Attachment to:

COMMENTS OF RADIATION, SCIENCE & HEALTH, INC. ON EPA's PROPOSED RULE ENTITLED "NATIONAL PRIMARY DRINKING WATER REGULATIONS; RADIONUCLIDES; NOTICE OF DATA AVAILABILITY," 65 FED. REG. 21,575 (April 21, 2000)

A. Animal and Plant Biology

Hundreds of scientifically valid studies in the peer-reviewed scientific literature have demonstrated that low level radiation produces beneficial health effects, or no health effects in animal and plant populations and in biological experiments. Dr. Hugh Henry, then at Oak Ridge National Laboratory summarized the scientific literature on low dose health effects. Henry, H. F. (1961) Is all nuclear radiation harmful?, J. Am. Med. Assoc., 176, 671 see Data Document §1.3 p.1; and 1.3.1 p.10; and the full paper attached. Dr. Henry states, inter alia:

> **"Internal Exposure - An evaluation of the effects of internal exposure from any radioactive material is complicated by the non-radioactive but toxic effects of the material itself. (Finkel 1953, 1958, 1959) has made the most extensive studies, and her work involved injecting 70-day-old mice with compounds of various alpha and beta emitters.**
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> **"With alpha emitters, she obtained life-lengthening for injections of plutonium 239 of 0.34 mc per kilogram of body weight, and less; polonium-210 of about 1.5 mc per kilogram of body weight, and less; and uranium-233 of about 2.0 mc per kilogram, and less. The maximum life extension was 14% for plutonium-239, 4% for uranium-233, and 7% for polonium-210. Life extension was not noted for radium-226, although there was little change for doses of as much as 12 mc per kilogram of body weight. Life-shortening effects were found at all levels down to 1.3 mc per kilogram of body weight for strontium-90 and 16 mc per kilogram of body-weight for calcium-45. In experiments with a mixture of strontium-90 chloride and yttrium-90 chloride, she stated that the lowest dose at which a statistically significant life-shortening effect was observed in the mouse was 44 mc per kilogram of body weight, this representing an end-of-life deposit of about 5 mc per kilogram of body weight."**
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> **"Rats fed uranium compounds by Hodge and co-workers gave indication of life-lengthening at low levels of ingestion and life-**

shortening at higher levels, the effect depending strongly upon the compound used (Voegtlin and Hodge 1953) Diets including as much as 20% of UO_2 or UF_4 have a possible slight life-lengthening effect, and there is a definite life-lengthening with diets of less than 0.05% of $U0_2F_2$ and 1.0% of $U0_2$ $(NO_3)_2$. $6H_20$. It was also observed in these studies that rats ingesting low levels of uranyl nitrate of about 0.1% of their diet, and less, for some 2 years actually had healthier-appearing kidneys than did their controls."

These studies and other equivalent studies clearly refute the premise that small amounts of internal radionuclides, that far exceed typical natural background levels, have an adverse effect on health.

Prof. Emeritus Don Luckey, Department of Biochemistry in the U. Missouri School of Medicine, summarized more than 2000 studies that demonstrate beneficial effects from "whole-body" doses, not including beneficial effects from organ doses. Luckey, T.D. (1991) Radiation Hormesis, CRC Press, Boca Raton, FL. see Data Document §1.3.1 p.4-8 (Errata: correct header ref from "1994" to "1991.") and Luckey, T.D. (1980) Hormesis with ionizing radiation, Boca Raton, FL: CRC Press. These results are summarized in the peer-reviewed literature in Luckey, T.D. (1982) Physiological Benefits from Levels of Ionizing Radiation, Health Physics. 43, pp771-789. see the full paper enclosed with these comments.)

Dr. Luckey reports on work by Egon Lorenz of the National Cancer Institute, and many others at the national laboratories and universities supported by the AEC Biology and Medicine programs. These studies demonstrate the beneficial effects of low-dose radiation exposures that include: reduced cancer incidence and mortality, increased mean life span, increased growth rates, increased size and weight, increased fertility and reproduction, and reduced mutations, along with enhanced physiological and biological functions. Luckey, T.D. (1995b) Live in harmony with ionizing radiation, In: Biological Effects of Low Level Ionizing Radiation and Molecular Biology Research (Z. Zu and S.Z. Liu, eds.). Norman Bethune Univ. Med. Sci., Changchun, pp40-7, 1. see Data Document § 1.3.1 p.3)

Dr. Luckey states that:

"The beneficial effect of low dose irradiation was discovered 100 years ago at the University of Missouri. Professor W. Shrader (1896) inoculated Guinea pigs with diphtheria bacillus. Unexposed controls died within 24 hours. When animals were exposed to X-rays before inoculation, they survived."

Dr. Luckey also presents a substantial summary of animal studies in Luckey, T.D (1984) Hormesis with High LET Radiation Induced Cancer, Z. Phys. Med. Baln. Med. Klim., (Sonderheft 1) Vol. 13, pp11-16. see Data Document § 1.3.1 p.21)
With respect to radionuclide ingestion, Dr. Luckey specifically reports that:

"Nishio et al, (1967) found that mice watered with 0.1 Ci 137Cs and 0.4 Ci 90Sr/L through several generations were more resistant than controls to Ehrlich acites tumor transplants."

Dr. Luckey reports that studies that fail to demonstrate beneficial effects are largely the result of using hybrid animals with deficient immune systems, keeping animals germ-free, and even studies that discard controls with early mortality. The physiological responses in animals and plants are shown to be equivalent to the effect of many natural elements and compounds that are essential nutrients at low levels and toxic at high levels. Studies directed by radiation protection interests selectively ignored work and led to defunding of research to document beneficial effects.

Professor Emeritus of the Central Laboratory for Radiological Protection of Poland, and Member and Former Chairman of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), Dr. Zbigniew Jaworowski, reports on early animal studies with internal radionuclides. Jaworowski, Z. (1995b) Stimulating effects of ionizing radiation: New issues for regulatory policy, Regulatory Toxicology and Pharmacology, 22:2. see Data Document § 1.4 p.1; and the full paper enclosed with these comments.

Dr. Jaworowski states that:

"In 1943, during the early stages of the Manhattan Project, it was found that the animals exposed to inhalation of uranium dust at levels that were expected to be fatal actually lived longer, appeared healthier, and had more offspring than the noncontaminated control animals. For years, these results were treated as an anomaly but later studies produced similar results (Brucer 1989). The first UNSCEAR report to the General Assembly of the United Nations presented the results of experiments showing longer survival times of mice and guinea pigs exposed to small doses of gamma radiation (UNSCEAR 1958)."

In the review of reduced cancer in the high radioactivity area of Kerala India, Drs. Balaram and Mani of the Regional Cancer Center of Kerala India document the literature on the lack of adverse effects on organisms and animals. Balaram, P. and Mani, K.S. (1994) Review Article: Low dose radiation A curse or a boon?, Nat. Med. J. India, 7, 4. see Data Document §1.3 p.3.

Drs. Sacher and Trucco of Argonne National Laboratory present results showing improved biological performance and survival from low-dose radiation. Sacher, G.A. and Trucco, E. (1966) A theory of the improved performance and survival produced by small doses of radiations and other poisons.

In addition, NO substantial or reproducible studies that demonstrate adverse health effects to plants and animals have been identified!

The LNTH can not be supported, and is demonstrated to be invalid, by such

consistent radiation health effects data despite limited research funding support and constraints on publication.

Specific studies of animal experiments have shown beneficial effects of low-dose radiation. For example, experiments by Dr. Egon Lorenz of the National Cancer Institute show the lack of adverse effects, and increased longevity. Lorenz, E. (1954) Biological Effects of External Gamma Radiation, Part I, (R. E. Zirkle, ed.), McGraw-Hill, New York, p24. see Data Document § 1.3.1 p.1; and Lorenz, E. (1950b) Some Biologic Effects of Long-Continued Irradiation, National Cancer Institute, Bethesda, Maryland, Feb 1950, pp176-185. see Data Document § 1.3.1 p.1-3)

Dr. Harold Boxenbaum presents evidence on mammalian aging, toxicity, and longevity hormesis. Boxenbaum, H. (1992) Hypothesis on Mammalian Aging, Toxicity, and Longevity Hormesis: Explication by a Generalized Gompertz Function Biological Effects of Low Level Exposures to Chemicals and Radiation, Lewis Publishers, Chelsea, Michigan, pp1-39. see Data Document § 1.3.1 p.9.

Dr. Boxenbaum states:

> **"Further support that radiation produces longevity hormesis is supplied... (I)n this case, the data deal with chipmunks living in the wild. The animals were live-trapped, irradiated with either a single-dose of 200 or 400 Roentgens gamma-radiation, except for controls, and then returned to the wild. It is readily apparent that gamma-radiation exposure, within the dose-range utilized, enhanced longevity."**

Dr. Ishii and colleagues in Japan report on animal experiments confirming applications to successfully treat cancer in humans. Ishii, K., Hosoi, Y., Yamada, S., Ono, T. and Sakamoto, K. (1996) Decreased Incidence of Thymic Lymphoma in AKR Mice as a Result of Chronic, Fractionated Low-Dose Total-Body X Irradiation, Rad Res., Vol. 146, No. 5, p582. see Data Document § 1.3.1 p.9.

Dr. Ishii states in the abstract:

> **"Male AKR mice were irradiated with 5 cGy three times a week or 15 cGy two times a week from 11 weeks of age for 40 weeks. The incidence of thymic lymphoma was 80.5% in sham-irradiated mice, 67.5% in mice irradiated with 5 cGy three times a week and 48.6% in mice irradiated with 15 cGy twice a week."**

Drs. Yoshio Hosoi and Kiyohiko Sakamoto of the Tohoku University School of Medicine document the effects of total body irradiation to suppress metastasis. Hosoi, Y. and Sakamoto, K. (1997) Suppression of spontaneous and artificial metastasis by low dose total body irradiation in mice, In: Low Doses of Ionizing Radiation: Biological Effects and Regulatory Control, IAEA-TECDOC-976, IAEA-CN-67/132, pp424-427. see Data

Dr. U. Yamamoto of the Faculty of Life Science at the Yasuda Women College in Hiroshima and Dr. T. Seyama of the Radiation Effects Research Foundation, Hiroshima demonstrated that ingestion of tritium in tritiated water by mice significantly reduced the tumor frequency. Yamamoto, O. and Seyama, T. (1997) Threshold Dose-Rate Observed by Administration of Tritiated Water in Mice for Radiation Risk, In: Low Doses of Ionizing Radiation: Biological Effects and Regulatory Control, IAEA-TECDOC-976, IAEA-CN-67/68, pp243-245. see Data Document § 1.3.1 p.12)

In reports on the effects on animals in high radiation areas, Dr. P.C. Kesavan in India reports on the lack of effects in rats living in the high background area for 800-1000 generations. Kesavan, P.C. (1997a) Indian research on high levels of natural radiation: pertinent observations for further studies, In: Elsevier Science B.V, High Levels of Natural Radiation, Radiation Dose and Health Effects, pp111-117. see Data Document § 1.3.1 p.12.

Also, Dr. M. Delpoux and colleagues in France and Belgium report on experiments with rabbits that show no adverse effects, and increased fertility in male rabbits consistent with hormesis, exposed to doses from the high background areas of France. Delpoux, M., Leonard, A., Dulieu, H. and Dalebroux, M. (1997) Experimental study of the genetic effects of high levels of natural radiation in South-France, In: High Levels of Natural Radiation-Radiation Dose and Health Effects, pp397-406. see Data Document § 1.3.1 p.12.

In a recent experiment with mice in France, Caratero A, Courtade M, Bonnet L, Planel H, Caratero C of the Laboratoire d'Histologie-Embryologie-Cytogenetique, Faculte de Medecine Toulouse-Rangueil, exposed groups of 300 mice each to radiation doses from thorium at background, at 7 cGy/yr, and at 14 cGy/yr and found that the exposed groups lived significantly longer than the group exposed to background radiation. Caratero, A., Courtade, M., Bonnet, L., Planel, H., and Caratero, C., (1998) Effect of a continuous gamma irradiation at a very low dose on the life span of mice, Gerontology; 44(5):pp272-6. see Data Document § 1.3.1 p.15.

Drs. E. Hahn and W. Ward of the Department of Radiation Biology and Biophysics, University of Rochester School of Medicine and Dentistry, report in 1967 that rats X-irradiated before mating have no adverse effect on reproductive factors up to 50 cGy. Hahn, E.W. and Ward, W.F. (1967) Increased litter size in the rat: X-irradiated during the estrous cycle before mating, Science, 157, pp956-957. see Data Document § 1.3.1 p.16.

Drs. John 'Jake' Spalding, Robert Thomas, and Gary Tietjen, of Los Alamos National Laboratory, document a life span study of mice to measure life-shortening as a function of dose, dose-rate, and age, in two replicates of almost 4,000 mice. The study, reported in 1982, show increased longevity of exposed mice except at the extreme doses: Spalding, J. F., Thomas, R. G. and Tietjen, G. L. (1982) Life span of C57 mice as influenced by

radiation dose, dose rate, and age at exposure, Los Alamos National Laboratory, Report LA-9528 October 1982. see Data Document § 1.3.1 p.16.

Dr. Spalding reports:

> **"This study was designed to measure the life shortening of C57BL/6J male mice as a result of exposure to five external doses from 60Co gamma radiation delivered at six different dose rates. Total doses ranged from 20 to 1620 rad at exposure rates ranging from 0.7 to 36000 R/day. The ages of the mice at exposure were newborn, 2, 6, or 15 months. Two replications were completed."**

And:

> **"Most of the irradiated animals lived longer or no differently than did the non-irradiated controls; however, in several cases differences were significant. For newborn mice exposed to 180 rad at 0.7 R/day, the life span was significantly longer than it was for controls. At all dose levels the 2-month age group lived significantly longer than did the median controls. Although there were no differences among 6-month-old mice, the 15-month group with the 20-rad dose lived significantly longer than did their controls."**

Dr. Jean René Maisin and André Wambersie of the Université Catholique de Louvain in Brussels, Belgium and Drs. Georg B. Gerber and Jan Vankerkom from the Radiation Protection Unit, Mol, Belgium report survival and causes of mortality in irradiated mice. Maisin, J.R., Gerber, G.B., Vankerkom, J. and Wambersie, A. (1996) Survival and diseases in C57BL mice exposed to X Rays or 3.1 MeV Neutrons at an age of 7 or 21 days, Radiat. Res. 146, pp153~60. see Data Document § 1.3.1 p.25.

Dr. Maisin and colleagues state:

> **"Survival and causes of mortality were studied in 7- or 2l-day old male C57BL/Cnb mice exposed to 0.5, 1 or 3 Gy of 250 kVp X rays or 0.125, 0.25, 0.5 or 1 Gy of accelerator neutrons (modal energy 3.1 MeV). A total of 1287 animals were used in the experiments. Survival of irradiated animals was reduced significantly only in the mice receiving the highest doses (1 Gy neutrons, 3 Gy X rays). Mice exposed to the lowest doses (0.125 Gy neutrons, 0.5 Gy X rays) lived significantly longer than controls, apparently reflecting a reduction in non-neoplastic lung and liver diseases. All malignant tumors increased significantly from (and including) doses of 0.5 Gy neutrons and 1 Gy X rays. … Based on percentage life shortening, it appears that exposure during infancy does not shorten total survival or survival from cancer much more than exposure of adults…."**

Dr. Georges Monchaux and Jean-Paul Morlier of the Département de Radiobiologie et

Radiopathologie, in France, report that the influence of exposure-rate on lung cancer induction in rats at relatively low cumulative exposures, comparable to lifetime exposures in high-radon houses or current underground mining exposures, the risk of lung cancer in rats decreases with exposure rates. Monchaux, G. and Morlier, J.P. (1999) Lung cancer induction in rats after exposure to radon progeny :The complex interplay between cumulative exposure and exposure rate. In: Proceedings on "The Effects of Low and Very Low Doses of Ionizing Radiation on Human Health," World Council of Nuclear Workers, June 16-18, St. Quentin en Yvelines, Versailles, France. Elsevier (in press). see Data Document § 1.3.1 p.28.

Dr. Monchaux and colleagues state:

> **"A trend towards increasing tumour risk with decreased exposure rate was observed in Sprague-Dawley rats… In contrast, the results obtained at low cumulative exposure, comparable to domestic indoor exposures showed no evidence of an inverse exposure-rate effect. Chronic radon exposure at 0.09 J h m^{-3} (25 WLM), protracted over a 18 months period, at a potential alpha energy concentration (PAEC) of 0.042 mJ m^{-3} (2 WL), resulted in fewer lung carcinomas in rats than a similar cumulative exposure protracted over 4 to 6 months at a PAEC of 2.1 mJ m^{-3} (100 WL). Moreover, the lung cancer incidence in rats exposed at low exposure rate (0.60%) was slightly lower than that in control animals (0.63%)… The significance of exposure rates in assessing the hazards of domestic radon exposure was addressed on biophysical grounds by Brenner, who concluded that, when cumulative exposures are sufficiently low that multiple traversals of target cells by alpha particles are rare - that is the case for typical domestic radon exposures -, all exposure-rate enhancement effects disappear. Our recent data in rats appear to follow the same trend."**

Research on paramecia by Dr. T.D. Luckey of the Dept. of Biochemistry, U. Missouri-Columbia School of Medicine, finds enhanced growth relative to organisms in normal background radiation from stimulation by ionizing radiation, and suppressed growth as a result of suppressing normal background radiation levels. Luckey, T.D. (1986) Ionizing Radiation Promotes Protozoan Reproduction, Rad. Res 108, pp215-221 see Data Document § 1.3.2 p.1

Dr. Luckey stated:

> **"Control populations [of T. pyriformis] increased from 200 to approximately 24,000/ml during 6 day incubation. The reproduction rate T. pyriformis was statistically lower ($P<0.01$) in subambient radiation than it was in near ambient radiation levels, 0.5 mrad/day (Fig. 3). Cultures irradiated at levels of 7.3 and 45 mrad/day reproduces faster ($P<0.01$) than did those at near ambient levels of radiation."**

Dr. Luckey had also reported additional similar results in Luckey, T.D., Johnson, W., Krueger, S., Tolo, D. and Vandenboom, B. (1978) Ionizing Radiation is Required for Optimum Reproduction in Paramecium bursaria. see Data Document § 1.3.2 p.5.

Dr. H. Planel and colleagues at the Laboratoire de Biologie Medicale in France produced experiments in lower order animals, on the effect of both low- to moderate-exposure doses, and on suppression of natural background levels. Planel, H., Soleilhavoup, J.P., Tixador, R., Conter, A., Croute, F. Caratero, C. and Gaubin, Y. (1987) Influence on Cell Proliferation of Background Radiation or Exposure to Very Low, Chronic g Radiation, Health Physics, Pergamon Press, N.Y. Vol. 52, No. 5, pp 571-578. see Data Document § 1.3.2 p.1.

Dr. D. Sugg and colleagues reported on the thriving aquatic animals exposed to the radionuclides at the site of the Chernobyl accident, and with reference to the Savannah River site. Sugg, D.W., Bickham, J.W., et al (1996) DNA Damage and radiocesium in channel catfish from Chernobyl, Environmental Toxicology and Chemistry, Vol. 15, No. 7, pp1057-1063. see Data Document § 1.3.2 p.6.

These studies consistently find that a continuum exists for stimulation by radiation. This includes deleterious effects from reducing radiation levels below normal background, with beneficial effects at multiples of background radiation, up to a level orders of magnitude above background at which the organisms demonstrate deleterious effects from high doses.

In health and medical research such results lead to research to establish the basis for vitamin, mineral, and other supplements to provide for nutrition and health. Such radiation research has been constrained.

It is also clear that many studies do not show null responses or hormetic effects. These studies generally do not include the low dose and dose-rate ranges and conditions of interest. This is largely due to radiation protection research bias to assess high doses to support radiation protection standards. However, "accidental" null and hormetic effects in animal studies are not considered in maintaining the LNT as the basis for costly radiation protection policies, and to support research directed to these ranges.

B. Cellular and Molecular Biology, Genetics, and Cancer Research

Drs. J.F. Townsend and T.D. Luckey, in the Dept. of Biochemistry of the U. Missouri-Columbia School of Medicine, reported in 1960 on the biological basis for hormesis. Townsend, J.F. and Luckey, T.D. (1960) Hormologosis in pharmacology, J. Am. Med. Assoc., 173: pp44-48. see Data Document § 1.4 p.1

Drs. Townsend and Luckey state:

"While studying fermentation in milk, Richert (1906) noted that heavy metals were stimulatory at concentrations lower than those which gave the harmful "oligodynamic action." Schulz (1888) and Branham (1929) have shown that most of the classic bactericidal agents exhibit hormesis in yeast. Antibiotics frequently cause a zone of accelerated growth in bacteriological assay work. Garrod's evidence (1951) indicates that this may have a direct effect on the cells. The antibiotic growth stimulation of laboratory and farm animals has recently been reviewed in full (Luckey 1959a)."

"Since pharmacology is the department of medicine which deals most directly with such chemical effects on cells and tissues, a survey of hormetics is appropriate in this field. From the generalized viewpoint of hormology, a study of the action of a variety of drugs in animal systems may add further evidence to the validity of the thesis and illustrate a common denominator in drug action. Goodman and Gilman (1955) recognize the phenomenon of hormesis as being of a general nature as is shown by the following statement: 'Quinine affects such a large variety of biological systems that it has been called a general protoplasmic poison; with some reservations this appraisal is probably correct. Like many other poisons of this sort, it may stimulate in low concentrations but depress in higher concentrations.'

"A possible mechanism of action at the cellular level has been suggested (Luckey 1959b) as follows: The stimulation reflects limitations in the ability of the organism to equate or modulate its response to a given stimulus at the lowest threshold of perception. If we assume that the response involves a chemical reaction, then the response of the organism is a unit (discontinuous) response, in which the lowest possible reaction would require one or more molecules to be changed. The release of an enzyme, proenzyme, hormone, or ribonucleic acid information molecule could quickly change the internal character of cells. The minimum response is an apparent overcompensation at the sensing threshold of the organism.

"The complexities of higher organisms lead to interactions between different cells and tissues. This allows a more complex reaction mechanism to be visualized. In spite of this, the over-all patterns of response are similar to those seen with micro-organisms. Irrespective of mechanism, common denominators evidently exist in the response of organisms to drugs at the threshold of perception and response levels."

"The fact that so many apparently unrelated stimuli produce the same general response (for example, stimulation followed by depression) at least suggests that there are a few fundamental processes by which the

cell responds to all such stimuli rather than myriad processes by which it responds to a wide variety of compounds. The complete pattern of drug-response patterns should be known. Demonstration of the uniformity of response in the face of diversity of stimulation …should point the way to a better understanding of drug action and allow some generalization in basic cellular physiology."

Professor Emeritus Dr. Zbigniew Jaworowski, Member and former Chairman of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and Head of the Central Laboratory for Radiological Protection in Poland, reports that UNSCEAR (1994) reviewed the most important publications on the stimulating effects of radiation ...effects were found at biochemical, cellular and organic levels, in cell cultures, bacteria, plants, and animals. Jaworowski, Z. (1995b) Stimulating effects of ionizing radiation: New issues for regulatory policy, Regulatory Toxicology and Pharmacology, 22:2. see Data Document § 1.5 p.1

Prof. Jaworowski states:

"UNSCEAR 1994 concentrates on the elucidation of mechanism by which radiation hormesis acts at the level of cell control systems such as protein synthesis, gene activation, DNA repair, stress-response protein production, radical detoxification, activation of membrane receptors, proliferation of splenocytes, and stimulation of the immune system."

Professor Emeritus Dr. Sohei Kondo reports on the role of apoptosis on the elimination of damaged cells. Kondo, S. (1988) Altruistic cell suicide in relation to radiation hormesis, Atomic Energy Research Institute, Kinki University, Osaka, Japan. Int. J. Radiat. Biol. Relat. Stud. Phys. Chem. Med. 53: pp95-102. See Data Document § 1.5 p.2.

Prof. Kondo states:

"The high radiosensitivity to killing of undifferentiated primordial cells (Bergonie and Tribondeau 1906) can be described as a manifestation of the suicide of injured cells for the benefit of an organism as a whole if their suicide stimulates proliferation of healthy cells to replace them, resulting in complete elimination of injury. This process is called cell replacement repair, to distinguish it from DNA repair which is rarely complete. 'Cell suicide', 'programmed death' and 'apoptosis' are terms used for the same type of active cell death. Cell suicide is not always altruistic. Altruistic suicide in Drosophila, mice, humans, plants, and E. coli is reviewed in this paper to illustrate its widely different facets. The hypothesis that in animals, radiation hormesis results from altruistic cell suicide is proposed. This hypothesis can explain the hormetic effect of low doses of radiation on the immune system in mice. In contrast, in plants, radiation hormesis seems to be mainly due to non-altruistic cell death."

"HORMESIS—'the stimulating effect of small doses of substances which in larger doses are inhibitory' (British Medical Dictionary Caxton Publ. Co., 1961)."

Dr. Shu-Zheng Liu, former President of Norman Bethune University, and Head of the Radiobiology Research Unit of Norman Bethune University and the Department of Health of China, and colleagues document the stimulatory effects of low dose radiation on immune functions. Liu, S.Z., Liu, W.H. and Sun, J.B. (1987) Radiation hormesis: its expression in the immune system, Health Phys 52:pp579-583. see Data Document § 1.5 p.3.

Dr. Liu and colleagues state:

"The effects of low-dose single and continuous whole-body irradiation on immune functions were studied in C57BL/6 mice. Plaque-forming cell reaction of the spleen was found to be stimulated by single doses of x rays in the range of 0.025 to 0.075 Gy and by continuous exposure to gamma rays with a cumulative dose of 0.065 Gy. The reactivity of thymocytes to interleukin 1 showed a dose-dependent depression in the dose range of 0.025 to 0.25 Gy, but there was an increase in cell number in the thymus between doses of 0.025 and 0.10 Gy, resulting in enhancement of reaction of the whole organ. Unscheduled DNA synthesis of spleen cells was stimulated by single irradiation with 0.05 Gy and continuous irradiation with a cumulative dose of 0.13 Gy. The implications of these immunologic changes under low-dose radiation are discussed."

Dr. Liu has more recently reported on extensive confirmations that low dose radiation stimulates immunological responses in bi-phasic modes for low doses vs. high doses. Liu, S.Z. (1997) Cellular and molecular basis of the stimulators effect of low dose radiation on immunity, In: Wei, L., Sugahara, T. and Tao, Z., High Levels of Natural Radiation 1996: Radiation Dose and Health Effects, Beijing, Elsevier, pp341-353. see Data Document § 1.5 p.8; and the full paper attached.

Dr. Liu states:

"It has been observed in human populations and animal studies that low dose radiation (LDR) could stimulate the immunological responses. The up-regulation of immunity following LDR involves a series of cellular and molecular reactions as well as their systemic regulation. The studies in our laboratory and elsewhere in recent years have convinced us that whole-body irradiation (WBI) with X- and g-rays in the dose range within 0.2 Gy has definite positive effect on the immune system which can be considered as beneficial to the organism."

Dr. T.D. Luckey also summarized the functions and the role of low-dose radiation on

the immune system. Luckey, T.D. (1991) Radiation Hormesis, CRC Press, Boca Raton, FL. see Data Document § 1.4 p.2-5.

Dr. Luckey summarizes existing immune response data as:

"Increased cell repair enzymes and enhanced immune competence are keys to understanding many physiologic effects of low doses of ionizing radiation. DNA repair enzymes are effective for exposures which are low enough to provide adequate time to repair one strand using the intact strand as a template; these function in most cells. (Frigerio 1976, Lesher 1967) Also important are repair of cell membranes, altered enzyme concentrations, and changed metabolic priorities.

"Immune functions which show radiation hormesis include radio-resistance, wound healing, resistance to infection, antibody formation, and lymphocyte proliferation, differentiation, and function. The net result is decreased debilitating infections and cancer from birth through midlife, and into senescence. This provides both increased quality of life and longer lifespan.

"The main organs of the immune system are the bone marrow which produces white blood cells, the thymus which hosts a reproductive frenzy for the maturation of lymphocytes into T cells, and centers for storage of debris, proliferation, and interaction of cells and products. The last includes spleen, bursa, tonsils, and other lymph nodes. Immunity also depends upon the white blood cells and their soluble products: antibodies (the humoral factor), lysozymes, opsinins, and small molecules of intercellular communication. Note that interaction of these important elements of the immune system are absent in radiation experiments with cells in culture."

"Exposure of mice to an acute dose of 0.5 Gy of X-radiation increased mitogen-stimulated proliferation of natural killer cells 145% above sham irradiated controls, p <0.001. (Liu 1989) Exposure of >4 Gy decreased natural kiIler cells. Similar differences in T cell reactions were found when young and old people from the high and low background areas of China were compared.

"Myeloid cells include several types of circulating white blood cells. Macrophages, or phagocytes, are important components of most tissues. For example, about 10% of cells in muscle are wandering macrophage cells. Macrophages search, identify, and engulf nonself intruders, including newly mutated cancer cells. They scavenge dead cells, particulates, and other waste, and transport such material to lymph nodes for storage or further processing. Their proteinaceous messages, left on cells not digested, help leukocytes to react to these intruders.

"Exposure of mice to 1 to 20 cGy of X ray-stimulated proliferation of

the bone marrow stem cells (Gidali 1979, Liu 85, Lorenz 1954, Martin 1955, Morris 1980, Murphy 1926, Pape 1950, 1951, Russ 1919, Sacchetti 1960, Thomas 1919, Trautmann 1953, Troup 1982, Zukhbaia 1989) his study with cancer transplantation, Murphy used 'stimulating' doses of X-rays with the knowledge that they gave increased circulating lymphocytes in blood." (Murphy 1915)"

"Irradiated individuals are protected from cancer induction by many different systems: increased total circulating leukocytes, reduction of antigen specific suppressor T cells, serum antigen-specific blocking factors, thymic hormones, and a variety of small molecules used as interleukocyte messengers. Cell mediated immune competence following whole-body exposure with single doses of 0.4 Gy in preimmunized mice has been studied extensively by the Hellstrom group. (Leon 1962, Lesher 1967) Tumors become established following the attachment of a layer of host antigen-antibody complex; this host facade on the surface of established tumors appears to fool the immune system into inactivity.

"Immune protection is most effective for small, newly initiated tumors. Growth inhibition, and even complete regression of newly transplanted tumors was reported following whole-body exposure of 4 Gy from 60-Co rays. (Hellstrom 1979)

"A clear example demonstrated some of the above interactions. (North 1982, 1985, 1986) Tumors were inserted intraperitoneally into four groups of mice. The tumors grew well in control mice and those exposed to 5 Gy of X-rays. They also grew well in mice injected intravenously with immune cells from mice which had the same tumor; these lymphocytes were activated toward this specific tumor. Presumably, the suppressor T cells of the host mice prevented effective action by these cells. When the immune cells were injected into irradiated mice, there were no host suppressor T cells to inhibit the helper T cells and the tumors were destroyed. Injection of lymphocytes from mice not bearing this tumor was not productive; there were no T cells specific for this tumor. A comparable strategy is being tried clinically."

> **"Following radiation damage, suppressor T cells are reduced in number and regenerate more slowly than helper T cells. This allows increased efficacy in the removal of newly formed cancers. Thus, the effects of whole body irradiation are well suited to the elimination of newly formed cancers. Cancer growth inhibition and even complete regression of transplanted tumors has been reported following whole-body exposure of mice and rats to 4 Gy gamma rays. (Anderson 1980a,b, 1982, 1988, Hellstrom 1979, 1983, North 1982, 1985, 1986, Westman 1923)"**

Dr. Luckey reports also that:

"The BEIR Committee accepted a threshold model for all physiologic effects except mutation and cancer; no decision was made for doses under 10 cGy (BEIR III, 1980). This committee ignored the fact that every major study on radiation-induced cancer, which utilized low doses whole-body exposure, produced some data showing that low and high exposures gave opposite results. The data consistently support hormesis in radiation-induced mutation."

Drs. E.I. Azzam, S.M. de Toledo, T. Gooding and J.B. Little, of the Department of Cancer Cell Biology, Laboratory of Radiobiology, Harvard School of Public Health, report that at doses where about 2% of the nuclei would be traversed by an α particle, induction of CDKN1A occurs in more cells than predicted. Furthermore, the induced cells are present in isolated aggregates of neighboring cells. Therefore, their studies at the gene expression level indicate that similar signaling pathways are induced in bystander cells that are not traversed by an a particle as in traversed cells, and that biological effects in cell populations are not restricted to the response of individual cells to the DNA damage they receive. This finding confirms that biological response can not be a linear function of damage to individual cells as a function of radiation dose. It also confirms the premise requiring intercellular communication and response that support the premise of whole tissue and organism response to enable stimulatory beneficial effects. Azzam, E.I., de Toledo, S.M., Gooding, T. and Little, J.B. (1998) Intercellular communication is involved in the bystander regulation of gene expression in human cells exposed to very low fluences of alpha particles, Radiat. Res. 150, pp497-504. see Data Document § 1.5 p.14.

Dr. Azzam and colleagues, then at the Chalk River Laboratories in Canada, found that initiation of cancer was reduced in experiments applying low doses of radiation to mouse embryo cells. Azzam, E.I., Raaphorst, G.P. and Mitchel, R.E.J. (1994) Occupational exposure to radiation induces an adaptive response in human lymphocytes, Int. J Radiat Biol. 1995 Feb;67(2):187-91

Dr. Azzam and colleagues state:

"We have monitored the end points of cellular survival, micronucleus formation and neoplastic transformation frequency to assess adaptation to ionizing radiation in the C3H 10T1/2 mouse embryo cell system. Plateau-phase cells were pre-exposed to an adapting dose of 0.1 to 1.5 Gy low-dose-rate gamma radiation 3.5 h prior to an acute challenge dose of 4 Gy. No adapting dose improved clonogenic survival detectably, whether the cells were plated immediately after the acute exposure or held in plateau phase for 3.5 h before plating. However, all chronic adapting doses resulted in both a reduction in micronucleus frequency in binucleate cells and about a twofold reduction in neoplastic transformation frequency per viable cell when cells were subsequently exposed to the 4-Gy challenge dose. Our data suggest that a low-dose-rate pre-exposure to ionizing

radiation induces an adaptive response in C3H 10T1/2 cells, and that this response enhances DNA double-strand break repair when cells are subsequently exposed to a second radiation dose. This enhanced repair appears to be error-free since these adapted cells are also less susceptible to radiation-induced neoplastic transformation."

Radiobiologist Prof. Emeritus Gunnar Walinder of Sweden reports on the current knowledge in biology that research on cancer at the level of the cell and tumor in whole organisms has established that carcinogenesis is a complex, iterative, progression that precludes the biological plausibility of the LNT as a plausible postulated stochastic "hit" to DNA that can progress to a cancer. This research rejects the proposition that a single hit on DNA that causes either a single- or double-strand break, with a presumed constant repair error rate, can lead to cancer. Walinder, G. (1987) Epistemological problems in assessing cancer risks at low radiation doses, Health Phys., 52, 5.

Biological evidence has established that 'whole' cell colonies and organisms have adaptive responses to radiation, for cells in which complex intracellular communications and responses are enabled, and for organisms in which immune responses are functional

Dr. Alexander Kuzin, Corresponding Member of the Academy of Sciences of Russia, Honorary Doctor of the Leeds University (England), State Prize Winner of the USSR (1987), Head of the Group of Radiational Biochemistry and Cellular Regulation, of the Institute of Biophysics, reports (1993) that: Kuzin, A.M., Ruda, V.P. and Mozgovoi, E.G. (1991) The role of receptors in radiation hormesis, Radiat. Environ. Biophys. 30, pp259-266. see Data Document § 1.4 p.6

Dr. Kuzin states:

"The different cellular responses to high (suppressive) and low (stimulant) doses of atomic radiation suggest understanding of radiation hormesis, since the well developed mechanisms of damaging effect of atomic radiation (radiodamage of DNA, chromosomal aberrations, death of radiosensitive cells) cannot explain the converse effects of low stimulant radiation doses. Here the direct or indirect excitation of membrane receptors comes to the foreground. The excitation activates membrane-bound enzymes which control many vitally important processes.

"Now that an increasing proportion of the general population is exposed to low chronic doses of ionizing radiation, the knowledge of radiation hormesis acquires great importance, particularly, for temporal predictions of its consequences. Although this problem is far from complete understanding, it is, undoubtedly, wrong to estimate the hazard of the low radiation doses by straight extrapolation of the data obtained with much higher doses during shorter time periods."

Drs. J. Smith-Sonneborn and Barbee, report that low dose radiation induces responses stress-induced protective proteins demonstrated in induced longevity in the Paramecium model system. Smith-Sonneborn, J. (1996) Heat shock proteins as an adaptive response: Oxidant and exercise induced stress response, 3rd BELLE Conference, Toxicological Defense Mechanisms and the Shape of Dose-Response Relationships. see Data Document § 1.4 p.1

Drs. J. Smith-Sonneborn and Barbee state:

> **"The global molecular response to stress includes a dramatic change in gene expression and elevated synthesis of heat shock or other stress-induced protective proteins. Stressors include heat, heavy metals, oxidants, bacterial and viral infection, and most recently, exercise. Oxidant damage and/or heat are major components in the induction of the adaptive protective response at appropriate challenge doses.**
>
> **"Radiation induces members of the heat shock family and the coordinated expression of antioxidant defenses. Exercise has been shown to induce both the cardioprotective heat shock proteins, antioxidants, and members of the ubiquitin family; (regulators involved in protein degradation, cell division, and differentiation).**
>
> **"The model system Paramecium was used to assess mechanisms involved in the beneficial effects of low doses of otherwise harmful agents; e.g. radiation induced increased longevity and peroxide induction of oxidative tolerance."**

Some cellular and molecular biology research that purports to support the LNT results from organisms and cell colonies in culture that fail to demonstrate biopositive responses because of the absence of the biological response capability.

Professor James Trosko, Department Of Pediatrics and Human Development, Michigan State University, and former Director of Research at RERF, and others, show that radiation damage effects only initiate at levels that exceed normal levels of oxidative damage; and that responses are triggered by intracellular signal transduction mechanisms that are epigenetic, not genotoxic in nature. As such, radiation doses sufficiently high to contribute to cancer are not the result of a toxic insult, but triggered by a non-stochastic epigenetic process. As long as damage frequencies are within the background rate of metabolic processes, which are factors of thousands to millions of times the natural radiation background rate, proliferation and adaptive functions in multicellular organisms regulate damaged cells through sharing reductants for repair and by triggering apoptosis. Biologically, cancer can not be caused by radiation at low doses. Trosko, J.E. (1996) Hierarchical/ Cybernetic Nature of Homeostatic Adaptation to Low Level Exposures to Oxidative Stress-inducing Agents. See Data Document § 1.5 p.4

Dr. Trosko states:

"The biological consequences to the low-level radiation which exceeds the background level of oxidative damage could be necrosis or apoptosis, cell proliferation or cell differentiation. These effects are triggered by oxidative stress-induced 'signal transduction' mechanisms, an epigenetic, not genotoxic, process. If these endpoints are not seen at frequencies above background levels in an organism, it is unlikely that low-level radiation would play a role in the multi-step processes of chronic diseases such as cancer. The mechanism linked to homeostatic regulation of proliferation and adaptive functions in a multicellular organism could provide protection of any one cell receiving deposited energy by the radiation tract through the sharing of reductants and by triggering apoptosis of target stem cells.

"Examples of the role of gap junctional intercellular communication in the 'adaptive response' of cells and the 'bystander' effect illustrate how the interaction of cells can modulate the effect of radiation on the single cell."

Current data from cellular and molecular biology is being reflected in models of biological processes and responses, and tumorigenesis. Simplified 2-stage models (representing the 3- to 6-stage cancer process) by Dr. Kenneth Bogen at LLNL reflect linear damage from radiation dose, with terms to reflect repair processes, including cell death by apoptosis and necrosis, along with tumorigenesis and wound repair. These models reflect the significant work scientifically establish the biological validity of the evidence for biopositive dose responses. Bogen, K.T. (1998) Mechanistic Model Predicts a U-Shaped Relation of Radon Exposure to Lung Cancer Risk Reflected in Combined Occupational and U.S. Residential Data, BELLE Newsletter, Vol 7 No. 2 see the full paper enclosed with these comments; and see the paper and responses to comments at http://www.belleonline.com/home72.html

Dr. Harald Rossi, Professor Emeritus of Columbia University, member of the International Committee on Radiation Units, the NCRP, and former member of ICRP, also published substantial criticisms of the LNT in recent years and showed that comprehensive reviews of existing data indicated hormesis in exposed populations. Dr. Rossi and Dr. Marco Zaider also of Columbia University, report on a critical review of the literature that leads to the conclusion that, at the radiation doses generally of concern in radiation protection (<2 Gy), protracted exposures to low linear energy transfer (LET) radiation (x-or gamma rays) does not appear to cause lung cancer. There is, in fact, indication of reduction of the natural incidence. Rossi, H. and Zaider, M. (1997) Radiogenic lung cancer: the effects of low doses of low linear energy transfer (LET) radiation, Radiat. Environ. Biophys. 36, pp85-88. see Data Document § 1.6 p.4

Drs. Rossi and Zaider state:

"We believe that we have reviewed most of the pertinent publications. [Ed. Note: 24 references] Those involving fluoroscopic patients are the most relevant ones because they refer to the conditions that concern radiation protection for long-term low level exposure with a relatively uniform dose distribution in the lung. In both cases, there appears to be a reduction of lung cancer at low doses. The probability that the RR is 1 or more at both 0.25 and 0.5 Gy (Fig.1) is negligibly small."

"Reduction of the cancer incidence at low or moderate doses has been observed in experimental radiobiology, where it was also found in the case of a lung tumor. (Ulrich 1976)

"Figure 1 also shows RR vs. dose as given by the risk factor of ICRP which, based on the postulate of 'linearity' permits extrapolation from the risk at high doses that can also be average doses. The evidence for no, and probably a negative risk of lung cancer at small doses not only conflicts with 'linearity' but also invalidates risk estimates based on non-uniform irradiation."

Dr. Rossi also presented a summary of low dose radiation risks. Rossi, H.H. (1999) Risks from less than 10 Millisievert, Rad. Prot. Dos. Vol. 83, No. 4, pp277-279., see the full paper attached.

The additional data in the partial summary of the extensive biology research that refutes the premise that a linear dose response exists for low dose radiation health effects incorporated in Data Document §§ 1.4, 1.5 and 1.6 are incorporated by reference.

C. Hormesis

A recent review of the "historical foundations" of radiation hormesis has been published by Edward Calabrese and Linda Baldwin. Calabrese, E.J., Baldwin, L.A. (2000) "Radiation Hormesis: its historical foundations as a biological hypothesis." And "Radiation hormesis: the demise of a legitimate hypothesis." And "Tales of two similar hypotheses: the rise and fall of chemical and radiation hormesis" In: Human and Experimental Toxicology Vol. 19, Number 1, pp41-97 see the Journal issue attached which includes summaries of the "historical foundations" of chemical hormesis.

Additional statements by RSH members on the foundations for hormesis and the failure of the radiation protection agencies to objectively document and assess the scientific bases of low dose radiation responses and health effects are provided: Luckey, T.D. (2000) Radiobiology Deceptions Reject Health, Proc. Nuclear Safety Res. Assn. Conference, Hiroshima, Japan, and; Muckerheide, J.B. (2000) Apply Radiation Health Effects Data to Contradict and Overturn Radiation Protection Policies and Rules, Proceedings of the 8th Int. Conf of Nuc. Eng., Baltimore MD, see the full papers attached.

D. Radium Ingestion Health Effects and Dose Limits

Until the 1930s, the invigorating effects of moderate levels of radiation on plant, animal, and human life enticed the public, unaware of potential long term effects of high doses. Then, in 1932 Eben Byers' died from using Radithor (starting in 1928 at age 51). Radithor is a patent medicine elixir that was made by William Bailey containing 1 µCi Ra-228 and 1 µCi Ra-226 in 1/2 oz distilled water. (Macklis, R., 1993, "The Great Radium Scandal", Scientific American, Aug., p 94). Enamored of its invigorating qualities, Mr. Byers used several bottles a day, giving it to friends by the case. In 1931 his bones deteriorated, causing his jaw to be removed with other disfiguring effects, leading to a notorious death.

The Food and Drug Administration (FDA) was seeking control of radiation use. Eben Byers was a highly recognized multi-millionaire industrialist, sportsman, and socialite. His gruesome death received national attention. FDA was able to achieve radiation control authority. The public largely abandoned radiation use except by medical direction. FDA, after Byers' overdose, did not study the health effects of persons who ingested the 400,000-500,000 vials of Radithor estimated sold (Mr. Byers used up to ~3,000), to assess their health or determine whether safe levels existed. Such studies would acknowledge the potential for dose significance. Others who had ingested Radithor, including William Bailey himself (who died in 1959 at age 64 reportedly from colon cancer), claimed to have ingested more Radithor than Byers, but not as quickly; and others accumulated much higher doses without adverse effects.

The Center for Human Radiobiology (CHR), established at Argonne National Laboratories, consolidated radium studies at Dr. Evans' retirement in 1970. As of 1979, as reported in an International Conference in Lake Geneva WI, there were 84 cancers in 4,076 radium cases exposed in the period from 1900-1950. Many were symptom-selected (including exhumations) limiting the epidemiological validity of the population. (Rowland, R.E., Stehney, A.F., and Lucas, H.F., "Dose Response Relationships for Radium-Induced Bone Sarcomas., In:Rundo, J., et al, Editors, 1983; "Radiobiology of Radium and the Actinides in Man", Proceedings of an International Conference, 11-16 October 1981; HPJ Vol 44 Suppl. 1)

There were 60 cancers in 1,953 cases where there was a measured dose. This included 1,468 young, female dial painters, with 42 cancers, NONE of which were below about 2000 rad. (A QF of 3-20 would convert that dose to 6,000 - 40,000 rem.) There was 1 cancer in 8 female non-dial-painter cases in the range of 1000-2000 rads. In 347 males, 3 cancers occurred in 16 cases in the range of 10,000-40,000 rads (30,000 to 800,000 rem?!), with none in 319 cases <10,000 rad (and none in 12 cases >40,000 rad). Rowland, R.E., *loc. cit.*

As Dr. Evans summarized for the Conference:

"...studies... continue to show no radiogenic tumors, or other effects, in hundreds [Note: 'thousands' in the U.S. alone] of persons whose effective initial body burden was less than about 50 µCi Ra-226 and whose cumulative skeletal average dose is less than about 1000 rad". Rundo, J., *loc. cit.*, p. 572

CHR was to be "an immortal organization" for radium case lifetimes, but Federal funding was severely cut in 1983 and subsequent years, and the acquisition of new cases terminated. The program was terminated in the late 1980s with more than 1,000 living cases being continuously exposed to their internal radium burden doses.

Notwithstanding these health effects results, EPA drinking water limits are 5 pCi/L Ra-226 (about 5 pCi/day, 2,000 pCi/yr), at significant public cost, while international radium studies data find zero health effects at exposures below 50,000,000 pCi Ra-226 equivalent systemic uptake in studies over 50 years. This equates to no effects for ingestion below 250,000,000 pCi Ra-226 equivalent, based on the established uptake factor of 20%.

Areas with high levels of radium in water also show no consequences, with related studies that would be more definitive also having lost Federal support.

Comparing drinking water standards to Radithor and Byers' death:

- ~5 pCi/day, vs. ~3,500,000 pCi Ra-226 equivalent in 1 Radithor vial;
- ~2,000 pCi/yr, vs. ~10,000,000,000 pCi Ra-226 equivalent in 3 yr ingested by Byers;
- 100s of people ingested >400,000 vials (>1,400,000,000,000 pCi Ra-226 equivalent)
- Plus many other radium sources in use.

Following the FDA receipt of authority over radioactivity, a report by the National Research Council in 1936 concluded that there were no beneficial effects of low doses of radiation. This report was led by a scientist that had demonstrated beneficial effects of radiation in her own work, and whose advisor had performed extensive experiments on the beneficial effects of radiation. Calabrese, E.J., Baldwin, L.A. (2000) Radiation hormesis: the demise of a legitimate hypothesis. In: Human and Experimental Toxicology Vol. 19, Number 1, pp41-97 (Journal issue attached.)

Similar results (at fewer orders of magnitude) apply to health effects data from the other exposed populations sources and doses:

D. BEIR IV Radium Dial Painter Dose-Response Results and Conclusions

The EPA references BEIR IV (NRC 1988) to state that the radium dial painter data can be represented by a linear dose-response model. However, as acknowledged by the EPA in 1991, in rejection of the recommendation of its SAB/RAC Committee to use the radium

dial painter data, the EPA correctly stated that to do so it would have to draw a straight line through very non-linear data, or abandon the linear dose-response model (which it refused to do).

Further, the source of the "linear" representation in the BEIR IV report misrepresents the scientific data. In a concise summary of the basis of this misrepresentation of the radium dial painter data presented in BEIR IV to enable this erroneous result and conclusion, Prof. Emeritus Dr. Otto Raabe, a radiobiologist at the Univ. of California, Davis, then President of the Health Physics Society, stated in an email on this issue as follows:

At 01:47 PM 4/19/99 -0500, Mike McNaughton wrote:
>Caution: the radium-dial painter data are consistent with the linear model. The data look inconsistent because they are drawn on a logarithmic graph. On this graph, the linear model transforms to an exponential, and it is possible to draw a reasonable "exponential" fit through these data.

April 21, 1999 Davis, CA

Actually, in the November 1974 issue of the Health Physics Journal Robley Evans showed definitively that NO linear model of radiation-induced bone cancer is consistent with the U.S. data on radium in people (Robley D. Evans, "Radium in Man", HEALTH PHYSICS 27:497-510, 1974). He used linear (not logarithmic) plots and rigorous mathematical tests of several hypothetical linear models (Figures 4 and 5 in his paper). His analysis demonstrated that it is highly unlikely that these data can be explained by any linear dose-response model and that all of the linear dose-response models were "strongly rejected by the chi-square test for goodness of fit."

By grouping the Evans data into six non-uniform dose groups selected so that only one dose group included no bone cancer cases (one with average skeletal alpha doses from zero to about 500 rad or 10,000 rem)and so that the next highest dose group included a few cases of bone cancer (cases were only observed for average skeletal alpha radiation doses that exceeded 1,000 rad or 20,000 rem), Chuck Mays and Ray Lloyd created the appealing, but misleading, linear plot shown on page 198 of BEIR IV. In their plot the "threshold" region, which is below 1,000 rad, is obscured near the origin since the abscissa is extended to 16,000 rad and only one dose group was assigned to this region. Their plot proves nothing about linearity. Evans's analysis shows that no linear model fits these data.

Otto
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