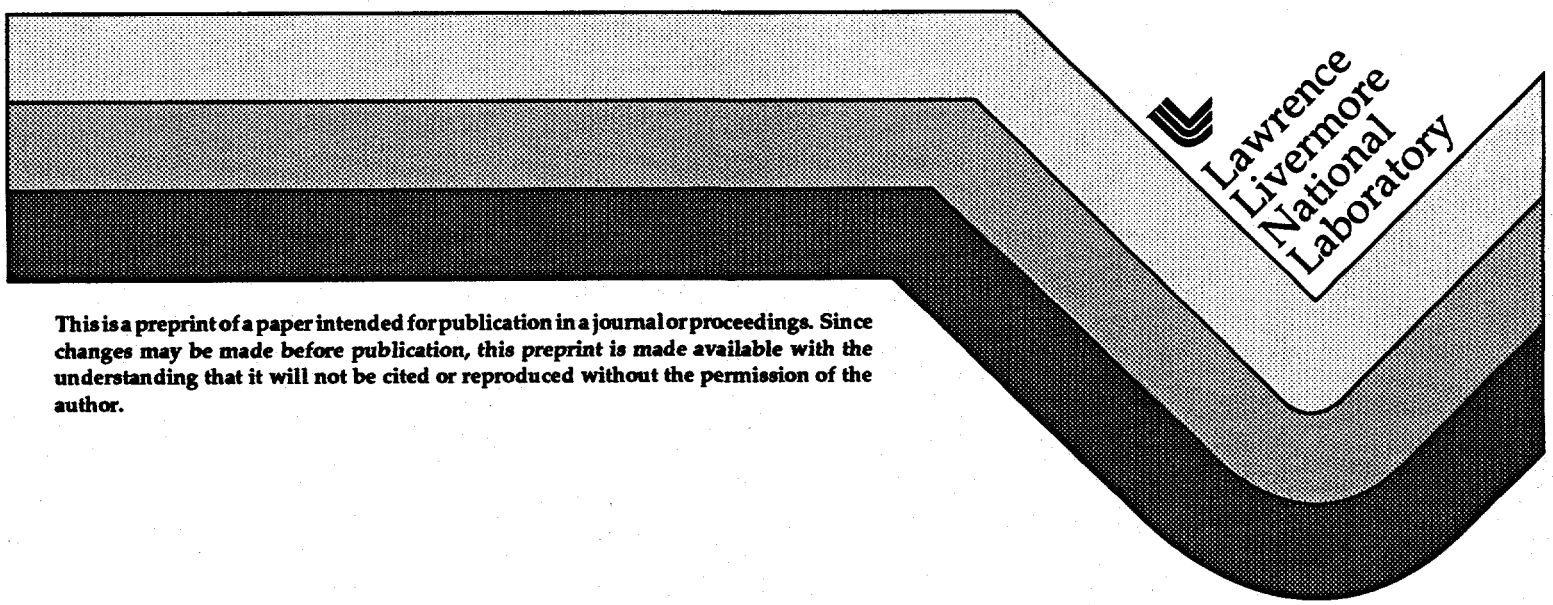
UCRL-JC-121795
PREPRINT
CONF-960306--12

Setting Standards for Radiation Protection: A Time for Change

H. Wade Patterson
David P. Hickman

This paper was prepared for submittal to the
Fourth International Conference on Nuclear Engineering
New Orleans, LA
March 10-14, 1996

January 1996



DISCLAIMER

Setting Standards for Radiation Protection: A Time for Change

H. Wade Patterson
David P. Hickman

Lawrence Livermore National Laboratory, Livermore, California, 94550

Abstract

In 1950, the International Commission on Radiation Protection (ICRP) recommended that "certain radiation effects are irreversible and cumulative." Furthermore, the ICRP "strongly recommended that every effort be made to reduce exposures to all types of ionizing radiations to the lowest possible level."[1] Then in 1954 , the ICRP published its assumption that human response to ionizing radiation was linear with dose, together with the recommendation that exposures be kept as low as practicable.[2] These concepts are still the foundation of radiation protection policy today, even though, as Evans[3] has stated, "The linear non-threshold (LNT) model was adopted specifically on a basis of mathematical simplicity, not from radio-biological data. . . ." Groups responsible for setting standards for radiation protection should be abreast of new developments and new data as they are published; however, this does not seem to be the case. For example, there have been many reports in scientific, peer-reviewed, and other publications during the last three decades that have shown the LNT model and the policy of As Low As Reasonably Achievable (ALARA) to be invalid. However, none of these reports has been refuted or even discussed by standard-setting groups. We believe this mandates a change in the standard-setting process.

*This work was performed under the auspices of the U.S. Department of Energy by Lawrence Livermore National Laboratory under contract No. W-7405-Eng-48.

Introduction

In 1950, the ICRP recommended that " certain radiation effects are irreversible and cumulative" and it further recommended that "every effort be made to reduce exposures to all types of ionizing radiations to the lowest possible level"[1]. Then in 1954 the International Commission on Radiation Protection (ICRP) published its assumption that human response to ionizing radiation was linear with dose, together with the recommendation that exposures be kept as low as practicable [2]. These concepts are still the foundations of radiation protection policy today, even though, research to date fails to support these assumptions. It should be axiomatic that groups responsible for setting radiation protection standards keep abreast of new developments and new data as they are published, however this seems not to be the case for radiation protection. Evans[3] stated, "The linear non-threshold (LNT) model was adopted specifically on a basis of mathematical simplicity, not from radio-biological data... "

There have been many reports in scientific, peer-reviewed, or other, publications during the last three decades, all of which show the LNT model and the policy of As Low As Reasonably Achievable (ALARA) to be invalid. And as far as we are aware, none have been refuted or even discussed by standard-setting groups. Rather, they seem to have been ignored. It is time to mandate a change in the standard-setting process, and begin to question and remove as necessary, the time (and research) withered concepts that have existed since the early years of radiation protection.

Historical review

It was thought for over 30 years after the discovery of x-rays and radioactivity that the somatic effects of radiation exposure in humans could be repaired; and for this period the concept of a "tolerance" dose was used to set protection standards. This view was revised, when in 1927, Hermann Muller published his results on the induction of mutations by radiation. Shortly thereafter and until the early 1950's protection standards were set to limit the number of recessive genes introduced into the gene "pool" by radiation exposure.

Then, by the mid-nineteen fifties, when it was recognized that "genetic damage.....is not a limiting factor" [4], radiation protection standards began to look at alternative effects such as life span shortening and cancer. The standards incorporated the concept that cancer induction has no threshold, and that all exposure carries some risk, while at the same time, recognizing that there was some evidence of repair and recovery from radiation effects. From this innocuous, but erroneous, assumption of linear non-threshold effects has grown the pernicious, but now official, policy of standard-setting groups. Based on these early concepts, the linear model must be used to fit exposure-response data over the entire dose range, even though there is no basis, other than "mathematical convenience" for so doing. An example of such policy was recently reiterated in the Federal Register, Fig. 1, despite a statement that human response to ionizing radiation is "non-linear."

Natural phenomena

There is abundant evidence that many natural processes are non-linear. As early as the 1800's both the principle of an optimum quantity (neither to little or too much) , and of a necessary minimum quantity, of environmental agents were expressed in the context of plant growth modeling [5]. An extension of this principle to mutagenic effects was given by Bowen and Tolley [6]. C.E.K. Mees, in "The Theory of the Photographic Process" [7], details the non-linearity of the relation "between the exposure given...and the density obtained" in a photographic emulsion. Here, a minimum amount of energy must be supplied to render a silver grain developable. This requirement of a minimum energy needed to cause an effect is common to other processes as well.

Continuing this line of reasoning, we know that all living organisms, DNA, and even molecules, are highly ordered systems, and it is clear that this order must be maintained if a system is to perpetuate itself. Scientific logic dictates that it is natural for such systems to develop a mechanism to routinely perform needed maintenance. And indeed, self-maintenance and repair are everywhere evident in living organisms. It is also natural that this repair mechanism can be stimulated by external forces and this too is everywhere evident. Hair regrows, skin is replaced, and neural pathways regenerate. All are evidence of both normal and stimulated repair, the expected response to a normal environment.

It has been speculated that such changes are mandated in order to induce adaptation and evolution. If this were so, then exposure to environmental agents, below the threshold for harm, may be vital to assure that needed adaptation in a constantly changing environment continues to occur.

A multitude of physical evidences demonstrate that a threshold exists, below which highly ordered systems will show no detrimental response. For example, oxidation induced by Brownian motion would turn our bodies into CO_2, H_2O, and a fine white inorganic powder, if a threshold did not exist.

Stated differently, we human beings have developed and adapted in such a way as to function best and to be fittest over a range of environmental agents. Ultraviolet light, temperature, pressure, and our response to trace elements are examples. Is it logical to assume that our response to radiation, another environmental agent, should be different?

Review of experimental radiobiological observations

Twenty years ago, Robley Evans' paper[2] showed the LNT model to be radiobiologically untenable. By extension this would invalidate the conceptual basis for ALARA. Nonetheless, the Committee on Biological Effects of Ionizing Radiation (BEIR) for the National Academy of Science) and the United Nations Sub-Committee on the Effects of Atomic Radiation (UNSCEAR) have chosen to use selected data, manipulated this data, and forced the data to fit the linear non-threshold model. To review this process, just re-read the Robley Evans paper.

Both ICRP and National Committee on Radiation Protection and Measurement (NCRP) base their "recommendations" on the BEIR and UNSCEAR report(s), and on Japanese data which is also forced to fit the linear non-threshold model. Fig. (2) In turn, regulatory bodies such as the United States Department of Energy and Environmental Protection Agency (USDOE and USEPA) justify their use of the LNT model and ALARA by referencing the ICRP, NCRP, BEIR and UNSCEAR reports.

Over the years many published papers, cited in the attached reference lists, from the U.S., China, India, Canada, Japan, and England also invalidate the linear non-threshold model. Most, if not all, have been published after peer review. Some show a downward trending response to radiation exposure, and others show a threshold. (See the first attached list of references for response versus exposure effects). These studies demonstrate that there is no relation whatsoever between the epidemologic or biological data and the linear non-threshold model. The published results from these studies, which demonstrate the fallacy of the LNT theory, include data on cancer incidence from both external and internal exposures, for both whole populations and occupationally exposed groups. Figs (3), (4), and (5) show such data; these are typical of the entire group of attached references. Equally important, an additional set of references provide evidence for both normal and stimulated repair of radiation damage. (See the second attached list of references for genetic repair of radiation effects.)

Summary

When we review the substantial list of published data as well as the author's conclusions given in the attached references and compare the data with the linear non-threshold, we can only conclude that the LNT model (and by inference ALARA) are wrong. Modeling, or fitting, or any other form of interpretation of this data need not be performed. Rather direct observation of the data allows one to easily conclude that the LNT model can not be used as a predictor of dose versus effect. As Richard Feynman said [8], "In general we look for a new law by the following process. First we guess it. Then we compute the consequences of the guess to see what would be implied if this law we guessed is right. Then we compare the result of the computation with nature, with experiment or experience, compare it directly with observation, to see if it works. If it disagrees with experiment it is wrong. In that simple statement is the key to science. It does not make any difference how beautiful your guess is. It does not make any difference how smart you are, who made the guess, or what his name is - if it disagrees with experiment it is wrong. That is all there is to it. ..."
Feynman continues, by stating; "Another thing I must point out is that you cannot prove a vague theory wrong. If the guess you make is poorly expressed and rather vague, and the method you use for figuring out the consequences is a little vague - you are not sure, and you say, 'I think everything's all right because it's all due to so and so, and such and such do this and that more or less, and I can sort of explain how this works..., then you see that this theory is good, because it cannot be proved wrong! Also if the process of computing the consequences is indefinite, then with a little skill any experimental results can be made to look like the expected consequences."

Feynman's view of science is directly applicable both to use and application of the LNT model and ALARA. Moreover, based on review of the data presented in the attached sets of references, there is unequivocal evidence that heterogeneous groups of humans have, without ill effect, tolerated chronic radiation exposures of at least 0.1 rad per year; and can tolerate acute exposures of at least 10 rad, also with no effect.

Recommendation

Although it may once have been prudent to assume that the linear model should be used, we now believe that, in the spirit of true science, it is obligatory to reject this policy. The promulgation of standards for radiation protection must be based on scientific observation rather than on an unsupported assumption and on subsequently begged assertions. Obviously a re-examination should be commissioned under the auspices of some entity other than those responsible for present standards. It seems apparent that these groups have failed to consider new developments and data, and that they would face a severe conflict of interest were they to be involved in a new review.

After such a review and reexamination it is to be hoped that the current application of the LNT model and the ALARA principle, together with its regularly ignored mandate to balance benefit against cost, would both be abandoned. We make no recommendation about methods and procedures to be used in any re-examination of the linear model by a newly constituted group, believing it is premature for us to propose specific solutions. Also, we have for this reason omitted any discussion about what model, if any, should replace the LNT, rather the approach should use good scientific principles in arriving at logically and unequivocally stated concepts.

References

1. International Commission on Radiation Protection, Recommendations of the International Commission on Radiation Protection, NBS Handbook 47, U.S. Department of Commerce, 1950 (issued June 29, 1951).
2. International Commission on Radiation Protection, Recommendations of the International Commission on Radiation Protection, (revised December 1, 1954), British Journal of Radiology, Supplement No. 6, 1955.
3. Evans, Robley D., Radium in man., Health Physics, 27, pp 497-510, 1974.
4. National Committee on Radiation Protection, NBS Handbook 59, U.S. Department of Commerce, (issued September 24, 1954).
5. C.A. Browne, Liebig and After Liebig, AAAS Publication 16, 1942.
6. W.M. Bowen, H.D. Tolley, Assessing Mutagenic Effect by Likelihood Methods, Proceedings of the 1980 DOE Statistical Symposium, CONF 801045.
7. C.E. Kenneth Mees, The Theory of the Photographic Process, edited by T.H. James with the technical assistance of Ardelle Kocher. Contributors: C.R. Berry [and others]. 3d ed. New York, Macmillan [1966].
8. (Excerpted from "The Character of Physical Law", MIT Press, Cambridge MA, 1965).

Figures

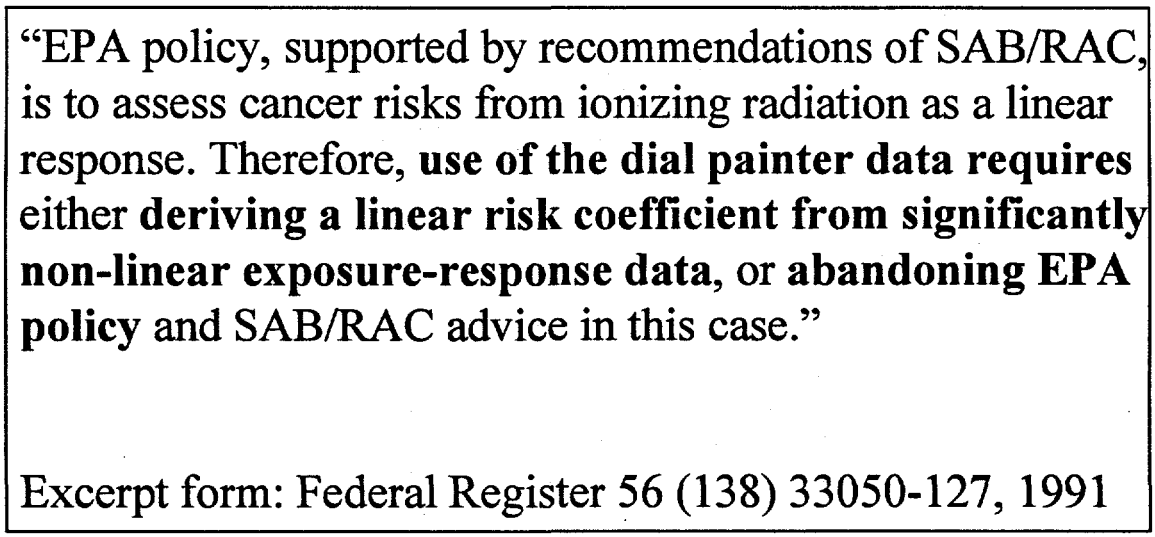


Fig. 1. EPA admission that the LNT model is not valid.

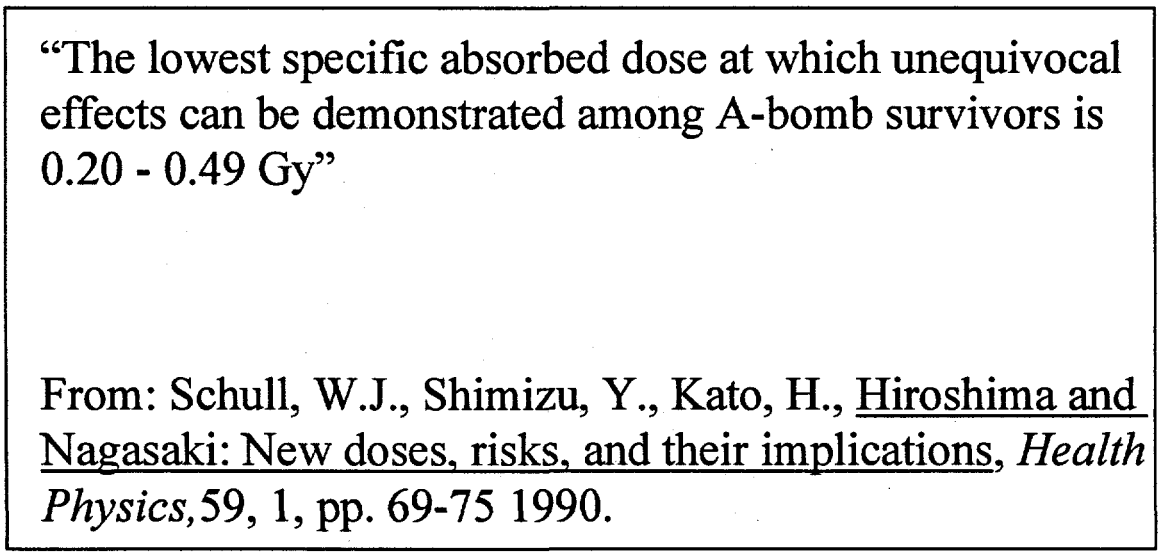


Fig. 2. The LNT model does not apply to the A-bomb survivor data.

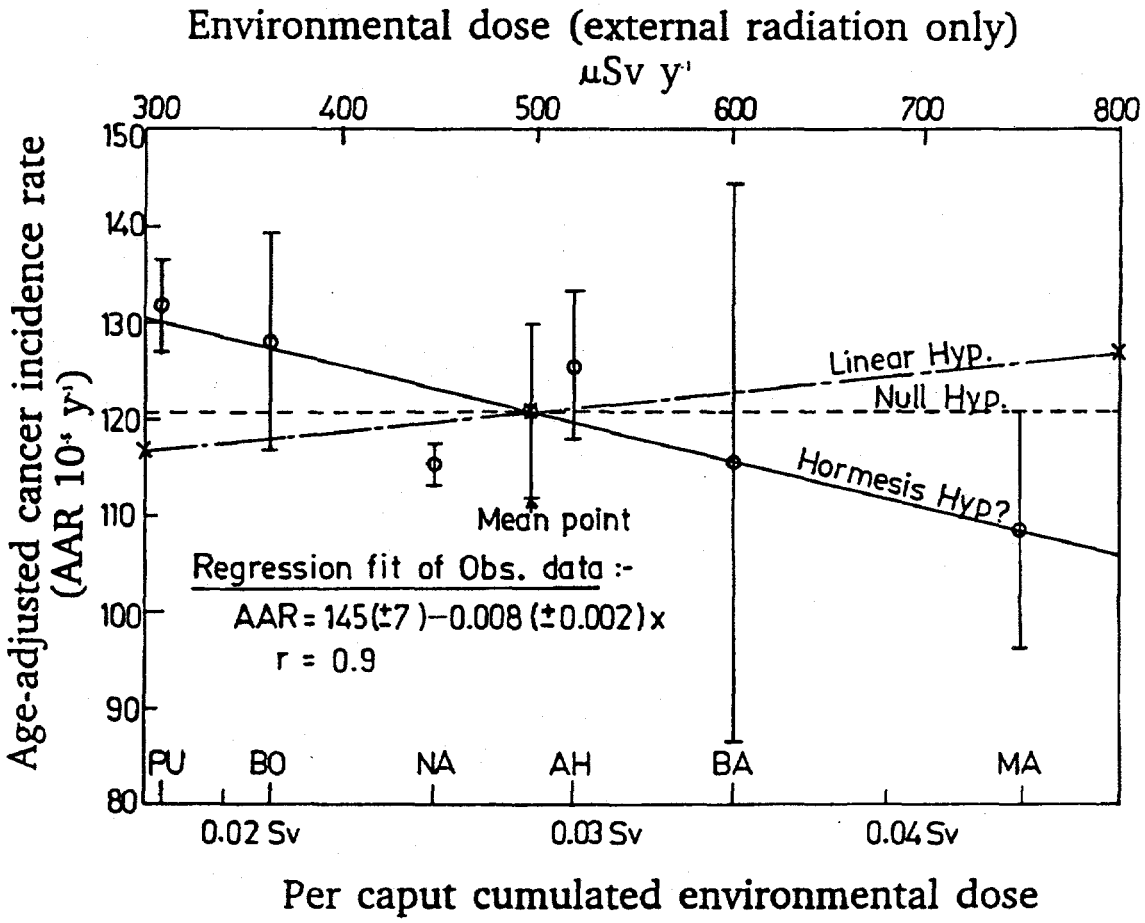


Fig. 3. Cancer risk from environmental radiation (external and internal) in age-standardized Indian populations.

Reprinted with permission from : Nambi, K.S.V., Soman, S.D., Further observations on environmental radiation and cancer in India, *Health Physics*, 59, 3, p 543, 1990.

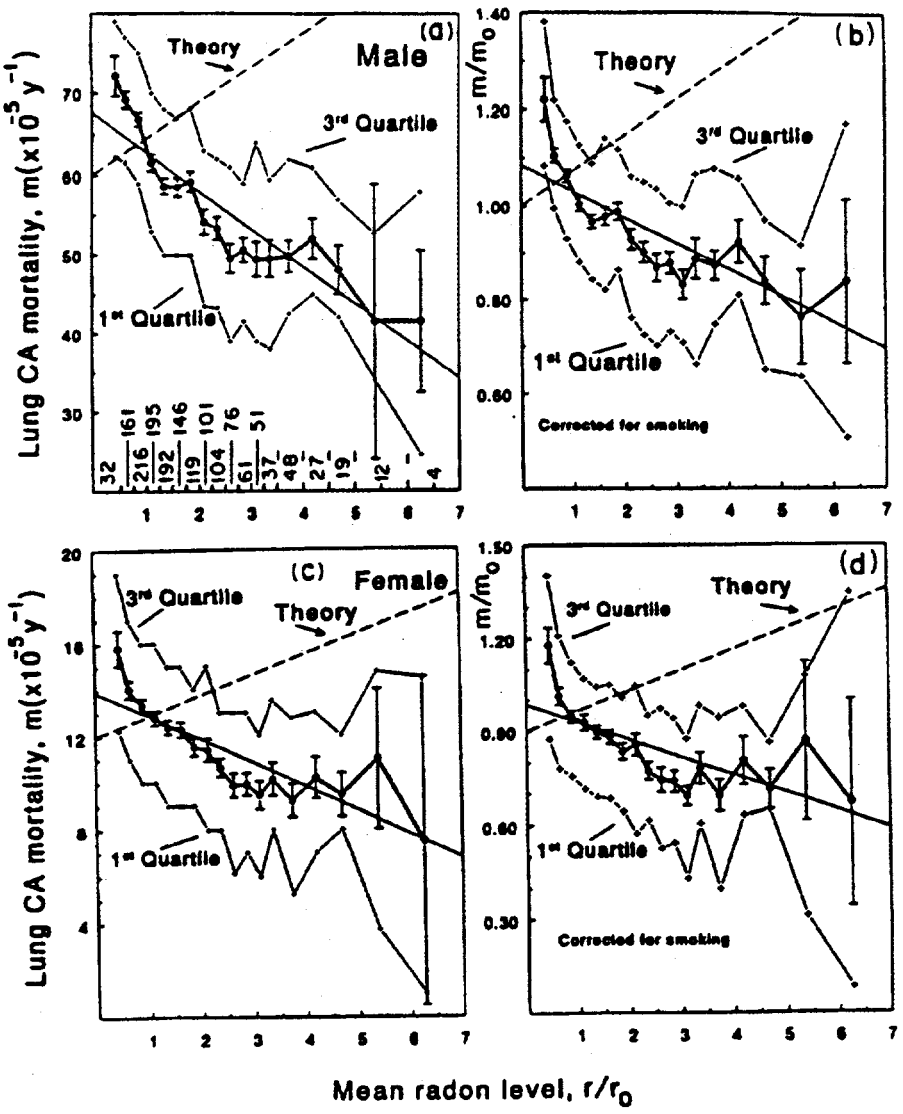


Fig. 4. Radon cancer mortality data disagrees with the LNT model.

Reprinted with permission from: Cohen, B.L., Test of the linear-no threshold theory of radiation carcinogenesis for inhaled radon decay products, *Health Physics*,68,2, p.158 1995.

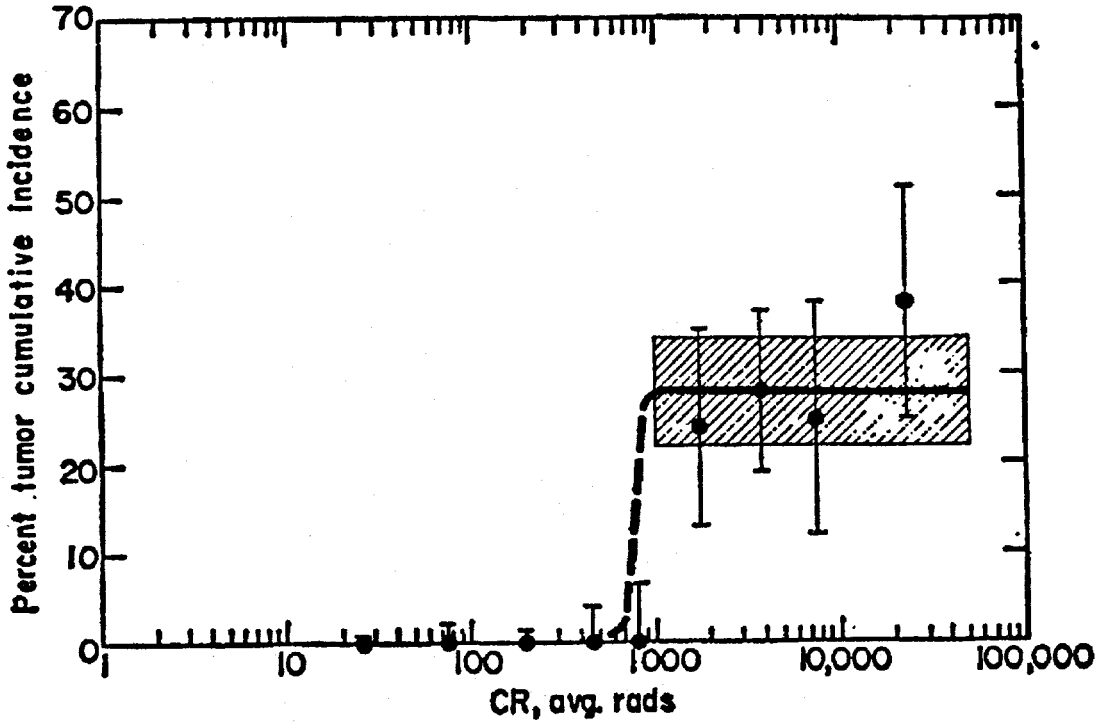


Fig. 5. Radium dose response data does not agree with the LNT model.

Selected references showing a downward trend in the exposure-response relation

1. Craig,L.; Seidman,H.
Leukemia and lymphoma mortality in relation to cosmic radiation.
Blood 17 : 319, 1961.

2. Frigerio,N.A.; Ekerman,K.F.; Stowe,R.S.
The Argonne Radiological Impact Program (ARIP), Part I.
Carcinogenic Hazard from Low-Level, Low-Rate Radiation;
ANL/ES-26 Part I, Environmental and Earth Sciences, Sept. 1973.

3. Frigerio,N.A.; Stowe,R.S.;
Carcinogenic and genetic hazard from background radiation.
IAEA Symposium, Biological and Environmental Effects of Low Level Radiation, vol. 2, pp 285-289, Vienna, 1976.

4. Spalding,J.F.; Thomas,R.G.; Tietjen,G.L.;
Life span of C57 mice as influenced by radiation dose, dose rate and age at exposure.
Report UC-48, LA 9528, Los Alamos National Laboratory, Los Alamos, 1982.

5. Abbat,J.D.; Hamilton,T.R.; Weeks,J.L.
Epidemiological studies in three corporations covering the Canadian nuclear fuel cycle.
pp 351-361, Biological Effects of Low Level Radiation. IAEA-STI/PUB 646, International Atomic Energy Agency, Vienna, 1983.

6. Haynes,R.M.
The distribution of domestic radon concentrations and lung cancer mortality in England and Wales.
Rad. Prot. Dosim., 25, 2, pp 93-96 1988.

7. Gilbert,E.S.; Fry,S.A.; Wiggs,L.D.; Voelz,G.L.; Peterson,G.R.
Analysis of combined mortality at the Hanford site, Oak Ridge National Laboratory, and Rocky Flats nuclear weapons Plant.
Radiation Research, 120 : 19, 1989.

8. Wei,L.X.; Zha,Y.R.; Tao ,Z.F.; He,W.H.; Chen,D.Q.; Yuan,Y.L.
Epidemiological investigation of radiological effects in high background radiation areas of Yangjiang, China.
Journal of Radiation Research, 31, 1, pp 119-136, 1990.

9. Dousset, M.
Radon in dwellings.
Aerobiologica 6:36-38, 1990

10. Nambi,K.S.V.; Soman,S.D.
Further observations on environmental radiation and cancer in India.
Health Physics, 59, 3, pp 339-344, 1990.

11. Wing,S.; Shy,C.M.; Wood,J.L.; Wolf,S.; Cragle,D.L.; Frome,E.L.
Mortality Among Workers at Oak Ridge National Laboratory.
JAMA, 265, No. 11 p. 1397, March 20, 1991

12. Jablon,S.; Hrubec,Z; Boice,Jr.;J.D.
Cancer in Populations Living Near Nuclear Facilities.
JAMA, 265, No. 11 p. 1403, March 20, 1991.

13. Chen,D.; Wei,L.
Chromosome aberration, cancer mortality and hormetic phenomena among inhabitants in areas of high background radiation in China.
Journal of Radiation Research, 32 Suppl. 2, pp 46-53, 1991.

14. Matanoski,G.M.
Health effects of low level radiation in shipyard workers.
E 1.99 DOE/EV/10095-T1 and T2, DOE Washington, 1991.

15. Kendall,G.M.; Muirhead,C.R.; MacGibbon,B.H.; et al.
Mortality and occupational exposure to radiation; first analysis of the National Registry for Radiation Workers.
Brit. Med. J. 304:220, 1992.

16. Shihab-Eldin,A.; Shlyakhter,A.; Wilson,R.
Is There a Large Risk of Radiation? A Critical Review of Pessimistic Claims.
Environment International, 18, pp. 117-151, 1992.

17. Latarjet, R.
Radiation carcinogenesis and radiation protection.
Cancer J., 5, pp 23-27, 1992.

18. Sohei Kondo
Health Effects of Low-Level Radiation, Kinki University Press, Osaka, and Medical Physics Publishing Co., Madison, WI., 1993.

19. Biological effects of low level exposures : dose-response relationships.
Edward J. Calabrese, editor. Boca Raton : Lewis Publishers, c1994.

20. Cohen,B.L.
Test of the linear-no threshold theory of radiation carcinogenesis for inhaled radon decay products. Health Physics, 68, 2, pp 157-174; 1995.

Selected references showing a threshold in the exposure-response relation.

21. Evans, Robley D.
Radium in man.
Health Physics, 27, pp 497-510, 1974.

22. Schull,W.J.; Shimizu,Y.; Kato,H.
Hiroshima and Nagasaki: New doses, risks, and their implications
Quotation from page 73.
"The lowest specific absorbed dose at which unequivocal effects can be demonstrated among the A-bomb survivors is 0.20-0.49 Gy."
Health Physics, 59, 1, pp. 69-75 1990.

23. Shimizu,Y.; Kato,H.; Schull,W.J.; Mabuchi,R.
Dose response analysis among atomic-bomb survivors exposed to low-level radiation.
pp 71-74 in Sugahara,T.; Sagan,L.A.;Aoyama,T., eds, Low Dose Irradiation and Biological Defense Mechanisms, Excerpta Medica, New York, 1992.

24. Thomas,R. G.
The US radium luminisers: A case for a policy of 'below regulatory concern'.
J. Radiol. Prot., 14, 2, pp 141-153, 1994.

25. Bond,V.P.; Benary,V.; Sondhaus,C.A.
A Different Perception of the Linear Non-threshold Hypothesis for Low-Dose Radiation.
Proc. Natl. Acad. Sci. USA, Vol. 88, pp. 8666-8670, October 1991.

26. Bond, V.P., Benary, V. Sondhaus, C.A., and Feinendegen, L.E.
The Meaning of Linear Dose-Response Relations, Made Evident by Use of Absorbed Dose to the Cell. Health Physics, v68, no.6, pp 786-792, 1995.

27. Excerpt from: Federal Register 56 (138) 33050-127,1991
"The Scientific Advisory Board (SAB)/Radiation Advisory Committee (RAC) urged the Environmental Protection Agency (EPA) to base its risk assessment for radium on human epidemiology data on radium watch dial painters, rather than on modeled estimates, and urged EPA to present its rationale for adopting the modeling approach for radium risk assessment. The SAB/RAC also requested that EPA better describe its dosimetric model in the revised criteria document, including calculated doses and risks to organs, and that if EPA continued to use the modeling approach, uncertainties in the modeling be addressed.
EPA Reply:
The Agency carefully reconsidered this issue. First it should be pointed out that all risk estimates are based on both epidemiologic data and require mathematical modeling. The EPA uses the wealth of epidemiologic data on human exposure and risk of radiogenic cancers, including radium dial painters and epidemiologic data on bone sarcomas resulting from injection of Ra-224.
The watch dial painter data indicate that the incidence of bone sarcomas may follow a dose-squared response, especially at higher exposures. EPA policy, supported by recommendations of SAB/RAC, is to assess cancer risks from ionizing radiation as a linear response. Therefore, use of the dial painter data requires either deriving a linear risk coefficient from significantly non-linear exposure-response data, or abandoning EPA policy and SAB/RAC advice in this case."

Selected References to the Genetic Repair of Radiation Damage:

Ruiz de Almodovar J.M.; Bush C.; Peacock J.H.; Steel G.G.; Whitaker S.J.; McMillan T.J.
Dose-rate effect for DNA damage induced by ionizing radiation in human tumor cells.
Radiation Research, 1994 Apr, 138(1 Suppl):S93-6.

Shadley J.D.
Chromosomal adaptive response in human lymphocytes.
Radiation Research, 1994 Apr, 138(1 Suppl):S9-12.

Joiner M.C.
Evidence for induced radioresistance from survival and other end points: an introduction.
Radiation Research, 1994 Apr, 138(1 Suppl):S5-8.

Nelson W.G.; Kastan M.B.
DNA strand breaks: the DNA template alterations that trigger p53-dependent DNA damage response pathways.
Molecular and Cellular Biology, 1994 Mar, 14(3):1815-23.

Caldecott K.W.; McKeown C.K.; Tucker J.D.; Ljungquist S.; Thompson L.H.
An interaction between the mammalian DNA repair protein XRCC1 and DNA ligase III.
Molecular and Cellular Biology, 1994 Jan, 14(1):68-76.

Joiner M.C.
Induced radioresistance: an overview and historical perspective.
International Journal of Radiation Biology, 1994 Jan, 65(1):79-84.

Kurimasa A.; Nagata Y.; Shimizu M.; Emi M.; Nakamura Y.; Oshimura M.
A human gene that restores the DNA-repair defect in SCID mice is located on 8p11.1-->q11.1.
Human Genetics, 1994 Jan, 93(1):21-6.

Kovacs M.S;. Evans J.W.; Johnstone I.M.; Brown J.M.
Radiation-induced damage, repair and exchange formation in different chromosomes of human fibroblasts determined by fluorescence in situ hybridization.
Radiation Research, 1994 Jan, 137(1):34-43.

Caldecott K.W.; McKeown C.K.; Tucker J.D.; Ljungquist S.; Thompson L.H.
An interaction between the mammalian DNA repair protein XRCC1 and DNA ligase III.
Molecular and Cellular Biology, 1994 Jan, 14(1):68-76.

Leadon S.A.; Cooper P.K.
Preferential repair of ionizing radiation-induced damage in the transcribed strand of an active human gene is defective in Cockayne syndrome.
Proceedings of the National Academy of Sciences of the United States of America, 1993 Nov 15, 90(22):10499-503.

Wolff S.; Afzal V.; Jostes R.F.; Wiencke J.K.
Indications of repair of radon-induced chromosome damage in human lymphocytes: an adaptive response induced by low doses of X-rays.
Environmental Health Perspectives, 1993 Oct, 101 Suppl 3:73-7.

Hafezparast M.; Kaur G.P.; Zdzienicka M.; Athwal R.S.; Lehmann A.R.; Jeggo P.A.
Subchromosomal localization of a gene (XRCC5) involved in double strand break repair to the region 2q34-36.
Somatic Cell and Molecular Genetics, 1993 Sep, 19(5):413-21.

Liu P.; Siciliano J.; White B.; Legerski R.; Callen D.; Reeders S.; Siciliano M.J.; Thompson L.H.
Regional mapping of human DNA excision repair gene ERCC4 to chromosome 16p13.13-p13.2.
Mutagenesis, 1993 May, 8(3):199-205.

Lutze L.H.; Cleaver J.E.; Morgan W.F.; Winegar R.A.
Mechanisms involved in rejoining DNA double-strand breaks induced by ionizing radiation and restriction enzymes.
Mutation Research, 1993 May, 299(3-4):225-32.

Zasukhina GD; Lvova GN; Vasileva IM; Sinelshchikova TA; And Others.
Adaptive Repair Induced By Small Doses Of Gamma-Radiation In DNA-Repair Deficient Human Cells.
Doklady Akademii Nauk, 1993 Apr, V329 N5:658-660.
Language: Russian.

Price A.
The Repair Of Ionising Radiation-Induced Damage To DNA.
Seminars In Cancer Biology, 1993 Apr, V4 N2:61-71.

Koudela K; Ryznar L; Kozubek S; Slotova J.
Induction Of Sos Repair By Ionizing Radiation - Results From Experiments At Accelerators.
Radiation And Environmental Biophysics, 1992 Oct, V31 N4:343-348.

Bases R; Franklin WA; Moy T; Mendez F.
Enhanced Excision Repair Activity In Mammalian Cells After Ionizing Radiation.
International Journal Of Radiation Biology, 1992 Oct, V62 N4:427-441.

Caldecott K.W.; Tucker J.D.; Thompson L.H.
Construction of human XRCC1 minigenes that fully correct the CHO DNA repair mutant EM9.
Nucleic Acids Research, 1992 Sep 11, 20(17):4575-9.

Wolff S.
Failla Memorial Lecture. Is radiation all bad? The search for adaptation.
Radiation Research, 1992 Aug, 131(2):117-23.

Yoo H. Li L.; Sacks P.G.; Thompson L.H.; Becker F.F.; Chan JY.
Alterations in expression and structure of the DNA repair gene XRCC1.
Biochemical and Biophysical Research Communications, 1992 Jul 31, 186(2):900-10.

Jeggo P.A.; Hafezparast M.; Thompson A.F.; Broughton B.C.; Kaur G.P.; Zdzienicka M.Z.; Athwal R.S.
Localization of a DNA repair gene (XRCC5) involved in double-strand-break rejoining to human chromosome 2.
Proceedings of the National Academy of Sciences of the United States of America, 1992 Jul 15, 89(14):6423-7.

van Loon A.A.; Timmerman A.J.; van der Schans G.P.; Lohman P.H.; Baan R.A.
Different repair kinetics of radiation-induced DNA lesions in human and murine white blood cells.
Carcinogenesis, 1992 Mar, 13(3):457-62.

Wolff S.; Jostes R.; Cross F.T.; Hui T.E.; Afzal V.; Wiencke J.K.
Adaptive response of human lymphocytes for the repair of radon-induced chromosomal damage.
Mutation Research, 1991 Sep-Oct, 250(1-2):299-306.

James SJ; Enger SM; Makinodan T.
DNA Strand Breaks And DNA Repair Response In Lymphocytes After Chronic Invivo Exposure To Very Low Doses Of Ionizing Radiation In Mice.
Mutation Research, 1991 Jul, V249 N1:255-263.

Thompson L.H.; Wu R.W.; Felton J.S.
Introduction of cytochrome P450IA2 metabolic capability into cell lines genetically matched for DNA repair proficiency/deficiency.
Proceedings of the National Academy of Sciences of the United States of America, 1991 May 1, 88(9):3827-31.

Thompson LH.
Properties and applications of human DNA repair genes.
Mutation Research, 1991 Apr, 247(2):213-9.

Wolff S.
Biological dosimetry with cytogenetic endpoints.
Progress in Clinical and Biological Research, 1991, 372:351-62.

Thompson L.H.; Brookman K.W.; Jones N.J. Allen S.A.; Carrano A.V.
Molecular cloning of the human XRCC1 gene, which corrects defective DNA strand break repair and sister chromatid exchange.
Molecular and Cellular Biology, 1990 Dec, 10(12):6160-71.

Dillehay LE.
A Model Of Cell Killing By Low-Dose-Rate Radiation Including Repair Of Sublethal Damage, G2 Block, And Cell Division.
Radiation Research, 1990 Nov, V124 N2:201-207.

Wolff S.; Afzal V.; Olivieri G.
Inducible repair of cytogenetic damage to human lymphocytes: adaptation to low-level exposures to DNA-damaging agents.
Progress in Clinical and Biological Research, 1990, 340B:397-405.

Troelstra C.; Odijk H.; de Wit J.; Westerveld A.; Thompson L.H.; Bootsma D.; Hoeijmakers J.H.
Molecular cloning of the human DNA excision repair gene ERCC-6.
Molecular and Cellular Biology, 1990 Nov, 10(11):5806-13.

Powell S; Mcmillan TJ.
DNA Damage And Repair Following Treatment With Ionizing Radiation.
Radiotherapy And Oncology, 1990 Oct, V19 N2:95-108.
Pub Type: Review.

Weber C.A.; Salazar E.P.; Stewart S.A.; Thompson lL.H.
ERCC2: cDNA cloning and molecular characterization of a human nucleotide excision repair gene with high homology to yeast RAD3.
Embo Journal, 1990 May, 9(5):1437-47.

Regan J.D.; Thompson L.H.; Carrier W.L.; Weber C.A. Francis A.A.; Zdzienicka M.Z.
Cyclobutane-pyrimidine dimer excision in UV-sensitive CHO mutants and the effect of the human ERCC2 repair gene.
Mutation Research, 1990 May, 235(3):157-63.

Calkins J.; Harrison W.; Einspenner M.
Reactivation of neutron killed mammalian cells by gamma irradiation: the observations, possible mechanism and implication.
Strahlentherapie und Onkologie, 1990 Jan, 166(1):22-5.

Liu SZ; Cai L; Sun JB.
Effect Of Low-Dose Radiation On Repair Of DNA And Chromosome Damage.
Acta Biologica Hungarica, 1990, V41 N1-3:149-157.