

SAB Working Review Draft Advisory dated November 15, 2006 for Radiation Advisory Committee Edits – Do Not Cite or Quote. This review draft is a work in progress, does not reflect consensus advice or recommendations, has not been reviewed or approved by the Science Advisory Board’s Charter Board, and does not represent EPA policy.



**UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460**

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

--- Working Review Draft ---

EPA-SAB-RAC-ADV-07-xxx

The Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460

Subject: Advisory on Agency Draft White Paper entitled “*Modifying Radiation Risk Models Based on BEIR VII,*”

Dear Administrator Johnson:

--- Letter to be Completed ---

NOTICE

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3 This advisory has been written as part of the activities of the EPA Science Advisory
4 Board (SAB), a public advisory group providing extramural scientific information and advice to
5 the Administrator and other officials of the Environmental Protection Agency. The SAB is
6 structured to provide balanced, expert assessment of scientific matters related to problems facing
7 the Agency. This advisory has not been reviewed for approval by the Agency and, hence, the
8 contents of this advisory do not necessarily represent the views and policies of the
9 Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal
10 government, nor does mention of trade names of commercial products constitute a
11 recommendation for use. Reports and advisories of the SAB are posted on the EPA website at
12 <http://www.epa.gov/sab>.

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3 **Science Advisory Board (SAB)**
4 **Radiation Advisory Committee (RAC)**
5

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**U.S. Environmental Protection Agency
Science Advisory Board**

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--- (Roster to be Inserted in Later Drafts) ---

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1. EXECUTIVE SUMMARY

--- (To be prepared in a Later Draft, once consensus language is agreed upon --- KJK) ---

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2. INTRODUCTION

2.1 Background

In 1994, the EPA published a report, referred to as the “Blue Book,” which lays out the EPA’s methodology for quantitatively estimating radiogenic cancer risks (U.S. EPA. 1994) <http://epa.gov/radiation/docs/assessment/402-r-93-076.pdf>. A follow-on report made minor adjustments to the previous estimates and presented a partial analysis of the uncertainties in the numerical estimates (U.S. EPA. 1999a) <http://epa.gov/radiation/docs/assessment/402-r-99-003.pdf>. Finally, the Agency published Federal Guidance Report 13 (U.S. EPA. 1999) <http://epa.gov/radiation/docs/federal/402-r-99-001.pdf> which utilized the previously published cancer risk models, in conjunction with International Commission on Radiological Protection (ICRP) dosimetric models and U.S. usage patterns, to obtain cancer risk estimates for over 800 radionuclides, and for several exposure pathways. These were later updated (U.S. EPA. 1999b) <http://epa.gov/radiation/federal/techdocs.htm#report13>.

The National Research Council recently released *Health Risks from Exposure to Low levels of Ionizing Radiation BEIR VII Phase 2* which primarily addresses cancer and genetic risks from low doses of low LET radiation (BEIR VII) (U.S. NAS/NRC. 2006) <http://newton.nap.edu/catalog/11340.html#toc>). In the EPA draft *White Paper: Modifying EPA Radiation Risk Models Based on BEIR VII*, the Agency proposes changes to the EPA’s methodology for estimating radiogenic cancers, based on the contents of BEIR VII (U.S. EPA. 2006a). The Agency expects to adopt the models and methodology recommended in BEIR VII, but believes that certain modifications and expansions are desirable or necessary for the EPA’s purposes.

2.1.1 Request for EPA Science Advisory Board (SAB) Review

The Radiation Advisory Committee (RAC) was initially briefed on the draft White Paper topic at its public planning meeting of December 21, 2005 which was held at the National Air and Environmental Radiation Laboratory (NAERL) in Montgomery, Alabama (see 70 Fed. Reg. 69550, November 16, 2005). ORIA issued its external draft White Paper entitled “*Modifying EPA Radiation Risk Models Based on BEIR VII*,” on August 1, 2006 (U.S. EPA. 2006a). The charge questions to the SAB were formally submitted on August 31, 2006 (U.S. EPA. 2006b).

The SAB RAC met in a public teleconference meeting on September 6, 2006 and conducted a face-to-face public meeting on September 26, 27 and 28, 2006 for this advisory (see 71 Fed. Reg. 45545, August 9, 2006). Additional public conference calls took place on November 28, 2006 and December 18, 2006 (see 71 Fed. Reg., 62590, October 26, 2006 and add additional meetings as appropriate - - - KJK). These notices, the charge to the RAC and other supplemental information may be found at the SAB’s Web site (<http://www.sab.gov/sab>).

2.2 Proposed EPA Adjustments and Extensions to BEIR VII Models

2.2.1 Current EPA Cancer Risk Models

For most cancer sites, radiation risk models are derived primarily from epidemiologic data from the Life Span Study (LSS) of the atomic bomb survivors. The EPA’s models for esophageal, stomach, colon, lung, ovarian, bladder, leukemia, and “residual” cancers were adapted from the models published by Land and Sinclair based on a fit to the linear, no-threshold fit to the LSS data (Land and Sinclair. 1991).

For each solid tumor site, gender, and age-at-exposure interval, there is a model providing a coefficient for the excess relative risk (ERR) per gray (Gy) for cancer mortality, which is assumed to be constant beginning at the end of a minimum latency period until the end of life. Land and Sinclair present two sets of models known as the “multiplicative” and the “National Institutes of Health (NIH)” models that differ in how one “transports” risk from the Japanese LSS population to the United States population. In the multiplicative model, it is assumed that the ERR/Gy is the same in all populations, whereas, in the NIH model, it is assumed that the excess absolute risk (EAR) is the same in different populations for the limited period of epidemiological follow-up. Given the scarcity of information on how radiogenic cancer risk varies between populations having differing baseline cancer rates, the EPA previously adopted an intermediate geometric mean coefficient “GMC” model for each site, where the ERR coefficients were taken to be the geometric mean of the corresponding ERR coefficients for both the multiplicative and the NIH models (U.S. EPA. 1994).

For leukemia, the treatment of the temporal response in the models was more complex, but the approach for transporting risk to the U.S. population was analogous. Following the approach of Land and Sinclair, the EPA also developed a GMC model for kidney cancer from the LSS data. The EPA’s models for other sites, including breast, liver, thyroid, bone, and skin were based on various authoritative reports (NCRP. 1980; NRC. 1988; ICRP. 1991a, b; Gilbert. 1991). Based primarily on ICRP recommendations at that time, for low doses and dose rates, each coefficient was reduced by a factor (DDREF) of two from that which would be obtained from a Linear Non Threshold (LNT) fit to the LSS data.

2.2.2 BEIR VII Models

BEIR VII cancer site-specific models derived from the LSS differ from those of Land and Sinclair in several notable ways: (1) they are derived primarily from data on cancer incidence rather than cancer mortality; (2) mathematical fitting is performed to better reflect the functional dependence of solid cancer risk on age at exposure and attained age, (i.e., age at diagnosis of a cancer or age at death due to cancer depending on the end-point of interest); (3) a weighted average of risk projection models is used to transport risk from the LSS to the U.S. population; (4) a value for the DDREF of 1.5 is estimated from the LSS and laboratory data; (5) quantitative uncertainty bounds are provided for the site-specific risk estimates in BEIR VII.

1 For breast cancer and thyroid cancer, BEIR VII risk models are based on pooled analyses
2 of data from the LSS cohort, together with data from epidemiologic studies of medically
3 irradiated cohorts (Preston et al. 2002; Ron et al. 1995).

4 5 **2.2.3 Proposed EPA adjustments and Extensions to BEIR VII Models**

6
7 In the draft *White Paper: Modifying EPA Radiation Risk Models Based on BEIR VII*
8 (U.S. EPA. 2006a), the Agency’s Office of Radiation and Indoor Air (ORIA) outlined proposed
9 changes in the EPA’s methodology for estimating radiogenic cancers, based on the contents of
10 BEIR VII and some ancillary information. For the most part, the Agency expects to adopt the
11 models and methodology recommended in BEIR VII; however, the Agency believes that certain
12 modifications and expansions are desirable or necessary for the EPA’s purposes.

13
14 One significant extension to be considered is the estimation of risks from exposures to
15 higher LET radiations, especially to alpha particles, but also to lower energy photons and beta
16 particles. An important expansion proposed by EPA to be considered is the estimation of risks
17 from exposures to alpha particles, and also to alpha emitters deposited in the lung and the bone.
18 BEIR VII does not present any risk estimates for radiogenic bone cancer. The EPA proposes to
19 estimate bone cancer risk from data on radium injected patients.

20
21 BEIR VII does not provide quantitative estimates of risk for skin cancer. It does not fully
22 address prenatal exposures. BEIR VII presents a model for estimating the risk of the radiogenic
23 thyroid cancer incidence, but not of mortality due to radiogenic thyroid cancer.

24
25 The EPA proposes to use somewhat different population statistics from BEIR VII.
26 Consideration is given to an alternative model for estimating radiogenic lung cancer. For breast
27 cancer, the EPA proposes an alternative method for estimating mortality, which takes into
28 account changes in incidence rates and survival rates over time.

29
30 At this point in its activity on this topic, the EPA is seeking advice from the Agency’s
31 Science Advisory Board’s Radiation Advisory Committee (RAC) on the application of BEIR
32 VII’s cancer risk estimates and on issues relating to these modifications and expansions. After
33 receiving the advisory review, the Agency plans to implement changes in their methodology
34 through the publication of a revised Blue Book, which it would expect to submit to the SAB’s
35 RAC or a specialty panel supplementing the RAC for final review. The revised Blue Book could
36 then serve as a basis for an updated version of FGR-13.

37 38 **2.2.4 Uncertainty Estimates**

39
40 BEIR VII provides quantitative uncertainty bounds for each of its risk coefficients,
41 however, no uncertainty was assigned to the form of the dose-response relationship. It was
42 implicitly assumed that the dose-response relationship is “linear-quadratic”, which allowed the
43 BEIR VII Committee to place uncertainty on bounds of the dose and dose-rate effectiveness
44 factor (DDREF). Mechanisms pertaining to the biological effects of low-level ionizing radiation
45 are being investigated, which could eventually mandate a different dose-response model,
46 potentially resulting in large changes in estimates of risk at low doses. Assigning probabilities to

1 alternative models would be highly subjective at this time. The EPA does not propose to
2 quantify the uncertainty pertaining to low-dose extrapolation, but it would provide a brief
3 discussion of the issue.

4 5 **2.2.5 Level of Review**

6
7 There are various levels of reviews which EPA can request from the SAB. These include
8 reviews, advisories, and commentaries. The request from EPA-ORIA was for an “advisory”
9 review of the draft White Paper. ORIA was interested in vetting ideas with a group of scientific
10 experts on how to incorporate the changes in cancer risk models described by BEIR VII and to
11 extend the BEIR VII models to areas not specifically addressed by the BEIR VII committee.
12 ORIA described it as kind of a “mid-course correction” which would allow the RAC to provide
13 advice on a series of questions which would guide the agency in incorporating the latest
14 scientific thinking into their risk estimates. The RAC was not asked to provide policy direction,
15 therefore the RAC did not consider the implications to EPA standards which may be an outcome
16 of the changes to the risk estimates. The RAC only considered the scientific evidence which
17 support the risk models for radiogenic cancer.

18 19 **2.2.6 Specific Charge to the Committee**

20
21
22 *1) BEIR VII provides incidence models for many cancer sites as a basis for calculating the*
23 *risk from low-dose, low-LET radiation. Please comment on EPA’s application of this overall*
24 *approach as described in the draft White Paper.*

25
26 *2) In addition to the overall approach described in BEIR VII, the draft White Paper presents*
27 *specific modifications and extensions. Please comment on the soundness of the following*
28 *proposals:*

29
30 *a. Calculation of the risk to the life table (stationary) population instead of the actual*
31 *U.S. population (see Sections II.A.-C.); this is consistent with our current approach.*

32
33 *b. Use of more recent incidence and mortality data from SEER and/or other sources*
34 *(see Section II.D.); BEIR VII used a previous version of SEER data for the years*
35 *1995-1999.*

36
37 *c. Method for combining BEIR VII’s models for projecting risk from Japanese A-bomb*
38 *survivors to U.S. population (see Section II.E.). In contrast to BEIR VII, we propose*
39 *to combine the two risk models before integration to calculate the lifetime*
40 *attributable risk.*

41
42 *d. Adoption of an alternative model for radiogenic lung cancer risk which may better*
43 *account for the effects of smoking than the BEIR VII approach (see Section II.G.).*

44
45 *e. Method for calculating breast cancer mortality risk, accounting for the relatively long*
46 *time from detection until death (see Section II.H.).*

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f. *Proposed approaches for extending risk estimates to radiations of different LET's - in particular, deriving site-specific risk estimates for alpha or x radiations based on models derived from the A-bomb survivors, who were primarily exposed to gamma rays (see Section III).*

g. *Estimation of risks for sites not specified in BEIR VII, specifically bone and skin, for which we propose to update our current approaches (see Sections III.A. and V, respectively).*

h. *Estimation of risk due to prenatal exposure. EPA's current lifetime risk estimates do not include risk from prenatal exposure, and BEIR VII does not provide them. The draft White Paper uses ICRP recommendations to project its risks of childhood cancers induced by in utero exposure. Please comment on the soundness of the approach described in the draft White Paper to apply ICRP as described in Section IV.*

3) *BEIR VII provides quantitative uncertainty bounds for each of its risk coefficients. EPA proposes to adopt this methodology with some additional discussion of the uncertainties not quantified in BEIR VII. Please comment on the adequacy of this approach (see Section II.K.).*

4) *In Section VI, the draft White Paper discusses some issues relating to radiogenic thyroid cancer. Does the RAC have any specific suggestions for dealing with this risk; e.g., does the RAC have any advice on gender specificity, effectiveness of iodine -131 compared to gamma rays, or estimation of thyroid cancer mortality?*

3. PHILOSOPHY OF APPROACH TO THE CHARGE

The SAB’s RAC wishes to stress the four following points as it addresss this topic:

- 1) the RAC accepts the use of LNT for standard setting for radiation protection purposes;
- 2) the RAC considers it is important that there be an open discussion of the limits of usefulness of the LNT as a function of dose. It is important to understand and recognize that at low doses, (i.e. those out of the range of observed increased cancer rates in epidemiological data, primarily below 0.2 Gy,) cancer risk estimates are derived by extrapolation from “real” data in the high dose range (primarily 0.5 – 4.0 Gy) to the low dose range based primarily on the model. Because of this, the EPA should add a cautionary note regarding using LNT in both high and low dose ranges;
- 3) the Agency needs to recognize and acknowledge that there are many experimental data (i.e. cellular or animal data, not epidemiologic/ human data) that suggest although the dose-response relationship for the initial interaction between radiation and cellular DNA damage is linear, the consequences (biological effects) of the resultant DNA damage is non-linear. There is a strong and growing data base (Need Reference - - -KJK) to support observations of non-linear dose-response relationships at low radiation doses; this should be recognized and discussed by the EPA; and
- 4) the RAC is concerned that issues be addressed regarding the influence of RBE and DDREF at high doses delivered at low dose rates (See Section 3.1, below).

3.1 Draft Discussion Relating to Specific Request

One of the major problems in making risk estimates following exposure to high total doses delivered at a low dose-rate, is distinguishing between the contribution of the RBE of the radiation involved and the DDREF of the radiation over high to the low total dose range. RBE is applied to the dose, and is a method of normalizing the results of exposure to a specific type of radiation to a single reference radiation, which could be gamma or x-rays. The value of the RBE provides a quantitative index of the effectiveness per unit of absorbed dose of any radiation. The DDREF is the ratio of the slope of the dose-response curve obtained for high dose, high dose-rate exposures to that obtained for low dose, low-dose rate exposures. The significance of the data in these low dose ranges is in many cases not different from zero. (Need help rewording – are we saying that the uncertainty is so great that the confidence interval includes zero?)

It has been demonstrated in a large number of cellular and animal systems that at doses of 0.2 – 1.0 Gy, there is a statistically significant increase in biological changes indicative of cancer following high dose-rate exposures, however there are no significant increases in the same biological changes following low dose-rate exposures. By increasing the total dose to higher values at the low dose-rate, where there are significant responses (at 1.0 – 1.0 Gy), the difference between the response for high and low dose-rate is much greater than the current

1 DDREF. An example would be to compare the effectiveness of exposure to 10 Gy of low-LET
2 radiation delivered acutely or chronically. An acute dose of 10.0 Gy from an internally
3 deposited radioactive material delivering a low-LET exposure is likely to result in death within a
4 period of weeks. However, the same dose at a low dose-rate over a life time, is likely to result in
5 little life shortening. Similar examples of very different responses as a function of dose rate can
6 be given. Many radiation-induced biological changes are observed that range from the induction
7 of DNA damage to the induction of cancers following exposure to high doses of low-LET
8 radiation delivered at high and low dose-rates. Thus, at high total doses the dose-rate
9 effectiveness factor (DDREF) is very large, which is not reflected in the current DDREF.

10
11 With respect to ORIA’s proposal to assign a value of 20 to the RBE, this is in reference
12 to chronic exposure to both the high and low-LET radiation. For example, when comparing the
13 internally deposited radioactive materials which deliver their dose at low dose rates over the life
14 time of the animals, it has been demonstrated that alpha particles are about 20 times as effective
15 as beta-gamma exposures. The high RBE is related to the very low effectiveness of the
16 protracted beta-gamma exposure in producing biological damage. However, this RBE is often
17 applied to evaluate the effectiveness of alpha particles relative to the a-bomb survivors exposed
18 to high does-rates. Since the biological responses to high dose-rate bata-gamma exposures show
19 very non-linear kinetics and the alpha responses are very linear, the RBE of 20 would not hold at
20 higher total doses.

21
22 The RAC suggests that the EPA/ORIA staff provide a thorough discussion of the RBE
23 and take dose-rate into consideration. The RAC took the position that unless there is compelling
24 evidence which would suggest that a change is necessary, EPA/ORIA should follow BEIR VII’s
25 recommendations or leave the numbers unchanged from their current version. The RAC does
26 not believe that EPA/ORIA presented a compelling case to assign a value of 20 to the RBE.

27 28 **3.2 Acknowledgement**

29
30 The document “*Modifying EPA Radiation Risk Models Based on BEIR VII*,” August 1,
31 2006 was well written and provided much needed background. Similarly, with the BEIR VII
32 report, presentations by the ORIA staff and other information provided to the RAC in the course
33 of the public meetings were found to be helpful. During the meetings, the ORIA staff worked
34 diligently to augment their draft *White Paper* with additional pieces of information that the RAC
35 felt were necessary to assist with the advisory. The staff took care to honor all the RAC’s
36 requests and demonstrated their patience as members sought to understand all that went into the
37 modified procedures being proposed.
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4. RESPONSE TO CHARGE QUESTION 1: APPLICATION OF THE OVERALL APPROACH AS DESCRIBED IN THE DRAFT WHITE PAPER

4.1 Response to Charge Question 1:

BEIR VII provides incidence models for many cancer sites as a basis for calculating the risk from low-dose, low-LET radiation. Please comment on EPA’s application of this overall approach as described in the draft White Paper.

The Radiation Advisory Committee (RAC) agrees with the EPA that the BEIR VII methodologies and data should be used wherever possible. The RAC accepts the EPA’s use of BEIR VII methodologies for deriving risk estimates for cancers of the stomach, colon, liver, prostate, uterus, ovary, bladder, and other solid tumors. However if one of the four following conditions apply, then the RAC agrees that the EPA is correct in deviating from the BEIR VII’s methodologies. The four possible conditions are:

- 1) Information and data are needed about subject matter not addressed in BEIR VII;
- 2) More recent or more relevant data exist which could improve or otherwise influence the risk estimates;
- 3) Compelling evidence suggests the use of a more appropriate scientific method; or
- 4) The EPA’s implementation requirements for practicality or applicability necessitate an adaptation or other alternative to BEIR VII methodologies.

The RAC grouped all of the charge issues according to these conditions. Under condition one, RAC considered prenatal exposures, bone and skin cancers, x- and alpha-particle radiations and tritium as areas not addressed by BEIR VII, and for which the EPA has a need to derive a basis for risk estimates. For condition two, the use of the most recent SEER data would improve the risk estimate. Examples of condition three issues where a more appropriate scientific method was considered were in development of lung and breast cancer risk estimates and the estimation of uncertainty. The most important example of condition four is the use of a stationary or a standard population.

The RAC concludes that the EPA’s use of the gray (Gy) as the unit of radiation absorbed dose is appropriate and agrees that modifying factors should be applied to the risk rather than dose.

The RAC’s approach to giving advice to the EPA is predicated on the basic premise that the risk estimates are for use in assessing population or average individual risk, rather than risk to a specific individual. This is because specific individuals may be more or less susceptible to radiation-induced cancer than the average individual.

1 **5. RESPONSE TO CHARGE QUESTION 2: WHITE PAPER**
2 **MODIFICATIONS AND EXTENSIONS**
3

4 **5.1 Response to Charge Question # 2**

5 *In addition to the overall approach described in BEIR VII, the draft White Paper presents*
6 *specific modifications and extensions. Please comment on the soundness of the following*
7 *proposals:*
8

- 9 a. *Calculation of the risk to the life table (stationary) population instead of the actual U.S.*
10 *population (see Sections II.A.-C.); this is consistent with our current approach.*
11
12 b. *Use of more recent incidence and mortality data from SEER and/or other sources (see*
13 *Section II.D.); BEIR VII used a previous version of SEER data for the years 1995-1999.*
14
15 c. *Method for combining BEIR VII’s models for projecting risk from Japanese A-bomb*
16 *survivors to U.S. population (see Section II.E.). In contrast to BEIR VII, we propose to*
17 *combine the two risk models before integration to calculate the lifetime attributable risk.*
18
19 d. *Adoption of an alternative model for radiogenic lung cancer risk which may better*
20 *account for the effects of smoking than the BEIR VII approach (see Section II.G.).*
21
22 e. *Method for calculating breast cancer mortality risk, accounting for the relatively long*
23 *time from detection until death (see Section II.H.).*
24
25 f. *Proposed approaches for extending risk estimates to radiations of different LET’s - in*
26 *particular, deriving site-specific risk estimates for alpha or x radiations based on models*
27 *derived from the A-bomb survivors, who were primarily exposed to gamma rays (see*
28 *Section III).*
29
30 g. *Estimation of risks for sites not specified in BEIR VII, specifically bone and skin, for*
31 *which we propose to update our current approaches (see Sections III.A. and V,*
32 *respectively).*
33
34 h. *Estimation of risk due to prenatal exposure. EPA’s current lifetime risk estimates do not*
35 *include risk from prenatal exposure, and BEIR VII does not provide them. The draft*
36 *White Paper uses ICRP recommendations to project its risks of childhood cancers*
37 *induced by in utero exposure. Please comment on the soundness of the approach*
38 *described in the draft White Paper to apply ICRP as described in Section IV.*
39

1 **5.2 Response to Charge Question # 2a**

2
3 *Calculation of the risk to the life table (stationary) population instead of the actual U.S.*
4 *population (see Sections II.A.-C.); this is consistent with our current approach.*

5
6 The RAC agrees that the proposed estimation of radiogenic cancer risks for the U.S.
7 population based on a standard stationary population based on the year 2000 deathrate, or fixed
8 cohort is a reasonable adaptation of the BEIR VII approach. Specifically, it avoids the potential
9 for changes over time in the baseline cancer rates among the actual U.S. population that may be
10 associated with changes in its racial, ethnic, cultural or other characteristics known to influence
11 population disease rates. It also is consistent with the EPA’s established approach to cancer risk
12 estimation from exposures to chemicals.
13

14 **5.3 Response to Charge Question #2b**

15
16 *A Use of more recent incidence and mortality data from SEER and/or other sources (see*
17 *Section II.D.); BEIR VII used a previous version of SEER data for the years 1995-1999.*

18
19 The RAC agrees that the EPA’s proposed use of the most current cancer-specific
20 incidence and mortality rates available is an appropriate and scientifically valid adaptation of the
21 BEIR VII approach.
22

23 It is anticipated that these data for the years 1995-2002 will be available for the final
24 calculations of radiogenic cancer incidence risk estimates from NCI’s SEER program. In
25 contrast, only data from this program for 1995-1999 were available to BEIR VII.
26

27 Although other potential sources of valid, nationally representative data will be
28 considered by the EPA, the RAC considers that the most current SEER data are adequate and
29 preferred for consistency with the BEIR VII approach. The EPA may want to consider the latest
30 vital statistics report produced from the 2000 census for mortality rates if they become available
31 before the final report is produced.
32

33 **5.4 Response to Charge Question #2c**

34 *Method for combining BEIR VII’s models for projecting risk from Japanese A-bomb*
35 *survivors to U.S. population (see Section II.E.). In contrast to BEIR VII, we propose to*
36 *combine the two risk models before integration to calculate the lifetime attributable risk.*

37
38 The RAC notes that there is considerable uncertainty in the application of risk estimates
39 developed from the Japanese atomic bomb survivors to the U.S. population. This uncertainty
40 results from different genetic and lifestyle characteristics of the two populations and differences
41 in the baseline cancer risks. The RAC agrees with the EPA’s proposed approach for projecting

1 risk estimates from the Japanese A-bomb survivors to the U.S. population by combining the age-
2 specific results from the EAR and ERR models using the weighted geometric mean before
3 calculating the lifetime attributable risk. This approach is a modification of that used in BEIR
4 VII but is consistent with the method used previously by the EPA in FGR13. The RAC notes that
5 the EPA’s proposed approach has only a small impact on the age-averaged lifetime attributable
6 risk calculations as compared to BEIR VII values, and that the EPA method has the advantage of
7 allowing the risk results from separate exposures to be integrated, enabling the risk from chronic
8 lifetime exposure to be calculated.
9

10 **5.5 Response to Charge Question #2d**

11 *Adoption of an alternative model for radiogenic lung cancer risk which may better account*
12 *for the effects of smoking than the BEIR VII approach (see Section II.G.).*
13

14 The RAC recommends that the EPA accept the use of BEIR VII approach unless the
15 following analyses provide compelling reasons to adopt another approach. The EPA ORIA staff
16 should:

- 17 ● Compare results of the calculation of LAR using BEIR VII weighting to 100% EAR model
18 and to alternative weighting schemes and/or the use of arithmetic, AM, or geometric, GM,
19 means.
20
- 21 ● Consider how the additive ERR model for smoking and radiation provides evidence for the
22 appropriate weighting scheme.
23
- 24 ● Consider papers additional to Pierce (2003) on the nature of the smoking /radiation
25 interaction.
26

27
28 The EPA ORIA staff will need to undertake the following steps in order for the RAC to
29 provide more specific advice:
30

- 31 1) Search literature for other smoking /radiation interaction analyses for lung cancer. (ORIA
32 has not yet provided this to the RAC)
33
- 34 2) Search other literature for RR between women and men for lung cancer due to exposure to
35 low LET radiation. (ORIA has not yet provided this to the RAC.)
36
- 37 3) Do LAR calculations for alternative weighting schemes. (ORIA has provided this.)
38
- 39 4) Examine the tradeoff between AM vs. GM combinations of EAR and ERR and weighting
40 schemes. Determine if there is a justification for using a GM in BEIR VII. (RAC is waiting
41 for the literature review to determine if there is “compelling evidence” to suggest that ORIA
42 make a change.)
43

1 **5.6 Response to Charge Question #2e**

2 *Method for calculating breast cancer mortality risk, accounting for the relatively long time*
3 *from detection until death (see Section II.H.).*
4

5 The RAC notes that the EPA adopts BEIR VII’s approach to estimating the risk of breast
6 cancer in females that differs from that used by BEIR VII to estimate the risks for the majority of
7 other solid cancers. However, the EPA identified issues relative particularly to the changing
8 clinical course of breast cancer in conjunction with a relatively long survival period, and
9 questions some aspects of BEIR VII’s risk estimation method for this site-specific cancer. The
10 EPA thus has identified several alternative methods for estimating the relative risk for radiogenic
11 breast cancer in an effort to take into account some of the temporal features that can influence
12 the cancer’s clinical course and hence the risk estimates. The RAC concurs with the EPA’s
13 decision to explore these alternative methods.
14

15 Specifically, the RAC likes the EPA’s proposal to relate current breast cancer mortality
16 rates to retrospective incidence rates rather than current incidence rates to better reflect the
17 influence of life style changes, earlier breast cancer detection and treatment that could influence
18 survival and hence mortality rates over an extended period.
19

20 The RAC notes the potential of development of second cancers during the cancer survival
21 period. Such an event could be spontaneous or related to treatment of cancer. In the case of
22 breast cancer, it could impact mortality reporting and loss of deaths attributed to breast cancer.
23

24 The RAC suggests that the EPA explore the feasibility of using the BEIR VII with the
25 proposed method (above) with retrospective lagging incidence rates relative to current mortality
26 rates.
27

28 **5.7 Response to Charge Question #2f**

29 *Proposed approaches for extending risk estimates to radiations of different LET’s - in*
30 *particular, deriving site-specific risk estimates for alpha or x radiations based on models*
31 *derived from the A-bomb survivors, who were primarily exposed to gamma rays (see Section*
32 *III).*
33

34 This charge question is directed toward evaluating the use of RBE and radiation
35 weighting factors to convert the risk derived from the A-bomb data to site-specific risks for alpha
36 particles and low energy photons and electrons. There are major problems with this approach.
37 First, the biological response to high and low LET radiation suggest that there are different
38 mechanisms of action for each of these exposures. For example, exposure of a single cell to a
39 single alpha particle produces a marked “bystander effect” while exposing cells to low LET
40 radiation is much less effective in producing such changes. Multiplying the risks from a single
41 acute exposure to low LET radiation to predict the risk from a non-uniform protracted exposure
42 to high-LET radiation from internally deposited alpha particles may neglect important factors.
43 As stated in the white paper, there are human populations that that have been exposed to

1 internally deposited radioactive materials and have cancer data associated with them. These data
2 provide information on the risks of alpha induced cancers in the bone and liver. Since there are
3 human populations and extensive animal studies on internally deposited radioactive materials
4 that emit alpha particles the RAC recommends that these data be used as supporting evidence for
5 extrapolation to determine the RBE’s that are used. Each cancer site has to be considered
6 individually and the RAC recommends that single values (i.e., 20) not be used to extrapolate
7 risks from external exposure to that for different radioisotopes and target tissues.

8
9 Biological effectiveness of alpha-emitters. The issue of estimation of lung cancer risks
10 for alpha-emitters is problematic because of the uncertainties and variability in the available
11 studies. In the past it has been assumed by ICRP and others that alpha-emitters have an RBE of
12 20 compared to exposures from gamma-emitters. BEIR VI provides us with estimates of lung
13 cancer in humans induced by exposure to the radon decay chain in which alpha- emitters
14 predominate. Using these data and the LSS data would suggest an RBE of 2-3 but most of the
15 dose is to the airways of the lung, whereas for many other alpha emitters the region of interest is
16 the dose to the periphery of the lung. Estimates of radiation risks for the periphery of the lung are
17 available from the Mayak worker studies. These suggest an RBE of about 10 for plutonium
18 workers compared to workers exposed to gamma emitters at Mayak. Unfortunately this study has
19 several methodological problems which are being addressed in further analyses of the data.
20 Animal studies in dogs and rodents comparing inhaled alpha-emitters with beta-gamma-emitters
21 have in some studies indicated an RBE of 20 or higher. This presents an uncomfortable situation
22 in that the data in people does not provide any clear indication for the RBE for lung and that the
23 estimates may be well below those currently recommended for people by the ICRP and other
24 groups. If reliance is placed solely on the human data one possible solution is to have different
25 RBEs for the airway of the lung and the periphery of the lung. A justification for different RBEs
26 is that the airways and the periphery of the lung are very different types of tissues, with different
27 functions and cell types. Also different types of tumors arise from the airways and periphery. If
28 this approach were adopted, an RBE for airways would be a value of 2-3. For the periphery of
29 the lung it would be best to continue to use a value of 20 until further analysis of the Mayak
30 workers or other populations are available for consideration. The RBEs would be used by
31 proportioning the risks for LSS lung cancer by the weights of the airways and the weight of the
32 lung periphery.

33
34 Biological effectiveness of low energy photons and electrons: The EPA White Paper
35 suggests that the relative biological effectiveness (RBE) for medical x rays is about 2 – 2.5.
36 However, x rays are not unique from gamma rays except for their production. Any risk estimate
37 associated with exposure to photons needs to be correlated with energy as opposed to method of
38 production. Subsequent guidance could make reference to specific applications such as low
39 energy photons from certain radionuclides or medical x rays. Such guidance would need to take
40 into consideration variation in the x-ray spectrum, which is caused by use of various targets and
41 filters that may increase the average photon energy to which patients are exposed.

42
43 Reviews by ICRU (1986) and Kocher et al. (2005) show that RBEs for low energy
44 photons, < 30 keV, and low energy electrons, <15 keV, are high when compared to higher

1 energy x rays and ^{60}Co gamma rays. A probability distribution by Kocher et al. (2005) showed
2 a median radiation effectiveness factor (REF) of approximately 2.4 for photons less than 30 keV
3 and for ^3H beta particles. Thus, an effectiveness factor for these low energy radiations in the
4 range of 2 to 2.5 seems reasonable.
5

6 **5.8 Response to Charge Question #2g**

7 *Estimation of risks for sites not specified in BEIR VII, specifically bone and skin, for which we*
8 *propose to update our current approaches (see Sections III.A. and V, respectively).*
9

10 Non-melanoma skin cancer (NMSC) is the most common cancer in the U.S. population.
11 Given the high and increasing incidence of NMSC EPA has no option but to provide risk
12 estimates for radiogenic NMSC in the US population.
13

14 The options include:

- 15 1) The use of the most current scientifically valid data available and base estimates on
16 updated data (Shore 1990, Shore 2001). This would seem to be a reasonable approach;
17 and/or
18
19 2) Provide risk estimates for NMSC induced by ionizing radiation (probably only for Basal
20 Cell Carcinoma (BCC)). BCC incidence as well as mortality data exist for photons and
21 should both be provided because of the low mortality associated with BCC.
22

23 Melanoma skin cancer is not considered to be induced by ionizing radiation.
24

25 Data for mortality due to bone cancer following exposure to ^{226}Ra , ^{228}Ra and ^{224}Ra are
26 available for humans. These data provide a basis for estimating risks from these isotopes that
27 distribute uniformly in the bone. They also should be compared to the derived risks using the A-
28 bomb data and help define the magnitude of the RBE and radiation weighting factor used. The
29 induction of bone cancer by radioisotopes that produce non-uniform surface deposition in the
30 bone such as ?? need to be considered as being in a different class from the isotopes that are
31 uniformly deposited in the bone. For isotopes for which there are no human data, the animal
32 data may help in determining the factors necessary to relate the risk from an isotope like ^{239}Pu or
33 the beta-emitting ^{90}Sr to that derived from the A-bomb data combining DDREF with radiation
34 weighting factors.
35

36 **5.9 Response to Charge Question #2h**

37 *Estimation of risk due to prenatal exposure. EPA’s current lifetime risk estimates do not*
38 *include risk from prenatal exposure, and BEIR VII does not provide them. The draft White*
39 *Paper uses ICRP recommendations to project its risks of childhood cancers induced by in*
40 *utero exposure. Please comment on the soundness of the approach described in the draft*
41 *White Paper to apply ICRP as described in Section IV.*
42

1 The RAC concludes that it would be reasonable for the EPA to base its risk estimates for
2 *in utero* radiation exposure on those recommended by the ICRP.

3
4 Rationale:

- 5 • BEIR VII does not provide risk estimates for *in utero* exposure to radiation, and the EPA
6 needs an estimate for its guidance documents.
- 7
8 • Few human data exist on which to base an estimate of radiogenic cancer risk for *in utero*
9 exposure to radiation. The primary sources of data are the Oxford survey of Childhood
10 Cancer (Mettler and Upton, 1995, Steward et al. 1958, Mole 1990, Doll and Wakeford
11 1997) and studies of Japanese atomic bomb survivors exposed during pregnancy
12 (Delongchamps et al, 1997). When all sources of uncertainty are taken into account, the
13 risk estimates from these studies are not incompatible with each other (Wakeford & Little
14 2002). ICRP has provided an absolute risk estimate for cancer risk of $6 \times 10^{-2} \text{ Gy}^{-1}$ from
15 ages 0-15 after *in utero* irradiation (ICRP 2001a; ICRP 2001b).
- 16
17 • Even though the risk from *in utero* exposure is a minor component of the overall
18 radiogenic cancer risk, a discussion of it should be included for completeness.
- 19
20
21

6. RESPONSE TO CHARGE QUESTION 3: UNCERTAINTIES NOT QUANTIFIED IN BEIR VII

Charge Question 3: *BEIR VII provides quantitative uncertainty bounds for each of its risk coefficients. EPA proposes to adopt this methodology with some additional discussion of the uncertainties not quantified in BEIR VII. Please comment on the adequacy of this approach (see Section II.K.).*

The RAC strongly endorses the EPA-ORIA’s desire to estimate uncertainty bounds for its radiogenic cancer risk estimates. Indeed, given the range of possible operational uses of the risk estimates, as much effort should go into estimating the uncertainty bounds as into producing the central or point risk estimates themselves.

Ideally, the uncertainty analysis would involve the development of a probability density function for (site-specific) estimated risk, rather than confidence intervals around a central or point risk estimate. Such an approach, which has previously been considered by other national and international committees, would facilitate risk estimation based on other than the average risk. For example, such an approach might facilitate the identification of a minimum cost-of-errors (or ‘loss’) risk estimate for operational use (e.g., in risk-informed regulation). However, the RAC believes that such an approach is not likely to be practically achievable, and endorses the EPA-ORIA’s approach (central risk estimate with uncertainty bounds, following BEIR VII).

The uncertainty bound estimates should incorporate, to the extent possible, *all* sources of error and/or uncertainty, including the three main sources identified in BEIR VII (sampling variability in the LSS data, transport of risk from LSS to U.S. population, and the appropriate value for DDREF **at both high and low doses of low-LET radiation** (or, equivalently, the **appropriate use of the LNT** dose-response model used for low-dose extrapolation)), but also considering other sources of error and/or uncertainty identified by the EPA-ORIA (including dosimetry (of which neutron RBE is a factor), disease detection, correct disease classification, temporal patterns, and appropriate RBE values). By “consider,” the RAC means that the EPA-ORIA should attempt to estimate, in a preliminary fashion, the relative magnitude of the contribution of the additional sources of error or uncertainty they identified to the overall uncertainty. Of importance, it is useful to try to estimate the independent contribution of these additional sources, most of which are likely partially correlated with those sources identified in BEIR VII. One possible way of estimating the magnitudes is via some modest simulation studies. Only if the independent contribution of any of these additional sources of error is potentially significant in magnitude should that source be included in the uncertainty analysis. In any event, the methods of uncertainty analysis should follow BEIR VII.

There is some value to producing two sets of uncertainty bounds, one representing the bounds on the (site-specific) central or point risk estimate for the method of combining the RR and AR that the EPA finally chooses to use, the other representing combinations ranging from 100% RR through 100% AR. The former gives a measure of the uncertainty of the central risk estimate derived from the method specifically used, and the latter gives an indication of the range in which the true value (independent of method) likely resides.

1
2 In coming to these recommendations, the RAC considered several additional
3 complications that could influence uncertainty. To begin with, **the significant biological**
4 **responses from the LSS** and other epidemiological data cover a finite range of individual doses.
5 The uncertainties associated with risk estimates are smallest within that range of doses **where**
6 **significant changes in cancer frequency can be detected.** At doses significantly above this
7 range, radiogenic cancer risk estimates are not meaningful, because acute fatalities dominate. At
8 doses below this range, there is, by definition, no directly demonstrated effect in humans, and
9 risk estimates must be based, explicitly or implicitly, on an assumed **LNT** dose-response model
10 and method of extrapolation from known, higher-dose/higher-response data. In such a situation,
11 lower-dose risk estimates may have larger relative uncertainties than higher-dose risk estimates
12 because of this extrapolation.

13
14 Said another way, assumptions about the biophysical model for radiogenic cancer
15 influence the choice of dose-response model, and errors in the assumptions and subsequent
16 choice of method of extrapolation amplify errors in the central or point risk estimates. The
17 biophysical model for radiogenic cancer intrinsic to LNT implies direct DNA damage to a single
18 cell as the initiating event, followed by clonal expansion. **It is well established that the initial**
19 **DNA damage increases linearly with dose. However, it is also well known that the**
20 **biological processing of that damage is non-linearly dependent on dose. Since dose-**
21 **dependent changes in gene and protein expression,** adaptive response, genomic instability,
22 and bystander effects **have been demonstrated in many biological systems,** they can change
23 the shape of the dose-response function at low doses. *(Such effects are likely only important*
24 *(relative to LNT) at low doses.) Perhaps we could delete this.* Of importance, the specific nature
25 of the original damage, and the mechanisms involved in the biological processing of that damage
26 change with dose. It is thus likely an oversimplification to suggest that only the magnitude of
27 somatic genetic changes is dose-dependent. It is further likely that errors in risk estimates
28 progressively increase (relatively) as dose decreases if these additional elements of the
29 biophysical model are more prominent at low doses.

30
31 Having said that, BEIR VII specifically considered adaptive response, genomic
32 instability, and bystander effects, and concluded that there is insufficient evidence to explicitly
33 add these effects to the dose-response model. The EPA-ORIA proposes **at the present time** to
34 follow BEIR VII **and use the LNT for calculation of radiation risk.** In the absence of
35 compelling scientific evidence to do otherwise, the RAC endorses the EPA-ORIA’s plan in this
36 regard. The RAC does recommend, however, that the EPA-ORIA include a (qualitative)
37 discussion of **modern molecular biological** concepts in its final report. As a cautionary note,
38 **we recommend that the EPA discuss** the application of its **LNT** risk estimates in very low-
39 dose settings **where there are no human cancer data and where the doses are a fraction of**
40 **the ever present background radiation exposure.**

41
42 It is important to note that there is indeed opportunity to include uncertainties in the
43 model – that is, uncertainties in high-dose versus low-dose behavior – in the overall uncertainty
44 analysis. In BEIR VII and the EPA-ORIA’s proposed approach to uncertainty estimation, this
45 “additional” uncertainty is contained within the uncertainty in the value for DDREF, since

1 DDREF is only invoked at lower doses. The RAC thus strongly endorses the EPA-ORIA’s
2 intention to include uncertainty in DDREF in the overall uncertainty analysis.
3

4 **The RAC suggests the need for an additional dose-rate effectiveness factor at higher**
5 **total doses delivered at low dose rates where the DDREF does not apply. In these higher**
6 **dose ranges the influence of dose-rate can be marked and a dose rate factor would much**
7 **higher than the 1.5 recommended by BEIR VII. This large dose rate factor can be very**
8 **important in predicting the biological damage in the event of nuclear accidents or terrorist**
9 **activities where large populations could be exposed to large doses at low dose-rates**

10
11 Uncertainties are also a function of time into the future, being smallest in the near time
12 frame. This is due to several factors, including changes in future (actual) populations (as opposed
13 to a ‘stationary population’), future background cancer incidence, and future medical advances
14 (since the case fatality rate may decrease as a result of better treatment interventions in the
15 future). Uncertainties thus become greater as the risk estimates are applied further into the future.
16 The RAC recommends that EPA-ORIA include a (qualitative) discussion of these concepts in its
17 final report.
18
19
20

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7. RESPONSE TO CHARGE QUESTION 4: ISSUES RELATING TO RADIOGENIC THYROID CANCER NOT QUANTIFIED IN BEIR VII

Charge Question 4: *In Section VI, the draft White Paper discusses some issues relating to radiogenic thyroid cancer. Does the RAC have any specific suggestions for dealing with this risk; e.g., does the RAC have any advice on gender specificity, effectiveness of iodine -131 compared to gamma rays, or estimation of thyroid cancer mortality?*

The RAC wishes to defer any advice to the EPA on this issue until the updated NCRP report on thyroid risk is published.

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APPENDIX A – BIOSKETCHES

**U.S. ENVIRONMENTAL PROTECTION AGENCY
SCIENCE ADVISORY BOARD
RADIATION ADVISORY COMMITTEE (RAC)**

--- (To be Added in Quality Review Draft) ---

APPENDIX B –ACRONYMS

1		
2		
3	A-Bomb	<u>A</u> tom <u>B</u> omb
4	AM	<u>A</u> rithmet <u>M</u> ean
5	AR	<u>A</u> bsol <u>R</u> isk
6	BCC	<u>B</u> asal <u>C</u> ell <u>C</u> arcinoma
7	BEIR	<u>B</u> iological <u>E</u> ffects of <u>I</u> onizing <u>R</u> adiation
8	BEIR VII	<i>Health Risks from Exposure to Low Levels of Ionizing Radiation BEIR VII</i>
9		<i>Phase 2</i>
10	CDC	<u>C</u> enters for <u>D</u> isease <u>C</u> ontrol
11	CFR	<u>C</u> ode of <u>F</u> ederal <u>R</u> egulations
12	Co	Chemical symbol for cobalt (⁶⁰ Co isotope)
13	DDREF	<u>D</u> ose and <u>D</u> ose- <u>R</u> ate <u>E</u> ffectiveness <u>F</u> actor
14	DFO	<u>D</u> esignated <u>F</u> ederal <u>O</u> fficer
15	DNA	<u>D</u> eoxyribonucleic <u>A</u> cid
16	EAR	<u>E</u> xcess <u>A</u> bsolute <u>R</u> isk
17	EPA	<u>E</u> nvironmental <u>P</u> rotection <u>A</u> gency (U.S. EPA)
18	ERR	<u>E</u> xcess <u>R</u> elative <u>R</u> isk
19	FR	<u>F</u> ederal <u>R</u> egister
20	FGR-13	Federal <u>G</u> uidance <u>R</u> eport <u>13</u>
21	GM	<u>G</u> eometric <u>M</u> ean
22	GMC	<u>G</u> eometric <u>M</u> ean <u>C</u> oefficient
23	GSD	<u>G</u> eometric <u>S</u> tandard <u>D</u> eviation
24	Gy	<u>G</u> ray
25	H	Chemical symbol for <u>H</u> ydrogen (³ H isotope)
26	I	Chemical symbol for <u>I</u> odine (¹³¹ I isotope)
27	ICRP	<u>I</u> nternational <u>C</u> ommission on <u>R</u> adiological <u>P</u> rotection
28	ICRU	<u>I</u> nternational <u>C</u> ommission on <u>R</u> adiation <u>U</u> nits and Measurements, Inc.
29	IREP	<u>I</u> nteractive <u>R</u> adio <u>E</u> pidemiological <u>P</u> rogram
30	keV	<u>k</u> ilo <u>e</u> lectron <u>V</u> olts
31	LAR	<u>L</u> ifetime <u>A</u> ttributable <u>R</u> isk
32	LET	<u>L</u> inear <u>E</u> nergy <u>T</u> ransfer
33	LNT	<u>L</u> inear <u>N</u> on <u>T</u> hreshold
34	LSS	<u>L</u> ife <u>S</u> pan <u>S</u> tudy
35	mSv	<u>m</u> illi- <u>S</u> ievert
36	NAS	<u>N</u> ational <u>A</u> cademy of <u>S</u> ciences (U.S. NAS)
37	NCHS	<u>N</u> ational <u>C</u> enter for <u>H</u> ealth <u>S</u> tatistics
38	NCI	<u>N</u> ational <u>C</u> ancer <u>I</u> nstitute
39	NCRP	<u>N</u> ational <u>C</u> ouncil on <u>R</u> adiation <u>P</u> rotection and Measurements
40	NIH	<u>N</u> ational <u>I</u> nstitutes of <u>H</u> ealth
41	NIOSH	<u>N</u> ational <u>I</u> nstitute for <u>O</u> ccupational <u>S</u> afety and <u>H</u> ealth
42	NMSC	<u>N</u> on- <u>M</u> elanoma <u>S</u> kin <u>C</u> ancer
43	NRC	<u>N</u> ational <u>R</u> esearch <u>C</u> ouncil
44	OAR	<u>O</u> ffice of <u>A</u> ir and <u>R</u> adiation (U.S. EPA/OAR)
45	ORIA	<u>O</u> ffice of <u>R</u> adiation and <u>I</u> ndoor <u>A</u> ir (U.S. EPA/OAR/ORIA)

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2	PAG	<u>P</u> rotective <u>A</u> ction <u>G</u> uide
3	Pu	Chemical symbol for <u>P</u> lутonium (²³⁹ Pu Isotope)
4	QA	<u>Q</u> uality <u>A</u> ssurance
5	QC	<u>Q</u> uality <u>C</u> ontrol
6	QA/QC	<u>Q</u> uality <u>A</u> ssurance/ <u>Q</u> uality <u>C</u> ontrol
7	R	<u>R</u> oentgen
8	Ra	Chemical symbol for <u>R</u> adium (Isotopes include ²²⁴ Ra, ²²⁶ Ra, ²²⁸ Ra, and ²³⁶ Ra)
9	RAC	<u>R</u> adiation <u>A</u> dvisory <u>C</u> ommittee (U.S. EPA/SAB/RAC)
10	rad	Traditional unit of <u>r</u> adiation absorbed dose in tissue (a dose of 100 rad is
11		equivalent to 1 gray (Gy) in SI units)
12	RBE	<u>R</u> elative <u>B</u> iological <u>E</u> ffectiveness
13	REF	<u>R</u> adiation <u>E</u> ffectiveness <u>F</u> actor
14	rem	<u>R</u> adiation equivalent in <u>m</u> an; traditional unit of effective dose equivalent (equals
15		rad x tissue weighting factor) (100 rem is equivalent to 1 Sievert (Sv))
16	R/h	<u>R</u> oentgen per <u>h</u> our; traditional measure of exposure rate
17	Rn	Chemical symbol for Radon
18	RR	<u>R</u> elative <u>R</u> isk
19	SAB	<u>S</u> cience <u>A</u> dvisory <u>B</u> oard (U.S. EPA/SAB)
20	SCC	<u>S</u> quamous <u>C</u> ell <u>C</u> arcinoma
21	SEER	<u>S</u> urveillance, <u>E</u> pidemiology, and <u>E</u> nd <u>R</u> esults
22	SI	<u>I</u> nternational <u>S</u> ystem of <u>U</u> nits (from NIST, as defined by the General Conference
23		of Weights & Measures in 1960)
24	Sr	Chemical Symbol for <u>S</u> trontium (⁹⁰ Sr Isotope)
25	Sv	<u>S</u> ievert, SI unit of effective dose equivalent in man (1 Sv is equivalent to 100 rem
26		in traditional units)
27	UNSCEAR	<u>U</u> nited <u>N</u> ations <u>S</u> cientific <u>C</u> ommittee on the <u>E</u> ffects of <u>A</u> tom ic <u>R</u> adiation
28	US	<u>U</u> nited <u>S</u> tates
29	WLM	<u>W</u> orking <u>L</u> evel <u>M</u> onths
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