(Pre-publication preprint.)

ASSESSING CHERNOBYL'S CANCER CONSEQUENCES:*

APPLICATION OF FOUR "LAWS" OF RADIATION CARCINOGENESIS

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Presentation at the 192nd National Meeting
The American Chemical Society
Anaheim, California
September 9, 1986

Symposium On Low-Level Radiation
Division of Chemical Health and Safety

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September 7 - 10:

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• Introduction

There exists as large a body of evidence for the human concerning the quantitative aspects of induction of cancer and leukemia by ionizing radiation as exists for any other carcinogen, probably an even larger body of evidence. In 1969, using the human evidence available to that time, Gofman and Tamplin[2] presented three generalizations or "laws" which permit quantitative assessment of the cancer toll which will follow human exposure to ionizing radiation under virtually all circumstances of exposure.

It is gratifying --- as a scientist, but not as a physician --- that all of the human epidemiological evidence which has accumulated since 1969 has provided support for the correctness and usefulness of the three laws. In other words, the laws have been correctly predicting what is being found. The laws apply as well for the incidence of cancer induction by radiation as for the mortality.

The validity of the laws made it possible to arrive in 1981 at a single ratio, called the Whole-Body Cancer-Dose for a population of mixed ages, with which ratio the cancer consequences of population exposures could be correctly assessed. The evidence supporting the laws and the step-by-step method of transforming the human evidence into the Whole-Body Cancer-Dose are presented in detail in Chapters 5-10 of Gofman[3].

In the first part of the present paper, the laws, the Whole-Body Cancer-Dose, and the status of challenges to their validity are discussed. Then the idea that cellular repair of DNA and chromosomes may provide some safe threshold dose of ionizing radiation, with respect to human carcinogenesis, is shown to be ruled out by human evidence which already exists. And lastly, specific errors in the reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[4, 5] and of the N.A.S. Committee on the Biological Effects of Ionizing Radiation (BEIR-3)[6] are identified because those errors would make assessments of Chernobyl's cancer consequences at least 16 to 25 times too low; the disparate risk-estimates are reconciled.

In the paper's second part, the Whole-Body Cancer-Dose is applied to the available dose-data from the Chernobyl accident, and the cancer-leukemia consequences are assessed in Table 6 for each of 30 countries; additionally, the methods for handling such data and making such calculations are shown step-by-step so that anyone anywhere can use them when and if a country provides more detailed dose-data.

(1) THE LAWS OF HUMAN CARCINOGENESIS FROM IONIZING RADIATION

• Generalization 1:

"All forms of cancer, in all probability, can be increased by ionizing radiation, and the correct way to describe the phenomenon is either in terms of the dose required to double the spontaneous mortality rate for each cancer, or alternatively, of the (percent) increase in mortality rate of such cancers per rad of exposure."

• Generalization 2:

"All forms of cancer show closely similar doubling doses and closely similar percentage increases in cancer mortality rate per rad " (at a given age).

• Generalization 3:

"Youthful subjects require less radiation to increase the (cancer) mortality rate by a specified fraction than do adults."

• Generalization 4, added in 1983 (Gofman, 1983)[7]:

"The peak percent increase in cancer rate per rad is reached grossly earlier for such high Linear-Energy-Transfer radiations as alpha-particle irradiation in contrast to the time to reach peak percents for low LET radiation."

The fourth law is not discussed or used in this analysis, whose particular application is Chernobyl. While it is clear that the accident caused measurable quantities of plutonium, americium, curium, and other alpha-emitters to fall in Scandinavia and elsewhere, daily data on air concentrations are not available. Since inhalation causes the predominant hazard from such nuclides, we are able to say nothing except that any cancers induced by the transuranics would be additional to those listed in Table 6.

• STATUS AND IMPLICATIONS OF THE FIRST LAW

• All Forms of Cancer

By now, the human evidence clearly shows that radiation exposure increases the frequency of virtually every major kind of cancer. By major kind, we mean those kinds which, combined, account for about 90% of cancer deaths. Prostate cancer[8] and uterine cancer[9] have recently joined the proven group.

By 1982, Dr. Edward Radford, Chairman of the BEIR-3 Committee, said "The point that I feel is important is the consistency with which radiation

has proved to be carcinogenic in man. It is far and away the most consistent agent that we know of to cause cancer of any type"[10]. In a 1985 report, the National Institutes of Health[11] acknowledged that "it is generally accepted that ionizing radiation may increase the risk of virtually any form of cancer."

• A Linear Dose-Response Relationship

When Generalization 1 speaks of the (percent) increase in mortality rate, it implies that there is only a single percent increase in cancer mortality rate, per rad of exposure, throughout the large dose-range of relevance for humans. A constant risk-per-rad at all dose-levels corresponds to direct proportionality, or the linear dose-response model. As more human evidence accumulates, it keeps confirming that the dose-effect relationship is either linear or quite possibly supralinear, but none of it suggests a quadratic curve with falling risk-per-rad as dose falls.

The most recent follow-up data from Hiroshima and Nagasaki again support linearity[12]. Dose-groups in that study range from about 13 rads to 300 rads. Among some 150 types of cancer, only the commonest types like breast cancer and thyroid cancer are providing enough cases to analyze separately.

In 1985, the N.I.H. Group[11] acknowledged that the evidence on breast and thyroid cancers "strongly favors linearity", and in 1983, Wakabayashi and co-workers reported that the data on all cancer-types combined among the atomic bomb survivors suggest a linear model, and in a linear-quadratic model, "the linear term is significant whereas the quadratic term is not"[12]. Indeed, the quadratic term was negative. Since the data from Hiroshima-Nagasaki show linearity for all cancer-types considered together, and since the data show linearity for breast and thyroid cancers each analyzed separately, it is obvious that the combined remaining types of cancer must also show direct proportionality between increased cancer risk and radiation dose.

In 1970, Stewart[37] had already demonstrated that linearity holds, for diagnostic X-rays, in the region of 0.25 to 1.25 rads.

• No Protection By Dividing Doses

Generalization 1 implies also that the rate of excess cancer per rad of dose is unaffected by the rate of dose-delivery; a single percent increase applies to a rad, whether it is delivered in one instant or over a year. Boice[13], in a careful study of breast-cancer induction in women, concluded:

"The observation that multiple low-dose exposures did not produce significantly fewer cancers per unit dose than less highly fractionated

larger exposures suggests that radiation damage is cumulative and that highly fractionated X-irradiation may be as effective in inducing breast cancer as single or less fractionated exposures."

In 1985, the N.I.H. Working Group[11] (p.26) acknowledged this study and others showing that, where human evidence exists on the question of divided doses, it shows no reduced risk per rad when a given dose is divided into smaller doses.

• No Harmless Dose, For Carcinogenesis

Generalization 1 implies that it holds at every dose, all the way down to zero dose. No exception for the lowest dose-range is suggested, and no completely harmless dose is implied. Rather, cancer-risk is proportional to dose, right down to the lowest conceivable dose . In Section 2 of this paper, we shall offer scientific disproof of the notion that a safe "threshold dose" exists with respect to human carcinogenesis.

• IMPLICATIONS AND STATUS OF THE SECOND LAW

One of the important consequences of the second law is that quantitative evidence about radiation-induction of common cancers can be extended to less common cancers for which statistically significant proof is likely to be absent. It is likely to be absent because radiation induces excess cancer in proportion to a cancer's spontaneous rate, as the first law says.

An early assault on Generalization 2 was made in 1972 by the BEIR-1 Committee[14], which reached retroactively into a study published in 1965 by Court-Brown and Doll[15] . BEIR-1 raised one of Court-Brown and Doll's crucial dose-estimates by four-fold. In 1980, the BEIR-3 Committee[6] quietly undid this challenge to Generalization 2 by restoring the dose in question approximately to its 1965 value (discussion in Gofman[3], 1981, pp. 327-32).

As of today, inspection of the combined evidence from Smith and Doll[16], Smith[17], Kato and Schull[18], Schull[19], and Wakabayashi and co-workers[12] provides no reason whatever to think that one type of cancer increases, over its spontaneous rate, by a different percentage per rad than any other type.

With respect to the irradiated ankylosing spondylitic patients, Smith[17] reports that " with the exception of tumors of the spinal cord and nerves, the excess mortality from cancers of heavily irradiated sites was increased roughly in proportion to the expected number of cancers of each site based on general population rates."

Reporting on the Hiroshima-Nagasaki series, Kato and Schull[18] find the same phenomenon (p.404):

"As shown in Figure 1, the 90 % confidence limits of the relative risks of breast, stomach, lung, and colon cancers overlap each other, and the relative risk of cancers of all sites (except leukemia) is within the confidence limit, so, statistically it cannot be said that the relative risk differs according to target organ."

In Schull's 1984 paper[19], he confirms that "there is as yet no statistically persuasive evidence that the relative mortality risk differs according to target organs."

These statements of Kato and Schull, based upon the single largest body of evidence in existence for human radiation carcinogenesis, provide powerful confirmation of Generalization 2. The reason for some of the early doubt was that for certain cancers, the number of cases available for analysis was very low, and so there were wide confidence limits for the percent increase per rad. With increased follow-up time, the number of cases also increased, the confirming evidence for Generalization 2 grew steadily, and now very strong statements are coming from Kato and Schull.

• STATUS AND IMPLICATIONS OF THE THIRD LAW

There exists virtually unanimous assent now to the third law. In 1985, the N.I.H. Working Group[11](p.18) joined the mainstream by saying, "One of the most interesting observations to come out of the Japanese A-bomb survivor studies, which are based on a large population of all ages in 1945, is that the risk of radiation-induced cancer depends strongly on age at exposure."

Indeed, we have shown (Gofman[3],1981, Chapter 9) that, when a population of normally mixed ages is irradiated, about 73 % of the radiation-induced cancers will develop in people at or below 20 years of age at exposure.

• IMPLICATIONS FOR ASSESSING CHERNOBYL

Although all three of these laws were regarded in 1969 as outside the mainstream of wisdom, the real-world human evidence accumulated by 1986 has made them the mainstream and has cast the contradictory propositions on the sandy fringes of the riverside. The out-dated propositions must be avoided, if society wants any realism in assessments of Chernobyl's cancer-consequences.

(2) DISPROOF OF A SAFE "THRESHOLD DOSE" FOR CARCINOGENESIS

• THE EXISTENCE OF REPAIR MECHANISMS

There has never existed any valid evidence at all for a safe "threshold dose" of radiation with respect to human carcinogenesis; major flaws in "threshold" studies using doses from natural background and using the very lowest dose-group of A-bomb survivors have been analysed in extenso in Gofman[3] (pp. 227-31, 386-7, 566-72, 672-83). But after the Chernobyl accident, the idea has been predictably voiced here and abroad that populations which received its fallout may be protected from any cancer consequences by the existence of some safe threshold dose. Indeed, some Soviet officials are apparently attempting to deny that the health of anyone outside the Soviet Union was injured[20], according to the Associated Press.

Once upon a time, the safe-dose idea had some plausibility, since repair mechanisms do exist for at least certain types of damage to chromosomes and genetic molecules, but in the light of accumulating human evidence --- both from epidemiology and from cell-studies in vitro --- the safe-dose idea with respect to human carcinogenesis has become indefensible, as we shall show.

• REPAIR-TIME AND REPAIR-DOMAINS

There are certain types of injury to DNA and chromosomes whose repair (and misrepair) can be observed and measured in cell studies. It is observed by workers in the field that it is probably failure to repair DNA, or its misrepair, which often causes the chromosome aberrations which endure[21, 22]. It is well known by now that chromosome deletions and translocations are highly associated with Wilm's tumor[23], retinoblastoma[24], kidney cancer[25], and numerous types of blood cancer[26]. It may turn out that certain disturbances in chromosomal material are key to cancers of all types[3].

With respect to the question of repair providing a safe threshold dose of radiation, studies of human cells in vitro, following X-irradiation, provide some important parameters.

(1) Such studies indicate that whatever repair is achieved is complete within 6 hours or less after irradiation, even at doses of 100 and 200 rads[21, 27, 28]. Indeed, almost all the repair occurs within the first 2 hours after irradiation, and by 3 hours, the repair-curve is flat. In Table 3, we have assumed that 8 hours are required for full repair, to be extremely cautious.

(2) Such studies confirm what we already know from chromosome studies of

living humans: not all injuries get repaired, even when cells have infinite repair-time, and some attempts to repair end in misrepairs.

(3) The cell studies suggest that each cell-nucleus has more than one set or team of repair enzymes. The speed at which the early repair is occurring even at doses of 100 and 200 rads per exposure makes it unreasonable to suppose that a single "crash-cart" of repair enzymes is servicing all the genes and all the chromosomes (see Table 1, Note 2). It is far more likely that each chromosome has at least one repair set of its own, and possibly many. In other words, it seems very unlikely that, with respect to repair, the entire nucleus is a single domain serviced by only one repair team or "crash-cart."

When we come to the epidemiological evidence, below, it will be clear how repair-speed and repair-domains are relevant to the indefensibility of a safe threshold dose.

• THE MINIMAL CHALLENGE TO REPAIR MECHANISMS

For repair to provide a safe threshold dose of radiation, below which no radiation-induced cancer occurs, repair of the relevant injuries must work perfectly below some dose or dose-rate. If human evidence shows that repair mechanisms are failing to prevent radiation-induced cancer when such mechanisms are faced with the least possible challenge, this amounts to proof that no safe dose exists with respect to human carcinogenesis.

So it is important to explore in detail what dose or dose-rate constitutes the least possible challenge to the repair mechanisms in the cell nucleus.

• One Primary Ionization Track Per Nucleus

By definition, a radiation dose of 100 millirads is simply the deposition of 10 ergs of energy per gram of tissue. The deposition is not uniform. The biologically important characteristics of low LET radiation are that its energy is carried through tissue by high-speed electrons and that the transfers of this energy occur in extremely localized or concentrated fashion, unlike the even diffusion of heat energy. A dose of 400 rads is equivalent in heat to only 0.001 calorie per gram of tissue --- enough to provoke the tiny fever of $0.001^{\circ}C$ --- yet 400 rads of ionizing radiation to the whole body will kill about half the humans acutely exposed to it.

Unlike toxic substances, which can be diluted indefinitely to lower and lower concentration in solution, ionizing radiation cannot be evenly diffused into cells. For low LET radiations such as X-rays, gamma rays, or beta particles, the minimal event is one primary ionization track. Either a cell nucleus experiences such a track, or it does not (discussion in Gofman[29] pp.275-6 and Gofman[3], pp.405-7).

The minimal possible challenge to the repair mechanism from ionizing radiation is therefore the traversal of the cell nucleus by just one primary ionization track.

• DOSE-RATES CORRESPONDING TO ONE PRIMARY TRACK PER NUCLEUS

At what dose or dose-rate does every cell-nucleus of an irradiated tissue experience, on the average, only one primary ionization track? Table 1 provides answers, which vary with the energy of the radiation, its linear energy transfer (LET), and the size of irradiated cells. Technical Appendix 1 shows how the answers were obtained, and the basis for 11.4 microns as the appropriate cell-size for most human tissues.

Table 1:

Millirads Causing Only One Primary Track Per Nucleus In All Cells Of Irradiated Tissue

	Cells 10 microns	Human Average Cells 11.4 microns	Cells 15 microns
Cesium-137	165 mrads	125 mrads	70 mrads
Radium-226	170 mrads	130 mrads	75 mrads
50 KEV X-rays	490 mrads	375 mrads	215 mrads
40 KEV X-rays	575 mrads	445 mrads	255 mrads
30 KEV X-rays	705 mrads	540 mrads	310 mrads

Note 1: At half the given doses, half the cell nuclei completely escape primary ionization tracks, and for those particular cells, the dose would be zero. Example: when tissue consisting of 11.4-micron cells receives a dose of 270 millirads from 30 KEV X-rays, half the cell-nuclei experience no primary track at all.

Note 2: 30 KEV represents the average energy of most medical diagnostic X-rays when peak kilovoltage is about 90 KEV, and 50 KEV represents the average energy used in cell irradiation studies when the peak kilovoltage is about 150 KEV. A dose of 100 rads from 50 KEV X-rays corresponds (from Table 1) to 100 rads / 0.375 rad per track = 267 primary tracks per nucleus.

As the dose to an irradiated tissue falls below the corresponding value given in Table 1, the number of injured nuclei falls, but the challenge to the repair system inside those nuclei which are hit does not fall because no cell nucleus --- and no repair mechanism --- can experience a challenge less than one primary ionization track. For 11.4-micron cells irradiated by 30 KEV X-rays, many nuclei receive no hits at all at a dose of 1 millirad, but for the particular nuclei which are hit, the injury experienced at 1 millirad is equal to the injury experienced at 540 millirads.

• DOSE-RATES CORRESPONDING TO ONE PRIMARY TRACK PER REPAIR-DOMAIN

For a given cell-size and type of radiation, Table 1 provides the doses at which the repair mechanism faces the minimal challenge if there is only one repair team or "crash-cart" available to the entire domain of the nucleus. But we find no evidence for only one team, and no evidence that experts in the field consider a single single "crash-cart" per nucleus to be plausible.

If there is more than one "crash-cart" for repair per nucleus, then the minimal challenge for the repair squad is one primary ionization track or hit per repair-domain, not per nucleus. For instance, if there were 10 repair-domains per nucleus, all the dose-entries in Table 1 would have to be raised by a factor of 10 in order for each repair-domain to experience the challenge of 1 primary ionization track.

In Table 2, the doses in Table 1 are adjusted to correspond with 23 repair-domains per nucleus (one domain per pair of chromosomes), which seems like a very conservative approach when 46 suggests itself as a more likely number, and when the true number may be hundreds or more. Both Table 1 and Table 2 are used for the analysis made in Table 3.

Table 2:

Rads Causing Only One Primary Track Per Repair-Domain In All Cells Of Irradiated Tissue

Basis: 23 repair-domains per cell-nucleus.

	Cells 10 microns	Human Average Cells 11.4 microns	Cells 15 microns
Cesium-137	3.8 rads	2.9 rads	1.6 rads
Radium-226	3.9 rads	3.0 rads	1.7 rads
50 KEV X-rays	11.3 rads	8.6 rads	4.9 rads
40 KEV X-rays	13.2 rads	10.2 rads	5.9 rads
30 KEV X-rays	16.2 rads	12.4 rads	7.1 rads

• DESCRIPTION OF LOW-DOSE EPIDEMIOLOGICAL EVIDENCE

Some values from Tables 1 and 2 can be applied to the existing epidemiological evidence of radiation carcinogenesis at very low doses in humans in order to answer the central question: is "repair" failing to prevent radiation-induced cancer even at doses and dose-rates which present the minimal challenge to repair systems --- namely, 1 primary ionization track or hit per repair-domain.

There are at least 5 human studies, widely recognized to be well done and "mainstream," which show radiation-induction of cancer at very low dose-rates. Four of the five studies are described in Gofman[3] ; the Baverstock study[30,31] of British luminisers appeared later.

In three of the five studies, the total accumulated dose was high, but the time-interval between small exposures was far more than ample for repair-mechanisms to achieve completely whatever protection they can against radiation-induced cancer.

Before testing the studies for their dose-rates, we shall briefly describe the nature of each.

• The Nova Scotia Fluoroscopy Study

Myrden and Hiltz[32] studied 243 women who (in the course of tuberculosis treatment) had chest fluoroscopies with the beam from front to back, and with an estimated dose to the breasts of 7.5 rads per fluoroscopy. Time between fluoroscopies was days or weeks, and the total dose accumulated per woman was about 850 rads. Breast cancer was observed at more than six times the expected rate during a limited follow-up period.

• The Israeli Scalp-Irradiation Study

Modan and co-workers [33] reported on the excess of thyroid cancer observed among almost 11,000 Israeli children who received X-radiation for treatment of tinea capitis (ringworm of the scalp). The estimated thyroid dose per child was 7.5 rads total. Thyroid cancer was observed at five times the expected rate during a limited follow-up period.

• The Massachusetts Fluoroscopy Study

Boice and Monson [34] also studied women who had received repeated chest fluoroscopy during tuberculosis treatment; in their series, the beam was usually from back to front , and the estimated breast-dose was 1.5 rads per exam. The accumulated breast dose was about 150 rads. Among the women whose average age was 20 years at the time of irradiation, breast cancer was observed at more than twice the expected rate during a limited follow-up period.

• In-Utero Irradiation

Stewart [35, 36, 37] compared the X-ray history of children who died of cancer or leukemia in childhood with the X-ray history of matched controls who had no malignant disease. She demonstrated that diagnostic pelvimetry, irradiating the fetus in utero, provoked about a 50% increase in the frequency of childhood cancer and leukemia. She was able to demonstrate an excess even from single-film exams, with an estimated dose to the fetus of 0.25 rad (250 millirads). Her work has been confirmed by several additional analysts and series [38, 39, 40, 41, 42, 43].

• The British Luminisers

In 1981 and 1983, Baverstock and co-workers [30, 31] reported highly significant proof of breast-cancer induction by radiation in female workers who applied radium-226 to luminous instruments in Great Britain. Baverstock was able to rule out internal radiation by alpha particles as the cause, and identified external gamma radiation as the source of the radiation injury. The dose-rate of external gamma radiation to the breasts was, by measurement, 500 millirads per week or less. For a 40-hour week, this represents a dose of 12.5 millirads per hour, or 100 millirads per 8 hours (the maximum time consumed by human cells in repairing X-ray injuries). The average dose-rate must have been even lower. Among the women whose average age was 20 years at the time of irradiation,

breast cancer was observed at twice the expected rate during a limited follow-up period. The total breast dose accumulated by the young women over the work-years was 40 rads.

• ANALYSIS OF THE EPIDEMIOLOGICAL EVIDENCE

In Table 3, the five studies described above have been tested to ascertain whether or not the excesses of cancer were observed at dose-rates representing the minimal possible challenge to repair-mechanisms in the nucleus. Most of the entries in Table 3 are "1 track," the minimal possible challenge. In other words, existing human evidence already is showing that repair is not able to prevent excess malignancy even at minimal dose-rates.

One can quibble about cell-size, uncertain dosimetry in one study or another, and number of repair-domains per nucleus, but even if one invokes the probably preposterous notion of a single "crash-cart," the evidence in Table 3 combines to tell realists that repair provides no safe threshold dose.

Repair must work perfectly in order to provide a harmless dose. If it were working perfectly at the minimal dose-rates considered in Table 3, then there would be no cumulative carcinogenic effect to observe from repeated exposures. Repair most probably does a lot of good for human health, but what epidemiologists observe here is the residual and unrepaired injury.

In the scalp irradiation series, the single "crash-cart" had infinite time to cope with only 14 primary tracks, but it could not. In the Nova Scotia series, the "crash-cart" had far more repair-time between exposures than the few hours in which repair is observed to be completed even in cell-studies performed at doses of about 200 rads (about 533 primary tracks per nucleus); yet repair could not cope successfully with only 14 tracks. In the Massachusetts series, the "crash-cart" failed to cope successfully with just 3 primary tracks.

When one also considers the evidence from the in-utero and British luminiser series, the story is clear --- if wishful thinking is banished from radiation science.

Table 3:

Primary Ionization Tracks Per Repair-Domain in Five Human Studies

	Single Repair-Domain Per Nucleus; Divisor from Table 1	23 Repair-Domains Per Nucleus; Divisor from Table 2
Nova Scotia Fluoroscopy Dose = 7.5 rads per exposure Energy = 30 KEV X-rays	7.5 / 0.540 = 13.88 14 tracks	7.5 / 12.4 = 0.60 1 track (Some domains having 0)
Israeli Scalp Irradiation Dose = 7.5 rads, one time Energy = 30 KEV X-rays	7.5 / 0.540 = 13.88 14 tracks	7.5 / 12.4 = 0.60 1 track (Some domains having 0)
Massachusetts Fluoroscopy Dose = 1.5 rads per exposure Energy = 30 KEV X-rays	1.5 / 0.540 = 2.77 3 tracks	1.5 / 12.4 = 0.12 1 track (Most domains having 0)
In-Utero Series Dose = 0.250 rad per single-film exam Energy = 30 KEV X-rays	0.250 / 0.540 = 0.46 1 track (Many nuclei having 0)	0.250 / 12.4 = 0.02 1 track (Most domains having 0)
British Luminisers Dose = 100 millirads per 8 hours Energy = Radium-226	0.100 / 0.130 = 0.77 1 track (Some nuclei having 0)	0.100 / 3.0 = 0.03 1 track (Most domains having 0)

• CONCLUSION ON THE "SAFE THRESHOLD"

The expression of radiation-induced cancer at minimal dose-rates shows that it is scientifically unreasonable --- even ludicrous --- to entertain hope of finding a "safe threshold" based upon "repair." Any repair-process which fails to prevent radiation-induced cancer at minimal delivery-rates of ionizing radiation is inherently unable to provide a safe or harmless dose. The combination of physics, epidemiology, and cell studies in vitro, provides overwhelming evidence that a safe "threshold dose" of ionizing radiation simply does not exist for human carcinogenesis.

In 1980, the BEIR-3 Committee [6] declared that, from epidemiologic science alone, "the existence or non-existence of a threshold dose is practically impossible to determine" because the size of the human series

required to achieve statistical significance would be huge if the threshold were very low. However, the determination is not impossible if the already existing human evidence from epidemiology is combined with the physics of ionizing radiation. The non-existence of a safe "threshold dose" for human carcinogenesis has now been determined.

• IMPLICATIONS FOR THE LINEAR DOSE-RESPONSE CURVE

Human epidemiological evidence supports the linear dose-response relationship (see Section 1 of this paper). Table 2 shows why the relationship must remain linear below a dose of 12.4 rads (for 30 KEV X-rays). At doses lower than 12.4 rads, the fraction of repair-domains which experience any traversal decreases in direct proportion to dose, while those domains which do experience traversal continue to experience the same minimal dose-rate they experienced at 12.4 rads, namely 1 primary ionization track per repair-domain. The combination of the existing human evidence and the information in Table 2 demonstrates that linearity cannot possibly exaggerate the carcinogenic effect of radiation at very low doses.

(3) RECONCILIATION OF DISPARATE RISK-ESTIMATES

Among the ways to express cancer-risk from radiation exposure are percent increase over the spontaneous rate per rad, doubling dose (the dose in person-rads which doubles the spontaneous rate), cancer-dose (the dose in person-rads which corresponds to 100 % chance of 1 fatal radiation-induced cancer in the population), and number of radiation-induced cancer deaths per million person-rads. Person-rads are simply the product of a dose and the number of people who receive it. One hundred person-rads could be the sum of 60 people each receiving 1.5 rads (90 person-rads) plus 2 people each receiving 5 rads (10 person-rads).

• CORRECTIONS OF THE UNSCEAR RISK-ESTIMATE

In 1977 and again in 1982, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) [4, 5] asserted that the cancer-dose for a population of mixed ages is 10,000 person-rads per fatal radiation-induced cancer.

• Error In the Leukemia-Dose

UNSCEAR's value for cancer-dose starts with its mistaken value for leukemia of 1 case per 50,000 person-rads. UNSCEAR, using data from the Japanese A-bomb survivors as followed up through 1972 (UNSCEAR[4], Appendix G, Table 4, p.372), arrived at the wrong value by invoking an especially large carcinogenic effect for neutrons, allegedly present in the Hiroshima dose. By 1982, it was revealed that neutrons were virtually absent[63].

In the meantime, a much larger set of leukemia data from the A-bomb survivors was published in 1978 by Beebe, Kato, and Land[44]. It was clear from that data, and also from the breast-cancer data in the A-bomb series[64], that neutrons --- if present --- were <u>not</u> having an especially large carcinogenic effect. Therefore, we considered the leukemia data[44] (the Life Span Study through 1974), and analysed it without invoking any "neutron effect." These data yield a Leukemia-Dose not of 50,000 but rather of 10,000 person-rads per case (Gofman[3]).

If UNSCEAR had simply analysed its more limited set of leukemia data (through 1972) for both Hiroshima and Nagasaki without invoking any "neutron effect" (since the effect was imaginary), it would have arrived at a Leukemia-Dose of 11,500 person-rads per case, a value remarkably close to the value of 10,000 person-rads derived from the longer follow-up period. There is no doubt that UNSCEAR's leukemia-dose is wrong by a factor of 5.

• Error In the Cancer-Leukemia Ratio

UNSCEAR's second error occurred when it asserted that the ultimate ratio of fatal cancers to leukemia in an exposed population would be 5. In other words, for every radiation-induced leukemia, the population would show 5 fatal radiation-induced cancers. This assertion is the basis for UNSCEAR's setting its cancer-dose at 10,000 person-rads per fatal case, compared with its leukemia-dose of 50,000 person-rads per case.

But UNSCEAR's ratio of 5 failed to take proper account of what everyone now recognizes: the rate of radiation-induced leukemia passes its peak 7.5 years after radiation exposure, but at 7.5 years post-irradiation the rate of radiation-induced cancer is just beginning its climb. The delay in the appearance of the solid tumors has been observed in numerous human studies of low LET exposure, not only in the A-bomb study.

By 1977, the radiation-induced leukemia cases among the A-bomb survivors had peaked, declined, and finished, whereas the rate of radiation-induced cancers was still rising with no end in sight. Follow-up studies published later[12, 18, 44, 45] show that new radiation-induced cancer cases are continuing to appear, and very markedly among those who were young at the time of exposure. The true ratio of cancer to leukemia keeps growing.

The leukemia-dose in the A-bomb series is fixed and verifiable because the leukemia "story" there is over. A cancer-dose based exclusively on that one series would be less firm because the cancer "story" there won't be over for another 30 years or so. It would be a serious mistake to base the human cancer-dose on a single series when there are many additional studies of exposed humans besides the A-bomb survivors. When the worldwide human evidence is considered (Gofman[3]), the data show that the leukemia dose is likely to be 6,500 person-rads per case compared with 268 person-rads per fatal cancer, which reflects a ratio of 24.3, not 5.

Because no lifespan follow-up study has been completed anywhere, the ratio of 24.3 necessarily reflects some projections based upon the three "laws" (Section 1), which have already proven repeatedly to predict correctly what the next human evidence will show. By contrast, there is no scientific basis for UNSCEAR's assertion that 5 should be regarded as the ultimate and universal value for the cancer-to-leukemia ratio.

Therefore it is fair to say that UNSCEAR's cancer-to-leukemia ratio is wrong by a factor of (24.3 / 5), or 4.9.

• Reconciliation Of the Disparity

If we take both of UNSCEAR's errors into account, the UNSCEAR value for cancer-dose is off by the product of 5 and 4.9, or 24.5. The corrected UNSCEAR value for a mixed-age population would be (10,000 person-rads per fatal cancer) divided by (24.5) = 408 person-rads per fatal cancer.

This is very close to 268 person-rads per fatal cancer, the value from Gofman used in this paper to analyse the consequences from the Chernobyl accident.

It is natural if scientists disagree about the exact size of UNSCEAR's errors, but it would be unthinkable for responsible members of the scientific community to use UNSCEAR's obviously mistaken cancer-dose as it is, simply because it carries UNSCEAR's authority.

• CORRECTIONS OF THE BEIR-3 RISK-ESTIMATE

It is well known that members of the Committee on the Biological Effects of Ionizing Radiation (BEIR-3) [6] were split over the risk-values in their final report, which included a vigorous dissenting appendix by the BEIR-3 Chairman, Dr. Edward P. Radford. After the recall of the Committee's 1979 report by the National Academy of Sciences and its appointment of a special committee to arrange a compromise, final values emerged in 1980 as if from a black box; no way was provided for the reader to check, follow, or replicate the analysis. However, what we can do is to show, quantitatively, how the BEIR-3 values of percent increase (over the spontaneous rate of cancer) per rad of exposure are about 16 times lower than the human evidence requires.

• Erroneous Incorporation of Zero-Values

Because the BEIR-3 Committee did not use Generalization 2, it reviewed the data for each type of cancer separately. It relied almost exclusively on the Hiroshima-Nagasaki data. Obviously, if one subdivides a set of evidence enough times, eventually the data in many of its sections will become statistically inconclusive. After the Committee subdivided the existing data by age at irradiation, by sex, and by site of cancer, it had categories with excess cancer-rates which were not provably significant, and it treated all those categories as zero excess-cancers-per-rad.

These zeroes were especially important among A-bomb survivors who were between ages 0 - 9 years in 1945. Although many studies of childhood exposure have shown that young children are far more sensitive than adults to radiation carcinogenesis (data in Gofman[3], 1981), the first law correctly states that the sensitivity is most fully manifest as they reach the adult years when their spontaneous cancer rate rises. Therefore, when a population of mixed ages is irradiated, the youngest children are the slowest in showing the full consequences.

Ignoring this, the BEIR-3 Committee put three shocking entries in its Table V-14, "Estimated Excess Cancer Incidence (Excluding Leukemia and Bone Cancer) per Million Persons per Year per Rad, 11-30 Years after Exposure, by Site, Sex, and Age at Exposure "[6].

For males 0 - 9 years old at exposure:

Lung cancer 0.00

For females 0 - 9 years old at exposure:

Lung cancer 0.00
Breast cancer 0.00

It is nothing short of fantastic to use risk values of zero for two of the most important cancers when the most sensitive age-group is irradiated. Such entries mistakenly and seriously lower BEIR's risk-value for a population of mixed ages. Such a blunder is particularly surprising when the 0 - 9

year age-group of A-bomb survivors was already showing, with other cancer sites, a risk-rate 5 to 10 times higher than adults (see Table 4).

Two other cancers of major importance were prematurely assigned zero values at every age by the BEIR-3 Committee: prostate and uterine cancers. The mistakes were soon evident to all. In 1983, Wakabayashi[12] reported that the excess of prostate cancer in the Nagasaki bomb-survivors was consistent with the excess observed for most of the common cancers. In 1984, Wagoner[9], reported on the significant excess of uterine cancer observed among women irradiated in the pelvis.

It is hardly surprising, after BEIR-3 made so many major omissions, that its risk-values are seriously at variance with reality. If it had used Generalization 2, it would have avoided those errors.

Because of our confidence in the three laws, we never corrupted our risk-estimates with obviously false zeroes. Instead, we presumed that the "missing" cancers would ultimately display the same behavior as all the other types. And ever since 1969, additional evidence has repeatedly validated the presumption. More recently, we predicted in 1981 (Gofman[3], 1981, p.260) that female A-bomb survivors who were 0 - 9 years old at irradiation "will demonstrate a startling number of breast cancers induced by radiation."

The very next year, Tokunaga and co-workers[45] demonstrated that additional follow-up of the A-bomb survivors showed, among the females 0 - 9 years old at the time of bombing, that the relative rate of radiation-induced breast cancer already exceeds the rate for any other age-group. Meanwhile, the BEIR-3 Committee had built into its risk-analysis an underestimate of gross proportions for the most important single form of cancer among women.

• Consequent Percent-Increase-per-Rad Values

As a result of BEIR's false zero values and its erroneous use of a quadratic term, it arrived at the following values for risk (Table V-19, BEIR-3 Report[6]).

For males: 0.11 percent increase per rad.
For females: 0.15 percent increase per rad.
Average : 0.13 percent increase per rad.

Since the BEIR-3 Committee relied almost exclusively on the Hiroshima-Nagasaki study, we will compare its value of 0.13 % with the actual evidence

for the period 1950 through 1974, calculated from Beebe, Kato, and Land [44] in Gofman [3], Chapter 6, and presented in Table 4 below.

Table 4:

Observed Rates of Excess Cancer Among A-Bomb Survivors, Through 1974

(1) Age Group	(2) Person-Years	(3) Crude Percent Increase per Rad	(4) Product (2) x (3)
0 to 9	362,100	3.26 %	1,180,446
10 to 19	398,500	1.42 %	565,870
20 to 34	363,100	0.63 %	228,753
35 to 49	386,300	0.33 %	127,479
50+	177,700	0.0 %	0
Sum	1,510,200		Sum 2,102,548

Table 4 leads to the following value:
Weighted Percent Increase per Rad = 2,102,548 / 1,510,200 = 1.39 %.
When this value is compared with BEIR's, BEIR is clearly too low by a factor of 1.39 / 0.13 = 10.7 times. But that is not all.

• Erroneous Values From Inadequate Follow-Up Time

The crude percent increase per rad of 1.39 % is itself too low by at least 50 %, because it is based on the fifth through thirtieth years following exposure; the percent is seriously lower than one based on the tenth through fiftieth years following exposure because (a) it is decreased by values from the 5th-10th years after exposure when the ratio of irradiated cases to unirradiated control cases (ratio of observed/expected) is 1.0 or just beginning to diverge gradually from unity, and (b) the irradiated children have not even reached ages where their special sensitivity adds hugely to a population's cancer toll. If 1.39 % is increased by a realistic 50 %,the value becomes 2.085 % per rad.

When 2.085 % is compared with the BEIR's 0. 13 %, BEIR is clearly too low by a factor of 2.085 / 0. 13 = 16 times.

It is regrettable that some analysts insist on using inadequate follow-up periods as a basis for publishing inordinately low cancer rates per rad. And there is no reason for taking such analyses seriously. BEIR's error of 16-fold accounts for the disparity between its cancer-dose of 4,425 person-rads (Table V-4)[6] and Gofman's 268 person-rads[3].

• Reconciliation of the 16-Fold Disparity

If the BEIR Committee had done its work correctly, its percent increase per rad would have been in the neighborhood of 2.085 %. And how would that have compared with the value presented by Gofman in 1981 for the Whole-Body Cancer-Dose?

It would have been in complete harmony. Our value of 268 person-rads per cancer fatality is derived from the male Cancer-Dose for a mixed age population of 235.0 person-rads, and the female Cancer-Dose of 300.2 person-rads (Gofman[3], 1981, p.294).

These, in turn correspond with a male doubling dose of 43.5 person-rads and a female doubling dose of 49.5 person-rads, for populations of mixed ages. And since these values produce a 100 % increase in cancer rate over its spontaneous rate, it follows that:

For males, percent increase per person-rad = 100 % / 43.5 person-rads,
= 2.3 % per person-rad.

For females, percent increase per person-rad = 100 % / 49.5 person-rads,
= 2.02 % per person-rad.

Average for males and females = 2.16 % per person-rad.

The difference between 2.16 % and 2.085 % is utterly negligible. The way to reconcile the 16-fold disparity is no mystery.

• CORRECTION OF THE RADFORD RISK-ESTIMATE

The Chairman of BEIR-3, Edward P. Radford, disavowed the introduction of a linear-quadratic model for risk, and stated in his 1980 dissent that the human evidence supports the linear dose-effect model. Radford's own analysis of percent increase in cancer rate per rad was (from his Table 1, BEIR-3 Report[6])

For males: 0.31 % per rad.
For females : 0.67 % per rad.
Average : 0.465 % per rad.

The ratio of the Gofman and Radford estimates was therefore 2.16 % / 0.465 %, which is 4.645, in 1980.

The origin of Radford's underestimate is clear (p.303, BEIR-3 Report[6]): he,too, mistakenly assigned risk-values of zero to several important cancers. In addition, he failed to correct evidence from a limited-follow-up period so that it could properly represent what it purported to represent: <u>total</u> excess cancer mortality in a population following its irradiation.

Subsequently, Radford[47] has raised his risk-value somewhat. It is presently only 3.7-fold lower than ours.

• COMPARISON WITH THE N.I.H. ESTIMATE

The 1985 report of the N.I.H. Working Group virtually adopted BEIR-3's risk values without challenge. However, the N.I.H. Report made three notable exceptions[11].

First, it explicitly objected (p.242) to BEIR's use of zero to describe breast cancer risk in females exposed between 0 - 9 years of age. In a classic understatement, it said, "The two studies make it plain that the BEIR coefficient of zero for women exposed under 10 years of age is inappropriate and should be replaced."

Secondly, on breast cancer, it conceded (p.55) that the human evidence demands the use of the linear model rather than the linear-quadratic model.

Thirdly, it made the same concession about thyroid cancer (p.55).

• Small Remaining Disparity on Breast and Thyroid Cancers

How, then, does N.I.H.'s evaluation of breast-cancer risk compare with our own ? To compare risk-values for exposure of 1 rad to the breasts at age 20, we can work from the respective PC (probability of causation) values for a breast cancer appearing at age 44, as an illustrative case.

PC = R / (S + R), where R = the share of causation contributed by the radiation exposure and S represents the "spontaneous" share contributed by other causes.

The N.I.H. Breast Value: N.I.H.[11] (p.244-5) gives R = 0.00606 for such an illustration, where the spontaneous share (normalized) = 1.0 Therefore, PC = 0.00606 / 1.00606, or a PC value of 0.602 %.

The Gofman Breast Value: (1981, Table 56b)[3] gives R = 2.01 , where the spontaneous share = 93.35 for the same illustrative case. Therefore, PC = 2.01/(93.35 + 2.01), or a PC value of 2.1 %.

The Breast Ratio: The ratio of Gofman to N.I.H. for breast cancer risk per rad is 2.1 / 0.602 = 3.5. As we shall see for the other cancer, it is the N.I.H. committee which has the higher value.

A comparable illustration can be explored for thyroid cancer. Consider a female, exposed at age ten to 5 thyroid-rads, who shows a thyroid cancer at age 25.

The N.I.H. Thyroid Value: N.I.H.(p.257) shows R = 0.297 where spontaneous is 1.0. Therefore PC = 0.297 / 1.297 = 0.23, or a PC of 23.0 %.

The Gofman Thyroid Value: The Gofman tables for this illustration show R = 0.22 for 1 thyroid-rad where spontaneous = 8.00. When R is multiplied by 5 for a dose of 5 thyroid-rads, PC = 1.1 /(8.00 + 1.1) = 0.121, or PC = 12.1 %.

The Thyroid Ratio: For this cancer, the N.I.H. risk value is higher than our own by a factor of 23.0 / 12.1 = 1.9-fold.

• SUMMARY ON RECONCILIATION

For the two types of cancer which provide enough data for separate analysis, we and the N.I.H. Committee arrived at a "wash", with virtually the same risk-values. In one case, the N.I.H. is 3.5-fold lower and in the other case, 1.9-fold higher. It is clear where the mainstream is flowing.

(4) THE CANCER-DOSE FOR MIXED AGES

The Whole-Body Cancer-Dose is a ratio: a whole-body dose in person-rads per one fatal radiation-induced cancer. Gofman [3] has demonstrated extensively how the Whole-Body Cancer-Dose is derived for each sex and at various ages from the existing human evidence. As Generalization 3 indicates, the Cancer-Dose is far lower for children than for adults, and is very high for adults age 50 and older. The range (Gofman[3], 1981, Tables 21 and 22) is from about 65 whole-body person-rads for newborns to about 20,000 whole-body person-rads at age 55.

Risk Possibly Underestimated: Up to the 40th year following exposure to low LET radiation, there is evidence that the observed / expected ratio for solid cancers is increasing[3, 9, 30, 61]. There are as yet no studies with follow-up for longer periods. But we have data for leukemia, where the O/E ratio peaks about 7.5 years following a single exposure and then declines. Using this decline as a model, we have included in the Whole-Body Cancer-Dose the assumption that the O/E ratio for solid cancers from low LET exposures will also begin declining after it peaks. This assumption may underestimate the risk from such exposures.

• THE CANCER-DOSE FOR A MIXTURE OF AGES

Following a situation like the Chernobyl accident, the world is interested in assessing the overall excess number of cancer fatalities and non-fatal cancer cases for populations in which all ages are present.

A Cancer-Dose for mixed ages is available (Gofman [3]).We simply took account of the distribution of persons by age and sex in a population (the U.S. population

was used) and then weighted the Whole-Body Cancer-Dose for each age by the fraction of the population at that age. In a population of constant size, the distribution also remains virtually constant. The error introduced by this approximation is trivial except in a population with age distributions grossly and permanently different from the U.S. (details in Gofman[3],1981). The result obtained from such weighting gave the following Whole-Body Cancer-Dose for a population of mixed ages (both sexes included) (Gofman[3],1981,p.294):

268 whole-body person-rads per fatal cancer, or
268,000 whole-body person-millirads per fatal cancer.

• LEUKEMIA-DOSE FOR A POPULATION

There is unanimous agreement on treating leukemia separately from the solid cancers, because leukemia behaves differently with respect to speed of appearance, duration of radiation effect, loss of life-expectancy, and the absence of a definitive age-trend with risk-per-rad. The Whole-Body Leukemia-Dose for a mixed-aged population is estimated at 6,500 person-rads, or 6,500,000 person-millirads per leukemia case (Gofman and O'Connor, 1985)[46] .

(5) THE CHERNOBYL ACCIDENT: CANCER-LEUKEMIA CONSEQUENCES

• ELEMENTS OF THE CALCULATION

If in each country, we knew the whole-body radiation dose received by each person and if we added up those doses for the entire population, we would know the total person-rads or person-millirads received in that country. For illustration, suppose we had a country with 10^8 person-millirads of whole-body dose. Then,

Fatal cancers = 10^8 person-millirads / 268,000 person-millirads per cancer
= 373 fatal cancers from the Chernobyl accident.

Since there will be approximately one non-fatal cancer induced by radiation for each fatal cancer, there would be an additional 373 non-fatal cancers.

Leukemia cases = 10^8 person-millirads / 6.5×10^6 person-millirads per leukemia
= 15 leukemia cases.

• SOURCES OF DOSE FROM A NUCLEAR POWER ACCIDENT

There are several sources of exposure in areas where fallout occurs:

(1) Direct radiation of the whole body from gamma rays in the cloud of radionuclides passing over a population;

(2) Inhalation of radioactive substances from the passing cloud and from radionuclides re-suspended after deposition on the ground;

(3) Direct external radiation of the whole body by gamma rays emitted from radionuclides deposited on the ground;

(4) Internal exposure by ingestion of radionuclides with milk, water, meat, fruits, and vegetables.

One or another source of exposure will dominate, according to the type of accident and where a population is located with respect to the event.

• RADIOCESIUM AS THE DOMINANT MENACE

In most countries receiving fallout from the Chernobyl accident, it is clear that the major doses will come from gamma rays emitted from radionuclides deposited on the ground, and from internal radiation via food and water.

Fallout measurements show that a large quantity of radioactive cesium did come out of the Chernobyl reactor. The dose received from cesium-137 ($T_{\frac{1}{2}}$ =30.2 years) and cesium-134 ($T_{\frac{1}{2}}$ = 2.3 years) will be the most important part of the whole-body exposures. Of course, we do not deny additional doses from other nuclides. Even without the incremental dose they inflict, we can reach a good appreciation of Chernobyl's cancer and leukemia consequences if we are able to calculate the doses delivered by the cesium-137 and cesium-134, both from direct gamma radiation from the ground and from these nuclides in the food chain.

(6) THE SOURCES OF FALLOUT DATA

There are two major sources of multi-nation information available. The first is a series of reports from the World Health Organization (WHO)[48] in Copenhagen, and the second is a series of reports from the United States Environmental Protection Agency (EPA) [49] in Washington, D.C. The organizations issued their last reports, respectively, on June 12 and June 30, 1986.

These reports rely upon measurements provided by the various reporting countries. Some countries reported data rather professionally. Others, such as East Germany, reported none at all. The Soviet Union, in spite of its assurances of being forthcoming with data, provided no cesium-137 or cesium-134 measurements at all, until the Soviet report in late August[65].

In addition to the WHO and EPA reports, there are reports for some single countries, most particularly Finland[50, 51] and the United Kingdom[52].

We will describe below the kinds of measurements available, and their use in estimating doses from cesium-137 and cesium-134. In this paper's Technical Appendix, dose data are described country-by-country.

• Opportunity for Future Measurements

We can state at the outset that all the requisite measurements for a perfect assessment of Chernobyl's cancer consequences are far from available. To match existing measurements with exact population distribution would require a grid over each country with measurements of both the population within a particular grid-location and the cesium-137 and cesium-134 deposition in that same grid-location. It is regrettable that society is not set up to provide such information on a timely basis. However, if the will exists to obtain such data, the opportunity has not been lost (see Section 8).

• THREE KINDS OF AVAILABLE DATA AND THEIR HANDLING

• (1) The Best Type of Data

Here the country reports the integrated deposition of cesium-137 and cesium-134 up through the entire period of significant fallout. Among the reports, this occurs relatively rarely. Some countries provide the deposition values for a very limited period of the fallout, so that the true total deposition must be higher than the values reported.

• (2) The Next Best Type Of Data

Here the country reports the values for gamma-ray exposure from the deposition of all radionuclides on the ground for a specified date following the accident. These data can be used effectively to obtain indirectly what the cesium-137 and cesium-134 depositions were at the same locations.

The basis for such conversion from external gamma-ray dose to cesium values resides in the provision by the Finnish Centre for Radiation and Nuclear Safety of values for the percent of the total gamma-ray dose which is to be assigned to cesium-137. In the Finns' first report[50], they provide the datum that 1.8% (1.7% - 1.9%) of the total gamma-ray dose for April 29, 1986 is to be assigned to gamma-rays from cesium-137's decay (via barium-137m). In their second report[51], they provide the datum that 11% of the total gamma-ray dose for May 6-7, 1986 is to be assigned to gamma-rays from cesium-137. By using the daily decay curve of of gamma-ray dose for Uusikaupunki for the first two weeks, it is possible to interpolate and extrapolate the percent of the gamma-dose to be assigned to

cesium-137 for dates others than those for which data are provided directly. These assignments are listed in Table 5.

Table 5:

Percent Of Gamma-Ray Dose Assigned To Cesium-137 Gamma-Rays

Date Of Gamma-Ray Measurement	Percent Of Measured Dose Assigned To Cesium-137
April 29,1986	1.8%
April 30,1986	4.1%
May 1,1986	4.9%
May 2,1986	5.8%
May 3,1986	6.8%
May 4,1986	8.0%
May 5,1986	9.1%
May 6,1986	10.2%
May 7,1986	11.7%
May 8,1986	12.9%
May 9,1986	14.4%
May 10,1986	16.0%
May 11,1986	17.3%
May 12,1986	18.8%

The reason for the rising percent of the gamma-ray dose assigned to cesium-137 is that the cesium-137 hardly changes its output of gamma-rays during this brief period of about two weeks, whereas many of the short-lived nuclides are decreasing their output due to substantial decay during the same time period.

• (3) The Last Type Of Data

Here we are not provided either with gamma-ray dose measurements or with radiocesium deposition measurements, but we are provided with iodine-131 deposition measurements. From analysing other data where both I-131 and Cs-137 deposition data are available, we are able to estimate Cs-137 deposition indirectly from I-131 deposition data.

(7) METHOD: ILLUSTRATIVE USE OF THE DATA

• METHOD 1: BEST TYPE OF DATA (DENMARK)

Cesium-137 integrated deposition is available. Denmark did provide such data.

Denmark provided (WHO June 5, 1986 Report)[48] the following data (from 10 stations for May 15 through May 27) for countrywide contamination in Becquerels/m^2 of surface soil:

	Mean	S.D.	Max
Cs-137	1075	758	2943
Cs-134	602	424	3477

These reports note that the above values are corrected with respect to cesium-137 still present from weapons-test fallout.

For Cs-137: (1075 Bq/m^2) x (27 Picocuries/Bq) = 29025 pCi/m^2.

For Cs-134: (602 Bq/m^2) x (27 Picocuries/Bq) = 16254 pCi/m^2.

• Cesium-137 Dose, Method 1

From data for worldwide fallout from weapons testing, described by UNSCEAR[4] and summarized in Gofman[3](1981, p.548), we calculate that the total absorbed dose commitment is 0.66 millirads for each 1000 pCi/m^2. This includes the dose commitment both from external radiation from Cs-137 gamma rays coming from the ground, adjusted by UNSCEAR for weathering to an average depth of 3 cm, for body-shielding, and for time spent indoors, and from internal radiation from Cs-137 ingested via the food chain.

Internal doses vary by soil type, and here we are using average values observed from weapons fallout. Unfortunately for people in the Ukraine, UNSCEAR[4] estimates that a much larger internal dose will be received from cesium there than in most areas, due to special soil characteristics there. But for average conditions, of the 0.66 millirads total dose commitment from each 1000 pCi/m^2, UNSCEAR's estimate is that 70 % is from external dose and 30 % from internal dose.

For Denmark, therefore, we can make the following calculation of dose.

External Cs-137 Dose Commitment

External dose = (total deposition) x (dose per unit of deposition) x (external share) = (29025 pCi/m^2) x (0.66 mrads per 1000 pCi/m^2) x (0.70) =13.4 millirads.

Internal Cs-137 Dose Commitment

The internal share changes to 30 %, and therefore internal dose = (29025 pCi/m^2) x (0.66 mrads per 1000 pCi/m^2) x (0.30) = 5.7 millirads.

• Cesium-134 Dose, Method 1

External Cs-134 Dose Commitment : There are two factors to consider in evaluating the dose from Cs-134 for the same number of picocuries/m^2 as for Cs-137.

(a) The total average gamma-ray energy per disintegration for each nuclide.
Ratio of gamma-ray energy Cs-134 / Cs-137 = 2.52

(b) The mean life of Cs-134 atoms versus Cs-137 atoms (mean life = half life / 0.693).
Ratio of mean life Cs-134 / Cs-137 = 0.076

Relative Dose-Effectiveness: The relative dose-effectiveness of Cs-134 versus Cs-137 per pCi/m^2 deposition is the product of factors (a) and (b). Dose-effectiveness Cs-134 / Cs-137 = (2.52) x (0.076) = 0.19.

Calculation of Dose: The external dose commitment from Cs-134 is: (Relative Deposition Cs-134/Cs-137) x (Dose-Effectiveness Factor) x (External Cs-137 Dose). For Denmark, we have therefore Cs-134 external dose = (16254 pCi per square meter / 29025 pCi per square meter) x (0.19) x (13.4 millirads) = 1.4 millirads.

Internal Cs-134 Dose Commitment: There are two factors to consider.
(a) The average peak beta energy of Cs-134 versus that of Cs-137; the predominant source of internal dosage is from disintegrations via beta particles.

Ratio of beta-particle energy Cs-134 / Cs-137 = 0.65 / 0.51 = 1.275. (Note that some handbooks give 1.17 MEV as the Cs-137 beta energy. This is true for only 8 % of the disintegrations; 92 % go via the 0.51 MEV disintegration pathway.)
(b) The ratio of mean-life, calculated above to be 0.076.

Relative Dose-Effectiveness: This factor is the product of (a) and (b). Dose-effectiveness Cs-134 / Cs-137 = (1.275) x (0.076) = 0.097

Calculation of Dose: The internal dose commitment from Cs-134 is (Relative Deposition of Cs-134 / Cs-137) x (Dose-Effectiveness Factor) x (Internal Cs-137 Dose). For Denmark we have, therefore, Cs-134 internal dose = (16254 pCi per m^2 / 29025 pCi per m^2) x (0.097) x (5.7 millirads) = 0.3 millirads.

• Combined Cs-137 and Cs-134 Doses, Method 1

The combined external and internal doses from both nuclides, in Denmark = (13.4 + 5.7 + 1.4 + 0.3) millirads = 20.8 millirads.

• Cancer and Leukemia Consequences, Denmark

$$\text{Fatal Cancers} = \frac{\text{(Population Size) x (Dose in millirads)}}{\text{(268,000 person-millirads per fatal cancer)}}$$

$$= \frac{(5.1 \times 10^6 \text{ persons}) \times (20.8 \text{ millirads})}{\text{(268,000 person-millirads per fatal cancer)}}$$

= 396 fatal cancers, which we round off to 400, in Denmark.

Non-fatal Cancers, additional = 400 cases.

$$\text{Leukemias} = \frac{(5.1 \times 10^6 \text{ persons}) \times (20.8 \text{ millirads})}{\text{(6,500,000 person-millirads per leukemia)}}$$

= 16.3 leukemias, rounded off to 16 cases.

This completes the analysis for Denmark, based upon what we are calling the best type of data, namely, integrated Cs-137 and Cs-134 deposition on the ground, averaged over the country.

• METHOD 2: NEXT BEST TYPE OF DATA (POLAND)

Gamma-ray exposure from deposition of all radionuclides on the ground is provided. Poland reports data usable for this illustrative example. The WHO[48] report of May 30, 1986, provides gamma-ray exposure from the ground for "all Poland" for the very early period, April 29, 1986, which is ideal since the 1.8 % factor for the contribution by cesium-137 applies correctly. Although we would much prefer to have separate gamma-ray measurements and population distributions for each part of Poland, such data are not supplied. The measurement supplied is, for "all Poland", a range of 20-1000 micro-roentgens per hour, or 20-1000 μR/hr. Subtracting 12 μR/hr for background, we have 8-988 μR/hr as the range, outdoors, free-in-air. So we use Criterion II (see Technical Appendix 2) to derive a gamma dose for Poland of 249 μR/hr.

• Cesium-137 Dose, Method 2

Since cesium-137's contribution to the gamma dose is 1.8%, the Cs-137 gamma dose = (0.018) x (249 μR/hr) = 4.48 μR/hr. We are interested in calculating the Cs-137 dose for the whole first year, and thereafter for the entire mean-life of the Cs-137 atoms. That mean-life is $T_{\frac{1}{2}}$ / 0.693, or about 43.5 years. The first-year dose is only 2.3 % of the total dose commitment. The total dose is 43.5 times the first year dose.

If all the deposited Cs-137 were to remain right on the surface for the first year (and thereafter), the calculation would simply involve multiplication of the early deposition dose by the number of hours in a year, 8760 hours per year. But the cesium-137 has been found to work its way into the soil during the first year, with the result that the average external dose is appreciably lower than it would be if the Cs-137 had all remained on the surface. How much lower?

Devell and co-workers [53] have given a value for external dose one meter above the ground of 0.0811 mR/yr per 1000 pCi/m^2 --- provided the Cs-137 remains on the surface of the earth for the entire period of one year.

Beck's work is cited by UNSCEAR [4] as leading to the conclusion that the cesium-137 works its way into soil, with the establishment of an exponential profile for the Cs-137 , with a mean depth of 3 cm. For the average dose in the first year, UNSCEAR gives a value of 0.033 mR/yr per 1000 pCi/m^2. The cesium apparently stabilizes at this distribution in soil, and the average value for the first year can be used for all the subsequent years in estimation of dose commitment over the mean-life of the Cs-137.

Therefore, the value we would get for external dose one meter above the ground for deposited Cs-137 is too high if we use the very early dose. The correction factor is 0.0811 / 0.033, or 2.46.

In our analysis, we have, for Poland, an external dose of 4.48 μR/hr. This must be divided by 2.46, yielding 1.821 μR/hr as the appropriate external dose per hour for the hours in the first year. Therefore, for the first year the total dose will be (8760 hrs) x (1.821 μR/hr), or 15,952 μR in the first year. And for the total dose commitment over the mean-life, we have (43.5 yrs) x (15,952 μR/yr), or 6.939×10^5 μR.

• Correction of the External Cs-137 Dose Commitment

The UNSCEAR [4] recommendation is that the external dose in μR should be reduced by a factor of 0.32 μrads/μR to take into account back scattering, shielding by the body itself, and time spent indoors, on the average. Therefore, the whole-body

absorbed dose from external Cs-137 = (0.32 μrads/uR) x (6.939 x 10^5 μR) , or 2.22 x 10^5 μrads. Thus, external Cs-137 dose = (2.22 x 10^5 μrads) x (1 mrad/1000 μrads), or 222 millirads.

• Internal Cs-137 Dose Commitment

Given the usual distribution of Cs-137 dose (70% external and 30 % internal), we must multiply the external dose by (0.3 / 0.7) or 0.43 to obtain the internal Cs-137 dose. Therefore, internal dose from Cs-137 = (222 mrads) x (0.43) = 95.5 millirads.

• Cesium -134 Dose, Method 2

Poland provides no data for the Cs-134 / Cs-137 ratio of deposition. We use the average value calculated from many other data in the WHO[48] and EPA[49] reports. Since the ratio is fixed in the reactor, use of the average ratio from such measurements is fully justified in the absence of actual measurements in a particular country. The average deposition ratio , Cs-134 / Cs-137 = 0.76.

• External Cs-134 Dose Commitment

The external Cs-134 dose is the (deposition ratio) x (dose-effectiveness factor) x (external Cs-137 dose). Borrowing the dose-effectiveness factor of 0.19 from Method 1, we calculate the external Cs-134 dose = (0.76) x (0.19) x (222 mrads) = 32.1 mrads.

• Internal Cs-134 Dose Commitment

This is the (deposition ratio) x (dose-effectiveness factor) x (internal Cs-137 dose). Borrowing the appropriate dose-effectiveness factor of 0.097 from Method 1, we calculate the internal Cs-134 dose = (0.76) x (0.097) x (95.5 millirads) = 7.0 mrads.

• Combined Cs-137 and Cs-134 Doses, Method 2

Total cesium dose, from above, = 222 + 95.5 + 32.1 + 7.0 = 356.6 millirads.

• Cancer and Leukemia Consequences, Poland

$$\text{Fatal Cancers} = \frac{\text{(Population Size)x(Dose in mrads)}}{\text{268,000 person-mrads per fatal cancer}} = \frac{(36.9 \times 10^6 \text{ persons}) \times (356.6 \text{ mrads})}{\text{268,000 person-mrads per fatal cancer}}$$

= 49,099 fatal cancers. This is rounded off to 49,000 fatal cancers.

Non-fatal cancers, additional, are 49,000 cases.

$$\text{Leukemias} = \frac{(36.9 \times 10^6 \text{ persons}) \times (356.6 \text{ mrads})}{\text{6,500,000 person-mrads per leukemia}} = \text{2025 leukemias (rounded off.)}$$

• METHOD 3: LAST TYPE OF DATA (ITALY)

This type of analysis is based upon Iodine-131 deposition on the ground, with conversion of such data to Cesium-137 deposition on the ground. Fortunately, there were few instances where this method had to be used. Italy was such a case. The EPA report of May 12, 1986,[49] provides values for I-131 deposition in five separate regions. The average is 269 nanocuries/m^2, or 269,000 pCi/m^2.

From excellent Swedish data on the ratio of I-131 to Cs-137 depositions,[49] daily, in the early days of the accident, we obtain a factor of 0.202 for converting from Iodine-131 deposition to Cesium-137 deposition. Therefore, average Cs-137 deposition in Italy = (0.202) x (269,000 pCi/m^2) = 54338 pCi/m^2. This value is used as if it were Type (1) data, (see especially EPA Report, May 12,1986).

• Cs-137 External and Internal Doses

Total Cs-137 dose = (0.66 mrads per 1000 pCi/m^2) x (54338 pCi/m^2) = 35.9 millirads. External share is 70%, or 25.1 millirads. Internal share is 30 %, or 10.8 millirads.

• Cs-134 External and Internal Doses

Using the deposition ratio from Method 2 and the dose-effectiveness ratio from Method 1, we obtain:

External Cs-134 dose = (0.76) x (0.19) x (25.1 mrads) = 3.6 millirads.

Internal Cs-134 dose = (0.76) x (0.097) x (10.8 mrads) = 0.8 millirads.

• Combined Cs-137 and Cs-134 Doses, Method 3

Total cesium dose, from above, = (25.1 + 10.8 + 3.6 + 0.8) millirads = 40.3 millirads.

• Cancer and Leukemia Consequences, Italy

$$\text{Fatal Cancers} = \frac{\text{(Population size) x (Dose in Millirads)}}{\text{(268,000 person-millirads per fatal cancer)}}$$

$$= \frac{(5.624 \times 10^7 \text{ persons}) \times (40.3 \text{ millirads})}{\text{(268,000 person-millirads per fatal cancer)}}$$

= 8457 fatal cancers, rounded off to 8450 fatal cancers.

Non-fatal Cancers, additional = 8450 cases.

$$\text{Leukemias} = \frac{(5.624 \times 10^7 \text{ persons}) \times (40.3 \text{ millirads})}{\text{(6,500,000 person-millirads per leukemia)}}$$

= 350 leukemias, rounded off.

• UNIFORM REDUCTION OF "FIRST-STEP" VALUES

The dose commitments from cesium derived above are not the final values used to assess the cancer consequences; they are "first-step" values. The final values are the entries in Table 6, which are lower.

We are confident that Methods 1, 2, and 3 provide very reasonable dose commitments from cesiums in the localities where some measurements were reported. But we could not know how representative those localities were for the whole country. For instance, the localities measured may sometimes have been the arbitrary locations of permanent monitoring equipment, or may often have been localities where rainfall produced much greater concern and much more fallout. The variability of fallout within some countries was illustrated by Poland, where gamma doses ranged from 8 μR/hour to 988 μR/hour on the same date. Therefore, before calculating "first-step" dose commitments, we tried to correct for such variability by using the two criteria stated at the beginning of Technical Appendix 2.

After obtaining "first-step" dose commitments for each country by Methods 1, 2 and 3, we obtained reasonable factors by which all "first-step" values could be reduced uniformly. We shall call these the "lowering factors."

• BASIS OF THE LOWERING FACTORS

The "first-step" dose commitments from cesiums correspond to "first-step" deposition-values for cesiums. These were easily obtained in picocuries per meter^2 for cesium-137 with a single equation. Because in Methods 2 and 3 the ratio is constant for the deposition of Cs-134 to Cs-137, the ratio of the dose commitment from each nuclide is likewise constant. The share of the nuclides' combined dose commitment which is contributed by the Cs-137 is always 0.89. And because a Cs-137 deposition of 1,000 pCi/m^2 gives an absorbed dose commitment of 0.66 millirads (see Method 1), the following equation can be applied for all countries where Methods 2 and 3 were used. Cesium-137 deposition in units of 1,000 pCi/m^2 = (0.89) x (dose commitment Cs-137 + Cs-134 in mrads) / 0.66 mrads per 1,000 pCi/m^2.

With this equation, we obtained average "first-step" values for cesium-137 deposition in pCi/m^2 for every country in Table 6. We multiplied by each country's area in meters^2 to get "first-step" values for <u>total</u> cesium-137 deposition in each country.

The sum of those "first-step" values was 2.73×10^6 curies of cesium-137 deposited in all the countries combined. By comparing this value with some conservative estimates of total cesium-137 released by the accident, we obtained two appropriate lowering factors which we applied to the "first-step" dose commitments.

• Cesium: Amount Released and Initial Inventory

Several groups have attempted to estimate the total quantity of cesium-137 released from the Chernobyl reactor. Knox [54] suggested a value of 3.0×10^6 curies. The Imperial College Group [55] in England suggested a much lower value of 1.4×10^6 curies released. It is hard to know whether one of these values is better than the other.

The initial inventory of Cs-137 at the time of the accident depends on the length of operation and refueling schedule. Estimates for the Chernobyl reactor have been offered [54, 56], based on approximately two years of operation, which place its cesium-137 inventory at 5.8×10^6 and 6.0×10^6 curies. However, because our objective is to determine a credible lower-limit on the cancer-consequences from the accident, we have used the much lower value of 3.53×10^6 curies as the cesium-137 inventory, which corresponds with one year's full-power operation.

From this minimal value, we are going to derive and apply (separately) two lowering factors. Their results are in good agreement with the Soviet report[65]; see foot of Table 6.

• FACTOR FOR CESIUM-137 DEPOSITION OF 1,990,000 CURIES

For one factor, we have assumed that 75% of the minimal cesium-137 inventory was released at the temperatures and disruption which occurred at the reactor: $(3.53 \times 10^6$ curies) x (0.75) = 2.65×10^6 curies released. After we assumed that 25% of this amount was deposited on lands and waters not considered in the areas of Table 6, the cesium-137 deposition was reduced to $(2.65 \times 10^6$ curies) x (0.75) = 1.99×10^6 curies. This compares with our "first-step" value of 2.73×10^6 curies deposited. Therefore this lowering factor for all the "first-step" dose commitments is (1.99 / 2.73) = 0.729.

• FACTOR FOR CESIUM-137 DEPOSITION OF 1,330,000 CURIES

For the other factor, we have assumed that 50% of the minimal cesium-137 inventory was released: $(3.53 \times 10^6$ curies) x (0.50) = 1.77×10^6 curies

released. Then this value was reduced for the 25% "loss" in areas not considered: (1.77×10^6 curies) x (0.75) = 1.33×10^6 curies deposited. Comparison with our "first-step" value of 2.73×10^6 curies leads to the lowering factor of (1.33 / 2.73) = 0.487.

• FINAL ENTRIES IN TABLE 6

After a dose commitment is lowered by one of the factors, it is multiplied by the country's population to obtain person-millirads, and then person-millirads are divided by 268,000 person-millirads per fatal cancer and 6,500,000 person-millirads per leukemia to obtain the entries for Table 6, as explained in Section 5 of this paper.

The two sets of entries for malignancies in Table 6 correspond to cesium-137 depositions of 1,990,000 curies and 1,330,000 curies respectively (Technical Appendix 2-B illustrates the country-by-country calculation). The lower value of 1.3 million curies is very close to the estimate by the Imperial College Group[55]. It may be much too low, especially if the initial Cs-137 inventory was about 6 million curies instead of the 3.53 million curies used in this paper.

Unfortunately, scientists must be skeptical about the validity of any Soviet statements concerning cesium-137 inventory or percentage released. Indeed, one must wonder how much the Soviets can know about the percentage released when the condition of their reactor is hidden under tons of sand, lead, and boron, and when the explosion rendered worthless any measurements at normal vents.

Moreover, the Soviets have an obvious interest in underestimating the amount of cesium released, and this interest is powerfully shared by many nuclear experts in other countries which have nuclear power plants, or plan to have them.

(8) RESULTING ASSESSMENT OF CHERNOBYL'S CANCER CONSEQUENCES

Table 6 shows that the Chernobyl accident will cause between 634,200 and 951,000 total cases of radiation-induced cancer, and between 13,100 and 19,500 cases of radiation-induced leukemia. (Table 6 is on page 39.) Which end of the range is the more credible?

• REALITY-CHECK ON TABLE 6's ASSESSMENT

For reasons of compassion, we would much prefer that the lower values from Table 6 be the true ones. On the other hand, we must recognize that it is the higher estimate which corresponds more closely with the "first-step" values derived from actual measurements (see Section 7 of this paper). And although we did not tabulate the results if the cesium-137 inventory was 6,000,000 curies instead of 3,530,000 curies, anyone can see by simple proportion that the total cancers would rise to a range of 1,000,000 to 1,600,000 from the same analysis.

In the absence of additional measurements, we will use the lower range based on the lower inventory.

A way does exist for the scientific community to make a reality-check on Table 6's assessment. The cesium-137 and cesium-134 are going to remain as fallout in the various regions for a long period of time. Even though cesium-137 measurements, made retroactively without "trays" to collect only fresh fallout, are complicated somewhat by residual Cs-137 from weapons-testing, the solution is still easy. An independent team of scientists could go to all the affected countries and measure the cesium-134 contamination, making samples which are coded and split before analysis whenever possible. There is no significant cesium-134 left from weapons-testing. From such measurements, reliable values of the Cs-137 fallout from Chernobyl could be obtained. The Soviet Union would necessarily have to agree to such testing by independent scientists. Whether that will ever come to pass in not known. But there can be no doubt that a correct final assessment of the cancer consequences from the Chernobyl accident can be validated if the will for such assessment exists.

Meanwhile, Table 6 reveals that a credible lower-limit on the cancer-consequences from the Chernobyl accident is:

317,100 fatal cancers
317,100 additional non-fatal cancers
13,100 leukemias.

647,300 malignancies.

It must be noted that the number 647,300 excludes cancers from the following

additional sources of exposure:

- (a) from external gamma-dose delivered from the ground by deposited radionuclides other than the radio-cesiums. This dose will add approximately 3% to each of the totals for malignancies in Table 6.

  For the lower estimate, the sum would become

  647,300 + 19,400 = 666,700 malignancies.

  For the higher estimate, the sum would become

  970,500 + 29,100 = 999,600 malignancies.

- (b) from inhalation and ingestion of the radio-iodines, which concentrate in the thyroid gland and can cause thyroid cancers and abnormalities;
- (c) from internal dose (via food, water, and inhalation) delivered from radionuclides other than radio-cesiums and radio-iodines;
- (d) from the passing radioactive cloud, which irradiated people directly with gamma rays.

• THE DISTRIBUTION OF DOSES OVER TIME

Exposure from Chernobyl's radioactive cloud occurred only once, but exposure from Chernobyl's cesium fallout extends through time, because of the 2.3 year half-life of cesium-134 and the 30.2 year half-life of cesium-137. Calculation shows (Technical Appendix 2-C) that approximately 50% of all the dose ever to be received from the cesiums from the accident will have been received in a little over ten years. About 2/3 of the dose ever to be received will have been received by about the 25th year after the accident. About 75% of the dose ever to be received will have been received by the 40th year.

The delivery of about 50% of the dose commitment during the first ten years after the accident means that about 50% of the cancers in Table 6 will result from that part of the exposure. However, the malignancies will definitely not appear simultaneously. Even if the dose had occurred in an instant instead of gradually over ten years, the leukemias would be spread over 25 years (with the peak excess about 7.5 years after the exposure), and the cancers would be spread over the

Table 6:

Cancer and Leukemia Tolls From the Chernobyl Nuclear Power Plant Accident
(Based Upon Dose Commitments In Millirads From Cesium-137 Plus Cesium-134)

Country or Region	Population	Method (see text)	Corresponding To Deposition Of 1,990,000 Curies Of Cesium-137				Corresponding To Deposition Of 1,330,000 Curies Of Cesium-137			
			Dose Commit. mrads	Fatal Cancers	Addit'l Non-fatal Cancers	Leuke-mias	Dose Commit. mrads	Fatal Cancers	Addit'l Non-fatal Cancers	Leuke-mias
Albania	2,500,000	(2)	12	110	110	5	8	73	73	3
Austria	7,600,000	(2)	174	4,900	4,900	200	116	3,300	3,300	135
Belgium	10,000,000	(1)	2	75	75	3	1.3	50	50	2
Bulgaria	8,600,000	(2)	172	5,500	5,500	225	115	3,700	3,700	150
Canada	22,125,000	(3)	0.4	33	33	1	0.3	22	22	1
*Czechosl.	15,500,000	(2)	52	3,000	3,000	125	35	2,000	2,000	83
Denmark	5,100,000	(1)	15	280	280	12	10	190	190	8
**Finland	4,800,000	(2)	249	4,450	4,450	180	166	3,000	3,000	120
France	54,540,000	(2)	58	11,800	11,800	480	39	7,900	7,900	320
Germany,W	61,400,000	(2)	172	39,400	39,400	1,600	115	26,300	26,300	1,100
Germany,E	17,100,000	(2)	201	12,800	12,800	530	134	8,600	8,600	350
Greece	9,700,000	(1)	3	110	110	5	2	72	72	3
Hungary	10,600,000	(2)	41	1,620	1,620	65	27	1,080	1,080	43
Ireland	3,100,000	(2)	1.3	15	15	1	0.9	10	10	0
*Italy	56,200,000	(3)	29	6,100	6,100	250	17	4,000	4,000	165
*Japan	119,500,000	(3)	0.8	360	360	15	0.5	240	240	10
S.Korea	33,900,000	(3)	0.6	75	75	3	0.4	50	50	2
Luxemb'rg	350,000	(2)	12	16	16	1	8	11	11	0
Nether'ds	14,400,000	(2)	12	640	640	26	8	430	430	17
Norway	4,130,000	(1)	86	1,300	1,300	55	57	880	880	37
***Poland	36,900,000	(2)	259	35,700	35,700	1,470	173	23,800	23,800	980
Romania	22,900,000	(2)	770	66,000	66,000	2,700	513	44,000	44,000	1,800
Spain	38,200,000	(2)	2.6	370	370	15	1.7	250	250	10
***Sweden	8,300,000	(1)	496	15,400	15,400	630	331	10,200	10,200	420
Switzer'd	6,500,000	(2)	236	5,700	5,700	240	157	3,800	3,800	160
Turkey	48,000,000	(2)	100	18,000	18,000	740	67	12,000	12,000	490
United K.	56,000,000	(2)	65	13,600	13,600	560	43	9,100	9,100	370
U.S.A.	235,000,000	(3)	0.05	44	44	2	0.03	29	29	1
*U.S.S.R.										
Ukraine	50,700,000	(2)	936	177,000	177,000	7,300	624	118,000	118,000	4,900
Byelor'a	9,900,000	(2)	714	26,400	26,400	1,100	476	17,600	17,600	730
Moldavia	4,080,000	(2)	125	1,900	1,900	80	83	1,300	1,300	55
Baltic R.	7,660,000	(2)	104	3,000	3,000	120	69	2,000	2,000	80
Moscow	8,400,000	(2)	40	1,250	1,250	50	27	830	830	35
Lening'd	4,700,000	(2)	148	2,600	2,600	110	100	1,700	1,700	75
Yugoslav.	23,000,000	(2)	185	15,900	15,900	650	123	10,600	10,600	430
Sum (all countries) (Rounded off)				475,500	475,500	19,500		317,100	317,100	13,100
			Total Malignancies = 970,500				Total Malignancies = 647,300			

*Czechoslovakia, Italy, Japan, USSR: The values in Table 6 are probably too low; details in Technical Appendix 2-A. We have no data for the area close to Chernobyl, and none for the Russian SSR except for Moscow and Leningrad.

**Finland: There have been serious inconsistencies in the Finnish data; details in Technical Appendix 2-A.

***Poland and Sweden:

Poland reported extremely high gamma-dose rates in Warsaw during the early days of the accident, but these values were later deleted from EPA reports as "too high" without any explanation (compare EPA reports of May 12 and 14 with the EPA report of June 4, 1986).

Sweden reported extremely high gamma measurements in Uppsala for April 29, but these high values simply disappeared from later reports without explanation (compare EPA reports of May 8 and 9 with EPA reports of May 12 and thereafter).

In epidemiological science, authorities cannot select only high measurements for checking; unless low measurements are checked for error with exactly the same amount of diligence, the net result is to create a bias toward lowering a whole set of measurements. Such practice is not acceptable in science.

August 22, 1986: The Soviets are estimating 1,000,000 curies of cesium-137 deposition within their own european regions [65]. Table 6 matches extremely well with the Soviet value. The higher estimate of dose and malignancies corresponds with cesium-137 deposition of 991,874 curies in european regions of the Soviet Union; see Technical Appendix 2-B. The lower estimate in Table 6 corresponds with 2/3 of that value, or 661,458 curies.

remaining lifespans of the irradiated population (with the peak excess occurring between 30-40 years after exposure).

• THE DISTRIBUTION OF IMPACT BY AGE

The third "law" of radiation carcinogenesis (Section 1 of this paper) means that children will be the most affected by the cesium fallout. Not only will they experience more fatal cases per 100,000 exposed individuals than will adults, but each cancer fatality means a far greater loss of lifespan for those irradiated young than for those irradiated at older ages. This point is demonstrated by considering three ages: newborn, age 25, and age 45 at irradiation.

When newborn males are irradiated, among those who do develop fatal radiation-induced cancer, the average loss of life expectancy is about 22.3 years. Half of those cases die before reaching age 54.5 years, and half die later.

By comparison, if irradiation occurs at age 25, among those who do develop fatal radiation-induced cancer, the average loss of life expectancy (for males) is 12.8 years. Half of such cases die before reaching age 67.5 years, and half die later.

And if irradiation occurs at age 45, among those who do develop fatal radiation-induced cancer, the average loss of life expectancy is about 8.7 years. Half of such cases die before reaching age 75.2 years, and half die later.

The calculations leading to the statements about loss of life expectancy are based upon Tables 21 and 56 in Gofman[3].

(9) DISCUSSION AND CONCLUSIONS

• THE SINGLE MOST SERIOUS INDUSTRIAL ACCIDENT EVER

It is correct to say that a single event --- the Chernobyl accident --- has caused between 600,000 and a million cases of cancer and leukemia. The radio-cesiums are on the ground, and humans are committed to receive the doses from them. To the extent that a share of the dose has already been received, a share of the malignancies is already underway, even though they will not become manifest, clinically, for years.

The Chernobyl accident obviously represents the most serious industrial tragedy in the history of mankind, and by a very large factor.

• THE QUALITY OF EVIDENCE

With respect to the proven human carcinogens, the existing quantitative evidence of human carcinogenesis by ionizing radiation is second to none (UNSCEAR[4], BEIR[6], GOFMAN[3], N.I.H.[11]). The data on ionizing radiation may be the strongest of all, and they cover virtually every site of human cancer. Moreover, several studies examine very low doses --- a total of 250 millirads in one series[25]; even the A-bomb survivors provide a large subset of people who received less than 20 rads of exposure[18]. In addition, studies of occupationally and medically exposed populations have contributed much evidence at low doses.

Coupled with the quantitative human evidence hard-won over the past half-century, the three generalizations described in this paper provide a very good assessment of the cancer consequences of the Chernobyl accident. The real problem we have in making such an assessment is simply the acquisition of dose data. The problem does not have to do with any mystery about consequences, once the doses are known.

• WHAT WE NEED, AND DO NOT NEED, TO ASSESS CHERNOBYL ACCIDENTS

On June 6, 1986, Mr. Stuart Loory, broadcasting from Moscow to many nations on the Cable News Network, reported that an agreement had been reached between Dr. Robert Gale of the U.S.A. and the Soviet Government to arrange for a lifetime study of the approximately 100,000 persons who received high doses from Chernobyl and were finally evacuated from the nearby area. Mr. Loory added that such a study might determine for radiation and cancer what we already know for cigarette smoking and cancer.

We can imagine nothing further from the truth than the suggestion that science has not yet firmly established a causal relationship between radiation exposure and human cancer. If the follow-up study of the Soviet high-dose group is promoted as necessary to establish this relationship, it will represent a cruel deception of mankind concerning the massive body of existing evidence which already demonstrates in quantitative detail the production of cancer by radiation, and at very low doses.

• A PREDICTION

We can predict with high confidence that an honest study of the proposed population sample will simply confirm --- but decades from now --- the magnitude of radiation production of cancer, a magnitude we know quite well prior to such

a study.

The existing human evidence provides a solid basis for assessing the Chernobyl toll. The credible lower-limit of malignancies from the cesium fallout is approximately 640,000 cases, and a credible upper-limit is probably 1,600,000 malignancies. Only additional and reliable measurements of cesium fallout, made by independent scientists, can narrow the range.

• IMPLICATIONS FOR MEDICAL, DENTAL, AND OCCUPATIONAL IRRADIATION

The findings in Section 2 of this paper that there cannot be a safe threshold dose of ionizing radiation with respect to human carcinogenesis, and that linearity cannot exaggerate the carcinogenic effect at very low doses, disprove the "hormetic" notion that exposure at low doses may protect humans against malignancies [62].

Also the findings of Section 2 have daily applicability for medical, dental, and occupational exposures. Although lip-service is generally paid to the absence of any safe dose, in reality the hazard at low doses is often dismissed as "purely theoretical." The findings presented here show why the hazard is not imaginary --- it is real.

The aggregate dose each year from diagnostic radiology is sufficient to cause about 78,000 radiation-induced cancers per year in the United States alone (Gofman-O'Connor [46], pp.365-70). Occupational exposures, in their aggregate, add another large number. The findings in Sections 2 and 3 of this paper provide ample evidence that measures to reduce individual doses would constitute a scientifically sound method of achieving large reductions in the human cancer-rate.

TECHNICAL APPENDIX 1
The Basis For Table 1

Part A of this Technical Appendix shows the basis for estimating the appropriate size of most human cells, and Part B demonstrates the series of calculations which produced Table 1 in the text.

• (A) DETERMINATION OF HUMAN CELL-SIZE

Because the size of human cells and their nuclei has an impact on the results in Table 1, the choice of appropriate size was not made casually. A search was made for electron micrographs of human tissue fixed with glutaraldehyde, for they should provide the most reliable dimensions in-situ for cell nuclei. Several histology atlases[57, 58, 59] and the twelve-volume work of Johannessen[60] were consulted for such micrographs. Specifically sought were micrographs from normal human tissue in which nuclei were prominent. Especially good were those micrographs where several nuclei were present, so that the one with the largest diameter could be chosen from the group. The largest was chosen because, in sectioning the tissue, some nuclei will not have been sectioned through their maximum nuclear dimension. The following nuclear dimensions were ascertained:

	Mean Nuclear Diameter
From 29 suitable micrographs of non-fetal human cells	5.9 micrometers
From 6 suitable micrographs of fetal human cells	6.1 micrometers
From 1 suitable micrograph of non-fetal human thyroid	5.7 micrometers
From 1 suitable micrograph of fetal human thyroid	6.9 micrometers
From 1 suitable micrograph of non-fetal human breast	5.5 micrometers

For the epidemiological studies evaluated in this paper, it is appropriate to start with the mean nuclear value, which is 5.9 micrometers. Two corrections of this dimension were made. First, because it is impossible to know that the nuclei pictured were cut exactly through the maximum dimension, a factor of 1.1 increase was applied. Second, because it is possible that fixation may have caused some shrinkage of cells, another factor of 1.1 increase was applied. The total correction applied = 1.1 x 1.1 = 1.21. So, diameter of nuclei for this paper is (5.9 μm) x (1.21) = 7.1 micrometers.

A very reasonable estimate, based on examining numerous cells in histology texts, is that cell-diameter is twice the nuclear diameter. Therefore, for this paper, cell-diameter is 14.2 micrometers.

A spherical nucleus with a diameter of 7.1 μm has the same volume as a cuboidal

nucleus of 5.7 μm per edge. A spherical cell with a diameter of 14.2 μm has the same volume as a cuboidal cell of 11.4 μm per edge.

• (B) CALCULATIONS PRODUCING TABLE 1

We shall present the calculations supporting the entries in Table 1 for the cell-size of 11.4 microns (cuboidal). The six steps are the same for cells of any other size. However, once a value for Step 5 has been obtained for one cell-size, the corresponding value for cells of any size can be obtained with the general formula presented here after Step 6.

• Expression Of 100 Millirads In MEV

1 rad means 100 ergs or 6.25×10^7 MEV delivered per gram of tissue.

100 millirads means the delivery of 6.25×10^6 MEV per gram of tissue.

• Step 1: Number Of Cell-Nuclei Per Gram Of Tissue

Density of tissue = 1.1 grams/cm^3.

Cell-size (cuboidal) = 11.4 micrometers per side.

Volume = 1481.5 μm^3.

Mass of one cell = $(1481.5\ \mu m^3)(1.1\ gms/10^{12}\ \mu m^3) = 1.630 \times 10^{-9}$ grams.

Number of cells per gram of tissue =

1.0 gram / (1.630×10^{-9} grams per cell) = 6.135×10^8 cells.

Number of nuclei per gram of tissue = 6.135×10^8 nuclei.

• Step 2: Number Of Primary Electrons Required To Deliver 6.25×10^6 MEV Per Gram Of Tissue

By using the energy per photo-electron below, we exaggerate the energy per primary electron since ionizing radiation converts also to Compton electrons of lower initial energy. Because the average energy is really somewhat lower than stated, all the values in Step 5 should be a bit lower and all the doses in Step 6 should be a little higher. We are using the higher energies in order to stay conservative in demonstrating the case against a safe "threshold dose."

For Cesium-137, the gamma ray is actually from Barium-137m decaying to Barium-137.

Radium's value of 0.596 MEV per photo-electron represents the weighted average gamma-ray energy from Radium-226 and its daughters.

50 KEV represents the average energy of photo-electrons when the peak kilovoltage of X-rays is about 150 KEV. 30 KEV represents the average energy of photo-electrons when the peak kilovoltage of X-rays is about 90 KEV (typical for medical diagnostic X-rays).

	(MEV per 100 mrads)	/ (MEV per electron)	=	(number of primary electrons for dose of 100 mrads)
Cesium-137	6.25×10^6 MEV	/ 0.662 MEV per e	=	9.44×10^7 electrons.
Radium-226	6.25×10^6 MEV	/ 0.596 MEV per e	=	1.05×10^8 electrons.
50 KEV X-rays	6.25×10^6 MEV	/ 0.050 MEV per e	=	1.25×10^8 electrons.
40 KEV X-rays	6.25×10^6 MEV	/ 0.040 MEV per e	=	1.56×10^8 electrons.
30 KEV X-rays	6.25×10^6 MEV	/ 0.030 MEV per e	=	2.08×10^8 electrons.

• Step 3: Number Of Cells Traversed By Each Primary Electron

The distance traveled by each type of photo-electron is its initial energy divided by the energy it loses per micrometer of tissue traversed (its linear energy transfer, or LET). The number of cells traversed is the distance divided by the cell-size of 11.4 micrometers.

	(initial energy in KEV)	/ (LET)	= distance per electron
Cs-137	(0.662 MEV x 1000 KEV/MEV)	/ (0.28 KEV/μm)	= 2,364 micrometers.
Ra-226	(0.596 MEV x 1000 KEV/MEV)	/ (0.29 KEV/μm)	= 2,055 micrometers.
50 KEV X-rays	(50 KEV)	/ (0.84 KEV/μm)	= 59.5 micrometers.
40 KEV X-rays	(40 KEV)	/ (1.00 KEV/μm)	= 40.0 micrometers.
30 KEV X-rays	(30 KEV)	/ (1.20 KEV/μm)	= 25.0 micrometers.

	(distance)	/ (11.4)	= (cells traversed by each primary electron)
Cesium-137	2,364 μm	/ 11.4 μm per cell	= 207.4 cells
Radium-226	2,055 μm	/ 11.4 μm per cell	= 180.3 cells
50 KEV X-rays	59.5 μm	/ 11.4 μm per cell	= 5.22 cells
40 KEV X-rays	40.0 μm	/ 11.4 μm per cell	= 3.51 cells
30 KEV X-rays	25.0 μm	/ 11.4 μm per cell	= 2.19 cells

• Step 4: Number Of Cell-Nuclei Traversed By Primary Electrons Delivering 100 Millirads To a Gram Of Tissue-Cells

An electron approaching normally to a cell "sees" a nuclear area which is ¼ the area of the total cell's area. Therefore, the number of nuclei traversed by primary electrons will be about ¼ of the number of cells traversed. In the calculation below, the number of primary electrons (from Step 2) times the number of cells traversed by each (from Step 3) provides the number of cells traversed, and this is reduced by a factor of 0.25 to obtain the number of nuclei traversed.

For 100 mrads:	(electrons)	x (cells per e)	x (0.25)	=	(number of nuclei traversed)
Cesium-137	(9.44×10^7)	(207.4)	(0.25)	=	4.89×10^8
Radium-226	(1.05×10^8)	(180.3)	(0.25)	=	4.73×10^8
50 KEV X-rays	(1.25×10^8)	(5.22)	(0.25)	=	1.63×10^8
40 KEV X-rays	(1.56×10^8)	(3.51)	(0.25)	=	1.37×10^8
30 KEV X-rays	(2.08×10^8)	(2.19)	(0.25)	=	1.14×10^8

• Step 5: Number Of Primary Tracks Per Cell-Nucleus At 100 Millirads

The number of primary ionization tracks which pass through a cell-nucleus is obtained by dividing the number of cell-nuclei traversed (from Step 4) by the number of nuclei present in a gram of tissue (from Step 1). Traversal by a primary ionization track is commonly called a "hit."

For 100 millirads:

Cesium-137	$(4.89 \times 10^8$ nuclei hit) / $(6.135 \times 10^8$ nuclei) = 0.7971 hit
Radium-226	$(4.73 \times 10^8$ nuclei hit) / $(6.135 \times 10^8$ nuclei) = 0.7710 hit
50 KEV X-rays	$(1.63 \times 10^8$ nuclei hit) / $(6.135 \times 10^8$ nuclei) = 0.2657 hit
40 KEV X-rays	$(1.37 \times 10^8$ nuclei hit) / $(6.135 \times 10^8$ nuclei) = 0.2233 hit
30 KEV X-rays	$(1.14 \times 10^8$ nuclei hit) / $(6.135 \times 10^8$ nuclei) = 0.1858 hit

Since a nucleus is either hit or it is not, the use of average values with fractional hits is just a preparatory device for Step 6.

• Step 6: Millirads Required To Cause an Average Of One Primary Ionization Track In All Cell-Nuclei Of an Irradiated Tissue

This value is the dose of 100 millirads divided by the average number of primary tracks per nucleus occurring at 100 millirads.

Cesium-137	When 100 mrads cause 0.7971 track, then 125 mrads cause 1 track.
Radium-226	When 100 mrads cause 0.7710 track, then 130 mrads cause 1 track.
50 KEV X-rays	When 100 mrads cause 0.2657 track, then 376 mrads cause 1 track.
40 KEV X-rays	When 100 mrads cause 0.2233 track, then 448 mrads cause 1 track.
30 KEV X-rays	When 100 mrads cause 0.1858 track, then 538 mrads cause 1 track.

• Formula To Convert Values For Other Cell-Sizes

The general relationship of values from Step 5, according to cell-size, is

$$\text{New Value} = \frac{(\text{Number of nuclei hit, old size}) \times (\text{old/new size})}{(\text{Number of nuclei present, old size}) \times (\text{old/new size})^3}$$

$$\text{New Value} = (\text{Old result from Step 5}) \times \frac{1}{(\text{old/new size})^2}$$

• Example: To convert the value in Step 5 from 11.4 micron cells to 10 micron cells, for 30 KEV X-rays, we write

$$\text{New Value} = (0.1858 \text{ hit}) \times \frac{1}{(11.4/10)^2} = 0.14296 \text{ hit.}$$

TECHNICAL APPENDIX 2
The Basis Of Table 6

Part A of this Technical Appendix shows the handling of fallout data, country by country.

Part B provides the area of each country and demonstrates how the 1,990,000 curies of cesium-137 are distributed country-by-country.

Part C shows the calculations supporting the statement that 50% of the dose commitment from the cesiums will occur during the first ten years after the Chernobyl accident.

• (A) TYPES, DATES, AND HANDLING OF FALLOUT MEASUREMENTS, BY COUNTRY

• General Criteria

Examination of all the fallout data from the various reporting countries shows that there is a high degree of variability of results within a single country. This is wholly expected, largely because rainfall can grossly increase deposition of radionuclides, and also because cloud plumes seldom cover a country uniformly.

In our endeavor to obtain the best representation of the average dose received by residents of any region, we have established some criteria for handling the limited quantity of fallout data provided from the various countries.

• Criterion I: Any country can be divided into four quadrants. When data are presented for each of the quadrants, we shall use the data as presented. When data are provided for three quadrants, we shall assign a zero value for the fourth quadrant, and then shall average all four values. When data are provided for two quadrants, we shall assign a zero value for the two remaining quadrants before averaging the four values. When data are provided for a single quadrant, we shall assign a zero value to each of the remaining quadrants before averaging. This set of procedures provides a cautious method of estimation.

• Criterion II: Some data are reported as a range of values. If values are provided within the range and if all four quadrants are represented, we shall average the values given. Where only the outer limits of the range are provided, we shall take these to represent two of the four quadrants of the country, and shall assign zero values to two other quadrants, and then average all four values.

• CONVERSION OF UNITS

There are 10^9 picocuries per millicurie.

There are 27 picocuries per Becquerel.

There are 100 rads per Gray.

There are 100 rems per Sievert.

There is 1 rem per rad, for gamma and for X-rays.

COUNTRY	SOURCE	COMMENTS
Albania	WHO Report June 12, 1986	Data are given for only one site. Therefore, three zeroes were assigned to other sites. The final result is 1/4 the value given for the one site. Date for the direct gamma dose was not given. Therefore, Cesium-137 % is taken as 1.8%, the lowest possible value. This effectively minimizes the fallout estimate.
Austria	EPA Report June 11, 1986	Excellent data are provided for the direct gamma dose. The average dose is based upon reports from 322 stations. The peak value for direct gamma dose was almost always for May 2,1986. Therefore, the appropriate Cesium-137 % is 5.8% of the total gamma dose.
Belgium	WHO Report June 5, 1986	Data are given as a range for Cs-137 deposition, for May 9 ,1986. Therefore, two zeroes were assigned, and the average of these plus the range limits were used to obtain average Cs-137 deposition.
Bulgaria	WHO Report June 5, 1986	Data for the direct gamma dose are given for five separated sites. The average of these is taken. Values are for May8, 1986. The appropriate Cs-137 % is 12.9% of the total gamma dose.
Canada	EPA Report June 11, 1986	Data are given for Iodine-131 deposition on the ground for nine widely separated locations--a reasonable representation of Canada. Most deposition values given were for May 12 or May 13. The conversion factor (in Method 3) for conversion from Iodine-131 to Cs-137 takes these dates into account appropriately.
Czecho-slovakia	WHO Report June 5, 1986	A single peak value of 200 uR/hr is given for the direct gamma dose. Three additional values of zero were assigned, giving an average value of 50 μR/hr for use in Method (2) calculation. Since the only indication for the date of this one reading was that it was before May 6, 1986, caution requires using the lowest Cs-137 %, the value of 1.8% of the total gamma dose. The effect is to make the cancer estimate given here too low, if the true date were later than April 29, 1986.
Denmark	WHO Report June 5, 1986	Excellent data are provided. The mean value for the integrated Cs-137 and Cs-134 depositions on the ground are provided for the period between May 7 and May 27, 1986. The data were obtained as a mean for 10 separate locations, labeled as "countrywide". It is not clear whether there may have been additional depositions before the May 7, 1986 date. If there were additional depositions, the Cs-137 and Cs-134 deposition totals here are too low, and the cancer estimates are also too low.
Finland	WHO Report May 30, 1986 and WHO Report June 5, 1986 plus Communication with Finnish Authorities. Also Finnish Reports: STUK-B-VALO 44 STUK-B-VALO 45	In the May 30, 1986 WHO report, the statement is made that "the deposition of Cs-137 varied between 100 and 1300 kBq/m^2." These values would lead to an extremely high cancer rate compared with the ones in Table 6 of this paper. In the June 5, 1986 WHO report, these data have just disappeared and the following data, bearing no resemblance, are presented for the cesiums: "Contamination of surface soil in kBq/m^2 (in-situ measurements) 6-7 May in Southern Finland was as follows: Cs-137 3 to 40 kBq/m^2; Cs-134 0.9 to 24 kBq/m^2." Inquiry produced from the Finnish Centre For Radiation and Nuclear Safety the reply that "WHO made an obvious error in their first figures from Finland. We straightened out that mistake, but why WHO did not inform in their next report about their misprint, I do not know." The letter, dated July 16, 1986, was signed by Olli Paakkola, Acting Director of the Surveillance Dept., Finnish Center for Radiation and Nuclear Safety. In the same letter, it is stated that "only half the country was affected by Chernobyl fallout," which is the basis for using half the area in Technical Appendix 2-B. Finnish authorities are designating one-third of the gamma-dose measured for Uusikaupunki as representative of Finnish exposures (STUK-B-VALO 45)[51]. That value is the basis for Method 2 calculations for Finland, and for the entries in Table 6.
France	EPA Report June 11, 1984	Only a single value is given for the direct gamma dose rate. It is for Paris for May 4, 1986. Three additional values were assigned as zero, and hence the average is 1/4 of the value for the Paris datum. The appropriate value for Cs-137 % of gamma dose rate is 8.0%. It is remarkable that France, a sophisticated nation in the field of nuclear power, provides so little data to WHO and the EPA.
Germany, East	No data provided	East Germany provided no data at all to the World Health Organization. Since it lies between Poland and West Germany, it is reasonable to assign it a dose intermediate to that of Poland and West Germany. Since West Poland most probably had a lesser dose than East Poland, we have weighted the West German dose twice as heavily as the Poland dose, to arrive a reasonable estimate for East Germany. It is certainly regrettable that the East German authorities saw fit to refuse to provide any measurements.
Germany, West	WHO Report May 30, 1986	The WHO Report provides an "average" value for the direct gamma dose rate for Southern Germany for May 4, 1986. A comparison of air values for many stations in Northern Germany showed that the fallout was heavier in the Southern region than the northern region. By using such comparisons, a value was estimated for Northern Germany. It appears that most of the data reviewed are for the eastern region of Southern Germany. Therefore, two additional values of zero were assigned for the western quadrants, north and south,in arriving at an appropriate value for the gamma dose rate. Since the gamma dose rate is reported for May 4, 1986, it is appropriate to take 8.0% as the Cs-137 % of the total gamma dose rate.
Greece	WHO Report May 30, 1986 and WHO Report June 5, 1986	A single value for Cs-137 deposition is given as follows: " May 9-11 0.8 kBq/m^2 " This is difficult to interpret, since the data as reported suggest that the value reflects only deposition for the period between May 9- May 11, rather than the entire surface contamination with Cs-137 on the ground. Nevertheless we have used this value here. Since only a single value is given, we have assigned a zero value to three other quadrants, giving a final value 1/4 that of the single value given. Both WHO Reports show the same inadequate statement concerning Cs-137 deposition.
Hungary	WHO Report June 5 ,1986	Direct gamma dose rates are presented as a range for May 1, 1986. Therefore, the outer limits of the range plus two assigned values of zero for two other quadrants are all used. The final average is 1/2 the mid-point of the given range. This was used in Method (2) calculation of Cs-137 deposition. For May 1, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 4.9 %.

COUNTRY	SOURCE	COMMENTS
Ireland	WHO Report June 12, 1986	A single measurement is given for direct gamma dose rate for May 7, 1986. Therefore, three additional values of zero were assigned for other quadrants, giving a final value to be used in Method (2) of 1/4 the measurement given. For May 7, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 11.7 %.
Italy	EPA Report May 12, 1986	Data are given for Iodine-131 depositions for five separate locations in Italy, for dates ranging from May 1 through May 3, 1986. To be cautious, we are treating these values as cumulative depositions, but if they are values for single days, we are underestimating Cs-137 deposition and dose by Method (3).
Japan	WHO Report June 5, 1986 and EPA Report June 11, 1986	Deposition of Iodine-131 is given for four separate locations in Japan. The results are given "by day", so that they may not reflect the cumulative deposition of Iodine-131. If this is true the Cs-137 deposition estimated by Method (3) is too low, and the cancer estimates presented here are also too low.
Korea (South)	WHO Report June 5,1986 and EPA Report June 11, 1986	No direct data are given for South Korea. However, measurement of Iodine-131 in air in Seoul, Korea is available for comparison with Iodine-131 in air in Kanagawa, Japan for the same day. So, an indirect calculation can be made based upon the Japanese deposition data plus the Korea-Japan comparison for air data. While this is not an ideal basis for calculation, it certainly gives the order-of-magnitude level for cancers in Korea.
Luxembourg	WHO Report June 5, 1986	A single direct gamma dose rate measurement is given for May 2, 1986. Therefore, a zero value was assigned for three additional quadrants, and the final average value used in Method (2) is 1/4 the given value. For May2, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 5.8%.
Norway	WHO Report June 5, 1986	Excellent data are given for Cs-137 deposition on the ground. The data are presented as the cumulative surface soil contamination by Cs-137 for the period between May 1 and May 22, 1986. The results are based upon 70 separate samples having been measured.
Netherlands	WHO Report June 5, 1986	A direct gamma dose rate is given for May 4, 1986 and thereafter. However, since it is not clear whether this dose is for a single location or is an average, we have, for caution, assigned three additional zero values to other quadrants. The final value used in Method(2) is, therefore, 1/4 of the given value. For May 4, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 8.0%.
Poland	WHO Report June 5, 1986	Multiple direct gamma-dose rate measurements are provided as a range for "all Poland". The two extremes of the range are taken and an additional two zero values are assigned to two quadrants. Therefore, the final value used in Method (2) calculations is 1/4 of the midpoint of the range given. For measurments made on April 29, 1986, the appropriate Cs-137 % of total gamma dose rate is 1.8%. Early EPA reports showed extremely high values for gamma-dose rates in Warsaw, Poland in the early period of fallout. These values were deleted in later EPA reports, as noted in Table 6. Inquiry revealed that EPA did not know the reason; "must be too high," was suggested.
Romania	WHO Report June 5, 1986	Multiple direct gamma dose rates are given as a range for the period April 29, 1986 through May 8 ,1986. The two extremes of the range for May 1 are used and zero values are assigned for two additional quadrants. The final value for May 1 used in Method (2) calculations is 1/4 of the midpoint of the range for that date. For May 1, 1986 measurements, the appropriate value for Cs-137 % of total gamma dose rate is 4.9%.
Spain	WHO Report June 12, 1986	Direct gamma dose rates are given as a range for the period April 29 to May 8 ,1986. The extremes of the range for April 29 are taken and zero values are assigned for two additional quadrants. Therefore, the final value used in Method (2) calculations is 1/4 of the midpoint of the range for April 29. For April 29 measurements, the appropriate value for Cs-137 % of total gamma dose rate is 1.8 %.
Sweden	WHO Report May 30, 1986	Detailed data are provided for Cs-137 deposition on the ground for eight separate stations. Four stations report deposition for May 15, 1986 and four other stations report deposition data for April 30, 1986. While the early data may be very much too low for measuring the cumulative deposition of Cs-137, those data were averaged in with the data for May 15. It is puzzling that Sweden did not continue reporting measurements after April 30 at four of the stations. Also it is puzzling that very high gamma-doses reported from Uppsala on April 29, in the EPA reports of May 8 and 9, simply disappeared as noted in Table 6. EPA is left in its May 12 report and thereafter with a single value for Uppsala (1,000 uR/hr on May 4) and no other data at all for that city. Since the eight stations reporting on cesium deposition were mainly in eastern Sweden, we elected to assign zero values for western Sweden. Therefore, the final value used is half the average for the eight reporting stations. This approach may underestimate radiation-induced cancers in Sweden. The basis for using half the area of Sweden in Technical Appendix 2-B is the map on page 32 of Hohenemser[56].
Switzerland	EPA Report June 11, 1986	Direct gamma dose rates are given for four parts of the country, central, east, west, and south. These values are for May 4, 1986. The average of these four gamma dose rates is used for indirect estimation of Cs-137 by Method (2). For May 4, 1986 measurements, the appropriate value for Cs-137 % of total gamma dose rate is 8.0 %.
Turkey	WHO Report June 12, 1986	A range of values for the direct gamma dose rate is given for the period May 4- May 7, 1986. The two extremes of the range are taken for May 4 and a zero value is assigned to two additional quadrants. The final value used in Method (2) calculations is, therefore, 1/4 of the mid-point of the range for May 4, 1986. For May 4, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 8.0 %.

COUNTRY	SOURCE	COMMENTS
United Kingdom	Report in NATURE Volume 321 15 May, 1986[52]	Fry, F.A., Clarke, R.H., and O'Riordan, M.C. published a paper entitled "Early Estimates of UK Radiation Doses from the Chernobyl Reactor". This useful paper provides representative data for gamma dose rates, weighted by population distribution for two major regions of the United Kingdom. "South" is the description of the region with 82.1% of the UK population, and "North" is the region with 17.9 % of the UK population (including the northwest of England, North Wales, Scotland, and Northern Ireland). These dose rates for May 2 ,1986 were used here for estimation of Cs-137 deposition by the indirect method (Method (2)). For May 2, 1986 measurements, the appropriate Cs-137 % of total gamma dose rates is 5.8 %.
United States	EPA Report May 11, 1986	Deposition of Iodine-131 on the ground is reported for fifteen widely separated stations in the United States. These data are satisfactory for indirect estimation of Cs-137 deposition by Method (3). The iodine-131 deposition data are for May 5- May 8, 1986, and I-131 to Cs-137 conversion factors for those dates were used.
Yugoslavia	WHO Report June 11, 1986	Direct gamma dose rates are provided for three separate regions. Peak gamma dose rates were reached May 2 - May 3, 1986. Two of the regions were close together, so the average of these two was used as a single value. Zero values were assigned to two additional quadrants. Then an average was taken of the four values so derived. This average was used in Method (2) calculation of Cs-137 deposition. For measurements of May 2, 1986, the appropriate value of Cs-137 % of total gamma dose rate is 5.8%.
U.S.S.R. Ukraine	WHO Report June 12, 1986	Direct gamma dose rates are reported for Oster, just north of Kiev, starting with May 9, 1986. The data for May 10 are used as a first step in the indirect estimation for Cs-137 deposition. A second usable value is that for Kishinev, Moldavia, which borders the Ukraine in the southwestern region. Therefore, we have assigned two zero values to cover the other quadrants of the Ukraine. The final average for gamma dose rate for May 10 is that obtained by averaging the values for Oster, for Kishinev, and the two assigned zero values. For May 10 measurements, the appropriate value for Cs-137 % of total gamma dose rate is 16.0 %. We should note that the Ukraine is one of the regions where Cs-137 remains available to plants through the root-soil pathway for longer periods than is the case elsewhere. As a result, our estimate of the internal dose from Cs-137 to residents of the Ukraine may be too low.
U.S.S.R. Byelo-russia	WHO Report June 12, 1986	Direct gamma dose rates are reported for Bialystok, Poland on the west border of Byelorussia. And, as mentioned above, direct gamma dose rates are available for Oster (north of Kiev, and 100 km south of the southern border of Byelorussia). It appears reasonable that the average of these two results can be used as representative of the southern 1/3 of Byelorussia. Therefore, we have assigned a zero value for each of the other 1/3 segments of Byelorussia. The final average is 1/3 of the value midway between the values for Bialystok and Oster. For measurements in Bialystok (data for April 29), the appropriate value for Cs-137 % of total gamma dose rate is 1.8 %. For Oster, as stated above (for May 10 measurements), the appropriate value for Cs-137 % of total gamma dose rate is 16.0 %. These adjustments were made before combining the Bialystok and Oster measurements.
U.S.S.R. Moldavian Republic	WHO Report June 12, 1986	Direct gamma dose rate data are provided by the Soviet Union for Kishinev, the capitol of the Moldavian Republic, starting with May 10, 1986. The data used here are for May 11, 1986. Three additional values of zero were assigned for other quadrants of Moldavia where we have no measurements. Therefore, the average value used in Method (2) calculations is 1/4 of the value for Kishinev. For measurements of May 11, 1986, the appropriate value for Cs-137 % of total gamma dose rate is 17.3 %.
U.S.S.R. Baltic Republics Latvia Lithuania Estonia	WHO Report June 12, 1986	No really useful data for Cs-137 or gamma dose rates are provided for the Baltic Republics. But, data are available for direct gamma dose rates for sites in Poland (Bialystok, Olsztyn) bordering these Republics, for Southern Finland not far from the northern part of these republics, and from Sweden to the northwest of these Republics. From all these data, a minimal estimate of 100 μR/hr as the peak direct gamma dose rate has been here assigned to the Baltic Republics. This appears cautious and reasonable. Further, to err on the side of underestimation of cesium dose, we shall assign this value for April 29, 1986, for which the appropriate value for Cs-137 % of total gamma dose rate is 1.8 %.
U.S.S.R. Moscow and Suburbs	EPA Report June 11, 1986	Some values for direct gamma dose rate are provided, starting with data for May 5, 1986. We shall used the May 5 data for indirect estimate of Cs-137 deposition by Method (2). For May 5 measurements, the appropriate value for Cs-137 % of total gamma dose rate is 9.1%.

Note: The values in Table 6 are rounded off. Some may have preferred that we do not round off so as to facilitate cross-checking between column entries. Others complain that the goodness of the data do not justify keeping the number of significant figures which would be present without rounding off. This dilemma is ever-present. The reader simply needs to keep in mind that rounding has been done, when the reader makes use of Table 6.

COUNTRY	SOURCE	COMMENTS
U.S.S.R. Leningrad and Suburbs	EPA Report June 11, 1986	Direct gamma dose rates are provided for May 2- May 7, 1986. The peak gamma dose rates are reported for May 7 ,1986, and these data are used in the indirect estimate of Cs-137 by Method (2). For May 7, 1986 measurements, the appropriate Cs-137 % of total gamma dose rate is 11.7 %.
U.S.S.R. Russian Soviet Republic		No really satisfactory data are available which enable us to provide any estimates for Cs-137 deposition in this largest of the Soviet Republics, aside from the data for Moscow and Leningrad, which are described above. This is regrettable, since this Russian Republic is not only the largest geographically, but is also the most populous of the Soviet Republics.
U.S.S.R. Chernobyl Region	No data	This area very near the Chernobyl nuclear power plant had some very high doses, since radiation sickness and deaths have occurred there. Since no data have been made available for this special region, no cancer calculations have been made.
U.S.S.R. All Other Soviet Republics	No data	No data have been provided for all these other Soviet Republics, nor are there any data for regions close by from which any reasonable estimates of Cs-137 deposition can be made. We therefore refrain from making any cancer calculations for these Republics.

• (B) DISTRIBUTION OF 1,990,000 CURIES OF CESIUM-137, BY COUNTRY

The tabulation below corresponds with the left-hand side of Table 6.

Country	Deposition (pCi/m^2)	Area in $meters^2$	Deposition Total, in Curies
Albania	1.618×10^4	2.886×10^{10}	467
Austria	2.346×10^5	8.417×10^{10}	19,746
Belgium	2.697×10^3	3.063×10^{10}	83
Bulgaria	2.319×10^5	1.113×10^{11}	25,810
Canada	0.539×10^3	1×10^{13}	539
Czechoslovakia	7.012×10^4	1.284×10^{11}	9,003
Denmark	2.022×10^4	4.324×10^{10}	874
Finland (½ area)*	3.358×10^5	1.692×10^{11}	56,817
France	7.821×10^4	5.491×10^{11}	42,945
Germany, West	2.319×10^5	2.495×10^{11}	57,859
Germany, East	2.710×10^5	1.086×10^{11}	29,431
Greece	4.045×10^3	1.325×10^{11}	536
Hungary	5.529×10^4	9.340×10^{10}	5,164
Ireland	1.753×10^3	7.055×10^{10}	124
Italy	3.911×10^4	3.024×10^{11}	11,827
Japan	1.079×10^3	3.738×10^{11}	403
Korea, South	0.809×10^3	9.887×10^{10}	80
Luxembourg	1.618×10^4	2.590×10^9	42
Netherlands	1.618×10^4	4.100×10^{10}	663
Norway	1.160×10^5	1.627×10^{11}	18,873
Poland	3.493×10^5	3.139×10^{11}	109,645
Romania	1.038×10^6	2.383×10^{11}	247,355
Spain	3.506×10^3	5.067×10^{11}	1,776
Sweden (½ area)*	6.688×10^5	2.258×10^{11}	151,015
Switzerland	3.182×10^5	4.145×10^{10}	13,189
Turkey	1.348×10^5	7.836×10^{11}	105,629
United Kingdom	8.765×10^4	2.45×10^{11}	21,474
United States	0.067×10^3	7.60×10^{12}	469
Ukraine	1.262×10^6	6.032×10^{11}	761,238
Byelorussia	9.628×10^5	2.083×10^{11}	200,551
Moldavia	1.686×10^5	3.370×10^{10}	5,662
Baltic Republics	1.402×10^5	1.742×10^{11}	24,423
Yugoslavia	2.495×10^5	2.568×10^{11}	64,072
Moscow and Leningrad not computed, because area is so small			
Sum of All Depositions, in Curies			1,987,784
$(0.75)(0.75)(3.53 \times 10^6) =$			1,985,625

* See Technical Appendix 2-A

Comparison with cesium-137 deposition from weapons fallout:

According to UNSCEAR[4] (p.146), the deposition of cesium-137 in the temperate latitudes of the northern hemisphere from all the atomspheric nuclear bomb-tests of the United States, Soviet Union, and Britain combined was 136,000 or 1.36×10^5 picocuries per square meter.

• (C) TIME-DISTRIBUTION FOR DOSE COMMITMENT FROM CESIUMS

Cesium-134 with its half-life of 2.3 years will deliver its committed dose to exposed populations very much earlier than is the case for cesium-137, with its half-life of 30.2 years. Calculations below show what fraction of the total dose commitment (over all time, from the cesiums combined) is delivered by the end of each decade following the accident. To calculate, we used the observations (from Section 7 of this paper) that

Cesium-134 (internal + external) accounts for 11% of the total dose from cesiums;
Cesium-137 (internal + external) accounts for 89% of the total dose from cesiums;
and of the 89%, the internal share is 30% and the external share is 70%.

• First Decade CESIUM-134 will deliver 94.6% of both its internal and external doses; this amounts to (0.946) x (11%) = 10.4% of the total dose from cesiums. CESIUM-137 will deliver approximately 95% of its internal dose in the first decade; this amounts to (0.95) x (0.30) x (89%) = 25.4% of the total dose from cesiums. CESIUM-137 will deliver 20.0% of its external dose in the first decade; this amounts to (0.20) x (0.70) x (89%) = 12.5% of the total dose from cesiums. COMBINED DELIVERY (%) BY THE END OF THE FIRST DECADE = 10.4 + 25.4 + 12.5 = 48.3% of total.

• Second Decade CESIUM-134 will deliver 5.4% of (11%) = 0.59% of the total dose. CESIUM-137 (internal) will deliver 5% of (0.30)(89%) = 1.34% of the total dose. CESIUM-137 (external) will deliver 16% of (0.70)(89%) = 10.0% of the total dose. COMBINED DELIVERY (%) BY THE END OF THE SECOND DECADE =

48.3 + 0.59 + 1.34 + 10.0 = 60.2% of the total dose committed.

• Third Decade The only new contribution will be from external cesium-137 because internal contributions from the cesiums are essentially over. CESIUM-137 will deliver 14% of (0.70)(89%) = 8.7%.

COMBINED DELIVERY (%) BY THE END OF THE THIRD DECADE =

60.2 + 8.7 = 69% of the total dose committed from the cesiums.

• Fourth Decade Additional contribution from external CESIUM-137 is 10% of (0.70)(89%) = 6.2% of the total dose.

COMBINED DELIVERY (%) BY THE END OF THE FOURTH DECADE =

69 + 6.2 = 75.2% of the total dose committed from the cesiums.

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John Gofman is Professor Emeritus of Medical Physics at the University of California at Berkeley, and lecturer at the Department of Medicine, University of California School of Medicine at San Francisco. Author of Radiation and Human Health, 1981.

He is the author of approximately 150 scientific papers in peer-review journals in the fields of nuclear/physical chemistry, coronary heart disease, ultracentrifugal analysis of the serum lipoproteins, the relationship of human chromosomes to cancer, and the biological effects of ionizing radiation with particular reference to cancer and leukemia induction. Since 1980, he has been serving as a peer-reviewer for Health Physics journal.

While a graduate student at Berkeley, he co-discovered Protactinium-232 and Uranium-232, Protactinium-233 and Uranium-233, and proved the slow and fast neutron fissionability of U-233. He worked during the war on the Manhattan Project atomic bombs, and developed some of the first methods for chemically extracting plutonium from irradiated uranium.

After graduating from medical school, he began his research on coronary heart disease and, by developing special flotation ultracentrifugal techniques, demonstrated the existence of high-density and low-density lipoproteins. His work on their chemistry and health consequences has been widely honored, including the Stouffer Prize (shared) in 1972 for outstanding contributions to research in arteriosclerosis, and selection in 1974 by the American College of Cardiology as one of the twenty-five leading researchers in cardiology of the past quarter-century.

Meanwhile, the Atomic Energy Commission asked him to establish the Biomedical Research Division at the Lawrence Livermore National Laboratory. From 1963 through 1969, he served as Associate Director of the Laboratory, and as the first Director of its Biomedical Division, where he also carried out his own laboratory research in cancer, chromosomes, and radiation, as well as his analyses of the data on the Japanese atomic-bomb survivors and other irradiated human populations. In 1969, he published his finding (with Dr. Arthur Tamplin) that exposure to ionizing radiation is far more serious than previously recognized, and stated the first three "laws" of radiation carcinogenesis.

In 1973, he returned to full-time teaching at the University of California at Berkeley, and continued independently to analyze the accumulating human evidence of health effects from radiation in the low-dose region. His book RADIATION AND HUMAN HEALTH (1981; still in print) integrated the existing worldwide evidence for the first time. It has been described as a "remarkable and important book" by the Journal of the American Medical Association (19 March 1982), and recommended by the journal of the American Nuclear Society Nuclear News (January 1982) as "not a tome to be treated casually, since there is a lot of material here which should be read carefully and given thought and evaluation."

His most recent book (1985) X-RAYS: HEALTH EFFECTS OF COMMON EXAMS (with O'Connor) has been selected by the Library Journal (15 April 1986) as one of the most useful and important reference books published during all of 1985. The New England Journal of Medicine (6 February 1986) said, "This book is practical and important. It is destined to represent a watershed in the controversial field of low-dosage radiobiology and will be of inestimable value to radiologists, other physicians, dentists, and patients."

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