UNITED STATES
NUCLEAR REGULATORY COMMISSION
WASHINGTON, D. C. 20555

NOV 03 1980

DSI PERSONNEL

Dr. Ross has recommended that all DSI personnel read the attached article, which is a very readible discussion of its subject. We expect to be giving a division seminar on this subject area sometime in the future, and you may find that the article raises some questions that you can ask at the seminar.

Bill

William E. Kreger, Assistant Director
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Enclosure 2

Health Physics Vol. 40, (October) pp. 689–703
Pergamon Press Ltd. Printed in the U.S.A.
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THE CANCER RISK FROM LOW-LEVEL RADIATION

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(*Received* 14 *December* 1979; *accepted* 10 *March* 1980)

Abstract—The various lines of evidence that lead to current estimates of the cancer risk from low-level radiation are reviewed. It is first shown why it is very difficult to get direct experimental evidence, so that much reliance is placed on extrapolation of data from high level radiation. The evidence that a linear extrapolation is conservative, i.e. more likely to over-estimate than to under-estimate the risk at low levels, is extensively reviewed. The "new evidence" that has been claimed to indicate that the linear extrapolation under-estimates effects at low levels is examined. Complications in deriving risks based on the linearity assumption are considered, and final estimates from various sources are presented.

I. EFFECTS AT HIGH LEVELS AND THE LINEARITY HYPOTHESIS

THERE HAVE been many situations in which large numbers of people have been exposed to high levels of radiation, and through studies of them (NAS72; UN77) the health effects of high-level radiation are rather well known. Some of these data are summarized in Table 1. Among the survivors of the atomic bomb attacks on Japan, there were 24,000 people who received an average exposure of 130 rem, and about 120 extra cancers developed among them up to 1972. There were 15,000 British patients treated with X-rays for ankylosing spondylitis (arthritis of the spine) with doses averaging 370 rem, and they had about 115 extra cancers. Over 900 Germans were treated for that same disease and for bone tuberculosis with injections of ^{224}Ra giving an average dose to the bone of 4400 rem,* and 45 of them got bone cancer (vs 0.1 expected). About 1700 U.S. women employed during the 1920s in painting radium on clock and watch dial numerals to make them self-luminous used their tongues to put a fine tip on the brush, getting radium into their bodies; their average bone dose was 17,000 rem and 48 of them died of bone cancer (vs 0.4 expected). Among 4100 U.S. uranium miners exposed to excess levels of radon gas due to poor mine ventilation, the average exposure to bronchial surfaces was 4700 rem and up to 1972 there were 135 lung cancer deaths among them vs 16 expected. There have been several other miner groups which have experienced excess lung cancers, like a group of 800 Canadian fluorspar miners whose average bronchial exposure was 2800 rem, resulting in 51 lung cancer deaths vs 2.8 expected. There have been a number of situations in which there have been high exposures resulting in something like 10 extra cancers, such as women in a Nova Scotia tuberculosis sanitorium exposed to excessive X-rays in the course of fluoroscopic examinations, U.S. women

*For loose discussion as in this section, we use 1 rad = 10 rem (ICRP59) for α-particle radiation of the lung and bone (with radium).

Table 1. Sources of information on risk of cancer from radiation (NAS72)

Type of cancer	Cause of exposure or X-ray treatments	Exposure date	Years after exposure considered	Number of subjects	Average dose (rem)	Cases observed	Cases expected	Cases 10^4 rem yr
Leukemia	A-bombs, Japan	1945	5–25	23,979	130	81	19.7	1.0
	Spondylitis	1935–54	0–25	14,554	372	52	5.5	0.9
	Menorrhagia	1940–60	0–24	2000	136	6	1.3	1.2
Bone	Ra^{226} intake	1915–35	11–56	775	17,000	48	0.4	0.11
	Ra^{224} treatments	1944–64	4–25	925	4410	45	0.12	0.84
	Spondylitis	1935–54	6–27	14,654	372	4	0.63	0.10
Breast	A-bombs, Japan	1945	16–25	12,000	125	26	12.9	0.9
	Fluoroscopy	1940–49	10–30	243	121	(22)	(4)	5.4
	Mastitis		10–29	606	200	11	4.2	6.0
Lung	Uranium mines	1920–63	6–50	4146	4680	135	16	0.53
	Fluorspar mines	1935–63	11–33	800	2770	51	2.8	1.6
	Metal mines		16–37	1759	1720	45	16	0.67
	spondylitis	1935–54	6–27	14,554	400	96	54	1.2
	A-bombs, Japan	1945	16–25	19,472	133	71	37	0.60
Gastro-intest.	A-bombs, Japan	1945	25	23,979	130	378	363	0.52
	Spondylitis	1935–54	11	14,554	375	53	34	0.81
Leukemia age 0–9	A-bombs, Japan	1945	6–24	4507	112	19	2.9	1.6
	Thymus X-rays	1926–57	0–35	1451	65	6	1.0	3.0
	Tinia capitis	1940–49	0–22	2043	30	4	0.9	3.4

treated with X-rays for inflammation of the breasts following childbirth, women treated with X-rays for gynecological maladies, various types of pelvic X-ray treatments, children treated with X-rays for enlargement of the thymus gland, infants radiated with X-rays for ringworm of the scalp, patients in several countries fed a thorium compound to aid in X-ray contrast studies and Marshall Island natives exposed to fallout from a nuclear bomb test. As a result of numerous studies of these groups, there is a great deal of information available on induction of cancer by high levels of radiation. This is periodically reviewed and updated by prestigious study-evaluation groups (NAS72; UNSCEAR77).

If one seeks to find similar information on low level radiation, one is immediately confronted with statistical limitations. For example, suppose one found a group of 10,000 white males who had received an extra 10 rem of whole body radiation. The easiest evidence to find would be excess leukemias because that disease develops earliest and is among the most sensitive to radiation. As a first approximation we might use the results of high level radiation studies that leukemias are induced at a rate of about 1.0×10^{-6}/yr per rem of exposure. We would then expect $10{,}000 \times 10 \times 10^{-6} = 0.1$ extra leukemias per yr among this group. In the absence of radiation, one would expect 0.88 leukemias if we take statistics for the entire U.S. In the 25 yr over which radiation is effective in causing leukemias, we then expect 22 ± 4.7 cases from natural causes vs 2.5 from the 10 rem radiation exposures. Clearly, the statistics here are marginal at best. But the problem goes much deeper—the total U.S. population is hardly a suitable control group. Cancer is largely caused by environmental factors and hence is subject to wide variations in incidence rates. Even for entire states, the 0.88 cases given above as the expectation varies from 1.0 (MN, DC) to 0.77 (ME, NM). In our above examples, this could vary the number of expected cases from 19 to 25, making it still more difficult to ascertain that there are 2 or 3 extra. Moreover, a group of people with 10 rem

of extra radiation would typically have more environmental factors in common than merely living in the same state.

Any experimental study of effects of low-level radiation would therefore need large populations, like millions of subjects, and there would still be considerable difficulty in selecting a control group. One way to achieve large numbers of subjects would be to use variations in natural radiation; for example citizens of Colorado, Wyoming and New Mexico are exposed to about 5 rem more than the U.S. average over their lifetimes. However, the leukemia rates in those states are considerably *below* the U.S. average, 8.11 vs 8.81×10^{-5}/yr for white males and 5.13 vs 5.74 for white females. The same is true for all cancers; the high natural radiation states have annual rates of 140×10^{-5} for white males and 114×10^{-5} for white females, while the U.S. average is 174 and 130 respectively. The fact that states with high natural radiation have considerably *lower* cancer rates than average is generally dismissed as indicating only that radiation is very far from being the principal cause of cancer, and this point is logically correct. However, this author is highly skeptical over whether that attitude would be accepted if states with high natural radiation happened to have somewhat *higher* than average cancer rates.

Since there is little direct evidence on effects of low-level radiation, the simplest option is to use our abundant data on effects of high-level radiation to derive estimates of effects of low levels by assuming a linear dose-effect relationship, i.e. if some high level dose D causes a cancer risk R, we assume that a dose 0.1D will cause a risk 0.1R, that a dose 0.01D will cause a risk 0.01R, etc down to extremely low doses.

This "linearity hypothesis" was recommended by the National Academy of Sciences Committee on Biological Effects of Ionizing Radiation (BEIR) in 1972 (NAS72) with a statement that it is a "conservative" approach, more likely to over-estimate than to under-estimate the effects of low levels. This statement seems to represent the general thinking in the involved scientific community, although there is considerable variation in opinions of *how much* more likely the over-estimate is. There is a considerable body of opinion that the over-estimate is gross, say by a factor of 2–10, while there is also an important body of opinion that linearity does not give an over-estimate. We now turn to a review of the evidence behind these positions.

2. EVIDENCE THAT LINEARITY IS CONSERVATIVE

In this section we review the principal evidence bearing on whether the linearity assumption is conservative as a means of estimating effects of low-level radiation. This evidence comes from various sources, each of which is discussed in a separate section.

(A) *Repair processes*

There is a great deal of evidence that nature provides mechanisms for repair of radiation damage to biological molecules. There have been many experiments in which it was shown that single doses in the 1000 rem range cause fatal radiation sickness in mice and other animals whereas fractionating these doses over several days or more does not (Fo77). Perhaps the best demonstration of repair mechanisms with regard to cancer is the dose rate effect, examples of which are shown in Figs. 1 and 2.

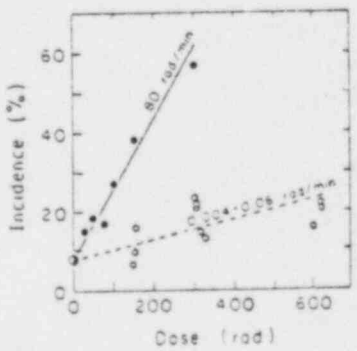

FIG. 1. Myeloid plus thymic leukemia in male RF mice exposed to X- or γ-rays (Up70). Plot shows percent incidence vs total dose for a high dose rate (80 rad/min) and a low dose rate (0.004–0.06 rad/min).

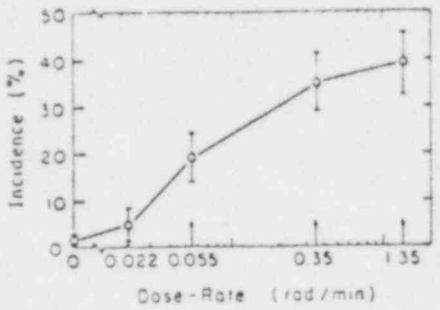

FIG. 2. Leukemia in CBA and C57Bl mice after a γ-ray dose of 1000 rad (Mo59). Plot shows percent incidence after 15 months vs dose rate.

We see there that a given dose of radiation is generally much less carcinogenic when spread out in time than when given rapidly; this implies that damage from earlier doses was repaired before the later doses were administered. The dose rate effect is well established in many animal studies with X-rays and γ-rays (Gr72; Sh66; Up70; Mo59) although there is some contrary evidence with α-particle radiation (Spe73; May78a) which will be explained later. It has been pointed out (Brow76) that effects from the high dose rates received by the Japanese A-bomb survivors were no larger than from low dose rate medical exposures, but this could have other explanations. Increased effects of high dose rates are well established in studies of genetic effects, a mutagenic process somewhat linked to cancer induction (Rus72).

In addition to this indirect evidence from dose rate effects, there is direct evidence for repair processes in that broken chromosomes have been observed to re-unite into a single strand (Le55; Wo61). There is also a vast amount of evidence for DNA repair in bacteria (Fo73; El77; UNSCEAR72; Mc66; To73).

In view of the well established existence of repair mechanisms, there is a general feeling that effects of low doses should be largely repaired, whereas repair of the much more extensive damage from high doses would be far less complete. This implies that the linearity hypothesis *over*-estimates effects of low-level radiation. On the other hand, it is argued that large doses may kill cells, which prevents them from becoming cancerous; this could cause linearity to under-estimate effects of small doses. However, there is good evidence that cell killing is not an important effect below about 100 rem (Mars78; El67), so if data in the 100 rem range is utilized, it would be difficult to use cell killing as a reason for linearity under-estimating effects of low-level radiation.

(3) *Mechanism for radiation induction of cancer*

One of the strongest reasons for believing that the linear hypothesis is conservative derives from our understanding of the processes by which radiation induces cancer (Ke72). Radiation affects matter largely by knocking electrons out of molecules and thereby disturbing molecular structure. In the process, the radiation gives up energy, transferring it to the material. The number of electrons knocked out of position is proportional to this energy deposited, and the latter is the basis for defining radiation dose. If the biological effects of radiation were simply due to single electrons being knocked out of position—this is called a "single hit" process—the cancer risk would be proportional to the total number of single hits, which is proportional to the energy deposited, regardless of the type of radiation. However, this is well known *not* to be the case (ICRP59); for a given energy deposited, α-particles and neutrons (known as high LET—linear energy transfer—radiation) are an order of magnitude more effective in doing biological damage than γ-rays or electrons (low LET). This is strong evidence that biological effects of radiation are not caused by single hits, but rather by multiple hits.

The basic difference between high and low LET radiation is that the former concentrates its damage within a much smaller volume of tissue. Since high LET is more effective, we may presume that effects are caused by multiple hits very close together, within some small sensitive volume.

Confirmatory evidence for a multi-hit process derives from the dose rate dependence discussed above. If cancer induction were a single hit process, it could not matter whether these hits were close together or far apart in time; each hit would have a certain probability of resulting in a cancer. However, as noted previously in connection with Figs 1 and 2, effects are much larger at high dose rate. With a multi-hit model, this is readily explainable by repair processes. If the two hits are well separated in time, damage from the first may be repaired before the second hit occurs.

Granted that radiation induced cancer is a multi-hit process (we assume a two-hit process), the multiple hits may be by the same particle of radiation or by separate particles. If they are by the same particle, effects are linearly proportional to dose, with the proportionality constant much larger for high LET than for low LET radiation because the former has a much better chance of making two hits close together.

On the other hand, if the two hits are by separate particles of radiation, effects are proportional to the square of the number of single hits,* and therefore proportional to the square of the dose, regardless of whether the radiation is high LET or low LET. These considerations lead to dose-effect curves like those shown in Fig. 3: at very low doses the linear term must be predominant, and at very high doses the term proportional to dose-squared (quadratic terms) must predominate. The transition between the two should occur at a dose where it is not unreasonable to expect two hits by separate particles of radiation within the sensitive volume. This would occur somewhere near the dose where there is an average of one hit per sensitive volume. The latter dose, of course, depends on the sensitive volume: for example, if it is 1 μ in diameter as suggested by some experiments (Ke72; Sc73; Ke75), the dose for an average of one hit within it is 8 rad for a 1-MeV γ-ray and 300 rad for a 5-MeV neutron whereas if the sensitive volume is 5 μ in diameter, the size of an entire cell nucleus, the required dose is 0.3 rad for a 1 MeV γ-ray and 12 rad for a 5-MeV neutron. We don't really know what the sensitive volume is, but these examples give the general impression that the transition from a linear to a quadratic dependence occurs at relatively low levels for β's and γ's, and at relatively high levels for α's and neutrons as shown in Fig. 3. This means that over the range of principal interest, the dose-effect curves should be linear for the latter and concave upward (quadratic) for the former, which leads to the conclusion that application of the linear hypothesis based on data at high doses will over-estimate effects of low doses for β- and γ-ray exposure.

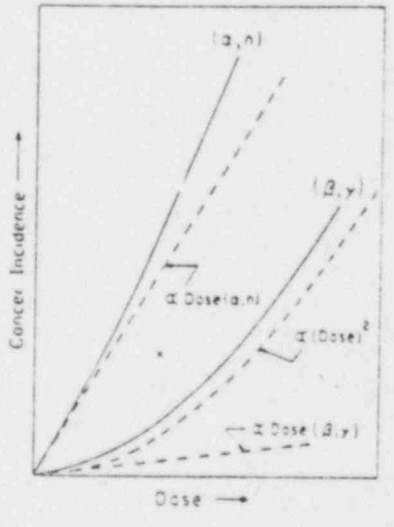

FIG. 3. Cancer incidence vs dose in a 2-hit model. The $\alpha(\text{DOSE})^2$ term is due to the two hits coming from different particles of radiation, while the α-DOSE terms are from both hits coming from the same particle; the latter is much larger for α-particles and neutrons (α,n) than for β's and γ's (β,γ). The total cancer incidence is the sum of the two terms, as shown by the solid lines for β,γ and for α,n.

*For example, if there were 100 targets, the chance of hitting a given pair in 10 shots is about $\frac{10}{100} \times \frac{10}{100} = 0.01$, whereas in 20 shots it is about $\frac{20}{100} \times \frac{20}{100} = 0.04$. Note that it increases as the square of the number of shots.

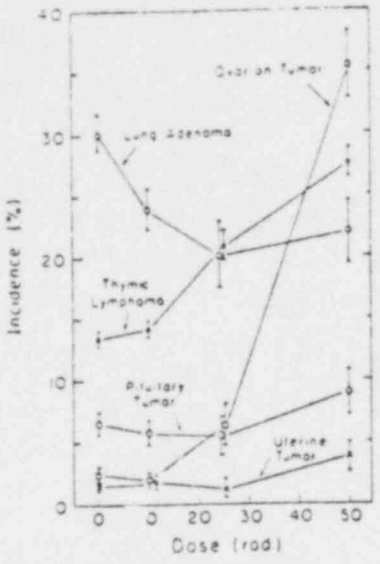

FIG. 4. Incidence of various neoplasms in γ-ray irradiated mice (U176) as a function of dose. Error bars are ± 1 S.D. In all cases, incidence is much increased at doses above 50 rad.

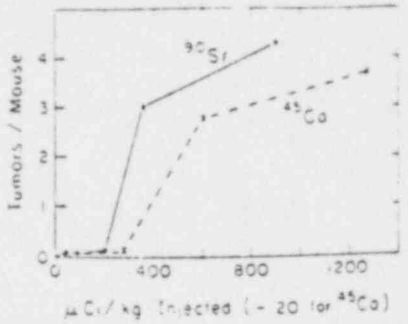

FIG. 5. Osteogenic sarcomas produced in CF1 female mice by intravenous injection of ^{90}Sr and ^{45}Ca at age 70 days (Fi68). Plot shows tumors per mouse vs radioactivity injected. Controls had 0.03 tumors per mouse. ^{90}Sr experiments used 810 mice and 150 controls; results for 1.3, 4.5 and 20 μCi/kg, not shown on plot, showed *no* excess over controls.

It may be noted that the above-outlined discussion assigns most α-particle radiation effects as due to multiple hits by the same α-particle at the same time. This implies that there would be no dose-rate effect for α-particles, in agreement with experimental evidence (Spe73). In fact there is some evidence that effects of α-particles are increased at lower dose rates (May78a).

(C) *Data on cancer induction by radiation*

One basis for judging the validity of the linearity hypothesis is to observe how well it behaves in explaining data down to the lowest doses at which effects are statistically meaningful. There is a rather large body of animal data extending down to the 10-rem region with reasonable statistical significance. These data for X, γ- and β-rays preponderantly indicate that linearity *over*-estimates effects at low levels (Up61; Fi68; Bu68; Mal69; May70; May72); examples are shown in Fig. 4 for external radiation and in Fig. 5 for exposure to irradiation by internally absorbed radionuclides. Note that these curves are concave upward. In some cases results are close to linear (St75), and there is one well known situation in which the observed dose-effect curve is concave downward (Sh69), the latter case involves mammary cancers in Sprague-Dawley rats, a special breed in which all females are virtually certain to die of that disease even in the absence of radiation exposure; it is widely recognized that this is an exceptional situation in many ways (Ke72).

The animal data for α-particle radiation usually show something close to a linear

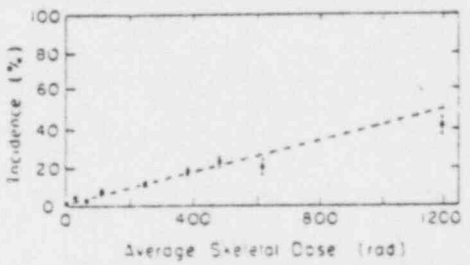

FIG. 6. Bone cancers induced in mice by injection of ^{226}Ra. Plot is percent incidence of bone sarcomas vs dose to bone in rad. Plot shown is work reported in (Fi69), adapted in (May 78b). Note that dose equivalent in rem is an order of magnitude larger than dose in rad shown on the plot. Dashed line is experimenter's straight line fit to data.

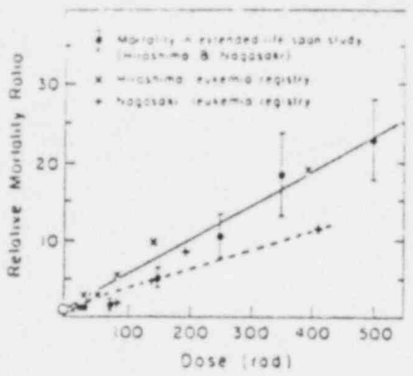

FIG. 7. Leukemia among Japanese A-bomb survivors (Be77). Solid circles are mortality data from leukemia among the "extended life span study" group chosen for careful analysis and follow-up, and solid line is a straight line between those data for high dose and zero dose (natural occurrence level). Other points are from the Hiroshima and Nagasaki leukemia registries analysed as described in text. Dashed line represents a straight line through data from Nagasaki leukemia registry.

dependence (May79a); an example (Fi69) is shown in Fig. 6. It should be noted that here, as in all other cases, there are no significant data below about 100 rem (~10 rad for α's).

Probably the best human data is that for leukemia among the Japanese A-bomb survivors (Be77), shown in Fig. 7. Unfortunately this has a controversial aspect. The mortality data for subjects in the group chosen for careful follow-up—the so-called "extended life-span study"—are shown by the solid circles with error bars, and they seem to indicate that linearity grossly over-estimates effects at low doses. However, there are leukemia registries in both Hiroshima and Nagasaki which keep track of the number of cases of leukemia among people living at various distances from the bomb explosion and compare them with the total population living at these distances (in 1950); the number of cases divided by the population is also plotted vs dose (as derived from the distance) in Fig. 7. There is no consideration as to age distribution, number of people who left the city, etc. but it is presumed that these factors do not vary systematically with dose. It is evident that these data are much better fit to a straight line than are the mortality data for the "extended life span" study. However, even the tumor registry data give no evidence that linearity *under*-estimates effects of low level radiation.

Data on bone cancer among the radium dial painters (Ro78) are plotted in Fig. 8, and we see there that the data points for low doses lie consistently below the prediction of the linearity hypothesis. While the statistical significance of this conclusion is not very great, there is certainly good evidence here that the linearity hypothesis is, if anything, conservative. Note that these data are for α-particle exposure.

(D) *Information from natural environmental exposure*

The clearest evidence that linearity *over*-estimates effects of low doses, if the basic assumptions of the study are accepted, comes from a comparison of radon-induced lung cancer between miners exposed to high doses and the non-cigarette smoking members of the

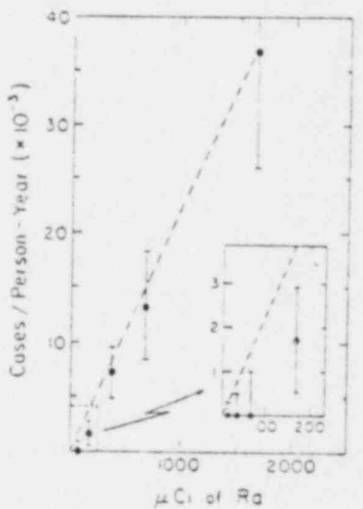

FIG. 8. Bone sarcomas among radium dial painters (Ro78). Main plot shows higher dose data used to obtain straight line fit, and inset compares this straight line with the low dose data.

general public exposed to natural radon in the environment at low doses (Co80). It turns out that 70% of the excess cancers among the miners were of one particular histological type, small cell-undifferentiated (SCU), and if linearity is applied to this disease based on the miner data, the number of SCU lung cancers due to normal environmental radon exposure among non-smokers is *over*-predicted by a factor of 8.

The significance of this conclusion depends on some basic assumptions that require justification. The most important assumption is that radon induced lung cancer has the same risk factor for smokers and non-smokers, i.e. that there is no synergism between smoking and radon exposure. Actually, there were indications in the early U.S. miner data that smoking accentuated the effects of radon, but the most recent data do not seem to support that viewpoint. One strong evidence against a smoking-radon synergism is that lung cancer incidence vs radon exposure is very similar between modern U.S. miners and a group of 19th century miners in the Erz Mountains of central Europe who suffered their fate before cigarette smoking began (in early 20th century). There is independent evidence against a smoking-radon synergism from the studies of the Japanese A-bomb survivors: the *difference* in lung cancer rates between those with very low and very high exposure is the same for smokers and non-smokers, although the percentage increase is only 40% for smokers vs 200% for non-smokers. If there were a synergism, the percentage increase should be about the same for the two groups. It may also be noted that the male female ratio of excess lung cancer from radiation was about unity, whereas that ratio in the general population was about 3, due to heavier smoking by males. If there were a strong synergism, these ratios should be the same.

Another basic assumption in the high-level vs low-level radon comparison was that the percentage of lung cancers that are SCU cell type does not decrease with decreasing dose. The evidence for this is that among the uranium miner victims, the fraction of the excess lung cancers that were SCU was 77% for the lowest dose group, 68% for the intermediate dose group, and 68% for the highest dose group (Ar74).

The above outlined evidence would seem to justify the conclusion that a linear dose-effect relationship normalized to the high dose data on miners over-estimates the effects of environmental radon exposure by a factor of 8.

The reason why this test is so sensitive is that average environmental radon exposures are quite high, well over 1 rem/yr to the bronchial epithelium. The lowest dose range for which there is significant evidence on the linearity hypothesis is leukemia caused by natural background γ-rays, levels of about 0.1 rem/yr.

Radiation induced leukemia is much better understood than other types of radiocarcinogenesis because it develops much sooner after exposure; thus we know the age dependence rather well. It has been rather easily diagnosed since the 1890s, which allows us to go back in time to the early years of this century when it was a much rarer disease than it is now. It is therefore possible to establish that a linear dose-effect relationship normalized to the high dose data on Japanese A-bomb survivors and radiation therapy patients requires that essentially all of the leukemia observed among 20–35-yr-old British females in the 1911–20 time period was due to natural environmental γ-ray exposure (Co80). This is evidence that the linear hypothesis does not under-estimate effects of radiation levels in the 2–3 rem range.

(E) *Latent period increases with decreasing dose*

An entirely separate reason for believing that the linear hypothesis is conservative derives from evidence that the latent period for radiocarcinogenesis, the delay between radiation exposure and development of cancer symptoms, increases as radiation dose decreases. There is evidence for this from many animal experiments (Up64; Hu67; Bu68; Do69; Hu69; Sh69; Ni70). An example is shown in Fig. 9 for injec-

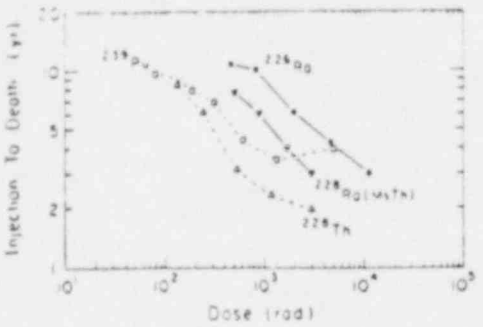

FIG. 9. Survival time for beagle dogs who developed bone cancer from injections of various α-emitting radioactive isotopes (Do69) vs dose to their bone at 1 yr before death. Note that ^{239}Pu and ^{228}Th are much more effective than ^{226}Ra because the former concentrate on the bone surfaces. These differences are taken into account in converting rad to rem.

tions of radioactivity into beagle dogs (Do69); each point represents the average time delay for a number of dogs, all of whom died of bone cancer. Dogs not injected (controls) had a post injection survival of 11.3 yr, so we may infer from Fig. 9 that doses very much below 100 rad to the bone would be totally ineffective.

There is evidence on this effect from human data for the group of German ankylosing spondylitis patients injected with ^{224}Ra (May78a), for skin cancer in Japanese radiological workers (Ki73), and for the U.S. radium dial painters (Ev74). Data for the last-named group are given in Fig. 10 which shows the time delay vs dose for each case: since there is no averaging, there are wide fluctuations, but when it is recognized that the data are cut off by time limitations on the study at an ordinate value of about 55 yr, it is reasonably clear that the trend of the data are as shown by the line, sloping upward to the left.

If the latent period increases with decreasing dose, at a rate even remotely approaching those indicated in Fig. 9 and 10, at low doses this latent period will far exceed the normal life span so there will be *no* cancers caused by low level radiation.

3. "NEW EVIDENCE" INDICATING LINEAR HYPOTHESIS *UNDER*-ESTIMATES EFFECTS

The news media have recently given heavy publicity to several reports from different sources purporting to indicate that the linear hypothesis grossly *under*-estimates health effects of low-level radiation. We here consider these reports, treating each in a separate section.

(A) *Mancuso–Stewart–Kneale (MSK) studies of Hanford workers*

Probably the best known report of the type under discussion is a study of the effects of occupational radiation exposure to workers at the Hanford Laboratory (near Richland, WA) by Mancuso *et al.* (Man77), which we refer to hereafter as MSK. It is based essentially on the 3500 male deaths from the work force of 25,000, searching for correlations between causes of death as obtained from death certificates and radiation exposures as recorded by film badges. MSK found that those who died of cancer had slightly higher radiation doses than those who died of other causes—2.1 vs 1.6 rad, and that those who received more radiation more frequently had cancer as their cause of death. Note that these statements do not necessarily imply that those who received more radiation had a larger probability of dying of cancer. In fact that is not the case, at least

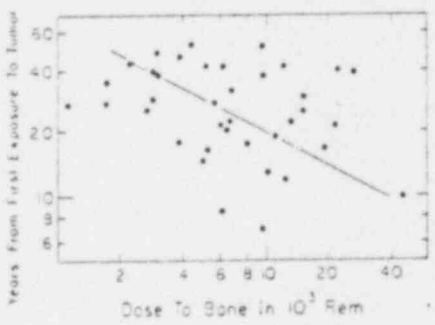

Fig. 10. Time between first radiation exposure and development of tumor for individual radium dial painters (Ev74). Line shows average trend in data considering that the ordinate is effectively cut off at about 55 yr by the length of the follow-up.

within the statistical accuracy of the available data (Gi78): the age-adjusted cancer mortality rates per yr per thousand white males aged 25–70 were 1.7 ± 0.4 for 0–2 rem exposure, 2.1 ± 0.8 for 2–5 rem, and 1.4 ± 0.6 for >5 rem, as compared to 2.1 for the total U.S. population (± denotes 95% confidence limits). There are some tricky aspects in considering only those who have died in that no consideration is given to the great majority of those exposed since they are still alive. Moreover, a higher proportion dying of cancer can mean either a higher probability of cancer death *or* a lower probability of dying from other causes.

MSK break their data down in various ways, and when this is done they obviously find some ages, some employment periods, some time interval between exposure and death, etc. which give larger results than average. This especially applies to types of cancer; for example, they find large excesses for cancer of the bone marrow (22 observed vs 13.4 expected), pancreas (49 obs. vs 37.3 exp.), and lung (192 obs. vs 144 exp.); they give no further consideration to types where results are in the opposite direction, as for lymphatic leukemia (3 obs. vs 9.4 exp.), other RES neoplasms (5 obs. vs 20.3 exp.), and genito-urinary cancers (15 obs. vs 30.9 exp.).

Since the difference in radiation dose between cancer and non-cancer deaths is so small and they explain the entire effect by these small differences, they naturally find very small "doubling doses". Some of these are listed in Table 2, in which we include also the results of their later revision (Kn78).

The MSK work has drawn criticism from a wide variety of sources (Gi77; Gi78; Mark78; Brod78; Sa78; Sa78a; Ki78; Re78a; Re78b; Pa78; An78; NR76; NR78; Rub78; Hu79; Ta79; Co78; Mo78; Ge78; Spi79; Gi79; Co80a). We attempt here to summarize it only briefly. There has been widespread criticism of the statistical methodology and handling of data; to cite one example, for multiple myeloma there were 8 cases among men with <1 rem exposure and 3 cases with about 30 rem, and this was treated, using averaging, as equivalent to 11 cases with 8 rem each. This procedure, plus some non-standard disease grouping—"bone marrow" cancer, which includes multiple myeloma, is not a standard classification—led to the estimate in Table 2 of 9.7 radiation induced deaths due to bone marrow cancer whereas the 3 myeloma victims were the only ones with appreciable exposure (An78).

There was no consideration given to the "healthy worker" effect (Gi78)—the fact that the Hanford workers had steady jobs means that they had less chronic disease than average which would result in their dying less frequently from some other causes and hence relatively more frequently from cancer. MSK used 1960 national statistics for comparisons, although most of the worker deaths occurred closer to 1970; since lung cancer was increasing rapidly over that period, this error alone explains their entire lung cancer effect (Hu79).

MSK paid no attention to the fact that dose correlates with many other factors such as years of service, job type and socioeconomic class, and these in turn correlate with exposures to other carcinogens and to general

Table 2. Results of study of Hanford workers by Mancuso et al. Table is from (Re78b)

Cancer type	Number of cases		Doubling dose (rad)	
	Total	Due to rad	(Man77)	(Kn78)
Bone marrow	14	9.7	0.8	3.6
Pancreas	31	6.0	7.4	15.6
Lung	130	12.6	6.1	13.7
All RES	47	11.1	2.5	—
All cancer	442	25.8	12.2	33.7

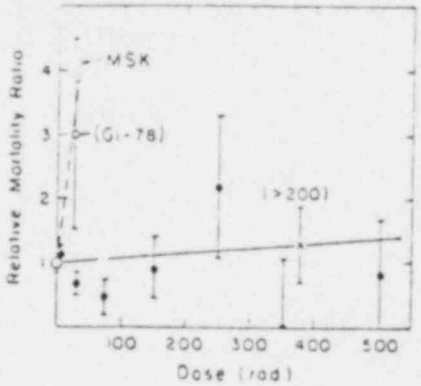

FIG. 11. Mortality from cancer of the pancreas among Japanese A-bomb survivors (black circles), among Hanford workers (Gi78, open square), and according to MSK (dashed line). Point labelled (>200) represents the average of all data above 200 rad. From (Co80b).

mortality from cancer; for example, technicians were exposed to much more radiation than office workers, and they were also exposed to far more chemical carcinogens. Several of the critics cited above pointed to lack of increased leukemia incidence as a strong point against the validity of the study. It was never made very clear what MSK meant by "doubling dose" as regards the role of natural background radiation, and in fact the derivation of doubling doses below total background exposure has a ring of unreality.

At least two independent analyses were made of the Hanford worker data used by MSK (Gi78; Hu79), and the conclusions of each were that the only results worthy of consideration were those for cancer of the pancreas and multiple myeloma. For the former there were 5 cases with exposures above 10 rad vs 1.4 expected, and for the latter there were 3 cases vs 0.4 expected (Hu79); for exposures above 15 rem there were 3 cases vs 1.0 expected for the former and 2 cases vs 0.4 expected for the latter (Gi78). These results are clearly not explainable as statistical fluctuations, although they could easily be due to a factor which correlates with radiation dose such as exposure to chemical carcinogens.

One way of deciding whether radiation is the causative agent is to check for evidence of these diseases among other groups exposed to radiation. Data on cancer of the pancreas among Japanese A-bomb survivors are shown in Fig. 11 along with the single data point from the Hanford workers [taken from (Gi78)], and the prediction from the MSK doubling dose (Co80a). It is clear that the MSK conclusion is grossly inconsistent with the A-bomb survivor data.

The Japanese A-bomb survivor data on multiple myeloma (Ni73) are much more sparse—4 cases vs 1.9 expected up to 1965— but doubling dose is of the order of 100 rad which is 30 times the MSK doubling dose for bone marrow cancers. The rate derived for the A-bomb survivors was 3×10^{-6} cases/man-rem. In studies of patients exposed in thorotrast treatments, the rate was only 0.25×10^{-6} cases/man-rem, and among the German patients treated for ankylosing spondylitis with ^{224}Ra, there were 54 bone cancers but no cases of multiple myeloma which corresponds to less than 0.04×10^{-6} cases/man-rem (May79b). Castleman searched the X-ray exposure history of multiple myeloma patients, and found no correlations (Ca79).

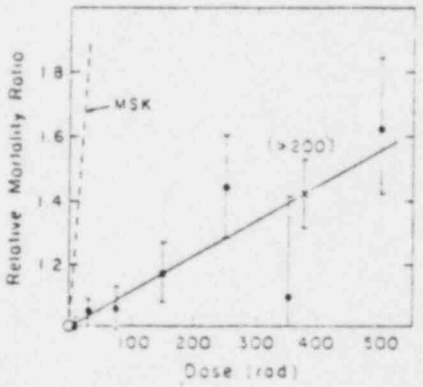

FIG. 12. Mortality from all cancers except leukemia among Japanese A-bomb survivors, and according to MSK (dashed line). Point labelled (>200) represents the average of all data above 200 rad. From (Co80a).

By very complicated and questionable procedures, MSK derived a doubling dose for all cancers as 12.2 rad (Man77), later revised to 32.7 rad (Kn78). The latter is shown in Fig. 12 along with the data from the Japanese A-bomb survivors for all cancers except leukemia (Be77). Clearly there is an enormous discrepancy here, by about a factor of 30 in doubling dose. It should be noted that MSK make no effort to compare their results with those for the Japanese A-bomb survivors or with any of the other groups with high radiation exposure.

The reaction to MSK of the prestigious national and international groups charged with evaluating health effects of radiation has been cool. The minutes of the Stockholm meeting of ICRP states "the Commission has concluded that the information available up to May 1978 does not call for changes in the risk factor given in ICRP Publication 26". The latter was published in January 1977 and MSK was published in November 1977. They made no mention of MSK.

The UKNRPB (Re78b) report on MSK concludes: "Despite the claims of the authors, a wide body of experts agree that there is no evidence in the Hanford data to support the suggestion that ICRP [26] values do seriously underestimate the risk."

The National Academy of Sciences BEIR Committee 1979 report has not yet appeared, but in a press conference it was stated that it found "no substance" in the MSK work.

(B) *Rotblat comments on A-bomb survivor data*

Rotblat has pointed out that the survivors of the A-bomb attacks on Japan are a select group in that they survived the injuries and trauma of the attack (Ro78a). He points out that for some types of cancer, evidence from medical patients treated with radiation indicates higher risks, and explains the difference as due to the fact that the A-bomb survivors are such a select group. He does not seem to consider the fact that the medical patients are also a select group in that they are already suffering from another serious disease. He concludes that if there had been no early deaths from injuries and trauma in the original bombing episodes, the cancer deaths from radiation would have been 5 times higher.

It is interesting to point out that the Rotblat effect should be very much larger for the high dose cases than for the low dose cases, since the former were close to ground zero while the latter were far away and thus much less likely to be directly injured by the blast, heat and other effects of the bomb (Co59). Thus, if Rotblat is correct, the data on the right side of Fig. 12 would be moved up by an order of magnitude while the low dose data would be little affected. This would give a curve very much concave upward. In any case, the effects of low doses would not be much changed.

The Rotblat thesis has not been accepted by the prestigious evaluation groups, and there has been little indication of significant acceptance by the scientific community.

(C) *Najarian studies of workers at Portsmouth Navy Yard*

Thomas Najarian, a physician practicing in Boston, got the impression from discussions with patients, that there might be an excess of leukemia among workers at the Portsmouth (NH) Naval shipyard where nuclear ships are serviced, and suspected that it might be due to their occupational radiation exposure. With the help of a team of reporters from the Boston Globe, he searched through 90,000 death certificates and found 1700 former Portsmouth workers. Of these, 22 died of leukemia as compared with only 5 expected, according to the February 1978 story in the Boston Globe; according to a more scientific account published in *Lancet* (Na78), there were 1450 workers identified, and among these there were 20 leukemias vs 10.8 expected. The latter number is subject to considerable variation depending on the control group chosen, as explained in section 1 above.

Age distribution is also a sensitive parameter (La78), as the leukemia rate increases rapidly

with increasing age—the average age of Portsmouth workers is clearly above the average age of all males which includes children. It was not explained how the "expected" number was derived. It is also well known that rates for various cancer types show strong occupational correlations; in a study in Washington State (Mi76), poultrymen were found to have over double the average leukemia rate, and such diverse groups as dairymen, bankers and bus drivers also seemed to suffer far above average leukemia rates.

Najarian attempted to separate out radiation workers by asking their close relatives whether they remembered them wearing film badges, a somewhat marginal methodology. He thereby identified 146 radiation worker deaths among which there were 6 leukemias vs 1.1 expected.

At this stage, data finally became available on doses measured with film badges, and it turned out that only 3 of the 6 radiation worker leukemia victims had any radiation exposure, and the average exposure for the group was 1.3 rem. This raises the question of the role of natural and medical X-ray radiation, which exposed the average worker to about 10 rem by the time of his death. How is it possible that 10 rem causes at most 1.1 leukemias, whereas an extra 1.3 rem to make the total 11.3 rem causes 6 leukemias? No imaginable dose-effect relationship could explain such a situation, especially in view of the variability of natural radiation with geography which is not correlated with leukemia rates.

It may be noted that there are many chemical carcinogens in a shipyard environment, including benzene and other organic solvents, welding debris and asbestos.

On 19 June 1979, Najarian, appearing before Sen. Edward Kennedy's subcommittee, withdrew most of his claims, and those not withdrawn were heavily criticized in other testimony. As reported in the Boston Globe which first promoted Najarian's work, a highly agitated Sen. Kennedy chided Najarian with "I don't think we ought to be alarming families unduly... we have seen you repudiate two areas of your study, and the National Cancer Institute has repudiated the third" (We79).

(D) *Bross re-analysis of Tri-State Study*

Bross and collaborators (Bros79) reported a re-analysis of the well known "Tri-State Study" carried out in NY, MD and MN in 1959–62 (Gr66). They emphasize that different people respond to radiation in different ways, and attempt to take this into account by considering 5 different categories of people—this multiplies the number of parameters available for adjustment by 5. They then consider the triple correlation between number of X-rays received, cancer and heart disease. They treat the problem with what they call a new statistical methodology, which they explain only cursorily. They finally conclude that the linear hypothesis under-estimates the effects of low-level radiation by an order of magnitude.

Immediately following the Bross article in *American Journal of Public Health*, there was a scathing critique of it by Boice and Land (Bo79) of National Cancer Institute. It is ironic that the *New York Times* published a long article on the Bross paper (NY79), but made no mention whatsoever of the Boice–Land critique.

Boice and Land questioned the Bross statistical methodology and method of choosing parameters (the latter seems to be highly arbitrary). They note that there are clearly too many parameters for the individual values of each to have statistical significance. They point out that there is a great deal of independent evidence that radiation does *not* cause heart disease; for example, among the Japanese A-bomb survivors, there is no more heart disease among those exposed to >200 rad than among those with less than 1 rad exposure.

They point out many problems in the data collection process: X-rays may have been given for a pre-leukemic disease—half of all X-rays were made within the 5 yr previous to development of leukemia; most information was obtained from interviews with relatives, a generally unreliable source, with a strong tend-

ency to look for causes; there may well have been a tendency for interviewers to probe harder for radiation information on those whom they knew to be leukemia victims than on controls; and the dosimetry was very crude. They also point out that the Bross analysis considers only 206 of the 399 cases in the Tri-State Study, and gives no explanation for not considering the others.

The National Academy of Sciences BEIR Committee Report considered the Bross paper and reported in a press conference that it found "no substance" in the work.

The 1979 paper by Bross and collaborators is the culmination of a series of previous papers re-analyzing the Tri-State Study (Ci68; Bros72; Bros77). The evolution of these, as well as a preliminary version of the 1979 paper, were heavily critiqued by Rothman (Ro78b) in which he emphasized that the whole approach was highly unscientific. Details of the Bross 1972 paper were critiqued by MacMahon Mac72) and by Mole (Mo74).

(E) *Lyon paper on excess childhood leukemia down-wind from Nevada Test Site*

Lyon *et al.* (Ly79) reported an analysis of mortality from childhood (age 0–14) leukemia in Utah, in which they divided the state into areas of high fallout and low fallout from the Nevada Test Site, and also considered time periods of high fallout (1951–58) and low fallout (before and after those dates). Where a person's pre-leukemia years were partially during low fallout and partially during high fallout time periods, the responsibility for the leukemia was divided proportionately between the two. Their results on childhood leukemia mortality rates are shown in Table 3(a).

There appears to be a large excess in the high fallout areas attributable to the high fallout time period which is not reflected in the data for the low fallout areas or for the total U.S. If the earlier and later time periods are assumed to represent the situation in the absence of fallout, this excess consists of 32 cases vs 13.1 expected.

On the other hand, it should be noted that the statistical uncertainty is rather high, enough so that even under the assumptions used there is a few percent chance that the entire effect is simply a statistical fluctuation. Moreover, the effect is not so much an excess over the U.S. average during the high fallout time period as it is a deficiency relative to the U.S. average before and after the high fallout time period.

It is interesting to include data for other childhood cancers as given in Table 3(b). Note that there was a sharp *drop* in the rate during the high fallout time period as compared with the before-after average. Land has pointed out (La79a) that this drop has about the same statistical significance as the increase for leukemia.

Table 3(a). Childhood leukemia mortality rates in high fallout and low fallout areas of Utah and in the U.S. Figures are rates per 10^5 population, and ± indicates 2 S.D.

Area	1944–50	1951–58	1959–75
High fallout	2.1 ± 1.56	4.39 ± 1.58	1.96 ± 1.23
Low fallout	3.84 ± 1.14	4.21 ± 0.70	3.28 ± 0.64
Total U.S.	3.2	3.5	3.1

Table 3(b). Childhood cancers other than leukemia (see caption for Table 3(a))

Area	1944–50	1951–58	1959–75
High fallout	6.36 ± 2.72	3.07 ± 1.37	3.05 ± 1.60
Low fallout	4.52 ± 1.25	4.33 ± 0.68	3.09 ± 0.62

The Lyon paper actually has little direct bearing on the question of effects of low level radiation since there are no data available on doses. However, the Utah situation has often been cited as evidence for increased danger from low level radiation, and the Lyon paper is the only one in the scientific literature on that subject. There has been widespread publicity about evidence for increased adult leukemia in Southern Utah (PBS79) due to fallout, but none of it seems to be of a quality worthy of scientific consideration.

(F) *Smoky Test*

In 1957, 3200 soldiers participated in a tactical warfare exercise in which they occupied an area in which a nuclear bomb had been detonated shortly before. Their radiation exposure as obtained from film badges averaged about 1 rem.

A group of these men was recently studied, and there were found to have been 8 leukemias among them. The number of cases expected is poorly known because this is highly sensitive to their age distribution which cannot be reconstructed because records were lost in a fire in St. Louis. If their average age was 22, only 1.8 cases would have been expected by now, but if their average age was 35, there would be 5 cases expected (Lap79).

If the facts as outlined were correct and there were 8 cases vs roughly 3 expected, one is faced with the idea that natural plus medical exposures of 10 rem (plus all other causes of leukemia) caused 3 cases while 1 rem additional caused 5 cases. No reasonable person could believe in such a dose-effect relationship, especially in view of the low leukemia rates in states with high natural background. If the extra leukemias were indeed caused by radiation, there must have been an error in dosimetry. It has been suggested that the soldiers involved may have inhaled or ingested radioactivity; this would not affect film badge readings, so their doses may have been much higher.

In any case it seems clear that the Smoky Test evidence sheds no light on the dose-effect relationship for low-level radiation, although a linkage has frequently been implied in press reports.

(G) *Reported excess leukemia due to mill tailings in Grand Junction, CO*

The ABC-TV "20–20" program has more than once presented the viewpoint that low-level radiation is much more dangerous than it is generally believed to be, with heavy emphasis on a report that leukemia rates are double what is expected in Grand Junction, CO (Cu78) where uranium ore-processing mill tailings were used in construction. The "expected" rate is the Colorado state average, and the result is based on 41 cases vs 20.1 expected in Mesa County (which includes Grand Junction) between 1970 and 1976.

It is widely believed that most leukemias are due to environmental factors, since, for example, the U.S. mortality rate from that disease has increased by a factor of five over the last half century, and the rates in different states vary by as much as 30% (with negligible statistical uncertainty). It thus seems reasonable to conclude that there may be a leukemogenic agent in the Mesa County environment. This, of course, does not imply that it is radiation.

A case-control study was carried out (Cu78) in which it was found that there was no significant difference between the leukemia victims and the control groups as regards exposure to tailings. This would seem to indicate that exposure to tailings is not the responsible factor. Further evidence for this is that the principal expected radiation effect from mill tailings is lung cancer (due to radon emissions), and there was no evidence for an increased level of lung cancer in Mesa County (Cu78).

Even after all of these facts became known, the ABC-TV "20–20" program referred to them as "inconclusive" and repeated their assessment that mill tailings are causing large numbers of leukemias in Grand Junction.

(H) *Excess lung cancer near Rocky Flats, CO*

The ABC-TV "20–20" program also featured evidence for increased cancer rates in Jefferson County, CO in which the Rocky Flats plutonium processing plant is situated. However, these charges do not seem to be verified by the statistics (La79). The total cancer mortality rate in Jefferson County is just equal to the Colorado State average which is somewhat below the U.S. average. Since the principal expected effect of plutonium in the environment is lung cancer, statistics on that disease are also of interest here: before the Rocky Flats plant was built, the lung cancer rate in Jefferson County was slightly greater than the U.S. average, but in recent years it has been significantly below the U.S. average. No information supporting the charges of increased cancer near Rocky Flats has appeared in the scientific literature.

(I) *Summary on "new evidence"*

None of the "new evidence" purporting to indicate that the linear hypothesis underestimates effects of low-level radiation seems to stand up under scientific scrutiny. None of it has been accepted by any of the prestigious evaluation committees such as BEIR, ICRP, etc. The publicity given to them by the news media thus seems to be completely unwarranted.

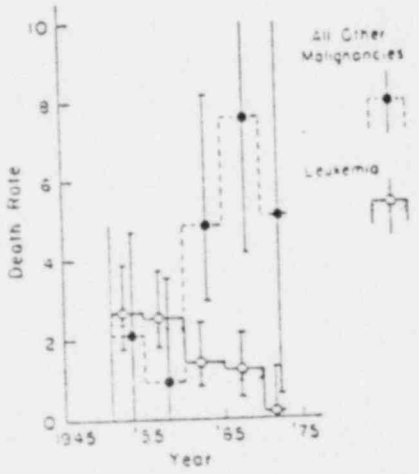

FIG. 13. Variation of death rates from leukemia and from all other cancers with time for Japanese A-bomb victims. Exposure was in 1945, and data begin in 1950. Ordinate is mortality rate/10^4 rad, and error bars indicate 90% confidence limits. From (UNSCEAR77).

4. ESTIMATION OF EFFECTS OF LOW-LEVEL RADIATION

We have reviewed a great deal of evidence indicating that the linear hypothesis gives a conservative estimate of the effects of low-level radiation. This leaves us with the problem of estimating risks per unit radiation dose for use with the linear hypothesis. As indicated previously, this is done by analysing effects of high-level radiation situations. From these analyses, one derives the number of fatalities per yr per rem of exposure. However, in the process of converting these numbers to a lifetime risk, there are several complications:

(1) *Latent period*

The mortality rates from leukemia and from all other cancers among the Japanese A-bomb survivors are shown vs time in Fig. 13. We see that for the solid tumors the rate was relatively low until 1960, 15 yr after the exposure, and then suddenly jumped to a much higher level. This implies that there is a long latent period during which cancers develop without presenting symptoms. This must be taken into account in any analysis. In many of the high radiation incidents, as with miners exposed to radon or radium dial painters exposed to internally deposited radioactivity, the exposure takes place over a very long time period, which greatly complicates the analysis.

(2) *Duration of risk*

Once the latent period is past and the risk approaches its full value, one must estimate how long this risk will persist. There is reasonable evidence for leukemia in Fig. 13 that the risk falls off with time and becomes much less after about 25 yr. For other cancers, however, there is little direct evidence on this question. There is a general impression that it lasts about

Table 4. Cases of all cancers but leukemia occurring between 1960 and 1970 among Japanese A-bomb survivors of various ages at time of exposure. Data are from (NAS72)

Age in 1945	Approximate number of subjects	Average dose (rem)	Cases observed	Cases expected	Number/ 10^6-rem-yr
0–9	5000	111	6	2.1	0.75
10–19	5500	157	25	16.9	0.72
20–34	5500	134	85	63.9	3.1
35–49	5800	122	286	251	5.4
50+	2500	106	221	210	5.3

30 yr, and this is often used in calculations. In most cases, results would not be very much different if the risk persists throughout the remainder of life.

(3) *Age dependence*

There is evidence in Table 1 that young children develop leukemia at about double the adult rate for a given exposure. For other cancers, the situations seems to be reversed as indicated by data for the Japanese A-bomb survivors in Table 4 where we see that those exposed as children experienced a lower rate by nearly an order of magnitude. On the other hand, it must be recognized that those exposed as children have not yet reached the age range where cancer is an important risk, whereas those exposed as adults are well into that age range. Because of this, the ratio of observed to expected numbers of cancers is actually higher among those exposed as children. It is an open question as to whether this high ratio or the low absolute incidence will persist when those exposed as children reach the age of high normal cancer risk.

In treating the problems of latent periods, duration of risk, and age dependence, one can adopt models (NAS72) or use piece-meal estimates (UNSCEAR77). In either case, it would be unrealistic to expect any high degree of accuracy. Moreover, data from different high radiation dose situations are often inconsistent, as may be seen from Table 1.

Nevertheless, BEIR, ICRP and UNSCEAR have all given estimates of age-averaged risks. These are listed in Table 5. We see that there is a reasonable degree of consistency among the three estimates. ICRP gives less detail than the other two, so more types of cancer are included in the "other" category.

It may be noted that the "totals", which represent the effect of radiation to the whole body, are not equal to the sum of the risks to the various organs. Part of the reason for this is that the totals represent a male-female average, whereas the breast cancer entry applies to females only. When this is corrected for, there is still a considerable discrepancy for the ICRP and UNSCEAR estimates, but they offer no explanation for it. Actually they seem to derive the total independently from summing the risks to each organ, and the uncertainties are large enough to cover the discrepancies. In any case, Table 5 gives the best information available for estimating effects of low level radiation.

An average radiation-induced cancer shortens the victim's life by about 20 yr, so if we adopt the BEIR estimate, 1 rem reduces life expectancy by $180 \times 10^{-6} \times 20\,\text{yr} = 1.3$ days.

Table 5. Estimates of cancer risk per 10^6 rem by the different evaluation groups (NAS72, ICRP77, UNSCEAR77). *Risks are averaged over ages, and the totals are sex-averaged*

Type of cancer	BEIR	ICRP	UNSCEAR
Leukemia	25	20	15–25
Lung	39	20	25–50
Breast (female)	90	25	60
Bone	6	5	2–5
G-I tract	30	—	25
Thyroid	—	5	5–15
Other	30	30	~25
Total	180	100	120

Table 6. Deaths from various causes among survivors of A-bomb attacks on Japan vs radiation exposure. Listings are total deaths 1950–74 [upper line] and ratio of these to the number expected in the absence of radiation [lower line]. The final column is the observed/expected ratio for the three highest dose groups divided by the observed/expected ratio for the zero exposure group. Data are from (Be77)

Disease	Radiation dose received (rad) 0	1–9	10–49	50–99	100–199	200–299	300–399	400+	200+ : 0
Leukemia	31	23	20	7	16	15	12	20	17.5
	0.50	062	0.76	0.96	2.8	5.9	10.3	17.6	
Other cancers	1586	881	704	202	159	80	28	58	1.42
	0.97	0.97	1.02	1.03	1.14	1.39	1.05	1.58	
All non-cancer	6367	3652	2684	747	513	202	91	149	0.98
	1.01	1.01	0.99	0.97	0.97	0.95	0.92	1.11	
Cerebrovascular	1891	1080	800	228	159	57	27	35	0.92
	1.01	1.01	0.99	0.99	1.03	0.92	0.64	0.89	
Circulatory system	1264	704	534	179	98	39	23	24	1.01
	1.01	0.98	0.98	1.17	0.96	0.97	1.23	0.93	
Blood and blood-forming organs	40	23	23	7	9	6	3	6	5.16
	0.77	0.81	1.05	1.12	2.04	3.19	3.59	5.44	
Digestive system	700	383	293	79	58	27	5	21	1.06
	1.02	0.97	1.00	0.95	1.01	1.16	0.47	1.40	
Other natural causes	2059	1158	798	204	140	57	24	48	0.92
	1.045	1.021	0.94	0.85	0.88	0.89	0.81	1.20	
Accidents, poisons, violence	546	324	195	63	46	12	6	18	0.83
	1.03	1.03	0.90	1.04	0.97	0.57	0.64	1.37	
Suicide	183	122	50	22	15	1	0	2	0.17
	1.09	1.18	0.71	1.09	0.91	0.13	0	0.44	

One mrem then reduces life expectancy by 2 min. By way of comparison, smoking a single cigarette reduces life expectancy by 10 min, and an overweight person reduces his life expectancy by 15 min for each extra 100 calories ingested (Co79a).

With present technology, low level radioactivity releases if all U.S. electric power were nuclear are estimated (APS78; Po76; NRC76a; UNSCEAR77) to cause an average population exposure of about 0.2 mrem/yr, or 40,000 man-rem yr in the U.S. This might then be expected to cause $180 \times 10^{-6} \times 40{,}000 = 7$ additional cancers per yr among the public. This corresponds to a loss of life expectancy for the average American of about 30 min, which represents a risk equal to that of smoking one cigarette every 20 yr, or of an overweight person gaining an additional 0.01 oz.

There has been speculation as to whether radiation causes diseases other than cancer in those exposed. The best evidence on this is from the Japanese A-bomb survivors, which is listed in Table 6 and compared there with the evidence on leukemia and other cancers. We see that, with the relatively minor exception of diseases of the blood and blood-forming organs which are closely related to leukemia, there is no indication that any somatic effect other than cancer is related to radiation.

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