



U. S. Department of Justice

Office of Legislative Affairs

Office of the Assistant Attorney General

Washington, D.C. 20530

March 26, 1997

Honorable Albert Gore
President
United States Senate
Washington, D.C. 20510

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Dear Mr. President:

Enclosed is a draft bill entitled the "Radiation Exposure Compensation Improvement Act of 1997." The Department of Justice requests that the draft bill be introduced, referred to the appropriate committee, and passed. We are forwarding an identical proposal to the Speaker of the House of Representatives.

This bill would amend various provisions of the Radiation Exposure Compensation Act of 1990, 42 U.S.C. § 2210 note, to ensure that the criteria for compensation are consistent with the latest scientific data and analytical methods. The bill would incorporate new eligibility criteria for uranium miners seeking compensation for lung cancer, including a new set of criteria defining eligibility for partial compensation, and expand existing eligibility criteria for downwind and onsite participant claimants. The bill would also clarify the circumstances under which an award due to an onsite participant under the Act must be offset by payments from third-parties or the Federal Government. The proposed legislation is designed to remedy a series of perceived shortcomings in the Act that may be frustrating the congressional intent to provide compassionate payments to individuals who suffer or have suffered from diseases presumably resulting from exposure to radiation in connection with the Federal Government's nuclear weapons testing program.

BACKGROUND

On October 15, 1990, Congress enacted and the President signed into law P.L. 101-426, the Radiation Exposure Compensation Act of 1990 ("RECA" or "Act"). The Act established a procedure to make partial restitution to three classes of individuals who were put at serious risk by the federal government's nuclear weapons testing program during the Cold War era, and who, presumably as a result of exposure to resulting radiation, developed certain diseases. The three classes of eligible claimants recognized in the Act are: individuals who lived a specified duration in certain counties downwind of the Nevada

Test Site during defined time periods when above-ground nuclear tests were conducted, and who suffer or suffered from any of thirteen radiogenic malignant diseases; Department of Defense and Department of Energy personnel and contractors physically present ("on site") at one of the federal government's nuclear weapons testing sites during an atmospheric detonation of a nuclear device, and who suffer or suffered from any of thirteen radiogenic malignant diseases; and individuals employed in underground uranium mines in the states of Arizona, Colorado, New Mexico, Utah and Wyoming during the period January 1, 1947 through December 31, 1971, and who suffer or suffered from either lung cancer or one of the following nonmalignant respiratory diseases: "fibrosis of the lung, pulmonary fibrosis, and corpulmonale related to fibrosis of the lung." (In this last category, "if the claimant, whether Indian or non-Indian, worked in an uranium mine located on or within an Indian Reservation, the term shall also include moderate or severe silicosis or pneumoconiosis;") In addition to the geographic, temporal and medical criteria noted above, the statute requires uranium miner claimants to prove that they were exposed to specified minimum levels of radiation in the course of their employment in underground uranium mines. The prescribed minimum exposure level varies depending on a claimant's smoking status and age at the time of onset of the disease.

The Act provides a set level of compensation to an eligible claimant in each class (defined to include the exposed individual or specified surviving beneficiaries): a "downwinder" receives \$50,000; an "on-site participant" receives \$75,000; and a uranium miner receives \$100,000. The Department of Justice administers the RECA through the Radiation Exposure Compensation Program ("Program").

The provisions of RECA defining the eligibility criteria for uranium miner claimants have increasingly been the subject of criticism by scientists, claimants, and interested parties in the affected areas. The principal criticism has been that the threshold radiation exposure criteria for lung cancer are inconsistent with the latest scientific information. Most recently, the Act's provisions relating to uranium miners (and the Department of Justice's implementing regulations) were reviewed by the President's Advisory Committee on Human Radiation Experiments, charged to investigate the history of human radiation experimentation conducted by the federal government during the Cold War period. In its Final Report, the Advisory Committee concluded that much of the criticism of the Act was justified. It recommended that the Administration undertake a review of the provisions of RECA governing compensation for uranium miners to ensure that they are fair, consistent with current scientific evidence, and compatible with the objectives of the Act.

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In response to this recommendation, the Administration created a committee of experts to review the provisions relating to uranium miners in light of the latest epidemiological data and analytical methods. The Committee concluded, among other things, that, based on the latest data, the present exposure criteria misrepresent the risk of lung cancer for many miners. As a consequence, the present criteria have the unfortunate and unjust effect of denying compensation to many miners who were subject to a considerable risk of lung cancer from exposure to radon progeny, and who later developed lung cancer.

Additionally, through its experience administering the Act, the Department of Justice has identified a number of statutory provisions relating to the downwind and onsite participant populations that should be modified to promote just compensation to deserving claimants. The Department believes that a number of these provisions have caused claimants undue hardship. First, leading scientists at the National Cancer Institute now inform the Department that the list of compensable radiogenic diseases for the downwind and onsite participant populations is no longer comprehensive, and, further, that some of the lifestyle conditions that will preclude recovery for an otherwise compensable disease are no longer accurate. The Department has had to deny compensation to a number of claimants who, based on the information provided by the NCI, should be eligible.

Second, through what is plainly an oversight in the original Act, onsite participants are not entitled to recover for childhood leukemia. While Congress, quite understandably, did not anticipate that any children would be present at a nuclear weapons testing facility during a detonation, the statute define "childhood," for purposes of this section, as exposure before age 21. The Department of Justice has denied claims, albeit a small number, from otherwise eligible veterans and government contractors who were present at a designated testing facility during an official test prior to age 21, and have contracted leukemia.

Third, the statute presently requires the Department to offset any award to an otherwise eligible claimant by any payments the claimant has received from some other source related to injuries covered by the Act. The Act specifically provides, however, that an award to downwinders or uranium miners not be offset by any payments received on a claim for workers compensation benefits. The offset provisions for onsite participants differ: they do not make explicit that workers compensation benefits are excluded from offset, and they require the offset of any payments to the claimant from the Federal Government based on injuries covered by the Act. This lack of parity has proved particularly frustrating to claimants and the Department. Pursuant to the explicit direction of the Act, the Department has had to offset awards to onsite participant

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claimants by Social Security disability payments; the Department does not offset awards to downwinders and uranium miners for similar disability payments.

PROPOSED LEGISLATION

The proposed bill would attempt to remedy the shortcomings in the Act relating to uranium miners by incorporating new eligibility criteria for lung cancer based on the latest epidemiological data and analytical methods.

The proposed amendments would add two additional sets of eligibility criteria to the existing exposure criteria for lung cancer, one based on minimum radiation exposure levels, the other on minimum duration of employment. These new criteria more accurately reflect the risk of lung cancer from uranium mining, and, therefore, more accurately provide for compensation to claimants whose lung cancer more likely than not was caused by their exposure to radon in uranium mines. The bill would allow a claimant to qualify by meeting either the existing exposure-based criteria, or either of the two new alternative criteria.

The proposed bill would also establish a new set of eligibility criteria that would provide partial compensation (\$50,000) to some claimants who presently do not qualify for the full compensation amount specified in the Act (\$100,000). These new eligibility criteria would take into account known uncertainties in the data, and resolve these uncertainties in favor of the miner-claimants. The proposed bill would, therefore, provide partial compensation to uranium miners whose exposure is the most likely cause of their lung cancer if they are given the benefit of known doubts.

The proposed bill would also eliminate some redundancy in the list of compensable nonmalignant respiratory disorders, and eliminate the statutory language limiting compensation for silicosis or pneumoconiosis to claimants employed in uranium mines located on an Indian Reservation.

Additionally, the bill would expand the list of compensable diseases for the downwind and onsite participant populations to be consistent with the latest information. The bill would add to the list of compensable diseases primary cancer of the male breast, and primary cancer of the salivary gland, if the claimant was not a smoker. It would also modify the list of lifestyle conditions that exclude otherwise eligible claimants by eliminating the requirement that claimants seeking compensation for pancreatic cancer not have a history of heavy coffee drinking.

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The proposed bill would also correct an oversight in the initial Act: it would expand the eligible population for childhood leukemia to include any onsite participant first exposed to radiation at one of the designated nuclear weapon detonation sites prior to age 21, and who meets the remaining statutory medical criteria.

Additionally, the proposed bill would modify the offset provisions relating to onsite participants. The bill would specifically exclude from offset, as present law already excludes for downwinders and uranium miners, any payments an onsite participant receives on a claim for workers compensation benefits based on injuries covered by the Act. The bill would also specifically limit the offset for payments by the Federal Government to payments made by the Department of Veteran Affairs. This would include amounts paid to the onsite participant under the Radiation Exposed Veteran's Compensation Act (REVCA), but exclude benefits paid under Social Security disability programs for the same compensable illness. These proposed amendments would put onsite participants on an equal footing with downwinders and uranium miners with respect to federal disability payments.

Finally, the proposed bill would make some technical amendments. The bill would amend the provision setting a twelve month period on the time within which the Department of Justice must process a claim. The bill would clarify that where the Department seeks necessary documentation from the claimant or some third-party, the limitations period is tolled.

Estimates of the number of claimants, who would be affected by the proposed legislation, and the budgetary impact necessarily involved substantial uncertainty. Nonetheless, the Administration estimates that approximately 600 claimants, the overwhelming number of them uranium miners, who do not presently qualify for compensation under RECA would receive compensation totalling approximately \$50 million dollars under the proposed legislation. These funds would be distributed over a period of approximately 15 years. The Administration believes that these estimates are conservative, and that the actual figures will prove to be lower.

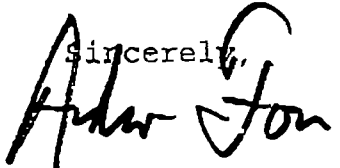
We believe that the enactment of the draft bill would be the best way to further promote the congressional objectives stated in the Radiation Exposure Compensation Act of 1990. The proposed bill will ensure that individuals presumably injured as a result of the Federal Government's nuclear weapons testing program are justly compensated.

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Thank you for your consideration of this important matter. The Office of Management and Budget advises that there is no objection to the presentation of this draft bill, and that enactment of this legislation would be in accord with the President's program.

Sincerely,

A handwritten signature in black ink, appearing to read "Andrew Fois". The signature is fluid and cursive, with a large initial "A" and a stylized "F".

Andrew Fois
Assistant Attorney General

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FINAL REPORT
OF THE
RADIATION EXPOSURE COMPENSATION
ACT COMMITTEE

SUBMITTED TO THE
HUMAN RADIATION INTERAGENCY
WORKING GROUP

JULY, 1996

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EXECUTIVE SUMMARY

On October 15, 1990, Congress passed the Radiation Exposure Compensation Act of 1990 (RECA), which provided for compassionate payments to individuals who suffered from specified diseases presumably as a result of exposure to radiation in connection with the federal government's nuclear weapons testing program. Among those eligible for compensation under the Act are individuals who were employed in underground uranium mines in Arizona, Colorado, New Mexico, Utah or Wyoming during the 1947 to 1971 time period, who were exposed to specified minimum levels of radon, and who contracted specified lung disorders. The Department of Justice administers the RECA through the Radiation Exposure Compensation Program (Program).

The provisions of the RECA defining compensation for uranium miners have been characterized by critics as unfair and inconsistent with current scientific information. The regulations of the Department of Justice implementing the statute have also been criticized as being unnecessarily stringent and unreasonably burdensome. These criticisms were noted, and in some cases affirmed, by the President's Advisory Committee on Human Radiation Experiments, charged by the President to investigate the history of human radiation experimentation conducted by the federal government during the Cold War period. In its Final Report, issued on October 3, 1995, the Advisory Committee recommended, among other things, that the Administration review the provisions of RECA governing compensation for uranium miners and the implementing regulations

to ensure that they are fair, consistent with current scientific evidence, and compatible with the objectives of the Act.

In response to this recommendation, the Human Radiation Interagency Working Group established a committee of government scientists and attorneys to study the RECA provisions relating to underground uranium miners and the Department's implementing regulations. The Interagency Working Group charged the committee to respond within six months with a list of recommended statutory amendments and modifications to the regulations. The accompanying Report is intended to fulfill this obligation.

Listed below is a summary of the Committee's recommended changes to the statute and implementing regulations.

I. Proposed Amendments to the Statute

RECOMMENDATION 1: Amend the Act to incorporate a new set of compensation criteria for lung cancer based on minimum levels of radon exposure

The legislative history of the RECA convinces us that Congress set the exposure criteria at a level it thought would compensate those miners whose lung cancer more likely than not was caused by their exposure to radon progeny in the mines. The latest epidemiological data suggest that the current exposure criteria do not accurately distinguish among lung cancer cases on a "more likely than not" basis, and, therefore, do not effectively implement congressional intent. The present statutory criteria, we conclude, exclude some deserving miners and inappropriately compensate others.

In place of the present criteria, we recommend alternative exposure-based criteria that more accurately effect congressional intent. To generate these criteria we modified a relative risk model developed by the National Research Council's Committee on the Biological Effects of Ionizing Radiations (BEIR IV), and applied it to updated cohorts of Colorado Plateau and New Mexico underground uranium miners. The model conditions compensation on the variables that the latest data indicate most significantly affect the risk of lung cancer in underground uranium miners: cumulative exposure to radon progeny, attained age (the age at which the claimant developed cancer), and time since last exposure.

RECOMMENDATION 2: Amend the Act to incorporate an alternative set of criteria conditioning compensation on minimum duration of employment

The present RECA criteria and the alternatives discussed in Recommendation 1, above, are based in part on a measure of the claimant's cumulative exposure to radon, which is calculated from historical measurements of radon levels in the mines. These historical measurements are widely believed to be inaccurate and imprecise measures of miners' true exposures; for many mines, measurements either do not exist at all or do not exist for all work areas. Using the modified BEIR-IV risk model, we generated compensation criteria for lung cancer based on duration of employment underground in uranium mines, instead of cumulative exposure.

The exposure-based criteria we propose better "fit" the observed data, and will, therefore, result in a more valid characterization of a miner's risk than the proposed employment duration-based criteria. Nonetheless, we recommend a claimant should be allowed to qualify under either the exposure or duration standard.

RECOMMENDATION 3: Amend the Act to condition compensation for nonmalignant respiratory disease on different criteria

The present statutory criteria require that a claimant prove exposure to minimum levels of radon as a condition for compensation for one of the specified nonmalignant respiratory diseases (NMRD) -- pulmonary fibrosis, cor pulmonale related to fibrosis, silicosis and pneumoconiosis. Although it is physiologically plausible that radon causes pulmonary fibrosis, or contributes to the development of silicosis, the two principal compensable NMRDs, the prevailing hypothesis in the scientific community is that the more likely cause of these diseases is exposure to various agents in the mining environment, most notably silica and other dusts. The radon levels in a mine are not believed to be a good proxy for dust levels, so there is no scientifically justifiable reason to tie NMRDs to radon exposure levels.

Unlike the case with lung cancer, above, we do not recommend specific alternative criteria in this case. It is unclear whether and to what extent reliable data are available, and whether appropriate standards can be derived from available data.

The Committee did not have the time to identify and analyze the extant data. We recommend that such a project be undertaken.

RECOMMENDATION 4: Amend the Act to extent compensation for silicosis or pneumoconiosis to all miners covered by the Act

The RECA limits compensation for two NMRDs -- silicosis and pneumoconiosis -- to miners who were employed in mines on an Indian reservation. The principal risk factor for silicosis (or pneumoconiosis) is the amount of silica released in the mining environment. The mines both off and on the Navajo Reservation were in silica-bearing rock, and there is no reason to believe the risk of silicosis was any greater in mines on than off the Reservation. We recommend that this statutory limitation be eliminated.

II. Recommended Modifications to the Department of Justice Regulations

RECOMMENDATION 5: Modify the regulations to presume all Native Americans are nonsmokers, absent evidence to the contrary

The existing compensation criteria for both lung cancer and NMRDs set different minimum exposure levels depending on a claimant's smoking status; smokers must meet a higher exposure threshold, presumably to offset the risks from smoking. The regulations define a smoker as one who has smoked one pack-year of cigarettes or more. Smoking status is generally derived from references in historical health records. Most smoking information in the records is self-reported. The overwhelming

number of Native American underground uranium miners were Navajo. Navajo generally smoked only for ceremonial purposes or not at all; those who smoked did so at substantially lower rates than their White counterparts. Nonetheless, many Navajo reported themselves as smokers, though their consumption was less than one pack-year. We recommend, therefore, that all Native American's be considered nonsmokers, absent evidence in any individual's record of a smoking rate greater than one pack-year.

RECOMMENDATION 6: Modify the definition of nonsmoker in the regulations to include certain ex-smokers

The regulations presume that once a smoker, always a smoker. The latest evidence suggests that the risk of lung cancer in an ex-smoker declines over time, approaching, though possibly never returning, to the background risk of a nonsmoker. The rate of decline of risk depends, among other factors, on the number of cigarettes smoked per day, the number of years the individual smoked, their age at time of cessation, and the years since cessation. We recommend that a claimant be classified as a nonsmoker if he can prove he stopped smoking at least 15 years before diagnosis of lung cancer.

RECOMMENDATION 7: Modify the regulations to allow proof of NMRD by HRCT scans

The regulations require that a claimant prove both physical changes indicating the presence of a compensable NMRD and functional pulmonary impairment. Proof of the presence of disease is made by submitting chest radiographs (x-rays)

interpreted by two "B"-readers, physicians specially certified by National Institute for Occupational Safety and Health (NIOSH) as proficient in the classification of radiographic evidence of pneumoconioses (occupational dust-induced lung diseases). It is well established that chest radiographs, even when interpreted by "B"-readers, have significant false negative and false positive rates. Thus, a claimant with evidence of functional impairment may be denied compensation because a chest radiograph does not show evidence of disease; conversely, a chest radiograph may falsely be read to show evidence of a compensable disease though that disease is not in fact present. Because of the false negative rate associated with the detection of pneumoconioses by chest radiograph, some critics argue that the Program should dispense with the requirement that the claimant submit radiographic evidence of disease process.

We recommend that the Program continue to require radiographic proof of disease. We further recommend that the Program accept high-resolution computed tomography (HRCT) scans as proof of change to lung structure where the claimant has proven physiological pulmonary impairment yet chest radiographs are negative. HRCT is a more sensitive and specific diagnostic technology: in an appreciable number of cases, it will pick up disease that does not appear on chest radiographs, and allows for more accurate diagnosis.

RECOMMENDATION 8: Modify the regulations to allow proof of disease process by biopsy from all miners

The regulations allow surviving beneficiaries to prove a deceased miner had a compensable NMRD through biopsy results, but do not allow living miners to submit this form of proof. This was done to avoid creating an incentive among living miners for invasive and dangerous surgery. Biopsy results, however, are the "gold" standard for diagnosis. We recommend that where a biopsy is performed for a medically-justified reason independent of the desire for compensation, it be accepted as proof of lung abnormality. We also recommend the Program reserve the right to demand appropriate medical evidence demonstrating the justification for the procedure.

RECOMMENDATION 9: Modify the regulations to establish a random audit procedure for "B"-reader reports

The diagnosis of fibrosis or pneumoconiosis by chest radiograph is an inexact science. It is well established that there is significant variability in radiographic interpretation among readers, including "B"-readers, particularly where the disease is in a mild state. The Program has the interpretive reports of some "B"-readers reviewed by experts selected by NIOSH where there may be a question about the accuracy of the interpretation. Some in the affected community perceive the Program as selectively reviewing the reports of the most "liberal" "B"-readers, and thus evidencing a bias against

compensation. Some critics also recommend dispensing with the "B"-reader requirement as unnecessary.

We recommend that the Program retain the requirement that chest radiographs be interpreted by certified "B"-readers. We further recommend that the Program adopt an audit procedure that will provide for the review of all "B"-readers, and thus protect against the possibility of improper application of the professional diagnostic standards, yet eliminate the possibility of selective enforcement.

RECOMMENDATION 10: Modify the pulmonary function standards in the regulations to define impairment consistent with the recommendations of the ATS

As noted in Recommendation 10, above, the regulations define impairment as 25% or more reduction in lung function as measured by pulmonary function tests. The American Thoracic Society (ATS) and American Medical Association (AMA) recommend guidelines that define impairment as more than a 20% reduction, measured against standard reference values. We recommend that the regulations be modified to define impairment consistent with the guidelines recommended by the ATS and AMA.

I. INTRODUCTION

On October 15, 1990, Congress enacted and the President signed into law P.L. 101-426, the Radiation Exposure Compensation Act of 1990 ("RECA" or "Act"). The Act established a procedure to make partial restitution to three classes of individuals who were put at serious risk by the federal government's nuclear weapons testing program during the Cold War era, and who, presumably as a result of exposure to resulting radiation, developed certain diseases.¹ The three classes of eligible claimants recognized in the Act are: individuals who lived downwind of the Nevada Test Site during designated time periods when above-ground nuclear tests were conducted, and who suffer(ed) from any of thirteen radiogenic malignant diseases; Department of Defense and Department of Energy personnel and contractors physically present at one of the federal government's nuclear testing sites during an atmospheric detonation of a nuclear device, and who suffer(ed) from any of thirteen radiogenic malignant disease; and individuals employed in underground uranium mines in five states during a designated time period, and who suffer(ed) from either lung cancer or a specified nonmalignant respiratory disease.² The Act provides a set level of compensation to an eligible claimant (defined to include the exposed individual or specified surviving beneficiaries) in each

¹ P.L. 101-426, § 2, 104 Stat. 920, 920 (1990) (codified at 42 U.S.C. § 2210 note).

² Id. §§ 4, 5, 104 Stat. at 921-22.

class: a "downwinder" receives \$50,000; an "on-site participant" receives \$75,000; and a uranium miner receives \$100,000.³

With regard to the class of uranium miners, Congress specifically found in the Act that:

[R]adiation released in underground uranium mines that provided uranium for the primary use and benefit of the nuclear weapons program of the United States Government exposed miners to large doses of radiation and other airborne hazards in the mine environment that are presumed to have produced an increased incidence of lung cancer and respiratory diseases among these miners; ⁴

The Act provides compensation to miners who were employed in underground uranium mines in the states of Arizona, Colorado, New Mexico, Utah and Wyoming during the period January 1, 1947 through December 31, 1971, and who contracted either lung cancer or one of the following nonmalignant respiratory diseases: "fibrosis of the lung, pulmonary fibrosis, and corpulmonale related to fibrosis of the lung; and if the claimant, whether Indian or non-Indian, worked in an uranium mine located on or within an Indian Reservation, the term shall also include moderate or severe silicosis or pneumoconiosis;"⁵ The Act further requires proof that the miner was exposed to specified minimum levels of radiation, the specific level depending on the

³ Id.

⁴ Id. § 2(a)(3), 104 Stat. at 920.

⁵ Id. § 5(b)(3), 104 Stat. at 923.

miner's age when the disease develops and whether the miner was a smoker or nonsmoker.⁶

The Act delegated responsibility for establishing regulations for the submission and payment of claims to the Attorney General.⁷ On April 10, 1992, the Department of Justice issued final regulations implementing the Act; the regulations became effective May 11, 1992.⁸ The regulations define the proof claimants are required to submit to demonstrate entitlement to compensation. The Attorney General established the Radiation Exposure Compensation Unit ("the Program") in the Civil Division of the Department of Justice to administer the Act and regulations. The Program has been accepting and paying qualifying claims since May 1992.

On October 3, 1995, the President's Advisory Committee on Human Radiation Experiments (Advisory Committee) submitted its Final Report to the President and the Human Radiation Interagency Working Group (IAWG). In its Final Report, the Advisory Committee documented the results of its investigation into various Cold War-era activities of the federal government, including the government's relationship to the uranium miners in the American West. The Advisory Committee concluded that the federal government wronged the uranium miners by allowing them

⁶ Id. § 5(a), 104 Stat. at 922.

⁷ Id. § 6, 104 Stat. at 923.

⁸ See 57 Fed. Reg. 12428, 12429 (1992) (codified at 28 C.F.R. § 79).

standards for compensation should be liberalized.¹²

In response to this recommendation the IAWG requested that the Department of Justice establish a committee of government scientists and attorneys to review the Act and the implementing regulations to ensure that they are fair, comparable to compensation standards for other radiation-exposed populations, and consistent with current scientific data. The IAWG requested that the designated committee recommend to the Administration within six months possible statutory changes to be proposed to Congress and appropriate amendments to the Department's existing regulations.

Acting upon this request, the Attorney General, through the offices of the Deputy Assistant Attorney General for the Civil Division, established the present Committee.¹³

The Committee, which first met in January 1996, has reviewed much of the published criticism of RECA. We reviewed all the critical testimony submitted to the Advisory Committee; the 1993 RECA oversight hearings before the Senate Committee on Labor and Human Resources; and many of the comments on the Department's regulations submitted by interested parties prior to and since

¹² Id. at 813. The Advisory Committee's recommendation regarding uranium miners is attached as Appendix B.

¹³ The Committee is composed of three attorneys from the Department of Justice; two scientists (one physician and one biostatistician) from the National Institute for Occupational Safety and Health (NIOSH); and two scientists (a physician and a biostatistician) from the National Cancer Institute (NCI). The names and affiliations of the Committee members are listed at the front of this Report.

the promulgation of the regulations. We spoke with leading researchers following the Colorado Plateau and New Mexico mining populations, Indian Health Service and private physicians who regularly treat uranium miners, officials and representatives of the Navajo Nation, former miners, claimants' attorneys, and other claimants' consultants and advocates. Some of the individuals with whom we spoke had previously provided testimony to the Advisory Committee and the 1993 congressional oversight hearings.

We also interviewed members of the Program staff to learn their concerns regarding the existing statutory and regulatory scheme, and to obtain the Program's response to the criticisms we heard. We feel obliged to note our perception that the Program staff often go to impressive and commendable lengths to aid claimants. In the course of our admittedly limited investigation, we found little evidence to support the perception in some of the affected communities that the Program staff is biased against compensation or seeks to deny compensation where possible. While we identified a number of possible shortcomings in the administration of the Act (which we passed on to the Program separately),¹⁴ we also heard from some attorneys and miners' advocates who regularly work with the Program that the

¹⁴ For example, it is possible that some of the staff are not fully aware of congressional sentiments expressed during the passage of the RECA urging those who administer the program to treat liberally the claims of Navajo miners, particularly where historical medical documentation is ambiguous. See, e.g., 136 CONG. REC. S11752-55 (daily ed. Aug. 1, 1990). On the other hand, the various administrative shortcomings we perceived appear to affect only a handful of claims.

staff are knowledgeable, helpful and professional.¹⁵ We surmise that the perceptions among some that the Program is biased against the miners is more the unfortunate result of the existing statutory and regulatory restrictions than any systematic bias in the administration.

We set forth in this Report our understanding and analysis of the major criticisms of the Act and implementing regulations, and recommendations for changes to both. The Report is preceded by a summary that lists our recommended changes to the Act and regulations, and is followed by an appendix (Appendix A) that contains a detailed explanation of the relative risk models and statistical analyses upon which we base parts of the Report.

As a final introductory matter, we wish to make explicit three caveats that should be borne in mind when reading the Report. First, the Committee recognizes that the recommendations we put forward may be modified in whole or in part by the Administration because of extrinsic considerations we did not include in our calculus. The Committee has operated on the premise that we were tasked to make recommendations regarding the RECA and implementing regulations that, based on our technical expertise, are warranted. Our recommendations rest on analyses replete with discretionary judgments, but the touchstone for these various judgments were scientific and technical criteria.

¹⁵ We would be remiss if we did not single out for mention Ms. Debra Hetrick, who provided the Committee with outstanding logistical support throughout our investigation. Her contribution and efforts are very much appreciated.

We do not believe we were empowered to make policy-based judgments. As a consequence, we did not consider various potentially relevant policy-based factors in our analysis of the issues -- indeed, we made a conscious effort to avoid doing so.

Second, the recommendations we put forth represent only the opinions of the members of the Committee; nothing in the Report should be construed to represent the opinions of any of the federal agencies or departments which employ the members of the Committee.

And, third, in the time allotted we could not investigate every criticism of the Act or the regulations brought to our attention, although we believe we have addressed the major criticisms. In a few instances, discussed below, we were able to determine that a criticism had merit and warrants changes to the statute, but for lack of time and resources, were unable to generate substitute criteria. In other instances, we concluded that the criticism did not raise issues of the same systemic importance as those discussed below, and, consequently, we simply did not have sufficient opportunity in the time allotted to determine whether the criticism was sound. The fact that we did not address an issue, therefore, does not imply that we found it without merit.

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II. ISSUES RELATING TO COMPENSATION FOR LUNG CANCER

A. Statutory and Regulatory Framework for Compensation

Congress explicitly recognized in the RECA a causal relationship between exposure to radiation in underground uranium mines and an increased risk of lung cancer.¹⁶ The Act provides for compensation to uranium miners who develop(ed) lung cancer if they can establish they were exposed to specified minimum levels of radiation. The statute sets the minimum level of radiation depending on the miner's age at the time the lung cancer is diagnosed (referred to as the miner's "attained age"), and his smoking status: a nonsmoking miner must have been exposed to 200 Working Level Months (WLMs)¹⁷ of radiation; a miner who smoked, and who developed lung cancer before age 45 must have been exposed to 300 WLMs of radiation; and a miner who smoked, and who

¹⁶ P.L. 101-426, § 2(a)(3), 104 Stat. at 920. Uranium deposits exist in geologic formations in the five states designated in the Act. See D.R. Shawe, et al., Uranium and Vanadium Deposits, in [P-2] The Geology of North America 103-24 (H.J. Glustoker, et al. eds., 1991). Uranium is unstable, and naturally decays over time through a well-defined series of reactions in which various forms of radiation are emitted. One of the intermediate products of this so-called "uranium cycle" is radium, which decays to radon, a gas. The radon diffuses through the work environment and is inhaled by miners. Radon itself decays into a series of progeny or daughter products, which give off alpha radiation. The carcinogenic potential of radon derives from the progeny's emission of alpha radiation in the lung. Throughout this report, we use the terms "exposure to radon" and "exposure to radon progeny" interchangeably.

¹⁷ A Working Level Month (WLM) is a time-integrated exposure measure. A working level is defined as any concentration of radon progeny that results in the release of 1.3×10^5 million electron volts (MeV) of alpha energy per liter of air. WLMs are calculated as the product of the number of months underground (170 hours/month) and the radon progeny concentration measured in WLMs. See 28 C.F.R. § 79.30(c), (d).

developed lung cancer after the age of 45, must have been exposed to 500 WLMs.¹⁸ The regulations define a smoker as "an individual who has smoked at least one (1) pack-year of cigarette products."¹⁹ A nonsmoker is defined as an individual who has never smoked cigarette products or has smoked less than one pack-year.²⁰

These exposure criteria, and to a slightly lesser extent the definition of smoking in the regulations, have been severely criticized. The principal criticisms have come from scientists who study the uranium mining population. They insist that the present exposure criteria are inconsistent with or unsupported by the latest scientific data. The present criteria, the scientists charge, have the unfortunate and unjust effect of denying compensation to many miners who were subject to a considerable risk of lung cancer from exposure to radon progeny, and who later developed lung cancer.

The critics charge that the present statutory criteria are defective in five fundamental ways. First, the latest data indicate that the numerical exposure minima misrepresent the risk of lung cancer for many miners. As a consequence, the Act effectively compensates only those miners who meet a threshold risk much greater than Congress intended. Second, the exposure

¹⁸ P.L. 101-426, § 5(a)(1), 104 Stat. at 922.

¹⁹ 28 C.F.R. § 79.31(f).

²⁰ Id. § 79.31(g). A pack-year of cigarette products is defined as smoking one pack of cigarettes (20) each day for one year.

minima vary depending on smoking status, though the latest data suggest that the risk of lung cancer due to exposure to radon may not be dependent on this variable. Third, the distinction in exposure minima for smokers based on whether lung cancer was diagnosed before or after age 45 is curious. Although the latest data do suggest attained age is an important variable, the critics note that there is little support in the data for the proposition that the risk of lung cancer varies dramatically only around age 45. Fourth, the Program uses actual and estimated historical radon level measurements in mines to compute miners' exposure. The existing measurement data, however, do not cover all mines or all work areas within mines. The resulting estimates of a miner's true exposure are imprecise and inaccurate, and, consequently, any calculated risk of lung cancer may be seriously in error. And, fifth, the existing exposure minima for uranium miners translate to a much higher risk than is imposed on either the downwinders or on-site participants for compensation.

These criticisms have been echoed most recently and forcefully by the President's Advisory Committee in their Final Report. They have also been voiced by almost every group with an interest in RECA, including the miners, their attorneys, advocacy groups, treating physicians, medical researchers and government officials.

In light of these criticisms, this Committee reviewed the latest epidemiologic data on lung cancer in underground uranium

miners employed in Colorado and New Mexico. We have concluded that most of the concerns raised about the present statutory criteria have merit, and warrant amendment of the Act. Our analysis of the epidemiological data convinces us that the present exposure minima underestimate the risk of lung cancer to some miners, with the result that these miners may unfairly be denied compensation though it is likely their exposure in fact is the cause of their lung cancer.²¹ Moreover, the criteria do not properly account for or incorporate two factors that significantly affect the risk of lung cancer in uranium miners: attained age independent of smoking and time since last exposure.

However, we have also concluded, contrary to many critics, that the distinction between smokers and nonsmokers should be retained. Based on our analysis of the data, we suggest two possible substitute compensation models -- one, similar to RECA, that bases compensation on cumulative minimum radiation exposure; and a second, as suggested by the Advisory Committee, that conditions compensation on duration of employment. We are convinced that either alternative model more accurately identifies those miners Congress intended to compensate: individuals whose lung cancer "more likely than not" was caused by exposure to radon in the uranium mines. We recommend that the Act be amended to incorporate both compensation models, so that a

²¹ In addition to the problem of underinclusiveness, as we demonstrate infra, the current criteria also tend toward overinclusiveness (compensation to undeserving miners), by failing to consider attained age and time since last exposure, independent of smoking.

miner qualifies for compensation if he satisfies the criteria of either model.

We also conclude that there is significant merit to the charge that the Act's compensation criteria for uranium miners are not comparable to the criteria for downwinders and on-site participants, to the gross disadvantage of the miners. We believe, however, that the respective compensation criteria and associated legislative history imply a desire by Congress to treat the covered classes differently. Because the issue is at heart a policy matter, we make no recommendation. We do put forth a number of options that would provide greater parity among the eligible groups, if so desired.

B. Fairness of the Present Statutory Compensation Criteria

Since the early 1970s, a substantial amount of research has been published, including numerous epidemiological studies of underground uranium miners, that convincingly demonstrates a causal link between exposure to radon and its decay products and the development of lung cancer.²² The results of this research

²² See, e.g., Jonathan M. Samet, et al., Uranium Mining and Lung Cancer in Navajo Men, 310 NEW ENGLAND J. OF MEDICINE 1481 (1984); F.D. LUNDIN, JR., ET AL., NATIONAL INSTITUTE OF OCCUPATIONAL SAFETY AND HEALTH-NATIONAL INSTITUTE FOR ENVIRONMENTAL HEALTH SCIENCES, JOINT MONOGRAPH No. 1, RADON DAUGHTER EXPOSURE AND RESPIRATORY CANCER, QUANTITATIVE AND TEMPORAL ASPECTS (1971). Two of the principal epidemiological studies defining the risks of radon are ongoing investigations of cohorts of uranium miners covered by the RECA. One study has focused on uranium miners employed underground in mines in the Colorado Plateau area. See Richard W. Hornung, et al., Modifiers of Exposure-Response Estimates for Lung Cancer Among Miners Exposed to Radon Progeny, 103 ENVIR. HEALTH PERSP. 49-53 (1995) (updating study); Robert J. Roscoe, et al., Lung Cancer

(continued...)

and a good deal of the literature were presented to Congress in the years preceding the passage of the RECA.²³

Since 1990, however, some of the principal studies have been updated and reanalyzed, producing important new information regarding the relative risk of lung cancer posed by exposure to radon. Data on lung cancer mortality in both the Colorado Plateau and New Mexico cohorts have been updated.²⁴ Most importantly, in 1994, scientists from the National Cancer Institute, in collaboration with the principal investigators of the eleven cohort studies of radon-exposed underground miners,

²² (...continued)
Mortality Among Nonsmoking Uranium Miners Exposed to Radon Daughters, 262 J. OF THE AM. MED. ASSOC. 629 (1988) (updating study); LUNDIN, supra (original study). The second has focused on underground uranium miners in New Mexico. Jonathan M. Samet, et al., Lung Cancer Mortality and Exposure to Radon Progeny in a Cohort of New Mexico Underground Uranium Miners, 61 HEALTH PHYSICS 745-752 (1991) [hereinafter Lung Cancer Mortality in New Mexico Uranium Miners] (updating study); Samet, Uranium Mining and Lung Cancer in Navajo Men, supra (original study).

²³ See Health Effects of Radiation Exposure Caused by Open Air Atomic Testing and Uranium Mining, Hearings before the Senate Committee on Labor and Human Resources, 101st Cong., 2d Sess. 25-44 [hereinafter Hearings on Health Effects of Radiation Exposure] (testimony of Victor Archer, M.D.); id. at 61-117 (appended studies); Impact of Past Uranium Practices: Hearings before the Subcomm. on Mineral Resources, Development and Production of the Senate Comm. on Energy and Natural Resources, 101st Cong., 2d Sess. 21-37 [hereinafter Hearings on Impact of Past Uranium Practices] (testimony of Richard W. Hornung, Dr.P.H.); id. at 50-131 (testimony of Lynda Taylor, with supporting studies).

²⁴ See Samet, Lung Cancer Mortality in New Mexico Uranium Miners, supra note 22, at 745-752; Hornung, supra note 22, at 49-53.

pooled and analyzed the original data from all eleven studies.²⁵ This 1994 NCI-led study refined an existing statistical model that describes the relationship between exposure to radon and the risk of lung cancer.²⁶

The President's Advisory Committee relied heavily on the 1994 NCI-led analysis in criticizing the present RECA criteria and recommending reform of the Act. Citing the NCI-led analysis as its principal if not sole support, the Advisory Committee concluded:

Since 1990, additional scientific information has become available to support the view that radon exposure is responsible for a much higher proportion of the lung cancer cases among the miners than had been previously thought. In particular, the [A]ct's current requirement of a minimum of 200 WLM ... exposure for nonsmokers or 300 to 500 WLM (depending on age) for smokers translates to quite large probabilities of causation, according to a recent report of the National Cancer Institute. That analysis finds little evidence to support a distinction between smokers and nonsmokers and suggests that a majority of lung cancer deaths among Colorado white miners and New Mexico Navajo miners are attributable to radon exposure. Furthermore, it finds that the lung cancer risk is strongly modified by a number of factors and uncertainties that are not accounted for in

²⁵ LUBIN, ET. AL, NATIONAL INSTITUTES OF HEALTH, RADON AND LUNG CANCER RISK: A JOINT ANALYSIS OF 11 UNDERGROUND MINER STUDIES (1994) (NIH Publication No. 94-3644).

²⁶ This statistical risk model was developed in 1988 by the National Research Council's Committee on the Biological Effects of Ionizing Radiations (BEIR IV). The BEIR IV model was based on a pooled analysis of four epidemiological studies of underground miners, including the uranium miners of the Colorado Plateau and New Mexico. The 1994 NCI-led pooled analysis included approximately eight times the data available to the BEIR IV Committee.

the total dose; thus, for many miners, the level of exposure that would merit compensation on the basis of the principle of "balance of probabilities" might be far lower than the present criteria.²⁷

This Committee agrees with the President's Advisory Committee that the BEIR-IV/NCI model is the best available tool by which to judge whether the present RECA compensation criteria (for lung cancer) fairly compensate miners. The model was developed by leading experts in the field and has been accepted by the scientific community. As recently refined by NCI, it employs the latest data and statistical methods. It should represent, therefore, the best estimate of the true risks of exposure to radon sustained by the uranium miners covered by RECA. Moreover, we adapted the BEIR-IV/NCI analysis to pooled data from the Colorado Plateau and New Mexico cohorts to derive risk models, and from these models compensation criteria that we propose in place of the existing statutory criteria.

The crucial question, however, is how one defines the "fairness" of the compensation criteria. The Committee believes that the fairness of the present statutory criteria, or any proposed alternative criteria, should be judged by the extent to which they effect the intent of Congress. We conclude, as did the Advisory Committee, that the present standards are not very fair by this definition: that is, they do not accurately provide for the compensation of those miners Congress intended to compensate. We propose instead two alternative sets of criteria

²⁷ FINAL REPORT, supra note 9, at 814.

that, either used alone or in combination, more accurately identify those miners whom Congress sought to compensate.

1. The Legislative History Reveals That Congress Sought to Compensate Uranium Miners On A Balance of Probabilities Standard

To determine the class of miners Congress intended to enfranchise through the statutory criteria in RECA, we examined both the text of the Act and its legislative history. Based upon this review, we have concluded that Congress intended in the RECA to compensate uranium miners whose lung cancer "more likely than not" was caused by exposure to radon and its decay products during employment in underground uranium mines.²⁸ In the statistical terms employed by the BEIR-IV and NCI analyses, this standard is equivalent to a probability of causation (PC) equal to or exceeding 0.5.²⁹

The Act itself provides some guidance as to congressional intent, but ultimately is ambiguous as to the underlying standard of causation. Congress included as one of the legislative

²⁸ Apparently, this is the same conclusion reached by the Advisory Committee. In its Final Report, the Advisory Committee recommended changes to the Act and regulations that it believed would provide compensation to all miners entitled under the "balance of probabilities" principle. See FINAL REPORT, supra note 9, at 814.

²⁹ The probability of causation (PC) can be thought of as the chance that a specific case of lung cancer in a subject population was caused by a given exposure. More formally, it is defined as the excess risk of lung cancer in the subject population (the risk in the exposed population minus the risk in the unexposed population) divided by the risk of lung cancer in the exposed population. See Ben Armstrong & Gilles Theriault, Compensating Lung Cancer Patients Occupationally Exposed to Coal Tar Pitch Volatiles, 53 OCCUPATIONAL ENVIR. MED. 160, 162 (1996).

findings that "radiation released in underground uranium mines ... and other airborne hazards in the mine environment ... together are presumed to have produced an increased incidence of lung cancer and respiratory diseases among miners"³⁰ It also found that "the lives and health of uranium miners and of individuals who were exposed to radiation were subjected to increased risk of injury and disease to serve the national security interests of the United States."³¹ While Congress acknowledged in these statements that miners were subject to an increased risk from exposure to radon, they leave unclear precisely how much risk, when followed by disease, warranted compensation. Yet, the fact that Congress explicitly conditioned compensation on specific minimum exposure criteria strongly implies that it intended to compensate only those miners in which the increased risk due to radon exposure -- the likelihood of causation -- exceeded some threshold level of confidence.

The history of the Act is extensive; legislative efforts to compensate uranium miners date back to the late 1970s. Specific minimum exposure standards first appeared in H.R. 5022, an earlier version of a compensation scheme introduced into the House of Representatives in July, 1988. The bill would have created a cause of action in the (then) United States Claims Court for uranium miners employed in mines in certain designated states within a specific time period, who contracted lung cancer

³⁰ P.L. 101-246, § 2(a)(3), 104 Stat. at 920.

³¹ Id. § 2(a)(5), 104 Stat. at 920.

or "other serious respiratory disease," and who were exposed to at least 100 WLMs if a nonsmoker or 250 WLMs if a smoker.³² The source of these specific exposure levels is not apparent, but subsequent testimony before two congressional committees suggests that the levels may have been approximations of exposures at which it was more likely than not that the subsequent development of lung cancer or a nonmalignant disease was due principally to the exposure. In February, 1988, in testimony before the Senate Committee on Labor and Human Resources, Dr. Victor Archer, a physician who has spent his career studying the incidence of disease among uranium miners, and who was formerly the principal investigator for the Public Health Service's prospective epidemiological study of uranium miners of the Colorado Plateau area, stated:

I have been involved in a number of workers' compensation cases in about five States in the West with uranium miners, and I have been frequently asked to determine whether the particular lung cancer was more likely or not caused by the radiation in the mines. For nonsmokers, this is quite easy if their radiation exposure has been above 100 Work Level Months, and among the nonmining population, the rate of lung cancer among nonsmokers is so extremely low. For heavy cigarette smokers, however, this is a little more difficult. Usually above 800 [sic] Work Level Months, it is more likely than not that the lung cancer is due to radiation. At Work Level Months below 300, then it is more often than not that cigarette smoking turns out to be the more important cause. This is not to say that lung cancer does not occur among miners who have had less than 300 Work Level Months, that it does not occur as a result of

³² H.R. 5022, 100th Cong., 2d Sess. (1988).

radiation, but in groups of heavy smokers with less than 300 Work Level Months, we have found that smoking does cause a higher fraction of the cases than the radiation does.³³

Less than one month later, before a different Senate committee, Dr. Richard Hornung, of the National Institute of Occupational Safety and Health, another leading researcher of the effects of radon, testified that a cumulative exposure to 120 WLMS would result in a doubling of the risk of lung cancer.³⁴ The exposure that causes an individual's risk to double, also called the "doubling dose," is the point at which it is more likely than not that the individual's exposure to radon was the cause of any resulting lung cancer.

³³ Hearings on Health Effects of Radiation Exposure, *supra* note 24, at 41. In a later colloquy with Senator Hatch, Dr. Archer made clear that in his opinion the association between lung cancer and radiation exposure for many miners was more than probable -- it was virtually certain:

Senator Hatch: And how would you rate the probability with regard to the contribution toward cancer? Would it also be a high probability?

Dr. Archer: Yes, extremely high.

Senator Hatch: To use your terms, almost a certainty?

Dr. Archer: Yes.

Senator Hatch: That's a fair statement?

Dr. Archer: Among those with higher exposures, yes, it is almost certainly due to radiation.

Id. at 43-44.

³⁴ Hearings on Impact of Past Uranium Practices, *supra* note 23, at 23, 31.

Thus, the exposure criteria suggested to Congress by the leading experts explicitly sought to distinguish for compensation those miners whose disease more probably was caused by their exposure. Significantly, the various legislative schemes proposed after 1988, including H.R. 2372, which became the RECA, all contained threshold exposure criteria equal to or greater than these suggested levels.

The legislative history of H.R. 2372 suggests strongly that the existing exposure minima were intended to define the class of miners who developed diseases probably caused by radiation. To begin with, the bill, as originally introduced in the House, established a single exposure criteria of 250 WLM, more than twice the level recommended by Drs. Archer and Hornung.³⁵

Additionally, during House consideration of the bill, one Representative expressed his concern that the bill did not require claimants to prove causation, and included diseases that the medical community did not yet agree were caused by

³⁵ H.R. 2372, 101st Cong., 2d Sess. § 5(a)(1) (1990).

radiation.³⁶ These concerns were immediately addressed by one of the bill's sponsors:

"Considering all those people who have suffered, I do not think that I would agree with the gentleman from Florida [Mr. James] as to whether there would be a preponderance of evidence, and the proximate cause would be difficult. We can all see that. But the case that was tried in Salt Lake City ... turned out the type of evidence that we feel is necessary.³⁷

While not without ambiguity, this response appears to assume that claimants would in effect be required to prove causation (in legal parlance, "proximate cause") by a "preponderance of the evidence," a standard legal description of a burden of proof equivalent to the "more likely than not" standard.

Senate consideration of H.R. 2372 more clearly reflects congressional understanding that the exposure criteria were intended to identify cases where exposure to radon was the cause of lung cancer or a nonmalignant disease. Senator Hatch, one of the sponsors of the bill, made it clear that the legislation

³⁶ "I admit that I have some problems with the lack of a requirement for proof of causation under the Compensation program which this bill creates, however, I understand the difficulty in proving such claims. I am aware that there is some confusion among our medical experts today as to which diseases are caused by exposure to radiation and the inclusion of some of those diseases and the coverage of claims program set forth in this bills troubles me to some extent. However, I have no proof as to the medical certainties that will deny the coverage of certain of these diseases any more that I have proof that will confirm a need for coverage of these diseases and, therefore, I am willing to err on the side of compassion rather than on the side of denial to those who have become ill." 136 Cong. Rec. 12636 (1990) (statement of Rep. James).

³⁷ Id. at 12638 (statement of Rep. Hansan).

demanded a high standard of association between exposure and disease:

In recognition of the fact that legal avenues are closed because of the Tort Claims Act and the fact that the Government's failure to monitor the downwind areas has made a case by case determination on the issue extremely difficult, the legislation now before us has been designed to provide compensation for those victims whose cancers were most likely to have been caused by their prolonged exposure to radiation. ... Uranium miners must have worked in mines during those times when there were minimum efforts to reduce the level of radium in the air and they must have been exposed to levels of radiation of at least 200 working level months, ...³⁸

The Senate amended the bill to add, inter alia, the present three-tiered exposure minima, dependent on age and smoking status, for uranium miners.³⁹ Explaining these amendments, Senator Hatch reiterated the requirement that the claimants' radiation exposures be the most likely cause of their compensable diseases:

In addition, as a request of the Senator from Wyoming, we have incorporated additional changes in the uranium miners section of the bill to ensure that the lung cancer and respiratory diseases for which compensation is provided are those which are most likely to have been caused by the radiation in the mines. Distinctions have been made with respect to exposure levels and whether or not the miners were smokers.⁴⁰

³⁸ 136 CONG. REC. S11752 (daily ed. Aug. 1, 1990).

³⁹ See id. at S11749-50 (substituting bill for H.R. 2372).

⁴⁰ Id. at S11752.

The purpose and effect of the Senate amendments were understood by the House as well when it reconsidered and passed H.R. 2372, as amended. Representative Owens, one of the bill's original sponsors, stated that "[o]ther changes were made in the House version to ensure that the lung cancers and respiratory diseases for which compensation is provided are those which are most likely to have been caused by the radiation in the mines."⁴¹

The purpose of the amendments was made even more explicit by Representative Kyle, who stated:

The one flaw I did find in the House version of the measure has now been corrected by the Senate. That flaw related to diseases that may have [been] brought on by an individual's own conduct -- conduct such as tobacco or alcohol abuse. ... We have to be sure that the Government's misconduct, not the individual's is the cause.⁴²

Additional evidence of congressional intent is apparent from a pair of bills introduced by Senator Hatch in 1989 and 1990, respectively, relatively contemporaneous with the introduction of H.R. 2372. S. 1994, introduced in November, 1989, would have

⁴¹ 136 CONG. REC. H8276 (daily ed. Sept. 27, 1990).

⁴² Id. at H8278. Indeed, the Senate amendments, excluding other possible causes for the various compensable diseases, apparently assuaged Representative James' concerns that claimants might be compensated though radiation was not the most likely or probable cause of the illness. During the House reconsideration of H.R. 2372, as amended, he stated that "I realize that this bill, as amended by the Senate, does include additional requirements for compensation. In this light, I would like to commend our colleagues in the Senate for their attempt to address the lack of requirement for proof of causation in the original House-passed Bill." Id. at H8275.

authorized miners to file petitions with Special Masters of the then United States Claims Court, who were authorized in the legislation to award money damages to miners employed in uranium mines located in designated states during the period January 1, 1947, and December 31, 1971, who contracted lung cancer "or other serious respiratory diseases," and who were exposed to at least 100 WLMs.⁴³ S. 2466, introduced shortly thereafter in April 1990 contained the same compensation criteria for miners with the exception that the minimum exposure level for compensation was set at 200 WLM.⁴⁴ The Senate Report recommending passage of S. 2466 makes absolutely clear the standard of proof underlying the compensation criteria for miners:

The law also requires a demonstration through a preponderance of the evidence that the nuclear fallout was the proximate cause of the cancers in question. ... In recognition of the fact that ... causation is such a difficult matter to prove on a case by case basis, especially in light of the Government's failure to monitor the downwind population, the Committee has designed this bill to provide compensation to those victims whose cancers were most likely to have been caused by their prolonged exposure to radiation. ⁴⁵

⁴³ S. 1994, 101st Cong., 1st Sess. § 2142(c)(1)(A)(ii)(I) (1989).

⁴⁴ S. 2466, 101st Cong., 2d Sess. § 2144(c)(1)(B)(ii) (1990).

⁴⁵ S. Rep. 101-264, 101st. Cong., 2d Sess. 23-27 (1990) (emphases added). The Report emphasized that the designated exposure level reflected a high degree of probability of association between exposure and disease:

Because of the possibility that any given cancer may or may not have been directly caused by low level radiation, and because

(continued...)

Moreover, S. 2466 would have directed the Secretary of Health and Human Services to prepare a report on "the existence of evidence or data supporting the conclusion that exposure of uranium miners to less than 200 working level months of radiation is associated with lung cancers or other serious respiratory disorders."⁴⁵ This requirement would seem to confirm that the bill's sponsors believed 200 WLM represented the point at which the causal association between exposure to radiation in the mines and the development of the designated diseases had been proven.

Finally, our conclusion that RECA is intended to compensate only those miners whose disease "more likely than not" was caused by their exposure to radon progeny is supported by the Senate's rejection of a compensation plan introduced by Senator Hatch in 1985 that would have allowed for payments to miners on a less rigorous standard. This legislative scheme would have created a commission empowered to pay damages to miners with lung cancer if

⁴⁵ (...continued)

this legislation attempts to compensate those victims with the highest probability of cancer due to ... uranium mining, the requirements for filing a petition have been narrowly drawn. ... The uranium mines found in the ... listed states were those with the highest levels of radon. The time periods for ... working in the mines are those periods when the levels of radiation were at their greatest. And finally, only those cancers which have strong evidence of being caused by radiation are included in the list of diseases.

Id. at 29.

⁴⁶ S. 2466, 101st Cong., 2d Sess. § 2141 (1990).

there was a "sizeable possibility" that the disease was the result of exposure to radiation in uranium mines in designated states during a specified time period; the commission was empowered to pay prorated damages to miners where the "sizeable possibility" of causation was less than fifty percent.⁴⁷

Numerous Senators spoke against the proposal for, among other reasons, providing compensation where the probability of causation was less than fifty percent. For example, Senator Simpson argued that

[T]his amendment allows awards even where the claimant is absolutely unable to show that his or her cancer was caused by the fallout under our traditional tort principles. In fact ... the amendment allows prorated awards where the probability of causation ... is less than 50 percent. There is a fundamental change in traditional court rules under this approach, where you would make awards based upon a probability of less than 50 percent.

...

This amendment would, if adopted, provide for compensation of individual claimants, even though those claimants could not establish that it was 'more likely than not' that their cancer was caused by exposure to fallout -- the standard traditionally imposed under our tort system as a condition of recovery. Under Section 209(c) of this amendment, the Commission would be authorized to provide prorated compensation for persons with probabilities of causation less than 50 percent. This provision represents a fundamental and grievous departure from the principles of fault and compensation that have served as the foundation of our tort

⁴⁷ S.J. Res. 77, 99th Cong., 1st Sess. (1985) (Amendment 772).

system, and in my judgment, the Congress should reject what has been proposed.⁴⁸

In sum, the available legislative history of proposals to compensate uranium miners strongly suggests that Congress intended the existing exposure criteria to compensate only miners whose disease "more probably than not" was caused by exposure to radiation in the mines.⁴⁹

2. The Present RECA Criteria for Lung Cancer Are Not Consistent With A Compensation Standard of PC Greater Than Or Equal To 0.5

The present RECA criteria reflect Congress' assumption that the relative risk of lung cancer among uranium miners is principally related to three factors: (1) a miner's cumulative exposure to radiation (measured in WLMS); (2) the miner's smoking status; and, (3), if the miner is a smoker, attained age, i.e., age at the date of diagnosis. More recent studies and data,

⁴⁸ 131 CONG. REC. 32080-82 (1985). See also id. at 32079 (statement of Sen. Stafford); id. at 32085 (statement of Sen. McClure).

⁴⁹ We recognize that the legislative history is not without ambiguity, and is susceptible to at least one alternative reading -- that Congress sought to compensate miners with lung cancer who were exposed sufficiently to radon progeny to elevate the probability of causation above the background risk of lung cancer in the non-exposed population, even if that probability of causation was less than 50%. E.g., P.L. 101-426 § 2(a)(3), 104 Stat. at 920 (explicit congressional finding in RECA that "radiation released in underground uranium mines that were providing uranium for the primary use and benefit of the nuclear weapons program of the United States Government ... are presumed to have produced an increased incidence of lung cancer and respiratory disorders among ... miners.") (emphasis added). The Committee believes that when one considers the entire legislative history, and particularly the Senate rejection of S.J. Res. 77, this interpretation is less plausible than the one on which we rely.

particularly the 1994 NCI-led analysis, have convinced the Committee that we can now offer Congress a more accurate assessment of the radon exposure-lung cancer relationship. While the NCI pooled analysis indicates that the first and third factors, and possibly the second, do significantly affect the relative risk of lung cancer, the present RECA criteria (1) do not properly reflect the effect of increasing age; and (2) do not account at all for an additional significant variable -- time since last exposure.

First, RECA does not properly account for the effect on the risk of lung cancer of attained age. The present criteria set one exposure standard for smokers under age 45, and a second, higher standard for smokers over age 45. Analysis of the miner studies indicates that the relative risk of lung cancer due to radon progeny exposure decreases with attained age in both smokers and non-smokers. The data do not indicate any dramatic changes in relative risk at age 45. Indeed, the decline in the relative risk continues throughout the middle and older ages. This is principally due to the fact that the background rate of lung cancer increases with age in smokers and non-smokers; the proportional increase in risk of lung cancer due to radon progeny does not keep pace with the increasing background risk.

Moreover, the present criteria necessarily imply that all smoking miners 45 years of age and older face the same or nearly the same risk of lung cancer: all such miners must meet the 500 WLM minimum exposure level standard, regardless of any other

factors. Yet, the BEIR-IV analysis conclusively demonstrated that a risk model that depends only on cumulative exposure will generate results that do not reasonably "fit" the observed data - - i.e., the model does not adequately describe the association between exposure and the development of lung cancer.⁵⁰

Second, the present criteria do not include an additional variable that has been demonstrated to have a significant effect on risk: the time since the miner's last exposure to radon in the mines. The BEIR-IV and NCI-led analyses indicate that the relative risk of lung cancer decreases as the time since exposure to radon increases. Thus, the relative risk of lung cancer due to radon exposure for a miner who has been out of the mines only 5 years is substantially greater than the relative risk for a miner with the same cumulative exposure whose last exposure was 30 years ago. To put it slightly differently, for two miners with lung cancer who had the same total exposure, there is a greater chance that radon exposure is the cause of the lung cancer in the miner who developed the disease within a few years of his last mining employment than in the case of the miner who developed lung cancer 20 or 30 years after last exposure.

To assess how well the present statutory criteria reasonably advance Congress' objective -- compensating miners with lung

⁵⁰ Indeed, the 1994 NCI-led analysis confirmed this conclusion. It found that a risk model dependent on a single, constant exposure level did not adequately fit the observed data in any of the eleven miner cohorts examined. This result also suggests, of course, that the statutory criterion holding all nonsmoking miners to a single exposure standard (200 WLM) is inconsistent with the known data.

cancer where the probability of causation due to radon exposure is greater than 0.5 -- we created the relative risk model described by the criteria (or a good first-order approximation). We applied this model to the known lung cancer cases in the pooled Colorado Plateau and New Mexico cohorts to determine which would qualify for compensation under the present statutory scheme. We did the same analysis with a risk model derived directly from the pooled cohorts, and based on the BEIR-IV and NCI-led models. The results of both models are compared in Figure 3 in Appendix A. The comparison shows that a large number of miners who met the requisite level of risk according to the model based on the BEIR-IV/NCI analyses are not eligible for compensation under the present statutory criteria (Quadrant A of Figure 3). On the other hand, a smaller number of miners are eligible for compensation under the RECA criteria though, according to the latest data, they do not have the requisite level of risk (Quadrant D of Figure 3). This indicates that the risk model based on the present statutory compensation criteria is not a good description of the relationship between exposure to radon and the development of lung cancer. The obvious implication is that the statute at present does not accurately identify (and compensate) those miners whom Congress sought to compensate.

C. Alternative Compensation Criteria

Because the present statutory criteria do not condition compensation on all the factors known to affect significantly the risk of lung cancer, and, as a consequence, do not effectively ensure that the appropriate group of miners is compensated, the Committee suggests modifications to the present statutory criteria that will yield results more faithful to Congress' intentions. We propose in this section two sets of criteria, either of which should produce a "fairer" distribution of compensation: one set is based in part on a miner's cumulative exposure to radon; the second set substitutes a duration of employment factor for cumulative exposure, due to the perceived shortcomings of the former. We recommend, however, that both sets be incorporated into any new statutory scheme, and that claimants who qualify under either set of criteria be eligible for compensation.

1. Description Of The Relative Risk Model Used To Derive Proposed Alternative Criteria, And Model Parameters

To derive more accurate compensation criteria, we employed a risk model based on the analytical approach developed in the BEIR-IV and NCI models. We applied this model to the pooled data from the Colorado Plateau and New Mexico cohorts. The patterns of risks identified by our model were consistent with those found by the BEIR-IV and NCI analyses. The analyses conclusively demonstrate that the relative risk of lung cancer increases with increasing exposure to radon progeny. The analyses also

demonstrate that the radon exposure-lung cancer relationship (exposure-response effect) is significantly affected by at least two factors: the miner's attained age, and the time since the miner's last exposure to radon in the mines. Although the risk of lung cancer due to radon exposure increases with cumulative exposure, the strength of that relationship decreases with attained age, and decreases with increasing time since last exposure.

The data, however, are ambiguous on the effect of smoking. Analyses of the various mining cohorts have demonstrated rather conclusively that there is a synergistic relationship between exposure to radon and smoking -- that is, the lung cancer risk to a uranium miner who smokes is greater than the sum of the risk for exposure to radon and the risk for smoking. The data do not conclusively indicate, however, the exact nature of the relationship.

An analysis of the data from the Colorado and New Mexico cohorts, which cover many of the miners eligible under the RECA, indicates that the relative risk of exposure jointly to radon progeny and smoking is consistent with a multiplicative association -- the combined risk is a product of the two risks. A multiplicative association implies that the proportional increase in risk of lung cancer due to radon progeny exposure is the same in nonsmokers and smokers.⁵¹ However, the data are

⁵¹ If the association is truly multiplicative, the proportional increase in the risk of lung cancer due to exposure (continued...)

"consistent with" a multiplicative relationship; one cannot conclude with statistical confidence that a multiplicative relationship is the true characterization of the association -- that is, the result could be due to chance alone.

Although the data are consistent with a multiplicative relationship, the best estimate of the association between the joint exposure to radon progeny and smoking and the risk of lung cancer is sub-multiplicative -- the combined risk is greater than the addition of the two risks, but less than the product of the risks. A sub-multiplicative relationship implies that the effect of radon exposure is greater in nonsmokers than in smokers; that an equivalent dose of exposure increases the relative risk of lung cancer in a nonsmoker more than in a smoker.

Although a sub-multiplicative association best describes the data, again, one cannot be sure, statistically speaking, that this describes the actual relationship. The data are only consistent with a sub-multiplicative relationship; the possibility such an association is due to chance cannot be statistically eliminated. More problematical, there is a limited amount of data on the incidence of lung cancer among nonsmokers, and the resulting lack of adequate statistical power makes it difficult to estimate precisely the strength of the association -

⁵¹(...continued)
to radon (i.e., the relative risk) will be the same in smoking and nonsmoking miners. The lung cancer rate in smokers is significantly greater (10 times or more) than in nonsmokers. Consequently, the absolute increase in the risk (i.e., the number of excess lung cancer deaths) will be greater in smokers than in nonsmokers.

- the "degree of submultiplicativeness." Because of the difficulty in estimating with precision the strength of the association, most researchers and committees examining mining populations have opted to apply a multiplicative model, and, therefore, do not differentiate between smokers and nonsmokers in determining the excess relative risk posed by exposure to radon progeny.⁵²

The conclusion in the BEIR-IV and NCI analyses that the data are consistent with a multiplicative relationship has led some scientists (and miners' advocates) to criticize the present statutory criteria as unfairly discriminating against miners who smoke. The critics charge that if the proportional effect of radon exposure is equivalent in both nonsmokers and smokers, the latter should not be required, as they are in RECA, to prove a greater amount of exposure. This position is well summarized by Dr. Jonathan Samet, one of the leading researchers on the incidence of disease in uranium mining populations, and one of the authors of the 1994 NCI pooled analysis:

I think another problem in the Act is the handling of smokers who, in essence, are penalized by their unfortunate addiction and are required to have higher levels of exposure and, in essence, a higher level of risk before they are compensated.

...

For radon progeny and cigarette smoking, the available evidence suggests synergism between

⁵² This is also the approach presently taken by the Environmental Protection Agency in devising appropriate risk models to estimate the lung cancer risk from residential radon exposure. See EPA, TECHNICAL SUPPORT DOCUMENT FOR 1992 CITIZEN'S GUIDE TO RADON (1992) (EPA Publication 400-R-92-011).

the two agents. In the presence in such synergism, the higher exposure requirements for smokers implies that for compensation they must accept a higher relative risk than never smokers, ...⁵³

The Advisory Committee echoed this same concern.⁵⁴ If these criticisms are correct, smoking should no longer be a factor in the compensation analysis.

Nonetheless, the Committee believes that the eligibility criteria should continue to include a miner's smoking status. We have concluded, for the reasons stated below, that the most appropriate risk model for the purposes of RECA should presume a sub-multiplicative effect between exposure to radon progeny and smoking.

First, as emphasized above, an analysis of the latest data from the Colorado Plateau and New Mexico cohorts suggests that a sub-multiplicative model is the best estimate of the association. Moreover, the 1994 NCI-led analysis confirmed that a sub-multiplicative association is consistent with the data from the six (of eleven) cohorts with smoking information. A pooled analysis of these six cohorts indicated that the exposure-response effect (the increase in the risk of lung cancer due to

⁵³ Oversight of Radiation Exposure Compensation Act: Hearings before the Senate Committee on Labor and Human Resources, 103rd Cong. 1st Sess. 19, 21-22 (1993) [hereinafter Hearings on Oversight of RECA].

⁵⁴ FINAL REPORT, supra note 9, at 814 ("[The NCI] analysis finds little evidence to support a distinction between smokers and nonsmokers").

exposure to radon progeny) in nonsmokers is approximately 3 times the effect in smokers, implying a sub-multiplicative association.

Second, the present statutory criteria would seem to reflect Congress' desire that miners not be compensated for lung cancers most likely caused by smoking. It is well established, of course, that smoking causes lung cancer. The greater exposure standards in the Act for smokers strongly suggest that Congress wanted at least some measure of assurance that miners would be compensated for the effects of exposure to radon, not smoking. Thus, where the data are uncertain it seems appropriate to resolve the question in favor of the policy expressed by Congress.

Third, if one derives a separate set of criteria for nonsmokers, the resulting exposure levels are substantially lower than those derived from the set of all miners without regard to smoking status. This necessarily implies that selection of a multiplicative association will work to the disadvantage of nonsmokers.⁵⁵ We believe that in resolving this uncertainty, we

⁵⁵ The converse is not necessarily true: selecting a submultiplicative association does not necessarily disadvantage smoking miners. If one develops compensation criteria for smokers based on data segregated by smoking status, the resulting exposure levels are not appreciably greater than levels generated for the combined data sets. This is because the majority of uranium miners were smokers. See Hornung, *supra* note 22, at 52 (83% of miners in the Colorado Plateau cohort reported they are current or former smokers as of 1986).

should favor nonsmokers, a population in which an alternative major risk factor for lung cancer is absent.⁵⁶

2. Proposed Alternative Compensation Criteria

Using the relative risk model described above, and explained in detail in Appendix A, we generated two sets of compensation criteria -- one based on a miner's cumulative radon exposure, the other on the miner's duration of employment underground -- that will more accurately reflect the intent of Congress than the present statutory criteria. Each set of criteria is captured in a matrix that identifies the relevant variables which must be known. (See Tables in Appendix A). Table 3(a) sets forth the criteria generated by the risk model based on a miner's cumulative radon exposure; Table 4(a) sets forth the criteria generated by the risk model based on a miner's duration of employment.

At the risk of complicating matters, however, we put forth two additional, alternative matrices for each set of criteria (Tables 3b-c; 4b-c); the selection of the appropriate matrix

⁵⁶ Our analysis of the cohort data suggested that there may be a somewhat greater exposure-response effect in Native American (Navajo) miners than in non-Native American miners. Thus, an equivalent exposure to radon progeny in the mines may increase the relative risk of lung cancer in Native American miners more than in non-Native American miners. However, the difference in the effect of radon was estimated very imprecisely and was not close to statistical significance. In the pooled data from the two cohorts, there was a total of 35 Native American lung cancer cases -- too few to determine to an acceptable degree of confidence any statistically different effects in Native American miners.

rests on a policy choice we leave to the Administration. These two additional matrices in each set contain the criterion representing the best estimate derived from the risk model but modified to take into account statistical uncertainty. The process of statistical risk estimation gives rise to multiple sources of uncertainty. Thus, the miner's true relative risk lies somewhere within an interval defined by the computed criterion multiplied or divided by an amount that represents the uncertainty, or potential error. The size of the interval will depend on the level of assurance desired that the true relative risk lies somewhere within the interval -- i.e., the extent to which the uncertainty is accounted for. The greater the desired assurance, the larger the interval.

One known source of uncertainty that we can reasonably approximate is the statistical sampling variability.⁵⁷ We recommend that the criteria generated by the risk model be modified to account for this uncertainty. The alternative matrices, therefore, contain criteria modified to account for this uncertainty to two separate levels of assurance: they represent the upper bound of the interval that captures the miner's true relative risk with either 80% assurance (Tables 3b

⁵⁷ This measure is different from the uncertainty due to the inaccuracy or imprecision of any individual miner's computed exposure to radon progeny. See infra pp. 40-41. The potential error introduced by the use of the mine data arises from the fact that the measured exposure level may not be a true measure of the miner's exposure. The uncertainty associated with statistical sampling, which we account for here, is due to the process of randomly sampling data.

and 4b), or 90% assurance (Tables 3c and 4c).⁵⁸ There is no overwhelming scientific justification for selecting one of these matrices over the other: the choice depends on the level of assurance desired, which we believe is ultimately a policy decision, and beyond our warrant.

Tables 3a-c set out exposure criteria that condition compensation for miners, distinguished by smoking status, on two variables: attained age and time since last exposure. We did not use individual values for the latter two variables; for simplicity, both attained age and time since last exposure were categorized into four and three levels, respectively. Thus, a miner's attained age is either less than 50, 50-59, 60-69, or over 70. Similarly, time since last exposure is measured as less than 10 years, 10-19 years, or 20 or more years. Tables containing individual exposure criterion for every possible combination of attained age and time since last exposure would be too cumbersome to use.

Numerous critics, including most recently the President's Advisory Committee, have expressed concern with compensation criteria based on cumulative exposures because of limitations

⁵⁸ The interval defined by the amount of uncertainty, of course, covers values both greater than and less than the listed criterion. Thus, it is equally probable that the miner's true relative risk lies above the criterion than below. Nonetheless, we recommend that the upper boundary be used because it minimizes the number of false negatives, i.e., claimants rejected though their true relative risk is equal to or greater than the designated criterion. As a consequence, of course, there will be an increase in the number of false positive claims compensated.

associated with the existing historical exposure data.⁵⁹ There is particular concern that historical measurements of radon levels do not exist for many mines or for many work areas within mines. These historical radon measurements are used by the Program to calculate a claimant's cumulative WLM exposure.⁶⁰ There is a distinct possibility, therefore, that the calculated estimates of claimants' exposures are inaccurate and imprecise. Thus, it is argued, some miners likely are being denied compensation unfairly because their calculated exposure -- which fails to meet the relevant statutory minimum -- is not accurate.

In response to this criticism, we created an alternative set of criteria, set out in Tables 4a-c, that relate the risk of lung cancer to a miner's duration of employment in underground uranium mines. These criteria are dependent on attained age, time since

⁵⁹ FINAL REPORT, supra note 9, at 814; Hearings on Oversight of RECA, supra note 53, at 20, 22 (testimony of Jonathan Samet, M.D., M.P.H.); Richard W. Hornung & Theodore J. Meinhardt, Quantitative Risk Assessment of Lung Cancer in U.S. Uranium Miners, 52 HEALTH PHYSICS 417, 422-24 (1987) (quantifying the error in mine exposure data).

⁶⁰ The Program calculates a claimant's cumulative exposure by aggregating exposures in mines where the claimant can prove employment over the period of employment. Exposure levels in a given mine and for a given year are generated by an algorithm developed by Dr. Archer and his colleagues for use in the Public Health Service study of the Colorado Plateau Miners. Actual measurements are used for a mine and year, where they exist; where no measurements exist for any given year in the mine, measurements from the same mine within a span of adjoining years (in either direction) are averaged and used. Where there is insufficient data for interpolation or projection, an average of measurements from mines in the same locality are used. Estimates of radon levels before 1950, when a systematic sampling program began, are based on knowledge of ore bodies, ventilation practices and earliest measurements. 28 C.F.R. § 79.34(g); see LUNDIN, supra note 22, at 32.

last exposure and the calendar year of first employment.⁶¹ This last factor is significant because the mean level of radon in the mines declined appreciably over the years.⁶² See Figure 1 in Appendix A. Again, for simplicity, we have grouped calendar years into categories that roughly reflect appreciable differences in the average radon level in mines.

Either set of criteria, we believe, represents a significant improvement over the present statutory scheme.⁶³ We recommend

⁶¹ As noted above, we accept the Advisory Committee's suggestion that the Act be modified to incorporate a threshold criterion based on duration of employment, as opposed to exposure to radon progeny. See FINAL REPORT, supra note 9, at 813. The data indicate, however, that a model based on a single factor such as duration of employment does not accurately describe the relative risk of lung cancer in miners. Cf. supra note 50 and accompanying text. Including the variables noted above enhances the accuracy of the relative risk model, ensuring a fairer distribution of compensation.

⁶² In this analysis, we used calendar year of first employment underground as a surrogate for each calendar year of underground employment. The data on the relevant mining cohorts reveal that only a small proportion of miners had significant exposure-free time periods in their work histories. Although it is reported that many miners were itinerant, gaps in work history were generally a matter of months. Because the radon levels in the mines generally declined over a miner's period of employment, the use of calendar year of first employment as a surrogate likely overestimates any given miner's exposure to radon.

Moreover, the experience of the Program, from what we have been told, suggests that for most miners, employment history and year of first mining can be determined with some accuracy -- at least greater accuracy than cumulative exposure to radon based on the historical mine measurements. Consequently, there is much less chance that a miner will be misclassified using these criteria.

⁶³ The criteria defined by Tables 3 and 4 were applied to the 470 known lung cancer cases occurring in the pooled Colorado Plateau and New Mexico data to determine how many would be eligible for compensation. Using the model based on cumulative
(continued...)

that both sets of criteria be used, so that a claimant who qualifies under either criteria is compensated.

On the one hand, the employment duration model has distinct advantages. As the Advisory Committee and others have noted, the use of the historical mine data is perceived as unfair by many. An approach based on employment duration is more transparent and intuitive, and may be more readily accepted. Moreover, the criteria generated by this model can be understood and used by miners and their survivors much more easily than exposure-based criteria to determine if they are eligible for compensation.

On the other hand, the exposure-based model has the advantage of being more accurate: it better "fits" the observed data, indicating that it more accurately describes the relationship between exposure to radon progeny and risk of lung cancer. This implies necessarily that use of this model will result in a fairer distribution of compensation.

We believe that if the employment duration-based criteria are used, the alternative exposure-based criteria must also be used. It is possible for a claimant to be denied compensation under the employment-based criteria we recommend (Tables 4a-c),

⁶³(...continued)
exposure: 388 cases (83%) would be eligible under Table 3(a) (no modification for uncertainty); 401 cases (85%) would be eligible under Table 3(b) (80% level of assurance); and 403 cases (86%), would be eligible under Table 3(c) (90% level of assurance). See Tables 5(a)-(c). Using the model based on duration of employment: 349 cases (74%), would be eligible under Table 4(a); 394 cases (84%) under Table 4(b) (80% assurance); and 408 (87%) under Table 4(c) (90% assurance). See Tables 6(a)-(c). Under the present RECA criteria, 68% of the observed lung cancers would be attributed to radon exposure and compensated.

yet qualify for compensation under the exposure-based criteria (Tables 3a-c). We believe it would be unjust to deny compensation to any claimant who can demonstrate eligibility under the exposure-based criteria. This model is the more accurate, so a claimant who can meet its criteria has demonstrated the most legitimate claim to compensation.

3. Proposed Modifications to the Definition of Smoking in the Regulations

Our recommendation that smoking continue to be a factor in the compensation calculus requires us to face two additional questions that relate to the definition of smoking in the regulations. As noted above, the regulations define a smoker as an individual who has smoked at least one (1) pack-year of cigarette products⁶⁴, and, correspondingly, a nonsmoker is defined as an individual who has never smoked cigarette products or has smoked less than one pack-year.⁶⁵ To determine if a claimant was/is a smoker, the regulations require him (or his surviving beneficiaries) to submit all medical records of specified types created within the six month period before and after the date of diagnosis of lung cancer.⁶⁶ If any of these records, or any of the historical health and mining survey records held by the Program, contain information indicating that

⁶⁴ 28 C.F.R. § 79.31(f).

⁶⁵ Id. § 79.31(g).

⁶⁶ Id. § 79.37(a).

the miner was a smoker, even if no smoking quantity is noted, he is presumed to be a smoker.⁶⁷

We have learned that in practice, however, the Program does not require the so-called "six months documents" from a miner if he is designated a nonsmoker in certain historical health survey databases held by the Program. Where these historical records code the miner as a smoker, he is presumed to be a smoker. Where the miner is not listed in any of these databases, or is listed but smoking information is either not provided or is unclear, the miner is required to submit the so-called "six months documents" to overcome a presumption of smoking.⁶⁸

This regulatory scheme has been heavily criticized in at least two respects: it discriminates against Navajo, who were generally nonsmokers or only occasional smokers; and it does not allow former smokers who stopped smoking for many years to be treated as nonsmokers. We believe both of these concerns are justified, and can be accounted for by modifications in the regulations.

⁶⁷ Id. § 79.37(b).

⁶⁸ The Program has imposed this presumption to force claimants to submit the "six months documents" when no or incomplete smoking information is available. Absent the presumption, a claimant who was a smoker is unlikely voluntarily to submit the necessary medical records documenting smoking. It seems reasonable to assume that some reference as to the claimant's smoking history will be made by a physician in the appropriate medical records within six months of the date of diagnosis of lung cancer. If no such reference exists, it seems reasonable to assume the claimant is a nonsmoker.

(a) Navajo and Smoking⁶⁹

Critics have charged that the definition of smoking, in combination with the presumption a miner is a smoker, unjustly results in the misclassification of many Navajo as smokers, which obviously increases the burden of proving eligibility. We heard from numerous credible sources (including Navajo Nation officials, present and former Indian Health Service physicians, and scientists who study the Navajo mining population), that the incidence of smoking among Navajo was very low, particularly among the generation that mined during the years specified in the statute. According to these sources, there is a cultural bias against smoking, so the majority of Navajo smoked only for ceremonial purposes if at all, and those who did smoke did so only occasionally and significantly fewer cigarettes than their non-Navajo counterparts.

However, many Navajo apparently classified themselves as smokers when asked about smoking status during a health study or in response to questioning by a physician. Thus, for many Navajo

⁶⁹ The significant majority of Native American underground uranium miners were Navajo, see Victor E. Archer, et al., Respiratory Disease Mortality Among Uranium Miners, 271 ANNALS N.Y. ACAD. SCI. 280, 290 (1976) (780 non-Whites in Colorado Plateau cohort, majority of which were Navajo); see also Robert J. Roscoe, et al., Mortality Among Navajo Uranium Miners, 85 AM. J. OF PUBLIC HEALTH 535, 536 (1995) (update of Colorado Plateau cohort; 775 of 779 non-Whites in cohort as of 1990 were Native Americans, majority of which were Navajo), and we have therefore focused on data specific to that Tribe. Because few non-Navajo Native Americans are represented in the underground mining cohorts, we have phrased this recommendation with specific reference to the Navajo.

there is a reference to smoking in the historical records that they cannot overcome.

The contention that Navajo were never-smokers or only light smokers is confirmed in the research literature published before the RECA was enacted.⁷⁰ The first reported study of the Colorado Plateau miners, published in 1971, found that 62% of the Native American miners in the cohort were never smokers, and the median consumption of cigarettes by Native American smokers was only 4 cigarettes per day. A later report summarized the limited additional data:

Of a sample of adult Navajos admitted to the Phoenix Public Health Service Indian Hospital from 1961 through 1965, only 4.3 per cent smoked more than one pack of cigarettes daily. In a 1977 survey of Navajos, only 13 per cent smoked and only 6 per cent of the smokers [.78% of the sample] consumed at least one pack daily.⁷¹

The cohort data we use to generate our alternative compensation criteria strongly bears out the concern that Navajo

⁷⁰ See Jonathan M. Samet, et al., Mortality From Lung Cancer and Chronic Obstructive Pulmonary Disease in New Mexico, 78 AM. J. OF PUBLIC HEALTH 1182-1186 (1988); Samet, Uranium Mining and Lung Cancer in Navajo Men, supra note 22, at 1483; Maurice L. Sievers, Cigarette and Alcohol Usage by Southwestern American Indians, 58 AM. J. OF PUBLIC HEALTH 71-82 (1968).

⁷¹ Samet, Uranium Mining and Lung Cancer in Navajo Men, supra note 22, at 1383. While it is arguable that the low incidence of smoking among Navajo generally does not translate to the Navajo males who mined uranium, this report suggests the contrary. The report was a population-based case-control study of 32 lung cancer cases among Navajo males. Of the 32 subjects with lung cancer, 23 reported uranium mining employment. Smoking data were available on 21 of the 23 uranium miners: 8 were never smokers, 8 consumed less than 3 cigarettes/day; and 5 consumed 4-8 cigarettes/day. Id.

are being misclassified. In the Colorado Plateau and New Mexico cohorts combined, 47% of Native Americans classified themselves as smokers; yet among this group of self-reported smokers, the average consumption of cigarettes was .3 packs per day, or 6 cigarettes. In contrast, 81% of non-Native Americans in the combined cohorts were ever smokers, and of this group the average consumption was 1.2 packs/day.

This suggests that many Navajo may be held to the present 300 WLM or 500 WLM standard though their smoking history suggests that they probably do not meet the definition of smoker in the regulations. Although it is generally believed that any smoking increases an individuals risk of lung cancer, the smoking rate among Navajo as borne out by the literature is so minor, and so much less than the average among White miners, that their increased risk due to smoking is relatively insignificant, as a group. We believe, consequently, it is inappropriate to presume on the basis of a bare self-reported reference in the historical records, that a Navajo meets the definition of smoking in the regulations. We believe it is more appropriate to presume the opposite: a Navajo miner should be presumed to be a nonsmoker absent evidence in the historical records specifically documenting cigarette consumption in excess of (or at a rate likely to lead to an excess of) one pack-year. We recommend,

therefore, that the regulations be modified accordingly to presume all Navajo are nonsmokers.⁷²

(b) Cessation of Smoking

The regulations irrevocably presume that a claimant who once was a smoker is always a smoker. No provisions are made for smokers who have stopped for such an extended period of time that the added risk of lung cancer due to their smoking is no longer significant. The absence of such a provision has been criticized, particularly in light of the minimal smoking history required to meet the definition of smoking in the regulations.

It is now accepted that the risk of lung cancer in ever smokers drops after the cessation of smoking.⁷³ The reduction in the risk of lung cancer can be quite appreciable over time,

⁷² The background rate of lung cancer among Navajo is quite low -- much lower than the rate for non-Hispanic Whites. See Charles L. Wiggins, et al., Cancer Mortality Among New Mexico's Hispanics, American Indians, and Non-Hispanic Whites, 1958-1987, 85 J. OF THE NAT'L CANCER INST. 1670, 1672 (1993). During the period 1958-62, the rate of cancer of the respiratory system (including cancers of the lung, trachea and bronchus) among all New Mexico Native Americans, a category composed primarily of Navajo, Pueblo and Apache, was 5.3/100,000; the rate among Non-Hispanic Whites was 30.1/100,000. Id. at 1670. During the period 1983-87, the rate among New Mexico Native Americans had increased to 13.1/100,000; the rate among New Mexico Non-Hispanic Whites had increased to 57.4/100,000. See also NEW MEXICO TUMOR REGISTRY, U. OF NEW MEXICO CANCER RESEARCH AND TREATMENT CENTER STATE OF NEW MEXICO: MALIGNANCIES DIAGNOSED 1993.

⁷³ American Thoracic Society, Cigarette Smoking and Health, 153 AM. J. OF RESP. CRIT. CARE MED. 861, 864 (1996).

though the risk is not generally believed ever to drop to the background risk of a never-smoker.⁷⁴

The extent of the reduction in risk and the rate of decline are believed to depend not only on the time since cessation of smoking, but also on a number of other factors, including the age at cessation, and the intensity and duration of cigarette use.⁷⁵ According to the data, for smokers who smoked 20 or more cigarettes per day, the relative risk of lung cancer decreases to less than two times the background rate of never-smokers after approximately twenty years.⁷⁶ At this point, where the relative risk in an ex-smoker is less than twice that of a never-smoker, one cannot say that if the ex-smoker develops lung cancer, it is more likely than not due to the smoking; in the statistical terms we have used above, below this point the probability of causation from smoking is less than 0.5. For smokers who smoked less than 20 cigarettes per day, the relevant point is less certain; based on the lower confidence limits of the available data, we estimate

⁷⁴ Ian T. Higgins & Ernst L. Wynder, Reduction in Risk of Lung Cancer Among Ex-smokers with Particular Reference to Histologic Type, 62 CANCER 2397, 2401 (1988); Eugene Rogot & James L. Murray, Smoking and Causes of Death Among U.S. Veterans: 16 Years of Observation, 85 PUB. HEALTH REP. 213, 219 (1980).

⁷⁵ See Lawrence Garfinkel & Edwin Silverberg, Lung Cancer and Smoking Trends in the United States Over the Past 25 Years, 41 CA-A CANCER JOURNAL FOR CLINICIANS 137, 143-44 (1991); U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, The Health Benefits of Smoking Cessation 122-126 (1990).

⁷⁶ Garfinkel & Silverberg, supra note 75, at 143; Higgins & Wynder, supra note 74, at 2398.

that the relative risk of lung cancer decreases to less than two after 15 years.⁷⁷

We recommend that the definition of smoking be modified to classify as a nonsmoker any ex-smoker who can prove through contemporaneous medical records that he stopped smoking at least 15 years prior to the diagnosis of lung cancer. We favor the 15 year threshold for two reasons. First, the data on smoking are uncertain. Second, the definition of smoking in the regulations covers a considerable range of smoking rates; similarly, the age distribution of ex-smoking miners at the time of cessation of smoking, given that the Act covers the period 1947 to the present, is likely quite broad. Both factors are known to affect the rate of decline in lung cancer risk. Their potential variability among the population at issue suggests to us erring on the side of liberality.

D. Comparison of the Compensable Risk Thresholds Required of Uranium Miners and Downwinders

A troubling criticism of the RECA is the charge that there is an unjustified disparity in the level of risk that the miners, on the one hand, and the downwinders, on the other hand, must establish to be eligible for compensation. The Act, critics maintain, imposes more stringent criteria on miners, effectively forcing them to demonstrate that they were subjected to a much

⁷⁷ See Higgins & Wydner, supra note 74, at 2398.

higher risk of disease as a result of their exposure than is required of downwinders.⁷⁸

The Committee believes that the criticism has merit. While we are sympathetic to the concern, for the reasons discussed below, we do not recommend any particular solution.

The Act does not impose any specific minimum exposure criteria for downwinders. To be eligible for compensation as a downwinder, an individual must demonstrate that he/she resided for a specified period of time within a defined geographic area during a designated period.⁷⁹

The legislative history suggests Congress did not require a specific dose threshold of downwinders because no reliable dose data reportedly were available from which to estimate their exposures and deduce the appropriate causal relationships.⁸⁰

⁷⁸ In its commentary accompanying Recommendation 7, the Advisory Committee noted that the compensation criteria for uranium miners in RECA are "far more stringent" than for the other two groups eligible for compensation. FINAL REPORT, supra note 9, at 814. The effect, the Advisory Committee noted elsewhere, is to set a much higher risk threshold for compensation for the miners than for either the downwinders or the on-site participants. Id. at 578. The exposures to ionizing radiation of residents of surrounding areas were likely lower than those of personnel stationed at the test sites. The disparity in comparable risk for compensation is greatest, therefore, between miners and downwinders. For this reason, we focus on downwinders and not on on-site participants.

⁷⁹ The Act requires the claimant to have been physically present in any of eight counties in Utah, six counties in Nevada, or a defined geographic area of Arizona for either a period of at least two years during the period January 21, 1951 to October 31, 1958, or for the entire period beginning June 30, 1962 and ending July 31, 1962. P.L. 101-426, § 4(a)(2), 104 Stat. 921.

⁸⁰ See 131 CONG. REC. 32075-77 (1985) (statement of Sen. Hatch).

The residency and proximity requirements are intended to serve as a proxy for exposure. As stated by Senator Hatch during the debate preceding the passage of the RECA, "[i]n order to qualify for compensation under this Bill, the fallout victims must have lived in the designated area long enough to have been exposed to repeated doses of fallout."⁸¹

The scientific literature, however, does not at present support an association between exposure to fallout and development of malignancies.⁸² Based on the latest data, the risk of developing any of the malignancies for which downwinders are eligible under RECA, as a result of estimated exposures typical of the downwind population, simply is not of the same magnitude as the proven risk of lung cancer associated with the doses of radon typical of the uranium mining population between the period 1947 and 1971.⁸³ As a consequence, downwinders are

⁸¹ 136 Cong. Rec. S11752 (daily ed. Aug. 1, 1990).

⁸² See John D. Boice, Jr. & Jay H. Lubin, Occupational and Environmental Radiation 15-18 (April 23, 1996) (manuscript submitted for publication; on file with the Committee) (citing studies).

⁸³ There are substantial differences between the two populations in both the effective dose of radiation received and the resulting cancer risk at threshold doses for compensation. It is estimated that 97% of the downwind population received an effective external radiation (gamma) dose of less than .01 Sievert (Sv); the 200 WLM exposure minimum in the Act corresponds to an effective dose of 1 Sv. Jonathan M. Samet & Fred A. Mettler, Jr., The Radiation Exposure Compensation Act: Well Intended But Needing Revision 5-6 (unpublished manuscript on file with the Committee). Miners are not compensated, therefore, until they have been subject to an effective dose 100 times that of downwinders.

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compensated though the risk resulting from the effective dose they received is many times lower than the risk to uranium miners at the exposure levels required for compensation.⁸⁴ Put another way, a miner has to prove a probability of causation between his exposure and resulting disease that is substantially greater than the probability of causation demanded of most downwinders between their likely exposure and any compensable radiogenic cancer.

It can be argued that the disparity in threshold risks for compensation between miners and downwinders may be justified in part by the fact that the former were exposed occupationally, while the latter were exposed environmentally and thus did not voluntarily assume any risk or receive any compensation for risks, known or unknown. Additionally, the disparity in risk is reflected in the different levels of compensation to the two

⁸³(...continued)

Additionally, an exposure of 500 WLM, the statutory minimum for most smoking miners, (assuming a 30 year-old miner exposed at a constant rate of 100 WLM for 5 years, and using the 1994 NCI risk model), increases a miner's estimated lifetime lung cancer risk, conservatively, by approximately 20 percent. *Id.* An effective dose of .01 Sv, on the other hand, is estimated to raise a downwinder's lifetime cancer risk by approximately .05 percent. *Id.* The incidence of lung cancer among uranium miners in the Colorado Plateau cohort is over 5 times the expected rate in the area population. Robert J. Roscoe, An Update of Mortality Among Uranium Miners from the Colorado Plateau Study Group, AM. J. OF INDUS. MED. (forthcoming 1996). On the other hand, the incidence of the compensable malignancies (including childhood leukemia and thyroid cancers) in the downwind population is not significantly elevated. See Boice & Lubin, supra note 82, at 15-18 (citing studies).

⁸⁴ Hearings on Oversight of RECA, supra note 53, at 18, 21 (testimony of Jonathan Samet, M.D., M.P.H.).

groups -- downwinders receive \$50,000 in compensation, one-half the amount paid to eligible miners.

Notwithstanding these differences, the lack of comparable treatment for miners seems unfair in light of the well-accepted association between exposure and lung cancer, and the documented history of the federal government's failure to protect the uranium mining population.⁸⁵ The Act curiously requires the one group that can conclusively establish that the government's misconduct proximately caused them substantial harm to meet the highest threshold for compensation.

Nonetheless, we have concluded that we can do no more than report the validity and potency of this criticism. We can conceive of only a limited number of ways to begin to address this problem within the existing statutory framework: all require the exercise of policy judgment.⁸⁶ The merits of various alternative approaches cannot be judged principally by reference to technical or scientific standards, as we believe is true in

⁸⁵ See FINAL REPORT, supra note 9, at 579.

⁸⁶ For example, the Act can be modified so that the level of compensation for miners is proportionate to the calculated probability of causation. Under this scheme, a miner would receive some percentage of a maximum amount depending on the strength of the causal association -- an estimate of the likelihood that the miner's exposure to radon caused his disease. This is apparently the approach recommended by a number of expert scientific bodies, including the National Council on Radiation Protection and Measurements. See Hearings on Oversight of RECA, supra note 53, at 18-19 (testimony of Jonathan Samet, M.D., M.P.H.); Samet & Mettler, supra note 83, at 6. Cf. Armstrong & Theriault, supra note 29, at 162. This solution, however, does not directly address the issue of the comparability in the level of risk required for compensation among the various eligible groups.

each case where we have advanced a recommendation. To determine which solution among the alternatives is "best" or most appropriate ultimately requires reference to political and social concerns. We do not believe we were empaneled to make such judgments, and respectfully defer to those with the appropriate expertise and responsibility.

III. ISSUES RELATING TO COMPENSATION FOR NONMALIGNANT RESPIRATORY DISEASES

A. Statutory and Regulatory Background

In addition to compensating cases of lung cancer, the Act compensates miners (or surviving family members) who suffer (or suffered) from specified nonmalignant respiratory diseases. The Act limits the compensable nonmalignant diseases to:

[F]ibrosis of the lung, pulmonary fibrosis, and corpulmonale related to fibrosis of the lung; and if the claimant, whether Indian or nonIndian, worked in an uranium mine located on or within an Indian Reservation, the term shall also include moderate or severe silicosis or pneumoconiosis; ⁸⁷

The Act explicitly conditions compensation for these nonmalignant disorders on proof the claimant was exposed to minimum levels of radiation; the required exposure levels are the same as those specified for compensation for lung cancer. Thus, to qualify for compensation for a nonmalignant disorder, a nonsmoking claimant must have been exposed to 200 WLMs of radiation in the mines; a smoking claimant who developed the nonmalignant disorder before age 45 must have been exposed to 300 WLMs of radiation; and a smoking claimant who developed the

⁸⁷ P.L. 101-426, § 5(b)(3), 104 Stat. at 923. The Advisory Committee did not specifically identify any of the nonmalignant disease provisions in the Act, or implementing regulations, as possibly unjust or inappropriate. Neither did the Interagency Working Group in its charge to the Committee. Nonetheless, we were confronted with numerous criticisms of the nonmalignant disease statutory provisions and corresponding regulations during our investigation, many of which raised issues of obvious importance to the administration of the Act. To perform our function competently, we conclude, we must address these issues.

nonmalignant disorder after age 45 must have been exposed to 500 WLMs of radiation.⁸⁸

The regulations require that a claimant with a compensable nonmalignant disease prove exposure in the same manner as a claimant seeking compensation for lung cancer.⁸⁹ That is, a claimant must first document underground uranium mining employment during the time period and within the states specified in the Act, either through information held by the Program or by submitting trustworthy historical records verifying employment.⁹⁰ The Program then calculates the claimant's cumulative exposure according to the algorithm described in the regulations.⁹¹ The regulations also require written medical documentation proving that the claimant suffers (or suffered) from one of the compensable nonmalignant diseases.⁹² To establish this criterion, the regulations presently require most claimants to submit appropriate medical documentation demonstrating both the presence of the nonmalignant disease and physiological (functional) impairment.⁹³ The claimant is

⁸⁸ Id. § 5(a), 104 Stat. at 922.

⁸⁹ See 28 C.F.R. § 79.34 (1995).

⁹⁰ Id. § 79.33.

⁹¹ See id. § 79.34.

⁹² Id. § 79.36.

⁹³ Id. § 79.36. Claimants who are or were part of the PHS or NIOSH health studies, or any other federally-funded health study, may not need to submit medical documentation. The Program will accept as proof of medical condition verification by the PHS, NIOSH or federally-funded study that they possess medical records of the claimant containing a diagnosis of a compensable

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required to prove the presence of the disease through chest radiographs (x-rays) interpreted by two certified "B"-readers -- physicians specially certified by National Institute for Occupational Safety and Health (NIOSH) as proficient in the classification of radiographic evidence of pneumoconioses -- and to prove impairment through pulmonary function tests or arterial blood gas studies documenting reduced pulmonary function.⁹⁴

A number of the statutory criteria for the nonmalignant disorders have been criticized as being scientifically unjustified or redundant. Additionally, the regulations defining proof of medical condition have been criticized. Advocates complain principally that the regulations should not require proof of disease process; that the proof of disease required is highly subjective; that the regulations do not allow claimants to submit accurate and trustworthy proof of disease process; and, that they establish standards for functional impairment that are ethnically-biased.

⁹³(...continued)
nonmalignant disease. Id. § 79.36(b),(c). Further, the regulations do not necessarily require proof of both physical abnormality and functional impairment in the case of a deceased miner. Surviving beneficiaries of a deceased miner are required to submit chest radiographs alone, or if they do not exist, other trustworthy medical records containing appropriate statements of diagnosis; proof of functional impairment is not required for obvious reasons. Id. § 79.36(d)(i). The regulations do require proof of both disease and functional impairment from all living miners. Id. § 79.36(d)(ii).

⁹⁴ Id. § 79.36(d)(ii).

B. Recommendations Regarding Statutory Compensation Criteria

1. Proof of Radon Progeny Exposure As A Condition For Compensation For Pulmonary Fibrosis

The Act, as explained above, explicitly ties compensation for pulmonary fibrosis⁹⁵ to proof the claimant was exposed to minimum levels of radon progeny.⁹⁶ Thus, the Act necessarily presumes that exposure to radon progeny causes or significantly contributes to the development of the specified nonmalignant diseases.

Critics have argued that the association between exposure to radon and pulmonary fibrosis is not justified by the available scientific evidence; that, instead, the risk of fibrosis is likely related to a combination of agents in the mining environment, (including, possibly, radon), some or all of which are not accounted for in the statutory criteria. This criticism was expressed most succinctly by Dr. Samet, in testimony before the Senate Committee on Labor and Human Resources:

The linking of compensation for non-malignant respiratory diseases and achieving exposure threshold for radon progeny is not biologically appropriate. Whether an entity of radon-related pulmonary fibrosis has clearly occurred in former underground uranium miners remains uncertain, and, in any case, radon progeny are only some of the pollutants in uranium mines that can cause lung damage. ... Criteria for compensating

⁹⁵ The Act designates both pulmonary fibrosis and fibrosis of the lung as compensable nonmalignant diseases. These terms refer to the same disease process. We recommend that the term "fibrosis of the lung" be deleted from the Act as redundant.

⁹⁶ See P.L. 101-426, § 5(a)(2)(A), (B), 104 Stat. at 922.

non-malignant respiratory disease in underground miners which are not based on exposure to radon progeny are needed. [The] linkage is really inappropriate on a medical basis.⁹⁷

The Committee believes that this criticism is justified, and recommends that the Act be modified to condition compensation for pulmonary fibrosis on some alternative compensation criteria. Based on our review of the published data, we believe that the alternative criteria should be based at least on a claimant's duration of employment. We have not had either the time or resources to locate and analyze sufficient reliable data, assuming it exists, to recommend alternative criteria.⁹⁸

The Committee recognizes that the legislatively-presumed statutory connection between exposure to radon and the development of fibrosis is not without support. By the time Congress enacted the RECA, it had before it considerable testimony and documentation supporting an association between exposure to radon progeny and fibrosis. In hearings before the Senate Committee on Labor and Human Resources in February, 1990, Dr. Victor Archer, a physician who has spent his career studying

⁹⁷ Oversight of Radiation Exposure Compensation Act: Hearings Before the Senate Committee on Labor and Human Resources, 103rd Cong., 1st Sess. 19, 22 [hereinafter Hearings on Oversight of RECA] (testimony of Jonathan Samet, M.D., M.P.H.).

⁹⁸ There appears to be very limited historical data on dust levels in the mines during much of the time period covered by the statute. Therefore, it is questionable whether a direct quantitative assessment of the association between uranium mining and nonmalignant respiratory diseases can be estimated. It is possible, though by no means obvious, that data from other hard rock mining can be used to generate appropriate criteria.

the incidence of disease among uranium miners, and who was formerly the principal investigator for the Public Health Service's prospective epidemiological study of uranium miners of the Colorado Plateau area, testified:

There have also been a large excess of deaths among uranium miners from other lung diseases, which we group together as non-malignant respiratory diseases, NMRD. This includes pulmonary fibrosis, radiation pneumonitis, emphysema, silicosis, chronic bronchitis, chronic obstructive lung disease, and cor pulmonale. We have shown that the radiation in the mines does contribute to this group of deaths. We cannot assign radiation to any particular one of these diseases, but it does seem to contribute somewhat to all of them. We also know that there are other contributors to these diseases, such as silicate dust, cigarette smoking, chronic infections, and we are unable to really separate them out as to causation.⁹⁹

Dr. Archer's testimony was based on the results of pioneering studies he and others published in the 1960s and 1970s.¹⁰⁰

⁹⁹ Health Effects of Radiation Exposure: Hearing Before the Senate Committee on Labor and Human Resources, 101st Cong., 2nd Sess. 35 (1990) [hereinafter Hearings on Health Effects of Radiation Exposure].

¹⁰⁰ See, e.g., Victor E. Archer, et al., Respiratory Disease Mortality Among Uranium Miners, 271 N.Y. ACAD. SCI. 280-293 (1976); Joseph K. Wagoner, et al., Mortality Patterns Among United States Uranium Miners And Millers, 1950 Through 1962; Preliminary Report in, 1 RADIOLOGICAL HEALTH AND SAFETY IN MINING AND MILLING OF NUCLEAR MATERIALS 37-48 (1964); Victor E. Archer, et al., Pulmonary Function Of Uranium Miners, 10 HEALTH PHYSICS 1183-1194 (1964); Victor E. Archer, et al., Epidemiological Studies of Some Non-Fatal Effects of Uranium Mining, in 1 RADIOLOGICAL HEALTH AND SAFETY IN MINING AND MILLING OF NUCLEAR MATERIALS 21-35 (1964).

These studies and others were summarized, cited and/or submitted to Congress in later hearings.¹⁰¹

Dr. Archer explicitly affirmed the association in the following colloquy with Senator Hatch:

Sen. Hatch: In your opinion, what nonmalignant respiratory diseases did the uranium miners develop as a result of exposure to radiation?

Dr. Archer: Well, it is the group of nonmalignant respiratory diseases which I mentioned earlier, primarily pulmonary fibrosis, emphysema, chronic bronchitis, chronic obstructive lung disease, and cor pulmonale.

Sen. Hatch: And these are the types of radiation diseases that the uranium miners developed as a result of exposure to radiation?

Dr. Archer: Well, I can't say that directly because of the contribution of other items to those diseases, but we did show epidemiologically that the exposure to radiation almost certainly contributed to the development of these diseases, and they are much higher among the uranium miners than among nonmining groups.

Sen. Hatch: So you were saying there is a high probability that the exposure to radiation contributed to these diseases, these nonmalignant diseases?

Dr. Archer: Yes, yes, that's correct.¹⁰²

Explicit support for this position came from a physician who treated Navajo miners during his tenure at the Indian Health

¹⁰¹ See Impact of Past Uranium Practices: Hearings Before the Subcomm. on Mineral Resources, Development and Production of the Senate Committee on Energy and Natural Resources, 101st Cong., 2nd Sess. at 59-60 (1990) [hereinafter Hearings on Impact of Past Uranium Practices] (testimony of Lynda Taylor). This same witness appended to her testimony a paper by Dr. Joseph K. Wagoner, S.D. Hyg., explaining these findings in some greater detail, and listing all appropriate references. See id. at 69-71, 96-97 (testimony of Lynda Taylor).

¹⁰² Hearings on Health Effects of Radiation Exposure, supra note 99, at 43-44.

Service facility at Shiprock, New Mexico in the late 1970s.¹⁰³

In written testimony submitted at the same hearing at which Dr.

Archer testified, Dr. Leon Gottlieb stated:

In our Shiprock Navajo Clinic for uranium miners, we observed also, in addition to lung cancer, other lung diseases associated with uranium mining and milling. These pulmonary disorders consisted of silicosis and pulmonary fibrosis (scarring of the lung tissue). These conditions were the sequelae of inhaling rock dust and radioactive particles (radon daughters). Hazardous exposure in uranium mines may lead to malignant or non-malignant lung disease. The malignant type obviously is cancer of the lung; the non-malignant type comprises silicosis of the lung (fibrosis), and radiation pneumonitis (fibrosis).¹⁰⁴

Much of the testimony submitted to Congress in support of an association, including the testimony cited above, can be interpreted as suggesting only that the development of various nonmalignant diseases appears to be related to some agents in the mining environment, including radiation, acting alone or in combination. There is only one reference in the legislative history of which the Committee is aware advocating that compensation for the nonmalignant disorders explicitly be tied to the same exposure standards set for lung cancer compensation. That reference, interestingly, is from Dr. Archer: in testimony advocating separate exposure standards for lung cancer based on a claimant's smoking status, Dr. Archer stated:

¹⁰³ See id. at 46-49.

¹⁰⁴ Id. at 46-47.

I have been involved in a number of workers' compensation cases in about five States in the West with uranium miners, and I have been frequently asked to determine whether the particular lung cancer was more likely or not caused by the radiation in the mines. For nonsmokers, this is quite easy if their radiation exposure has been above 100 Work Level Months, ... For heavy cigarette smokers, however, this is a little more difficult. Usually above 800 [sic] Work Level Months, it is more likely than not that the lung cancer is due to radiation. But we cannot make that sort of division for the NMRD cases, so that I would recommend that if any thresholds for exposure are used, that they be set the same as for the lung cancers.¹⁰⁵

However, the most recent and analytically rigorous review of the pertinent scientific information on this issue is found in a 1992 report prepared by Dr. Samet and colleagues at the University of New Mexico School of Medicine for the Department of Health and Human Services in response to a congressional mandate in the RECA.¹⁰⁶ This report concludes that the presumed

¹⁰⁵ Id. at 41 (emphasis added).

¹⁰⁶ See JONATHAN M. SAMET & STEVEN Q. SIMPSON, REVIEW OF SILICOSIS AND OTHER PNEUMOCONIOSES IN UNDERGROUND URANIUM MINERS (Aug. 7, 1992) (Final Report Task 1); JONATHAN M. SAMET & MARION V. MORGAN, REVIEW OF SILICOSIS AND OTHER PNEUMOCONIOSES IN UNDERGROUND URANIUM MINERS: A REVIEW OF DATA SOURCES (Aug. 7, 1992) (Final Report Task 2).

Section 12 of the Act specifically provides that:

The Secretary of Health and Human Services shall submit a report on the incidence of radiation related moderate or severe silicosis and pneumoconiosis in uranium miners employed in the uranium mines that are defined in section 5 and are located off the Indian reservations.

P.L. 101-426, § 12(a), 104 Stat. at 926.

association between exposure to radon and the development of pulmonary fibrosis has not been conclusively established.¹⁰⁷ The report further concludes, consistent with the earlier hypothesis of Dr. Archer, that the disease process in miners is possibly related to exposure to other agents in the mining environment. The authors of the report noted that the existence of a distinct disease of radiation-induced pulmonary fibrosis is plausible, given our present understanding of pathogenetic mechanisms of lung injury, and the results of animal studies that report radiation-induced changes to lung structure that could be precursors of interstitial fibrosis.¹⁰⁸ Nonetheless, they concluded, the latest and most scientifically sound epidemiological studies of uranium miners cannot establish the existence of the disease because one cannot separate out the effects of radon exposure from the effects of other known or potentially toxic agents in the mining environment, such as silica, diesel exhaust, dust and other particles and gases.¹⁰⁹ The prior studies originally relied on by Dr. Archer and others, the report maintains, suffer from sufficient methodological shortcomings to render their results ambiguous.¹¹⁰

No data or analyses contradicting the conclusions of the 1992 HHS Report have been brought to the Committee's attention.

¹⁰⁷ Samet & Simpson, supra note 106, at 31-33.

¹⁰⁸ Id. at 31-33.

¹⁰⁹ Id. at 24-25, 33.

¹¹⁰ Id. at 27-29, 32.

Because the Act presently conditions compensation for nonmalignant disorders on criteria possibly unrelated to the development of fibrosis, it may unjustly deny compensation to miners whose nonmalignant disorders were likely caused by their mining experience. We have concluded, therefore, that the Act would more accurately compensate claimants who developed fibrosis as a result of their mining employment if the present exposure-based criteria were replaced with alternative criteria, such as duration of employment, that better represent the relevant risks posed by the mining environment.

2. Proof of Radon Exposure As A Condition For Compensation For Silicosis or Pneumoconiosis

As explained above, the Act explicitly ties compensation for all the nonmalignant respiratory diseases, including silicosis and pneumoconiosis, to proof that the claimant was exposed to minimum levels of radon progeny during employment in the mines.¹¹¹ The Act limits compensation for these two diseases, moreover, to miners who were employed in mines on Indian Reservations.¹¹²

As was the case with fibrosis, above, the Committee heard from numerous critics that the Act inappropriately presumes an association between exposure to radon and silicosis that is not medically justified. Indeed, the criticisms suggest that the case for reform of the Act is even more compelling for these two

¹¹¹ See P.L. 101-426, § 5(a)(2)(A), (B), 104 Stat. at 922.

¹¹² Id.

nonmalignant diseases. Again, these criticisms were brought out by Dr. Samet during his testimony at the Senate hearings on oversight of RECA in 1993:

Silicosis is caused by inhalation of crystalline silicon dioxide and levels of exposures to radon progeny are not known to be strongly associated with those of silicosis exposure. ... Silica causes silicosis; radon exposure does not cause silicosis. So the requirement that, there, in fact, be a threshold value or any particular value of radiation exposure before a miner receive compensation for silicosis is not correct.¹¹³

The Committee also heard or reviewed comments from most every interested group critical of the limitation on compensation for silicosis and pneumoconiosis to mines located on an Indian Reservation.¹¹⁴

The Committee believes that these criticisms are justified. We recommend, first, that the Act be modified to incorporate alternative compensation criteria for silicosis;¹¹⁵ and second,

¹¹³ See, e.g., Hearings on Oversight of RECA, supra note 97, at 22.

¹¹⁴ See id. at 16 (testimony of Louise Abel, M.D.) ("[T]he Reservation does not have any particular patent on causing silicosis. ... We have many uranium miners -- not many, some of the uranium miners who worked all over the Southwest, travelled around and worked off the reservation, who contracted silicosis"); id. at 19 (testimony of Jonathan Samet, M.D., M.P.H.) ("I certainly concur with Dr. Abel's statement concerning the requirement that those who receive compensation for silicosis have worked in the past on Indian Reservation lands. Certainly, silicosis extends well beyond the boundaries of the reservation.").

¹¹⁵ Pneumoconiosis is the condition characterized by the deposition in the lung of substantial amounts of particulate matter, usually dust of occupational or environmental origin, and
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that compensation for silicosis or pneumoconiosis be extended to all claimants who were employed in underground uranium mines in any of the five states specified in the Act.

Although the Act explicitly provides compensation for silicosis and pneumoconiosis to any miner, whether Native American or not, who was employed in mines on an Indian Reservation, there is no explicit indication in the legislative history that Congress intended to link compensation for silicosis and pneumoconiosis to radiation exposure. The legislative history suggests that these two nonmalignant diseases were added to the Act at the last moment principally because of reports that Native American miners suffered an increased incidence of these diseases. As Senator Hatch explained just prior to the Senate's passage of the RECA:

Finally, at the request of several of the co-sponsors to this measure, I have included two additional uranium mining related diseases for which compensation will be available, if the miners worked in uranium mines that were located on or within an Indian Reservation. As I have noted several times before, many of the uranium miners involved in the production of uranium were Native Americans from some of the Southwestern Indian Tribes. Furthermore, many of the mines were located within their reservations. Given the special trust

¹¹⁵(...continued)

the subsequent tissue reaction. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1318 (27th ed. 1988). Silicosis is the specific pneumoconiosis associated with inhalation of silica dust (crystalline silicon dioxide). See Marvin R. Balaan & Daniel E. Banks, Silicosis, in ENVIRONMENTAL AND OCCUPATIONAL MEDICINE 345 (William N. Rom ed., 2d ed. 1992). The Committee did not hear any information suggesting that uranium miners are at an increased risk of any pneumoconiosis other than silicosis. Consequently, compensation standards for silicosis are all that are recommended.

relationship of the United States Government over the activity of these tribal members, it is only fair that we also include compensation for these diseases. These mines were some of the earliest and consequently most contaminated mines. In addition, they were not subject to any form of state regulation.¹¹⁶

There is little if any evidence in the legislative history supporting a direct association between radiation exposure and silicosis. Most of the testimony on nonmalignant diseases, including the testimony of Dr. Archer cited above, suggests that radiation at best contributed to the development of silicosis.¹¹⁷ For example, Leonard Haskie, Interim Chairman of the Navajo Tribal Council testified before the Senate Committee on Energy and Natural Resources in March, 1990, that

"[s]erious respiratory diseases such as ... silicosis, fibrosis, and others became prevalent [among Navajo miners]. Studies show these diseases are caused by exposure to radiation and radioactive dust particles present in the mines. ... Unfortunately, many Navajo uranium miners are suffering from

¹¹⁶ 136 CONG. REC. S11752 (daily ed. Aug. 1, 1990). See also id. at S11753 (statement of Sen. DeConcini); id. at S11752 (colloquy between Senators Hatch and Domenici); 136 CONG. REC. H8276 (daily ed. Sept. 27, 1990) (statement of Rep. Rhodes). The various congressional statements supporting the addition of these two diseases focus on the risk to Native Americans. Nonetheless, Congress heard testimony from a number of witnesses that both Native American and non-Native American uranium miners were at an increased risk of developing silicosis. See Hearings on Impact of Past Uranium Practices, supra note 101, at 10-11, 17 (testimony of Leonard Haskie, Interim Chairman of the Navajo Tribal Council); id. at 59 (testimony of Lynda Taylor); Hearings on the Health Effects of Radiation Exposure, supra note 99, at 35 (testimony of Victor Archer, M.D.); id. at 46-47 (testimony of Leon Gottlieb, M.D.).

¹¹⁷ See supra pp. 62-63.

serious respiratory diseases to which radiation is only a contributing factor."¹¹⁸

Similarly, another witness at the same hearing, testified:

Although the Act clearly calls for compensating former uranium miners with some non-malignant respiratory diseases, the Act appears to include only those non-malignant diseases which have been linked to radiation exposure, such as fibrosis. Other diseases found in excess among this population such as silicosis may be left out because a clear relationship between radiation cause and effect has not been established. The point here is that silicosis as well as other non-malignant respiratory diseases, have been clearly linked to working in these early uranium mines, most probably associated with dust from blasting.¹¹⁹

Most of the uranium mines in the designated states were located in mineral belts comprised of ore-bearing sandstone, which contains silica dust.¹²⁰ The silica-containing mineral belts were mined throughout portions of the West; they were not restricted to areas within Indian Reservations.¹²¹ As a result, individuals who were employed in underground uranium mines in the states covered by the Act are at risk of developing silicosis.¹²² The incidence of silicosis among uranium miners

¹¹⁸ Hearings on Impact of Past Uranium Practices, supra note 101, at 10-11.

¹¹⁹ Id. at 59 (testimony of Lynda Taylor).

¹²⁰ D.R. Shawe, et al, Uranium and Vanadium Deposits, in [P-2] THE GEOLOGY OF NORTH AMERICA 103-24 (H.J. Gluskoter, et al., eds., 1991).

¹²¹ Id.

¹²² See Samet & Simpson, supra note 106, at 2.

has been documented in epidemiological studies and by clinicians in mining areas.¹²³ While it has long been known that silicosis is caused by exposure to dust containing silica, according to the 1992 HHS report to Congress it is still uncertain if or how exposure to radon progeny affects a miner's risk of developing silicosis.¹²⁴

The Committee believes that the demonstrated incidence of silicosis among uranium miners, and the almost complete absence of data indicating if and how radon exposure affects the risk of silicosis, counsel strongly against the present statutory scheme that compensates silicosis purely as a function of exposure to radon. We recommend, again, that compensation for silicosis (or pneumoconiosis), as with the other nonmalignant respiratory diseases, be conditioned on alternative criteria, such as duration of employment for designated period(s) of time. Moreover, because the geological information indicates that uranium mines both on and off Indian reservations were in sandstone-bearing ore, posing a risk of silicosis to the miners, the Committee recommends that the Act be amended to eliminate the geographic limitation on compensation for silicosis and pneumoconiosis.

Again, as was the case with fibrosis above, we cannot recommend specific alternative compensation criteria for these two diseases. Although some historical dust data does exist, we

¹²³ Id. at 31.

¹²⁴ Id. at 31.

do not know if there is a sufficient amount, or if it is sufficiently accurate, to serve as a basis for quantitative analysis.¹²⁵

C. Recommendations for Modification of the Existing Regulations Governing Proof of A Nonmalignant Respiratory Disease

The bulk of the Committee's recommendations relating to nonmalignant respiratory disorders propose changes to the existing regulations that govern the proof required of a claimant to establish the compensable medical condition. During the course of our review, the Committee heard numerous criticisms of these regulations and the manner in which they were being administrated by the Program. The Committee has concluded that some, though not all, of the criticisms are well founded and warrant changes. We also concluded, however, that some of the criticized regulations effect a reasonable balance between the statutory mandate to compensate deserving miners and legitimate policies of the Program, given the medical evidence that can reasonably be demanded of claimants.

1. Requirement That Claimant Prove Functional Impairment in Addition to the Presence of Disease

The regulations require living miners seeking compensation for a nonmalignant disorder to submit reliable written medical records documenting both the presence of a compensable disease process and

¹²⁵ See supra p. 61.

reduction in pulmonary function.¹²⁶ Proof of disease is made by submitting chest radiographs evidencing the disease; proof of functional impairment is made by submitting the results of one of two clinical tests showing a reduction in pulmonary function.¹²⁷ Thus, an individual with a mild case of a nonmalignant disorder detectable by chest radiograph but with no appreciable reduction in lung function would not qualify under the regulations.

The requirement that a claimant prove both structural abnormality and functional impairment has been criticized, chiefly by claimants' attorneys, as going beyond the text of the statute. The critics point to the explicit language of the statute, which requires only "written medical documentation that [the miner], ... developed a nonmalignant respiratory disease"¹²⁸ This language does not impose any impairment requirement, the critics maintain; claimants should be entitled to compensation if they can produce either radiographic evidence of the disease process or the appropriate clinical evidence of reduced pulmonary function.

The Committee finds this criticism without merit: we believe that the regulations accurately and appropriately express the intent of Congress. Although the Act does not explicitly include a requirement

¹²⁶ See 28 C.F.R. § 79.36(d)(1)(ii). If the claimant is deceased, the eligible beneficiary(ies) need only submit one of a list of appropriate medical records containing a diagnosis of the compensable nonmalignant disease or sufficient information from which experts designated by the Department can make a diagnosis of the disease. Id. § 79.36(d)(1).

¹²⁷ See id.

¹²⁸ P.L. 101-426, § 5(a)(2)(A), (B), 104 Stat. at 922 (emphasis added).

that a claimant with a nonmalignant disease prove impairment, the Committee believes such a requirement is clearly implied in the statutory scheme.

To begin with, the Act all but demands a functional impairment requirement in the case of silicosis and pneumoconiosis: the Act specifically defines the term nonmalignant respiratory disorder to include only "moderate or severe silicosis or pneumoconiosis."¹²⁹ A uniform system for classifying the extent of a pneumoconiotic disease process as evidenced on chest radiographs does exist; in fact, this system is used by the Program for the interpretation of chest radiographs for proof of disease.¹³⁰ This classification scheme, however, does not purport to grade the severity of the disease process, or distinguish radiographic evidence of disease as mild, moderate or severe.¹³¹ On the other hand, these distinctions are terms employed by physicians to differentiate the degree of functional impairment resulting from respiratory disease.¹³² This suggests to us that

¹²⁹ Id. § 5(b)(3), 104 Stat. at 923.

¹³⁰ See 28 C.F.R. § 79.36(d)(1)(ii)(C); see also infra pp. 71-72.

¹³¹ In fact, it is well established that there is a limited correlation between radiographic evidence of pneumoconiosis and functional impairment. See W.G.B. Graham, Silicosis, 13 CLINICS IN CHEST MEDICINE 253 (1992); J.M. Peters, Silicosis, in OCCUPATIONAL RESPIRATORY DISEASES 227 (J. Merchant ed. 1986).

¹³² There is no single, official definition of mild, moderate or severe pulmonary impairment. The existing regulations define moderate or severe impairment as a reduction in pulmonary function equal to or greater than 25%. See 28 C.F.R. § 79.36(d)(1)(ii)(B). The American Thoracic Society (ATS) guidelines, on the other hand, recommend that a patient be categorized as suffering mild impairment if their pulmonary

(continued...)

Congress intended proof of at least silicosis and pneumoconiosis to be made by reference to impairment.

The legislative history supports this interpretation. During the Senate's debate leading to the passage of the Act, Senator DeConcini stated: "As I understand it, the respiratory diseases that are most prevalent among these Native Americans miners are silicosis or pneumoconiosis. There is a requirement in the Act that those eligible for compensation have a moderate or severe case because the intent of this legislation is to compensate those individuals with both serious respiratory diseases."¹³³

Moreover, in 1992, during the Senate's consideration of amendments to the RECA, Senator McCain stated:

[W]hen the Congress enacted the Radiation Exposure Compensation Act, that Act included a Native American provision which extended compensation to any former uranium miner who is a member of an Indian tribe and has contracted moderate or severe silicosis or pneumoconiosis, in addition to lung

¹³² (...continued)
function is reduced by 21% to 40%; moderate impairment if their pulmonary function is reduced by 41% to 49%; and severe impairment if the reduction in function exceeds 50%. American Thoracic Society, Evaluation of Impairment/Disability Secondary to Respiratory Disorders, 133 AM. REV. RESPIR. DIS. 1205-06 (1986). These standards are consistent with those recommended by the American Medical Association. See AMA, GUIDELINES TO THE EVALUATION OF PERMANENT IMPAIRMENT § 5.3, at 161-163 (4th ed. 1993). While the decision to adopt a slightly more stringent standard at the outset of the Program is understandable, the Committee recommends that the regulations be modified to adopt the ATS definitions of disease. Consistent with the well-documented healthy worker effect, there is reason to believe most of the miners had above-average pulmonary function, and therefore, have to suffer a greater than average reduction in function to meet the definition of moderate impairment. This supports using the more liberal definitions recommended by the ATS.

¹³³ 136 CONG. REC. S11753 (daily ed. Aug. 1, 1990).

cancer and other serious respiratory diseases. ...
In keeping with the intent of the Act, compensation
is limited to those individuals that are severely
disabled.¹³⁴

This intent seems evident to us not only because of the express limitations on recovery for silicosis and pneumoconiosis, but in light of the severity of the other compensable medical conditions in the Act. In addition to the nonmalignant respiratory disorders, uranium miners are compensated for lung cancer, a malignancy that is often fatal or at least life threatening, and which in most cases seriously and irrevocably disrupts and impairs the quality of life of the individual afflicted.¹³⁵ Moreover, the diseases for which the other two classes of claimants covered by the Act are compensated are, like lung cancer, malignant, life-threatening conditions which are either accompanied by, or the treatment of which causes, significant and often debilitating side effects.¹³⁶ It does not seem reasonable to us that Congress

¹³⁴ 138 CONG. REC. S1322 (daily ed. Feb. 7, 1992) (emphasis added).

¹³⁵ See D.E. Midthun & J.R. Jett, Clinical Presentation of Lung Cancer, in LUNG CANCER: PRINCIPLES AND PRACTICE 421-435 (H.I. Pass, et al., eds., 1996) (5 year survival rate of 15%).

¹³⁶ The compensable diseases for the two other classes of claimants covered by the Act -- downwinders and on-site participants -- are, subject to certain limitations, leukemia (other than chronic lymphocytic leukemia); multiple myeloma; lymphomas (other than Hodgkin's disease); and primary cancer of the thyroid, female breast, esophagus, stomach, pharynx, small intestine, pancreas, bile ducts, gall bladder and liver. P.L. 101-426, § 4(b)(2), 104 Stat. at 922. With the exception of childhood leukemia and cancers of the thyroid and female breast, the 5-year survival rates for these malignancies is 50% or less. For cancer of the pancreas, bile ducts, liver, gall bladder and esophagus, the 5-year survival rate is 10% or less. American Cancer Society, Cancer Journal for Clinicians 5-27 (1996).

2. Regulations Governing Proof of Disease Process

The regulations require proof that a miner suffers or suffered from one of the compensable nonmalignant diseases. For living miners, this is done by submitting a chest radiograph evidencing changes to the lung structure consistent with the specified diseases.¹³⁹ Beneficiaries of deceased miners need only submit one of a series of specified medical records containing a diagnosis of a compensable disease, including, if they exist and are of appropriate quality, chest radiographs.¹⁴⁰

The regulations specifically set out the technical standards that constitute radiographic proof of disease, and the necessary documentation. A claimant must submit a radiograph and interpretative reports of at least 2 certified "B"-readers concluding that the radiograph demonstrates the existence of fibrosis of at least category 1/0 according to a widely-accepted classification scheme for assessing the existence and extent of pneumoconioses.¹⁴¹ Under this classification scheme, a radiograph is given one of 12 grades according to the degree of "profusion," which is an assessment of the concentration of small opacities that mark scarring of the lungs, as

¹³⁸ (...continued)
Staples, et al., High Resolution Computed Tomography and Lung Function in Asbestos-Exposed Workers With Normal Chest Radiographs, 139 AM. REV. RESP. DIS. 1502, 1507 (1989) (same).

¹³⁹ See 28 C.F.R. § 79.26(d).

¹⁴⁰ Id. § 79.36(d)(1)(i).

¹⁴¹ Id. § 79.36(d)(1)(ii)(A).

compared with standard radiographs.¹⁴² A radiograph graded as category 0 (profusion scores of 0/-, 0/0, 0/1) is considered to be consistent with normal lung structure. A classification of 1/0 means that the radiograph reveals abnormalities in the lung structure consistent with fibrosis or pneumoconiosis.¹⁴³ The regulations thus require the lowest score that is consistent with the existence of a compensable disease.

The regulations require that the radiograph be classified by a certified "B"-reader.¹⁴⁴ A "B"-reader is a physician who has been certified by NIOSH as having demonstrated proficiency in the application of the ILO classification system of radiographs for pneumoconioses.¹⁴⁵

Critics have raised four major objections to this regulatory scheme governing proof of disease. First, it is inappropriate, burdensome and unwarranted to require claimants who can prove pulmonary impairment as required in the regulations to also submit proof of disease process. Second, the regulations should allow claimants to prove disease by means

¹⁴² See James A. Merchant & David A. Schwartz, Chest Radiography for Assessment of Pneumoconiosis, in ENVIRONMENTAL AND OCCUPATIONAL MEDICINE, supra note 115, at 216-17. The scheme is known by the initials of the organization that established and periodically updates the guidelines, the International Labor Office (ILO). Under the ILO system, the reader also classifies the size and shape of the small opacities, and the extent of the profusion (the areas of the lung in which the opacities are observed). Id. at 216-17.

¹⁴³ See M. Remy-Jardin, et al., Computer Tomographic Evaluation of Silicosis and Coal Worker's Pneumoconiosis, 30 RADIOLOGICAL CLINICS OF NORTH AMERICA 1155, 1174 (1992); Gevenois, supra note 138, at 355.

¹⁴⁴ 28 C.F.R. § 79.36(d)(1)(i)(A).

¹⁴⁵ See AMA, supra note 132, § 5.1, at 158; see also 28 C.F.R. § 79.31(r).

of high-resolution computed tomography (HRCT) scans in addition to chest radiographs. Third, the regulations should allow claimants to prove disease by means of biopsy. And, fourth, the requirement that chest radiographs be interpreted only by "B"-readers is unnecessary and oppressive to claimants, and is being administered arbitrarily by the Program. We will take each in turn.

(a) Regulation Requiring Proof of Disease Process Where
A Claimant Can Prove Impairment

The Committee heard from some critics who found fault with the regulations in the converse situation to that addressed immediately above: that where claimants can prove functional impairment through the required clinical tests, they should not also be required to prove the presence of the nonmalignant disease. The concern in these circumstances arises from limitations of chest radiographs in detecting interstitial lung diseases such as fibrosis and silicosis, coupled with the belief that proof of disease is unnecessary where functional impairment has been verified. Critics contend that requiring all claimants to prove disease has the effect of denying timely compensation to those claimants who suffer from a compensable disease and are impaired, but may not develop radiographic evidence for some time -- possibly after the claimant is too disabled to benefit from the compensation.

It is well established that chest radiographs have a high rate of "false negatives" -- that is, they falsely report the absence of disease that in fact exists. The medical literature reports that chest radiographs will appear normal in as much as 10-18% of patients with

interstitial lung diseases proven by biopsy results.¹⁴⁶ Moreover, studies have demonstrated that persons exposed to dusts occupationally may suffer from significantly reduced pulmonary function years before the disease becomes evident on a chest radiograph.¹⁴⁷ The Committee believes it is quite likely, therefore, as critics charge, that some claimants with a compensable nonmalignant disease are not being timely compensated despite clinically-evident impairment.

Nonetheless, the Committee recommends that claimants continue to be required to submit radiographic evidence of disease. We believe it would be inappropriate to rely solely on proof of physiologic impairment for at least two reasons. First, the tests accepted by the Program for proof of impairment -- pulmonary function tests and arterial blood gas studies -- are not specific for the statutorily-specified nonmalignant diseases. These tests reveal a reduction in the patient's pulmonary function, but do not identify the specific etiology or disease process that caused the reduction. These tests will detect reduced pulmonary function due to both restrictive lung diseases² -- a class that includes, but is not limited to, interstitial fibrotic disorders -- and obstructive lung diseases -- a class that includes chronic obstructive

¹⁴⁶ See, e.g., S. Padley, et al., Review Article: Current Indication for High-Resolution Computer Tomography Scanning of the Lung, 68 BRITISH JOURNAL OF RADIOLOGY 105 (Feb. 1995); David D. Hansell & Ian H. Kerr, The Role of High Resolution Computer Tomography in the Diagnosis of Interstitial Lung Disease, 46 THORAX 77 (1991); N. L. Muller & B. N. Ostrow, High-Resolution Computer Tomography of Chronic Interstitial Lung Disease, 12 CLINICS IN CHEST MEDICINE 97 (March 1991); see also infra p. 85 (factors limiting sensitivity of radiographs).

¹⁴⁷ See Peters, supra note 131, at 227; see also Staples, supra note 138, at 1507 (citing studies).

pulmonary disease.¹⁴⁸ It is well documented that the majority of miners were smokers, putting many at an increased risk of obstructive disease.¹⁴⁹ Consequently, if the Program were to rely solely on proof of functional impairment, there is a serious risk that an appreciable number of miners would be compensated improperly.¹⁵⁰

Second, although the problem of false negative chest radiographs is compelling, the Committee does not believe the appropriate answer is to eliminate the requirement that the claimant prove disease. Instead, as discussed below, we recommend that claimants be allowed in some circumstances to demonstrate the presence of disease by HRCT. This imaging technique is often more sensitive and specific than a chest radiograph; most claimants with even low levels of fibrosis, silicosis or pneumoconiosis will be able to demonstrate sufficient abnormalities to justify compensation.

¹⁴⁸ See Stuart M. Garay, Pulmonary Function Testing, in ENVIRONMENTAL AND OCCUPATIONAL MEDICINE 183, 187 (William N. Rom, ed., 2d ed. 1992).

¹⁴⁹ Richard W. Hornung, et al., Modifiers of Exposure-Response Estimates for Lung Cancer Among Miners Exposed to Radon Progeny, 103 ENVIRON. HEALTH PERSPECT. 49, 52 (1995); F.D. LUNDIN, JR., ET AL., NATIONAL INSTITUTE OF OCCUPATIONAL SAFETY AND HEALTH-NATIONAL INSTITUTE FOR ENVIRONMENTAL HEALTH SCIENCES, JOINT MONOGRAPH No. 1, RADON DAUGHTER EXPOSURE AND RESPIRATORY CANCER, QUANTITATIVE AND TEMPORAL ASPECTS 19-20. Data suggest that dust or other agents in the mining environment can significantly increase the severity of impairment otherwise resulting from some obstructive lung diseases. M.R. Becklake, The Work Relatedness of Airways Dysfunction, in PROCEEDINGS OF THE 9TH INTERNATIONAL SYMPOSIUM ON EPIDEMIOLOGY IN OCCUPATIONAL HEALTH (1994) (National Institute for Occupational Safety and Health, DHHS Publication No. 94-112). The Act does not compensate miners for any obstructive lung diseases.

¹⁵⁰ Cf. Padley, supra note 146, at 105 (reporting that in patients with suspected silicosis who smoke, pulmonary function correlates more accurately with the severity of underlying emphysema than with profusion seen on a radiograph).

(b) Lack of Authority in the Regulations for Proof of
Disease By HRCT Scans

The regulations require claimants to prove disease process by chest radiographs.¹⁵¹ No provisions exist for the submission of other evidence of lung abnormality. There is relative unanimity among all interested groups, including physicians who treat miners and medical researchers who study the mining population, that a complete ban on proof by modalities other than chest radiograph is unwarranted. The objection rests on the accepted proposition, noted above, that chest radiographs have a significant false negative rate for detection of interstitial lung diseases such as fibrosis and silicosis. Critics advocate that the Program modify the regulations to accept HRCT, which they maintain is an accepted and available diagnostic technology of greater sensitivity and specificity than chest radiography. While HRCT is not itself without limitations, it is argued, miners should not be denied the ability to prove eligibility by means of available and reliable medical tests. The Committee agrees.

The Program's reliance on chest radiographs is not unreasonable. Chest radiographs are the modality used most often in the assessment of interstitial lung disease¹⁵², and, when coded by the ILO classification, are generally accepted as the basis for a diagnosis of pneumoconiosis without additional histologic proof.¹⁵³

¹⁵¹ 28 C.F.R. § 79.36(d)(1)(ii)(A).

¹⁵² See Muller & Ostrow, supra note 146, at 97.

¹⁵³ Staples, supra note 138, at 1507; Remy-Jardin, supra note 143, at 1173; Gevenoís, supra note 138, at 351.

Nonetheless, it is well recognized that chest radiographs and the interpretative process suffer from significant limitations. Radiographs have only limited resolving power; the two dimensional nature of the medium results in the superimposition of structures, obscuring significant portions of the lung; and the radiographic image is affected by the background density of the lung, and the shape and composition of lesions. The ILO classification scheme, created for descriptive and semiquantitative analysis, not diagnostic purposes, permits substantial reader error and interreader variability, particularly at low profusion scores.

Because of these limitations on the sensitivity (the ability to detect disease) and selectivity (the ability to differentiate among diseases) of chest radiographs, it is often difficult to make a confident and accurate diagnosis of pneumoconiosis on the basis of a radiograph alone.¹⁵⁴ Indeed, the medical literature documents that chest radiographs have an appreciable false negative rate, and underestimate the incidence of diseases such as fibrosis and silicosis.¹⁵⁵

High-resolution computed tomography is now widely accepted as a reliable diagnostic modality. Because of its significantly greater resolving power, HRCT is more selective and more sensitive than chest

¹⁵⁴ Hansell & Kerr, supra note 146, at 77; Muller & Ostrow, supra note 146, at 97.

¹⁵⁵ See supra pp. 73-74. Chest radiographs have also been found to have a significant false positive rate. Studies suggest that in as much as 10-20% of cases, if not more, a diagnosis of interstitial lung disease by chest radiograph is proved incorrect. See Padley, supra note 146, at 105; Gevenois, supra note 138, at 355.

radiographs, and allows for a better assessment of the type and severity of lung abnormalities. Consequently, HRCT allows for a more confident and specific diagnosis than a chest radiograph, and one that is more accurate.¹⁵⁶ Of particular significance, recent studies demonstrate that HRCT is more sensitive than chest radiographs in detecting interstitial lung diseases. Studies indicate that HRCT detects significantly more evidence of pneumoconiosis (such as silicosis) than a radiograph, resulting in earlier detection and detection of disease in a more mild state.¹⁵⁷ HRCT also significantly reduces the variability of diagnosis among readers.¹⁵⁸

There are, however, problems associated with the use of HRCT evidence, as even those advocating their use concede. HRCT scans are costly¹⁵⁹ and entail yet an additional dose of radiation to the claimant.¹⁶⁰ Equally problematic, there apparently are no official,

¹⁵⁶ Padley, supra note 146, at 105; Hansell & Kerr, supra note 146, at 84; Muller & Ostrow, supra note 146, at 100-01.

¹⁵⁷ See Gevenois, supra note 138, at 352-55; Remy-Jardin, supra note 143, at 1174.

¹⁵⁸ Gevenois, supra note 138, at 355.

¹⁵⁹ A limited and unscientific sample of hospitals with HRCT capability in the Southwest Colorado-Northwest New Mexico area indicates that the average cost is approximately \$660.

¹⁶⁰ See Joan F. Leonard & Philip A. Templeton, Pulmonary Imaging Techniques in the Diagnosis of Occupational Interstitial Lung Disease, 7 OCCUPATIONAL MEDICINE: STATE OF THE ART REVIEWS 241-42 (1992); Muller & Ostrow, supra note 146, at 99-100.

uniform, accepted standards for the diagnosis of interstitial lung diseases by HRCT.¹⁶¹

In light of the many advantages of HRCT over chest radiographs, and the strong advocacy of treating physicians and researchers in the affected areas, the Committee recommends that the regulations be modified to allow claimants to submit HRCT evidence as proof of disease. The increased sensitivity and selectivity of HRCT should reduce significantly the number of claimants who are unjustly denied compensation because of the absence of evidence or misdiagnosis of radiographs.¹⁶²

We do not recommend that claimants be required to submit HRCT evidence in every case: we recommend that it be accepted only in those circumstances where a claimant has demonstrated sufficient impairment to qualify, but has a chest radiograph that does not indicate fibrosis, silicosis or pneumoconiosis. The Program should continue to use the chest radiograph in the first instance to identify fibrosis or

¹⁶¹ Remy-Jardin, supra note 143, at 1157. Some advocates have expressed concern that HRCT equipment may not be sufficiently available in the areas with significant claimant populations. This is clearly an appropriate consideration, but the Committee believes the technology is sufficiently available in the area covered by the statute to be of benefit to many claimants. We have confirmed that HRCT scanners are available at facilities in Denver, Durango and Grand Junction, Colorado; in Albuquerque and Farmington, New Mexico; and at the Indian Health Service Hospital (Northern Navajo Medical Center) in Shiprock, New Mexico.

¹⁶² In those circumstances where HRCT evidence is accepted, of course, there is the possibility that the more powerful diagnostic tool will reveal that the source of the claimant's physiological impairment is a noncompensable disease process. Thus, HRCT use will benefit the Program by reducing the risk that a claimant is improperly compensated because of a false positive diagnosis of fibrosis or silicosis.

silicosis; radiographs are widely available and inexpensive. We also believe the Program should continue to accept as proof of disease a radiograph interpreted as showing profusion of 1/0 or more, as the regulations presently provide, though there is an acknowledged risk of a false positive interpretation. In our opinion, it is only where a claimant is impaired because of a proven lung disease, and the radiograph does not show a compensable abnormality, that the cost of the HRCT and additional radiation dose is justified.

We believe that the disadvantages of HRCT evidence noted above are not insuperable obstacles. First, the lack of official standards for the diagnosis of pneumoconiosis by HRCT, such as an ILO-type classification scheme, should not be a major problem. The increased sensitivity of HRCT reveals significant morphologic detail of the lung, and accepted descriptions of the appearance of normal and abnormal lung anatomy on HRCT have been published.¹⁶³

To minimize, if not avoid the problem entirely, we recommend that the Program establish a panel of appropriately-credentialed radiologists to interpret the HRCT evidence. We recognize that this is at odds with the philosophy governing the submission of radiographic evidence: the regulations allow claimants to identify and obtain reports from B-readers of their choosing. In the case of HRCTs, however, we believe centralizing the task of interpretation is warranted. The use of HRCT to diagnose pneumoconiosis is a relatively recent phenomenon; while its effectiveness and reliability for this purpose has been established and

¹⁶³ See, e.g., W. R. Webb, High Resolution Computed Tomography of the Lung: Normal and Abnormal Anatomy, 26 SEMINARS IN ROENTGENOLOGY 110 (1991).

accepted by the specialist medical community, there is some concern that it is not so common nor widespread that sufficient expertise has developed in all communities. A single panel of authorities will allow for consistent application of diagnostic and interpretative standards.¹⁶⁴ It will also lessen the cost to claimants.

Second, while the cost of an HRCT scan is significant, we do not believe it is excessive given the potential compensation at stake. Indeed, we have been informed by a number of attorneys representing claimants that miners presently are obtaining HRCT scans. Moreover, it is not a large class of claimants who will be forced to bear the cost if our recommendation is accepted.

(c) Lack of Authority in the Regulations for Proof of
Disease By Biopsy in Living Miners

The regulations allow the surviving beneficiaries of a deceased miner to prove disease by means of a pathology report of tissue biopsy.¹⁶⁵ The regulations do not allow a living miner to submit the same evidence.¹⁶⁶ Presumably, this restriction was designed to discourage claimants from undergoing dangerous and invasive surgery

¹⁶⁴ We leave to the Program the development of an appropriate protocol for selecting and reading the HRCT scans. We note that similar panels have been devised for diagnosing asbestos-related diseases in administrative compensation schemes settling class action litigation. We appreciate the concerns expressed by some miners and their representatives over the creation of some distant, nameless "Supreme Court" for diagnostic purposes. We believe this concern can be met simply by devising a fair system for selecting independent, acknowledged experts to serve on the panel, including, as appropriate, experts from the five mining states specified in the Act.

¹⁶⁵ 28 C.F.R. § 79.36(d)(1)(i)(A).

¹⁶⁶ Id. § 79.36(d)(1)(ii).

solely to establish their right to compensation. A number of interested parties have criticized this restriction, pointing out that where a biopsy result exists, there is no good reason why the Program should not accept it as proof of disease. We agree.

The most accurate basis for diagnosis of disease is by a tissue biopsy; it is the medical equivalent of the "gold standard." The sensitivity of other diagnostic modalities, such as chest radiographs or HRCT scans, is often measured against findings on biopsy.¹⁶⁷

We believe the Program's concern that it not create an incentive for unnecessary and dangerous surgery through the regulations is fully justified. However, where a tissue biopsy is performed for a medically-justified reason and not principally out of a desire to establish eligibility for compensation, we see little reason to ignore definitive evidence and allow a deserving claimant to be denied compensation.¹⁶⁸

We recommend, therefore, that the regulations be modified to allow for living miners to submit tissue biopsy results as proof they have developed a compensable nonmalignant disease. We also believe it is not unreasonable to rely on the ethics and sound judgment of physicians not

¹⁶⁷ See, e.g., Padley, supra note 146, at 105; Leonard & Templeton, supra note 160, at 241-42; Muller & Ostrow, supra note 146, at 100-01.

¹⁶⁸ In light of our recommendation above that the Program accept HRCT evidence, we expect that few claimants will need to submit biopsy results. Cf. Jonathan B. Orens, et al., The Sensitivity of High-Resolution CT in Detecting Idiopathic Pulmonary Fibrosis Proved By Open Lung Biopsy, 108 CHEST 109, 109 (1995) (because of resolving power of HRCT, necessity of performing biopsy to establish diagnosis of interstitial lung disease questioned); Hansell & Kerr, supra note 146, at 84 (HRCT may obviate the need for lung biopsies in patients with clinical evidence of lung abnormality and a normal chest radiograph).

to perform biopsies where medically unwarranted. The potential consequences of performing unnecessary surgery should prove a sufficient check. Nonetheless, the Program should be able to guard against the problem in a relatively simple fashion. The regulations should specify, first, that biopsies can be submitted only if performed for independent, medically-justified reasons; and second, the Program can, either routinely or where appropriate, require the claimant to submit medical documentation from the treating physician explaining and justifying the reason for the biopsy. The documentation can be audited by experts at NCI, NIOSH, or other appropriate federal agency.

(d) Requirement That Chest Radiographs Be Interpreted Only By "B"-Readers

As explained above, the regulations require that the claimant's chest radiograph be interpreted by "B"-readers.¹⁶⁹ A report finding fibrosis or silicosis by a treating physician or radiologist is not sufficient.¹⁷⁰

This requirement is the object of considerable criticism. The criticism seems directed to two distinct though related issues: first, critics argued that the "B"-reader requirement is an unnecessary imposition on claimants, and is of correspondingly little benefit to the Program; and second, they accuse the Program of abusing its discretion by selectively having reports of some "B"-readers reinterpreted by its own in-house experts, almost always resulting in the denial of the

¹⁶⁹ See supra pp. 50-51, 71-72.

¹⁷⁰ See 28 C.F.R. § 79.36(d)(1)(i)(C), (d)(1)(ii)(A). The regulations advertise that a list of certified readers is available from the Program. Id. § 79.31(r).

claim. The Committee concludes that the "B"-reader requirement is worthwhile and should be retained, but recommends that the Program establish a fair and random audit procedure to ensure that "B"-readers are properly interpreting radiographs.

As discussed above, the diagnosis of pneumoconiosis by chest radiography is an inexact science. It is well established that there is significant variability in radiographic interpretation among readers, including "B"-readers. This variability contributes to the documented observation that the diagnosis of pneumoconiosis by chest radiograph has appreciable associated false negative and false positive rates.¹⁷¹ As a rule, however, trained and certified "B" readers demonstrate less inter- and intra-reader variability in their readings than untrained and uncertified readers. The Program presumably requires reports of B-readers as a means of minimizing the risk of an erroneous diagnosis, with obvious benefits to both claimants and the Program.

The controversy over the use of "B"-readers arises, of course, because the Program relies on chest radiographs as proof of disease in the majority of cases. The controversy could be practically eliminated by demanding HRCT evidence or other more powerful radiographic evidence of disease in every case. The Committee believes this would be inappropriate: we recommend that the Program retain the present regulation requiring chest radiographs of claimants with nonmalignant disorders. The risk of misdiagnosis notwithstanding, the chest radiograph is an accepted and valid technique for the diagnosis of

¹⁷¹ See generally supra pp. 81-82, 85 & note 155.

pneumoconiosis.¹⁷² If HRCT scans are accepted from claimants with proven impairment and negative chest radiographs, the risk of misdiagnosis is almost entirely on the Program: a claimant with a negative chest radiograph can submit HRCT evidence to prove that the earlier result is a false negative; but claimants with positive radiographs (graded as profusion of 1/0 or greater) are not required to submit HRCT evidence, and are compensated despite the risk of a false positive diagnosis. Such a system does not strike us as unfair.

Moreover, HRCT scans are much more expensive than chest radiographs, and subject the claimant to an additional dose of radiation.¹⁷³ The Committee believes that it is neither reasonable nor necessary to impose these burdens on all claimants.

The Committee also believes that the Program is justified in continuing to require the chest radiograph to be interpreted by "B"-readers. By definition, "B"-readers have been certified by the government as having special proficiency in the classification of radiographs for the presence of pneumoconiosis not generally shared by all physicians and radiologists. "B"-readers have demonstrated knowledge of the appropriate standards for the radiographic classification of pneumoconiosis, and, more importantly, an ability to apply those standards within an acceptable degree of variation. Relying on "B"-readers may not eliminate inter-reader variability,¹⁷⁴ but it

¹⁷² See supra pp. 79-80, 84.

¹⁷³ See supra p. 86.

¹⁷⁴ The rate of misdiagnosis and, consequently, the greatest degree of interreader variability, occurs at the lower
(continued...)

does seem a reasonable way of limiting these risks. While the risk of misdiagnosis is present even if the Program relies on "B"-readers' expertise, there is plainly a much greater risk if one accepts the interpretations of physicians who do not have that expertise, or at least have not allowed their expertise to be verified.

On the other hand, the Committee expresses its concern with the Program's apparent practice of selectively re-reading the reports of B-readers. We heard from a number of critics who charge the Program with re-reading reports of "B"-readers that tend to be more "liberal" in their interpretation. The Program admits that they believe a small number of B-readers are not consistent and forthright in applying the appropriate interpretative standards. The Program admits that in many cases where they receive positive reports from these readers, they have the underlying chest radiographs reinterpreted by "B"-readers who serve as consultants to NIOSH.

The Committee has no doubt that the Program is motivated by a sincere, good-faith desire to ensure that the appropriate diagnostic standards are being accurately and consistently applied. This is surely a legitimate and important programmatic end, particularly given the substantial sums involved, and the relatively claimant-friendly standard for proof of disease (1/0 profusion or greater). Yet, the practice of selectively auditing only some "B"-readers, and only those "biased" in

¹⁷⁴ (...continued)
profusion scores (less than 1/0), which indicates, in most cases, a milder stage of disease. See Leonard & Templeton, supra note 160, at 241-42; Remy-Jardin, supra note 143, at 1174. It is precisely in these cases, of course, that we recommend HRCT evidence be accepted. This, again, all but eliminates the risks to the claimant posed by faulty "B" readings.

favor of claimants, may be contrary to the spirit of the regulations, and has apparently led many in the affected communities to conclude that the Program is biased against compensation.¹⁷⁵

The regulations were obviously designed to allow claimants to submit interpretative reports from "B"-readers of their own choosing.¹⁷⁶ Claimants should not be penalized for seeking out the most "liberal" certified readers, thus maximizing the possibility of compensation. They have done nothing more than the regulations provide.

We believe that the Program can and should audit "B"-readers, but should do so by means of a system that eliminates the potential for selective enforcement of the diagnostic standards. This should, in turn, eliminate the appearance that the Program is abusing its discretion. We offer two such systems as suggestions:

First, the Program can establish a procedure in which the interpretative reports are randomly audited. That is, every xth case will be sent to an independent panel of "B"-readers to be reinterpreted, regardless of who the "B"-reader is. The claimants' positive readings would be disregarded only if some percentage of the panel (majority, two-thirds, etc) conclude that the radiograph evidences profusion less

¹⁷⁵ We wish to reiterate that we have seen no credible evidence indicating the Department is biased against compensation or seeks to deny claims wherever possible. In fact, from what we have seen, and what we have been told by many credible sources, the exact opposite is true. It appears to us that the Program goes to some lengths to assist claimants, particularly those not represented by counsel, to gather the necessary proof to establish eligibility. We are of the impression that where deserving claimants are denied compensation, it is not the good faith of the Program staff that is in question, but the statutory criteria or regulations.

¹⁷⁶ See 28 C.F.R. § 79.36(d)(1)(ii)(A).

than 1/0. Over time, one would expect all readers who regularly provide reports for claimants will be audited. Those who provide the most readings, and thus pose the greatest risk to the Program of misdiagnosis, will (over time) be audited most often.

There are obvious limitations to this system, however: it may be a considerable period of time before a problematic reader is audited; more importantly, the focus is on the reading and not the "B"-reader, who is free to misdiagnose additional radiographs until audited again.

Second, the Program can audit (and reaudit) every "B"-reader after he/she submits x reports. Each time a "B"-reader submits a predetermined number of reports,¹⁷⁷ the underlying radiographs would be sent to an independent panel of "B"-readers. The panel would review the films to determine whether the audited reader's interpretations, as a whole, were reasonable: that is, were they within the range of scores one would expect from a reader conscientiously and forthrightly applying the accepted classification standards.¹⁷⁸ If the panel concludes they were not, the Program would then refuse to accept readings from that "B"-reader for a specific period of time.

¹⁷⁷ As an additional security measure, the interval between audits for any given "B"-reader could vary, as well. The interval could be randomly selected each time within a defined range.

¹⁷⁸ This review would be something akin to the "abuse of discretion" standard employed by appellate courts reviewing a variety of lower court determinations. Under this standard, for example, an appellate court may reverse the factual findings of a district court only if it finds the findings "clearly erroneous." See Cooter & Gell v. Hartmarx Corp., 496 U.S. 384, 401 (1990). This standard requires the appellate court to uphold any lower court determination that falls within the range of permissible conclusions. Id. at 400.

This second procedure likely offers a better check than the first proposed audit process. It allows the Program to "decertify" readers who systematically misclassify the compensable diseases, (either by overestimating or underestimating their incidence), and thus represent a real, long-term threat to the integrity of the Program. Because the panel would review all of the audited readers' interpretations since the last audit, the reader cannot interpret radiographs likely to be audited conscientiously, and others more liberally or conservatively. Additionally, all B-readers would be audited, eliminating the appearance of bias. And, finally, because the audit would only "decertify" readers who have demonstrated repeated unacceptable professional judgment, claimants would still be allowed to take advantage of "B"-readers who tend to interpret more liberally, but within accepted professional bounds.¹⁷⁹

D. Regulations Regarding Proof of Impairment

The regulations require all living miners to submit the results of physiologic testing evidencing significantly reduced lung function.¹⁸⁰ This is done by producing the results of either pulmonary function tests or arterial blood gas studies.¹⁸¹ During our investigation, medical researchers at the University of New Mexico School of Medicine brought

¹⁷⁹ We do not mean to imply by these suggested audit procedures that the Program should be stripped of the ability to audit readings under any other circumstances. The Program should have the right to have any given reading audited if there is evidence, other than the identity of the reader, to suggest it is untrustworthy.

¹⁸⁰ 28 C.F.R. § 79.36(d)(1)(ii).

¹⁸¹ Id. § 79.36(d)(1)(ii)(B), (C).

to our attention two problems in this area. Both have to do with the definition of "normal" lung function employed by the Program.

1. Pulmonary Function Test Standards

Claimants can prove impairment by undergoing tests of pulmonary function.¹⁸² A claimant's lung function as measured by these tests is compared with predicted values based on sex, age and height. These predicted values are derived from external populations without evidence of pulmonary disease that are considered to be "normal."¹⁸³ The regulations consider a claimant to be impaired for purposes of compensation if his lung function is equal to or less than 75% of the predicted value (normed for age, sex and height).¹⁸⁴

The predicted values adopted in the regulations were derived in a widely used study of non-Hispanic Whites in the Tucson, Arizona

¹⁸² See 28 C.F.R. § 79.36(d)(1)(ii)(B). More precisely, these tests provide a quantitative assessment of the volume changes achieved by various breathing maneuvers. Garay, supra note 147, at 189. They provide information for an analysis of volume-time and flow-volume relationships, allowing an assessment of the respiratory system in action. Id. See generally American Thoracic Society, Standardization of Spirometry - 1987 Update, 136 AM. REV. RESPIR. DIS. 1285-98 (1987).

¹⁸³ Bruce A. Shaffer, et al., Prediction of Lung Function in Hispanics Using Local Ethnic-Specific and External Non-ethnic-specific Prediction Equations, 147 AM. REV. RESPIR. DIS. 1349, 1349 (1993).

¹⁸⁴ 28 C.F.R. § 79.36(d)(1)(ii)(B).

area.¹⁸⁵ However, it is now acknowledged that the "normal" values for pulmonary function tests can vary by racial and ethnic differences.¹⁸⁶

Predicted values for healthy Native Americans of the Southwest have been generated in at least one study.¹⁸⁷ This study concludes that the predicted values for the two pulmonary function measures employed in the regulations, FEV₁ (forced expiratory volume in 1 second) and FVC (forced vital capacity), may be sufficiently different between male Native Americans and Non-Hispanic Whites to warrant separate ethnic-specific standards.¹⁸⁸ This study suggests that the predicted values for these two measures in Native Americans are sometimes greater than the predicted values for non-Hispanic Whites.¹⁸⁹ Consequently, it is possible that a Native American miner with greater than 25% reduction in spirometric values will fall above the minimum value in the regulations, and erroneously be judged to have "normal" pulmonary function.

¹⁸⁵ See R.J. Knudson, et. al., Changes in the Normal Maximal Expiratory Flow-volume Curve With Growth and Aging, 127 AM. REV. RESPIR. DIS. 725 (1983).

¹⁸⁶ See AMA, supra note 131, at 160; Arnold D. Renzetti, et al., Evaluation of Impairment/Disability Secondary to Respiratory Disorders: ATS Statement, 133 AM. REV. RESPIR. DIS. 1205, 1206 (1986) (official statement of the American Thoracic Society).

¹⁸⁷ See Robert O. Crapo, et al., Normal Spirometric Values in Healthy American Indians, 30 J. OF OCCUPATIONAL MED. 556 (1988). The authors derived "normal" predicted pulmonary function values for Native Americans by performing spirometry for 300 Native Americans. Of the test group, 75% of the women and 85% of the men were Navajo. Id. at 557. The overwhelming number of Native American underground uranium miners were Navajo. See supra note 68.

¹⁸⁸ Id. at 559.

¹⁸⁹ Id. at 560.

The Committee is not convinced, however, that the one existing study of Native Americans is sufficiently compelling to justify at this time modifying the regulations to incorporate separate predicted values for Native Americans. For one thing, the extent to which the predicted values generated by the Crapo study are accurate for an older population, such as the miner-claimants, is not clear. The median age of the participants in the Crapo study is significantly lower than the age of most claimants seeking compensation for pulmonary function disorders.¹⁹⁰ Additionally, it is unclear whether the predicted values for the FVC and FEV₁ measures used by the Program differ significantly from those derived by the Crapo study,¹⁹¹ which suggests that ethnic-specific equations will in practice help few if any miners.

Additional studies that use a more representative, older population sample may conclusively demonstrate that there are significant differences in pulmonary function between Native American and White miners, thereby warranting separate standards. We recognize that some may argue for adopting separate ethnic-specific standards now even though a difference in "normal" pulmonary function has not been conclusively established, and any such difference appears small. Such an approach would err on the side of benefiting Native Americans, which would be consistent with the expressed intent of Congress to view evidence in a light most favorable to this class because of the federal

¹⁹⁰ The age distribution of the male subjects in the study ranged from 18-66. The median age of the male subjects was 33.9 +/- 12.8 years. Id. at 558.

¹⁹¹ Id. at 559.

government's breach of its special trust relationship.¹⁹² Nonetheless, we believe that a stronger, more compelling scientific basis than presently exists in the literature is both a necessary and appropriate prerequisite for the adoption of this ethnic-based classification. We recommend, therefore, that the Program reconsider this question when additional data becomes available.

2. Required Reduction in Lung Function to Qualify As Impaired

As set out above, the regulations define pulmonary impairment for purposes of compensation as either an FEV₁ or FVC result equal to or less than 75% of the predicted value. It was pointed out to the Committee that this standard is not quite consistent with the definition of impairment adopted by the American Thoracic Society (ATS). The ATS, in a 1986 official statement, defined "normal" pulmonary impairment as FVC and FEV₁ greater than or equal to 80% of predicted value.¹⁹³ A claimant is classified as impaired if either measure is below 80%; the degree of impairment -- mild, moderate or severe -- is proportional to the percentage loss of pulmonary function.¹⁹⁴

We recommend that the regulations be modified to comport with the definition of impairment adopted by the ATS. Indeed, the regulations expressly require that the pulmonary function tests be performed in

¹⁹² See 136 Cong. Rec. at S11754 (daily ed. Aug. 1, 1990) (colloquy between Senators Hatch, DeConcini, Inoyue and McCain).

¹⁹³ Renzetti, supra note 186, at 1206.

¹⁹⁴ Id. See supra note 132.

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APPENDIX A

Appendix

Establishment of Data-Based Criteria for Radon-Associated Lung Cancer

Introduction

This appendix presents the scientific basis for suggested revisions of criteria for the compensation of lung cancer among underground uranium miners from the eligible regions of the U.S. Radioactive radon (more specifically radon-222, hereafter referred to as radon) is an inert gas and is a decay product of radium-226, the fifth progeny of uranium-238 which is ubiquitous in the crustal rock of the earth. Radon can migrate from soils and rocks and accumulate in enclosed areas, such as homes and underground mines. The carcinogenic potential of radon derives principally from alpha particles (helium nuclei) emitted during radioactive decay of the short-lived progeny of radon-222, that is, polonium-218 and polonium-214.

Since the 1970s, a substantial amount of research, epidemiologic studies in humans, experimental animal studies, and *in vitro* cellular studies, has been published, that establishes the causal link between exposure to radon and its decay products and the development of lung cancer. Periodically, the body of research material has been reviewed by expert committees. Reviews have been conducted by the National Council on Radiation Protection and Measurements (NCRP 1984) and by the International Commission on Radiation Protection (ICRP 1987). In 1988, the fourth National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiations (BEIR IV) evaluated the scientific literature, and for the first time, conducted a pooled analysis of original data from four epidemiologic studies of

miners, including a study of Colorado Plateau uranium miners (NRC 1988). The BEIR IV Committee reiterated the causal link of radon progeny exposure to lung cancer risk, and developed a risk model that described the exposure disease relationship. The BEIR IV model has been the principal model used for the evaluation of radon risks from exposures in mines and in homes. More recently, building on the work in the BEIR IV Report, scientists of the U.S. National Cancer Institute in collaboration with principal investigators of all (eleven) cohort studies of radon-exposed underground miners conducted a pooled analysis of original data (Lubin et al. 1994; 1995), including recent updates of studies of Colorado Plateau uranium miners (Hornung and Meinhardt 1987) and New Mexico uranium miners (Samet et al. 1991). The pooled analysis included a total of nearly 1.2 million person-years of observation and over 2,700 lung cancer deaths, nearly eight times the data available to the BEIR IV Committee.

The BEIR IV analysis and the NCI-led analysis showed that relative risk (RR) of lung cancer was strongly related to cumulative radon progeny exposure in units of Working Level Months¹ (WLM). The analyses indicated that the exposure-response relationship in the RR was complicated by a dependence on other factors, notably that the exposure-response relationship declined with current age (referred to as attained age) and time since the exposure occurred. These patterns indicate that the effects of exposure vary in a complex way as an individual ages. In terms of the RR, the effects of exposure lessen as the age of a miner increases and with time since cessation of radon progeny exposure. The analyses also found

One Working Level (WL) equals any combination of radon progeny in one liter of air which results in the emission of 130,000 MeV of energy from alpha particles. WLM is the product of time, in units of 170 hours, and WL.

an inverse exposure-rate effect, that is, for the same total exposure, exposures occurring over a shorter duration of time (at a higher exposure rate) were less harmful than exposures occurring over a longer duration (at a lower exposure rate). In the analysis of the Colorado and New Mexico data which is presented below, the inverse exposure-rate effect was not statistically significant and will be omitted. The pooled analyses also found that risk from radon progeny exposure did not depend on the age at which a miner was first exposed.

The procedures used in the BEIR IV and NCI-led pooled analyses are the same as the methods used in the analysis presented in this appendix. Specific information is provided in those reports.

RECA does not establish an entitlement program whereby all lung cancer cases are compensated, but seeks to establish a program of fairly compensating lung cancer cases for harm done during the mining of uranium. In this regard, the most appropriate basis for making such a determination is the application of the most relevant data. There have been two epidemiologic studies of uranium miners in the four state region of Arizona, Colorado, New Mexico, and Utah. One study was initiated in the 1960s by the U.S. Public Health Service and currently is being conducted by the National Institute for Occupational Safety and Health (Lundin et al. 1971; Hornung and Meinhardt 1987). A second study was initiated at the University of New Mexico (Samet et al. 1991). The study populations constitute a substantial fraction of all miners eligible for compensation under RECA, and thus are directly relevant to establishing factors predictive of lung cancer occurrence. The two studies include nearly 190,000 person-years of observation and 470 lung cancer cases, and provide the data used in our evaluation of compensation criteria.

The analyses presented in this appendix use outcome and exposure information as provided by study investigators. For comparing the likelihood that an observed lung cancer in a miner is due to radon progeny exposure using non-WLM-based criteria, we assume that at a minimum a worker is able to document work history, including calendar years of underground employment.

This appendix considers five topics related to the evaluation of whether an observed lung cancer in a miner is the result of exposure to radon and its decay products accrued during time as an underground miner.

- (1) Pooling data from two studies of uranium miners, risk models are developed that reflect the causal association between radon progeny exposure and lung cancer occurrence. These models form the basis for recommending new guidelines for compensation. Because the risk model is derived directly from an analysis of a substantial fraction of all eligible miners, the recommended criteria incorporates a direct evaluation of the likelihood that a lung cancer was the result of exposure to radon progeny.
- (2) The scientific basis for the joint association of radon progeny exposure and cigarette use and the occurrence of lung cancer is reviewed. Based on analysis of available miner data, the most appropriate description of the joint RRs can be interpreted to specifying eligibility criteria for non-smokers which are less stringent than for smokers. However, results to date indicate that due to chance alone, the joint RRs are also interpretable as consistent with using the same compensation criteria for smokers and non-smokers.
- (3) Evidence indicates that Native Americans do not smoke or are generally light smokers. Anecdotal reports suggest that radon progeny exposure may be especially deleterious in

Native Americans. Data are examined and recommendations are suggested.

- (4) In response to concerns of the President's Advisor Panel on Human Radiation Experimentation on uncertainties associated with the estimation of a worker's exposure, an alternative to a WLM-based risk model is developed. This non-WLM based model is used to recommend an alternative scheme for establishing compensation eligibility when information on mine location is missing or when measurement data are missing or inadequate for accurate estimation of WLM.
- (5) RECA criteria for compensation require that all exposures occurs prior to 1972, that for non-smokers total cumulative exposure should equal or exceed 200 WLM, and that for smokers exposure should equal or exceed 500 WLM or if lung cancer occurs under 45 years of age exposure should equal or exceed 300 WLM. Based on current epidemiologic findings, these criteria do not reflect a scientifically sound attribution of risk of lung cancer risk from radon progeny exposure, and therefore do not appropriately discriminate lung cancer cases that are more likely than not due to radon progeny exposure.

One issue of importance is the requisite "fairness" of the RECA compensation criteria for lung cancers in uranium miners. It is worth considering what is meant by fairness in the context of lung cancer attributable to radon progeny exposure. Compensation for lung cancer is based on whether it was more likely than not due to radon progeny exposure. This stipulation can be reflected in the probability of causation (PC), which is a probabilistic statement of the likelihood that a lung cancer case is due to radon progeny exposure. A PC equal to or greater than 0.5 indicates that the disease event was more likely than not to have

been due to the causal agent.

The validity of a PC is based on the validity of the model used to describe the disease-exposure association. If the model omits important factors or misspecifies the functional form of the relationship, then PCs computed from the model may not measure the true likelihood of the event being caused by the exposure. With observational data and without a precise understanding of the biological mechanisms of the carcinogenesis process at the level of the cell and of the organ system, the true risk relationship, and thus the true PC, can never be determined with absolute certainty. However, a risk model that provides a more accurate characterization of the observed data will necessarily result in a computed PC which has greater validity.

In the view of this Committee, fairness in the compensation criteria is reflected in the PC. The more accurate the model used to compute PCs the greater fairness there is in the determination of eligibility. The risk model which is developed below fits the pooled Colorado and New Mexico miner data significantly better than the model implied by the RECA criteria. Thus, criteria based on the new risk model would imply a greater fairness than the RECA criteria.

Current scientific understanding about the relationship between radon progeny exposure and lung cancer does not support the RECA criteria, and therefore the RECA criteria should not be viewed as the benchmark of fairness by which to judge any new criteria. It is not the case that new criteria can be fair only if greater numbers of lung cancer cases are compensated. As a result, using criteria based on a new risk model, some lung cancer cases may be eligible for compensation who would do not satisfy current RECA criteria.

Conversely, some lung cancer cases who would not be eligible under a new scheme, indicating that their lung cancer is more likely than not due to factors other than radon progeny exposure, may be eligible under current RECA criteria.

Finally, true dose to lung tissue from radon and its progeny can never be known with certainty. WLM estimates in the miner data are approximations and are themselves subject to uncertainty. Thus, any WLM-based model which is developed, while having greater validity than the RECA-based model, cannot be considered as the “gold standard” that identifies the true level of risk.

Before proceeding, we briefly review the scientific basis for the causal relationship between radon progeny exposure in miners and lung cancer, define the data available for analysis, and describe the methods of analysis, including the computation of the PC.

Scientific basis for the association of radon progeny exposure and lung cancer

Effects of radon progeny exposure in underground miners

Since the mid-1980s, expert committees have sought to characterize risk of lung cancer from exposure to radon and radon progeny. Two early reports, by the National Council on Radiation Protection and Measurements (NCRP 1984) and by the International Commission on Radiation Protection (ICRP 1987), relied on heuristic summarizations of existing results in the literature. The NCRP committee assumed that the effects of exposure directly added to the age-specific lung cancer rate in non-exposed workers, and assumed no synergistic effects of cigarette smoking. In the latter report, the ICRP committee assumed a constant RR model, whereby the effects of exposure multiple the age-specific lung cancer rate in non-exposed

workers. For exposures in miners, the multiplicative effects were assumed unchanged for all ages and without regard to years since last exposure. Recent analyses have shown that these simple characterizations of risk from radon progeny exposure are incorrect. In 1988, the NAS BEIR IV Committee was the first expert committee to conduct original analyses using modern statistical methodology. The model linking WLM and lung cancer risk was complex. The BEIR IV analysis showed that over a broad range of exposures RRs were generally consistent with a linear relationship; however, they also showed that the RR relationship was not constant, but decreased with current age of the individual and with time since the exposure occurred. These patterns of variation in the exposure-response relationship have since been sustained in a pooled analysis by Lubin et al. (1994; 1995) using data from 11 cohort studies, including the four in the BEIR IV Report.

Effects of smoking on risk of lung cancer due to radon progeny exposure

Cigarette smoking is the major cause of lung cancer. Several investigators have considered the effects of smoking on the estimates of risk from exposure to radon progeny. Analyses have provided convincing evidence that, although the majority of miners were smokers, the increased risk with WLM exposure is not due solely to smoking. Based on formal analyses, with the Colorado study providing the most complete information, it has generally been concluded that there is a synergistic relationship for WLM and smoking, that is, the lung cancer risk for exposure to both agents is greater than the sum of the risks for each exposure individually.

Analyses support a joint association which is most likely intermediate between multiplicative and additive, although analyses also indicate that the association is consistent

with a multiplicative association. An association which is less than multiplicative implies that the RR for radon progeny is greater in non-smokers than in smokers. In the pooled analysis of 11 cohort studies, non-smokers had a 3-fold greater exposure-response relationship than smokers. Because radon progeny exposure multiplies the background disease rate and because smokers have a much higher lung cancer rate than non-smokers, a “greater than additive” association results in a radiogenic excess that is substantially greater in smokers than in non-smokers.

Effect of radon progeny exposure in Native Americans

Because of small numbers of study subjects, analyses of Native American miners have been limited. There have been case reports of lung cancer among Native Americans of the Southwest, who were involved in mining. In the most complete analysis, Samet et al. (1984) reported on 32 lung cancer cases diagnosed between 1969 and 1982, in which 23 had a documented history of uranium mining, while none of the 64 matched controls had been uranium miners. This suggests a high RR with uranium mining in Native Americans, who traditionally do not smoke or are light smokers. However, results are insufficient for quantitative risk estimation or for comparison with non-Native American miners.

Cohort studies of U.S. uranium miners

Colorado Plateau uranium miners

Cohort definition and follow-up

The Colorado Plateau uranium miners study was one of the earliest of the modern epidemiologic studies to document increased lung cancer risk with exposure to radon progeny

(Lundin et al. 1971). Extensions of the follow-up through 1977, 1982, 1987 and 1990 were subsequently carried out. Initially established by the U.S. Public Health Service (PHS), the cohort follow-up is now under the auspices of the U.S. National Institute for Occupational Safety and Health. Work history information was available through 1969; little additional exposure is thought to have occurred after that time since many workers had retired and many uranium mines in the Colorado Plateau had closed. In contrast to the pooled analysis by Lubin et al. (1994), we include all miners and all exposures in the analysis.

The current analysis utilizes the most recent update with follow-up through 1990. A total of 115 workers, including 12 lung cancer cases, in the Colorado cohort qualified for and were included in the cohort study of New Mexico uranium miners (defined as all underground miners medically evaluated at the Grants Clinic, Grants, New Mexico). These miners were included only once in the pooled analysis.

Members of the Colorado Plateau study cohort included workers in the four States of Arizona, Colorado, New Mexico, and Utah, who had completed at least one month of underground uranium mining, who volunteered for at least one medical examination between 1950 and 1960, and who provided personal and occupational data of sufficient detail for follow-up and for exposure estimation. Information on follow-up and ascertainment of vital status was obtained from records of mining companies, state vital statistics offices, the U.S. Social Security Administration, the Internal Revenue Service, the Veteran's Administration and, most recently, the National Death Index, and by direct contact. Cause of death was determined from State death certificates.

Estimation of radon progeny exposure

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Prior to 1951, only a few measurements of radon were made. During the period 1951-60, radon progeny measurements were made by the PHS, several State agencies and the U.S. Bureau of Mines. Data were considered representative of areas in which most of the miners worked. After 1960 and until the latest samples were collected in 1968, most samples were taken by mine inspectors. Between 1951-68, almost 43,000 measurements of radon progeny in 2,500 mines were obtained. However, as noted by Holaday (NRC 1988, quoted by Lundin et al. 1971), most measurements were made for control purposes and may have overestimated exposure to workers.

A little over 10% of the miners had their exposure estimated entirely from measurements taken in the mines. Measures of exposure to radon progeny were generally collected using the Kusnetz method, which was an area sample based on alpha counts collected using a filter/pump apparatus. When multiple samples were available in a given mine and year, the arithmetic mean was used to characterize annual WL levels. When measurements were not available for a given mine and year, three different methods were used to assign WL levels. (1) When data for a mine were incomplete, the annual average WL was used for the level within a maximum of two years from the measured year. (2) When gaps of greater than four years existed for a mine, averages of other mines in the same geographic area were used to estimate annual WL levels. (3) If no data existed in adjacent years or within the geographic region, estimates of WL were made based on knowledge of ore bodies, ventilation practices, and other factors thought to influence radon progeny levels.

The amount of time each miner worked underground was obtained from several sources. Initial data were collected at the time of the physical examination, one of the defining criteria

for enrollment into the cohort. This information was used to provide work histories to that point in uranium mines, as well as past employment in other hard rock mines. Additional data were collected from mining company records and from an annual census taken by the PHS and by the Colorado Bureau of Mines.

The number of hours per month underground was taken to be 170. No adjustment was made for vacations, sick leave, or overtime. However, gaps in employment histories of one month or more were recorded and, when available, taken into account in the exposure estimates.

When a census identified a miner with no work history at a particular mine, the miner was assumed to have worked in that mine for a period of six months prior and subsequent to the census date.

A relatively small proportion of the study group had WLM estimates based completely on measured data. While extrapolated data may be subject to greater error, the extent of misspecification depended largely on estimation procedure. An analysis of error found a coefficient of variation of 112% that included sampling error, counting error, and environmental fluctuations over each year (Hornung 1985). The coefficient of variation in the three estimation procedures ranged from 121% to 186%. When all estimation procedures were averaged over the number of measurements and years worked for each miner, the coefficient of variation for cumulative WLM was approximately 97%.

Exposure rates in WL have decreased dramatically over time. Figure 1 shows mean exposure rates in WL for 1950-69. The primary factor in the decline was the introduction of ventilation systems in mines. There is approximately a 20-fold reduction in mean WL for the

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mines over this time period. Figure 2 shows the number of measurements made in the mines over this same period of time. The number of measurements peaked in the late 1950s and early 1960s and declined thereafter. These figures make it clear that if compensation criteria are based on duration of time spent in underground, as suggested by the Advisory Panel, calendar period of mining must also be considered.

Estimation of other mine exposures

For the present analysis, data were available on years of previous underground hard rock mining, as well as an estimate of WLM accumulated in these mines. There were no data available on other specific exposures that may have been encountered in the uranium mines, such as arsenic-containing dusts or silica, or in the previous hard rock mines.

Information on tobacco use

Data on smoking were obtained at the annual censuses of miners and from mail questionnaires, obtained on one to four occasions between 1950 and 1960 and at other times between 1963-69 (NRC 1988; Whittemore and McMillan 1983).

New Mexico uranium miners

Cohort definition and follow-up

The State of New Mexico was one of the four States included in the Colorado Plateau uranium miners study of the PHS. In 1977 a separate study cohort was established by investigators from the University of New Mexico. The cohort consisted of men who had undergone a mining-related physical examination at the Grants Clinic in Grants, New Mexico and who had worked at least one year underground in New Mexico prior to December 31,

1976 (Samet et al. 1991). The Grants Clinic opened in 1957 and performed most pre-employment as well as follow-up examinations for area mines. Although the geographic area of the New Mexico study was spatially related to the source area of workers for the Colorado Plateau cohort and although some workers qualified for inclusion in both cohorts, the New Mexico workers had substantially lower mean exposures.

Vital status was determined by searching New Mexico vital statistics records, the New Mexico Tumor Registry and State drivers' license records. The cohort was also matched against the records of the Social Security Administration and the U.S. National Death Index. Death certificates were obtained for the deceased and cause of death ascertained. After excluding men with missing work history, birth year, or follow-up information, vital status of 3,469 workers could be ascertained; 390 workers have a verified cause of death.

Estimation of radon progeny exposure

Measurements of radon and its progeny were obtained from various sources. Prior to 1968, data on radon progeny came from the State Mine Inspector, the State Health and Environment Department, and records from the Colorado Plateau study. These data were in the form of annual estimates for individual mines.

From 1967-1985, estimates of WLM exposure were taken from the WLM reports for individual miners from mining companies in the area, submitted to the U.S. Bureau of Mines or its successor agencies, the Mining Enforcement and Safety Administration, and the Mine Safety and Health Administration. Records were generally complete for the larger mining companies, while generally not available for the smaller companies (Morgan and Samet 1986).

Estimation of other mine exposures

For each worker, information from Clinic records was available on whether he previously worked in a hard rock mine.

Information on tobacco use

Clinic records indicated smoking status; current smoker, former smoker, or never smoked.

Material and methods of analysis

Pooled miner data

We developed risk models by directly pooling data from the Colorado and New Mexico studies. In the Colorado and New Mexico studies, there was a total of 7,475 miners, who accrued 187,406 person-years of observation and 470 lung cancer deaths. There were 951 Native American miners with a total of 35 lung cancers. For Colorado and New Mexico miners, mean cumulative exposures among exposed were 681 and 110 WLM, respectively.

Relative risk regression models

Poisson regression methods (more correctly called piecewise-exponential regression models) were applied to the pooled data. Methods are based on a RR model which is linear in WLM exposure, but allows for variations with other factors. These methods are similar to and are detailed in the BEIR IV Report and Lubin et al. (1994). The background risk of lung cancer among “non-exposed” miners was specified using factors for study, attained age, calendar year, previous hard rock mining experience, smoking status, and ethnicity (whites and Native Americans). These factors were adjusted in the modeling by categorizing the variables and including stratum-specific parameters.

RECA criteria and characterization of lung cancer risk

RECA criteria were meant to identify lung cancer cases which were more likely than not due to radon progeny exposure. This can be restated as a PC greater than or equal to 0.5. Since a linear model for the RR in WLM is a good first order approximation, RECA criteria can be re-expressed in terms of the RR of lung cancer for cumulative exposure (w), smoking status and age. For non-smokers, the specification of a PC of 0.5 at 200 WLM implies a linear RR model which is independent of age, namely,

$$RR_{ns}(w) = 1 + 0.005 \times w$$

For smokers, the specification of a PC of 0.5 at 300 WLM for ages under 45 years and 500 WLM at ages 45 years and older implies the following RR models,

$$RR_{smk,<45}(w) = 1 + 0.0033 \times w$$

for under age 45 years, and

$$RR_{smk,\geq 45}(w) = 1 + 0.002 \times w$$

for ages 45 years and older.

Direct analysis of data reveals that these RR patterns do not provide an adequate fit. Compared to the models developed below, the RECA-based model for the RR is statistically rejected, $p < 0.001$.

For current ages 45 years and over (the majority of miners), the 500 WLM criterion implies that the excess RR of lung cancer increases 0.2% per WLM and that the increase is constant with age and other factors. The BEIR IV Report (1988) demonstrated conclusively that a model in which the RR depends only on WLM does not adequately describe the

association between radon progeny exposure and lung cancer. The BEIR IV analysis indicated that the RR of lung cancer increases with increasing WLM (as implied in the RECA-based model), but that the exposure-response relationship decreases with attained age and time since the exposure occurred. In the more recent analysis of 11 miner cohorts, analysis showed that in every cohort examined, a constant relative risk model did not provide an adequate fit to the data.

Among miners in the pooled Colorado and New Mexico studies, the RECA criterion that distinguishes lung cancer events under age 45 years has little impact on eligibility for compensation. Of the 470 lung cancer deaths in the joint population, 32 lung cancer cases occurred under age 45 years, with 27 lung cancer cases in smokers. All five non-smokers qualify for compensation. Of the 27 smokers, twenty qualify under RECA criteria. All, but two workers, had exposures above 500 WLM (and thus qualified under the general exposure criterion). Only two smokers under age 45 had exposures in the 200-300 WLM range. (Using the model defined below, all 27 smokers would be eligible for compensation, since their disease occurred at such young ages, when lung cancer is usually quite rare.)

Definition of probability of causation

Probability of causation is the chance that an observed lung cancer was due to radon progeny exposure, i.e., PC is the increase in the probability of lung cancer due to exposure. Formally, for a lung cancer cases occurring at age a , for an individual with WLM exposure w , and with other factors z , the PC is defined as

$$PC(w,z) = \frac{P(\text{lung cancer given } w \text{ and } z) - P(\text{lung cancer given } w=0 \text{ and } z)}{P(\text{lung cancer given } w \text{ and } z)}$$

Dividing the numerator and denominator by the disease probability in the non-exposed, one obtains the PC as a function of the RR, namely,

$$PC(w,z) = \frac{RR(w,z) - 1}{RR(w,z)}$$

For a given level of exposure w and values for other factors z , the RR with exposure is determined by the risk model.

Topic 1: Modeling lung cancer risk and WLM

In modeling risk in miners, we have the benefit of having data on a substantial proportion of the population of interest, thus obviating the need for an extreme extrapolation from one population to another, possibly unrelated, population. Thus, risk levels can be based directly on relevant epidemiologic data. While the Colorado study provides limited information for exposures below 100-200 WLM, there are substantial data from New Mexico study in this range, where exposures were lower. In the New Mexico miners, mean year of first employment occurred about a decade later, in 1965, after substantial control measures had been put in place. Patterns of risk with exposure in the Colorado and New Mexico studies were consistent with patterns found in other populations of radon-exposed miners. This suggests that the observed patterns of risk in the data are not artifacts, but likely reflect underlying biological processes.

It is worth noting that, while there has been considerable advances in recent years in understanding the nature of the carcinogenic process from exposure to alpha particles,

knowledge is still incomplete. There are currently no biological markers of disease that can be used to attribute a particular case to radon progeny exposure, that is, given biological material from a lung cancer, it cannot be deduced whether the tumor was “caused” by radon exposure.

Development of risk model

Previous analysis of the Colorado data indicated that the exposure-response was non-linear, particularly at high exposures. Analyses also revealed that the exposure-response relationship varied with attained age and time since last exposure. Although our intent was to characterize risk as precisely as possible, we made several simplifications to the models to enhance their usefulness in developing a practical scheme based on PCs.

For simplicity, age was categorized into four levels, <50, 50-59, 60-69 and ≥ 70 years of age, and time since last exposure was categorized into three levels, <10, 10-19 and ≥ 20 years since last exposure. The following RR model along with several variants were fit to the data:

$$RR = 1 + \beta \times w^{\kappa} \times \phi_{\text{tsle}} \times \theta_{\text{age}} \quad (1)$$

where w is cumulative WLM and β the exposure-response parameter, κ is a “non-linear” effect of exposure, ϕ_{tsle} denotes the effects of the three time since last exposure categories, $\phi_{<10}$, ϕ_{10-19} , $\phi_{\geq 20}$, and θ_{age} denotes the effects of the four age categories, $\theta_{<50}$, θ_{50-59} , θ_{60-69} , and $\theta_{\geq 70}$. For identifiability, we set $\phi_{<5}=1.0$ and $\theta_{<50}=1.0$. Results of the model fitting are shown in Table 1. The table shows that the exposure-response relationship was significantly modified by both age and time since last exposure. These models were fit assuming a multiplicative association for radon progeny exposure and smoking status.

Based on model A in Table 1, approximately 77% of lung cancer cases can be attributed

to radon progeny exposure.

Cohort differences

The populations from the two studies arise from the same mining area of the U.S., although different criteria which were used to define entry into the cohort meant that mean exposure differed substantially. Model (1) was modified to test for cohort differences by replacing the exposure-response parameter β with study-specific parameters, β_{CO} and β_{NM} . The estimates were similar; the exposure-response estimate was about 30% greater for New Mexico, but a score test revealed that this difference was not statistically significant, $p=0.75$.

Topic 2: Cigarette smoking and lung cancer risk due to radon

Published analyses of the Colorado data and the New Mexico indicate that data are consistent with a multiplicative relationship between smoking and WLM, but that the data are also statistically consistent with a relationship less than multiplicative. A multiplicative relationship implies that the proportion of lung cancers due to radon progeny exposure is same in smokers and non-smokers. (Because the lung cancer rate is substantially greater in smokers, the model also implies that the absolute number of lung cancer cases due to radon is greater in smokers than non-smokers.) In determining risk, a multiplicative association suggests that the same model, and therefore the same compensation criteria, should be applied to smokers and non-smokers.

Analyses indicate that the most appropriate model is less than multiplicative. This implies that the increase in the RR with radon progeny exposure is somewhat greater in non-smokers than in smokers. The difficulty in applying a sub-multiplicative relationship is

the limited amount of data on lung cancer risk in non-smokers. This leads to a lack of adequate power to estimate precisely the nature of the joint association. An estimate of the “degree of sub-multiplicativeness” depends heavily on the postulated form for the joint association. Since a multiplicative model is consistent, the difficulty in the estimation of any particular sub-multiplicative model has lead most researchers and expert committees to apply the same risk model to smokers and non-smokers. This is the approach taken by the Environmental Protection Agency in applying miner-based risk models in the estimation of lung cancer risk from residential radon exposure.

Although the precise form for the joint RR is uncertain, current analyses indicate that the absolute risk of lung cancer due to radon progeny exposure and to cigarette smoking do not simply add. In the general population the lung cancer rate among smokers is 10-20 fold greater than the lung cancer rate among non-smokers. An additive RR association for WLM and smoking would imply that the excess rate of lung cancer due to radon is the same in smokers and in non-smokers. Data indicate that more radon-attributable lung cancer cases occur among smokers than non-smokers.

In the pooled analysis, Lubin et al. (1994; 1995) separated data into smokers and non-smokers and found that the exposure-response in non-smokers was about three times the exposure-response in smoker, indicative of a sub-multiplicative association.

Because there is evidence that Native American miners either did not smoke or were light smoker, Native American miners with missing data on smoking was classified as a non-smoker. In the pooled data, 427 miners, including 6 lung cancer cases, had no information on smoking, and were omitted from the smoking analysis.

There were 464 lung cancer cases with information on smoking habits, with 47 cases among non-smokers. With so few non-smoking lung cancer cases, we could not fit a separate model for non-smoking miners. To adjust the model for smoking status, model (1) was fit to all data, Table 1. (Smoking was still included as a stratification factor in the background.) Parameter values for the effects of non-linearity (κ), age and time since exposure were then fixed, with only the β parameter free to be estimated. The model was modified by replacing the exposure-response parameter β with smoking-specific parameters, β_{ns} and β_{smk} , then fit to the data on miners with smoking information. The changes in the β_{ns} and β_{smk} relative to the overall β were then applied to the model for all data (Table 1 column A).

In the miners with smoking information, relative to the overall β , the estimate for smokers was reduced by a factor of 0.8, while the estimate for non-smokers was increase by a factor of 3.0. For all data combined, the results in Table 1 column A were modified to reflect smoking status, $\beta_{ns}=3.0 \times 0.22=0.66$ replaces β for non-smokers and $\beta_{smk}=0.8 \times 0.22=0.18$ for smokers for the computation of PCs.

Topic 3: Radon risk among Native Americans

As indicated previously, there was little quantitative information that allows comparisons of lung cancer risk with radon progeny exposure among Native American miners with non-Native American miners. There was suggestive evidence that among smokers and among non-smokers the estimated exposure-response for Native American miners was greater than for non-Native American miners; however, the difference was not statistically significant ($p=0.18$). In addition, the relative impact of smoking on the estimated radon

exposure-response for Native-American miners was less than for non-Native American miners, i.e., the difference in the exposure-response by smoking status was smaller for Native American miners than for non-Native American miners, suggesting less of a difference between Native American smokers and non-smokers. Since the data on Native American miners are too limited to estimate an exposure-response trend specific for ethnicity and since Native Americans who do smoke are generally light smokers, we classify all Native American miners as non-smokers for risk estimation purposes.

Topic 4: Risk modeling based on duration of exposure and year first exposed

The President's Advisory Panel voiced concern about the misclassification of exposures and the inability to document exposure rates in miners for individuals seeking compensation. While some estimates of exposures may over-estimate true exposures, there was particular concern about possible under-estimation of exposures, and the possibility of unfair denial of compensation. The major source of error in exposure assessment is the uncertainty in exposure rates for the various mines and tunnels.

The risk models presented in Table 1 were based on the association of WLM and lung cancer risk, with modification by age and time since last exposure. This approach may be termed a science-based approach, in that the goal is to explore the underlying relationship between exposure and disease, and provide insights into the carcinogenic process. For the purpose of compensation, however, the calculation of a miner's WLM is often problematic and perceived to be unfair by applicants. An alternative approach relates cancer risk directly to duration of exposure and calendar year of employment, along with other factors. It would

be expected that duration of exposure and year could be known and verified with great accuracy, and therefore subject to less misclassification, and would be preceived to be fairer by applicants. The approach has the advantage that the calculation of risk appears more transparent.

It should be noted that WLM is not a true measure of lung dose, but is a surrogate of the true lung dose. Likewise, exposure determined in terms of duration of underground employment (and calendar year of first exposure) is also a surrogate of the true lung dose. Because exposure rate is ignored, it must be expected that duration of exposure (and calendar year of first exposure) may be less adequate than WLM as a surrogate for the true dose.

For simplicity in risk modeling and in risk determination, we use year of first exposure, rather than year of exposure. Since periods of work histories which were free of exposure constitute a relatively small proportion of total exposure durations, modeling duration and year exposed, or duration and year first exposed will define similar levels of risk. We defined three categories for year of first radon progeny exposure, <195-54, 1955-59, and ≥ 1960 . The RR of lung cancer was modeled in duration of exposure (denoted by d), as follows

$$RR = 1 + \beta \times d^k \times \gamma_{yfe} \times \phi_{tsle} \times \theta_{age} \times \tau^{(age - 60)} \quad (2)$$

where in contrast to model (1) β now denotes the duration-response parameter. The parameters γ_{yfe} denotes three parameters for categories of year of first radon exposure, <195-54, 1955-59, ≥ 1960 , while ϕ_{tsle} and θ_{age} represent parameters for categories of time since last exposure categories and attained age, and τ represents a parameter of the effect of continuous age minus 60 years. An empirical evaluation indicated that the inclusion of continuous age significantly improved model fit.

Results of modeling are shown in Table 2. Non-linearity of the duration-response relationship is only marginally significant, while year first exposed, time since last exposure, and attained age are significant modifiers of risk.

Although not directly comparable, the deviance (a measure of model fit) was substantially smaller using model (1) than model (2); for the “full” models, deviance values were 2,509.9 on 17,218 degrees of freedom for the WLM-based model, and 2569.9 on 17,216 degrees of freedom for the duration-based model without continuous age and 2557.7 on 17,215 degrees of freedom for the duration-based model with continuous age. This suggests that model (2) does not fit the data quite as well as model (1). The attributable risk of lung cancer using the duration-year first exposure model A in Table 2 was 73%, similar to the 77% attributable risk based on model (1) using WLM. (Note this is slightly higher than reported in Lubin et al. (1994), due to the inclusion of all exposure data. Lubin et al. excluded exposures above 3,200 WLM.)

Modification of the β estimate in the duration-based model (2) for smoking status was carried out as for model (1). Model (2) was fit to the miners with smoking information, all parameters, except β , were fixed. The model then was refit with β_{smk} and β_{ns} replacing β . Relative to the overall β , the estimate for smokers was reduced by a factor of 0.9, while the estimate for non-smokers was increase by a factor of 2.4. For all data combined, the results in Table 2 column A were modified to reflect smoking status, $\beta_{\text{ns}}=2.4 \times 22.3=54.4$ (with rounding) replaces β for non-smokers and $\beta_{\text{smk}}=0.9 \times 22.3=19.0$ for smokers for the computation of PCs.

The President’s Advisory Panel was concerned by the purported difficulty computing an

accurate WLM for a miner with lung cancer. In response to their concerns, the Committee developed a risk model based on duration of underground mining and year of first employment underground, factors which are likely known with a high degree of accuracy. However, another source of uncertainty that has yet to be considered arises from statistical sampling variability. The (asymptotic) statistical variability is captured in the covariance matrix of the parameter estimates. However, for the complex models described in Tables 1 and 2, calculating the variance estimate for a given set of covariates (age, time since last exposure, and WLM, or duration of exposure and year of first exposure, as well as smoking status) is not a simple matter, since one must account for the variance and covariance in the estimation of all the parameters. As suggested in the BEIR IV Report, a simple informal approximation is available, recognizing that statistical uncertainty is only one of many sources of uncertainty that burden accurate risk estimation. Stratifying on categories of attained age, calendar year of follow-up, study, ethnicity, previous mining experience and smoking status, we fit a simple constant RR model in WLM, namely,

$$RR = 1 + \beta \times w$$

Although this model does not represent the appropriate RR relationship, the estimate of β does represent a kind of average excess risk for the study population as a whole over the period of observation. Further, the estimate of variance then represents the approximate sampling variability for the data. In fitting the constant RR model in WLM to the data from the two cohorts, we obtained a multiplicative standard error of 1.25 for the estimate of β . Thus, to ensure that the computed RR for a miner captures his true level of risk with 80 percent assurance or 90 percent assurance the excess RR in model (1), i.e., $RR-1$, should be

multiplied by the factor $1.3=1.25^{1.28}$ or $1.4=1.25^{1.64}$, respectively. Because the approach is an approximation and the sampling variability is only one of the multiple sources of uncertainty in risk estimation, the limits are referred to as levels of assurance rather than levels of confidence which carries the connotation of a statistical confidence interval, which has a precise sampling interpretation and beyond our intended usage.

Model (2) in duration of exposure and year of first exposure lends itself to a similar approximation, using a constant RR model in duration of exposure, as opposed to WLM. In this case, the 80 percent assurance or 90 percent assurance levels are given by multiplying the excess RR in model (2) by the factors 2.2 or 2.8, respectively.

Adjusting the compensation criteria by a level of assurance decreases the number of false negative claims, i.e., rejecting claimants deserving of compensation. As a consequence, however, there is an increase in the number of false positive claims, i.e., compensating claimants who fails to meet a more likely than not criterion.

Topic 5: Comparisons PCs for data-based risk model and RECA-based models

As indicated above, the RECA-based model does not adequately account for patterns of risk, which therefore implies that RECA criteria do not adequately and fairly identify high risk workers. Using the 470 lung cancer deaths in the Colorado and New Mexico studies, we compute PCs using three different approaches: (i) based on the RRs derived from the RECA criteria, denoted PC_{RECA} ; (ii) based on fitting model (1) which utilizes the relationship of risk with WLM and other factors, denoted PC_{WLM} ; and (iii) based on model (2) which utilizes the relationship of risk with duration of exposure, calendar year of first exposure and other

factors, denoted PC_{dur} .

While PC_{WLM} is computed from what is thought to be the most appropriate model, any comparisons among these PC values is problematic, since the “true” relationship between exposure and disease cannot be known precisely.

Figure 3 compares PC_{RECA} with PC_{WLM} . Dotted lines denote a PC value of 0.5. The PC_{WLM} points follow curved paths of related values because of the categorical representations of age and time since last exposure. There were 296 lung cancer cases with both PC_{RECA} and PC_{WLM} greater than or equal to 0.5, termed the “true positives”; 22 cases with $PC_{RECA} \geq 0.5$ and $PC_{WLM} < 0.5$, termed the “false positives”, who would be compensated under RECA criteria, but not under the more appropriate risk model; 92 cases with $PC_{RECA} < 0.5$ and $PC_{WLM} \geq 0.5$, termed “false negatives”, who would not be compensated under RECA criteria, but would be compensated under the more appropriate risk model; and 60 cases with $PC_{RECA} < 0.5$ and $PC_{WLM} < 0.5$, termed “true negatives”, who would not be compensated under either approach. Out of 388 (296+92) lung cancer cases who would be compensated based on the more scientifically-based model, 92 or 24% would be ineligible under RECA criteria.

As indicated, the age 45 criterion for smokers has little practical impact on determining compensation. The question might arise whether any single WLM could ever adequately identify high-risk cases. Figure 4 plots PC_{WLM} , based on model (1), by cumulative WLM exposure. As illustrated, a simple criterion based on a single WLM (which would be represented by a vertical line) is insufficient to segregate lung cancer cases with high and low values for PC. For example, several cases with an estimated exposure of over 1,000 WLM had a value for PC_{WLM} of less than 0.5. These cases are individuals over the age of 70

at lung cancer occurrence and with more than 20 years since last exposure.

The correlation coefficient for PC_{WLM} and PC_{dur} was 0.88. Figure 5 shows a plot of PC_{WLM} and PC_{dur} for the 470 lung cancer cases. (The correlation coefficient for PC_{WLM} and PC_{RECA} is 0.65 and for PC_{dur} and PC_{RECA} is 0.57.) PC_{WLM} and PC_{dur} agree on the compensation classification of 89% (419/470) of cases. Using PC_{dur} as the sole basis for compensation, 2% (6/349) of lung cancers would be inappropriately compensated, while 45 or 12% (45/388) would be inappropriately rejected.

The rate of rejecting deserving lung cancer cases under a PC_{dur} scheme could be reduced by lowering the 0.5 criterion for the PC, with the corresponding consequence of increasing the number of non-deserving cases who are compensated. However, as suggested by Figure 4, no obvious and reasonable reduced standard (any vertical line) would capture a large majority of incorrectly rejected cases without markedly increasing the number of inappropriately compensated cases.

Development of model-based compensation criteria

The characterization of lung cancer events that are more likely than not due to radon progeny exposure can be re-expressed as $PC \geq 0.5$, which is equivalent to all cases with a computed $RR \geq 2.0$. Based on model (1), this characterization can be defined in terms of the level of WLM exposure for fixed categories of time since last exposure and age by solving the expression, $PC = (RR - 1)/RR$, for w , i.e.,

Tables of exposures for fixed categories of time since last exposure and attained age were computed for $RR = 2.0$. Table 3a shows WLM values for smokers, where $\beta_{smk} (=0.8 \times \beta)$

$$w = \left[\frac{RR - 1}{\beta \times \phi_{tse} \times \theta_{age}} \right]^{1/\kappa}$$

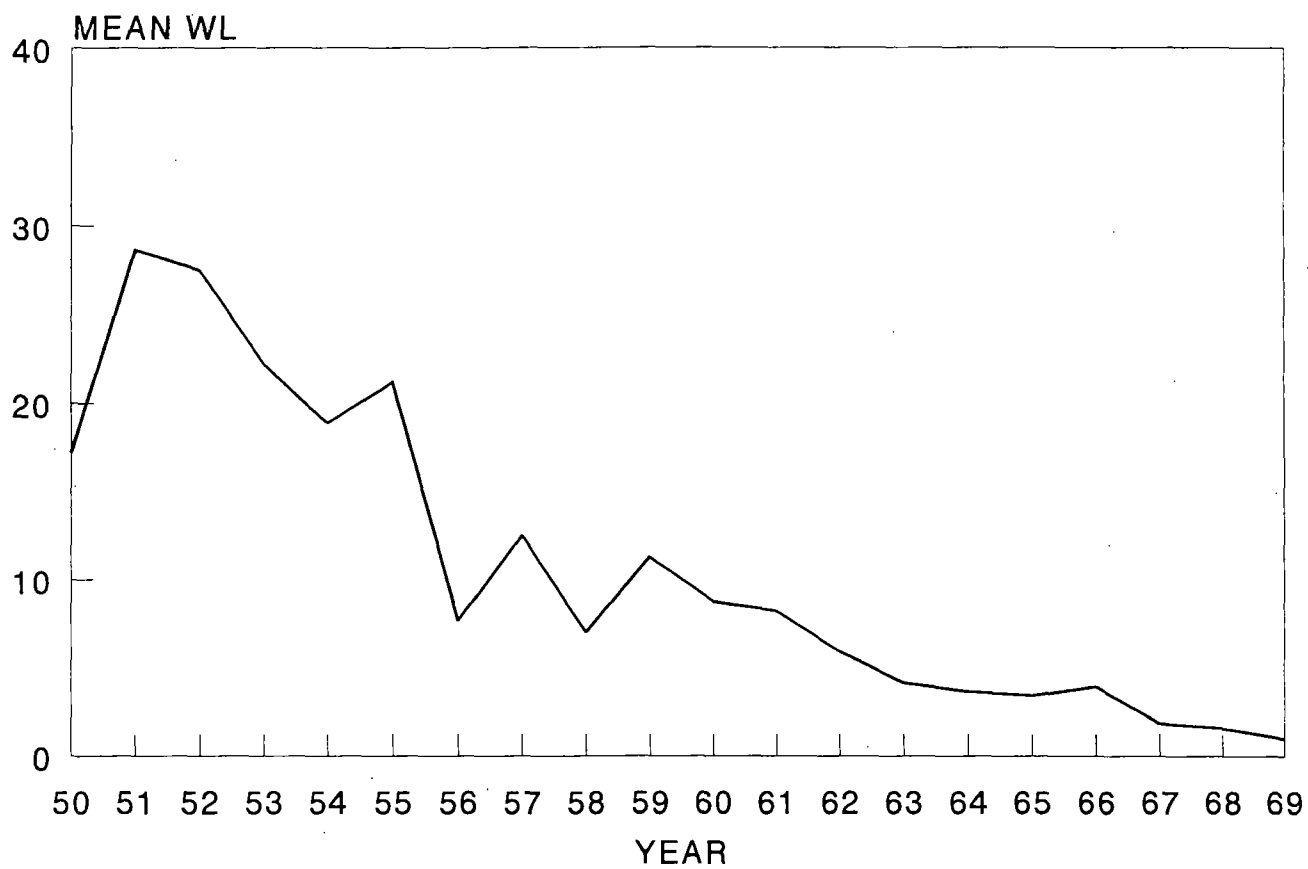
replaces β from Table 1, and for non-smokers, where β_{ns} ($=3.0 \times \beta$) replaces β . The table illustrates the impact of age at lung cancer and time since last exposure. The older the case occurs and the more distant in time since the last exposure the less the likelihood that the observed disease was due to radon progeny exposure, and therefore the greater the WLM required to meet the more likely than not criterion. The values of WLM that are required to meet the "more likely than not" criterion with 80 percent and 90 percent assurance are shown in Tables 3b and 3c, respectively.

A similar approach for adjusting the risk model for smoking status was used with model (2), using 0.9 and 2.4 as adjustment factors for β for smokers and non-smokers, respectively. Table 4a shows duration of exposure in years that would be required to meet a "more likely than not" criterion of lung cancer causality. The values of duration that are required to meet the "more likely than not" criterion with 80 percent and 90 percent assurance are shown in Tables 4b and 4c, respectively.

The criteria defined by Tables 3 and 4 were applied to the 470 lung cancer cases occurring in the pooled Colorado and New Mexico data (Tables 5 and 6). Using model (1) and the WLM-based approach, 388 (83%) cases would be eligible for compensation, with the numbers increasing to 401 (85%) or 403 (86%) after accounting for a 80 percent or 90 percent level of assurance in the RRs. Under model (2) and the duration/year first exposed-based approach, 349 (74%) cases would be eligible, with the number increasing to 394 (84%) or 408 (87%) after accounting for the level of assurance.

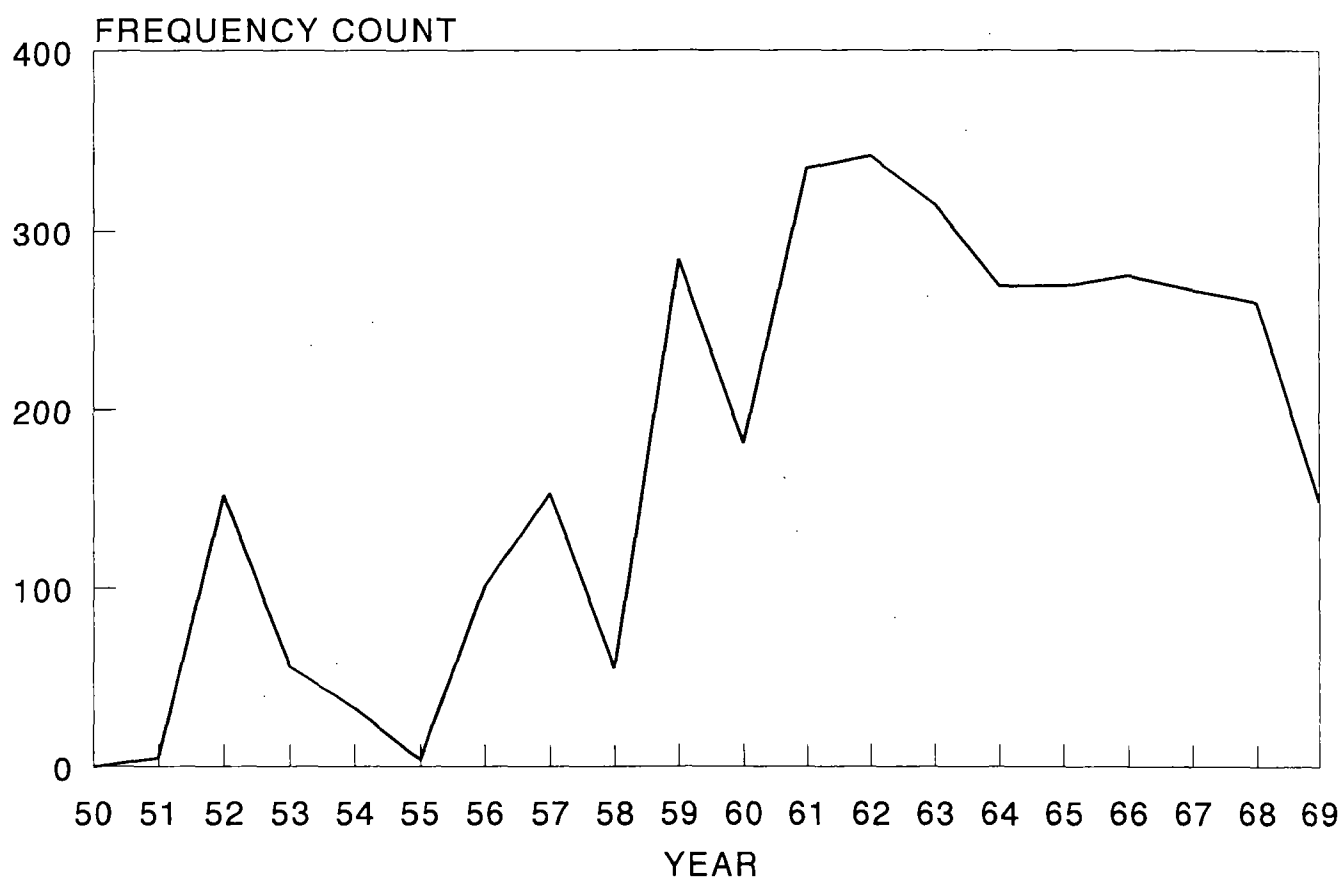
FIGURES

Figure 1: Mean levels of radon decay products measured in Working Levels (WL) in uranium mines surveyed during the period 1950-1969



PHOTOCOPY
PRESERVATION

Figure 2: Number of measurements of radon decay products in uranium mines surveyed during the period 1950-1969



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Figure 3: Comparison of PC_{RECA} and PC_{WLM}

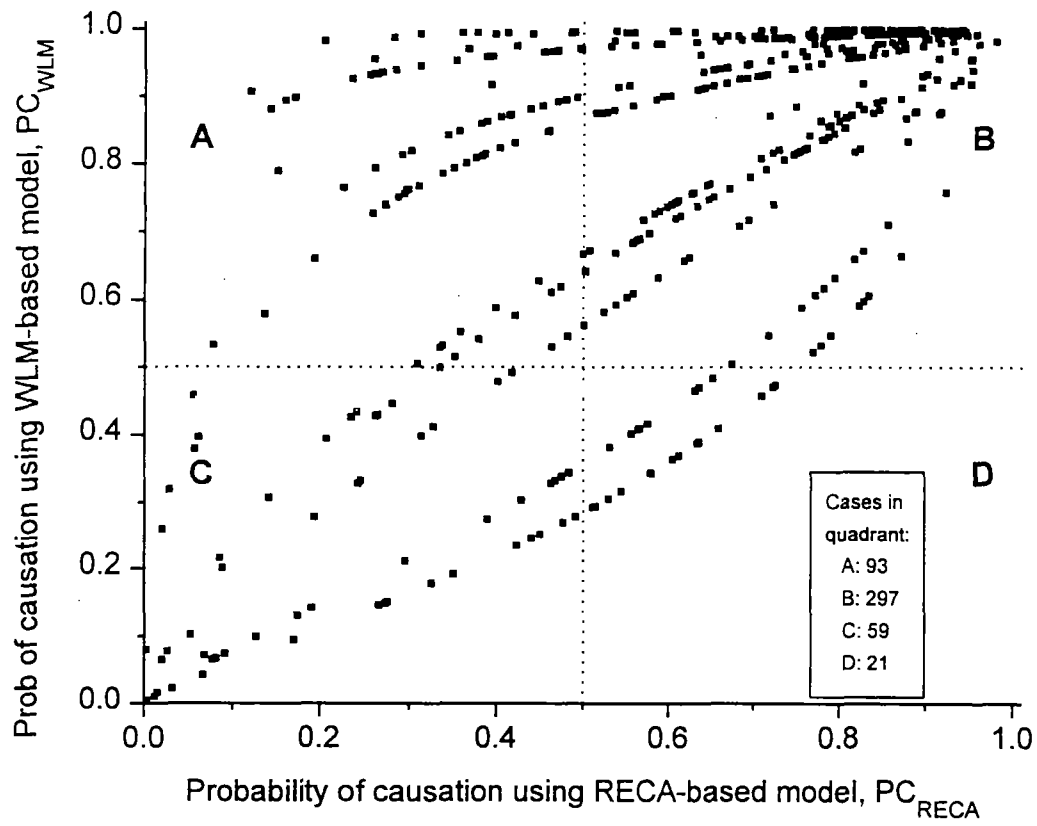


Figure 4: PC_{WLM} by cumulative radon progeny exposure

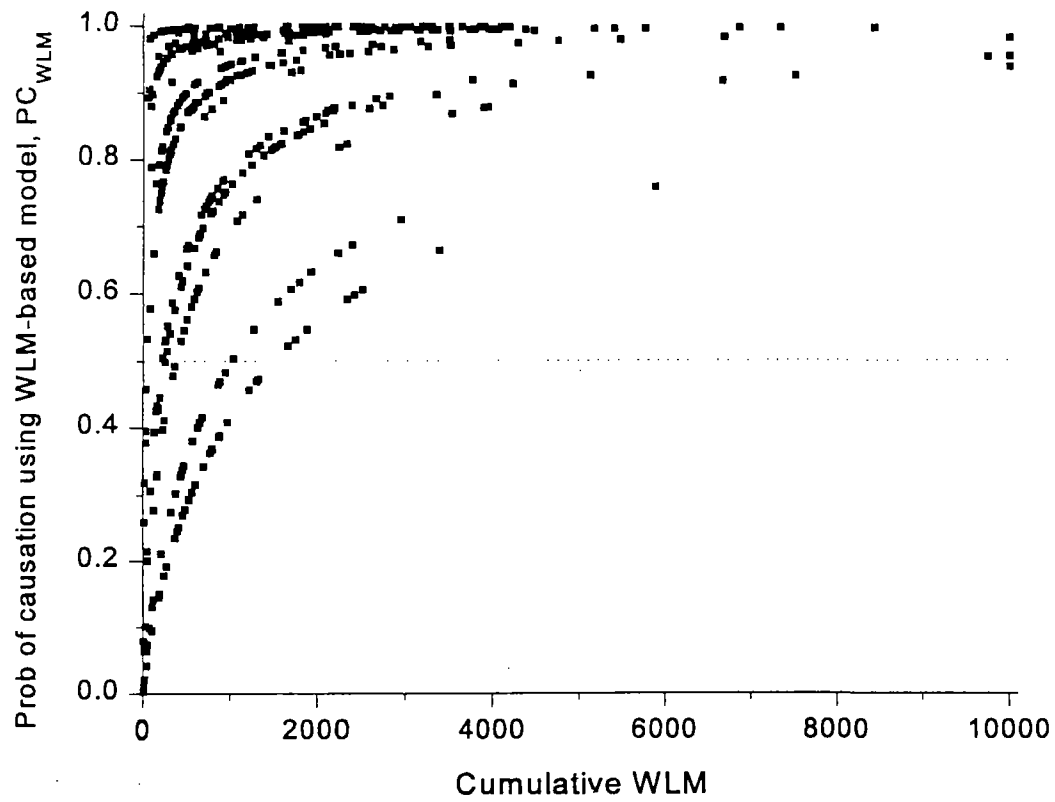
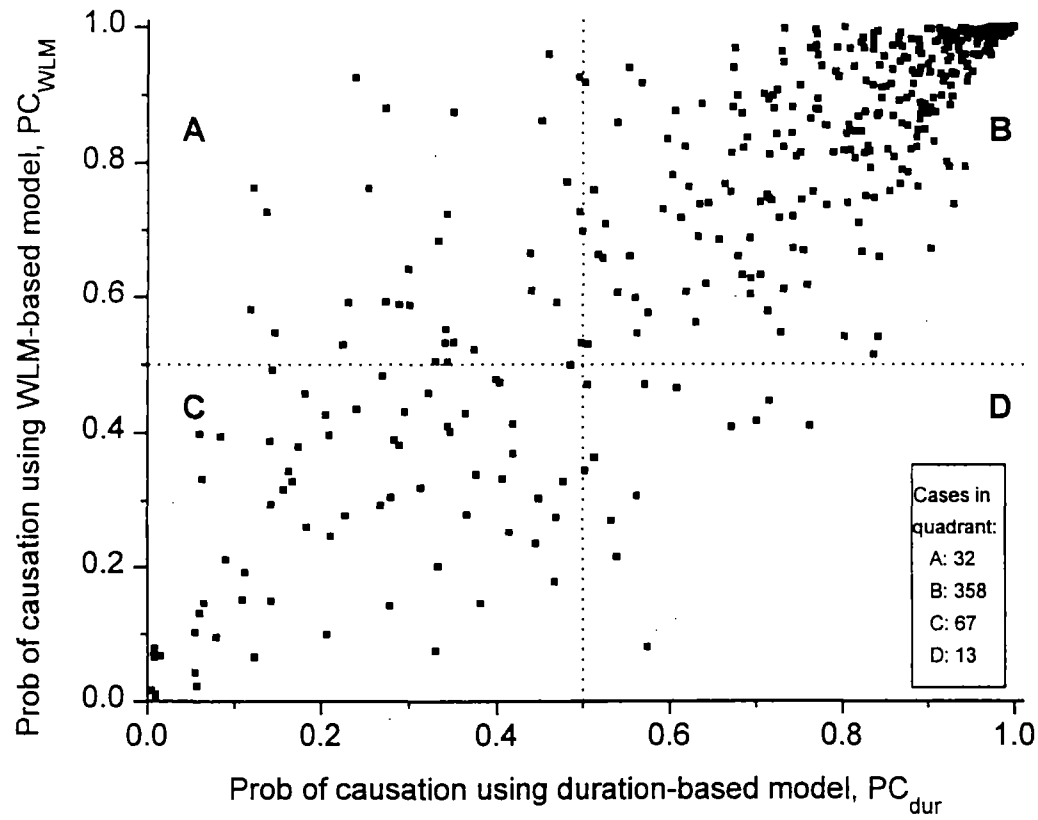


Figure 5: Comparison\$ of PC_{WLM} and PC_{dur}



TABLES

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Table 1: Parameter estimates from lung cancer risk model^a in cumulative WLM.

Parameters	Models			
	A	B	C	E
β	0.219	0.07	0.114	0.078
κ	0.845		0.907	0.781
Time since last exposure ^b				
$\phi_{<10}$	1.00	1.00		1.00
ϕ_{10-19}	0.53	0.52		0.48
ϕ_{20+}	0.16	0.15		0.14
Attained age ^c				
$\theta_{<50}$	1.00	1.00	1.00	
θ_{50-59}	0.34	0.256	0.24	
θ_{60-69}	0.10	0.07	0.04	
$\theta_{\geq 70}$	0.07	0.06	0.02	
Change in deviance ^d	- ^e	4.2	51.2	14.8

^a Fitted risk model, $RR = 1 + \beta \times w^* \times \phi_{tsle} \times \theta_{age}$, where w is WLM and $tsle$ is time since last exposure.

^b Data include 177, 172, and 121 lung cancer cases in each category, respectively.

^c Data included 83, 173, 133, and 81 lung cancer cases in each category, respectively.

^d Change in deviance tests difference in model fit compared with model in column A. Value is asymptotically chi-squared with degrees of freedom equal to the number of omitted parameters.

^e Deviance of model was 2,509.9 on 17,218 degrees of freedom.

Table 2: Parameter estimates from lung cancer risk model^a in duration of exposure and year of first exposure.

Parameters	Models					
	A	B	C	D	E	F
β	19.83	2.563	5.261	1.276	0.930	0.326
κ	1.171	1.259		1.511	1.081	1.686
Year of first exposure ^b						
<1955	1.00	1.00	1.00		1.00	1.00
1955-59	0.75	0.73	0.66		0.72	0.90
≥ 1960	0.24	0.19	0.17		0.14	0.20
Time since last exposure						
$\phi_{<10}$	1.00	1.00	1.00	1.00		
ϕ_{10-19}	0.61	0.66	0.60	0.77	0.57	
ϕ_{20+}	0.26	0.30	0.25	0.39	0.23	
Attained age group						
$\theta_{<50}$	1.00	1.00	1.00	1.00		1.00
θ_{50-59}	0.09	0.30	0.35	0.22		0.36
θ_{60-69}	0.01	0.10	0.13	0.08		0.14
$\theta_{\geq 70}$	0.003	0.06	0.09	0.04		0.04
Attained age - 60						
τ	1.09					
Change in deviance ^c	- ^d	12.2	14.9	26.4	26.3	27.8

^a Fitted risk model, $RR = 1 + \beta \times d^{\kappa} \times \gamma_{yfe} \times \phi_{tsle} \times \theta_{age} \times \tau^{(age-60)}$, where d is duration of exposure, yfe is calendar year of first exposure, and tsle is time since last exposure.

^b Data include 310, 141, and 19 lung cancer cases in each category.

^c Change in deviance tests difference in model fit compared with model in column A. Value is asymptotically chi-squared with degrees of freedom equal to the number of omitted parameters.

^d Deviance of model was 2,557.7 on 17,215 degrees of freedom.

Table 3a: WLM for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<50	8	17	68	2	3	14
50-59	28	60	243	6	12	49
60-69	121	259	1047	24	52	211
≥70	174	376	1,503	35	75	303

^a Model used in the model to compute PC=0.5, $RR = 1 + \beta \times w^* \times \phi_{isle} \times \theta_{age}$ with time since last exposure and age defined by categories. For calculations, β in Table 1 is replaced by β_{smk} or β_{ns} as described in the text.

Table 3b: WLM for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5. Estimated relative risks are increased to incorporate statistical uncertainty at the 80 percent level.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<50	6	12	50	1	3	10
50-59	21	44	178	4	9	35
60-69	89	190	767	18	38	154
≥70	127	273	1,102	26	55	222

^a Model used in the model to compute PC=0.5, $RR = 1 + \beta \times w^* \times \phi_{isle} \times \theta_{age}$ with time since last exposure and age defined by categories. For calculations, β in Table 1 is replaced by $\beta \times \exp[1.28 \times SE(\beta)]$ then adjusted to β_{smk} or β_{ns} as described in the text.

Table 3c: WLM for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5. Estimated relative risks are increased to incorporate statistical uncertainty at the 90 percent level.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<50	5	11	46	1	2	9
50-59	19	40	163	4	8	33
60-69	81	174	703	16	35	141
≥70	117	250	1,010	24	50	203
^a Model used in the model to compute PC=0.5, $RR = 1 + \beta \times w^* \times \phi_{t_{sic}} \times \theta_{age}$ with time since last exposure and age defined by categories. For calculations, β in Table 1 is replaced by $\beta \times \exp[1.64 \times SE(\beta)]$ then adjusted to β_{smk} or β_{ns} as described in the text.						

Table 4a: Years of exposure by year first exposed for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<i>First exposed: <1955</i>						
<50	0.0 ^b	0.1	0.1	0.0	0.0	0.0
50-59	0.4	0.7	1.4	0.2	0.3	0.6
60-69	2.6	3.9	8.2	1.1	1.7	3.6
≥70	4.1	6.3	13.2	1.8	2.7	5.7
<i>First exposed: 1955-59</i>						
<50	0.0	0.1	0.1	0.0	0.0	0.1
50-59	0.6	0.8	1.8	0.2	0.4	0.8
60-69	3.3	5.0	10.5	1.4	2.2	4.5
≥70	5.2	8.0	16.8	2.3	3.5	7.3
<i>First exposed: ≥1960</i>						
<50	0.1	0.2	0.3	0.0	0.1	0.1
50-59	1.5	2.2	4.7	0.6	1.0	2.0
60-69	8.7	13.3	27.8	3.8	5.8	12.0
≥70	14.0	21.4	44.7	6.0	9.2	19.3

^a Model used in the model to compute $PC=0.5$, $RR = 1 + \beta \times d^x \times \gamma_{yfe} \times \phi_{use} \times \theta_{age} \times \tau^{(age-60)}$ with time since last exposure, and year first exposed defined by categories. For calculations, β in Table 2 is replaced by β_{smk} or β_{ns} as described in the text. For computing RR with age categories, mean ages 44.5, 54.8, 63.9, and 75.7 used.

^b A value of 0.0 years exposed denotes exposure to radon progeny which is less than 0.05 years (or 0.6 mos = 0.05×12 , or 102 working hrs = 170×0.6)

Table 4b: Years of exposure by year first exposed for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5. Estimated relative risks are increased to incorporate statistical uncertainty at the 80 percent level.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<i>First exposed: <1955</i>						
<50	0.0 ^b	0.00	0.1	0.0	0.0	0.0
50-59	0.2	0.3	0.7	0.1	0.2	0.3
60-69	1.3	2.0	4.2	0.6	0.9	1.8
≥70	2.1	3.2	6.7	0.9	1.4	2.9
<i>First exposed: 1955-59</i>						
<50	0.0	0.0	0.1	0.0	0.0	0.0
50-59	0.3	0.4	0.9	0.1	0.2	0.4
60-69	1.7	2.6	5.3	0.7	1.1	2.3
≥70	2.7	4.1	8.6	1.2	1.8	3.7
<i>First exposed: ≥1960</i>						
<50	0.1	0.1	0.2	0.0	0.0	0.1
50-59	0.8	1.1	2.4	0.3	0.5	1.0
60-69	4.4	6.8	14.2	1.9	2.9	6.1
≥70	7.1	10.9	22.8	3.1	4.7	9.9

^a Model used in the model to compute PC=0.5, $RR = 1 + \beta \times d^* \times \gamma_{yfe} \times \phi_{isle} \times \theta_{age} \times \tau^{(age - 60)}$ with time since last exposure, and year first exposed defined by categories. For calculations, β in Table 2 is replaced by $\beta \times \exp[1.28 \times SE(\beta)]$ then adjusted to β_{smk} or β_{ns} as described in the text. For computing RR with age categories, mean ages 44.5, 54.8, 63.9, and 75.7 used.

^b A value of 0.0 years exposed denotes exposure to radon progeny which is less than 0.05 years (or 0.6 mos = 0.05×12, or 102 working hrs = 170×0.6)

Table 4c: Years of exposure by year first exposed for which a lung cancer case is "more likely than not" due to radon progeny exposure^a, based on probability of causation exceeding 0.5. Estimated relative risks are increased to incorporate statistical uncertainty at the 90 percent level.

Age at disease	Years since last radon progeny exposure					
	Smokers			Non-smokers		
	<10	10-19	≥20	<10	10-19	≥20
<i>First exposed: <1955</i>						
<50	0.0 ^b	0.00	0.0	0.0	0.0	0.0
50-59	0.2	0.3	0.6	0.1	0.2	0.3
60-69	1.1	1.6	3.4	0.5	0.7	1.5
≥70	1.7	2.6	5.5	0.7	1.1	2.4
<i>First exposed: 1955-59</i>						
<50	0.0	0.0	0.1	0.0	0.0	0.0
50-59	0.2	0.4	0.7	0.1	0.2	0.3
60-69	1.4	2.1	4.3	0.6	0.9	1.9
≥70	2.2	3.3	7.0	0.9	1.4	3.0
<i>First exposed: ≥1960</i>						
<50	0.0	0.1	0.1	0.0	0.0	0.1
50-59	0.6	0.9	1.9	0.3	0.4	0.8
60-69	3.6	5.5	11.5	1.6	2.4	5.0
≥70	5.8	8.8	18.5	2.5	3.8	8.0

^a Model used in the model to compute PC=0.5, $RR = 1 + \beta \times d^* \times \gamma_{yfe} \times \phi_{tisle} \times \theta_{age} \times \tau^{(age - 60)}$ with time since last exposure, and year first exposed defined by categories. For calculations, β in Table 2 is replaced by $\beta \times \exp[1.64 \times SE(\beta)]$ then adjusted to β_{smk} or β_{ns} as described in the text. For computing RR with age categories, mean ages 44.5, 54.8, 63.9, and 75.7 used.

^b A value of 0.0 years exposed denotes exposure to radon progeny which is less than 0.05 years (or 0.6 mos = 0.05×12 , or 102 working hrs = 170×0.6)

Table 5a: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Cases from pooled Colorado and NM studies.

Age at disease	Years since last exposure			Total
	<10	10-19	≥20	
<50	57/57	19/19	6/7	82/83
50-59	71/71	62/64	32/38	165/173
60-69	38/42	47/54	13/37	98/133
≥70	7/7	27/35	9/39	43/81
Total	173/177	155/172	60/121	388/470

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Table 5b: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Estimated relative risks are increased to incorporate statistical uncertainty at the 80 percent level. Cases from pooled Colorado and NM studies.

Age at disease	Years since last exposure			Total
	<10	10-19	≥20	
<50	57/57	19/19	6/7	82/83
50-59	71/71	62/64	33/38	166/173
60-69	39/42	49/54	17/37	105/133
≥70	7/7	29/35	12/39	48/81
Total	174/177	159/172	68/121	401/470

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Table 5c: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Estimated relative risks are increased to incorporate statistical uncertainty at the 90 percent level. Cases from pooled Colorado and NM studies.

Age at disease	Years since last exposure			Total
	<10	10-19	≥20	
<50	57/57	19/19	6/7	82/83
50-59	71/71	62/64	33/38	166/173
60-69	39/42	51/54	17/37	107/133
≥70	7/7	29/35	12/39	48/81
Total	174/177	161/172	68/121	403/470

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Table 6a: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Cases from pooled Colorado and New Mexico studies.

Age at disease	Years since last exposure			
	<10	10-19	≥20	Total
<i>First exposed: <1955</i>				
<50	39/39	9/9	4/4	52/52
50-59	52/54	38/40	23/29	113/123
60-69	21/23	32/34	12/24	65/81
≥70	3/7	13/22	0/25	16/54
Total	115/123	92/105	39/82	
<i>First exposed: 1955-59</i>				
<50	14/14	8/8	2/2	24/24
50-59	14/14	22/23	4/9	40/46
60-69	13/17	11/18	1/11	25/46
≥70	0/0	3/12	0/13	3/25
Total	41/45	44/61	7/35	
<i>First exposed: ≥1960</i>				
<50	4/4	2/2	1/1	7/7
50-59	3/3	1/1	0/0	4/4
60-69	0/2	0/2	0/2	0/6
≥70	0/0	0/1	0/1	0/2
Total	7/9	3/6	1/4	
Total-all	163/177	139/172	47/121	349/470

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Table 6b: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Estimated relative risks are increased to incorporate statistical uncertainty at the 80 percent level. Cases from pooled Colorado and New Mexico studies.

Age at disease	Years since last exposure			Total
	<10	10-19	≥20	
<i>First exposed: <1955</i>				
<50	39/39	9/9	4/4	52/52
50-59	54/54	38/40	26/29	118/123
60-69	23/23	33/34	16/24	72/81
≥70	4/7	19/22	9/25	32/54
Total	120/123	99/105	55/82	
<i>First exposed: 1955-59</i>				
<50	14/14	8/8	2/2	24/24
50-59	14/14	22/23	6/9	42/46
60-69	15/17	13/18	5/11	33/46
≥70	0/0	8/12	2/13	10/25
Total	43/45	51/61	15/35	
<i>First exposed: ≥1960</i>				
<50	4/4	2/2	1/1	7/7
50-59	3/3	1/1	0/0	4/4
60-69	0/2	0/2	0/2	0/6
≥70	0/0	0/1	0/1	0/2
Total	7/9	3/6	1/4	
Total-all	170/177	153/172	71/121	394/470

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Table 6c: Lung cancer cases which are "more likely than not" due to radon progeny exposure and total cases by age and years since last radon exposure. Estimated relative risks are increased to incorporate statistical uncertainty at the 90 percent level. Cases from pooled Colorado and New Mexico studies.

Age at disease	Years since last exposure			
	<10	10-19	≥20	Total
<i>First exposed: <1955</i>				
<50	39/39	9/9	4/4	52/52
50-59	54/54	38/40	26/29	118/123
60-69	23/23	34/34	16/24	73/81
≥70	5/7	19/22	14/25	38/54
Total	121/123	100/105	60/82	
<i>First exposed: 1955-59</i>				
<50	14/14	8/8	2/2	24/24
50-59	14/14	22/23	8/9	44/46
60-69	16/17	15/18	6/11	37/46
≥70	0/0	9/12	2/13	11/25
Total	44/45	54/61	18/35	
<i>First exposed: ≥1960</i>				
<50	4/4	2/2	1/1	7/7
50-59	3/3	1/1	0/0	4/4
60-69	0/2	0/2	0/2	0/6
≥70	0/0	0/1	0/1	0/2
Total	7/9	3/6	1/4	
Total-all	172/177	157/172	79/121	408/470

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APPENDIX B

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concerns about the adequacy and operation of the current laws, including, specifically, record-keeping practices. Although the Committee did not have the time or resources to pursue these concerns to the degree they merit, we believe that the concerns expressed by veterans and their family members deserve attention, and we urge the Human Radiation Interagency Working Group in conjunction with Congress to address these concerns promptly. The concerns reported to us include the following:

1. The listing of diseases for which relief is automatically provided--the "presumptive" diseases provided for in the 1988 law--is incomplete and inadequate.
2. The standard of proof for those without a presumptive disease is impossible to meet and, given the questionable condition of the exposure records retained by the government, inappropriate.
3. The statutes are limited and inequitable in their coverage; for example, the inclusion of those exposed at atmospheric tests does not protect those who were exposed to equal amounts of radiation in activities such as cleanup at Enewetak atoll.
4. The time and expense needed to prosecute a claim is too great. For example, veterans whose claims are initially denied at the VA regional offices and are seeking appeal of the initial decision receive a form letter stating that it will take at least twenty-four months to process their appeal.
5. Time and money spent on contractors and consultants in administering the program would be better spent on directly aiding veterans and their survivors.

Recommendation 7

The Advisory Committee recommends to the Human Radiation Interagency Working Group that it, together with Congress, give serious consideration to amending the provisions of the Radiation Exposure Compensation Act of 1990 relating to uranium miners in order to provide compensation to *all* miners who develop lung cancer after some minimal duration of employment underground (such as one year), without requiring a specific level of exposure. The act should also be reviewed to determine whether the documentation standards for compensation should be liberalized.

The uranium miners were exposed to extremely high levels of radon daughters, which were recognized at the time to be hazardous yet were not controlled by the government, despite the availability of feasible means to

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ventilate the mines. Furthermore, the government studied the miners without disclosing the purposes of the examinations or warning them of the hazards to which they were exposed. As a result of their continued exposure, hundreds of miners developed lung cancer or nonmalignant respiratory diseases that could have been prevented, and many of them have died.

In recognition of this tragedy, Congress included provisions for compensating certain uranium miners in the Radiation Exposure Compensation Act of 1990 (RECA). However, the criteria for compensation set in this act were far more stringent than for the two other groups (atomic veterans and downwinders of the Nevada Test Site) for which compensation was provided, despite the fact that the risks were far higher for the uranium miners.

Since 1990, additional scientific information has become available to support the view that radon exposure is responsible for a much higher proportion of the lung cancer cases among the miners than had been previously thought. In particular, the act's current requirement of a minimum of 200 WLM (working level months) exposure for nonsmokers or 300 to 500 WLM (depending on age) for smokers translates to quite large probabilities of causation, according to a recent report by the National Cancer Institute.⁵ That analysis finds little evidence to support a distinction between smokers and nonsmokers and suggests that a majority of lung cancer deaths among Colorado white miners and New Mexico Navajo miners are attributable to radon exposure. Furthermore, it finds that the lung cancer risk is strongly modified by a number of factors and uncertainties that are not accounted for in the total dose: thus, for many miners, the level of exposure that would merit compensation on the basis of the principle of "balance of probabilities" might be far lower than the present criteria. In particular, no exposure measurements are available for 90 percent of the years in most mines, so that any requirement to reconstruct exposure histories is likely to require some degree of extrapolation or estimation and be quite uncertain. Furthermore, many mines have since gone out of business, so that records needed to establish an exposure history are simply unavailable.

Also since 1990, there has been considerable experience with the administration of the act, and apparently much of it has been negative. The Advisory Committee took extensive testimony regarding the difficulties faced by miners in meeting the documentation requirements, particularly those related to the requirement to provide a reconstruction of their radon dose. For these practical reasons, and in light of the additional information, we suggest that the requirement that a miner demonstrate that he had been exposed to a certain minimum cumulative dose be replaced by a simple requirement that he worked underground for a certain minimum length of time. Since more than half the lung cancer deaths in the cohort who worked at least one month underground appear to be attributable to radon, we suggest that minimum length of service be set quite low, preferably not more than a year. At most this should then lead to

compensation being awarded to twice as many miners as would be entitled to it under the balance of probabilities principle, while not denying it to any who are entitled to it.

The grave injustice that the government did to the uranium miners, by failing to take action to control the hazard and by failing to warn the miners of the hazard, should not be compounded by unreasonable barriers to receiving the compensation the miners deserve for the wrongs and harms inflicted upon them as they served their country.

Recommendation 8

The Advisory Committee supports the Department of Energy's program of medical monitoring and treatment for the exposed inhabitants of the Marshall Islands atolls of Rongelap and Utirik and recommends that this program be continued as long as any member of the exposed population remains alive. Furthermore, the Advisory Committee recommends that the program be reviewed to determine if it is appropriate to add to the program the populations of other atolls to the south and east of the blast whose inhabitants may have received exposures sufficient to cause excess thyroid abnormalities. The Advisory Committee also recommends that consideration be given to the involvement of the Marshall Islanders in the design of any further medical research to be conducted upon them and the Advisory Committee recommends that the Human Radiation Interagency Working Group consider establishing an independent panel to review the status and adequacy of the current program of medical monitoring and medical care provided by the United States to the exposed population of the Marshall Islands.

The 1954 Bravo hydrogen bomb test caused the populations of several Marshall Islands atolls to be exposed to hazardous levels of radiation. The United States has provided a medical follow-up program that combines research on radiation effects with treatment for radiation-related illnesses. It is noteworthy that as a result of the ongoing program to study radiation effects, many cases of thyroid disease were detected and treated, but not all exposed Marshallese received the benefits of the program. The people of Ailuk, for example, who according to early reports received about the same exposure as the people of Utirik, were never evacuated from their atoll and were not followed up medically, even though they received a radiation dose of more than six roentgens. Moreover, an epidemiological study reported in the *Journal of the American Medical Association* in 1987 demonstrated that inhabitants of several atolls to the east and south of Bikini had elevated levels of thyroid disease and that there was a "strong inverse linear relationship" between incidence of thyroid nodules and