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Stimulating Effects of Ionizing Radiation: New Issue for Regulatory Policy

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The projects of radiation protection regulations usually invoke as their scientific basis the documents of the United Nations Scienti c Committee on the Effects of Atomic Radiation (UNSCEAR). These projects and UNSCEAR activity, however, are sometimes out of step. For example, in the recent *Notice on the Federal Radiation Protection Guidance for the Exposure of the General Public* prepared by the U.S. Environmental Protection Agency (EPA, 1994), four UNSCEAR documents, from 1977, 1982, 1986, and 1988, are used to support a need for revision of the current radiation standards. The most recent UNSCEAR document from 94, however, on the adaptive effects of low doses of ation, is not taken into account in this *Notice*, in which a new radiation limit for the public of 1 mSv per year (or 70 mSv in a lifetime) is proposed. Implementation of such a low radiation standard, which is only about 3% of the natural radiation background in many regions of the world, would bring enormous costs for society, and it would be ethically fair only through a large reduction of identifiable health hazards. The UNSCEAR 1994 document does not provide scientific evidence that such a low radiation standard is desirable.

The ur UNSCEAR documents quoted in the EPA *Notice* as estimating that the risks "of cancer have increased roughly threefold and have become more certain" were critically examined by UNSCEAR during the past 3 years, especially the interpretation of the results of epidemiological studies in Hiroshima and Nagasaki. In its 1994 document UNSCEAR concluded that it recognizes that the limitation of the Japanese data is their inability to address directly the effects of low-dose exposures. UNSCEAR (1993) introduced a factor by which cancer risk estimates derived from studies at high doses should be reduced to derive estimates for low doses. The committee suggested that the reduction factor of 2 should be applied for all doses below 200 mSv.

The most important message of the recent UN- AR (1994) document, however, is the recognition ne existence of stimulating and adaptive effects of ionizing radiation. During the past 4 decades these effects were ignored in radiation protection philosophy and practice.

THE FALLACY OF EXTRAPOLATING TO ZERO

Since 1959 the International Commission on Radiological Protection (ICRP) based its rules on an assumption that the relationship between dose and effect is a straight line, so that even a dose close to zero has some detrimental effect (ICRP, 1959). This assumption is called the "linear hypothesis" which states that there is no dose threshold or limit below which the effects observed at high doses cease to appear. The "effects" in this hypothesis are assumed to be only the detrimental ones, such as decrease in life duration, occurrence of cancers, and genetic damage. It also implies that no new effects occur at low doses. In this paper[1] the evidence showing that such new effects occur and that they may lead to the health advantage in populations exposed to low doses of ionizing radiation is discussed.

Over the years the working assumption of ICRP of 1959 came to be regarded as a scientifically documented fact by the mass media, public opinion, regulatory bodies, and even many scientists. The no-threshold principle, however, belongs to the realm of administration and it is not a scientific principle.

At the time the no-threshold hypothesis became the backbone of the philosophy and policy of radiation protection, the main scientific evidence came from epidemiological studies of atomic bomb survivors in Hiroshima and Nagasaki who were irradiated with doses up to about 6000 mSv. These studies indicated that the cancers were induced by single radiation doses hundreds and thousands of times higher than the worldwide average annual dose from natural background radiation, i.e., 2.4 mSv (or 168 mSv over a 70-year lifetime). Whether any cancers are induced by background radiation or ordinary exposure of the general popula-

[1] This article is based on the author's paper published in *Nukleonika, Int. J. Nucl. Res.* 41(1), 1995.

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tion to man-made radiation, however, was not indicated by the Hiroshima and Nagasaki studies.

The ICRP assumption on linearity was not very realistic. It was generally accepted, however, because it simplified regulatory work by allowing extrapolation from the high-dose region—about which we had reliable epidemiological data—to the unknown, small-dose region. The original purpose was to regulate the exposure to radiation of a relatively small group of occupationally exposed persons and it did not involve exceedingly high costs to society.

In the 1970s, however, ICRP extended the no-threshold principle to exposure of the general population to man-made radiation, and in the 1980s it extended the principle to limiting the exposure to natural sources of radiation. The dose limit for the public was set at 50 mSv over a lifetime (ICRP, 1984). This value is less than one-third of the global average lifetime dose from background radiation (168 mSv for 70 years) and many tens or hundreds of times lower than the lifetime dose in many regions of the world.

Limiting exposure below the levels of natural radiation at which millions of people have lived since time immemorial is a logical consequence of the administrative assumption from 1959: if each dose is detrimental, then one should also attempt to decrease the risk of background radiation from Mother Nature or the risk of man-made radiation even at such trivial levels as 1 mSv per year.

Yet such reasoning was less than palatable to many scientists associated with radiation protection. This was not only because of the epistemological problem of trespassing beyond the limits of knowledge, as pointed out by Weinberg (1972) and by Walinder (1987), or more recently by Seiler and Alvarez (1994), but also because of the absurd practical consequences and the moral aspects.

As demonstrated by Walinder, on the complementarity principle, the stochastic phenomenon of radiation carcinogenesis cannot be predicted for an open system, such as a human being or a population. It can only be done if the radiation dose is much more powerful than the natural dose, combined with other carcinogenic factors, which we meet in our daily lives. A conception that mathematical models adapted for high-dose effects can be limitlessly extrapolated to low doses and still represent a biological reality is epistemologically unacceptable (Walinder, 1987). The absurd practical consequences were exposed by the Chernobyl accident.

Standing on these practical grounds alone, several speakers at the Seventh World Congress of the International Radiation Protection Association and International Conference on Radiation Protection in Nuclear Energy in Sydney, Australia, in 1988 criticized the no-threshold principle (e.g., Alexander, 1988). And long before that Professor W. V. Mayneord, one of the most notable persons in radiation protection and a former member of the United Kingdom delegation to UNSCEAR and of ICRP (Mayneord, 1964), stated: "I have always felt that the argument that because at higher values of dose an observed effect is proportional to dose, at very low doses there is necessarily some 'effect' of dose, however small, is nonsense."

THE CHERNOBYL CASE

The absurdity of the no-threshold principle was brought to light after the Chernobyl catastrophe in 1986. A giant global monitoring network for radioactive contamination gathered a vast amount of data on radiation doses from Chernobyl fallout to the population of the Northern Hemisphere. For example it was found that people living in the United States would receive a dose of "Chernobyl radiation" over the next 50 years approaching 0.0046 mSv, or 0.004% of their natural background dose (120 mSv). The population of the rest of the Northern Hemisphere would receive 0.3%, and in the European part of the former Soviet Union the population would receive 5.0%.

These minute doses were then used to calculate the number of cancer deaths predicted by the linear no-threshold hypothesis over the next 50 years. For example, it was calculated that 30 more cancer deaths would occur in the United States, 28,000 more in the Northern Hemisphere, and 25,400 more in formerly Soviet Europe (Goldman *et al.*, 1987).

These numbers were derived by simply multiplying the Chernobyl doses by the number of people living in each region and by a cancer risk factor based on epidemiological studies of 75,000 atomic bomb survivors in Hiroshima and Nagasaki. The bomb survivors, however, were irradiated in a fraction of a second with doses more than 50,000 times higher than the total cumulative dose that the U.S. inhabitants will receive from the Chernobyl fallout over 50 years. No epidemiological data exist to indicate that a linear dose–effect relationship holds in this situation.

Only in a few of the estimates of the Chernobyl radiation mortality were readers informed that there is a probability of a zero effect, and such statements were never cited by the media, which reported tens of thousands of future Chernobyl-radiation-caused deaths as fact, not hypothetical extrapolations.

The media also ignore epidemiological studies from different parts of the world where the background radiation is 100 to 1000% more than the global average—not 0.004 or 0.3 or 5%—yet no higher cancer death rate has ever been observed. On the contrary, the cancer death rate is often lower than in less radioactive regions.

Dr. Lauriston Taylor, the former president of the U.S. National Council on Radiological Protection and Measurements, defined applications of the linear, no-threshold dose–effect relationship to such calculations

TABLE 1

eath from Breast Cancer among 31,710 Canadian women Exposed to Radiation at Fluoroscopic Examinations

Dose (mGy)	Standardized rate per 10^3 person-years
0–90	578.6
100–190	421.8
200–290	560.7
300–390	650.7
400–490	610.0
700–990	1362
1000–2900	1382
3000–5990	2334
6000–10,000	8000
>10,000	20,620

Note. Authors concluded that: "The data were most consistent with linear dose response relation" and did not comment on the hormetic effect evident at 100–190 mGy dose range. Adapted from Miller *et al.*, 1989.

as "deeply immoral uses of our scientific heritage" (Taylor, 1980).

The no-threshold arithmetic was also applied to the local population around Chernobyl and led to a decision the Supreme Soviet (but against the advice of the ng Soviet scientists (Ilyin, 1993) to evacuate about ,000 inhabitants of Ukraine and Belarus, causing unspeakable suffering and a loss of many billions of dollars, equivalent to about 1.5% of the GNP of the whole of the former Soviet Union (ICP, 199).

The intervention level for evacuation was a 70-year lifetime radiation dose of 350 mSv, or about twice the world average natural background dose (168 mSv). All families with pregnant women and children less than 12 years of age were relocated from areas with cesium-137 contamination greater than 550 kBq/m^2. Yet the cesium-137 body burden in children still living in these areas was found to range between 40 and 2250 Bq, which is less than the natural burden of radioactive potassium-40 (4000 Bq) in adults. Radiocesium body burdens of several thousand bequerels are now common in Northern Canada and were as high as 100,000 Bq during weapons tests in the 1960s (Tracy *et al.*, 1994).

Given the actual figures of natural radiation, one might ask why governments of various countries do not relocate populations living in areas where lifetime exposure to natural radiation exceeds 350 mSv? For example, why isn't everyone evacuated from Norway, where the average lifetime dose is 365 mSv (Henriksen and Saxebøl, 1988) and in some districts 1500 mSv rli, 1995)? Should not the regions of India with than 2000 mSv (Sunta, 1990) be depopulated?

What about the areas of Iran with more than 3000 mSv? Perhaps in Iran, for example, the government decided not to follow the ICRP guidelines when it observed that in the city of Ramsar several generations in one household have been receiving average individual lifetime doses of natural radiation of 17,000 mSv, 240 times the current ICRP limit. Yet these individuals show no increased incidence of disease, and some of them have lived to be 110 years of age (Sohrabi, 1990).

Using the no-threshold principle to calculate precise numbers of imaginary victims of Chernobyl fallout is like counting the number of dead among a small group of suicidal persons who each consume 50,000 tablets of aspirin in one session, and stating that the same number of deaths will occur in another group, 50,000 times greater, whose members each consume a single aspirin tablet, divided over the next 50 years into 18,250 daily doses (the number of days in 50 years).

Even before eating all the 50,000 tablets at one session most of the persons in the first group would die, while the members of the more moderate group will show no detrimental effects and may, in fact, have health benefits if they increase their consumption to one tablet per day.

HORMESIS: STIMULATORY AND ADAPTIVE EFFECTS

The aspirin example is used to illustrate the well-known fact that it is the *dose* that makes an agent or a substance harmful, imperceptible, or beneficial. Beneficial and protective effects of low doses of radiation were known long ago. In plants, such effects were

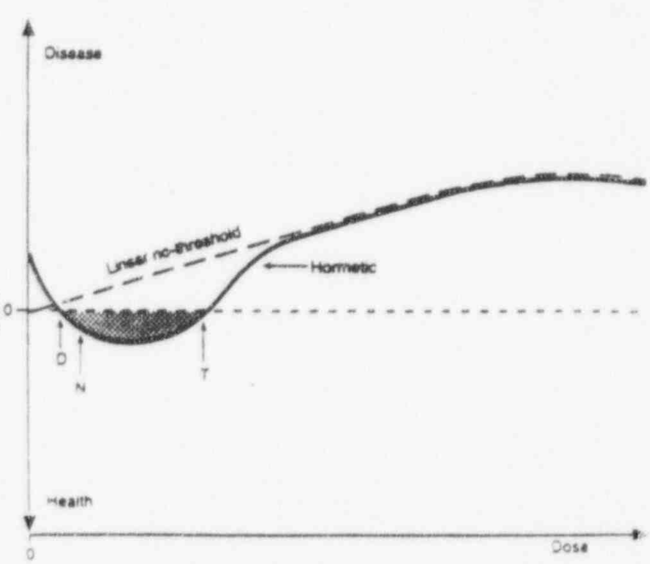

FIG. 1. Diagram of general biological response to chemical and physical agents. Deficit of an agent (dose less than D) causes deficiency symptoms; small doses (between D and T) are vital for good health (shaded area); doses higher than T cause toxic or other harmful effects. N is probably average global natural radiation dose. Dotted and solid lines represent linear no-threshold and hormetic dose–effect relationships, respectively.

TABLE 2
Frequency of Genetic Effects of Radiation (%) in Children of Atomic Attack Survivors in Hiroshima and Nagasaki (Parents Exposed to Dose of 400–600 mSv)

Effect	Unexposed	Exposed	References
Death of liveborn children 1946–1958	7.35	7.08	Yoshimoto *et al.*, 1991
Chromosomal aberrations	0.31	0.22	Awa *et al.*, 1989
Aneuploidy	0.30	0.23	Awa *et al.*, 1989
Mutations in blood proteins	6.4×10^{-6}	4.5×10^{-6}	Neel *et al.*, 1988

observed soon after the discovery of Roentgen radiation in 1895 (Atkinson, 1898).

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).

Since the 1960s, such effects have been ignored in radiation protection practice, while research on stimulating and adaptive effects of radiation, the *radiation hormesis*, has continued over several decades. The results of more than 1200 published papers on hormesis were recently reviewed by Luckey (1990)—many of them in an excellent book by Kondo (1993). The studies on hormesis were also presented at four international conferences (Oakland, CA, 1985; Frankfurt, Germany, 1987; Kyoto, Japan, 1992; and Changchun, China, 1993). It is astonishing, however, that even recently the obvious hormetic effects appearing in epidemiological studies were often not noticed, not only by the readers, but by the authors themselves (see for example Table 1).

Radiation hormesis goes beyond the notion that radiation has no deleterious effects at small doses: at small doses new stimulatory effects occur that are not observed at high doses, and these new effects may be beneficial to the organisms (Fig. 1).

Recognition of the existence of hormesis opens up an important new field of research. If hormesis did not exist, it would not be possible to demonstrate rigorously a true threshold. This follows from the purely statistical difficulty of proving that there is an absolute equality of an effect in an epidemiological study at zero dose and at an elevated dose. If hormesis does cause a decrease in cancer incidence or a decrease in other deleterious effects in a population exposed to small doses of radiation or other agent, however, there may be a statistically significant difference at an acceptable confidence level (Webster, 1993). For instance, it would require a vast number of people to detect in a population in which the normal frequency of cancer death is 20%, an increase in frequency of 0.005% (due to additional exposure of 1 mSv per year, predicted by the no-threshold linear extrapolation (EPA, 1994)),[2] which is $\frac{1}{4000}$ of normal frequency. Due to the hormetic effect, however, the decrease in cancer death at the dose range of 1 to 10 mSv may reach perhaps a relatively large $\frac{1}{4}$ of the normal incidence, as suggested by epidemiological studies in high background areas in China (see below). This may solve the problem of the prohibitively large populations needed to resolve the threshold dilemma.

Part of the information on (unexpected) positive genetic effects of ionizing doses of radiation comes from Hiroshima and Nagasaki. The data from these two cities show that acute irradiation with moderate doses does not produce any major negative effect on the health of the following generation. Any minor negative effects that might be produced are so small that they are submerged in the background noise of naturally occurring mutational effects; they cannot be demonstrated even by the refined epidemiological methods that have been used. What can be demonstrated, on the other hand, are the positive effects. Among the children of parents who survived the atomic bombings in Hiroshima and Nagasaki there were about 4% fewer deaths between 1946 and 1958 than among the children of parents unexposed to radiation from atomic bombs, 23% less aneuploidy, 29% fewer chromosomal aberrations, and 30% fewer mutations in blood proteins (Table 2). Similarly unexpected results were obtained in one of the best studies in human genetics carried out in Hungary before and after the Chernobyl accident. Several serious congenital anomalies occurred after the Chernobyl accident with lower frequency than before the accident (Table 3).

The epidemiological data from Japanese atomic bomb survivers show that persons irradiated with low

[2] UNSCEAR (1988, Paragraphs 266–270) discourages such numerical estimates of cancer death, which are not expected to be noticeable in health statistics. The committee refrains from such estimates of detriment in its publications because of their great uncertainty.

TABLE 3
Number of Birth Defects and Anomalies (per 10,000) in Hungary Following the Chernobyl Accident

Birth defect	1980–1985	May 1, 1986–April 30, 1987	May 1, 1987–April 30, 1988
Number of live births	807,939	126,708	125,514
Down's syndrome	8.44	7.27	6.77
Retinoblastoma	0.32	0.16	0.08
Wilms tumor	0.80	0.47	0.56

Note. Average Chernobyl radiation dose in various parts of Hungary ranged from about 60 to >500 μSv per capita (Feher, 1988). After Czeizel, 1989.

doses (but more than 10 times higher than the proposed EPA annual standard) have 40% lower mortality rate than unexposed persons. The mortality rate was also slightly lower in persons who were irradiated with doses of >10 mSv than with doses of <5 mSv (Table 4). As shown in Fig. 2, the relative risk of cancer death in male survivors of the Nagasaki bombing was lower at the dose range of 10 to 490 mSv than that in nonirradiated persons.

The UNSCEAR (1994) document reviewed the most important publications on the stimulating effects of radiation. These effects were found at biochemical, cellular, and organic levels, in cell cultures, bacteria, plants, and animals. In mammals radiation hormesis enhances defense reactions against neoplastic and infectious diseases, increases longevity, and improves fertility. For example, in an experiment with mice the incidence of leukemias, cancers, and sarcomas was lower in animals irradiated with cesium-137 gamma radiation doses of 2.5 to 20 mSv than it was in nonirradiated controls. The number of all malignant neoplasms in animals exposed to a single dose of 10 mSv was more than 30% lower than in nonirradiated controls (Table 5). In several experiments, small initial radiation doses have been shown to improve the survival of animals subsequently irradiated with large, near lethal doses. In other experiments, an increased life span was found in animals irradiated with doses between 250 and 3000 mSv.

Of special interest is a group of French studies discussed in UNSCEAR (1994). These studies, started in the early 1960s, indicate that protozoa and bacteria exposed to artificially *lowered* levels of natural radiation demonstrate deficiency symptoms expressed as dramatically decreased proliferation (Fig. 3). This indicates that ionizing radiation may be essential for life. Indeed, this might be expected. Living organisms developed under constant exposure to ionizing radiation, which was higher in the past than now. The early organisms had to protect themselves against the adverse effects of radiation to survive, and they probably began to use the radiation to their advantage. Similarly, at some point, organisms learned to use high-dose ultraviolet radiation in the process of photosynthesis, which became the basis for almost all life.

The 1994 UNSCEAR document 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.

Perhaps most interesting, however, are the results of studies on human populations. The UNSCEAR report states that among nuclear attack survivors from Hiroshima and Nagasaki who received doses of <200 mSv

TABLE 4
Annual Mortality Rates in Nagasaki (per 100,000) in 1970–1976

Sex	Unexposed inhabitants of Nagasaki	Bomb survivors <5 mSv	Bomb survivors >10 mSv
Male	45,731	27,551	25,835
Female	27,291	18,970	17,957
M—percentage of unexposed	100	63	59.9

Note. After Kondo, 1993.

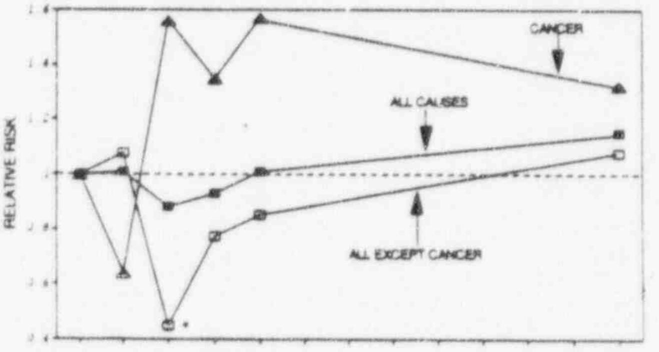

FIG. 2. Relative risk for death from all causes, from all causes except cancer, and from cancer alone in male survivors of the atomic bombing of Nagasaki. *$P < 0.05$. Adapted from Kondo (1993).

TABLE 5

Incidence of Neoplasms (%) after a Single Exposure of 3-Month-Old Mice to ^{137}Cs Gamma Radiation

Dose (mSv)	Leukemias	Carcinomas and sarcomas	All neoplasms
0	20.93	16.3	33.19
2.5	18.18	14.04	28.51
5.0	15.48	8.79	23.01
10	15.04	8.94	21.95
20	13.82	14.29	26.27
40	26.57	16.08	39.16
60	44.68	14.36	55.32

Note. Adapted from UNSCEAR (1994).

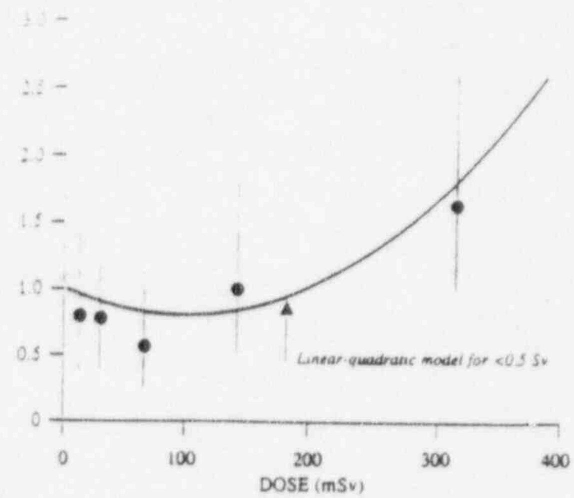

FIG. 4. Mortality risk from leukemia in Hiroshima and Nagasaki and dose–response curve. After UNSCEAR (1994).

(<200 times higher than the proposed EPA annual limit) there was no increase in the number of total cancer deaths. In fact, mortality caused by leukemia was less in this population at doses <100 mSv than among the nonirradiated inhabitants of these Japanese cities, which is not statistically significant (Fig. 4).

Probably the best radioepidemiological study at low doses to date has been carried out in China. Between 1970 and 1986, 74,000 people in Yangjiang county, which has a high level of natural background radiation (5.5 mSv per year), were compared to 77,000 people in two adjacent low-background counties (Enping and Taishan, 2.1 mSv per year). In the high-background Yangjiang county, the inhabitants receive a 70-year lifetime dose of 385 mSv, which is higher than the intervention level for evacuation adopted for Chernobyl, and 5.5 times higher than the dose limit proposed in the EPA (1994) *Notice*.

Should the Chinese government follow the Soviet example and evacuate Yangjiang county? The epidemiological data show that there is no reason to do so. In an age group of 10 to 79 years the general (nonleukemia) cancer mortality was 14.6% lower in the high-background county than in the low-background counties. The leukemia mortality among men was 15% lower and among women 60% lower in Yangjiang (Wei *et al.*, 1990).

From among several studies of people occupationally exposed to low radiation doses discussed in UNSCEAR (1994), I present here data on mortality of 13,491 employees of the Atomic Energy of Canada Limited (Gribbin *et al.*, 1992). Among these persons, 5504 were not exposed to gamma radiation. The mean radiation dose of exposed persons was 49 mSv for men and 5.5 mSv for women. As shown in Table 6 the mortality due to all leukemias in the exposed group was only 32% of that in the general Canadian population. The observed mortality among employees of AECL from all cancers and from all noncancer diseases was also less than expected.

The most recent data showing hormetic effects in humans come from the former Soviet Union. In September 1957, inhabitants of 22 villages in the Eastern Urals were irradiated with high radiation doses of up to 1500 mSv, the result of a radioactivity release from a thermal explosion in a Soviet military nuclear facility. About 10,000 people were evacuated and their cancer mortality was studied during the next 30 years.

From this group, 7852 of the persons studied were divided into three exposure groups: those who received

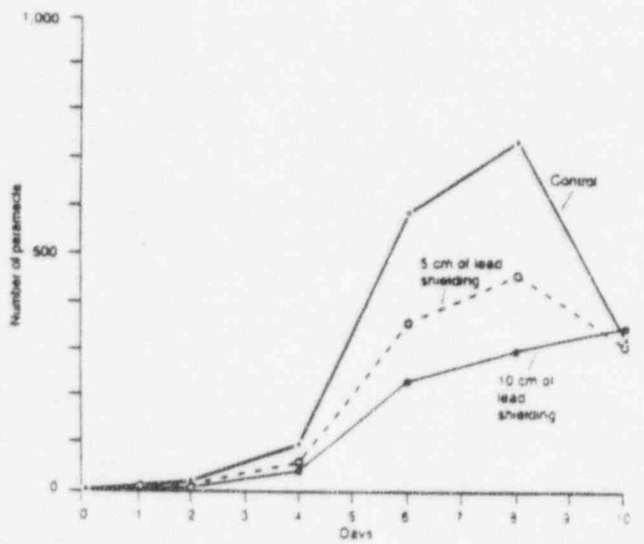

FIG. 3. Effect of shielding on proliferation of *Paramecium tetraurelia* cultured in identical chambers which were shielded with either 5 or 10 cm of lead. The nonshielded control animals were exposed to the normal natural gamma radiation rate of 1.75 mGy per year and animals shielded by 10 cm of lead were exposed to 0.3 mGy per year. On the eighth day, the proliferation of paramecia in the chambers shielded with 5 and 10 cm of lead was 60 and 40%, respectively, of proliferation of the nonshielded control animals. After Planel *et al.*, 1987.

TABLE 6
Standardized Mortality Ratios (SMR) for Leukemias in 9997 Male Employees of Atomic Energy of Canada, Limited

Cause of death	Nonexposed		Exposed	
	SMR	Observed/expected	SMR	Observed/expected
Lymphocytic leukemias	2.25	2/0.85	—	0/2.40
Myeloid leukemias	1.47	2/1.36	0.57	2/3.50
Other leukemias	0.72	1/1.40	0.28	1/3.59
All leukemias	1.37	5/3.64	0.32	3/9.50

Note. After Gribbin *et al.*, 1992.

average doses of 496, 120, and 40 mSv. Tumor-related mortality in the 496-mSv group was 28% lower than in the nonirradiated control population from the same region; in the 120 mSv group it was 39% lower, and in the 40-mSv group it was 27% lower. In the first two groups, the difference from the controls was statistically significant (Kostyuchenko and Krestinina, 1994).

Most of the epidemiological studies of a relationship between the radon levels in homes and lung cancer also seem to be in disagreement with the no-threshold principle, and some may suggest a hormetic effect. Of 19 studies reviewed in UNSCEAR (1994), 8 indicated increase in lung cancer mortality or incidence in ulations exposed to higher than average radon lev- .., and 11 studies indicated lower mortality or incidence or no association of lung cancer and radon levels.

In the United States, a study that covered 89% of the population found that the people living in houses with radon air concentrations that were higher than the average level had a lower mortality from lung cancer (Cohen, 1993).

In China, a meticulous study measured the radon level for 1 year in the houses of several hundred women with lung cancers and in homes of a similar number of healthy women. The results demonstrated at a 95% confidence level that women who lived in high-level radon houses (more than 350 Bq/m^3) had a 30% lower lung cancer risk than those living in low-level radon houses (4 to 70 Bq/m^3).

This result is opposite to the no-threshold principle estimate, according to which the lung cancer risk in the high-radon houses should be 80% higher than the normal risk (Blot *et al.*, 1990).

Similarly, in one region of Japan with an average indoor radon level of 35 Bq/m^3, the lung cancer incidence was 51% of that in a low-level radon region (11 Bq/m^3), and the mortality caused by all types of cancer was 37% lower (Mifune *et al.*, 1992). Similar results showing a lack of positive correlation between lung can- d indoor radon levels were reported from Canada, ·n, Denmark, Finland, France, and Great Britain (see UNSCEAR, 1994, for references).

Despite the evidence from these studies, the U.S. Environmental Protection Agency has recommended remedial action when indoor radon concentrations reach 150 Bq/m^3. The EPA considers that remedial action at any level down to 70 Bq/m^3 would be cost effective, even for the cost of reducing the level from 150 to 70 Bq/m^3 at approximately $2 million per life hypothetically saved (Schiager, 1992). Each human life hypothetically saved by implementing the U.S. Nuclear Regulatory Commission's regulations costs about $2.5 billion (Cohen, 1992). Such spending is morally questionable. Studies of radiation hormesis suggest that such expenditures may be futile and actually have an adverse effect on the health of the population.

The recognition by UNSCEAR, the most distinguished international scientific body on the matters of ionizing radiation, of the possibility that low doses of radiation may result in changes in cells and organisms which reflect an ability to adapt to the effects of radiation, may inspire the authorities to begin a more realistic approach to problems of estimating and managing the risks of ionizing radiation. The past 4 decades witnessed regulatory activity, stemming from the linearity principle, steadily decreasing radiation standards to an absurd subnatural level of 1 mSv per year. The time is ripe for renunciation of linearity principle in radiation protection of the public and for considering a practical threshold dose as a basis for radiation standards.

REFERENCES

Alexander, R. E. (1988). The linear non-threshold hypothesis. In *Radiation Protection in Nuclear Energy*, pp. 483–486. International Atomic Energy Agency, Vienna.

Atkinson, G. F. (1898). Report upon some preliminary experiments with Roentgen rays on plants. *Science* **7,** 7.

Awa, A. A., Honda, T., Nerishi, S., Sofuni, T., Shimba, H., Ohtaki, K., Nakano, M., Kodama, Y., Ytoh, M., and Hamilton, H. B. (1989). *Cytogenic Study of the Offsprings of Atomic Bomb Survivors, Hiroshima and Nagasaki.* Radiation Effects Research Foundation, Hiroshima. Report RERF TR 21-88, pp. 1–20.

Baarli, J. (1995). Personal communication.

Blot, W. J., Xu, Z.-Y., Boice, J. D., Jr., Zhao, D.-Z., Stone, B. J., Sun, J., Jing, L.-B., Fraumeni, J. F., Jr. (1990). Indoor radon and lung cancer in China. *J. Natl. Cancer Inst.* **82**(12), 1025–1030.

Brucer, M. (1989). Letter to the editor of *Time* magazine, quoted in *Access to Energy* **16**(7), March 1989.

Cohen, B. L. (1992). Perspectives on the cost effectiveness of life saving. In *Rational Readings on Environmental Concerns* (J. H. Lehr, Ed.), pp. 461–473. Van Nostrand-Reinhold, New York.

Cohen, B. L. (1993). Relationship between exposure to radon and various types of cancer. *Health Phys.* **65**(5), 529–531.

Czeizel, A. (1989). Hungarian surveillance of germinal mutations. *Hum. Genet.* **82**, 359–366.

Environmental Protection Agency (EPA) (1994). Federal radiation protection guidance for exposure of the general public: Notice. *Fed. Regist.* **59**(246), 66414–66428.

Feher, I. (1988). Experience in Hungary on the radiological consequences of the Chernobyl accident. *Environ. Int.* **14**(2), 113–135.

Goldman, M., Catlin, R., and Anspaugh, L. (1987). *Health and Environmental Consequences of the Chernobyl Nuclear Power Plant Accident*, Report DOE/RR-0232, p. 209. U.S. Department of Energy, Washington, DC.

Gribbin, M. A., Howe, G. R., and Weeks, J. L. (1992). *A Study of the Mortality of AECL Employees. V. The Second Analysis: Mortality during the Period 1950–1985.* Report No. AECL-10615, p. 48. Atomic Energy of Canada Limited.

Henriksen, T., and Saxebøl, G. (1988). Fallout and radiation doses in Norway after the Chernobyl accident. *Environ. Int.* **14**(2), 157–163.

Ilyin, L. A. (1993). Personal communication.

International Chernobyl Project (ICP) (1991). *International Advisory Committee Technical Report.* Vol. III, Annex G2. IAEA, Vienna.

International Commission on Radiological Protection (ICRP) (1959). *Recommendations of the International Commission on Radiological Protection.* ICRP Publication 1. Pergamon, London.

International Commission on Radiological Protection (ICRP) (1984). Principles for limiting exposure of the public to natural sources of Radiation: Statement from the 1983 Washington Meeting of the ICRP. ICRP Publication 39. *Ann. ICRP* **14**(1), i–vii.

Kondo, S. (1993). *Health Effects of Low-Level Radiation*, p. 213. Kinki Univ. Press, Osaka, Japan.

Kostyuchenko, V. A., and Krestinina, L. Yu. (1994). Long-term irradiation effects in the population evacuated from the East-Urals radioactive trace area. *Sci. Total Environ.* **142**, 119–125.

Luckey, T. D. (1990). *Radiation Hormesis.* CRC Press, Boca Raton, FL.

Mayneord, W. V. (1964). *Radiation and Health*, p. 140. The Rock Carling Fellowship, The Nuffield Provincial Hospital Trust.

Mifune, M., Sobue, T., Arimoto, H., Komoto, Y., Sohei, K., and Tanooka, H. (1992). Cancer mortality survey in a spa area (Misasa, Japan) with a high radon background. *Jpn. J. Cancer Res.* **83**, 1–5.

Miller, A. B., Howe, G. R., Sherman, G. J., Lindsay, J. P., Yaffe, M. J., Dinner, P. J., Risch, H. A., and Preston, D. L. (1989). Mortality from breast cancer after irradiation during fluoroscopic examinations in patients being treated for tuberculosis. *N. Engl. J. M.* **321**, 1285–1289.

Neel, J. V., Satoh, C., Goriki, K., Asakawa, J., Fujita, M., Takahashi, N., Kageoka, T., and Hazama, R. (1988). Search for mutations altering protein change and/or function in children of atomic bomb survivors: Final report. *Am. J. Hum. Genet.* **42**, 663–676.

Planel, H., Soleilhavoup, J. P., Taxidor, R., Richoilley, G., Conter, A., Croute, F., Caratero, C., and Gaubin, Y. (1987). Influence on cell proliferation of background radiation or exposure to very low chronic γ radiation. *Health Phys.* **52**(5), 571–578.

Schiager, K. J. (1992). Radon—Risk and reason. In *Rational Readings on Environmental Concerns* (J. H. Lehr, Ed.), pp. 619–626. Van Nostrand-Reinhold, New York.

Seiler, F. A., and Alvarez, J. L. (1994). The scientific method in risk assessment. *Technol. J. Franklin Inst.* **331A**, 53–58.

Sohrabi, M. (1990). Recent radiological studies of high level natural radiation areas of Ramsar. *Proceedings, International Conference on High Levels of Natural Radiation*, pp. 39–45. IAEA, Vienna.

Sunta, C. M. (1990). A review of the studies of high background areas of the S-W coast of India. *Proceedings, International Conference on High Levels of Natural Radiation*, pp. 71–86. IAEA, Vienna.

Taylor, L. S. (1980). *Proceedings 5th International Congress of the International Radiation Protection Assoc.*, Vol. 1, Some nonscientific influences on radiation protection standards and practice, pp. 307–319. The Israel Health Physics Society, Jerusalem.

Tracy, B. L., Kramer, G. H., and Gamarnik, K. (1994). Radiocesium in children from Belarus. *Health Phys.* **66**(4), 439–443.

Walinder, G. (1987). Epistemological problems in assessing cancer risks at low radiation doses. *Health Phys.* **52**(5), 675–678.

Webster, E. W. (1993). Hormesis and radiation protection. *Invest. Radiol.* **28**(5), 451–453.

Wei, L., Zha, Y., Tao, Z., He, W., Chen, D., and Yua, Y. (1990). Epidemiological investigation of radiological effects in high background radiation areas of Yangjiang, China. *J. Radiat. Res.* **31**, 119–136.

Weinberg, A. M. (1972). Science and trans-science. *Minerva* **10**, 209–222.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (1958). *Report of the United Nations Scientific Committee on the Effects of Atomic Radiation.* United Nations, General Assembly Official Records: 13th Session, Suppl. 17 (A/3838), p. 228.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (1993). *Sources and Effects of Ionizing Radiation*, p. 922. UNSCEAR, New York.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (1994). *Sources and Effects of Ionizing Radiation*, p. 272. UNSCEAR, New York.

Yoshimoto, Y., Schull, W. J., Kato, H., and Neel, J. V. (1991). *Mortality among the Offspring (F1) of Atomic Bomb Survivors, 1946–1985.* Radiation Effects Research Foundation, Hiroshima, Report RERF TR 1-91, pp. 1–27.

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