshold effect down to cumulative exposures of 20 WLM (70 mJ h/m^3) (Cross, 1990).

Verification of these assumptions is important because it applies to such a large number of people, i.e., the lognormal distribution of measured radon levels in homes suggests that most of the population will be exposed to the lowest radon progeny levels. Reducing the uncertainties in our current understanding can yield important societal benefits, either in terms of the potentially preventable lung disease caused by underestimating the risk, or conversely, by allocating large amounts of limited resources without achieving any benefit through overestimating the risks.

Although the experiments needed to address the issue of biological effects of low-level radiation exposure are difficult to conduct, an effort should be directed toward studies designed to quantitatively address the uncertainties. One such approach might be to utilize an animal population to ensure that uncertainties are not dominated by statistical considerations. However, such an effort would have to carefully consider the applicability of the results for estimating human health responses.

3.3.4.2 Effects of Exposure Rate on Radon Risk Estimates

There is evidence from epidemiological, *in vivo*, and *in vitro* research that decreasing the dose rate of alpha-particle (radon-progeny daughter) exposure will increase the carcinogenic effect per unit dose. This inverse relationship means that extrapolating from the miner situation to the environmental situation requires an understanding of how the relative risks change with dose rate. In the past few years, *in vitro* studies of oncogenic transformation have led to a considerable increase in understanding of the underlying processes involved. Applying these insights to the radon problem has yet to be done (Brenner, 1994).

The miner epidemiological data clearly show that lung-cancer induction from radon-progeny alpha-particle exposure depends on the exposure rate and that the risk increases as the exposure rate decreases. This phenomenon has been interpreted to suggest that radon risks per unit dose from domestic exposure (which occur at low exposure rates), will be underestimated by risk estimates derived from miner studies (which are at higher exposure rates). On the other hand,

there are approximately two orders of magnitude difference between the low exposure rate in mines at which an effect has been seen and the exposure rate in an average residential dwelling. It is not clear that the inverse dose rate effect can be extrapolated over such a range of exposure rates.

Another variable affecting this phenomenon is the absolute exposure level to radon-progeny. On basic biophysical grounds, when the exposure is sufficiently low such that multiple traversals of target cells are rare, the exposure rate effect should disappear. This is the case for typical domestic radon exposures, but not for most miner exposures. Thus there are additional uncertainties associated with use of risk estimates based on the miner data, where absolute dose rates are higher than dose rates for domestic radon exposures.

These dose rate patterns have been observed in lung cancer induction studies by radon in rats. For example at high levels of cumulative exposure (> 600 WLM; >2100 mJ h/m^3) an inverse dose rate effect has been seen in going from exposure rates of 500 WLM/week to 50 WLM/week (1770 mJ h/m^3-week to 177 mJ h/m^3-week). Exposure rates down to 5 WLM/week (18 mJ h/m^3-week) - comparable to underground miner exposure rates - showed a tapering off of the exposure rate effect (Cross, 1990). On the other hand, in another study rats exposed to 25 WLM (90 mJ h/m^3) at a rate of 2 WL (40μJ/m^3) showed no increase in lung cancer compared to control animals, whereas rats given 25 WLM (90 mJ h/m^3) at 100 WL (2000 μJ/m^3) had about a three-fold excess lung cancer incidence - i.e. there was no evidence of an inverse dose rate effect at low cumulative exposures and, at the lowest rate, there was no increased lung cancer incidence (Morlier et al., 1992). Thus extrapolations from the higher-dose rate/high dose miner data to the lower dose-rate/low dose domestic situation will almost certainly require a model relating effectiveness to dose rate.

3.3.4.3 The Alpha-Particle Quality Factor

There are currently two possible approaches towards estimation of lung cancer risk for exposure to low levels of radon progeny. One is an epidemiological approach, in which lung cancer mortality is assessed in cohorts of underground miners who have been exposed to high levels of radon. The miner results are then extrapolated to the environmental situation. Recent results from epidemiological studies of lung cancer risk from residential radon exposures have not altered the bases for this extrapolation.

A second approach to radon risk estimates is the so-called dosimetric approach. In this approach to radon risk estimation, the radon exposure is related to an absorbed dose to the segmental bronchial epithelium (or any other organ that might be considered at risk), which is then converted to equivalent dose, which is in turn related to risk, via risk factors derived

essentially from the Hiroshima and Nagasaki atomic bomb survivor data; (risks estimates from the atomic bomb survivors are assumed to represent better statistics and dosimetry, and longer follow up than for miners).

An essential step in this process is the conversion from dose to equivalent dose, which requires an assumption about the relative biological effectiveness (for oncogenic endpoints of interest) of low doses of radon-progeny alpha-particles relative to x- and gamma radiation. This conversion factor, known as the quality factor (or, more recently, the radiation weighting factor) is not well known, with estimates varying from 2 to 50. Recent *in-vitro*-based experimental results have suggested a quality factor of ≈ 10 is likely to be more appropriate (Brenner et al., 1995) for radon progeny.

It is important, from the standpoint of credibility, that radon risk estimates derived from the epidemiological approach should be broadly consistent with risk estimates based, through the dosimetric approach, on the atomic bomb data. For non-lung cancer end-points, such as gastrointestinal (GI) cancer arising from the ingestion of radon in drinking water, the use of the dosimetric approach for risk estimation is the only feasible method, as there are no epidemiological data on which one can base GI-tract cancers due to radon ingestion. Comparison of miner-based domestic radon risk estimates with estimates scaled from data on atomic bomb survivors thus represents an important test of the reliability of these risk estimates. However, there is currently a significant discrepancy of about a factor of 3-5 between domestic radon risk estimates generated by these two independent methods.

The quality factor is only one of several uncertainties inherent in extrapolating from atomic-bomb-based data to the domestic radon situation. The other major uncertainty is the correction for dose rate and the dose/dose rate effectiveness factor (DDREF) for alpha particles, as noted in the previous section. Although the DDREF thus far has only been invoked for low-LET radiation (NRC, 1990), there may be valid reasons for considering a DDREF for high-LET radiation also, providing that the doses and dose rates are sufficiently low. The recent French animal study results have a bearing on this issue (Morlier et al., 1992).

3.4 Factors Affecting Concentrations, Exposures, and Exposure Assessment

Radon concentrations are known to vary both spatially and temporally within a specific house, and from house to house even within the same region. In some cases, these variations can be substantial, making the assessment of average concentrations and exposures difficult and uncertain. In addition, because the measurement of radon is a surrogate for assessment of radon decay product concentrations and their associated health risks, these uncertainties are reflected in

the assessment of the risks. The lung deposition of the radon progeny and the associated radiation dosimetry depend upon the size of progeny (see, for example, the discussion of dosimetry in NRC, 1991). The size and concentration of the airborne progeny species - sometimes referred to as 'unattached' or 'attached'- are, in turn, strongly dependent on the concentration and size distribution of the indoor aerosols. These co-contaminants in indoor air are themselves variable both spatially and temporally, adding further uncertainty to the overall concentration-exposure-risk assessment. In order to have a comprehensive understanding of the uncertainties affecting the radon risk estimates, additional research is needed in critical areas of radon measurement and in the application of those measurements to making assessments of exposure for individuals and for the general population.

One goal of the research described in this section is to improve quantification of the uncertainties ultimately associated with assessing the risks from radon progeny exposures. Such uncertainties are inherent in defining the risks to populations or to individual members of the population. Another goal is to develop methods to locate those individual members of the population with the greatest risk. Although reduction of such risks has a negligible impact on the overall population risk, the role of uncertainty is less important in these individual cases where average indoor radon [progeny] concentrations equal or exceed levels at which occupational exposures are regulated (0.1 WL (2 $\mu J/m^3$) or about 20 pCi/L (740 Bq/m^3) (U.S. CFR, 1990).

3.4.1 High Radon Regions

Indoor radon concentrations vary from house to house. The sources of this variability are complex and often interrelated. The characteristics of the soil and the underlying geology help determine the distribution and concentration of the source term and the ability of radon to migrate into houses. The building substructure type (e.g. basement, slab-on-grade or crawlspace), the nature of the building-soil interface, and meteorological conditions are important factors in determining radon entry into buildings. Some studies have shown an association of indoor radon concentrations with specific geological parameters, such as surficial radium concentrations, although most of these studies have been limited to just one or a few parameters.

3.4.1.1 Identification of Regions with the Greatest Potential for Elevated Indoor Radon Concentrations

There appear to be characteristics common to homes in which radon concentrations are highest. One of the most prominent is that these homes are constructed in areas where geologic and soil conditions contribute greatly to the radon source term and to radon transport through the soil and entry into buildings. Meteorological conditions also play an important role in developing the pressure differences between the inside and the outside of a building, leading to advective flow

of radon-bearing soil gas into the building. These various factors are often interrelated, making the task of sorting out the relative importance of these influencing factors difficult. Nevertheless, the value of identifying radon-prone areas has been recognized (e.g., ICRP, 1993).

A general evaluation of radon risk at a county-level for individual states has been completed in a cooperative effort between EPA and United States Geological Survey (USGS) and used as input for developing building codes (Schumann, 1993). Currently, refined studies are being coordinated between EPA, Lawrence Berkeley Laboratory, and USGS to identify, at a sub-county scale, the specific characteristics that have the greatest influence on radon potential. Data to be examined will include soil, house, and geological features. The second stage is to develop a predictive model based on the associations developed from the initial data examination. The model would then be tested in various regions of the country using available data bases, including radon measurement data. A refined model could then be used, limited by the availability and resolution of input data, to locate those regions with the highest radon potential. The objective is to define those regions in which a manageable, yet intensive follow-up radon testing program could be used to help find the individual houses with highest concentrations.

3.4.2 Measurement and Interpretation Methods

Risks associated with radon (as a surrogate for radon decay product concentrations) are based on the total integrated exposure - essentially the product of concentration and the time spent at that concentration. Lifetime exposures will be the sum over many exposure conditions, integrating across spatial and temporal factors, including home, school, and workplace exposure environments. This concept in part, has been the basis for previous Radiation Advisory Committee recommendations that the most accurate assessment of exposure, and hence risk, will be based on long-duration, time-averaged concentrations (see, for example, reports 18, 24, 26 and 35, listed in Appendix A). The Radiation Advisory Committee has recognized that practical considerations sometimes argue against a long testing period, and the committee has provided recommendations applicable to short term testing.

A number of recently published studies have consistently indicated that for tests performed in individual houses, results of short-duration measurements do not provide accurate predictions of long-term concentrations (Condon et al., 1990; Harley et al., 1991; McDonough et al., 1991; Steck, 1990; Klotz et al., 1993; White, et al, 1994). Ratios between the short-term measurement of concentration and the long-term measurement range from less than 1 to more than 5, depending upon the season in which the short-term measurement was conducted and its location within the house (basement, first-floor, etc.). The data presented in two such studies, comparing two-day radon measurements conducted with charcoal canisters with longer-term alpha-track

detectors (ATD) measurements, further suggest that the ratios of the test results are concentration dependent (Klotz et al., 1993; White et al., 1994).

That there is a significant difference between short-term and long-term measurements is also indicated by comparing the results of the National Residential Radon Survey (NRRS), where 6 percent of the homes exceeded 4 pCi/L (Marcinowski et al., 1994; U.S. EPA, 1992a), with the results from short-term screening measurements made as part of the surveys conducted in approximately 30 states, where nearly 21 percent of the homes exceeded 4 pCi/L (150 Bq/m^3) (White et al., 1992). A difference of about a factor of 3 in short-and long-term measurement results has also been noted by the EPA (U.S. EPA, 1989), which cautions that "Short-term tests and long-term tests should be interpreted differently."

3.4.2.1 Accuracy and Precision of Radon Measurement Devices and Protocols

The Radiation Advisory Committee has previously recommended that EPA conduct studies directed toward improving both the precision and accuracy of the various measurement methods, testing protocols, and interpretive procedures (report 24 in Appendix A). In this context, the Committee has noted that the measure of accuracy is provided by comparison with a long integration time concentration measurement. Precision, on the other hand, would capture both the inherent precision of a particular test device when multiple measurements are made under identical conditions and the variability in concentrations over short period of time.

A key goal would be to determine what the accuracy criterion is or should be, in light of the available measurement methods and the variability in long-term radon concentrations (see, for example, Martz et al., 1991). Such a criterion might be concentration dependent, reflecting variations in device sensitivity and limits of detection, and the need for greatest accuracy (least uncertainty) for concentrations in the vicinity of the EPA guideline. Improvements in the accuracy of long-term measurement methods also appear to be possible, based on reports of variability in device-to-device response (Yeager et al., 1991b; Pearson et al., 1992).

Methods for interpreting short-term measurements deserve further investigation, with the goal of improving the utility of the measurements, either as estimators of longer-term average radon concentrations (hence providing a better basis for mitigation decision-making) or as a means of deciding whether further testing is needed. One obvious possibility would be to examine whether a statistically robust set of 'correction factors' could be developed that would permit adjustments for sampling season, measurement location within the house, and across different climate zones.

Studies conducted as part of the Florida Radon Research Program, jointly sponsored by the EPA and the State of Florida, suggest that it might be possible to establish threshold values for

short-term radon concentration measurements. These thresholds, which are device and protocol dependent, would provide either an upper or lower limit for the measured concentration (for a given statistical confidence level and desired average long-term concentration guideline). Measurements falling below the lower threshold would thus be interpreted as predicting that the long-term average radon concentration will be below the guideline concentration (e.g. 4 pCi/L; 150 Bq/m^{3}) and, conversely, measurements above the upper threshold could be reasonably interpreted as indicating that the long-term average concentration is above the guideline (see McDonough et al., 1991; Roessler et al., 1990). Based on RAC recommendations, the Agency has proposed a study to address many of these concerns.

3.4.2.2 Adequacy of Sampling Methods for Radon in Drinking Water

Little is known about the variability of radon in groundwater over time, especially under situations of variable use, as might be the case for a private well supplying domestic water for a residence. Most assessments of radon in water are based on grab samples, although there is little information in the literature about the extent of temporal variability. The adequacy of grab samples for analyses of radon in water should be studied, including the recording of temporal variations of radon and radium concentrations in well water over daily, weekly, and seasonal cycles. The study should also evaluate the potential for loss of radon during sample collection, as well as other factors that might produce either false positive or false negative results (see, for example, Burkhart et al, 1991). Some important areas of study regarding sampling and analytical methods include minimum flushing times and comparisons of liquid scintillation and emanation methods (see Vitz, 1991).

3.4.3 Factors Affecting Total Exposure and Dose

A major problem arises in the use of the exposure-response results obtained from the epidemiology of underground miner populations for estimating health risks in the general population because of the limited amount of information on the conditions that existed in the mines during the exposures. Similarly, there is a lack of current data characterizing exposure conditions in homes, schools, and workplace environments (NRC, 1991). As noted earlier, the sizes and concentrations of indoor aerosols have a particularly important effect on the activity-weighted size distributions of the radioactive aerosols conferring the risk. These factors also affect the equilibrium factor often used to associate radon decay product concentrations with the underlying concentrations of the radon parent. An equilibrium factor of 0.5 is often used in this association and in estimates of risk (Puskin, 1992; U.S. EPA, 1992d). Characterizing the variability in these conditions will more accurately portray indoor environments and will reduce the uncertainties of the risk estimates.

3.4.3.1 Exposure to Radon Progeny in Mine and Indoor Atmospheres

There have been efforts over the past several years to measure the behavior of the indoor aerosol, both radioactive and non-radioactive, in normally occupied homes, and new systems have been developed to measure activity-weighted size distributions (Hopke et al., 1992a). One system has been used to measure over 500 activity-weighted size distributions in 8 normally occupied homes and 2 unoccupied houses in which particles are generated (Li and Hopke, 1991a; Hopke et al., 1992a, 1992b; Hopke et al., 1993, 1994; Wasiolek et al., 1992, 1993). The non-radioactive aerosol size distribution measured every 3 minutes in 4 occupied homes in eastern Pennsylvania yielded approximately 30,000 size distributions from which the indoor radioactive size distribution can be estimated (Knutson et al., 1993). However, all of these occupied houses are in the northeastern United States and do not fully represent the full range of housing stock and indoor aerosol conditions within the U.S. A systematic survey of homes covering the range of major indoor aerosol sources, including indoor and outdoor combustion sources, cooking, etc., needs to be performed to assess the range of exposure/dose relationships that might be applied to the estimation of the population dose that results from the exposure to indoor radon.

Although there is an insufficient set of measurements to make population estimates of the exposure/dose relationships within the U.S. housing stock, even fewer data from measurements of mine conditions are available. With the closure of uranium mines in the U.S. and the change in the conditions under which mining took place over the past 30 years, it is not possible to make measurements under conditions similar to those that existed in the early Colorado Plateau mines. Although there has been reanalysis of the measurements made in mines of the Grants mineral belt in New Mexico in the early 1970s (Knutson and George, 1992), it is not possible to retrospectively apply modern measurement technology to the mine atmospheres to which the U.S. miner cohorts have been exposed. However active mining continues in the Elliot Lake region of Ontario and some of the mines in which other underground miner cohorts worked are still accessible. Thus, it may be possible to assess modern mine conditions. Some of these measurements could help to provide important input for comparative dosimetry between homes and mines. It is important to determine the feasibility of making such measurements.

In addition there needs to be a series of measurements of the typical conditions found in workplaces other than mines. There may be situations in which the nature of the work generates particles that significantly change the exposure/dose relationship. Currently there is no information available on either industrial or non- industrial radioactive aerosols although it may be possible to make some estimation based on industrial hygiene measurements of size distributions in the workplace. It may be worthwhile to search the industrial hygiene literature to identify specific workplace aerosol characteristics from which the attached radon progeny activities can be estimated. An effort in this area should be initiated.

An ancillary problem regarding the exposure/dose relationship for both indoor and mine atmospheres is the determination of the particle growth that occurs when particles are exposed to the >99% relative humidity present in the human respiratory system. Measurements of hygroscopic growth of a series of combustion particle sources has recently been reported (Li and Hopke, 1993) as has the results for a series of consumer products that generate aerosols (Li and Hopke, 1994). Additional materials that are found in typical indoor situations are currently under study. However, analogous studies of the hygroscopicity of mine aerosols have not been done and represent an additional source of uncertainty in the extrapolation of the underground miner risk estimates to the indoor atmosphere.

3.4.3.2 Contribution of Radon-220 to Radiation Exposures Indoors

Radioactive decay products of radon-220 (often called 'thoron' because it arises as part of the radioactive decay series that begins with naturally-occurring thorium-232) are thought to contribute 10 to 15% of the effective dose equivalent attributed to radon (UNSCEAR 1988). However, very few measurements of radon-220 or its decay products in indoor environments have been reported (see, for example, Schery 1989; Reineking et al., 1992; Martz et al., 1990). One important issue is the contribution of various sources to the indoor concentrations. Because radon-220 has a short, 55-second half-life, its transport is limited and diffusion has been thought to be the dominant mode of migration. However, evidence that advective flow of soil gas may also be an important contributor has recently been reported (Schery 1989; Martz et al., 1990). A related question is whether radon-220 concentrations are correlated with radon-222 concentrations, particularly if advective transport is a significant contributor. Some evidence for correlation between radon-222 radioactive decay products and those from radon-220 has been reported, but overall, the data are equivocal (see discussion in Sextro, 1994). Better information on the sources of radon-220 will also have implications for the choice of control method, as discussed in section 3.6.

3.5 Ingestion of Radon in Drinking Water

Concerns have been raised about exposure to radon from drinking water (U.S. EPA, 1991). Potential health risks include lung cancer associated with exposure to airborne radon released from the water as it is used indoors and gastrointestinal (GI) cancer due to ingestion of radon remaining in the water. A recent risk analysis suggests that the risk for GI cancer from ingested radon accounts for about 1/3 of the total risk (per unit radon concentration in water), while the risk for lung cancer resulting from airborne radon released from household water use accounts for about 2/3 of this risk, based on the distribution of the uncertainties (U.S. EPA, 1993a). The uncertainty analysis shows that the overall uncertainties associated with the estimates of GI cancer are much larger than the uncertainties associated with the lung cancer estimates, and unlike lung cancer, the direct evidence for GI cancer is very weak. Even though the overall risks associated with radon ingestion are small, the Subcommittee has noted further research that could reduce the attendant uncertainties, thus improving the scientific basis for regulation.

3.5.1 Gastrointestinal and Non-Gastrointestinal Cancer

Unlike radon progeny-induced lung cancer where a causal relationship has been established in animals and humans (NRC, 1988), evidence supporting the risk of GI cancer development following radon ingestion is much more limited (Crawford-Brown, 1990, 1991; U.S. EPA, 1993a). The main basis for concern regarding stomach and other GI cancers is theoretical.

Recent calculations based on biokinetics studies using a xenon model suggest that a significant component of the ingested radon is distributed to various GI organs (Crawford-Brown, 1990, 1991; Correia et al., 1987).

With regard to non-gastrointestinal effects, the biokinetics studies of radon gas transport suggest that radon reaching the intestine would be absorbed into the bloodstream, circulated to all organs and rapidly exhaled by the lung. The liver is assumed to receive a radon dose comparable to the lung. Some of the radon absorbed by the lung is also distributed to other organs prior to recirculation back to the lung and subsequent exhalation. These studies (see Crawford-Brown, 1990, 1991) suggest that the biological half-life of radon in the body is brief, between 30 and 50 minutes.

Additional research should focus on *in vitro* and *in vivo* mechanistic studies. However, epidemiological research could also provide insight into these alternative endpoints. This research could be comprised of both new study designs and a careful review of existing epidemiological databases.

3.5.1.1 Stomach Dosimetry

Experimental evidence is needed to evaluate the dosimetry of the stomach, especially the lumen to capillary gradient (Crawford-Brown, 1990, 1991; U.S. EPA, 1993a). The assumed gradient makes about a factor of 3 difference in the stomach dose calculation, and verification of the assumption is important to decrease uncertainty in estimating dose. Additional evaluations of the intestinal dosimetry should be performed in concert with the stomach dose evaluation and should account for the tissue heterogeneity in the GI system.

The research should include additional information on radon ingestion biokinetics studies, including the addition of mass balance determinations that would include measurements of exhalation of radon. These detailed biokinetics studies should include a careful calibration of the measurement system, that is, the relationship between the measured image density and the activity in the organ. Present calculations of ingestion doses are based primarily on a single study of the biokinetics of radon, and verification of the results of that study is needed. Other biokinetics studies should be conducted to explore the dose/distribution issues relating to "real world" complex exposure scenarios (multiple exposure pathways and multiple radioisotope elements) and to investigate other factors, such as age dependence.

3.3.1.2 Epidemiological Evidence for Stomach and Other Gastrointestinal Cancers

A review of data bases of both mining and selected general populations exposed to high radon/progeny would be useful to examine evidence for an increased risk of stomach and other gastrointestinal cancers that have been postulated by some modeling studies (Crawford-Brown, 1990, 1991; U.S. EPA, 1993a). Only a few epidemiological studies have provided very weak, suggestive evidence of the association between radon exposure and gastrointestinal cancer. A study by Hodgson and Jones (1990) identified an elevated but not statistically significant rise in stomach cancer among UK tin miners, Lawter et al. (1985) found significant excess stomach cancer mortality among Finnish-born Minnesota underground and above ground hematite miners. A third, albeit ecological, study found significant differences in sex-specific, age-adjusted stomach cancer mortality rates among New Mexico counties with significant deposits of uranium (Wilkenson, 1985).

Additional studies are essential, however, to support a hypothesis that these excess stomach cancers observed are related to environmental radon exposures, as there is some evidence that the excess stomach cancers may be attributable to mining in general, For example, an elevated incidence of stomach cancer was noted by BEIR IV (NRC, 1988) among some non-uranium-mining populations (Enterline, 1972; Muller et al., 1985, 1986). Chen et al. (1990) noted no excess stomach cancer in hematite miners exposed to elevated radon levels and who had increased incidences of lung cancer. The Committee acknowledges that conducting new epidemiological studies to examine the relationship between radon exposure and stomach cancer may be very difficult.

3.6 Exposure/Dose/Risk Reduction Methods

Methods to reduce radon or radon progeny concentrations, as with other indoor pollutants, rely on either source control or, once dispersed into the indoor environment, reduction of concentrations by removal methods. Such techniques can be applied either on a retrofit basis or incorporated directly as part of new building construction methods. Reduction of health risks associated with radon is generally approached through reducing concentrations of radon, or in some cases, by reducing radon decay product concentrations directly. These methods lead to reduction in exposures to radon progeny; reduction of exposures to other pollutants thought to be co-carcinogenic or synergistic, such as tobacco smoke, appear to reduce radon-associated risks as well, although there is little detailed information about these effects.

Although there is a growing body of both laboratory and 'as practiced' field data on the effectiveness of radon control methods, there are few data on the efficacy or practicality of many of the techniques over the lifetime of the application. Many of the retrofit applications, for example, rely on mechanical (fan-powered) systems for which there is little information on system longevity.

3.6.1 Source Control for Retrofit and New Applications

It is generally understood that the soil adjacent to the building substructure is the predominant source of radon in buildings and that advective flow of radon-bearing soil gas is the principal means of entry for those buildings in which indoor concentrations exceed 1 - 2 pCi/L (37 - 74 Bq/m^3). The most important exception to this general rule are those buildings -- principally dwellings served by individual ground-water wells -- in which radon-in-water concentrations are elevated, leading to elevated concentrations in indoor air. (As a rule of thumb, the ratio of radon concentration in water to the resultant concentration in air is about 10,000 to 1 for a typical dwelling.) Transport of radon by diffusion either from or through building materials is typically small and, when combined with usual building ventilation rates, does not yield elevated indoor concentrations. On the other hand, attempts to reduce indoor concentrations below 1 to 2 pCi/L (37 - 74 Bq/m^3) will likely require that other radon entry mechanisms, such as outdoor air and diffusive sources of radon, be addressed.

Control of radon entry into buildings generally relies on reducing the size and/or number of flow paths through the substructure and into the building interior along with a means to reverse the natural pressure gradient between the building exterior and interior. Sealing techniques are often not sufficient by themselves, but can greatly enhance the efficacy of control of the pressure gradient. In existing houses, retrofit techniques are sometimes limited by the inability to successfully control (reverse) pressure-driven flow at all entry points. In new buildings, radon resistant techniques can be incorporated directly into the design and/or construction process.

3.6.1.1 Durability, Long-Term Performance, and Adverse Effects of Active Mitigation Systems

The use of active components, such as fans and pressure alarms, in radon mitigation systems is wide-spread. However, there is currently very little information on the durability of these devices (which typically run continuously lest the homeowner forgets to turn the device back on); what little data are available suggest that the failure rate for fans and fan components is fairly high (Scott and Robertson, 1990; Yeager et al., 1991b). (System failure warning devices are available to alert occupants if a system is not working properly.) Follow-up testing on previously installed mitigation systems should be undertaken to provide information on the long-term reliability of active system components. In addition, there is some evidence that the performance of the mitigation system may change with time, due to changes in the air permeability of the underlying soil, to the settling of the house, or to other features (Dudney et al., 1990; Harrje et al., 1990; Roessler et al., 1992; Prill et al., 1990).

In addition to the changes in the performance of the mitigation system, long-term operation of a mitigation system could lead to structural or soil changes. Basement pressurization has been used in some installations to prevent advective radon entry. Typically, these systems supply conditioned air from the first floor; such air contains moisture which could condense on cold exterior building surfaces, potentially leading to structural damage. Subslab depressurization draws air through the soil, exhausting it out the top of the vent stack. This air flow could cause additional drying of the soil, particularly that below the slab which is shielded from rainfall. It is not known whether such changes in moisture content might lead to changes in the bearing strength of the soil, resulting in structural changes in the building. Large changes in soil moisture could also change the operation of a subslab mitigation system or in the transport potential of soil gas and radon. Research is needed to help define the potential for such changes.

Radon entry into buildings often depends upon features of the building-soil interface, such as cracks or gaps in the building shell, the permeability of the soil immediately adjacent to the opening, etc. High permeability layers, such as an aggregate below a slab, are often desirable to increase the efficacy of active mitigation systems. However, such layers have been shown to enhance radon entry in situations where no active mitigation system is present (Revzan and Fisk, 1992; Bonnefous et al., 1993; Robinson et al., 1993). Buildings change with age or even seasonally with changing temperature and moisture conditions in the soil or in the building structure itself. No systematic information is available to aid in determining whether these changes might defeat or compromise radon mitigation systems or radon-resistant construction features in new buildings. Critical to this understanding is whether most of the important changes due to aging occur within the first year or so after construction, or continue to occur for the life of the building. The development of a strategy for control and the adoption of design elements could depend upon this understanding.

There is some evidence that active depressurization systems can cause or exacerbate existing combustion appliance backdrafting, leading to spillage of combustion products indoors (Wiggers, 1992). Backdrafting is recognized as an important issue in cold climates, even without the presence of a radon mitigation system (Swinton and White, 1986; Moffatt, 1986). Measurements of air sources and flows in subslab mitigation systems have indicated that, in some cases, more than half the air flowing out of the active system originates in the basement (Clarkin et al., 1990). Although there are several possible methods to ameliorate these conditions, considerations of comfort and energy conservation complicate the problem. Further examination of this problem is warranted due to the potentially serious nature of appliance backdrafting.

An important distinction between most residential and non-residential buildings is the presence of mechanical heating, ventilating and air-conditioning (HVAC) systems in the latter. A variety of mechanical ventilation systems are used in non-residential buildings and, in several cases, the

operation of these systems has been linked to occurrences of elevated radon concentrations (Leovic et al., 1990; Saum et al., 1990). These conditions can arise when the mechanical system is exhausting more air than it is supplying; such imbalanced systems can also adversely affect other indoor air pollutant concentrations in addition to radon, because ventilation flows are often lower than needed to maintain acceptable concentrations (Brennan et al., 1992). Restoring proper balance between the supply and exhaust flows has often led to reductions in radon concentrations and to general improvements in indoor air quality. However, before this approach can be relied upon to provide consistent and stable control for radon, additional research is needed on identifying the source and frequency of the various failure modes, particularly with respect to the maintenance of system performance and the factors affecting pressure imbalance leading to enhanced radon entry.

3.6.1.2 Passive Radon Mitigation Methods

In principle, purely passive techniques to limit radon entry into buildings offer a number of advantages, probably the most significant of which is that no further attention need be paid to the system once it has been installed in an existing building or incorporated as part of the design and construction of a new building. There is, in addition, no direct or indirect energy penalty (as there is with an active, fan-powered system). Such systems have seen only limited application and only limited success (see review in Henschel, 1993). The operating parameters and conditions have not been sufficiently explored to determine under what circumstances such a system might be successful and the long-term system performance has not been established.

Although diffusive radon entry is usually regarded as a negligible source of radon in buildings with elevated concentrations, it is potentially a much more important source for buildings with concentrations below 1 or 2 pCi/L (37 or 74 Bq/m^3), particularly if soil radium or radon gas concentrations are considerably elevated above typical values. Building materials themselves usually contribute little to the total radon diffusive flux density; rather, most comes from radon in the surrounding soil. However, it is the diffusivity of the materials themselves - usually concrete in contact with the soil - that retards radon migration. Observations of high concrete diffusivities have been reported (Rogers et al., 1994). Techniques to reduce the diffusivity of building materials or elements in contact with the soil should be investigated. Another approach would be to evaluate techniques to limit soil radon concentrations adjacent to the building substructure.

3.6.1.3 Radon-Resistant New Construction

As with existing buildings, limiting radon entry in new buildings can be achieved with either passive or active methods. Passive techniques, including barriers to entry and methods to reduce the differential driving pressures, may have the greatest applicability in new construction.

Application to new buildings offers a number of advantages, in principle, where a number of features can be combined at the design and construction stage, such as barriers to advective soil gas flow, passive stacks to reduce or reverse the pressure gradient across the building shell and vents to supply low-radon concentration air to the subslab region. However, very few houses have been built incorporating such passive features, and more research is needed to provide information on the dynamic behavior of the house and control system. Experimental studies combined with parametric modeling could help elucidate the performance parameters and help determine under what conditions application of a passive approach would be successful.

Active (i.e., fan-powered) systems can also be incorporated in new construction. Experience with retrofit systems has indicated that two of the most important considerations for successful use of such systems are good subslab communication (usually achieved with a subslab aggregate layer) and a floor slab in which cracks or other penetrations have been sealed (Henschel, 1993).

3.6.1.4 Control of Radon-220

As noted in the discussion in section 3.4.3.2, radon-220 and its radioactive decay products are estimated to contribute 10 to 15% of the average effective dose equivalent attributed to radon exposures. Very little is known about the occurrence or the range of concentrations of either radon-220 or its decay products indoors. However, to the extent that it may pose a problem in some situations, research on control of these concentrations should be undertaken. There is some evidence that subslab mitigation systems provide some control of radon-220 entry into buildings (Li et al., 1992). However these results are only for a few houses and not all of the mitigation systems examined performed similarly. Ventilation methods will help reduce indoor radon-220 progeny concentrations but will not have an effect on radon-220 concentrations due to its short 55-second half-life.

3.6.2 Radon and/or Radon Decay Product Reduction Methods

Beyond the source control techniques discussed above, indoor radon concentrations are determined by the balance between the source term and the removal term, which in most cases is the ventilation rate. Ventilation also affects the radon progeny concentrations and may, under certain circumstances, affect progeny concentrations differentially, though at ventilation rates typical of buildings, the equilibrium conditions between radon parent and the subsequent radioactive progeny are not altered significantly. There are practical considerations -usually related to comfort and to energy use - that limit ventilation. In addition, natural ventilation is driven by the same interior-to-exterior pressure differences that drive radon entry. Mechanical ventilation systems, such as those found in non-residential buildings, may produce additional pressure differences, depending upon the balance between supply and exhaust air flows.

Other techniques have been proposed, including sorption of radon on carbon filters (Wasiolek et al., 1991) and removal of airborne radon progeny by air cleaning. Previous investigations have demonstrated that air cleaning systems can effectively remove radon decay products from indoor air. Reductions in the activity concentrations can be in the 60 to 80% range (Hopke et al., 1990). However, it is important to recognize that the decay product concentrations are not the only factors that determine the dose that the progeny deliver to the cell of the bronchial epithelium. Particle size is also a critical factor in the evaluation of the exposure/dose/risk relationship. The decay products associated with aerosols in the smallest size range (the ultra-fine or so-called "unattached" decay products) (Hopke, 1992) have much higher mobilities in the air and can more effectively deposit in the respiratory system. Thus, reductions in radon decay product concentrations do not necessarily translate into similar reductions in dose.

Because airborne radon decay products can readily attach to particles present in the indoor air, the effects of air cleaning are complicated. At the same time the air cleaner removes the radon progeny from the air, the airborne particles are also removed. Since the radon progeny are continually formed from the radioactive decay of radon, the fraction of ultrafine radioactive aerosols will increase as the particles are removed. Although there are fewer total airborne decay products, they may be more effective in depositing their radiation dose to the lung tissue. In some cases, doses were estimated to increase in spite of a lower airborne radioactivity concentration (see Sextro et al., 1986; and review by Hopke et al., 1990).

3.6.2.1 Radon Concentration Control

The ventilation rate of a building is an important controlling element for indoor radon concentrations. Some energy conservation measures are designed to reduce energy losses by reducing "uncontrolled" natural ventilation. In retrofit situations, such weatherization measures may reduce ventilation rates by about 20 percent; in new construction even larger changes are possible, achieving a factor of two or more reduction. In some cases, such as the Model Conservation Standards adopted in the Pacific Northwest, mechanical ventilation is sometimes used to supplement the natural ventilation rate in order to ensure that other indoor air pollutant concentrations remain low. The available evidence suggests that the resulting reduced ventilation rates in either new or existing houses do not necessarily lead to increased radon concentrations, in part because of changes in the driving forces for both ventilation and for advective radon entry (Turk et al., 1987; Siniscalchi, 1990; Siniscalchi et al., 1990). However, additional data are needed to understand these interactions in detail, particularly in formulating strategies to reduce radon concentrations in new houses through changes in building codes and practices. The growing use of energy conservation measures in new house construction should provide a source of data on the interaction of energy conservation, ventilation and radon entry in new houses. Such

studies should include housing in which mechanical ventilation is used to supplement the (low) natural ventilation rate.

In non-residential buildings, ventilation is usually provided by a mechanical heating, ventilating and air-conditioning (HVAC) system. When performing as designed, most HVAC systems can maintain ventilation rates that will keep radon concentrations (and usually other potential indoor contaminant concentrations as well) low. However, such systems can become imbalanced when not properly maintained, resulting in both reduced flow rates for ventilation air and, as noted earlier, increased building depressurization, thus potentially increasing the radon source term. Further research on the frequency and causes of system failures or performance degradation would be of considerable benefit in designing new systems and in maintaining existing installations.

3.6.2.2 Airborne Radon Decay Product Removal

Recently, a number of studies have reported on the use of air cleaners to mitigate the health effects from radon decay products in indoor air. These studies have measured both radon concentrations and the activity-weighted size distribution of the radon progeny in realistic environments (Li and Hopke, 1991b, 1992; Hopke et al., 1993, 1994). In this way the dosimetric implications of the changes in size distributions that are induced during air cleaner operation can be more fully evaluated.

The reduction in exposure and dose observed in studies of the effect of air cleaning suggests that these systems may have some utility in homes where the radon concentration is no more than 2 times the action level at which radon mitigation is recommended and could provide some assistance as an interim remedy while radon entry reduction techniques (i.e. subslab depressurization) are being constructed. However, these air cleaners do not appear to be capable of a sufficient level of dose reduction that they could serve as a primary system for the mitigation of the health risk arising from the exposure to radon decay products in indoor air. It may be possible to increase the effectiveness of room air cleaners through optimization of their role in the plateout of the ultrafine particle progeny as well as through new designs in air cleaners (Loreth and Torok, 1993; Torok and Loreth, 1993; Ilmasti and Raunemaa, 1993) and their optimization would be useful, but it appears that the utility of this approach will be limited to lower concentration situations. Further study of the performance of air cleaners in reducing airborne radon progeny concentrations and the related reductions in estimated dose would be useful, especially in optimizing the depositional removal of the ultrafine radon progeny aerosol.

3.6.3 Reduction in Water-Borne Radon Concentrations

An increasing number of homeowners who rely on private wells as their source of drinking water are evaluating their water for the presence of radon. Removing radon from the well water before it enters the house is the most effective treatment. The type of method used depends upon the radon concentration in the well water and the volume of water used by the household. Two radon removal methods are generally used, adsorption of radon by granular activated charcoal (GAC) and water aeration. Each has its advantages and disadvantages and further research or engineering design will be useful.

3.6.3.1 Removal of Radon From Domestic Water

Some mitigation contractors use GAC to reduce radon in water levels up to 20,000 pCi/L (740 kBg/m^3). The mitigation industry now offers a confusing array of GAC filter systems ranging from single tanks of different height/width ratios to multiple tank systems. Recommendations vary as to the appropriate frequency for changing of the filter medium. Additional data are needed to determine the most cost-effective combination of tank shape/number and filter changing frequency. The efficacy of the various styles of GAC filtration systems in reducing radon and radium in water concentrations needs to be documented. Consideration must also be given to the build-up of radon decay products on the GAC and the resulting radiation field around the GAC, as well as the disposal of used GAC.

Use of aeration treatment systems for reduction of radon concentrations in household well water is also increasing. Homeowners are offered a wide variety of treatment systems. Unfortunately, radon removal efficiency of some of these systems is low (Dembek et al., 1993). Moreover, some aeration system installations have actually resulted in higher indoor radon concentrations (Bell and Dembek, 1991). The section on measurement of radon in water (see Section 3.4.2.2) discusses the problems encountered in evaluating wells with significant temporal variation in radon concentrations (Dupuy et al., 1992; McHone et al., 1992; Siniscalchi et al., 1992). Mitigation systems must be designed to effectively treat wells with these significant temporal variations (Dembek et al., 1993). The use of supplemental treatment systems for uranium and radium mitigation also need to be evaluated. More studies are needed to identify the most effective design, and to reduce the cost of this expensive treatment technology.

3.7 Risk Communication and Evaluation

To date, research on risk communication at the U.S. EPA has been scattered throughout the agency. One major, ongoing, cooperative agreement at Rutgers, "Communicating Effectively about Risk Magnitudes", has produced a series of studies and reports related to the perception

and communication of radon risk (Sandman et al., 1994; Weinstein, 1988; Weinstein and Sandman, 1991, 1992; Weinstein et al., 1988, 1989a, 1989b, 1990, 1991a, 1991b). Currently, the Risk Communication project in EPA's Office of Planning and Policy Evaluation (OPPE) is putting most of its effort into two large projects: a) "A Research Agenda for Risk Communication in Government Agencies" also called "Defining practitioners' risk communication needs," (Chess et al. at Rutgers) and b) "Explaining Uncertainty in Health Risk Assessment" (Slovic et al. at Decision Research). The first project has provided much pertinent information for EPA risk communication research priorities but has not yet concluded (the proceedings from a workshop funded under this project are forthcoming in *Risk Analysis*). The second project has examined what happens with the perceived credibility of information sources when uncertainty in estimates is discussed. (Johnson and Slovic, 1994)

The agency's decision to assign high priorities to these research topics is laudable and is consistent with the specific research recommendations that follow. These projects have the potential to help illuminate the perceived conflict between the primary candidate goals for radon risk communication at EPA: a) persuading the public to test, and to mitigate if high levels of radon are found, b) educating people to enable them to make decisions about radon that are in their own best interest, and c) facilitating participative decision making. The Subcommittee recommends that EPA increase its reliance on empirical evaluations, increase opportunities for public participation, and increase use of peer review from the scientific community, to address effectively the challenges described below.

3.7.1. Communicating Technical Information and Scientific Uncertainty

Choosing what information to include in a risk communication is a matter of balance and focus (Atman et al., 1994). Too much information can be as devastating as too little for the recipient (Fischhoff, 1987). The effectiveness of risk communication is limited by the attention and interest of the public, as well as by how people process information. These raise several important questions regarding the communication of scientific uncertainty and technical information. What criteria should be used for deciding when to report scientific uncertainty? What effect does communicating scientific uncertainty (quantitatively or qualitatively) have on: (a) agency credibility and public trust, (b) public understanding, and (c) testing and mitigation?

A recent study of the role of technical information (Johnson et al., 1992) highlights some of the controversies surrounding technical content choices for risk communications. Communicating accurate scientific information has often been equated with supplying facts, often with much detail and sometimes with great complexity. However research findings in this area should be interpreted cautiously, as information that is technical according to one definition may not seem technical according to another, or be perceived as technical by the audience.

There is anecdotal evidence that people say that they do not intend to do anything about a specific risk, such as radon, because there is too much scientific disagreement, uncertainty, or ambiguity about the risk, relative to the amount known about other, presumably graver, risks. Thus it is tempting to conclude that information cannot be complete (that is, include technical details or information about scientific uncertainties) if it is to be persuasive, and that complete information (that is, including uncertainties) will not be persuasive. However, completeness can be defined with respect to many goals (see below). More importantly, systematic research on this question is sparse. As stated above, research currently sponsored by EPA addresses some of these issues. There are, however, multiple aspects to this perceived conflict. If an agency's risk estimates are reported as reliable, but are changed over time, the agency's credibility may suffer. On the other hand, conveying the uncertainty in risk estimates may require communicating more technical information, which may in itself influence risk perceptions, decisions and actions.

3.7.1.1 Communication of Technical Information

Some research results support the use of more detailed, or specific, information. In the Johnson et al. (1992) study, what the researchers intended as technical detail was not necessarily perceived by the participants as technical detail, nor was what the researchers intended as technical detail correlated with perceived risk. Perceived technical detail does correlate positively (if weakly) with perceived risk and testing intentions. Weinstein et al., (1990) find that public concern and intention to test are increased by mentioning a) that the specific area the subject lives in is a high-risk area, and b) the high fatality rates for lung cancer and the relative seriousness of radon as compared to other environmental hazards. These pieces of information are likely to increase the specificity of the general knowledge people already have (e.g., that radon is dangerous). Thus, in this study (Weinstein et al., 1990) more specific information was used effectively to create a sense of threat (technical difficulty was not a focus of this research). A study by Leferman Associates (1990) finds that people prefer more specific information about health risks from radon (i.e., that exposure can cause lung cancer) over more general information (i.e., that one can die from exposure to radon). However research in this area is limited and studies should be undertaken to further explore the use of detailed technical information.

3.7.1.2 Communication of Scientific Uncertainty

Uncertainty is inherent to any risk management process, including risk communication. Morgan and Henrion (1990) classify uncertainty in empirical quantities by source, all of which are relevant for radon risk communications: statistical variation, subjective judgment, linguistic imprecision, variability, inherent randomness, disagreement and approximation. In a multistage process, such as the radon testing and mitigation process, uncertainty can play very different roles at different stages. For example, EPA may recommend unconditionally that everyone who dwells

at or below the second story of their dwelling test for radon, but still convey that results from a single passive, short-term test is relatively unreliable and should be checked with a second, longer test. Scientific uncertainty, including that about radon, is by nature difficult to understand and changes as the science evolves. Nevertheless, uncertainty should not be glossed over: "Risk messages and supporting materials should not minimize the existence of uncertainty. Data gaps and areas of significant disagreement among experts should be disclosed. Some indication of the level of confidence of estimates and the significance of scientific uncertainty should be conveyed." (NRC, 1989, p. 170).

Recent studies by Johnson and Slovic (1994) indicate that it is difficult to get people to notice that a risk range rather than a simple point estimate of risk has been communicated. When people do notice, they may pay attention to the highest risk estimate provided (i.e., the upper bound). Overall, however, these researchers found no strong results concerning the effects of communicating uncertainty on agency credibility or trust. Although there was some tendency for perceived agency honesty to increase and perceived agency competence to decrease when uncertainty in risk estimates was communicated, prior attitudes proved to be the most important determinants of such perceptions.

Viscusi et al. (1991) examined the effects of communicating ambiguity in risk estimates on the perceived equivalence of environmental risks in two different areas. They found that communicating ambiguity in risk estimates does not affect the median of the risk estimates made by individuals, but does tend to lead to an increase in the mean of the group's risk estimates, because a minority of study participants focus on the upper bounds of the risk ranges.

Further research is needed on the effects of communicating uncertainty on risk attitudes and behaviors, and attitudes toward the communicating agency, as well as how uncertainty can be conveyed most effectively. The experiments discussed above use stimuli that consist of sets of point estimates of risk, which are either bounds on ranges, or point estimates from different scientists or studies. Simple graphics are also used as experimental stimuli by Johnson and Slovic (1994). Alternatives to these include communicating uncertainty graphically in other formats (see, e.g., Ibrekk and Morgan, 1987), qualitatively, or with the inclusion of more information about why risk estimates differ. The list of sources of uncertainty from Morgan and Henrion (1990) suggests other ways in which these studies could be extended, including, for example, indication of how much subjective judgment has been used, or, conversely, how much agreement between agencies or between scientists there is for a given risk estimate. Studies that do not rely solely on hypothetical scenarios might reveal more about context-dependency for the effects of communicating uncertainty. This research is likely to be most useful and relevant if it entails close collaboration between technical experts, policy makers, and risk communication designers.

3.7.2. Motivating Public Action: Goals for Risk Communication

EPA's goal is to reduce radon-related health risks. This goal does not in itself constrain the risk communication approaches that EPA chooses. Among the issues facing EPA risk communicators are a) how target audience(s) can be educated about indoor radon exposure, effects, test results, and mitigation possibilities most effectively, and b) what types or methods of risk communication(s) will best inform and support radon risk reduction decisions.

Three candidate goals for any risk communication, and radon in particular, are education, advocacy, and decision making partnership (Covello et al., 1986; NRC, 1989). Any of these three could be considered as an intermediate goal for the ultimate reduction of radon-related health risks. The goal of public education, which could also be called decision-making support, or a science education perspective, entails answering questions such as how best to communicate risk and scientific uncertainty about the risks from indoor radon, how best to improve lay knowledge of risk processes, and how to provide a basis for appropriate action. As currently defined, the goal of advocacy, or what might be called a public health perspective, appears to be to persuade the public to test and to reduce indoor radon concentrations to 4 pCi/L, or lower. An increase in meaningful and effective public participation can address the third goal. Because both behavioral decision theory and social cognition theories (Holtgrave et al., in press) suggest that these three goals are not incompatible, further research could help resolve the perception of conflict among them. In addition, explicit and realistic time horizons for risk reduction and public education goals, combined with careful empirical evaluation, would allow the agency to identify measurable risk communication objectives, and to evaluate progress toward those objectives.

3.7.2.1 Education vs. Persuasion as Risk Communication Approaches

Communications are most likely to lead to action or influence intentions to act if they include both a motivational and a self-efficacy (the feeling that one can effectively do something about the risk) component (Earle et al., 1990; Witte, 1992) The motivational component is designed to evoke involvement of the audience and can be positive or negative. A relatively recent review of research on the use of fear- arousal health promotion campaigns concludes that positive reinforcement may prove more effective than fear-arousal (Job, 1988). In those cases where fear-arousal has been effective, specific risk-reduction behavior with short-term effects has been offered as part of the communication, and positive feedback on the effectiveness of that behavior has been offered to reinforce behavior and offset fear. Witte (1992) argues that the interaction of threat and self-efficacy determines the success of fear appeals.

Earle et al. (1990) conclude that risk communication must evoke involvement and personal relevance, and inform the recipient how to control the risk. If threat and positive reinforcement

both arouse involvement and personal relevance, and risk control information conveys efficacy, these lines of research indicate that persuasion and education goals are compatible. Using decision analysis and a "mental models" approach to guide risk communication design (Morgan et al., 1992) provides conclusions consistent with these.

While a public health perspective may seem innocuous, the use of persuasion by any agency or organization outside the health profession is liable to be ill-received by many, and judged unethical by some. In risk communication, this potential increases when the communicator attempts to target motive rather than reason, for example, with fear-arousal. Evaluation of the radon advertising campaign produced by the Advertising Council provides a case in point. EPA reports that the first wave of this campaign, which used the "x-ray" theme, increased public awareness of the risk from radon by 14% and in pretests increased the intention of the public to call for radon information. However, while 74% of the test respondents mentioned health concerns, only 21% noted the need to test. (Excerpt from Advertising Council presentation, courtesy of Mike Walker, U.S. EPA, 1993.) The numbers can be interpreted as an indication of success, in that they illustrate increased awareness of radon. They can also be interpreted as indicating a weakness of the commercial, in that the numbers show that the importance of testing was not as strongly conveyed. An efficacy component sufficient to offset fear was not evident in the evaluation. However, the Advertising Council campaign did successfully provoke strong, critical responses from one audience - radon scientists and researchers (Cole, 1993).

Risk comparisons are frequently used in risk communications with the intent to motivate and involve communication recipients who are at risk. Perhaps partly due to the Advertising Council campaign, there has been some concern about the use of comparisons with "x-rays" in radon risk communications. In one recent empirical study comparisons of radon with x-rays did not affect attitudes toward x-rays, nor did any of several risk comparisons tested affect attitudes toward nuclear power (Weinstein and Sandman, 1992). In fact, in this study the specific risk comparisons did not influence attitudes - including those toward radon - much at all. Perhaps more importantly, the separation of comparisons into two charts, one for smokers and one for non-smokers, did not result in what might be considered a desirable difference in radon risks perceived by smokers, relative to those perceived by non-smokers. Whether separate risk comparisons were used to motivate smokers, or simply to provide lay readers with a more accurate and discriminating understanding about their personal risk from radon, they appear to have failed. Another example of the effects of risk comparisons is provided by a focus group study of the *Home Buyers' and Sellers' Guide to Radon.* Although such focus group studies cannot on their own be interpreted as representative of communication recipients' attitudes or responses, they are a praiseworthy beginning for empirical evaluation. In this study, participants complained that the histogram showing the estimated deaths caused by radon vs. other causes seemed designed to scare, rather than to inform, because radon was the second highest cause

shown (Leferman Associates, 1992, p 17). Much remains to be learned about risk comparisons (Roth et al., 1990), which continue to be used despite a lack of understanding about their effects.

In sum, research results on the use of fear-arousal are ambiguous at best; the literature indicates that persuasion and education are not conflicting goals. To resolve these issues, further research is needed on how to communicate risk effectively with regards to EPA's overall goal of reducing the health risks from radon. Effects on both people's assessment of their ability to control the risk and their sense of personal involvement need to be assessed.

3.7.2.2 Sequential Decision-Making

Goals for risk communications should take into account intervening and subsequent risk control stages and objectives. Controlling the risk from indoor radon involves the multistage process of recognizing the risk, as well as testing, retesting when appropriate, mitigation, and maintenance of any controls, including periodic retesting. Communications that target one stage, such as the first stage of risk awareness, may not influence people who are at another. Stage models of risk reduction, such as the precaution adoption process model (Holtgrave, et al., in press; Weinstein 1988; Weinstein and Sandman, 1992), clarify that risk control involves a series of sequential decisions and behaviors, one or more of which may be effectively communicated to the target audience by any given risk communication. For example, one study found that perceived mitigation difficulty is unrelated to interest in radon testing, and, more generally, that people do not seem to consider the difficulty of risk reduction when they are deciding whether to test for a risk (Weinstein et al., 1990). The relationship between various stages of the risk reduction process is unclear and further research is needed to both understand these relationships and better target risk communications to the different stages.

3.7.3 Public Participation

Mechanisms for public participation include public commentary; the partners program (Wagner, 1993) which promotes public participation by a wide range of interest groups, from the American Lung Association to IBM; and contributions by private citizens and others at publicly announced meetings in which EPA actions or analyses are reviewed, such as Science Advisory Board meetings. As part of the Radon Program Review (U.S. EPA, 1992b) five meetings of invited stakeholders, including representatives from academia, non-profit groups, and private industry, were convened. In other domains, EPA has used Citizen Advisory Groups and Task Forces for public participation (Lynn and Busenberg, in press). More research is needed on increasing and improving the quality of public participation in risk management and EPA priority-setting regarding radon.

3.7.3.1 Incorporation of Public Participation in Decision-Making at EPA

As described earlier, risk communication is often conceptualized as a two-way process, in which the party responsible for communicating risk may be receptive to input from the target audience. More appropriate in a policy context is a framework in which democratic processes are among the risk communicator's objectives. Risk communication development and evaluation necessarily become iterative and interactive in this framework; shared values are established and risk management is informed not only by the understanding of experts and risk professionals, but also by the understanding, or mental models of the risk held by those at risk (Bostrom et al., 1992; Fischhoff et al., 1993; Morgan et al., 1992). The public participation implicated by such an approach reaches further than that evident in most agency risk communication practices.

EPA has acknowledged both openly and tacitly that the source of the message and its context can influence how it is received by a target audience. The partners program promotes participation by specific public and private interest groups by establishing a strong network of affiliates who share EPA efforts, enabling them to coordinate multiple communication channels and to dilute potential credibility concerns (Wagner, 1993). EPA cannot possibly review all of the communications released by partners; although the partners program may gain the agency some credibility, credibility costs, especially in the context of scientific and technical accuracy, need to be examined and evaluated carefully. Also, many of the groups involved in this program receive funding from EPA. Given limited EPA resources, activities funded by EPA will necessarily be restricted in scope and nature. Although the partners program effectively creates a network for reaching the public with radon information, it is limited as a mechanism for encouraging public participation and input to decision-making and priority setting. Public commentary and other forums for public comment can have limited impact on agency decisions when their consideration is left to agency discretion. Empirical studies of public participation mechanisms are needed to establish their effectiveness and appropriateness, with regards to both outcomes and processes.

4. ADDITIONAL RADON RESEARCH TOPICS CONSIDERED

In developing the list of consensus research topics described in Section 3, the Subcommittee considered a broad range of research issues and topics. As described earlier, the consensus list was built from a number of considerations, including agreement among the Subcommittee about the importance and relevance of research on the issue. This section presents briefly some of the additional ideas discussed by the Subcommittee but for which no consensus was reached.

4.1 Epidemiology

4.1.1 Lung Cancer Types

Epidemiological studies should pay particular attention to the types of cancers identified, their locations in the lung and their probable sites of origin. A recent trend in the spectra of lung cancers being seen in nonsmoking female and male humans has been toward increasing incidence of adenocarcinoma at the expense of decreasing squamous cell carcinoma. The meaning of this trend is not clear, but it does imply that both the conducting airways and the parenchymal lung are at risk of developing lung cancer. This is an important shift, because it also implies that radon-induced lung cancers may not be strictly limited to the bronchi. Ongoing and future epidemiological studies should be encouraged to pay particular attention to the types of cancers identified, their locations in the lung, and their probable sites of origin. This information will be important to use in conjunction with improved regional radiation dosimetry to determine if there are differences in radiosensitivity of the different epithelial cell populations of the lung.

4.1.2 Baseline Lung Cancer Rate

A review of lung cancer incidence should be conducted to estimate the baseline cancer rates, separating out smoking and radon related causes. The incidence of lung cancer increase in specific age population groups has increased with the prevalence of smoking. By studying the increase, a determination of background lung cancer can be estimated that will be a combination of radon and baseline lung cancers. The rate of increase in smoking and the rate of increase in lung cancer could reveal the synergistic relationship (degree or power of multiplicity) between smoking and lung cancer.

4.1.3 Rank Lung Cancer Parameters

The numerous parameters for carcinogenic models should be critically reviewed and ranked so that those most influential for radon risk estimates can be used for direct comparison to epidemiological data. In recent years, models of carcinogenesis involving large numbers (in the tens) of parameters, have been developed. While of considerable theoretical interest, such approaches alone are unlikely to be useful for developing radon risk estimates, because there are too many parameters to enable them to be determined by comparison with epidemiological data.

4.2 Factors Affecting Total Exposure and Dose

4.2.1 Role of Non-residential Radon Exposures

Current estimates of radon exposure are based upon an assumed population average of 75 percent occupancy of a home. This implies that 25 percent of the time, or about 42 hours a week, exposures occur at other concentration levels. For individual risk, this is a significant percentage of time. If the bulk of the 25 percent is spent indoors, at school or in an office below the third floor, those concentration levels may vary somewhat from that encountered in the home. If the individual works in an outdoor environment, the levels may be less. If the individual is an underground miner, the levels may be much greater. The issue of non-residential radon exposure is an important factor to be included in an uncertainty analysis evaluating risk on both a population and individual basis.

4.2.2 Nasal and Oral Effects on Respiratory Deposition of Particles

Additional information is needed on respiratory deposition of particles less than 0.1 micrometer in aerodynamic diameter. In this size range, nasal and oral effects may be important. Nasal deposition has been relatively well characterized using nasal casts and limited human experiments. Experiments with monkey casts and *in vitro* measurements have shown that magnetic resonance imaging (MRI) casts give an excellent reproduction of the *in vitro* results. The human studies fall within the 95% confidence bound of the mathematical model developed with the casts. They may fall above the cast deposition because of the additional deposition in the oral cavity on expiration. There are some uncertainties left in oral deposition. Collaborative experiments in multiple laboratories did not yield the same degree of agreement as obtained for the nasal casts and further studies of both existing casts and casts that reflect the range of month and tongue positions are needed.

4.2.3 Distribution of Ambient Radon Levels

The distribution of ambient levels of radon should be characterized, including variability in vertical profiles and inversion characteristics in areas of high radon potential especially those associated with topographically low lying areas. Very cursory studies conducted to date indicate that there is a range of outdoor concentrations from about 0.1 to more than 2 pCi/L (4 to more than 75 Bq/m^3). These studies were conducted frequently at open sites, such as airports. Studies have also indicated that various regions of the country experience radon increases in ambient air when certain meteorological conditions dominate, such as temperature inversions. The scale of these inversions should be investigated because they can occur in any low land area at night. Air concentrations of greater than 25 pCi/L (925 Bq/m^3) have been recorded as being sustained over significant periods of time (days).

4.2.4 Effects of Population Mobility On Total Exposure

The US population of the United States is highly mobile, moving on an average of every 7 years; this can have a significant impact on estimating lifetime exposure. The effect of mobility on the estimates of total exposure has been treated theoretically in Lubin et al. (1990), for use in epidemiological studies (though this analysis did not include latency effects). In addition, estimates of the mobility effect on lifetime cumulative exposures to radon have been made for states where mobility data are available (Liu et al, 1993; Gadgil et al., 1993). This factor should be evaluated and considered in the population risk estimates and their related uncertainty analyses.

4.3 Radon and Radon Progeny in Drinking Water

4.3.1 Transfer Factor For Radon In Water

The transfer factor for release of radon from water to air has not been measured in a large number of houses, particularly where radon concentrations in water are quite elevated. A significant number of homes with private wells have been found where the radon concentration in water exceeds 250,000 pCi/L (20 GBq/m^3). These homes are often clustered, such as in New England and Colorado. For example, at a site near Lyons, CO radon concentrations in water have been found to exceed 1,000,000 pCi/L (80 GBq/m^3). This information is important for determining exposure at the high end of the radon-in-water concentration spectrum and could shed some light on both ingestion and inhalation factors.

4.3.2 Influence of Showering on the Dose

There have been only a few measurements of concentrations and activity-weighted size distributions for radon decay products during bathing or showering. Because the decay product sizes are critical to determining the dose, such measurements should be made under a number of representative conditions including various parameters, such as ventilation rate, water temperature, shower flow rate, etc.

4.3.3 Dose from Ingested Radon Progeny

Additional biokinetics studies are needed to identify the gastrointestinal dose distribution of ingested radon progeny (Crawford-Brown, 1990, 1991). Research is also needed on the contribution to dose/risk of ingestion of radium and other radionuclides found - in some cases concurrent with radon - in drinking water from well-water sources.

4.4 Radiation Biology

4.4.1 Epidemiological Evidence for Hematopoietic Cancers

There is considerable uncertainty in the role of radon and progeny in inducing diseases of the blood-forming tissues. Stated differently, what is the leukemogenic effect of alpha-particle radiation from ingested or inhaled radionuclides (Doll and Darby, 1991). Currently, the most convincing evidence for a particle-induced leukemia comes from followup studies of patients injected intravascularly with the X-ray contrast agent Thorotrast, which is a colloidal suspension of radioactive, nanometer-sized thorium dioxide particles. Follow-up studies of populations in Germany, Portugal and Denmark have shown significant increases in leukemia originating in the bone marrow (acute myeloid and chronic myeloid leukemia, multiple myeloma and hemangiosarcomas limited to the bone marrow) and in lymphoid tissues (thymoma, reticulosarcoma and acute lymphoid leukemia). BEIR IV estimated a linear lifetime risk coefficient of 50-60 leukemia cases per 10^4 person-Gray (BEIR IV, 1988). On the other hand, exposure to the alpha-emitting bone-seeking radionuclides radium-224 and radium-226 have produced only very weak evidence for leukemogenesis. In particular, no excess has been reported for occupationally exposed radium dial painters in either the USA or the UK (Spiers et al., 1983; Baverstock and Papworth, 1989).

Concern for leukemia from indoor exposure to radon and progeny thus far is based on the results of ecological epidemiological studies that related disease incidence to either average radon air concentrations (Henshaw et al., 1990) or radium/radon concentrations in drinking water (Lyman et al., 1985; Collman et al., 1991). These data must be interpreted cautiously, particularly

from the perspective of causation, being ecological studies. One study group (Bridges et al., 1991) measured radon concentrations in domestic environments and used these data to assess leukemia risk in the general population exposed to radon. They found a statistically significant relationship between the logarithm of the mutation frequency in circulating lymphocytes and the domestic radon concentrations. More studies are needed to determine if the observed associations are causal and to understand their meaning in the analysis of radon exposure and leukemia risk.

The data from these domestic studies appear to be in contradiction with the lack of observed leukemia in underground mining populations to date. However, it is acknowledged that these data bases have not been studied in sufficient detail to examine for the possibility of radon-induced leukemia in these populations. Such a detailed re-examination is considered a worthwhile approach to addressing the issue of radon-induced leukemia from indoor exposure.

4.4.2 Genotoxic and Mutagenic Effects

Human studies have revealed some genotoxic and mutagenic effects associated with radon exposures. A dose-response increase in chromosome aberrations of peripheral blood lymphocytes was seen in the lowest four dose groups of Colorado uranium mine workers (Brandom et al., 1978). On the other hand, these effects might be related to the concurrent gamma radiation fields that are also present in the mines.

An increased frequency of chromosomal aberrations was also found in peripheral blood lymphocytes of "radon therapy" workers exposed to 3,000 pCi/L (110 kBq/m^3) in a former gold mine (Pohl-Ruling and Fisher, 1979). An increase in DNA repair rate was higher in juvenile lymphocytes compared with fully differentiated lymphocytes, suggesting that repair enzymes were activated in response to radon-induced DNA damage (Tuschl et al., 1983). A 68-year old woman developed acute myelomonocytic leukemia with chromosomal aberrations following 21 years of "radon therapy" (Rechavi et al., 1990). A statistically significant relationship was also identified between the log mutant frequency of peripheral lymphocytes and domestic radon concentrations as measured by passive alpha-track detectors (Bridges et al., 1991).

The authors of another recent study using x-ray and/or radon progeny exposures of Chinese hamster ovary cells came to the following conclusions: a) Alpha-particles are in general more effective cytotoxic, clastogenic and mutagenic agents than x-rays; b) The lethal and clastogenic lesion induced by both x-rays and alpha particles is probably a DNA double strand break, c) DNA double strand breaks affect the clastogenic and cytotoxic effects of x-rays and alpha-radiation, not their mutagenic effects (Shadley et al., 1991). Although the authors attributed the results to natural background gamma radiation, rabbits exposed to "low normal background" radon

concentrations (28 WLM (100 mJ h/m^3) during 28 months) developed lymphocyte chromosomal aberrations while rabbits exposed to a "controlled (10.7 WLM (38 mJ h/m^3) during 1 month) exposure" did not (Leonard et al., 1981).

4.4.3 Induced Repair Mechanisms

There has been some interest in the hypothesis that a very small dose of X-rays can induce biological repair mechanisms, and thus decrease the response of a cell system to further radiation insult, including alpha particles (Wolff et al., 1991). In this particular study, chromatic deletions were reduced by 50% when the cells were irradiated with 2 cGy of x-ray radiation prior to radon exposure. The authors suggested the existence of a putative chromosomal repair process. It is not known how general this mechanism might be, nor whether it applies to oncogenic endpoints, but further investigations are likely to shed light on repair mechanisms for alpha-particle induced damage.

4.5 Risk Communication

4.5.1 Policy Choices and Target Audiences

According to recent survey results, awareness of radon varies tremendously by subpopulation, and is much greater among whites than among blacks or Hispanics (CRCPD, 1993). EPA is now extending its radon risk communication activities to target specific subpopulations, such as "hot spots," schools, and Hispanic populations. Each of these subpopulations has distinguishing characteristics that might be considered in risk communication design. Thus, not only does this outreach require EPA to successfully communicate with other agencies and policy makers what has already been learned about risk communication, but it also requires a careful examination of the appropriateness of generalizations from those findings. For example, an action level of 4 pCi/L (150 Bq/m^3) for classrooms is unlikely to be comparable to the same action level for homes, given the numerous differences between the two in terms of exposures to individuals.

The U.S. EPA estimates that about nine million U.S. homes have been tested for radon (CRCPD, 1993). Another recent study found that less than a third of a sample of homeowners contacted 3 months after they received test results over 20 pCi/L (740 Bq/m^3) planned to reduce their radon levels (Field et al., 1993). Why haven't more people tested? An EPA sponsored survey of the risk communication literature several years ago produced several explanations, some of which still pertain, and most of which appear to continue to influence risk communication efforts at EPA (Sjoberg, 1989).

More recent research on radon and health protective behavior (e.g., Weinstein et al., 1989b; 1990; 1991b) has suggested additional reasons people may not have tested for radon: a) people don't know what is involved (in other words - how to test); b) the nature of the hazard and risk from radon isn't understood (Bostrom et al., 1992; Field et al., 1993); c) radon is intangible, poses no immediate risk, is not imposed by anyone, and is easier to ignore than to manage; and d) people have other priorities. Moreover, when it comes to personal risks, people worry more about and prioritize economic and social risks above individually managed environmental risks (Doyle et al., 1991; Fischer et al., 1991; MacGregor, 1991).

Some of these - such as how to test for radon and what the hazardous process is - are easier to research and manage than others - such as what to do about radon being intangible, or whether one can or should change people's priorities. Sjoberg (1989) concludes in a review of radon risk communication efforts that "...information campaigns for motivating voluntary changes in behavior are likely to have limited success. Legislation has often turned out to be necessary in order to make a difference..." (p. 95). At least two countervailing arguments should be reviewed: a) The effects of information campaigns should perhaps be evaluated in a different time frame than the effects of legislation. b) Legislation imposes costs that information does not (e.g., loss of freedom of choice, imposition of financial costs for potentially ineffective prevention measures). Agency risk management policy choices should not be based on benefits alone (i.e., risk reduction from radon).

4.5.2 Risk Communication Evaluation

In defining radon risk communication research needs and evaluating risk communication effectiveness, the Agency can choose a) to focus on long or short term goals and objectives, and b) to prioritize either basic or very issue- specific risk communication research. It can be argued that research on effecting and evaluating communications about radon cannot be directed and prioritized until radon risk communication goals and objectives are clearly defined, both substantively and temporally.

Much risk communication research on radon has focused on specific messages in specific media (e.g., specific radon brochures, tables and risk comparison charts), and has been difficult to generalize to broader issues, such as how best to achieve radon risk communication goals, or how and when risk comparisons effectively motivate risk communication recipients. A research program based on empirical tests of risk communication theories is likely to be more informative and useful in the long run than studies that focus almost exclusively on specific, applied issues.

It is essential that risk communication efforts and research be evaluated empirically (Rohrmann, 1992; Fisher et al., 1991). Evaluation of risk communications provides a basis for

budget allocations and effective and efficient risk communication practices. This can only happen, however, if the evaluations are themselves relevant and valid. Risk communications are evaluated in light of specific objectives, which necessarily constrain both the methods and the metrics used, and hence the usefulness of the evaluation for assessing effectiveness according to alternative objectives. Some of these limitations are evident in the discussions above.

In the U.S. EPA Radon Program's plan for implementing recommendations resulting from the Radon Program Review (EPA, 1992b), several measurable objectives for the program are identified. These include awareness, testing, mitigation, school testing, radon resistant new construction, and laws or policies relating to new construction or real estate. All of these can be considered some measure of the effectiveness of various kinds of risk communication, although awareness, testing, and mitigation are most obviously related to the two kinds of goals identified above. None of these, however, appears likely to provide an adequate measure of informational or educational goals. It is also unclear if the CRCPD Radon Risk Communication and Results Study has the power or is appropriate to detect annual changes in testing and mitigation. To evaluate these, future residential radon surveys should be considered.

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APPENDIX A: EPA-SAB-REPORTS ON RADON AND RADIATION

1. Pre-RAC Report C	Report on the Scientific Basis of EPA's Proposed National Emmissions Standards for Hazardous Air Pollutants for Radionuclides (Aug. 1984).
2. Pre-RAC-86-005	Emergency Criteria Applicable to Elevated Indoor Radon Concentrations in Structures Built on the Reading Prong (Nov.5, 1995).
3. SAB-RAC-86-028	Review of Proposal Entitled Health Effects of Waterborne Radon (Sept. 5, 1986).
4. SAB-EC-87-002	Review of the the Research Program of Indoor Air Quality (Oct. 1986).
5. SAB-RAC-87-016	Review of EPA Radon Mitigation Test Matrix (Jan. 12, 1986).
6. SAB-RAC-87-035	Review of the Office of Drinking Water's Assessment of Radionuclides in Drinking Water and Four Draft Criteria Documents: Man-Made Occurrences, Uranium, Radium, Radon (July 27, 1987).
7. SAB-RAC-88-001	Review of Revised Plan for the Idaho Radionuclide Exposure Study (Oct. 9, 1987).
8. SAB-RAC-88-002	Review of the Office of Radiation Programs' National Radon Survey Design (Oct.9, 1987).
9. SAB-RAC-88-009	Review of the Radon Mitigation Research Program (Dec. 22, 1987).
10.SAB-RAC-88-026	Use of the Effective Dose Equivalent Concept (RAC-Initiated letter, Apr. 27, 1988).
11.SAB-RAC-88-041	Review of Office of Radiation Programs' Low-LET Risk Estimate for Regulatory Purposes (Sept. 9, 1988).
12.SAB-RAC-88-042	Review of Office of Radiation Programs' Radon Risk Estimate (Letter Report, Sept. 9, 1988).

13.SAB-EEC-89-012 Resolution on the Use of Mathematical Models by EPA for Regulatory Assessment and Decision-making (Jan. 1989)

14.EPA-SAB-RAC-89-003 National Emission Standards for Hazardous Air Pollutants (NESHAP): Standards for Radionuclides - Review of Assessment Methodologies (Nov. 10, 1988).

15.EPA-SAB-RAC-89-017 Review of the ORP's Radon Measurement Proficiency Program (Apr. 25, 1989).

16.EPA-SAB-RAC-89-024 Review of the Office of Radiation Programs' NESHAPS Background Information Document (June 30, 1989).

17.EPA-SAB--LTR-91-001 Letter Report on Radon Risk Estimates for General Population and Smokers, Non-Smokers, and Children (Letter Rep. Jan. 22, 1991).

18.EPA-SAB-RAC-92-008 Report on Correlation of Short- and Long-term Test Results for Indoor Radon (Dec. 9, 1991).

19.EPA-SAB-RAC-COM-92-001 Status of EPA Radionuclide Models (RAC-initiated commentary, Jan. 9, 1992).

20.EPA-SAB-RAC-LTR-92-003 Revised Radon Risk Estimates and Associated Uncertainties (Letter report. Jan. 9, 1992).

21.EPA-SAB-RAC-92-009 Review of the Office of Drinking Water's Criteria Documents and Related Reports for Uranium, Radon, and Man-made Beta-Gamma Emitters (Jan. 9, 1992).

22.EPA-SAB-RAC-92-012 ORP's Design for the National Survey for Radon in Schools (Jan. 9, 1992).

23.EPA-SAB-RAC-LTR-92-004 Review of Idaho Radionuclide Study (letter report, Jan. 21, 1992; mis-dated 1991).

24.EPA-SAB-RAC-LTR-92-005 Review of Draft Revised *Citizen's Guide to Radon* (letter report, Jan. 29, 1992).

25.EPA-SAB-RAC-COM-92-003 Reducing Risks from Radon; Water Criteria Documents (RAC-initiated commentary, Jan. 29, 1992).

26.EPA-SAB-RAC-LTR-92-010 Review of the Draft Revised *Homebuyer's and Seller's Guide to Radon* (letter report May 22, 1992).

27.EPA-SAB-RAC-COM-92-007 Harmonizing Chemical and Radiation Risk-Reduction Strategies (RAC-initiated commentary, May 18, 1992).

28.EPA-SAB-RAC-LTR-92-018 Drinking Water Treatment Wastes Containing NORM (letter report, Sept. 30, 1992).

29.EPA-SAB-RAC-LTR-93-004 Evaluation of EPA's Proposed Methodology for Estimating Radiogenic Cancer Risk (letter report Dec. 1992).

30.EPA-SAB-RAC-CON-93-002 Notification of a Consultation on a Congressionally Mandated Study of Radon in Water (Jan. 29, 1993).

31.EPA-SAB-RAC-COM-03-001 Radon Mitigation Research: Preliminary Finding (RAC-initiated commentary, Apr. 16, 1993).

32.EPA-SAB-RAC-93-014 Review of Uncertainty of Risks Associated with Exposure to Radon - "Chafee-Lautenberg Multi-Media Risk Study" (July 9, 1993). (cf.EC-93-010 (July 30, 1993).

33.EPA-SAB-RAC-COM-93-006 Quantitative Uncertainty Analysis for Radiological Assessments (RAC-initiated commentary, July 23, 1993).

34.EPA-SAB-DWC-93-015 Review of Issues Related to the Cost of Mitigating Indoor Radon Resulting from Drinking Water (July, 1993)

35.EPA-SAB-RAC-LTR-94-006 Re: ORIA'S Radon Measurement Protocol Evaluation Study (letter report, Jan. 28, 1994).

APPENDIX B: CONVERSION FACTORS

(Radio)activity:

1 becquerel (Bq)	=1 disintegration per second
(Bq is the primary SI* unit)	
1 curie (Ci)	=3.7 x 10^{10} disintegrations per second
	=3.7 x 10^{10} Bq
1 picocurie (pCi)	=2.22 disintegrations min^{-1}
	=0.037 Bq

Activity Concentration:

1 Bqm^{-3} (SI unit)	=0.027 pCi L^{-1}
1 pCi L^{-1}	=37 Bq m^{-3}

Conversion between radon (in activity concentration) and radon progeny (in working levels or joules m^{-3}): where 1 Working Level (WL) = 2.08 x 10^{-5} J m^{-3} (SI units)

1 Bq m^{-3} ^{222}Rn	=1.35 x 10^{-4} WL=2.81 nJ m^{-3} at F**=0.5
1 pCi L^{-1} ^{222}Rn	= 5.0 x 10^{-3} WL at F = 0.5
1 Bq m^{-3} ^{220}Rn	=1.81 x 10^{-4} WL=3.77 nJ m^{-3} at F = 0.05
1 pCi L^{-1} ^{220}Rn	= 6.7 x 10^{-3} WL at F = 0.05[b]

Exposure:

1 Woring Level Month (WLM) = 1 WL for 170h
So WLM = WL x (exposure time in h/170h)
1 WL =3.54 x 10^{-3} J h m^{-3} SI units
working 170 h per month for 1 y at 1 WL = 12 WLM
living for 1 y at 1 WL (100% occupancy) = 515 WLM

For 1 y exposure at 100% occupancy; F=0.5 for ^{222}Rn; F=0.05 for ^{220}Rn

Exposure (1 Bq m^{-3} ^{222}Rn)	=2.46 x 10^{-5} J h m^{-3} = 7.0x10^{-3} WLM
Exposure (1 pCiL^{-1} ^{222}Rn)	=0.26 WLM
Exposure (1 Bq m^{-3} ^{220}Rn)	=3.30 x 10^{-5} Jhm^{-3} = 9.3 x 10^{-3} WLM
Exposure (1 pCiL^{-1} ^{220}Rn)	=0.35 WLM

*Measurement units adopted by the International System of Units.
**Equilibrium factor F is the ratio of the radon progeny concentration to the corresponding radon concentration.

APPENDIX C: GLOSSARY OF TERMS

additive (risk model)	This model assumes that the combined risk from exposures to two (or more) risk factors is the sum of the risks associated with each separately.
adduct	A foreign molecule attached, in this case, to DNA
adenocarcinoma	A malignant tumor originating in glandular or ducted epithelium.
advection	Mass transport via bulk flow processes. In the case of radon or soil gas flow, advective transport is driven by pressure differences.
basal cell	In this case, the cells at bottom of the lung epithelium.
carcinogen	An agent that may cause cancer.
chromosomal aberration	Modification of the normal chromosome, due to deletion, duplication or rearrangement of genetic material.
co-carcinogen	An agent, which itself is not carcinogenic, that can augment the carinogenic process.
codon	The basic three-nucleotide unit of DNA, responsible for specific amino acids.
cohort	A group of individuals who share some common characteristic.
DDREF	Dose/Dose Rate Effectiveness Factor, a factor by which the effect caused by a specific dose of radiation (at a high dose rate) is reduced when the same dose is given at a low dose rate.
epithelium	In this case, the tissue or membrane forming the lining of the bronchus of the lung.
G: C, T:A	Guanine: cytosine, thymine: adenine
gastrointestinal (tract)	The stomach and intestines

gene deletion	The partial or complete loss of a specific DNA sequence.
genotoxin	An agent that may cause stable, heritable changes in the DNA of germ cells.
half-life	The time required for half of a given amount of a substance to disappear. The radioactive half-life is the time it takes for half of the radioactivity to decay. The biological half-life is the time it takes for the body to eliminate half of a chemical substance.
hematopoietic (cancer)	Cancer of the blood-forming tissue, for example, leukemia.
heterozygote	An individual in whom the genes derived from each parent are different.
histology	The study of tissue.
hygroscopic	The ability to readily take up and retain water.
in-vitro	In a cell culture, outside a living organism.
in-vivo	In (or with) a living organism.
LET	Linear Energy Transfer, the average amount of energy transferred per unit track length. Low-LET radiation is characteristic of electrons, gamma-and X-radiation where the distance between ionizing events is large on the scale of the cellular nucleus. High-LET radiation is characteristic of charged particles such as protons or alpha articles where the distance between ionizing events is small on the scale of the cellular nucleus.
lumen	The space in the interior of a tubular structure.
lymphocytes	The principal cell type in the lymphatic system.
morphometry	The measurement of different organs or organ components.

multiplicative (model)	This model assumes that the combined relative risk from exposures to two (or more) risk factors is the product of the relative risks associated with each separately.
mutagen	An agent that increases the frequency of mutations above the spontaneous rate.
neoplastic cells	Tumor cells.
oncogenic transformation	Alteration of cells to a cancerous state.
p53 tumor suppressor gene	A gene which, when altered, is found in many cancers.
parenchymal	Relating to the specific cells of a gland or organ.
point mutation	An alteration or loss of one (or a few) nucleotides in a gene.
promoter	An agent which is not itself carcinogenic but which can amplify the effect of a carcinogen by increasing the probability of late-stage cellular changes needed to complete the carcinogen process.
quality factor	A factor, Q, which depends upon LET (e.g. radiation type) by which absorbed doses are multiplied to obtain (for radiation protection purposes) correspondence with the degree of biological effect produced by X-or low energy gamma-radiation. Dose equivalent (in Sievert) = absorbed dose (in Gray) X Q.
radon progeny	The radioactive decay products of radon. For radon-222, these consist of the radioactive isotopes polonium-218, lead-214, bismuth-214, polonium-214, lead-210, bismuth 210, polonium-210, and stable lead-206. Radiation damage produced by alpha particles from the radioactive decay of polonium-218 and polonium-214 deposited in the lung can initiate lung cancer.
secretory cells	Cells producing substances which are then used by that organ.
transversion	A change from one nucleotide to another in the same position within a gene.

unattached fraction	Those radon progeny that are not associated with other aerosols in the air. This term has been replaced by the term 'ultrafine' aerosol, which more accurately describes these molecular clusters. These clusters can range in size from less than 1 nm to a few nm in diameter. Because such particles are highly mobile, they have a high probability of being deposited and retained in the lung.
working level month (WLM)	A measure of exposure resulting from inhalation of airborne radon progeny with a concentration of 1 working level for 170 hours - equivalent to the number of working hours in a month.

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