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STANDARDS

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UNITED STATES OF AMERICA  
NUCLEAR REGULATORY COMMISSION

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BRIEFING ON DEVELOPMENT OF RADIATION  
PROTECTION STANDARDS

- - - -

PUBLIC MEETING

Nuclear Regulatory Commission  
One White Flint North  
Rockville, Maryland

Wednesday, August 1, 1990

The Commission met in open session,  
pursuant to notice, at 10:00 a.m., Kenneth M. Carr,  
Chairman, presiding.

COMMISSIONERS PRESENT:

KENNETH M. CARR, Chairman of the Commission  
KENNETH C. ROGERS, Commissioner  
JAMES R. CURTISS, Commissioner  
FORREST J. REMICK, Commissioner

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## STAFF AND PRESENTERS SEATED AT THE COMMISSION TABLE:

SAMUEL J. CHILK, Secretary

MARTIN MALSCH, Deputy General Counsel

DR. ARTHUR UPTON, BEIR V Committee

DR. WARREN SINCLAIR, President, National Council on  
Radiation Protection and Measurements

DR. WILLIAM ELLETT, BEIR V Committee

CHARLES MEINHOLD, Division Director, Radiological  
Sciences Division, Brookhaven National Laboratory

DR. BILL MORRIS, Director, Division of Reg.  
Applications, RES

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## P-R-O-C-E-E-D-I-N-G-S

10:00 a.m.

CHAIRMAN CARR: Good morning, ladies and gentlemen.

This morning the Commission will be briefed about the development of radiation protection standards by representatives of the organizations that helped shape these standards. The purpose of the briefing is to describe the process by which radiation protection standards are developed based on estimates of the risks associated with ionizing radiation and to highlight the special precautions that are implemented throughout this process to ensure protection of the public health and safety and the environment.

This topic is especially relevant at this time given the Commission's recent approval of the final radiation production standards in 10 CFR Part 20 which should be published in the near future.

Joining us today for the briefing are Doctor Arthur Upton and Doctor William Ellett from the National Research Council's Committee on the Biological Effects of Ionizing Radiation, the BEIR V Committee; Doctor Warren Sinclair and Mr. Charlie Meinhold from the International Commission on Radiological Protection and the National Council on

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1 Radiation Protection and Measurements; and Doctor Bill  
2 Morris from the NRC staff.

3 Welcome, gentlemen.

4 Do any of my fellow Commissioners have any  
5 opening comments?

6 If not, please proceed, Doctor Upton.

7 DOCTOR UPTON: Thank you, Mr. Chairman.

8 (Slide) May I start with the first slide,  
9 please?

10 The first slide provides the membership,  
11 lists the membership of the so-called BEIR V  
12 Committee. The BEIR V Committee, B-E-I-R stands for  
13 Biological Effects of Ionizing Radiation. The  
14 Committee represents the fifth incarnation, so to  
15 speak, of an expert group that convened under the  
16 auspices of the National Academy of Sciences National  
17 Research Council to review the status of our knowledge  
18 of the health effects of low-level ionizing radiation.

19 The forerunner of the BEIR V Committee was  
20 the BEIR III Committee which provided a comprehensive  
21 review published in 1980. Concern about the problem,  
22 of course, goes back to the post-war era when the  
23 testing of nuclear weapons in the atmosphere led to  
24 rising levels of global fallout. It was the  
25 geneticists then who suggested that this increase in

1 the background level above and beyond natural  
2 background posed a threat to future generations. But  
3 we didn't think at that time there were threshold  
4 doses for mutagenic effects, that any level of  
5 background radiation would carry some genetic risk.

6 More recently, it seemed that the  
7 carcinogenic effects, if anything, might be more  
8 important. In the decade since the BEIR III Committee  
9 and the BEIR V Committee, reassessment of the A-bomb  
10 dosimetry in Hiroshima and Nagasaki, as I'll point out  
11 in a moment, suggested that the risks might be larger  
12 than had been thought before. There was another  
13 decade of follow-up of A-bomb survivors and other  
14 irradiated populations. So, it was time to reassess  
15 the subject.

16 (Slide) If I could have the next slide,  
17 please.

18 This brings out that in Hiroshima the dose  
19 equivalent shown in the lower right-hand corner with a  
20 new dosimetry, the DS86 dosimetry shown on the right  
21 was only about two-thirds of what was thought to be  
22 true in 1980, principally because of the much smaller  
23 neutron component in the radiations. In the upper  
24 left, we see the gamma ray component in the gray bar  
25 and the neutron component in the black bar. As you

1 can see, with the new dosimetry, the DS86 dosimetry,  
2 the neutron component was much, much smaller.

3 Historically, the risks to the population  
4 at Hiroshima for a given distance or a given dose  
5 seems substantially higher in Hiroshima and that had  
6 been attributed to the contribution of the neutrons to  
7 the dose there, the neutrons being known to be more  
8 effective than gamma rays. With the greatly reduced  
9 neutron component and the reduced dose equivalent  
10 shown in the lower right-hand corner, for a given  
11 total dose the effects now seem substantially larger.

12 (Slide) Next, please.

13 This set of bar graphs is the counterpart  
14 for Nagasaki. Again, in the upper left, the neutron  
15 component is substantially smaller but it was always  
16 relatively small. Finally in the lower right,  
17 weighting the neutron component with an appropriate  
18 quality or RBE factor, the dose equivalent today is  
19 thought to be substantially smaller. So, these  
20 factors led to some expansion of risks needed  
21 evaluation at this time.

22 (Slide) Next, please.

23 Another factor is that as the populations  
24 in Hiroshima have been followed, the magnitude of the  
25 radiation-induced excess cancer risk has grown with



1 attained age. This is a figure from the BEIR III  
2 report and at the top of the figure you'll see what  
3 are the two alternative models that were presumed to  
4 be applicable. The so-called absolute risk model on  
5 the left held that following a latent period, the  
6 cancer risk would appear and then would essentially  
7 parallel the natural baseline risk shown in the dashed  
8 line. Projected over a lifetime then, there would be  
9 a given additional number of cancers each year  
10 attributable to a given dose.

11 On the right-hand side, at the top, you  
12 see the so-called relative risk model or  
13 multiplicative risk model where after the elapse of  
14 the latent period and the appearance of a radiation  
15 induced cancer excess, cancer excess grows each year  
16 with attained age, so that the separation between the  
17 radiation exposed population on the top and the  
18 baseline population shown in the dashed line, that  
19 separation grows. The excess then is a given  
20 percentage or a given additional fraction of the  
21 natural incidence.

22 It would appear for the solid tumors in  
23 Japan that the relative risk model or the  
24 multiplicative risk model fits the data somewhat  
25 better over the last decade. Historically in the BEIR

1 III report, that relative risk model led to  
2 substantially higher lifetime risk estimates than did  
3 the absolute risk model or additive risk model.  
4 That's another reason why the risks seem higher today  
5 than they seemed before.

6 For leukemias, shown in the lower figures,  
7 it was thought at the time of BEIR III and still  
8 appears to be the case that the risk makes its  
9 appearance after a latent period and then decays with  
10 time so that ultimately the risk is much smaller than  
11 it was during the peak period of excess leukemia  
12 incidence.

13 (Slide) Next, please.

14 Another factor. At the time of the BEIR  
15 III report, it wasn't clear which of the hypothetical  
16 models relating incidence to dose was indeed the  
17 correct model. This is a figure from the BEIR III  
18 report showing in the upper right hand figure the so-  
19 called linear non-threshold model with increasing dose  
20 plotted on the horizontal. The risk of cancer rises  
21 in proportion to dose.

22 The so-called quadratic model is shown at  
23 the lower left where the risk goes up as the square of  
24 the dose. At the lower right, one has the linear  
25 quadratic model, risk rising linearly with dose in the

1 low dose range, low to intermediate, and then the  
2 curve bends upward as the square term or quadratic  
3 term takes hold, and then in the upper left linear  
4 quadratic model with a cell killing term to account  
5 for the saturation of high doses.

6 In the case of the BEIR V Committee, the  
7 linear quadratic model would appear to fit the  
8 leukemia data best, as it seemed to in BEIR III. Most  
9 of the leukemias have occurred, we're over the peak  
10 now. That's not surprising. Looking at the solid  
11 tumors, the Committee thought that the linear model in  
12 the upper right fit the data better than the others.  
13 So, the use of the linear model contrasts with the  
14 model that was used in BEIR III. BEIR III used the  
15 linear quadratic both for leukemia and solid tumors.  
16 Linear models for solid tumors is another factor for  
17 an increased risk projection, as Doctor Ellett will  
18 bring out.

19 (Slide) Next, please. May we have the  
20 next slide, please?

21 We're dealing now with many cancers, not  
22 just leukemia. Leukemia is shown at the top.  
23 Relative risk of leukemia is highest, but cancers of  
24 many sites and the number of radiation-induced  
25 sites -- the number of sites at which the excess has

1 appeared continues to grow with time as one follows  
2 the population. Suffice it to say not all forms of  
3 cancer are increased in frequency.

4 (Slide) Next, please.

5 Doctor Ellett will review the models that  
6 were used for estimating the cancer risks. Let me say  
7 just a word or two about the genetic risk estimates.

8 There's been little change over the past  
9 20 years in genetic risk estimates. They've been  
10 based largely on the information coming out of  
11 experiments with laboratory animals, principally mice.  
12 We do now have substantial data from the children of  
13 the A-bomb survivors in which no genetic detriment has  
14 been observable, despite substantial efforts to  
15 identify it. So, we're still making estimates largely  
16 on the basis of the mouse data and you'll notice in  
17 the BEIR III estimates the largest category of genetic  
18 detriment attributed to radiation was the so-called  
19 regularly inherited diseases. Some 20 to 200  
20 additional cases per million live born per rem per  
21 generation were thought to be attributable to the  
22 radiation.

23 (Slide) In BEIR V -- next slide,  
24 please -- there was no effort to estimate the genetic  
25 detriment as expressed in the regularly inherited

1 diseases principally because we simply don't know to  
2 what extent this type of disease is attributable to  
3 newly produced mutations. Most of us carry  
4 detrimental genes. Most of the common diseases of old  
5 age, arthritis, heart disease, cancer, are thought to  
6 have some genetic component, presumably for the most  
7 part in the regularly inherited category.

8 But if we eliminate those, as the BEIR V  
9 Committee did, there really is no substantial  
10 difference between the new genetic estimates and those  
11 that we've had in the past.

12 (Slide) Next, please.

13 Another category of radiation-induced harm  
14 was prominent in the review of the BEIR V Committee  
15 and that is depicted here. This is a curve relating  
16 the incidence or prevalence of severe mental  
17 retardation to dose plotted on the horizontal scale.  
18 One can see at the top, the line at the top, that in  
19 those A-bomb survivors who were irradiated between the  
20 8th and the 15th week of gestation, the frequency of  
21 severe mental retardation increased steeply with dose,  
22 amounting to some 40 percent of the dose of the gray.

23 The data don't indicate clearly whether  
24 there's a threshold in the range of 10 to 20 gray.  
25 That can't be excluded. But neither did the data rule

1 out the possibility of some linear slope in the low  
2 dose domain.

3 A smaller excess was seen in children  
4 irradiated between the 16th and the 25th week. But  
5 children irradiated earlier than the eighth week,  
6 early in the first trimester or after the 25th week of  
7 development, that is the third trimester, there was no  
8 demonstrable effect on the frequency of this severe  
9 developmental disturbance.

10 (Slide) The effect on developing brain--  
11 next, please -- is also manifested in a decreased IQ  
12 score, IQ plotted on the vertical. One sees again in  
13 that middle age group, the 8 to 15 week group, a very  
14 pronounced dose-dependent decrease in IQ scores. Seen  
15 also in the 16 to 25 week group, not evident in the 0  
16 to 7 or in the 26 plus.

17 (Slide) So, I think we have evidence  
18 reinforced by the next slide, please, which is school  
19 achievement test scores for the high sensitivity of  
20 the developing brain, especially at a critical stage  
21 in fetal development. These are school achievement  
22 scores, again showing in the sensitive age groups, the  
23 8 to 15, 16 to 25 week age groups dose-dependent  
24 decrements.

25 End of slides, please.

1 I think we don't know from these data that  
2 there is any substantial risk down in the millisievert  
3 range, a few millirem, but clearly as one gets up on  
4 the dose curve, one needs to be concerned about damage  
5 to the developing brain at this critical stage in  
6 organogenesis.

7 In summary then, I think, before turning  
8 the floor over to Doctor Ellett, we've seen increases  
9 in risk estimates for cancer, increases in the risk  
10 estimates for the developing brain at substantial  
11 doses of radiation, no evidence for an increase in the  
12 risk of genetic detriment.

13 I think I'd stop and let Doctor Ellett  
14 explain the cancer risk estimates, if I may, please,  
15 Mr. Chairman.

16 CHAIRMAN CARR: Please.

17 DOCTOR ELLETT: (Slide) If I could have  
18 the first slide, please.

19 I'd like to look a little bit more  
20 carefully at -- that's lovely but it's upside down. I  
21 brought in my slides late, so I was asking for  
22 problems.

23 What we're going to see eventually is the  
24 curve for dose response for leukemia. I'd  
25 particularly like you to notice that the curve seems

1 to be very linear up to about 300 rem. Then there's a  
2 break that indicates that there may be a quadratic--  
3 there is a quadratic component. You can show this  
4 with a high degree of statistical certainty at high  
5 doses. Then there's a decrease at higher doses. Now,  
6 this is in complete contrast to what we saw for solid  
7 cancers where the curve just goes up straight linear  
8 and then just levels off at high doses.

9 What the Committee did was restrict their  
10 analysis to doses below 400 rem so that they were just  
11 looking at the part of the curve before it flattened  
12 out.

13 I think we'll just have to hold it a  
14 minute. Ah, there we are.

15 It's really very linear up to about 300  
16 rem. It isn't true that we're just looking at doses  
17 at 100 rads or so. This is all high dose effects.  
18 The points on that curve are significant up to above  
19 30 rad and it's certainly linear down to about ten rad  
20 or so. We know nothing at doses lower than that.

21 (Slide) There's been a slow increase of  
22 solid cancers over time. If we could look at the next  
23 slide, please. This is historical data up to 1982  
24 from the A-bomb survivors. Estimated risk is shown on  
25 the horizontal slide at the top. This is time in



1 years going down vertically. You can see the leukemia  
2 has fallen off very rapidly with time, but it's still  
3 statistically significant and it is continuing on a  
4 much lower rate in A-bomb survivors. The relative  
5 risk for solid cancers is slowly decreasing as the  
6 population ages.

7 The BEIR Committee was, I think, very -- I  
8 must say I'm not a member of the BEIR Committee. I  
9 was the Academy study director on this, but I was  
10 impressed by the way the Committee looked after the  
11 uncertainties of the risk estimates. They aren't  
12 coming out with just magic numbers, point to risk  
13 estimates and say, "Well, this many people are going  
14 to die per rem." They looked at the whole uncertainty  
15 and the data they were using and the models they were  
16 using and got a pretty good picture of what the  
17 distribution of risks were for a given dose.

18 (Slide) All this is shown in the next  
19 slide. This is the histogram for solid cancers. This  
20 is for acute dose of ten rem to the whole populations,  
21 males on top, females on the bottom. The 90 percent  
22 confidence interval is about a factor of 2 on each  
23 side of the mean, which is 800 cases, early deaths or  
24 excess deaths per million person rem. So, the  
25 Committee makes no claim that their risk estimates are

1 exact, but they do claim that they have looked at the  
2 sources of error in the risk estimates, including the  
3 errors that are inherent in the assumptions they made.

4 (Slide) Cancer risks show a pretty strong  
5 effect of age of exposure, as shown in the next slide.  
6 This is for females. You can see the risks are  
7 particularly high for females under 30 years of age.  
8 Except for lung cancer, they've pretty much decreased  
9 by mid-life, for exposures occurring in mid-life and  
10 later. Respiratory cancer is a late-occurring cancer.  
11 Now, the curve for men looks about the same as this  
12 except digestive cancers are half as large,  
13 respiratory cancer is twice as large.

14 Notice that breast cancer does not seem to  
15 be too important with the latest data where it used to  
16 be rather dominant for female cancer risk before.

17 (Slide) Looking at the area under these  
18 curves, total risk is a function of age of exposure,  
19 as shown on the next slide. This is age of exposure,  
20 5, 15, 25, 35. Forty-five is the same as 35  
21 essentially. You can see that for children, the risks  
22 seem to be about three times higher than for adults,  
23 but there's still a pretty good risk up until age 30  
24 and then it starts to fall off. I think this has some  
25 implications for how you consider exposures for

1 occupational purposes.

2 I think if the models are held constant, a  
3 linear response, a relative risk model, that's  
4 assumptions made by the BEIR V Committee on, I think,  
5 rather strong evidence, risk estimates haven't changed  
6 too much over the years. They've been fairly stable,  
7 as shown in the next slide.

8 (Slide) BEIR I model was 690 cancers per  
9 million person rem. BEIR III was 500. BEIR V, it's  
10 up to about 790. This is the average for males and  
11 females. There was a difference in the way of  
12 accounting for deaths in BEIR V that makes comparisons  
13 a little bit hard. BEIR V looked at the excess  
14 deaths, BEIR Committees I and III look at early  
15 deaths, people that would have died of cancer in all  
16 probability later, but they died early due to cancer.  
17 That increased the risk estimates by about 25 percent.

18 (Slide) Finally, I'd like to look at the  
19 last slide where I show the range of risk estimates by  
20 various BIER Committees in the sense they're becoming  
21 more precise. BEIR I and BEIR III had no idea of what  
22 the uncertainty was in their risk estimates. They  
23 just provided different models. As you can see for  
24 BEIR III, risks per million person rem varied anywhere  
25 from 10 cancers to 500 cancers. Now, I maintain

1 that's a range so wide that it isn't really very  
2 useful for setting radiation protection standards.

3 BEIR V, I think, had a lot more data to  
4 work with. Perhaps the data was better analyzed.  
5 Error intervals are about a factor of two on either  
6 side of the point estimate of 800 cases. But I think  
7 that gives us an idea of what kind of uncertainty  
8 we're dealing with when we're talking about radiation  
9 risks from acute exposures.

10 Something that I think everyone has to  
11 bear in mind is that these risks are the risks for  
12 acute exposures, risks that are thought to be lower at  
13 low dose rates. How much lower is something that the  
14 Committee decided they could not specify except to say  
15 perhaps it was as much as a factor of two or more.  
16 There is very little scientific information on what  
17 number you should pick for a dose rate effectiveness  
18 factor, how much you should reduce the risk at low  
19 doses and low dose rates. My own view is this varies  
20 with different cancers and there is no magic number.

21 COMMISSIONER ROGERS: What was your point  
22 for defining acute? That was greater than what  
23 amount?

24 DOCTOR ELLETT: Why did they say that it  
25 was --

1 COMMISSIONER ROGERS: Well, how did you  
2 define acute? What was your definition of acute?

3 DOCTOR ELLETT: Oh. Really, I think if  
4 you do it operationally it's in terms of the exposures  
5 that were received by the Japanese which are really  
6 very high dose rate exposures over a --

7 COMMISSIONER ROGERS: Well, I mean in  
8 terms of numbers, rem or something of this sort.

9 DOCTOR ELLETT: Well, acute would mean it  
10 occurred over a very -- this is, I think, an important  
11 distinction this Committee made. They maintained  
12 doses, no matter how small, even a rad at very high  
13 dose rates, would be an acute exposure. A small dose  
14 isn't necessarily going to be less damaging. If a  
15 dose is distributed over time, and they used the  
16 example of a year, then the effects become less.

17 COMMISSIONER ROGERS: What is a small  
18 time? How small is small?

19 DOCTOR ELLETT: I'll go to my radiation  
20 pathology expert for that.

21 COMMISSIONER ROGERS: I mean are we  
22 talking about --

23 DOCTOR ELLETT: I would guess --

24 COMMISSIONER ROGERS: -- seconds, micro  
25 seconds, weeks, days --

1 DOCTOR ELLETT: Days.

2 COMMISSIONER ROGERS: -- months? I mean  
3 what's small here?

4 DOCTOR UPTON: I don't think the Committee  
5 really got into this discussion. Perhaps it should  
6 have. I think from my point of view one would want to  
7 approach this in terms of microdosimetry, energy  
8 deposition per sensitive target per unit time. And if  
9 one does this, I think one has different time scales  
10 for different kinds of radiation probably, different  
11 distributions.

12 I think you're leading into a set of  
13 questions which the BEIR V Committee really didn't  
14 address systematically and I think it brings out the  
15 importance of the ICRP and NCRP which must confront  
16 these issues of dose rate effects, spacial and  
17 temporal distribution of dose critically if they're  
18 going to come up with recommendations that are useful  
19 and practical spheres.

20 COMMISSIONER ROGERS: Well, just to get  
21 some feeling about where you begin to move from one  
22 regime into the other. When you're talking about  
23 acute, what's not acute? If you're talking about  
24 short time, what is a short time versus a long time  
25 and just get a layman's grasp of what we're talking

1 about in terms of scales here.

2 DOCTOR UPTON: Commissioner Rogers, as I  
3 say, I don't think the Committee wrestled with this  
4 issue systematically and I would venture to say that  
5 different radiobiologists would provide different  
6 answers. I'm not sure how helpful I can be.

7 COMMISSIONER ROGERS: Okay.

8 CHAIRMAN CARR: That completes your  
9 presentation?

10 DOCTOR ELLETT: Thank you.

11 COMMISSIONER REMICK: Just a question on  
12 administrative effectiveness factor. You mentioned--  
13 I don't know if you said recommended two or more. In  
14 the report itself I see figures like 2 to 10. Are  
15 those the same or should I --

16 DOCTOR ELLETT: They made no  
17 recommendation on this on purpose because they did not  
18 think there was a scientific basis for picking a  
19 particular number. I think the executive summary has  
20 words to perhaps as much as a factor of two or more.  
21 Later in the report, I believe it says between two and  
22 ten.

23 COMMISSIONER REMICK: Okay.

24 DOCTOR UPTON: Coming back to the issue of  
25 acute versus less acute, sub-acute, chronic, it may

1 depend on the cell which you're thinking about or the  
2 target organ, the rate at which lesions can be  
3 repaired. I think you're putting your finger on a  
4 very important point that I think needs more attention  
5 systematically in research. I don't think we've  
6 really addressed this critically yet.

7 DOCTOR ELLETT: I think the Committee was  
8 a little bit surprised when they looked at breast  
9 cancer. They thought they would see a dose rate  
10 effect because the Japanese received the dose in  
11 milliseconds to a second or so. People that had  
12 received radiation in their tuberculosis treatment,  
13 small doses over a long period of time, the difference  
14 in cancer risk in these two groups was insignificant.  
15 There was no dose rate effect and this makes people  
16 pause a bit. You can argue for various reasons why  
17 this would be so, but I think on animal data you would  
18 certainly have expected a larger dose rate effect.

19 CHAIRMAN CARR: Okay. Let's proceed.

20 DOCTOR SINCLAIR: Thank you, gentlemen.

21 (Slide) Could I have the first slide,  
22 please?

23 Gentlemen, I'm going to be talking about  
24 estimates of cancer risk and detriment as the basis of  
25 ICRP and NCRP recommendations. Inevitably, I'll be



1 covering some points that my colleagues from the BEIR  
2 Committee have already made to you, but I expect to do  
3 it from a different perspective, that of someone who's  
4 looking toward the recommendations that ICRP and NCRP  
5 will have to make, which Charlie Meinhold is going to  
6 talk about after me.

7 (Slide) First of all, what are the--  
8 next slide, please.

9 What are the concerns in low dose  
10 radiation protection? Well, they're primarily  
11 stochastic effects. No threshold is assumed, the  
12 magnitude of the effect is the same at all doses, the  
13 frequency is proportional to dose at low doses. Those  
14 are our assumptions. The principal effects under that  
15 category are hereditary effects and the induction of  
16 cancer. Hereditary effects, as we've just heard, have  
17 not changed recently, so we're going to concentrate on  
18 the induction of cancer.

19 There are two special problems that should  
20 be mentioned, but I'll not deal with them here and  
21 that's the risk of mental retardation in the fetus,  
22 which Doctor Upton has discussed, and deterministic  
23 effects. Direct effects and damage to tissue are not  
24 an issue in low dose radiation protection, broadly  
25 speaking that is, because the doses we expect we're

1 dealing with are below the thresholds for those  
2 effects. They would occur in accidents, but not  
3 elsewhere.

4 CHAIRMAN CARR: Let me ask you, are your  
5 low doses the same as the BEIR V's low doses? I  
6 understood BEIR V's low doses were down to ten rad and  
7 they didn't really get into lower than that. Is that  
8 where your cutoff is or do you start --

9 DOCTOR SINCLAIR: Well, no. We  
10 consider -- in assuming no threshold, we consider that  
11 our low doses are down in the region of one rad or  
12 less. The exposures that people might get  
13 occupationally in particular and some exposure --

14 CHAIRMAN CARR: I'm trying to figure out  
15 there where you say deterministic effects are not a  
16 concern because the limits are below thresholds, I  
17 don't know what limits we're talking about.

18 DOCTOR SINCLAIR: Well, the limits for  
19 occupational presently at 5 rems a year and even for  
20 the public at 100 millirems a year for manmade sources  
21 are way below the 50 to 200 rad threshold levels of  
22 effects like cataract --

23 CHAIRMAN CARR: Okay.

24 DOCTOR SINCLAIR: -- and so on. So,  
25 they're not our concern. They would be in an

1 accident, but they're not in low dose.

2 (Slide) Now, there are two points I'd  
3 like to make before we go to some of the numbers.  
4 First of all, on the next slide, we can see the  
5 exposed populations for risk estimation. The Japanese  
6 survivors come first and they always head the list.  
7 But I wanted to make the point that we have lots of  
8 information from other sources as well, mostly  
9 medical, and some of the studies are quite important  
10 and back up what we believe we know from the Japanese  
11 survivors. There's quite a list of them there, as you  
12 can see.

13 (Slide) The next slide, please.

14 This demonstrates another point that I  
15 think one needs to bear in mind in considering the  
16 induction of cancer by radiation. If a population  
17 were irradiated at time zero on that graph, we  
18 wouldn't see anything, wouldn't see any leukemias for  
19 two years because there's a latent period at least  
20 that long. But then it rises rather rapidly for  
21 leukemia, as you can see, to a peak at about 5 or 7  
22 years and then falls off almost to zero -- it  
23 shouldn't go quite to zero on that thing -- after  
24 about 30 years.

25 Solid tumors, on the other hand, have a

1 latent period of about ten years and then they rise  
2 more or less proportionally to the way spontaneous  
3 cancers occur in the population as we age.

4 CHAIRMAN CARR: Now, this is incidents?

5 DOCTOR SINCLAIR: It would be incidents.

6 CHAIRMAN CARR: And you were dealing in  
7 deaths, Doctor Upton?

8 DOCTOR UPTON: Primarily, yes.

9 DOCTOR SINCLAIR: Shape wouldn't be very  
10 much the same, wouldn't be much different if it were  
11 not. The leukemia, for example, incidents and  
12 mortality are essentially the same.

13 CHAIRMAN CARR: Okay.

14 DOCTOR SINCLAIR: But some of the other  
15 cancer is not. An important point to note, beyond 40  
16 years we don't yet know precisely how that curve will  
17 go. There's some evidence that it's beginning to drop  
18 off, but we just don't know as yet because the  
19 Japanese population which we follow the most closely  
20 hasn't reached there. Furthermore, the Japanese  
21 population, as we know, is at this point in time  
22 somewhat less than 40 percent of them have died. So  
23 we have to project our lifetime risks to the others  
24 and that's where these projections by relative risk  
25 and multiplicative models come in.

1 (Slide) Next slide, please.

2 Back in 1977, the ICRP and not only the  
3 ICRP but UNSCEAR and the preferred model at the time  
4 for the BEIR Committee and NCRP all considered that  
5 the risk from induced cancer was about  $10^{-2}$ . That's  
6 one percent per sievert and that was an average value  
7 of one and a half for females and one for males and  
8 then rounded off and that's the number we used for a  
9 long time for radiation protection.

10 (Slide) What's happened since then? On  
11 the next slide we see that the epidemiological  
12 information since 1977, of course, includes an  
13 extensive update in the Japanese A-bomb survivors, but  
14 update in all the other clinical studies too which are  
15 still being followed, particularly the ankylosing  
16 spondylitics in the United Kingdom, an important  
17 source of information; the International Cervix  
18 Series, which has become available since 1977; and  
19 other clinical updates in particular organs such as  
20 the breast and the thyroid.

21 (Slide) The next one, please.

22 The Japanese survivors, as we've seen, are  
23 the most important for risk estimation. They provide  
24 the largest sample and over the broadest dose range.  
25 So, we have to concentrate on them and we have three

1 new cycles of Japanese data since the 1977  
2 evaluations. The increase in the solid tumor database  
3 is from not much more than 100 to a little bit less  
4 than 300 between a factor of two and three. That's a  
5 big increase in --

6 CHAIRMAN CARR: Per what?

7 DOCTOR SINCLAIR: Well, these are the  
8 excess tumors.

9 CHAIRMAN CARR: Per --

10 DOCTOR SINCLAIR: Not per anything, the  
11 absolute number of excess tumors at this point in  
12 time.

13 CHAIRMAN CARR: That's got to be over more  
14 than one person.

15 DOCTOR SINCLAIR: Well, at this point in  
16 time, I think something like in the Japanese it would  
17 have been between 5 and 6,000 cancer deaths in this  
18 population. About 300 of those are attributable to  
19 solid tumor excess exposure, all exposures.

20 There's more information on the age  
21 dependence because even the youngest age groups are  
22 now beginning to reach the age when they are more  
23 likely to get cancer. There's more information on the  
24 time course, of course, because we have 11 more years  
25 and we've heard about the revisions in the survivor

1 dosimetry. We believe the DS86 system is a much more  
2 sophisticated one than its predecessor and it has  
3 increased the risks by -- of the order of one and a  
4 half to two times, depending which organ you're  
5 talking about.

6 (Slide) Amongst the evaluations of risk,  
7 the next one, please, the Radiation Effects Research  
8 Foundation do their own evaluations and they did this  
9 in 1988 and came to the conclusion that the total risk  
10 in a population of all ages for high dose rate  
11 exposure was about 12 percent per sievert, the third  
12 number down there, 11 percent of its solid tumors, one  
13 percent leukemia. If one were to divide by a dose  
14 rate effectiveness factor and they didn't opine on  
15 that, they simply said other people have used about  
16 two and a half, so if we did use that then we'd get  
17 about five percent per sievert and if we'd applied  
18 this to an adult population without the younger, more  
19 sensitive people in it, we would have got three  
20 percent per sievert. That's, of course, substantially  
21 higher than our normal risk up till that time.

22 (Slide) The next one, please.

23 And the UNSCEAR Committee looked at all  
24 the human sources of exposure that I listed in the  
25 second slide, decided that the Japanese sample was so

1 much superior to the others that they would  
2 concentrate on it for their number generating, but  
3 they would use, for example, the ankylosing  
4 spondylitics and the International Cervix Series to  
5 support that information.

6 They used two projection models to get to  
7 this lifetime risk, the additive model not favored so  
8 much today and the multiplicative model, which is, and  
9 obtained about 11 percent per sievert as the number  
10 for high dose, high dose rate in a population of all  
11 ages. For a working population, the same number is  
12 eight percent per sievert. And for low dose, low dose  
13 rate, they didn't actually do an evaluation of the  
14 situation, they simply quoted others and mainly the  
15 NCRP, as a matter of fact, and said, "We should divide  
16 by something. We think it's between two and ten  
17 because that range has been used by others."

18 CHAIRMAN CARR: Before you leave that one,  
19 let me get the high dose rate and low dose -- I mean  
20 high dose and low dose separated. I understand the  
21 rate at the bomb was instantaneously nearly. What's  
22 the high dose there you're talking about?

23 DOCTOR SINCLAIR: Well, you're talking  
24 about if it's 400 rad, 400 rad point in  
25 Hiroshima/Nagasaki was delivered in a second or less.



1 CHAIRMAN CARR: Yes. And the low dose --

2 DOCTOR SINCLAIR: Lose dose, lose dose  
3 rate we would consider to be the low doses that we get  
4 in occupational and otherwise, which are of the order  
5 of fractions of a rem per year usually.

6 CHAIRMAN CARR: So you're talking millirad  
7 there, huh?

8 DOCTOR SINCLAIR: Yes.

9 CHAIRMAN CARR: Okay.

10 DOCTOR SINCLAIR: Five rems a year is the  
11 limit. We'd still consider that a low dose rate.

12 (Slide) The next one, please.

13 Then we have the BEIR Committee gave us  
14 three particular numbers in the top range there which  
15 we reinterpret to compare with UNSCEAR and one can't  
16 do this exactly for some of the reasons that Doctors  
17 Upton and Ellett have already mentioned, but it comes  
18 out at about 9 percent per sievert for a population of  
19 all ages and it would have been less than that, about  
20 seven percent per sievert for the working population.  
21 The BEIR Committee did look at the question of dose  
22 rate effectiveness factors, decided they couldn't come  
23 up with a number, again for reasons which have been  
24 mentioned, and their advice was two or more.

25 Well, this is a rather important number

1 and it's important in radiation protection because we  
2 do need nominal values throughout. The advice that  
3 UNSCEAR and BEIR have offered us isn't sufficient for  
4 us to do that. A decision has to be made by  
5 protection people.

6 (Slide) On the next slide, I've tried to  
7 show you what it is we're talking about in this dose  
8 rate effectiveness factor. The top line, if we have  
9 data from high dose rate sources, it would be up in  
10 the top region of that top curve and we'd draw a  
11 straight line down to zero and delta would be the risk  
12 coefficient for that high dose, high dose rate  
13 circumstance.

14 As we get information at lower and lower  
15 doses and particularly from the laboratory in animals  
16 and cell studies, we see that the shape of the curve  
17 is not a simple linear. It's most often linear  
18 quadratic, although it differs in different biological  
19 systems. So, we believe that the linear quadratic  
20 curve there, which ultimately gives you this slope  
21 alpha, would represent the situation better. Then the  
22 dose rate effectiveness factor is the ratio between  
23 delta and alpha, high dose rate information over the  
24 expected low dose rate information.

25 As I say, we get quite a bit of that

1 information from the laboratory and that's the primary  
2 source of information there. One of the major  
3 evaluations of that was done by the NCRP in 1980.

4 (Slide) The next slide, please.

5 The next slide lists what people have  
6 actually used, what other organizations have actually  
7 used in the past for this dose rate effectiveness  
8 factor, starting with UNSCEAR in 1977 used two and a  
9 half. The BIER Committee, in using a linear quadratic  
10 effectively in 1980, the BEIR III Committee was 2.25.  
11 NCRP did this comprehensive evaluation in 1980 and  
12 came to the conclusion that the range covered by  
13 animal and cellular data was between 2 and 10 and that  
14 the 2s were really different from the 10s and depended  
15 more on the system, so that simple averaging was  
16 really not advisable.

17 Now, there's one point about that. The  
18 animal data and the cellular data normally cover a  
19 substantially broader dose range than the human  
20 information. Since the dose rate effectiveness factor  
21 itself depends on dose, one might expect to get  
22 somewhat higher values in these animal systems than in  
23 human ones. That indeed turns out to be the case  
24 because our human experience, if you look down a bit  
25 on the slide, for breast and thyroid in studies made

1 about a decade or so ago, there was no effect of  
2 fractionation in those studies whatsoever. Now, they  
3 were not terribly precise studies because they can't  
4 be in humans, but one could not have assumed from that  
5 a dose rate effectiveness factor of more than one.

6 More recent studies, one on the breast  
7 dose rate, does indicate possibly a factor of three,  
8 and studies with  $I^{131}$  and comparing it with external  
9 radiation, which may not be entirely due to dose rate  
10 but that would be a factor in it, have shown about  
11 four. But one has to see that the human experience is  
12 down near that end.

13 Then for Hiroshima/Nagasaki itself, as  
14 you've already heard, the leukemia fits quite well to  
15 a dose rate effectiveness factor which would come down  
16 at about two. The best fit for the solid tumors is  
17 still linear, which would give us about one. Now,  
18 both of those, of course, are based on data which is  
19 statistically not very precise. So you could stretch  
20 the solid tumors to two and the leukemias to a bit  
21 more than two.

22 (Slide) Considering all that -- could I  
23 have the next slide, please? Considering the fact  
24 that in the animal data they tend to be a broader dose  
25 range which might give you higher values, considering

1 that the human data don't give you much -- although  
2 not very precise -- don't give you much more than two.

3 The ICRP decided that they would use 2,  
4 and this is their estimate ~~of-their-estimate~~ of the  
5 risks for low dose, low dose rate. They start with--  
6 by averaging UNSCEAR and BEIR, although that also  
7 could be done a number of ways, and say that the high  
8 dose, high dose rate for a population of all ages the  
9 risk is about 10 percent per sievert, and for a  
10 working population it's about 8 percent per sievert.

11 And I think it's worth mentioning that  
12 probably the working population number is a little  
13 better known than the total population, simply because  
14 included in the total population are the younger age  
15 groups. They have to be projected the most and  
16 they're the least certain. Then ICRP decided that the  
17 dose rate effectiveness factor they would use was 2.  
18 And therefore, for a population of all ages 5 percent  
19 per sievert, for the working population 4 percent per  
20 sievert.

21 (Slide) But that isn't all that you need  
22 for a working protection system. ICRP had to go  
23 further and examine the details of the various  
24 different organs, which is shown on the next slide.  
25 They determined these by comparing transfer models

1 from the Japanese population to various others,  
2 including the United States, the United Kingdom,  
3 Puerto Rico, and China, and averaged the results of  
4 their determinations in order to get what they  
5 consider to be best numbers for the organ risks. They  
6 add up in the right-hand column there to 510,000,  
7 which is the 5 percent per sievert for the whole  
8 population of all ages. You'll notice most of them  
9 are higher. Bone marrow is higher. Lung is higher.  
10 But not all of them. Bone surfaces are about the  
11 same. Breast is about the same. Thyroid is about the  
12 same. So in dealing with particular organs one needs  
13 to look specifically at what the best values seem to  
14 be.

15 Well, in order to get a weighting system  
16 out of that, ICRP realizes of course that these organ  
17 risks are not very precise, not as precise or not as  
18 well known as the total risk.

19 (Slide) Could I have the next slide,  
20 please?

21 So for weighting factors, they decided to  
22 form only four groups of weights and these are grouped  
23 in .01 for the bone surfaces and the skin, .05 for the  
24 bladder, breast, liver, esophagus, thyroid and all  
25 remainder tissues, and .12 for bone marrow, colon,

1 lung and stomach, and .20 for the gonads. Now you  
2 have to take the total detriment into account in  
3 deriving that, and so far I've talked only about the  
4 fatal cancer risk.

5 (Slide) If we go over to the last slide,  
6 we see that the ICRP's estimate of the detriment  
7 includes 1 percent for serious hereditary disease in a  
8 population of all ages, 5 percent for cancer  
9 mortality, as we've seen, 1.5 percent for cancer  
10 morbidity by using a formulation which seems to have  
11 been fairly acceptable to make an estimate of the  
12 measure of detriment that results from a cancer that  
13 is cured but was caused by radiation for a total of  
14 7.5 percent for the detriment for a population of all  
15 ages. For the working population the corresponding  
16 numbers, all a bit less, add up to about 5.8 percent.

17 But these numbers, of course, are some  
18 three or four times higher than the nominal detriment  
19 of 1.65 percent that ICRP used in 1977. There are  
20 some different things in them of course, in addition  
21 to the increase in cancer risk. But they did behoove  
22 both protection bodies, both ICRP and NCRP, to  
23 consider what should be done about this with respect  
24 to recommendations, and I'll leave that to Mr.  
25 Meinhold to talk about.

1 COMMISSIONER ROGERS: Before you leave  
2 that graph, do you really believe these numbers to  
3 something like a tenth of a percent?

4 DOCTOR SINCLAIR: Well, I'm tempted to say  
5 of course not, but the formulation that one uses in  
6 the cancer morbidity, for example, is one that simply  
7 gives you that sort of number. It's not easy to round  
8 it down to 1 percent or up to 2.

9 COMMISSIONER ROGERS: Well, it just sort  
10 of looks like it's a mix of numbers that are known to  
11 different -- or believed to different extents.

12 DOCTOR SINCLAIR: Well, I think that's the  
13 one that is the weakest from that point of view,  
14 because it appears to be the most precise. And in  
15 fact, of course, it's based entirely on the way in  
16 which you judge the -- the risk of death, actually,  
17 from the given cancer is the deciding factor and it  
18 varies a great deal of course between leukemia with a  
19 lethality ratio of about .99, the thyroid with a  
20 lethality of about .10, and the skin with a lethality  
21 of less than 1 percent. And we try to correct for  
22 that, but it appears more precise than it is.

23 CHAIRMAN CARR: Thank you.

24 Doctor Meinhold?

25 MR. MEINHOLD: (Slide) Could I have the



1 first slide, please?

2 I'd like to begin by complimenting the  
3 Commission on adoption of revised Part 20. I think  
4 that will do a great deal and I'm very happy to see  
5 that and I don't want anything that I'll be saying  
6 about what --

7 CHAIRMAN CARR: You've seen it?

8 MR. MEINHOLD: Yes, I have.

9 CHAIRMAN CARR: Oh, okay.

10 MR. MEINHOLD: I don't want to say  
11 anything that -- anything we're going to be saying  
12 here to suggest that we would disagree with that  
13 decision at this time, because it's -- I think it's an  
14 excellent approach.

15 In view of the fact that the time is  
16 short, I won't be talking about the second through the  
17 sixth slide, which really were the basis for the  
18 ICRP's 1977 recommendations. You know all of those,  
19 because that's basically what went into your revision  
20 of Part 20 and also into the Presidential guidance  
21 that EPA put out in 1987. So I'm really going to use  
22 that as a base time. The 1977 ICRP publication is the  
23 one that you've been dealing with recently in terms of  
24 your new guidance, but we're asking, then, what's  
25 happened since then, what's going on.

1                   (Slide) I've listed in the next slide,  
2 please, the four major issues that we face in terms of  
3 radiation protection standards and whether or not we  
4 need to change our basic recommendations. And the  
5 first of those is that industry is becoming safer, and  
6 is you recall we used safe industry as our criteria.  
7 And of course, you've heard a lot about the new  
8 Japanese survivor data. You've heard something about  
9 the projection models and the change of emphasis that  
10 ICRP and NCRP would use. And of course, you've heard  
11 something of the fetal risk concern.

12                   (Slide) The most important of these, if  
13 we could go to the next slide, please, the most  
14 important perhaps that the public saw even before we  
15 got the Japanese data is this question of accidents  
16 frequencies going down in the safe industries, and  
17 I've just given you a rough-cut from the National  
18 Safety Council kind of data showing you that all of  
19 these are decreasing at a rate of about 2 percent, 2  
20 or 3 percent per year.

21                   (Slide) And of course this curve, if we  
22 go to the next slide as well, this distribution merely  
23 shows you two things, that they're all going down in  
24 terms of their likelihood of death in those industries  
25 and secondly that the safe industries are still

1 roughly in the range of  $1 \times 10^{-4}$ , but with a trend down.

2 CHAIRMAN CARR: Those numbers are per  
3 million people?

4 MR. MEINHOLD: Yes.

5 CHAIRMAN CARR: Per year?

6 MR. MEINHOLD: Right. So if you look at  
7 the first one would have been -- for instance, service  
8 would have been  $1.14 \times 10^{-4}$ , which is the kind of number  
9 you're probably used to hearing in terms of the fatal  
10 accidents.

11 (Slide) If we could go to the next slide,  
12 the NCRP issued Report 91 in 1987 in which -- and if  
13 we could go to the next slide, please -- in which the  
14 NCRP was beginning to be aware, of course, of changes  
15 but felt that it was time for us to revise our  
16 recommendations. And in that one, we did adopt for a  
17 nominal risk, the risk of  $10^{-4}$  per rem, which was the  
18 1977 ICRP and UNSCEAR evaluation. We also noted that  
19 the annual dose equivalent to monitored workers was in  
20 the order of 230 millirem, and so we felt that it was  
21 still reasonable to stay at that time with 5 rem per  
22 year or 50 millisieverts.

23 (Slide) The next slide I think indicates  
24 some of the concerns that we have, and that of course  
25 was that the risk estimates were likely to increase.

1 We knew that the new data was in Japan. We just  
2 couldn't get a quantitative handle on the numbers.

3 (Slide) And we also knew about the safe  
4 industries, if I could go to the next slide, please.  
5 So we took three steps, some of which you've taken in  
6 your revision to Part 20, of course, discontinuing the  
7 age minus 18 recommendation times 5. But secondly,  
8 and we think this is terribly important and I believe  
9 you've stressed this as well in your division, the  
10 upper boundary nature of the dose limit, that it's  
11 simply an inappropriate quantity to use for design  
12 criteria, that sort of thing, that it's a boundary  
13 condition to whatever approach you take to your design  
14 and that it's really trying to point out that the dose  
15 limit itself is the edge. It isn't a desire or a  
16 goal.

17 (Slide) And then the next slide is one in  
18 which we probably could have made our job a lot easier  
19 today. In fact, I believe that if in 1977 we had made  
20 this discussion about keeping individuals below their  
21 dose limit, below their age in terms of tens of  
22 millisievert or in rem, if we had made this  
23 recommendation at that time to apply to the individual  
24 we probably would be in -- wouldn't have to revise in  
25 '91 at this time because it probably takes care of the

1       problem.

2               The committee I of NCRP is reviewing this  
3       data right now to see what we should do about it, and  
4       I can't even tell you any more than that. But at this  
5       time we really thought we'd like to do it, but we got  
6       some static from our own counsel in terms of whether  
7       it was necessary at that time.

8               But we're saying here that one of the ways  
9       in which you can assure that the lifetime risk to the  
10      individual is controlled is to put essentially a limit  
11      on him that is -- in effect assures a lifetime limit  
12      of something in the order of 65 or 70 rem. We felt  
13      this was preferable to a set limit of 100 rem, which  
14      would mean that at some age he gets to 100 rem and  
15      he'd be out of work. This way, God willing, he's  
16      always going to have next year when he gets a year  
17      older and he would be available -- he would have,  
18      then, another rem available in terms of his working  
19      lifetime limit. But we did not make that a specific  
20      limit on the worker but only in terms of how an  
21      operation should be run. I suspect that we'll move  
22      perhaps strongly in that regard.

23              With regard to protection of the embryo  
24      and fetus, in 1987 when we wrote -- when this document  
25      was written there was a new concern about the fetal

1 risk in terms of the brain, mental retardation problem,  
2 and in fact the suggestion that it might have been a  
3 threshold effect, a deterministic threshold effect.

4 (Slide) And as a result, if we could go  
5 to the next slide, we did recommend two things, one of  
6 which was that the total to the fetus should be no  
7 more than 500 millirem. But a more important one and  
8 perhaps more restrictive was that after diagnosis it  
9 should be limited in terms of 50 millirem in any one  
10 month.

11 (Slide) The next slide -- and actually  
12 the next two -- but the next slide shows -- what I'd  
13 like to point out are some, if you like, secondary  
14 considerations that were given in NCRP '91. But I  
15 think it's terribly important that the NCRP considers  
16 that we ought to be looking at this case of over-  
17 exposure not in terms of a worker and his health risk  
18 but in terms of the operation and the way in which it  
19 resulted in his over-exposure.

20 So we're really saying that an exposure in  
21 excess of the limit probably doesn't affect his  
22 lifetime risk at all. If he gets 6 rem in one year,  
23 it's certainly going to be offset by the less than 6  
24 rem he gets in other years. But more importantly it  
25 suggests an inadequate system of protection and that

1 his return to work should be almost entirely based on  
2 improved control of the work place, unless the  
3 exposures are accidental and very serious.

4 (Slide) The next slide also is a--  
5 constitutes one of the sections in NCRP '91, which I  
6 think we need to give special consideration to as we  
7 think about lowering dose limits. In 1987 we said  
8 that we based our 5 rem on safe industries and we said  
9 that that's normally easily obtainable. That's one of  
10 the value judgments that's made is if we have safe  
11 industries we ought to be able to accomplish that.  
12 And we saw even then that it wasn't possible with  
13 space flights, and in fact NCRP has done some work for  
14 NASA to develop different limits for astronauts  
15 primarily on this basis.

16 And so we're saying that you could have  
17 new limits based on informed consent of the workers  
18 and a demonstrated need. There's nothing magic about  
19 this as long as we say that safe industry is our  
20 basis, and so in fact if you don't want to have a safe  
21 industry criteria you could have something else which  
22 you might have for a given situation. In fact, under  
23 some conditions it might be reactor maintenance  
24 workers that can't live by the dose limits and some  
25 special requirements would be required, again focusing

1 on the lifetime risk when you do this.

2 I'd like to shift now to the ICRP 1990  
3 draft, which Warren alluded to in his discussion of  
4 the risks. And I ought to say that it's very  
5 important whenever we consider radiation protection  
6 recommendations that we begin as we did in this  
7 discussion with the biology. You have to know what we  
8 know about the risk before we can begin.

9 But in looking at this, in 1977 the ICRP  
10 said that they were going to do this comparison with  
11 safe industries. They get concerned about two aspects  
12 of this. One of them was that, particularly for the  
13 ICRP, they're not uniform worldwide. And secondly,  
14 they aren't uniform in time, as we've just seen. And  
15 they also were concerned about the fact that the  
16 mortality data applies to the average, that is the  
17 risk number, whereas the dose limit applies to the  
18 individual.

19 Now there is a little bit of a circular  
20 reasoning on that, of course, because in that safe  
21 industry there are also individuals who are at the  
22 high end of the risk. But the fact is we've set our  
23 limit in publication 26 based on what we expect the  
24 average to be, and in fact the Commission is concerned  
25 about that.



1 (Slide) So if we look at the next slide,  
2 we see that in 1977 the limit was judged against the  
3 average, but that in 1990 we also want to look at the  
4 maximum. That is, what is the -- what would the  
5 worker -- what would his reaction be to the range?

6 Obviously, if the average is going to be  
7 safe industry there will be an average, an individual  
8 who will be, say, at some factor above that, perhaps 5  
9 or so. And when the Commission looked at this they  
10 looked primarily at studies done in the United Kingdom  
11 in which they looked at perhaps some different words.

12 The English language has a strange set of  
13 words with tolerable, intolerable, acceptable and  
14 unacceptable. They don't work well. And so we really  
15 see that unacceptable is probably right at the edge of  
16 the unsafe industries, that is the deep sea divers,  
17 the deep sea fishermen, and some of the farming  
18 industries. Tolerable is probably closer to what we  
19 called acceptable in the past, which is in the order  
20 of  $10^{-4}$  because of course even in the  $10^{-4}$  industry  
21 we're working all the time to reduce the accidents and  
22 to improve safety. So it's clearly not acceptable,  
23 but tolerable in the sense that people will go to work  
24 without -- yes sir?

25 COMMISSIONER REMICK: Question. Is that

1 10<sup>-4</sup> per year?

2 MR. MEINHOLD: Yes.

3 COMMISSIONER REMICK: Okay.

4 MR. MEINHOLD: Yes, it is. And acceptable  
5 is probably a factor of ten below that, 10<sup>-5</sup>, when  
6 people don't even think about whether or not there's  
7 any need to improve it.

8 CHAIRMAN CARR: Did you also look at non-  
9 occupational risks?

10 MR. MEINHOLD: Yes. Yes. And of course,  
11 the driving back and forth to work is one of the  
12 controlling risks. It's about 10<sup>-4</sup> by itself.

13 (Slide) If I could go to the next slide,  
14 one of the things that comes out of the new risk  
15 projection models is that it has to change a little  
16 bit how we look at the basis for our limit. If you  
17 look at the lifetime risk, we see that using the  
18 additive model it would -- and again, I take -- I  
19 agree that we've got too many significant figures--  
20 but if we look at the 5.66 against the 8.56, we see  
21 that, yes, the multiplicative model has increased our  
22 expectation of lifetime -- of a probability of cancer.

23 When we come down to the third -- to the  
24 second line, I'm sorry, we see that the loss of  
25 lifetime goes from 20 years under the additive model

1 to 13 years under the multiplicative model. And of  
2 course, it's because the relative model essentially  
3 says you'll get the cancer at the same time you'd get  
4 it naturally, which would be at older ages.

5 And so if you look at the next two, you  
6 see that loss of life expectancy, the difference  
7 between the 1.12 and the 20 of course is that that is  
8 the average for everybody that gets exposed. The 20  
9 is for those people that die from the exposure. So  
10 there's a -- the 20 years is loss of lifetime if you  
11 die from the attributable cancer. So it's an  
12 important thing to remember that maybe we can't look  
13 just at the first line, but we have to look at some of  
14 these other considerations since it's a multifaceted  
15 problem.

16 (Slide) So we look at the next slide, and  
17 what the Commission did, the International Commission,  
18 was look at these in terms of some suggested or trial  
19 limits. And among other things -- this isn't the only  
20 data that was used, because of course we used the  
21 morbidity data and some other considerations -- but  
22 primarily they said let's look at what we get for  
23 those different values and compare those with what we  
24 had in 1977, compare them with safe industries and all  
25 the rest of it.

1                   And so you see that there's a distribution  
2 there. Obviously the loss of lifetime isn't going to  
3 be very sensitive because that's going to happen when  
4 you get the cancer, and the same is true of the  
5 attributable age. But the loss of life expectancy and  
6 the lifetime risk, of course, are going to change.

7                   Well, given those, the Commission said,  
8 well, our best judgement in terms of the value that  
9 takes into account all of these attributes is given in  
10 the next slide.

11                  CHAIRMAN CARR: Let me look, before you go  
12 on to that one. I'm not sure I understand. What's  
13 the units on loss of lifetime? Years?

14                  MR. MEINHOLD: Yes.

15                  CHAIRMAN CARR: Okay. And the loss of  
16 life expectancy?

17                  MR. MEINHOLD: Is years.

18                  CHAIRMAN CARR: Is also years?

19                  MR. MEINHOLD: Right. And the difference  
20 between those, of course, the point -- the loss of  
21 life expectancy would be if everyone who is exposed  
22 the average would be .23, whereas the 13 would be, if  
23 you like the average, those who die from the -- who  
24 have the attributable death. So it would -- the 13 is  
25 the people that actually suffer the attributable

1 death. The .23 would be everybody.

2 CHAIRMAN CARR: When I read it, is what  
3 you're telling me what you intended to tell me? I  
4 shouldn't make any differentiation between the 10 and  
5 the 50?

6 MR. MEINHOLD: Well, on that basis, on the  
7 basis of loss of lifetime and the most probably age,  
8 but remember that the lifetime risk is greater, of  
9 course, between the 10 and the 50, so for that one  
10 you'd have to put some weight to it. For the loss of  
11 life expectancy, you see, it's about five times  
12 greater. So it depends upon how you would weight --

13 CHAIRMAN CARR: If I'm most probably going  
14 to die at 78 anyway --

15 MR. MEINHOLD: Right.

16 CHAIRMAN CARR: -- I don't know whether I  
17 worry about it or not.

18 MR. MEINHOLD: That is --

19 CHAIRMAN CARR: That's what you're trying  
20 to tell me?

21 MR. MEINHOLD: That's one of the things  
22 that need to be considered when you're trying to pick  
23 out which of these values -- that's my point. That,  
24 among others, is what you would have to consider. It  
25 isn't just the risk. It's also all of these

1 considerations.

2 CHAIRMAN CARR: That's the most probably  
3 age that I might die from cancer?

4 MR. MEINHOLD: Right.

5 CHAIRMAN CARR: Okay.

6 MR. MEINHOLD: Okay.

7 (Slide) The next slide, then, gets us to  
8 the values that the ICRP has recommended. They  
9 recommended essentially 10 rem in 5 years, and 5 rem  
10 in any 1 year. The other values, the annual dose  
11 equivalent for the limbs, the skin and the hands and  
12 feet are based primarily on the deterministic effects  
13 which Doctor Sinclair decided not to tell you about.

14 (Slide) The next is the occupational  
15 exposure of women, which is a very difficult problem  
16 in terms of the social impacts as well as the fetus.  
17 And the Commission has decided primarily that if  
18 they're not -- for women that are not pregnant they  
19 will have the same as men. There is no restriction on  
20 what we used to call women of reproductive age or  
21 capacity or whatever, and so -- because the  
22 consideration is that the probability of a fetus  
23 surviving an exposure which would be detrimental in  
24 that first two months or so probably isn't going to be  
25 an impact on the woman.

1           The second is that when the pregnancy has  
2       been, if you like, diagnosed and declared, then limit  
3       the dose equivalent to the mother's abdomen to 200  
4       millirem and the fetus to -- it implies also the fetus  
5       to something in the order of 100 millirem.

6           (Slide) I've given on the next slide--  
7       just a quick one, although we haven't discussed the  
8       basis -- the public dose limits, and one of the  
9       reasons is that those haven't changed because we did  
10      lower those. Both the NCRP and ICRP have lowered  
11      those to 100 millirem sometime in the last five or six  
12      years. The only difference for the ICRP is that it  
13      allowed excursions to 500 with an idea that you could  
14      average it over the lifetime, and now we're saying  
15      it's got to be restricted in terms of the time, that  
16      you can average it to only five years.

17           (Slide) If I could go to the next slide,  
18      perhaps we could talk a little bit about the system of  
19      protection. I've tried to diagram here sort of the  
20      conceptual way the Commission looks at exposures. And  
21      primarily we look at the fact that there is the source  
22      of exposure; there is the environment through which  
23      that exposure is received and the individual who  
24      receives it.

25           Now if we look at those sources such as--

1 I've used accelerators, but it could be teletherapy  
2 units or it could be steam generators or whatever  
3 else -- if we look at that kind of a source, we say  
4 that we can control that at the source. We can  
5 control it in the environment. That's really, if you  
6 like, our occupational case where we can put more  
7 shielding around it. We can ventilate. We can do  
8 whatever else. And we can control the worker. We can  
9 control his access, his training, and all the rest of  
10 it.

11 If we come to the top one, we're talking  
12 there about essentially remediation. That is, the  
13 source already exists. We can't control the source.  
14 It's in the ground or in the soil. We can work with  
15 the home. We can improve ventilation in the basement.  
16 We can seal up the cracks and that sort of thing.

17 And we have very little control over the  
18 public. That is, we don't control who comes and goes  
19 in the house and the rest of it. So we have much less  
20 control over the public. In fact, as a general  
21 principle, the Commission believes that almost always  
22 you need to control public exposure at the source,  
23 whereas occupational exposures you can control all the  
24 way through.

25 If we look at waste disposal, the reason I



1 put that on here is because we have a new category of  
2 what we call "potential exposures," that is exposures  
3 which may not happen. That could be a vault that's  
4 been controlled by an interlock system and you're  
5 asking about the failure of the interlock system.  
6 Here, you're talking about the failure of the waste  
7 disposal system, that is intrusion or explosion or  
8 whatever else, and there you again want to control  
9 that to the degree that you can at the source. And  
10 you can control it perhaps in the environment and at  
11 the worker. But again, as with the radon case,  
12 whenever it's a public exposure it's much better done  
13 at the source.

14 (Slide) Now we have divided these up, if  
15 I go to the next slide -- we have divided them up in a  
16 number of ways. Clearly we've stayed with our  
17 occupational, medical, and public exposure which is  
18 the traditional way to divide up exposures. But you  
19 also see that we've done it in terms of these  
20 classifications I've just discussed:

21 Practices or planned exposures, which are  
22 the occupational or even the public exposure in which  
23 you determine that you're going to release radioactive  
24 material at a given rate from a facility;

25 Then there are the potential exposures,

1 and I think these are very important areas where we're  
2 looking not at what is being received but what the  
3 probability of the release times the probability of  
4 the detriment, and that would be a waste disposal  
5 facility, interlock facility, or in your case a  
6 reactor accident of some kind;

7 The last one is the intervention, and here  
8 is a new concept that the Commission is trying to  
9 introduce in order to eliminate some of the confusion  
10 in for instance Chernobyl or other places in which  
11 radioactivity is in the environment. It wasn't  
12 planned. It's not a potential exposure. It's there  
13 like the radon case in the home. And so intervention  
14 is a different matter than the first two. Practices  
15 and potential exposure can be handled in terms of the  
16 normal operating situation, whereas intervention is a  
17 different beast, and that's how you need to be looking  
18 at recovery operations, if you will.

19 (Slide) If we go to the next slide -- the  
20 slide I should have begun with if I was a loyal ICRP  
21 person -- is to start with the three tenets, which is  
22 justification, optimization of protection, and the  
23 dose limits. But these are the basic tenets of  
24 radiation protection, optimization derived primarily  
25 from the fact that we have adopted an assumption of a

1 linear response and therefore any exposure causes some  
2 detriment. But you don't want to spend unnecessary  
3 resources and so you try to make sure that you have  
4 optimized protection looking at the economic factors,  
5 the social factors and whatever else.

6 (Slide) If I could go to the next slide,  
7 I did want to show the difference between the way in  
8 which one looks at justification and optimization in  
9 the intervention case. And here the justification  
10 isn't that I'm going to introduce a new source and is  
11 it worthwhile to do it. The radiation exists, and so  
12 all the justification is is am I going to do more harm  
13 than good -- excuse me, do more good than harm,  
14 because anything else is a spurious question. I mean,  
15 that's the only issue. Can I help? If I can help, I  
16 do it.

17 And the second one, the optimization, is  
18 merely that I do the best way I can. It's kind of a  
19 different concept. And we don't believe that dose  
20 limits apply, that new specific intervention levels  
21 need to be derived. Just as the radon levels in homes  
22 in the United States, the intervention levels would be  
23 much higher than any limit that we'd put on  
24 controllable -- or the practices that would result in  
25 people being exposed due to somebody else's

1 activities.

2 Well, I think that's pretty much the level  
3 of what I wanted to get into in the time we have  
4 available.

5 CHAIRMAN CARR: Thank you very much.

6 Questions, Commissioner Rogers?

7 COMMISSIONER ROGERS: On this last subject  
8 of justification, which is something we've wrestled  
9 with a great deal and I'm very interested in, how  
10 would you classify -- you've indicated how you'd deal  
11 with the justification question in the intervention  
12 classification, but how about practices? How would  
13 you deal with the issue of justification? How would  
14 you approach that?

15 MR. MEINHOLD: Well, let me say first that  
16 it's not just a radiation protection issue. It's much  
17 broader than that. And it really says that the public  
18 good is going to be greater than the harm. That's  
19 basically what -- but that harm is all of the costs.  
20 It's the cost of producing whatever it is you're going  
21 to do. It's the cost of protection. It's the cost of  
22 the detriment. You see, there are many costs  
23 involved.

24 I think a good example that I've always  
25 used for this is baggage x-ray machines in airports.

1       What is the justification? Well, the justification is  
2       made by FAA and a few others and government who say  
3       that it's very important to reduce the threat of  
4       hijacking for either perceived reasons, for actual  
5       reasons, for the cost of airplanes. As a protection  
6       person, I don't need to know what their values are for  
7       those. All I want to make sure is that they  
8       understand if you like the detriment and what we can  
9       do to reduce it.

10               Now once they decide that given a  
11       reasonable estimate of what the exposures would be  
12       that they're willing to accept that detriment along  
13       with all the other costs of introducing baggage x-ray,  
14       then I say to them now you must optimize it. You must  
15       now say, well, what's the total amount of collective  
16       dose I'm going to get from this baggage x-ray and do I  
17       add a flying spot x-ray machine which is going to cost  
18       me \$10 million to develop. And we say, yes, you  
19       should. And that's the optimization part of it. And  
20       the dose limits part would still come in to be sure  
21       that no individual member of the public -- the  
22       constraint on that would be that no individual member  
23       of the public, no matter how justified it is, should  
24       exceed 100 millirem per year. So that's the system,  
25       if you like, applied to a very specific case.

1                   COMMISSIONER ROGERS: Yes, but there are  
2 of course many other possibilities that one might have  
3 to consider. And the question of justification  
4 sometimes becomes really a very difficult debate as to  
5 who's going to make that decision and on what basis  
6 that practice might be justified.

7                   MR. MEINHOLD: I suppose it's the  
8 licensing authority.

9                   COMMISSIONER ROGERS: I beg your pardon?

10                  MR. MEINHOLD: I suppose it's the  
11 licensing authority.

12                  COMMISSIONER ROGERS: But on what basis?

13                  MR. MEINHOLD: All of the above.

14                  COMMISSIONER ROGERS: As soon as you start  
15 to move out of a purely technical basis for decision-  
16 making, you get into a wide open arena that a  
17 technical person is no more qualified to make that  
18 decision than anybody else in some ways. And it is a  
19 very sticky issue and it is one that we have wrestled  
20 with.

21                  MR. MEINHOLD: And of course the question  
22 is, is there a sort of trivial use of radiation that  
23 we would discourage even under your BRC philosophy.

24                  And the one I always use for my own  
25 purposes is if a manufacturer decided he would paint

1 the eyeballs of dolls with tritium to make they glow  
2 at night. How would I buy that? And some how or  
3 other, no matter what the dose is, it seems to me to  
4 be kind of a dopey thing to do. And those are the  
5 kinds of decisions I guess you're saying. It's a  
6 dopey thing to do, rather than I can calculate the  
7 dose and I can calculate the detriment and I'm going  
8 to say what that is.

9 But I think those are the kinds of  
10 judgments that are in justification. And as I said,  
11 they are not -- I don't think they're at all radiation  
12 protection alone issues. They're much broader than  
13 that.

14 COMMISSIONER ROGERS: Well, we could talk  
15 on it all day.

16 MR. MEINHOLD: Yes.

17 COMMISSIONER ROGERS: I'm sure it is a  
18 sticky issue.

19 You didn't discuss it here, but in reading  
20 the transcript of your presentation to the ACNW,  
21 Doctor Upton, I know at the end of that you really put  
22 your finger on a serious problem with respect to who's  
23 going to be around to do BEIR XV when the time comes  
24 and what the sources of qualified people will be to  
25 supply that need. And I know that's not what we're

1 really principally concerned about here today, but I  
2 wonder if you could say just a little bit about how  
3 you think that problem might be solved?

4 DOCTOR UPTON: Well, Commissioner Rogers,  
5 I'm not sure I know how the problem could be solved.  
6 I think that we are seeing the disappearance of a  
7 generation of people who've come into the radiation  
8 research area following the World War II impetus from  
9 atomic energy. And unlike the situation in other  
10 branches of science where there has been systematic  
11 effort on the part of Uncle Sam to support training to  
12 produce people, I don't think that same intensity of  
13 effort has been allocated to the problem of radiation  
14 protection, assessment of radiation risks, and the  
15 development of alternatives and standards.

16 So my own view is that there really ought  
17 to be a national study of the need for such people,  
18 not just to rely on Art Upton's top of the head view  
19 of things but a systematic examination of where we  
20 stand as a country. How many people are in the  
21 pipeline? Are they enough? What is the anticipated  
22 need for such people? And then, how do we produce  
23 them if we do need them?

24 I think that just as after World War II  
25 people came into this field because it was a new



1 field, there was obvious need for effort, I think if  
2 the nation identifies a national need, develops the  
3 training programs, the career opportunities that would  
4 be necessary, people will come into it.

5 CHAIRMAN CARR: Before we run into too  
6 much of Doctor Morris' time, let's get Doctor Morris  
7 on the record here.

8 DOCTOR MORRIS: Thank you.

9 The purpose of my presentation today would  
10 be to provide an overview of the process that's  
11 followed in developing regulatory standards for  
12 radiation protection and to provide a description of  
13 the NRC involvement in that process at all stages.

14 The key message I want to leave with you  
15 today I think is evident here from what's been  
16 discussed at the table, that this is a careful and  
17 deliberate process that not only involves the  
18 generation and development of basic scientific and  
19 technical information but involves the development of  
20 an international and national consensus on what kinds  
21 of recommendations are appropriate for radiation  
22 protection based on this information. And then  
23 finally, it involves the development of regulatory  
24 criteria that are reasonable, practical to implement  
25 in the field.

1           And so as we've looked at the evolution of  
2           the information from BEIR V and grappled with the  
3           implications of that for the BRC policy statement and  
4           10 CFR Part 20, that message I think has to be  
5           recalled, that there are many steps before we would  
6           now take actions involving use of BEIR V type  
7           information in our regulations. I think I'll try to  
8           lay out some of those steps now.

9           I want to point out that one part of this  
10          process is the development of basic scientific and  
11          technical information. The staff is involved in  
12          monitoring that process at various stages. For  
13          instance, we were aware through the staff review of  
14          technical information of the new dosimetry from the  
15          bomb survivors and of the developments of  
16          recommendations by UNSCEAR and the BEIR V and were  
17          able to provide the Commission with recommendations on  
18          how to deal with those early on. We were able to  
19          incorporate some of that thinking into the proposals  
20          in the BRC policy statement.

21          But in addition to monitoring these  
22          developments, the Agency supports development of  
23          scientific and technical information through research  
24          programs, for instance. We have programs who look at  
25          the way -- how you would go about calculating the dose

1 to the embryo/fetus based on the intake to the  
2 pregnant woman. We have studies going on in  
3 Brookhaven on the effectiveness of various specific  
4 measures to achieve doses that are ALARA throughout  
5 the world, and that's very useful research we think.  
6 So the Agency is involved at the very underpinnings of  
7 the development of this information.

8 In addition, as we look at the development  
9 of recommendations and guidance by standards writing  
10 committees such as ICRP and NCRP and others, the staff  
11 monitors those developments carefully at the  
12 professional staff technically and in the managerial  
13 level to do that. And in addition, we support those  
14 developments through individuals' participation in the  
15 various committees and through, in some instances,  
16 funding of those committees.

17 For example, we had Dick Cunningham, who  
18 works on the committee on applications of ICRP  
19 recommendations. We occasionally provide funding to  
20 the ICRP. There are eight different staff  
21 participants in working groups from the NCRP, although  
22 we have no member of the council itself, and we  
23 provide general funding amounting to \$150,000.00 per  
24 year approximately to the NCRP to support its work.

25 We have had staff members participating in

1 the development of International Atomic Energy Agency  
2 recommendations. Important among those  
3 recommendations were the safety guide '89 used as a  
4 reference point for developing the BRC policy. We're  
5 now -- staff is now working on a rulemaking to  
6 incorporate into Part 71 the recommendations from the  
7 IAEA on transportation.

8 We participate in the Committee on  
9 Interagency Radiation Research and Policy  
10 Coordination. About ten individuals participate in  
11 various committees or working groups of that  
12 organization. We support CIRRPC at about \$100,000.00  
13 a year. And I also mention here, although this isn't  
14 something that the staff involves itself in, the work  
15 of the Environmental Protection Agency developing  
16 generally applicable environmental standards that also  
17 are adopted as part of our regulations.

18 The final point is that the step that is  
19 taken historically before we would, say, adopt the  
20 guidance of these committees is to develop federal  
21 guidance, signed by the President. That is done  
22 involving various agencies working in committees  
23 chaired by the Environmental Protection Agency.  
24 Currently we are participating in efforts to develop  
25 guidance on the protection of the general public. I

1 would imagine that at some point in the future, the  
2 EPA would convene a committee to deal with the newer  
3 recommendations of the ICRP and the NCRP.

4 One point I wanted to mention too is that  
5 the standards that we're talking about are not just  
6 Part 20. There are a number of places in our  
7 regulations where we would have what would be  
8 considered radiation protection standards, Parts 34,  
9 35, 39, and in a number of regulatory guides. So, all  
10 of that is part of the pattern that we're talking  
11 about.

12 (Slide) Go on to the next viewgraph.

13 A key part of what we do in developing our  
14 regulatory standards and regulations is the evaluation  
15 of operating experience. One example of that is the  
16 radiation exposure information reporting system that  
17 gives us insights about the degree to which the  
18 licensees are achieving doses that are ALARA and this  
19 provides insight on the impacts of potential  
20 reductions, for instance, in dose limits.

21 Also, for instance, our examination of  
22 operating experience was able to focus on the issue of  
23 hot particles and how we might want to look at  
24 revising the skin dose limits in Part 20 in view of  
25 that experience.

1           Based on all these elements, the basic  
2       scientific and technical information, the  
3       recommendations of various groups and the evaluation  
4       of operating experience, then the agency identifies  
5       those areas where it believes it needs to modify its  
6       regulations and that can come either from petitions,  
7       from directions from the Commission or from  
8       recommendations from the EDO to the Commission. But  
9       in each case, there's a deliberate step where we  
10      decide that we do need to do something with  
11      regulations to incorporate this evolving guidance.

12           When we have made that decision, we go  
13      into the rulemaking process, of course, and there we  
14      are involved in analyzing benefits and impacts as  
15      exemplified by the regulatory impact analysis that we  
16      develop for every regulation and regulatory guide. We  
17      try to develop these regulations through the public  
18      comment process, sometimes involving public workshops,  
19      to show that the requirements are reasonable,  
20      inspectable, practical to implement. In certain  
21      instances, when we're trying to provide additional  
22      guidance for broadly applicable criteria that will be  
23      consistent with the dose limits, we will include  
24      margins to address uncertainties that are involved in  
25      trying to envelope a large range of operating

1 situations.

2 So, again, as I say, this is a process  
3 that we go through that involves a number of careful  
4 steps and I think that we have seen a kind of a  
5 snapshot of where we are today with regard to the  
6 evolution of the response to the BEIR V, UNSCEAR  
7 information and the development of recommendations by  
8 these ICRP and NCRP.

9 CHAIRMAN CARR: Thank you very much.

10 Go ahead, Commissioner Rogers.

11 COMMISSIONER ROGERS: I got started too  
12 soon.

13 CHAIRMAN CARR: Well, I started you too  
14 soon.

15 COMMISSIONER ROGERS: Well, I don't want  
16 to take up too much time. But it seemed to me in  
17 looking at some of the material in the BEIR V Report,  
18 I don't know whether it was the report itself or  
19 whether it was your presentation to ACNW, I seem to  
20 recollect that you said something to the effect, and  
21 please correct me if I'm wrong here, that some of the  
22 data which you were able to extract with respect to  
23 excess cancers you probably could never have seen if  
24 you hadn't known there'd been a bomb dropped on that  
25 population. Those numbers were so small that if you

1 hadn't known that there was an initiating event that  
2 then gave you the impetus to make a study, that if  
3 you'd simply been studying that population and looking  
4 at it, it wouldn't have told you that something had  
5 happened.

6 DOCTOR ELLETT: I think leukemia would  
7 have. The other things, that might be true.

8 What do you think?

9 DOCTOR UPTON: That's right. I think as  
10 Doctor Sinclair brought out, there have been roughly  
11 6,000 deaths from cancer in the study population,  
12 roughly 100,000 people in the two cities. Using the  
13 risk models that we've discussed, it has been  
14 estimated that there are perhaps 300 and some  
15 radiation-induced cancers out of that 6,000, of the  
16 order of 5 percent perhaps.

17 Epidemiological tools are just not  
18 sensitive enough to recognize that small an excess.  
19 There's enough difference between different cities in  
20 Japan so that an extra 300 out of 6,000 wouldn't  
21 attract any attention except as Doctor Ellett points  
22 out, that the excess of leukemia is large enough so  
23 that one would wonder what was going on there. But  
24 the total cancer excess would not attract attention.

25 CHAIRMAN CARR: Does the Japanese



1 population risk of cancer deaths, normally is it  
2 comparable to ours?

3 DOCTOR UPTON: Roughly comparable, yes.  
4 Different sites, much higher risk of gastrointestinal  
5 cancer, stomach, esophagus in Japan, higher risk of  
6 colon and breast in this country.

7 CHAIRMAN CARR: You had a comment?

8 DOCTOR SINCLAIR: Well, I just wanted to  
9 mention there's an additional point there. It's not  
10 just the total number of cancers, which is pretty  
11 small, but it's highly dose related. We know that the  
12 distribution of those cancers across a dose, and that  
13 makes it almost incontrovertible that it was radiation  
14 that caused those few. I think it's helped us to see  
15 it very much.

16 COMMISSIONER ROGERS: Well, I'm not being  
17 critical. I'm just trying to say we always have to  
18 put this whole question, these health effect questions  
19 in some broad context in which to understand them.  
20 This whole question of when something cannot be  
21 measured in terms of its consequences, but one can  
22 calculate it, how do you deal with that? How do you  
23 really practically deal with this kind of a situation,  
24 which is what we're dealing with right now in things?

25 DOCTOR SINCLAIR: Well, I think it's quite

1 possible, just to emphasize the point, that if there  
2 had been a five percent increase in the population and  
3 we didn't have any bomb to attribute it to, it would  
4 be very difficult to see because we don't know rates  
5 even in different parts of the country that well. But  
6 I think, as Doctor Ellett's pointed out, the leukemia  
7 rather specific and the dose-related nature of the  
8 other cancers does give us a great deal of confidence  
9 that that's what they were due to.

10 CHAIRMAN CARR: Do you have that much  
11 confidence that you know what the dose rate was for  
12 those people?

13 DOCTOR SINCLAIR: The dose rate?

14 CHAIRMAN CARR: The doses.

15 DOCTOR SINCLAIR: I have a fair amount of  
16 confidence in the DS86, yes. It has limits, of  
17 course. But, you know, with the extra confirmation  
18 that we've -- well, first of all, DS86 is a  
19 tremendously comprehensive evaluation. Now, there's  
20 ten years of dosimetry work in there involving a  
21 considerable number of --

22 CHAIRMAN CARR: But the exposure levels  
23 those people were exposed to is kind of --

24 DOCTOR SINCLAIR: Well, they range from  
25 less than a rad to over 400. We think we know those

1 numbers to the order of 25 to 30 percent now. And we  
2 have direct experimental confirmation, which we've  
3 never had before, of the gamma rays from measurements  
4 of thermal luminescent materials that were present at  
5 the site. That was never available to us before  
6 because the technique wasn't sensitive enough. It  
7 confirms the calculations which are fairly  
8 sophisticated now to within about ten or 15 percent or  
9 so.

10 CHAIRMAN CARR: Excluding shielding that  
11 you don't know about.

12 DOCTOR SINCLAIR: Well, shielding is a  
13 bigger uncertainty and it's a bigger uncertainty with  
14 some members of the sample that we haven't yet been  
15 able to put into the sample because we haven't figured  
16 out exactly how to have them reach the precision of  
17 the dosimetry system itself. But we're doing that  
18 slowly with various approximations.

19 CHAIRMAN CARR: Do you want to comment on  
20 that, Doctor Upton?

21 DOCTOR UPTON: No, the area of dosimetry  
22 is outside my field of expertise. But I think, as has  
23 been brought out, where we see the big effects are in  
24 those small numbers of people who got the big doses in  
25 close to ground zero. There's no question about the

1 excess there. But most of the people, of course, were  
2 out at greater distances and the effect falls off with  
3 distance with dose, so that looking at the whole  
4 population it's diluted out. You wouldn't see it.

5 But looking at the heavily irradiated  
6 population, there is a definite effect. For leukemia,  
7 many times normal incidence, many times normal.

8 COMMISSIONER ROGERS: I'll pass it.

9 CHAIRMAN CARR: Commissioner Remick?

10 COMMISSIONER REMICK: First, I'd like to  
11 add my personal welcome to such a distinguished group  
12 and thank you for coming in and spending time with us.  
13 It's been extremely helpful.

14 Doctor Meinhold, you touched upon  
15 something I've been curious about for a long time and  
16 that is the dose to astronauts. You point out that  
17 they don't fit the typical occupational mold. Do you  
18 know what a typical case might be?

19 MR. MEINHOLD: Well, I'll let Doctor  
20 Sinclair -- he's been heavily involved in that  
21 Committee activity, so we'll let him.

22 COMMISSIONER REMICK: Okay.

23 DOCTOR SINCLAIR: Well, we simply had to  
24 have a different approach to them because normally, of  
25 course, we use for radiation protection occupationally

1 on the ground an annual limit to control the exposures  
2 of the individuals. It's not easy to translate that  
3 straight into space for various different reasons.  
4 They have missions rather than years. In considering  
5 the space station, for example, we were already  
6 informed that they were likely to have 90 day tours of  
7 duty, as one example.

8 We decided then that the only way to go  
9 was to fix a career limit which would have a certain  
10 percentage risk. How do we get at that? We have in  
11 essence in worker populations three major groups:  
12 shall we say the very safe industries working in  
13 offices and so on; the not so safe but very normal  
14 occupations; and the least safe which are not normal  
15 either, test pilots and all that sort of thing.

16 For various reasons, we considered that  
17 the astronauts could not be put in the safest category  
18 because considering the various risks of other kinds  
19 that they had, which were much larger, it would be  
20 ridiculous to limit them, absurdly. On the other  
21 hand, you couldn't put them up in a test pilot  
22 category for radiation either because they already did  
23 have a big risk of a test pilot character going for  
24 them. So, we decided on an intermediate level which  
25 worked out to be at that time three percent career

1 limit. Then we decided as a function of age how much  
2 that would translate into a career limit and it worked  
3 out at numbers from 100 rems upwards in terms of a  
4 career limit which, by the way, none of them have  
5 approached yet. They're not even close. I think  
6 around 10 rems or so is the highest --

7 CHAIRMAN CARR: Do they wear dosimetry?

8 DOCTOR SINCLAIR: Beg your pardon?

9 CHAIRMAN CARR: Do they take dosimetry on  
10 the trips?

11 DOCTOR SINCLAIR: Yes. Yes, they do.  
12 We're facing considerations now, of course, of what  
13 the exposures will be in a Mars mission. That will  
14 certainly be up there.

15 COMMISSIONER REMICK: Has the NCRP done  
16 anything with airline pilots, similarly looking at  
17 risk and amount of exposure?

18 DOCTOR SINCLAIR: We haven't. We have a  
19 study in prospect right now.

20 COMMISSIONER REMICK: And one final  
21 question. What is the status of the ICRP draft 1990  
22 report? Where is that?

23 MR. MEINHOLD: The expectation is that it  
24 will be approved at the end of this year and probably  
25 be available earlier next year.

1 COMMISSIONER REMICK: Are you able to  
2 predict any possible changes or do you think it will  
3 go as recommended?

4 MR. MEINHOLD: I think the dose limits  
5 will go pretty much as recommended and the weighting  
6 factors as Doctor Sinclair presented. So, I think--  
7 by the way, I should point out this is the first time  
8 ever the ICRP has sent its documents out for  
9 discussion. They went all over the world, which had  
10 tremendous benefits which both Doctor Sinclair and I  
11 are very proud of because we beat pretty hard on the  
12 Commission to allow that to happen.

13 The fact is most people who commented were  
14 more concerned about clarity than they were about the  
15 answers. So, I think in general the final document  
16 won't be terribly different in terms of the numbers.  
17 Hopefully it'll be a little clearer.

18 COMMISSIONER REMICK: Thank you.

19 CHAIRMAN CARR: Commissioner Curtiss?

20 COMMISSIONER CURTISS: I just have a  
21 couple of questions here.

22 Doctor Meinhold, your suggestion that we  
23 take a look at occupational dose limit in the context  
24 of the remaining age of the individual touched on a  
25 concern, if I understand what you're saying, that I've

1 had in the past and that's the assumption that we as  
2 regulators use in terms of the exposure of the  
3 individual, in particular the concept of the maximal  
4 exposed individual, full-time exposure, 24 hours a day  
5 for a 70 year lifetime.

6 I wonder if you could expand upon that  
7 concept and maybe touch on the notion of MEI, maximal  
8 exposed individual, and what you see. Is there an  
9 emerging consensus about the use of that concept or  
10 the departure from MEI to some other more realistic  
11 and accurate approach?

12 MR. MEINHOLD: I maybe don't follow you  
13 exactly, but I think you're dealing with really what  
14 the ICRP was concerned about in its old recommendation  
15 in which it set a dose limit for the average -- based  
16 on the average exposure, assuming a distribution of  
17 exposed workers, and that that distribution would be  
18 similar to safe industry distribution.

19 The new ICRP recommendation, and I think  
20 the lifetime limit for the NCRP are both aimed at  
21 looking at that individual who might be exposed at the  
22 maximum level over his working lifetime. It's those  
23 two considerations which lead you to what ICRP has now  
24 said is a limit of intolerability, which is the  
25 maximal exposed individual which would be reached at



1        their consideration that at two rem per year, and what  
2        NCRP will be looking at in terms of its present  
3        guidance of 5 rem and to your age, which again -- the  
4        5 rem per year allows the flexibility for any worker.  
5        The age limit is this maximum exposed individual  
6        protected so that over his working life he doesn't  
7        exceed two or three percent in terms of his lifetime  
8        risk of cancer.

9                So, I think they both are maximum exposed  
10       individual concepts now.

11               COMMISSIONER CURTISS:    Maybe the question  
12       that I have goes to the more generic issue of whether  
13       the concept of the maximal exposed individual, seven  
14       year exposure for 24 hours a day from your perspective  
15       is a reasonable one.

16               MR. MEINHOLD:    I think it's unreasonable  
17       but not -- that may be wrong.    I don't think it's very  
18       likely, but I think it's a possibility.    The thing  
19       that I think that I would drive toward is that for the  
20       NCRP approach, that is some annual limit -- let us use  
21       5 since that's what's in '91, that will normally  
22       result in exposures far less than one rem to most of  
23       the individuals.    I believe that there's normally a  
24       distribution of exposures not just in the work force,  
25       but in the worker.    That is, he will get most of the

1 exposure from age 23 to 35 and then he becomes a  
2 commissioner or something and doesn't have to get  
3 exposure.

4 COMMISSIONER ROGERS: That's a fact.

5 MR. MEINHOLD: But the fact is that I  
6 think there is that distribution but it doesn't mean  
7 that there aren't a few guys out there who like to  
8 clean steam generators and they get a lot of money for  
9 it and that's their livelihood. When they look to  
10 other alternatives, they find they can't make anywhere  
11 near as much money.

12 Is it possible? Yeah, I guess it probably  
13 is. It may be possible for -- well, just if the dose  
14 limits -- for instance, the ICRP numbers of two rem  
15 per year under some of the new aircraft that are being  
16 discussed by the industry, it may well be that the  
17 pilots will be a problem in terms of dose limits. And  
18 they could do it over their careers, from the time  
19 they're perhaps 30 to 65.

20 So, I don't know the answer to your  
21 question.

22 COMMISSIONER CURTISS: Okay.

23 MR. MEINHOLD: I don't think it's  
24 expected, but I think there could be individuals and  
25 the fact is that I think the flexibility of the five

1 rem per year and the age takes care of both of these  
2 comfortably. It means you probably aren't going to  
3 have any difficulty with the five rem as a normal  
4 limit for most individuals and for that strange  
5 individual we're worried about, he'll be protected by  
6 the lifetime limit.

7 COMMISSIONER CURTISS: Doctor Upton,  
8 you've described the process that you've gone through  
9 as one that's continuing and I suspect with the  
10 additional data that will come in will continue to  
11 examine these questions. I wonder if you could say a  
12 word or two about the significance of the Chernobyl  
13 health effects data and in particular the value of  
14 that data from the standpoint of filling whatever gaps  
15 in the information we have now and that over the  
16 course of the next several years we might find to be  
17 beneficial.

18 DOCTOR UPTON: Thank you, Commissioner  
19 Curtiss. I think that if it is possible to quantify  
20 the doses to individuals in the area around Chernobyl  
21 or individuals involved in the cleanup of the  
22 accident, the fire fighting and so on, then the study,  
23 the long-term study of that population would be  
24 scientifically useful. Radiation was received over a  
25 matter of days or weeks or even longer, unlike the

1 situation in Hiroshima/Nagasaki. I understand that  
2 there were substantial doses to a substantial number  
3 of people. Perhaps the total collective population  
4 dose approaches that that we had in  
5 Hiroshima/Nagasaki.

6 So, epidemiologically, I think it deserves  
7 careful consideration. I'm not as close to the  
8 situation as Doctor Sinclair is. I believe he's been  
9 over and visited the site, talked to people there and  
10 his opinion is probably better informed than mine.

11 Warren?

12 DOCTOR SINCLAIR: Well, I think at the  
13 present time the situation is not clear about what we  
14 can expect in the way of epidemiological studies from  
15 the Russians. It started off with what was called an  
16 all union center in Kiev which was to be set up to  
17 look at all the likely groups and register some at  
18 least 200,000 people who might have had more than  
19 minor exposures.

20 But since the political changes in the  
21 Soviet Union, the republics have decided to do these  
22 things by themselves and not as an all union affair.  
23 The center at Kiev has become for the Ukraine only.  
24 Byelo Russia, which is one of the other -- there are  
25 three republics with hot spots in them. Byelo Russia

1 and Russia are setting up separate studies to examine  
2 the effects in their own people from their own hot  
3 spots.

4 And at this point in time, I'm not very  
5 clear, and I'm not sure if anybody is, about what the  
6 exposed groups have been exposed to. We thought at  
7 one stage that it was a very nice epidemiological  
8 package, if you like, in the people who were evacuated  
9 from Pripyat and around Chernobyl. There were about  
10 24,000 of those people who seemed to have average  
11 doses of about 45 rads. Not a bad little package.  
12 But I understand those doses have since been revised  
13 downwards and that in the hot spots that have been  
14 found in Byelo Russia and Russia, which were the  
15 result of rainfall because they're quite distant from  
16 Chernobyl relatively, I don't know what the exposures  
17 are and I haven't got a good record of them.

18 So perhaps somebody does know, but it's  
19 not me at this point in time, I'm afraid. I don't  
20 have too much hope that we'll get a lot from those  
21 studies.

22 COMMISSIONER CURTISS: That's all I have.

23 CHAIRMAN CARR: Doctor Upton, the BEIR V  
24 Report states that no increase in the frequency of  
25 cancer has been documented in populations residing in

1 areas of high natural background radiation. To a non-  
2 specialist, there appears to be a logical disconnect  
3 between this statement and the downward extrapolations  
4 of the risk associated with the higher radiation  
5 doses. Why aren't the results of the background  
6 studies given greater weight in developing these risk  
7 coefficients?

8 DOCTOR UPTON: Well, the report, Mr.  
9 Chairman, does stress that the epidemiological data  
10 don't exclude zero risk at levels of natural  
11 background. The problem, I think, is that as one goes  
12 to lower and lower levels of exposure one gets down  
13 into the noise level, if you will, and it's simply not  
14 possible to distinguish effects that are so small.

15 There has been a study of thyroid nodules  
16 in women residing in a region of China where the  
17 natural background levels are elevated and there dose  
18 to 70 years is estimated to approach something of the  
19 order of 9 rads. Nine rads given to a child, that  
20 leads to an appreciable excess of thyroid nodules and  
21 thyroid cancers. If there is no demonstrable excess  
22 in women in China, argues that at that low level  
23 exposure, rate of exposure, the 9 rads over a  
24 lifetime, the radiation is very much less effective.  
25 That's a possibility, as it's been brought out. We

1 really don't know as we go down to lower and lower  
2 dose rates that the risks will be as large as  
3 suggested by the linear extrapolation.

4 CHAIRMAN CARR: Well, did the Committee  
5 consider the epidemiological follow-up studies of  
6 patients who received up to 50 R for thyroid therapy  
7 in diagnosis?

8 DOCTOR UPTON: Yes. Yes. That  
9 information was considered. There is a reference to  
10 the work by Hoel. I believe that's the study that  
11 you're referring to. I think it's been brought out  
12 earlier today that there are some suggestions in the  
13 epidemiological literature that protracted irradiation  
14 is less effective. I think Doctor Sinclair said  
15 perhaps by a factor of four irradiation from  
16 radioiodine over a long period of time could be, over  
17 a lifetime, much less effective than a factor of four.

18 There are uncertainties there and I think  
19 that was one of the reasons why the Committee argued  
20 that for highly protracted irradiation one should  
21 assume that these risk estimates are likely to be high  
22 by a factor of two or more.

23 CHAIRMAN CARR: Doctor Ellett?

24 DOCTOR ELLETT: Mr. Chairman, I think the  
25 Committee really discussed that thyroid paper in some

1 detail and their conclusions on thyroid cancer was  
2 that it was really something that had to be studied,  
3 that there was conflicting evidence on both sides.

4 I must point out that that study in Sweden  
5 is by no means complete. People have been followed a  
6 long time. There have been other reports quite  
7 similar from studies in the United States and when the  
8 people have been followed for 30 or 40 years, the  
9 excess appeared. It takes a long time. But I don't  
10 think it's too safe to look at an early  
11 epidemiological study and conclude there's no effect.

12 CHAIRMAN CARR: Okay. On one of your  
13 slides, and I may have mis-looked it -- you know, the  
14 press release when the BEIR V Report came out said the  
15 risk from radiation, ionizing radiation, was four  
16 times greater than previously estimated. But one of  
17 your slides kind of indicated that the BEIR V  
18 Committee was in the range of all the rest of the risk  
19 estimates from the I, II, III, IV committees.

20 DOCTOR ELLETT: I'm glad you asked that  
21 question. The BEIR III Committee came out with a  
22 preferred risk model based on a linear quadratic  
23 response that gave -- that was the one newspapers and  
24 regulatory agencies alike used as the BEIR III risk  
25 estimates. The BEIR V Committee's risk estimates are



1 about three to four times higher than that preferred  
2 one. If you go to the same model, you get about the  
3 same answer. But the BEIR III Committee used a linear  
4 quadratic model.

5 Now, I think it's interesting that that  
6 model had, in a sense, a dose rate effectiveness  
7 factor of 2 and a half built into it, as I believe  
8 Doctor Sinclair pointed out. If you apply a dose rate  
9 effectiveness factor of two, if you will, to the BEIR  
10 V Committee's risk estimates, you'll find that their  
11 risk estimates are higher by about a factor of two.  
12 That might have been a better way to put it in the  
13 report. I don't know. The Committee did this both  
14 ways really and finally decided you should really  
15 compare it to the preferred model. That was the one  
16 people knew, not the highest number in the BEIR III  
17 Report.

18 CHAIRMAN CARR: Okay. I guess I'm trying  
19 to figure out whose slides these were. I guess these  
20 were yours, Doctor Sinclair. On the one on risks of  
21 cancer after one rad of whole body irradiation, that  
22 doesn't have anything in it on rate, dose rate of that  
23 one rad?

24 DOCTOR SINCLAIR: No, it doesn't. It has  
25 numbers over on the left-hand side which I'm --

1                   CHAIRMAN CARR: It says an annual risk.

2                   DOCTOR SINCLAIR: -- not at all proud of  
3 because it's an old slide based on the old numbers and  
4 I haven't redrawn it yet. So, it's the shape of the  
5 curve and it's time relationship that's important  
6 rather than the absolute value of the risk.

7                   CHAIRMAN CARR: Okay. I guess I  
8 understand.

9                   And Doctor Upton, given the large amounts  
10 of uncertainties in the risk associated with the low  
11 doses of ionizing radiation, what research would you  
12 think the Committee would recommend that we pursue to  
13 attempt to reduce those uncertainties? Is there  
14 something we can spend our money on to kind of  
15 eliminate that uncertainty?

16                  DOCTOR UPTON: Well, it's been brought  
17 out -- Mr. Chairman, it's been brought out a number of  
18 times elsewhere that more than half of the atomic bomb  
19 survivors who were put into the study in 1950 are  
20 still surviving. Much of the uncertainty in our  
21 estimates really relates to what's going to happen as  
22 those survivors age.

23                  CHAIRMAN CARR: We've got to wait for time  
24 then.

25                  DOCTOR UPTON: And we need to look at

1 human populations where there is good reason to think  
2 we can harvest information. Reference has been made  
3 to the Chernobyl population, that there are  
4 uncertainties there. Clearly, I think, it's very  
5 important to continue to follow the A-bomb survivors  
6 meticulously for another 20 years. That will help a  
7 whole lot in narrowing the uncertainties. But again,  
8 as you point out, that's extrapolation from  
9 instantaneous exposure, relatively high doses --

10 CHAIRMAN CARR: Well, has anybody taken  
11 these guys who love to clean steam generators that  
12 Doctor Meinhold talked about --

13 DOCTOR UPTON: I think it's important to  
14 follow the --

15 CHAIRMAN CARR: -- and watching that where  
16 we've got a good record of how much they've been  
17 exposed to?

18 DOCTOR UPTON: I think it's very important  
19 to follow the occupationally exposed populations. I  
20 myself doubt that we can refine risk estimates very  
21 much that way, but we can help to set upper limits.  
22 If, as some allege, the BEIR V estimates are too low,  
23 then studying radiation worker populations and cooling  
24 data from different countries, that analysis ought to  
25 help reassure us that these estimates are not --

1                   CHAIRMAN CARR: Certainly we've got better  
2 data on those people than we do on Chernobyl for  
3 instance --

4                   DOCTOR UPTON: Indeed.

5                   CHAIRMAN CARR: -- or even the bomb  
6 survivors.

7                   DOCTOR ELLETT: Well, this is a place  
8 where I think the Commission could really help  
9 science. We have a committee at the Academy that's  
10 looking now at the study of U.S. utility workers for  
11 radiation effects. One of the major difficulties in  
12 performing a study like this is the availability of  
13 data for the contract workers who get most of the  
14 dose. This data is held by the Nuclear Regulatory  
15 Commission. It's in the computer. The people at NRC  
16 have been very good about coming in and briefing us  
17 about it. But the problem is Commission's regulations  
18 do not allow the release of this data for  
19 epidemiological studies.

20                   Health organizations like Health and Human  
21 Services --

22                   CHAIRMAN CARR: You're talking to the  
23 right guys.

24                   DOCTOR ELLETT: -- do. This can be done.  
25 It's just a question of looking at how the data could

1 be released for studies. It's important.

2 CHAIRMAN CARR: Okay. Early on I remember  
3 when the original Nautilus crew was put under an  
4 intensive program, all of them, for cataracts. We  
5 were all base lined and logged and looked at for years  
6 and they finally quit that because they weren't  
7 finding anything. But if we've got a good database to  
8 start with, it looks like we ought to start following  
9 it. I don't know that anybody's doing that.

10 MR. MEINHOLD: Part of the difficulty, of  
11 course, is that the exposure in the industry really  
12 started in the middle '70s, your large numbers of  
13 people. As Doctor Sinclair's slide shows, solid  
14 tumors take a long time to get started.

15 CHAIRMAN CARR: All the more reason we  
16 ought to start tracking them.

17 MR. MEINHOLD: Yes, you've got to start  
18 tracking them. That's why the study is difficult.  
19 Even though there's a lot of exposure, there hasn't  
20 been a lot of time.

21 CHAIRMAN CARR: But a lot of us are beyond  
22 that age-related routine where it starts.

23 Let's see, I think that finished up my--  
24 so you're not sure you can give us any specifics on  
25 the research needs then that we need to dig into?

1 DOCTOR UPTON: Well, certainly the follow-  
2 up of irradiated human populations where the data can  
3 be harvested, that's very important. I think that  
4 we're just on the dawn of an enormous explosion of  
5 knowledge relating to cancer, getting to identify the  
6 genes that are involved, develop tools to detect  
7 genetic change with high sensitivity.

8 So, I think that the epidemiological  
9 studies need to be paralleled by the studies in the  
10 laboratory with cultured cells, with animal systems.  
11 There's a spectrum of research, all of which will help  
12 to narrow our uncertainties. That's why I would think  
13 that it would make sense for a national study to take  
14 a look at where the science is, where the problems  
15 are, what is being done to address those problems.  
16 Are they being pursued vigorously? Do we have the  
17 scientific talent we need? If the answers are we need  
18 more of this or that, then let's develop a national  
19 plan to address those needs.

20 CHAIRMAN CARR: Thank you.

21 Any other questions?

22 Well, let me thank you all for this  
23 informative briefing about the development of  
24 radiation protection standards. The mission of the  
25 U.S. Nuclear Regulatory Commission is to protect the

1 public and the environment from potential hazards  
2 associated with the uses of nuclear materials.

3 Radiation protection standards are an  
4 integral and essential part of our regulatory program  
5 to ensure the protection of the public. Your briefing  
6 today has helped us to understand the process through  
7 which expert bodies like the National Research  
8 Council, NCRP and the ICRP derive estimates of the  
9 risks associated with ionizing radiation and formulate  
10 recommended standards for radiation protection.

11 Your briefing has also highlighted the  
12 many precautions taken throughout the process to  
13 ensure proper protection of the public in light of the  
14 uncertainties associated with the low doses and dose  
15 rates typically associated with licensed and exempted  
16 nuclear activities.

17 Given the large uncertainties that still  
18 exist at these low doses, we need to continue to focus  
19 our collective efforts on reducing these uncertainties  
20 and on sustaining the scientific capability to improve  
21 our understanding of the health and environmental  
22 significance of ionizing radiation.

23 I thank you for your presentations today  
24 and for the careful and thorough work of your  
25 organizations over the years in support of radiation

NEAL R. GROSS  
1323 Rhode Island Avenue, N.W.  
Washington, D.C. 20005  
(202) 234-4433

1 protection programs of this Agency and the many users  
2 of radioactive materials.

3 Unless there are additional comments, we  
4 stand adjourned.

5 (Whereupon, at 12:04 p.m., the above-  
6 entitled matter was concluded.)  
7  
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CERTIFICATE OF TRANSCRIBER

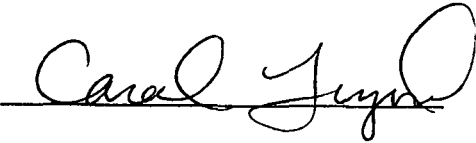
This is to certify that the attached events of a meeting  
of the United States Nuclear Regulatory Commission entitled:

TITLE OF MEETING: BRIEFING ON DEVELOPMENT OF RADIATION PROTECTION  
STANDARDS

PLACE OF MEETING: ROCKVILLE, MARYLAND

DATE OF MEETING: AUGUST 1, 1990

were transcribed by me. I further certify that said transcription  
is accurate and complete, to the best of my ability, and that the  
transcript is a true and accurate record of the foregoing events.



Reporter's name: Peter Lynch

**NEAL R. GROSS**  
COURT REPORTERS AND TRANSCRIBERS  
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WASHINGTON, D.C. 20005

SCHEDULING NOTES

Title: Briefing on Development of Radiation Protection Standards

Scheduled: 10:00 a.m., Wednesday, August 1, 1990 (OPEN)

Duration: Approx 1-1/2 hrs

Participants: SUMMARY OF BEIR V RESULTS 30 mins

- Dr. Arthur C. Upton  
Chairman  
Department of Environmental Medicine  
New York University Medical Center

- Dr. William H. Ellett  
Senior Program Officer  
Board of Radiation Effects Research  
National Research Council

DEVELOPMENT OF ICRP AND NCRP RECOMMENDATIONS 30 mins  
REGARDING RADIATION PROTECTION

- Dr. Warren Sinclair  
President  
National Council on Radiation  
Protection and Management

- Mr. Charlie Meinhold  
Division Head  
Radiological Science Division  
Brookhaven National Laboratory

NRC Staff

Dr. Bill M. Morris 10 mins

- Development of regulatory standards for  
radiation protection

## THE BEIR V COMMITTEE

A. Upton (Chair)  
D. Hartl (Vice Chair)  
B. Becker  
K. Clifton  
C. Denniston  
E. Epp  
J. Fabrikant  
D. Grahn

E. Hall  
D. Herbert  
D. Hoel  
G. Howe  
S. Jablon  
A. Kennedy  
A. Knudson  
D. Thomas

D. Preston, Scientific Advisor

## **TYPES OF RADIATION INDUCED CANCER MODELED**

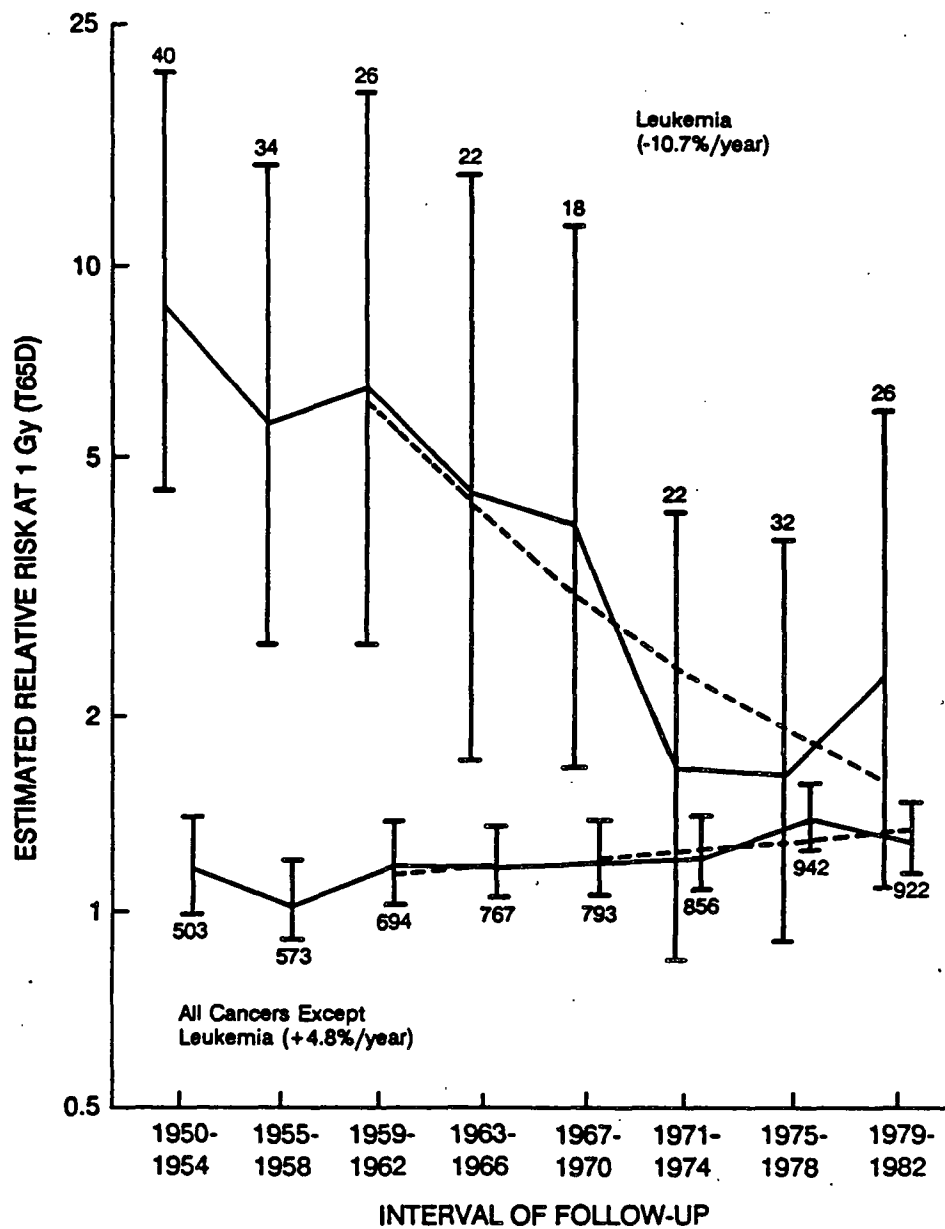
**Leukemia**

**Breast**

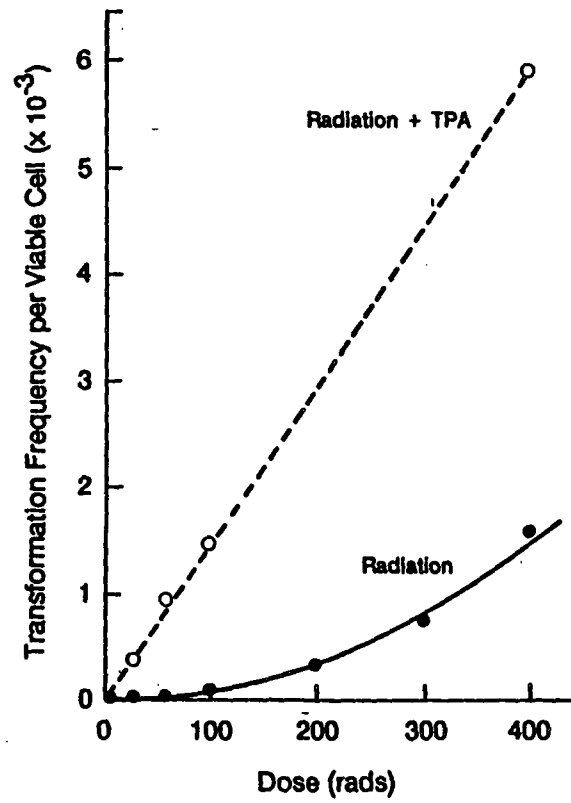
**Respiratory System**

**Digestive System**

**All others (as a group)**

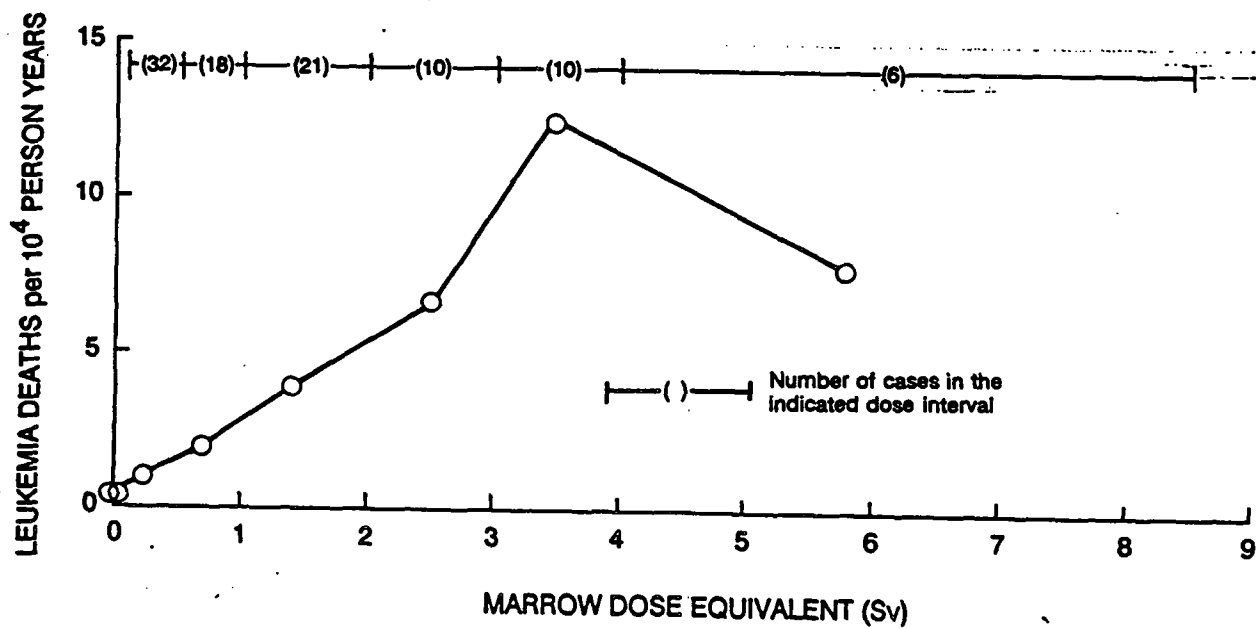


W471 fig 4.5-2



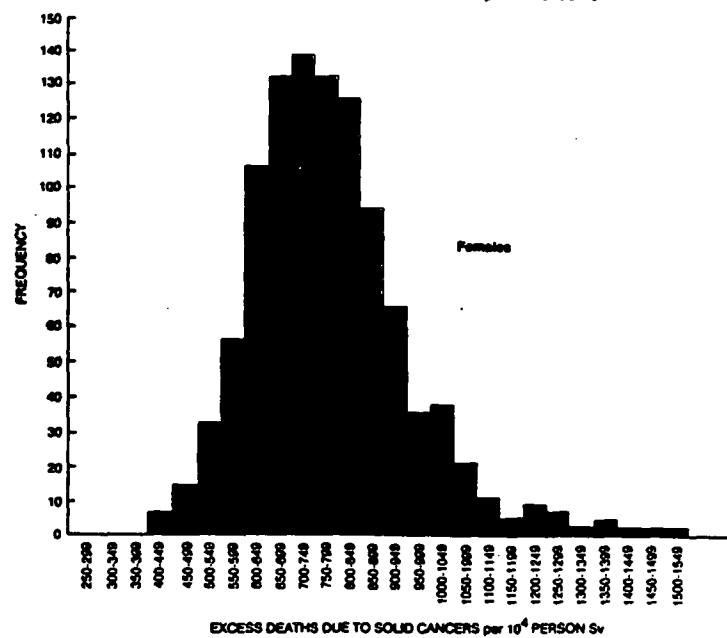
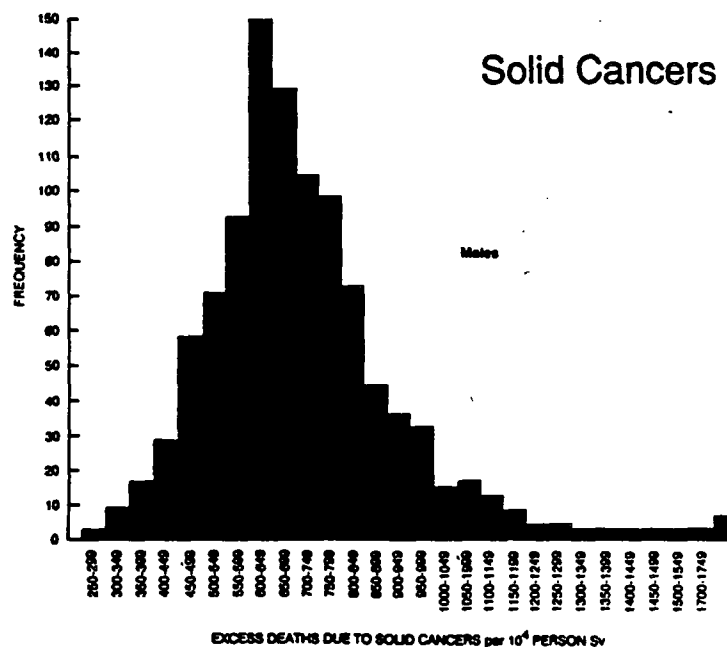
W471 fig. 3-4

Prentiss: double check  
percentage



5-1

W47108





## **CANCER MORTALITY - ACUTE DOSE**

**0.1 Sv to 100,000 persons of a given sex**

<b>Male</b>		<b>Female</b>	
<b>Leukemia</b>	<b>Solid</b>	<b>Leukemia</b>	<b>Solid</b>
<b>110</b>	<b>660</b>	<b>80</b>	<b>730</b>
<b>TOTAL</b>	<b>770</b>		<b>810</b>
<b>AVERAGE</b>	<b>8 x 10<sup>(-4)</sup> per rem</b>		

**MORTALITY BY TYPE OF SOLID CANCER**  
**FEMALES**

<b>Breast</b>	<b>Respiratory</b>	<b>Digestive</b>	<b>Other</b>
<b>70</b>	<b>150</b>	<b>290</b>	<b>220</b>

**TOTAL MORTALITY  
BY AGE AT EXPOSURE**

**FEMALES**

<b>5</b>	<b>1532</b>	<b>55</b>	<b>505</b>
<b>15</b>	<b>1566</b>	<b>65</b>	<b>386</b>
<b>25</b>	<b>1178</b>	<b>75</b>	<b>227</b>
<b>35</b>	<b>557</b>	<b>85</b>	<b>90</b>

# **TOTAL CANCER MORTALITY - MALES**

**1 rad per year - age 18-65**

**2880  
(2150-5460)**

**14% of normal expectation**

**Average years of life lost  
per excess cancer - 15 years**

## COMPARISON OF BEIR CTTES' RISK ESTIMATES

Linear Relative Risk Models - Lifetime Plateau  
(Cancer fatalities per million person rem)

BEIR-I 1971	690	Early Deaths 1970 S.Pop
BEIR-III 1980	500	Early Deaths 1970 S.Pop
BEIR-V 1990	790 (1000)	Excess Deaths 1980 S.Pop (Early Deaths)

# **RANGE OF CA RISK ESTIMATES BY BEIR COMMITTEES**

**(Cancer fatalities per 1,000,000 person rem)**

**BEIR-I    130-690    L (Additive, 30 yr.)-(RR,Life)**

**BEIR-III    10-500    QL (Additive)-L (RR,Life)**

**BEIR-V    400-1700    90% Credibility interval  
              (540-1240)    90% stat.confidence interval**

# **ESTIMATES OF GENETIC RISK**

**cases per 1,000,000 liveborn  
(1 rem per generation)**

	<b>First Generation</b>	<b>Equilibrium</b>
<b>Autosomal</b>	<b>6-35</b>	<b>100</b>
<b>Congenital Abnormalities</b>	<b>&lt;10</b>	<b>10-100</b>
<b>Unbalanced Translocations</b>	<b>&lt;5</b>	<b>5+</b>
<b>Others</b>	<b>&lt;3</b>	<b>6+</b>

# **ESTIMATES OF CANCER RISK AND DETRIMENT - THE BASIS OF ICRP AND NCRP RECOMMENDATIONS**

**Warren K. Sinclair  
President**

**National Council on Radiation  
Protection and Measurements**

**Presentation to the  
Commissioners of the  
Nuclear Regulatory Commission  
August 1, 1990**



# Concerns in Low Dose Radiation Protection

## Stochastic Effects

- No threshold
- Magnitude of effect same at all doses
- Frequency proportional to dose at low doses

## Hereditary Effects

## Induction of Cancer

## Special Concern

- Risk of Mental Retardation in Fetus
- Deterministic (nonstochastic) effects are not a concern because limits are below thresholds.

# **Exposed Human Populations for Risk Estimation**

## **A Bombs**

**Japanese Survivors  
Marshall Islanders**

## **Medical Therapy**

**Pelvic Radiotherapy  
Spinal Radiotherapy (A.S.)  
Neck, Chest Radiotherapy (thyroid)  
Scalp Irradiation (tinea capitis)  
Breast Radiotherapy  
Radium <sup>224</sup> Treatment**

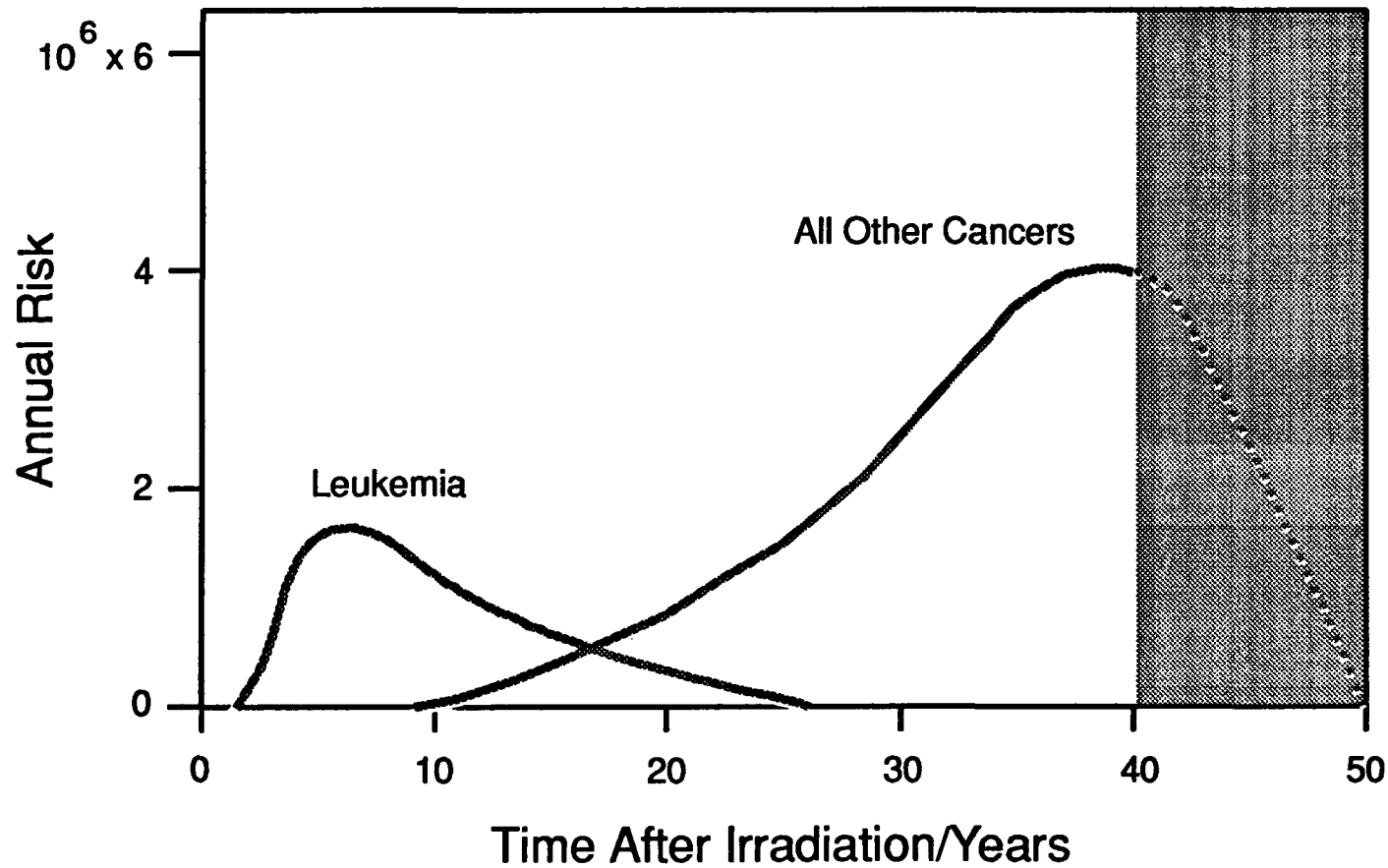
## **Medical Diagnosis**

**Multiple Fluoroscopies  
Pre-natal Irradiation  
Thorotrast Injections**

## **Occupational**

**Uranium Miners  
Radium <sup>226</sup> Ingestion**

# Risk of Cancer After 1 Rad, Whole Body Irradiation



## **From ICRP Publication 26 1977**

For the purposes of radiation protection involving individuals, the Commission concludes that the mortality risk factor for radiation induced cancers is about  $10^{-2} \text{ Sv}^{-1}$

(This value is rounded from the value of  $1.25 \times 10^{-2} \text{ Sv}^{-1}$  obtained from the average of  $1.5 \times 10^{-2} \text{ Sv}^{-1}$  for females and  $1.0 \times 10^{-2} \text{ Sv}^{-1}$  for males)

# **Epidemiological Information Since 1977**

- Update in Japanese A Bomb Survivors
- Update in Clinical Studies such as Ankylosing Spondylitis in U.K.
- International Cervix Series
- Other Clinical Updates, such as on Breast and Thyroid

## **New Information on Cancer Risk in the Japanese A-Bomb Survivors (Since 1977-80)**

1. Three new cycles of Japanese data,  
1975-78, 1979-82, 1983-85
2. Increase in excess solid tumors from  
 $\sim 100$  to  $\sim 300$
3. More information on age dependence
4. More information on time course
5. Revisions in the survivor dosimetry  
(DS86 vs. T65D)

## **Lifetime Risks in the Japanese (Preston & Pierce, RR, 1988)**

### **Population of All Ages**

---

Linear Extrapolation	solid tumors	11%/Sv
	leukemia	1%/Sv
	Total	12%/Sv
Nonlinear Extrapolation	Total	~ 5%/Sv

(DREF ~ 2.5)

### **Adult Population**

---

Nonlinear Extrapolation	Total	~ 3%/Sv
-------------------------	-------	---------

(DREF ~ 2.5)

# **UNSCEAR 1988**

## **Probability of Lifetime Excess Cancer Mortality**

High Dose, High Dose Rate

Population of All Ages (Japan)

Additive	4%/Sv
----------	-------

Multiplicative	11%/Sv
----------------	--------

Working Population

Additive	4%/Sv
----------	-------

Multiplicative	8%/Sv
----------------	-------

Low Dose, Low Dose Rate

Divide by DREF of 2 to 10



# **BEIR V 1989**

## **Probability of Lifetime Excess Cancer Mortality**

0.1 Sv U.S. Population (all ages)	0.8%
1 mSv/y Lifetime Exposure (all ages)	0.56%
10 mSv/y Exposure (age 18-65)	3.0%

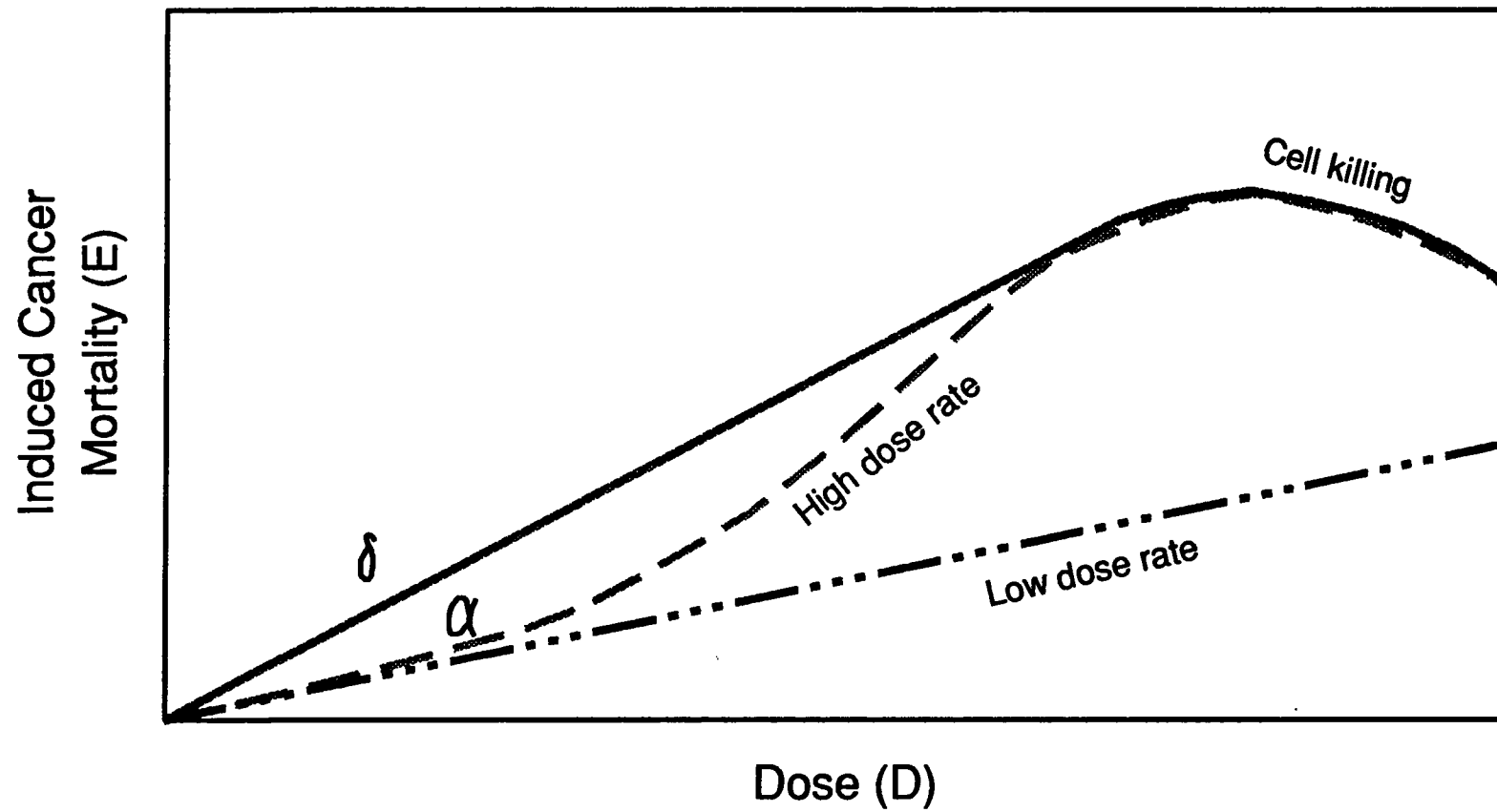
High Dose, High Dose Rate

Population of all Ages (U.S.)	9%/Sv
Working Population	

Low Dose, Low Dose Rate

Divide by DREF of 2 or more

$$E = \alpha D + \beta D^2$$



## Dose Rate Effectiveness Factors

UNSCEAR	1977	2.5
BEIR	1980	2.25
NCRP 64	1980	2-10
NIH	1985	2.3
UNSCEAR	1986	Up to 5
UNSCEAR	1988	2 to 10
BEIR	1989	2 or more
Human experience, breast, thyroid		~1- ~ 3
Hiroshima - Nagasaki	1987-89	
	Leukemia	~2
	Solid Tumors	~1

# **ICRP**

## **Risk Estimates for Radiation Protection**

High Dose, High Dose Rate

Population of All Ages

* Average UNSCEAR (mult)	11%	10%/Sv
--------------------------	-----	--------

Beir V	9%
--------	----

Working Population

UNSCEAR (multi)	8%/Sv
-----------------	-------

Low Dose, Low Dose Rate

Divide by DREF of 2

Population of all ages	5%/Sv
------------------------	-------

Working Population	4%/Sv
--------------------	-------

\* and others

# **Lifetime Mortality in 10,000 per Gy of Low Dose Rate Low-LET Radiation**

	ICRP 26	ICRP 1990
Bone Marrow	20	70
Bone Surfaces	5	5
Bladder	-	25
Breast	25	20
Colon	-	80
Liver	-	15
Lung	20	80
Oesophagus	-	30
Ovary	-	10
Skin	-	2
Stomach	-	105
Thyroid	5	8
Remainder	50	50
<b>TOTAL</b>	<b>125</b>	<b>500</b>

## **Tissue Weighting Factor ( $w_T$ )**

0.01 bone surfaces, skin

0.05 bladder, breast, liver, oesophagus, thyroid,  
remainder

0.12 bone marrow, colon, lung, stomach

0.20 gonads

---

1.00 whole body

## **ICRP Detriment (Low Dose)**

<b>Population of All Ages</b>	<b>per Sv</b>
<b>Serious Hereditary Disease</b>	<b>1%</b>
<b>Cancer Mortality</b>	<b>5%</b>
<b>Cancer Morbidity</b>	<b>1.5%</b>
	<hr/>
	<b>7.5%</b>
 <b>Working Population</b>	
<b>Serious Hereditary Disease</b>	<b>0.6%</b>
<b>Cancer Mortality</b>	<b>4%</b>
<b>Cancer Morbidity</b>	<b>1.2%</b>
	<hr/>
	<b>5.8%</b>

# **NCRP AND ICRP APPROACH TO SETTING DOSE LIMITS**

**AUGUST 1, 1990**

**Charles B. Meinhold  
Radiological Sciences Division  
Brookhaven National Laboratory  
Upton, New York**



## **ICRP PUB 26 (1977)**

- **Coherent system of dose limitation recommendations**
- **Total cancer and genetic risk to the first two generations (stochastic effects) must be compatible with the fatal accident rates in safe industries.**
- **Deterministic or nonstochastic effects must be avoided.**

## **ICRP PUB 26 (1977)**

- **50 mSv/yr (5 rem/yr)**
- **Since average exposure is  $<10$  mSv (1 rem)/yr, the average risk is  $<10^{-4}$ /yr.**
- **This is consistent with workers in safe industry.**

## **EFFECTIVE DOSE EQUIVALENT**

$$H_E = \sum_T w_T H_T$$

**Total risk for all stochastic effects**

$$\text{ALI}$$
$$H_{E,50} \leq 50 \text{ mSv (5 rem)}$$

### **Tritium**

$$\text{ALI} = 3 \times 10^9 \text{ Bq (80 mCi)}$$
$$H_{50} = 50 \text{ mSv (5 rem)}$$

### **Iodine-131**

$$\text{ALI} = 4 \times 10^6 \text{ Bq (100 } \mu\text{Ci)}$$
$$H_{50} = 2 \text{ Sv (200 rem)}$$

## **ALI**

**Therefore, a nonstochastic limit**

$$H_{50} \leq .5 \text{ Sv (50 rem)}$$

**Tritium  $3 \times 10^9 \text{ Bq (80 mCi)}$**

**Iodine-131  $1 \times 10^6 \text{ Bq (25 mCi)}$**

## **1977 - 1990**

- **Industry becoming safer.**
- **New Japanese survivor data.**
- **New risk projection models.**
- **Fetal risk concern.**

# **TRENDS WITH TIME IN FATAL ACCIDENT RATE**

**1957-1980**

	<b>Mean Rate over period (<math>10^{-6}y^{-1}</math>)</b>	<b>Annual Change of Rate</b>
<b>All groups</b>	<b>182</b>	<b><math>-2.4 \pm 0.1</math> (SE)</b>
<b>Trade</b>	<b>75</b>	<b><math>-2.3 \pm 0.1</math> (SE)</b>
<b>Manufacture</b>	<b>98</b>	<b><math>-1.6 \pm 0.2</math> (SE)</b>
<b>Service</b>	<b>114</b>	<b><math>-3.0 \pm 0.2</math> (SE)</b>
<b>Government</b>	<b>128</b>	<b><math>-1.2 \pm 0.2</math> (SE)</b>

# **TRENDS WITH TIME IN FATAL ACCIDENT RATE 1957-1980**

	Mean Rate over period ( $10^{-6}y^{-1}$ )	Annual Change of Rate
Transport & public utilities	362	$-1.2 \pm 0.3$ (SE)
Construction	677	$-1.4 \pm 0.2$ (SE)
Mines and quarries	940	$-2.5 \pm 0.5$ (SE)
Agriculture (1973-80)	561	$-0.3 \pm 1.0$ (SE)



**NCRP REPORT 91**

**RECOMMENDATIONS ON LIMITS FOR  
EXPOSURE TO  
IONIZING RADIATION**

**JUNE 1987**

## **OCCUPATIONAL EFFECTIVE DOSE EQUIVALENT LIMIT**

- **Risk of  $10^{-2} \text{ Sv}^{-1}$  ( $10^{-4} \text{ rem}^{-1}$ )**
- **Average annual dose equivalent = 2.3 mSv (230 mrem)**

**Therefore, 50 mSv (5 rem)**

## **OCCUPATIONAL DOSE LIMIT**

**But:**

- **Risk estimates are likely to increase.**
- **Safe industries becoming safer.**

## **OCCUPATIONAL DOSE LIMIT**

**First**

**Discontinue (Age-18) 5 rem**

**Second**

**Stresses upper boundary nature of  
dose limit**

**"It is only when the cost of further  
dose reduction is truly unreasonable  
that the limit should be approached."**

## **OCCUPATIONAL DOSE LIMIT**

### **Third**

**Operations should be aimed at keeping individual lifetime exposures in tens of mSv below his or her age in years.**

**For example: Worker at age 50 should have a lifetime dose equivalent of less than 500 mSv (50 rem).**

## **PROTECTION OF EMBRYO/FETUS**

- 1. Less concern about teratogenic effects.**
- 2. New concern about the developing central nervous system.**

**8-15 weeks =  $.4 \text{ Sv}^{-1}$  ( $4 \times 10^{-3} \text{ rem}^{-1}$ )**

## **EMBRYO/FETUS**

**Total = 5 mSv (.5 rem)**

**After diagnosis .5 mSv (.05 rem) in  
any month.**

## **EXPOSURE IN EXCESS OF THE LIMITS: OCCUPATIONAL**

- 1. Unlikely to affect lifetime risk.**
- 2. Suggests inadequate system of protection.**
- 3. Return to work based on improved control of workplace.**



## **SPECIAL DOSE EQUIVALENT LIMITS**

- 1. 5 rem (50 mSv) based on safe industries.**
- 2. Normally easily obtainable.**
- 3. Exceptions, i.e., space flight.**
- 4. New limits based on informed consent of workers and demonstrated need.**
- 5. Focus on lifetime risk.**

**ICRP**  
**1990 DRAFT**

## **DOSE LIMITS IN OCCUPATIONAL EXPOSURE**

**1977 Comparison with safe industry but:**

- 1. Standards for safe industries neither constant nor uniform worldwide.**
- 2. The mortality data apply to average while dose limits apply to the individual.**

## **CRITERIA**

**1977** Limit judged against the average  
**1990** Limit also judged against the  
maximum

**(Limit of Tolerability)**

<b>Unacceptable</b>	<b><math>10^{-3}</math></b>
<b>Tolerable</b>	<b><math>10^{-4}</math></b>
<b>Acceptable</b>	<b><math>10^{-5}</math></b>

## **MULTI-ATTRIBUTE (50 mSv) 18-65**

	<u>Additive</u>	<u>Multiplicative</u>
<b>Lifetime risk (%)</b>	<b>5.66</b>	<b>8.56</b>
<b>Loss of lifetime</b>	<b>20</b>	<b>13</b>
<b>Loss of life expectancy</b>	<b>1.12</b>	<b>1.11</b>
<b>Most probable age of attributable death</b>	<b>68</b>	<b>77</b>

## MULTI-ATTRIBUTE ANNUAL DOSE FROM 18-65

	10 mSv	20 mSv	50 mSv
<b>Lifetime risk (%)</b>	<b>1.8</b>	<b>3.6</b>	<b>8.6</b>
<b>Loss of lifetime</b>	<b>13</b>	<b>13</b>	<b>13</b>
<b>Loss of life expectancy</b>	<b>.23</b>	<b>.46</b>	<b>1.11</b>
<b>Most probable age of attributable death</b>	<b>78</b>	<b>78</b>	<b>78</b>

# **RECOMMENDED DOSE LIMITS**

## **Occupational**

**Effective Dose      100 mSv in 5 years**  
**50 mSv in any 1 year**

**Annual dose equivalent in**  
**the lens of the eye      200 mSv**  
**the skin (100 cm<sup>2</sup>)      500 mSv**  
**the hands and feet      500 mSv**

## **OCCUPATIONAL EXPOSURE OF WOMEN**

- **Not Pregnant - Same as men**
- **Pregnant - Limit dose equivalent to the mother's abdomen to 2 mSv (200 mrem)**



# **RECOMMENDED DOSE LIMITS**

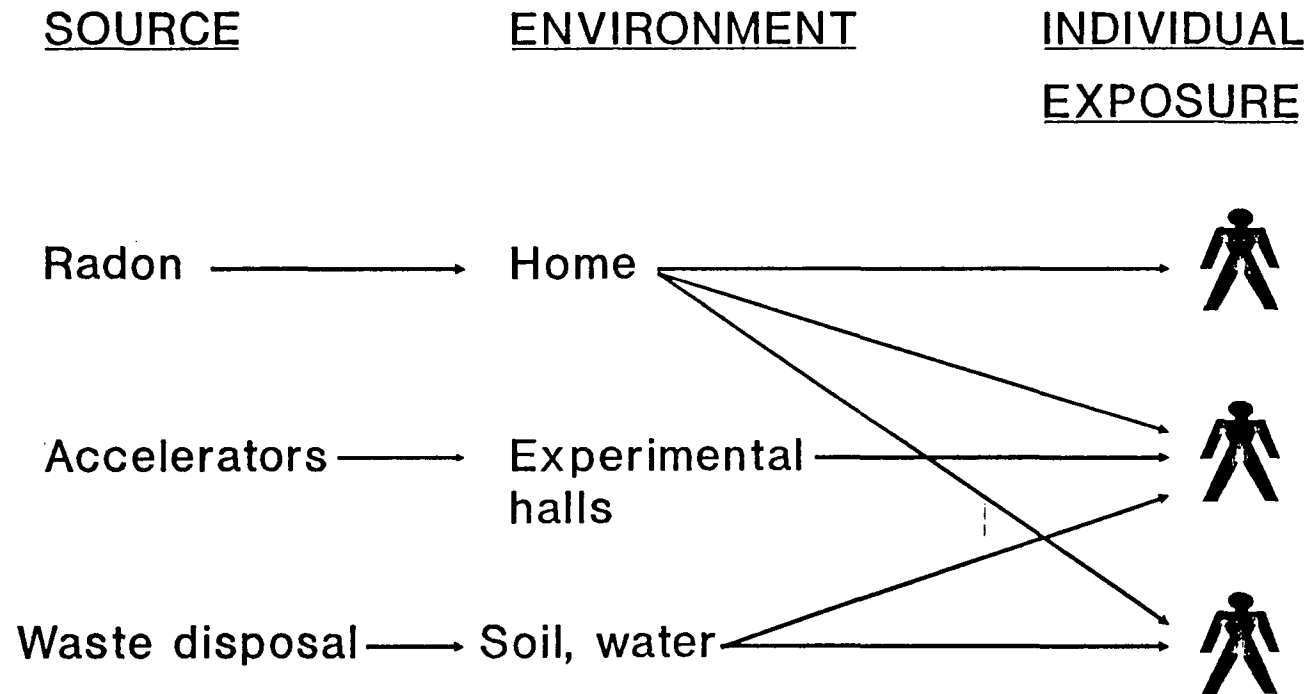
## **Public**

<b>Effective Dose</b>	<b>1 mSv per year, averaged over any 5 consecutive years</b>
-----------------------	--

### **Annual dose equivalent in**

<b>the lens of the eye</b>	<b>20 mSv</b>
<b>the skin (100 cm<sup>2</sup>)</b>	<b>50 mSv</b>
<b>the hands</b>	<b>50 mSv</b>

# NETWORK OF HUMAN EXPOSURE



## **CLASSIFICATION**

- **Practices (planned)**  
x-ray facilities, accelerators
- **Potential**  
waste disposal facilities, interlocked  
exposure facilities
- **Intervention (pre-existing)**  
radon in existing homes-post  
accident, etc.

## **PRACTICES AND POTENTIAL EXPOSURE REQUIREMENTS**

- **Justification of the practice.**
- **Optimization of the protection.**
- **Dose limit.**

## **INTERVENTION**

- 1. Justification - do more good than harm.**
- 2. Optimization - form, scale, and duration.**
- 3. Limitation - situation specific, intervention levels.**

**BRIEFING ON DEVELOPMENT OF REGULATORY  
STANDARDS FOR RADIATION PROTECTION**

**AUGUST 1, 1990**

**BILL MORRIS**

Contact: Bill Morris  
Phone: 492-3750

# **DEVELOPMENT OF REGULATORY STANDARDS FOR RADIATION PROTECTION**

- O MONITOR AND SUPPORT DEVELOPMENT OF  
SCIENTIFIC AND TECHNICAL INFORMATION**
  
- O MONITOR AND SUPPORT DEVELOPMENT OF  
GUIDANCE, RECOMMENDATIONS AND STANDARDS**
  - INTERNATIONAL COMMISSION ON  
RADIOLOGICAL PROTECTION**
  - NATIONAL COUNCIL ON RADIATION  
PROTECTION**
  - INTERNATIONAL ATOMIC ENERGY AGENCY**
  - ENVIRONMENTAL PROTECTION AGENCY**
  - COMMITTEE ON INTERAGENCY RADIATION  
RESEARCH AND POLICY COORDINATION**
  - FEDERAL GUIDANCE**

# **DEVELOPMENT OF REGULATORY STANDARDS FOR RADIATION PROTECTION (CONTINUED)**

- 0 EVALUATION OF OPERATING EXPERIENCE**
- 0 IDENTIFICATION OF POTENTIAL MODIFICATIONS  
TO REGULATIONS**
- 0 MODIFY NRC REGULATIONS AND REGULATORY  
GUIDANCE**
  - ANALYZE BENEFITS AND IMPACTS**
  - INCLUDE MARGINS TO ADDRESS  
UNCERTAINTIES IN IMPLEMENTING  
RECOMMENDATIONS**
  - ASSURE THAT REQUIREMENTS ARE  
REASONABLE, INSPECTABLE, AND PRACTICAL  
TO IMPLEMENT**



---

HEALTH EFFECTS OF  
EXPOSURE TO  
LOW LEVELS OF  
**IONIZING  
RADIATION**

BEIR V

Committee on the Biological Effects  
of Ionizing Radiations  
Board on Radiation Effects Research  
Commission on Life Sciences  
National Research Council

NATIONAL ACADEMY PRESS  
Washington, D.C. 1990

---

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## THE BEIR V REPORT

The BEIR V report updates earlier estimates of the risks of somatic and genetic effects of low-level irradiation, taking into account new information gained during the decade since completion of the BEIR III study.

For estimating the risks of carcinogenic effects, the BEIR V Committee placed primary reliance on the a-bomb survivor data, since they provide the only coherent body of information on the effects of a wide range of reasonably well quantified doses of whole-body radiation in persons of both sexes and all ages. For comparative purposes, data from other irradiated populations also were analyzed.

The a-bomb survivor experience was assessed with the use of a machine-readable database, obtained from RERF, comprising a total of 3399 records on survivors, grouped by age at exposure, time after exposure, DS86 dose, city, and sex. The kerma categories in the records were replaced by corresponding organ doses, and an RBE of 20 was assigned to the neutrons (other RBE values also were evaluated for their influence on the resulting risk estimates). The records were truncated at 4 Gy, data for higher doses being considered of doubtful reliability.

The rates of mortality from various forms of cancer were analyzed in relation to age at irradiation, time after irradiation, sex, dose, and other variables, with a view toward their compatibility with relative (multiplicative) and absolute (additive) risk models. Parameter estimates for these models were then derived from the data, and the results were evaluated for goodness of fit. To assure sufficient numbers of cases for adequate modeling, cancer deaths were ultimately combined into the following five diagnostic categories: "leukemia"

(excluding CLL), "cancer of the breast", "cancer of the digestive system", "cancer of the respiratory tract", and "other cancers".

Various exposure-time-response models were fit to the data, using the AMFIT program, which fits a general form of "Poisson regression" model. The observed number of events in each cell of the cross-tabulation were thus treated as a Poisson variate with parameters given by the predicted number of events under the model; i.e., the product of the person-years in that cell times the fitted rate. Although the BEIR III report included risk estimates for cancer derived by the additive projection model as well as by the multiplicative projection model, the BEIR V committee found neither model to fit the data unless modified appropriately for sex, age at irradiation, and time after exposure. Given such modifications, the multiplicative risk model was found to provide a more parsimonious description of the data and to be less subject to error resulting from misclassification of causes of death as discussed below. Hence the BEIR V Committee's preferred risk estimates (Tables 1,2) were based on the latter type of model.

For a given radiation dose equivalent  $d$  in sievert (Sv) the individual's age-specific cancer risk  $\gamma(d)$  was expressed as:

$$\gamma(d) = \gamma_0[1 + f(d)g(\beta)]. \quad (1)$$

where  $\gamma$  denotes the age-specific background risk of death due to a specific cancer for an individual at a given age -- which also depends upon the individual's sex and birth cohort (year of birth),  $f(d)$  represents a function of the dose  $d$  which is always a linear or linear-quadratic function; i.e.,  $f(d) = \alpha_1 d$  or  $f(d) = \alpha_1 d + \alpha_2 d^2$ . In general, the excess risk function,  $g(\beta)$  was observed to depend upon a number of parameters; for example, sex, attained age, age-at exposure, and time after exposure. The age-specific risk could also be

modeled as an additive risk:

$$\gamma(d) = \gamma_0 + f(d)g(\beta). \quad (2)$$

Although both models gave similar results, as expected -- since the function  $g(\beta)$  was allowed to depend on age, time, etc. -- this would not have been the case if  $g(\beta)$  were restricted to having a constant value other than for sex and age at exposure.

The models were fitted using maximum likelihood -- i.e., the values of the unknown parameters which maximized the probability of the observed number of cases (the "likelihood function") were taken as the best estimates, and, where applicable, confidence limits and significance tests were derived from standard large-sample statistical theory.

It was expected that the form of the background term might vary considerably between populations at risk and would not be of particular interest in terms of radiation risk. Hence the committee chose not to model it, but rather to estimate the baseline rate nonparametrically by allowing for a large number of multiplicative rate parameters, as is often done when fitting hazard models to ungrouped data.

Each model was then described by the Committee in terms of "point" estimates of its various parameters, their respective standard errors and significance tests, and an overall "deviance" for the model as a whole. Because of the extreme sparseness of the data, comparison of deviance to its degrees of freedom was not used as a test of fit of the model; however, since differences in deviance between nested alternative models (pairs of models for which all terms in one model were included in the other) have an asymptotic chi squared distribution, with degrees of freedom equal to the difference in the degrees of freedom between the models being compared, this test was used to

assess the improvement in fit as a result of adding terms to the dose-response function and used repeatedly by the committee to minimize potential over-specification of the models.

Approximate confidence limits on parameter estimates were constructed in the usual way, by adding and subtracting the standard error times 1.65 (for 90% confidence) or 1.96 (for 95% confidence). However, in cases where the Committee had reason to believe that the use of a normal distribution to estimate confidence limits was not valid, it reported "likelihood based" limits found by searching iteratively for the parameter values that led to a corresponding increase in the deviance.

For leukemia, the Committee's preferred model was a relative risk model (equation 1) with terms for dose, dose<sup>2</sup>, age at exposure, time after exposure (minimum latency of 2 years is assumed), and interaction effects. Between individuals exposed before age 20 and those exposed later in life, there appeared to be no effect of age at exposure but simply a different time pattern within each group; hence a simple step function was found to fit both groups rather well (splines can be used to smooth the transitions when desired--e.g., in the calculation of probability of causation).

The parameters for the leukemia model were as follows:

$$f(d) = \alpha_1 d + \alpha_2 d^2$$

$$g(\beta) = \begin{cases} \exp[\beta_1 I(T \leq 15) + \beta_2 I(15 < T < 25)] & \text{if } E \leq 20 \\ \exp[\beta_3 I(T \leq 25) + \beta_4 I(25 < T \leq 30)] & \text{if } E > 20, \end{cases} \quad (3)$$

where  $T$  is years after exposure,  $E$  is age at exposure, and the indicator function  $I(T \leq 15)$  is defined as 1 if  $T \leq 15$  and 0 if  $T > 15$ . The estimated parameter values and their standard errors, in parenthesis, are:

$$\alpha_1 = 0.243(0.291), \alpha_2 = 0.271(0.314),$$

$$\beta_1 = 4.885(1.349), \beta_2 = 2.380(1.311), \beta_3 = 2.367(1.121),$$

$$\beta_4 = 1.638(1.321).$$

The standard errors for the dose-effect coefficients, estimated by means of the likelihood method mentioned above, are both imprecise and highly skewed.

For all cancers other than leukemia, a 10-year minimum latency was assumed; this was done simply by excluding all observations (cases and person-years) less than 10 years after exposure. For purposes of overall nonleukemia cancer risk estimation, the committee simply chose to sum the risks of the components of the nonleukemia cancer group (i.e. respiratory cancer, digestive cancer, etc.), each of which was estimated by the models described below.

For cancer of the respiratory tract (ICD 160-163): The Committee's preferred model was a relative risk model (equation 1) with terms as follows:

$$f(d) = \alpha_1 d$$

$$g(\beta) = \exp[\beta_1 \ln(T/20) + \beta_2 I(s)], \quad (4)$$

where  $T$  = years after exposure and  $I(S) = 1$  if female, 0 if male, with  $\alpha_1 = 0.636(0.291)$ ,  $\beta_1 = 1.437(0.910)$ ,  $\beta_2 = 0.711(0.610)$ .

Under the Committee's model, the relative risk for this site decreases with time after exposure; i.e., the coefficient for time after exposure, -1.437, means that the relative risk decreases by a factor of about 5 over the period of 10-to-30 years post-exposure. The Committee noted that few data are available, as yet, on respiratory cancer among those exposed as children, and that the relative risk is 2 times higher for females (owing to their much lower baseline rates) than for males, although the observed absolute excess risks are similar.

When testing departures from a constant relative risk model, the addition of a parameter for time after exposure resulted in the greatest improvement in



describing the data, a finding consistent with the decreasing relative risk observed in the Ankylosing Spondylitis study (Da87), which influenced the Committee's choice of parameters. The inclusion of a parameter for sex did not greatly improve the model's fit to the data significantly but caused some improvement. There was no improvement when a term for age-at-exposure was added to the regression model, its value being sufficiently close to zero as to have no influence on the estimated risk.

For cancer of the female breast (ICU 174), the model was based on a parallel analysis of several cohorts. The important modifying factors were found to be age at exposure and time after exposure. The dependence of risk on age at exposure was complex, doubtless being heavily influenced by the woman's hormonal and reproductive status at that time. Lacking data on these biological variables, the Committee found that the best fit was obtained with the use of an indicator variable for age-at-exposure less than 16, together with additional indicator or trend variables depending on the data set. Both incidence and mortality models were developed. Although these differed, the highest risks were seen in women under 15-20 years of age at exposure. Risks were low in women exposed at ages greater than 40, suggesting that risks decrease with age at exposure. Finally, risks were found to decrease with time after exposure in all age groups.

The preferred model for breast cancer age-specific mortality (female only) was a relative risk model (equation 1) with terms as follows:

$$f(d) = \alpha_d$$

$$g(\beta) = \begin{cases} \exp[\beta_1 + \beta_2 \ln(T/20) + \beta_3 \ln^2(T/20)] & \text{if } E < 15 \\ \exp[\beta_2 \ln(T/20) + \beta_3 \ln^2(T/20) + \beta_4(E - 15)] & \text{if } E \geq 15, \end{cases} \quad (5)$$

where  $E$  is age at exposure and  $T$  is years after exposure with  $\alpha_1 = 1.220(0.610)$ ,  $\beta_1 = 1.385(0.554)$ ,  $\beta_2 = -0.104(0.804)$ ,  $\beta_3 = -2.212(1.376)$ ,  $\beta_4 = -0.0628(0.0321)$ .

For cancer of the digestive system (ICD 150-159), the most significant aspect of the LSS data was found to be the greatly increased risk (factor of 7) for those exposed under the age of 30, although the Committee had no explanation for it. There was no evidence of a significant change in the relative risk with time after exposure.

The Committee's preferred model is as follows:

$$f(d) = \alpha_1 d \quad (6)$$

$$g(\beta) = \exp[\beta_1 I(S) + \sigma E]$$

where  $I(S)$  equals 1 for females and 0 for males and

$$\sigma E = \begin{cases} 0 & \text{if } E \leq 25 \\ \beta_2(E - 25) & \text{if } 25 < E \leq 35 \\ 10\beta_2 & \text{if } E > 35 \end{cases}$$

with  $E$  = age at exposure. The estimated parameter values are  $\alpha_1 = 0.809(9.327)$ ,  $\beta_1 = 0.553(0.462)$ ,  $\beta_2 = -0.198(0.0628)$ .

For cancers other than those listed above (ICD 140-209 less those listed above), the excess was found to contribute significantly to the total radiation-induced cancer burden. Finer subdivision of the group did not, however, provide sufficient cases for modeling individual substituent sites. When attempted, the models were unstable, resulting in risk estimates for which there was little confidence. The general group of "other cancers" was reasonably fit by a simple model with only a negative linear effect by age-at-exposure at ages greater than 10. There was no evidence of either an effect by sex or by time after

exposure.

The preferred model is as follows:

$$f(d) = \alpha_1 d$$

$$g(\beta) = 1 \text{ if } E \leq 10 \text{ and } \exp [\beta_1 (E - 10)] \text{ if } E > 10, \quad (7)$$

where  $E$  = age at exposure and  $\alpha_1 = 1.220(0.519)$ ,  $\beta_1 = -0.0464(0.0234)$ .

As concerns the influence of dose rate on the carcinogenic effectiveness of radiation, the BEIR V Committee expressed the view that low-LET radiation can be expected to decrease in effectiveness when highly protracted, possibly by a factor of 2 or more for certain neoplasms, if the carcinogenic response of human tissues in the low-to-intermediate dose range is consistent with that which has been observed in experimental model systems (NCRP, 1980; UN, 1986). The Committee refrained, however, from specifying a precise value for the DREF, except in the case of leukemia, where its preferred linear-quadratic model (equation 3) contained an implicit DREF of approximately 2.

For heritable radiation-induced detriment (Table 3), the BEIR V Committee's risk estimates differ from those of the BEIR III Committee in including no allowance for effects on the incidence of multifactorial diseases, which were considered by the BEIR V Committee to be too uncertain to quantify. In other respects, the estimates of the two Committees were similar.

For radiation injury to the developing embryo the BEIR V Committee's risk estimates were larger than those of the BEIR III Committee, reflecting new information (Figs. 1-3) on the incidence and severity of mental retardation in prenatally irradiated a-bomb survivors.

Table 1. Estimated excess lifetime mortality from cancers of various organ systems after acute exposure to 0.1 Gy acute whole-body low-LET radiation, in relation to age at exposure and sex (from BEIR, 1989)<sup>a</sup>

<u>Males</u> (deaths per 10 <sup>6</sup> )							
<u>Age at Exposure</u>	<u>Total</u>	<u>Leukemia<sup>b</sup></u>	<u>Nonleukemia<sup>c</sup></u>	<u>Respiratory</u>	<u>Digestive</u>	<u>Other</u>	
5	1276	111	1165	17	361	787	
15	1144	109	1035	54	369	612	
25	921	36	885	124	389	372	
35	566	62	504	243	28	233	
45	600	108	492	353	22	117	
55	616	166	450	393	15	42	
65	481	191	290	272	11	7	
75	258	165	93	90	5	---	
85	110	96	14	17	---	---	
Average <sup>d</sup>	760	110	650	190	170	300	

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<u>Females</u> (deaths per 10 <sup>6</sup> )							
<u>Age at Exposure</u>	<u>Total</u>	<u>Leukemia<sup>b</sup></u>	<u>Nonleukemia<sup>c</sup></u>	<u>Breast</u>	<u>Respiratory</u>	<u>Digestive</u>	<u>Other</u>
5	1532	75	1457	129	48	655	625
15	1566	72	1494	295	70	653	476
25	1178	29	1149	52	125	679	293
35	557	46	511	43	208	73	187
45	541	73	468	20	277	71	100
55	505	117	388	6	273	64	45
65	386	146	240	---	172	52	16
75	227	127	100	---	72	26	3
85	90	73	17	---	15	4	---
Average <sup>d</sup>	810	80	730	70	150	290	220

<sup>a</sup> Based on a single exposure to radiation, and on a lifetable weighted average over each of the age groups listed, in a stationary population having U.S. mortality rates.

<sup>b</sup> Estimates for leukemia are based on the use of a linear-quadratic model and therefore include an implicit DREF of approximately 2.0. Estimates for solid tumors are based on the use of a linear model and therefore include a DREF of 1.0.

<sup>c</sup> Based on the sum of cancers of respiratory tract, digestive tract, breast, and other organs.

<sup>d</sup> Values rounded to nearest 10.

Table 2. Projected lifetime cancer mortality and associated loss of life expectancy from continuous whole-body irradiation in a population of both sexes (BEIR V, 1989).

	Exposure Throughout Life (1mSv/yr)	Exposure from Age 18 to Age 65 (10 mSv/Yr)
Excess Cancer Deaths		
No. per 10 <sup>4</sup>	56	320
% of normal	3	16
Loss of Life Expectancy (Yr)		
Average per person exposed	0.2	0.5
Average per excess death	17	16

(Calculations based on cancer and survival rates for the U.S. population and on use of the risk models presented in Table 10, which include an implicit DREF of about 2.0 for leukemia and DREF of 1 for solid tumors).

Table 3. Estimated genetic effects of 10 mSv (1 rem) per generation.

Type of Genetic Disorder	Natural Prevalence (per 10 <sup>6</sup> )	Additional Cases per 10 <sup>6</sup> Liveborn/rem/Generation			
		First Generation		Equilibrium	
		<u>BEIR III</u>	<u>BEIR IV</u>	<u>BEIR III</u>	<u>BEIR V</u>
Autosomal dominant and x-linked	10,000	5-65	6-35	40-200	100
Recessive	2,500	< 1	< 1	very slow increase	very slow increase
Congenital anomalies	20,000- 30,000	<10	-	slight increase	-
Chromosomal abnormalities	4,400	-	< 6	-	< 1
Subtotal		~10-70	~10-40	~40-200	~100
Multifactorial	650,000	-	-	20-900	-
Total	~700,000	~10-70	~10-40	~60-1,100	~100

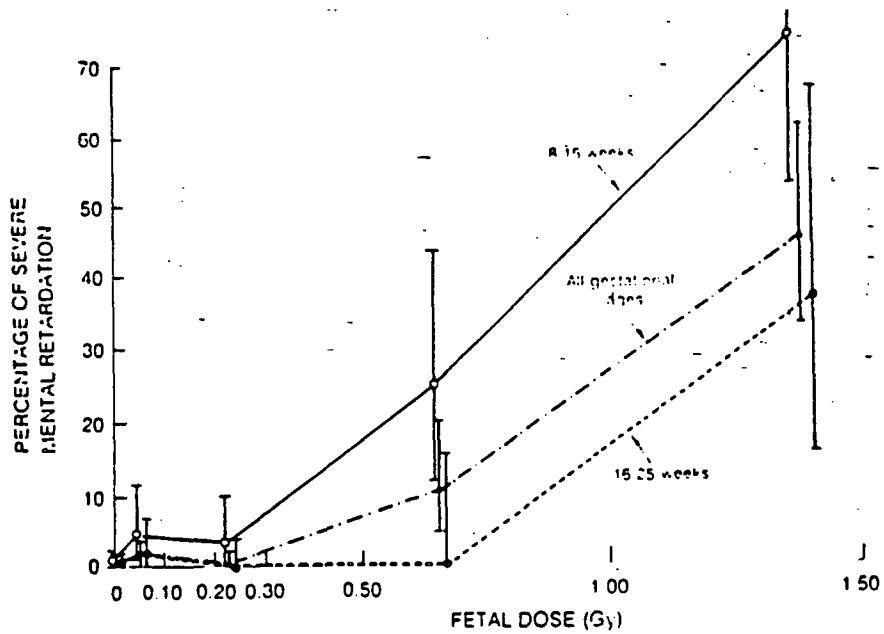


Figure 1. The prevalence of severe mental retardation among a-bomb survivors exposed in utero, by dose and gestational age, in Hiroshima and Nagasaki. The vertical lines indicate 90% confidence intervals.

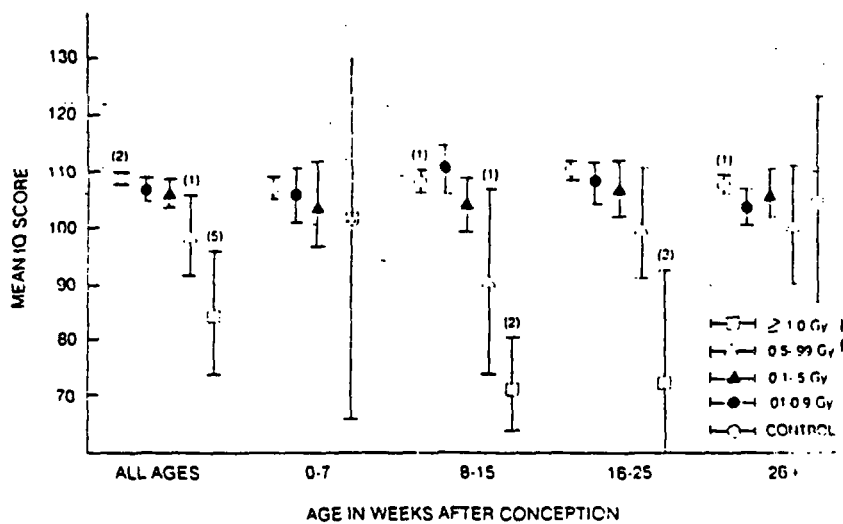


Figure 2. Mean IQ scores and 95% confidence limits, by gestational age in weeks and fetal dose, in a-bomb survivors exposed in utero in Hiroshima and Nagasaki. The numbers in parentheses are severely retarded cases, IQ  $\leq 64$ .



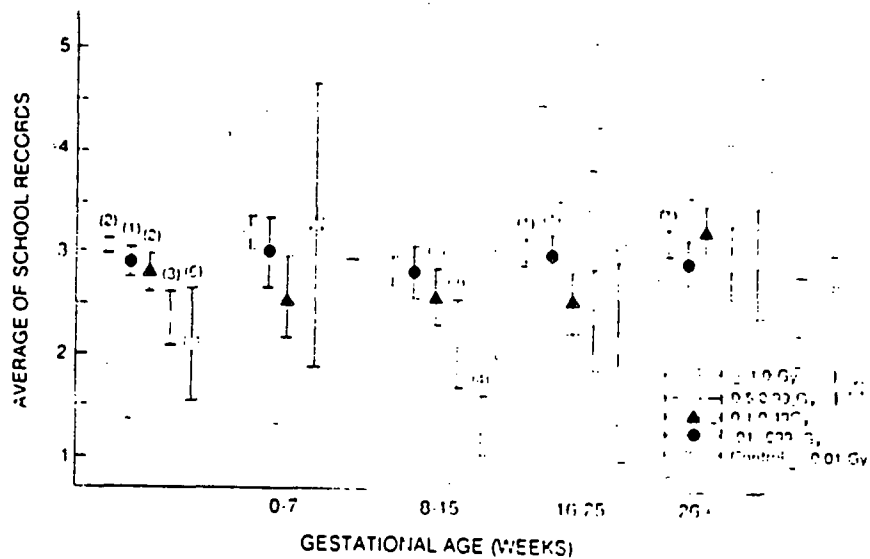


Figure 3. Average school subject score in the first grade with 95% confidence limits, by gestational age and fetal dose, in a-bomb survivors exposed in utero in Hiroshima and Nagasaki.

## ABSTRACT

### BEIR V Estimates of Excess Cancer Mortality

W. H. Ellett, D. G. Hoel, and R. D. Cooper

The BEIR V committee initially modeled each of the major types of cancer (leukemia, breast cancer, lung cancer, cancers of the digestive system, and a residual group "all other cancers") in terms of organ dose equivalent and a full set of modifying factors (e.g. sex, age at exposure, age at risk, and time after exposure). Modifying factors which proved not to be important for a particular cancer site were eliminated in subsequent models. In this way, a group of "preferred" relative risk models which include temporal factors were developed which can be used to calculate the risk of cancer mortality due to an exposure occurring at any age and time interval. This paper reports on the time and age dependence of the committee's risk models and presents risk estimates with their 90% confidence intervals for exposures occurring at various ages.

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BEIR V Estimates of Excess Cancer Mortality

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In 1986, a consortium of U.S. Federal Agencies asked the National Research Council to organize a new Committee on the Biological Effects of Ionizing Radiations (BEIR) to develop risk estimates de-novo for cancer due to low LET radiation.<sup>(1)</sup> The timing of this request was largely due to the assignment of new estimated organ doses to the individual A-bomb survivors in the Radiation Effects Research Foundation (RERF) Life Span Study. In addition, 11 more years of survivor mortality experience had occurred since collection of mortality data for the 1980 BEIR III report.<sup>(2)</sup> In this time interval, the number of deaths due to cancer had nearly doubled. The cohort of about 76,000 survivors having newly estimated doses was defined more rigidly than previously in that persons without significant shielding information were excluded. While this truncation of the cohort largely affected distal survivors who had small doses and contributed little, if anything, to the dose response, it also excluded survivors who were self reported to be in the open but had not received flash burns. In addition, persons who died at ages greater than 75 years were excluded from the committee's analyses as there is reason to believe the information on cause of death is considerably less reliable for this age group.

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The BEIR V Committee did not confine its data sources to Japan but also considered data bases for a number of other epidemiological follow-up studies carried out in Great Britain, Canada, Israel, and the United States. The availability of original data permitted the committee's risk estimates to be based on their own biometric analyses rather than on the conclusions reached by other investigators. Moreover, a variety of source materials allowed intercomparisons of results from studies of different ethnic groups. Nevertheless, with the exception of breast cancer, the committee's risk estimates of cancer mortality are largely based on the A-bomb survivor data (see footnote).

#### The Committee's Approach to Risk Modeling

Preliminary examination of the A-bomb survivor data indicated that, with the exception of leukemia, the dose response for mortality due to all cancers combined was linear at organ doses below 4 Sv but flattened out at larger doses. RERF investigators have attributed this decrease in effect per unit dose at high doses to large random errors in the doses assigned to heavily exposed persons,<sup>(3)</sup> but cell killing may also be a contributing factor. Because interests have centered on effects at low doses, the committee decided to limit the data set to organ doses below 4 Sv. This resulted in the exclusion of 2 cases of death from leukemia and 22 cases of death due to solid cancers. Results of tests made at various maximum dose levels are described in Table 1 where it is seen that the coefficient for dose square is negative and not statistically significant for all solid cancers combined. In contrast, the leukemia data showed a linear quadratic response when bone

marrow doses greater than 5 Sv were excluded. At higher doses, the response for leukemia decreases rather rapidly as has been observed in several animal studies.

The committee fitted a number of dose-response models to the mortality data for all solid cancers combined to test for the effect of such temporal modifying factors as age at exposure, age at risk, etc. Although most of these models gave about the same estimates of lifetime risk, none was really satisfactory. When all solid cancers were analyzed as a single group, the goodness of fit was not impressive and many of the models appeared to emphasize the risk to those exposed as children. When more detailed studies of cancer by site indicated there were often large differences among cancer types in their temporal responses, the attempt to model solid tumors as a group was abandoned. Although this move limited the precision of the final risk estimates, it was a wise choice. The average response over time often has little utility in formulating radiation control decisions or in deciding on compensation for possible radiation injury.

Nevertheless, modeling the dose response for individual cancers from a limited data set also has limitations. The committee fitted models to ten sites, or groups of sites, with the number of cancer deaths ranging from 34 to 2034. Clearly, the larger groups produced more stable risk estimates. In the end, a balance was struck between specificity and stability by modeling cancer mortality in just five groups: leukemia, breast cancer, cancers of the respiratory system, cancers of the digestive system, and a residual group of "all other cancers." While this is not an optimal grouping for calculating

probability of causation, it was considered to be the best that can be done at this time. No doubt, further follow up of the A-bomb survivors will allow greater specificity.

#### Time and Age Dependence for Cancers at Specified Sites

All of the models preferred by the committee are on a relative risk scale. In general, the radiogenic excess among survivors in the Life Span Study has increased over time much like the age-specific baseline rates among the unexposed. Relative risk models could be fitted with weaker modifying factors for age and time than absolute excess risk models and, judged on the basis of deviance, usually yielded a better fit to the data. Moreover, it is necessary to model the baseline cancer rate rather precisely when temporal factors are included in absolute risk models. The committee's experience indicated this can be an appreciable source of uncertainty in the final risk estimates.

On a relative risk scale, the committee's general model for age-specific cancer mortality is:

$$\lambda(d) = \lambda_0 [1 + f(d)g(\beta)]$$

where  $\lambda(d)$  is the age-specific cancer risk for an individual of a given age,  $\lambda_0$  is the age-specific baseline rate for a specific cancer,  $f(d)$  represents a linear or linear quadratic function of the dose equivalent, and  $g(\beta)$  depends on a number of modifying factors such as sex, attained age, age at exposure, and time since exposure. Models were fitted using maximum likelihood. For all cancers except leukemia, the best fit to data was provided by  $f(d) = \alpha_1 d$ . Addition of a quadratic term,  $\alpha_2 d^2$ , increased the initial slope as  $\alpha_2$  was invariably negative. In no case was  $\alpha_2$  statistically significant.

Details of the committee's model are presented in the BEIR V report.<sup>(1)</sup> Because the variation of  $g(\beta)$  with cancer types can be important in the design of efficient sampling strategies for dose ascertainment and risk projection, special emphasis is given to it here. For cancers of the digestive system, the relative risk is higher for females than males:

$$g(\beta) = \exp[\beta_1 I(S) + \delta_E]$$

where  $\beta_1$  is 0.55,  $E$  is age at exposure, and  $I(S)$  equals 1 for females and zero for males. There is also a marked decrease in risk for those individuals exposed at more than 30 years of age, i.e.  $\delta_E = 0$  if  $E \leq 25$ ;  $\beta_2(E-25)$  if  $25 < E \leq 35$ ; and  $10\beta_2$  if  $E > 35$ ; where  $\beta_2$  is nearly -0.2.

Similarly, there is a decrease in the relative risk for increasing age at exposure in the group "all other cancers."

$$g(\beta) = 1 \text{ if } E \leq 10 \text{ and } \exp[\beta_1(E-10)] \text{ if } E > 10 \text{ where } \beta_1 \text{ is } -0.046$$

The committee's preferred model for cancers of the respiratory system differs significantly from the usual model where the relative risk is modulated by age of exposure. Such models invariably project a very large lifetime risk for those exposed as children. Since A-bomb survivors who were exposed as children are just now entering the age range at which lung cancer occurs, it is obvious that such projections are based on the experience of older persons not those exposed as children. In contrast to the usual model, the one preferred by the committee has no parameter for age at exposure. In fact, when a term for age of exposure is added to this model, its maximum likelihood estimated value is so close to zero as to have no influence on the risk.

For cancer of respiratory system, the committee's model is:

$$g(\beta) = \exp[\beta_1 \ln(T/20) + \beta_2 I(S)]$$

where T is years after exposure and I(S) equals 1 for females, and zero for males. With this model, the relative risk decreases rapidly with time after exposure,  $\beta_1 = -1.44$ . Of all factors tested, the factor for time after exposure produced the greatest improvement in fit, as compared to a constant relative risk model. This improvement, however, was not significant at a 5% confidence level. The decision to include this factor was largely based on the decline in the relative risk of lung cancer observed in the Ankylosing Spondylitis study.<sup>(5)</sup> Future follow up will, no doubt, refine the estimate of  $\beta_1$  which has a large standard deviation,  $\pm 0.91$ . While the factor for sex,  $\beta_1 = .71$ , was also not highly significant, it did improve the fit of the model to the data.

The committee was fortunate in having four data sets for breast cancer mortality: the A-bomb survivors, irradiated postpartum mastitis patients, and Canadian and U.S. tuberculosis patients who received radiation to the breast for medical purposes. Although the exposure pattern for these cohorts ranged from high doses delivered nearly instantaneously to a rem or less every few weeks, there was no significant effect of dose rate. In contrast, experimental studies of breast cancer in rodents show a large dose rate effect. Obviously, care must be exercised in extrapolating animal data to radiocarcinogenesis in humans.



The time pattern of radiogenic breast cancer is complex, in all likelihood, because of the age variation in hormonal factors in the population at risk. The committee's model reflects this complexity in that age at exposure, E, and time since exposure, T, are strong modifiers of the relative risk.

$$g(\beta) = \begin{cases} \exp[\beta_1 + \beta_2 \ln(T/20) + \beta_3 \ln^2(T/20)] & \text{if } E \leq 15 \\ \exp[\beta_2 \ln(T/20) + \beta_3 \ln^2(T/20) + \beta_4(E-15)] & \text{if } E > 15 \end{cases}$$

where  $\beta_1$  is -1.4,  $\beta_2$  is -0.10,  $\beta_3$  is -2.2, and  $\beta_4$  is -0.062. The committee modeled both breast cancer mortality and incidence but found no evidence for an increased effect among those less than 10 years of age at exposure. The major breakpoint in age dependence appeared to be at about 15 years of age, but this could not be tested in detail as the data available to the committee were in 5-year intervals. In Figure 1, the projected breast cancer incidence pattern of U.S. women is shown as a function of attained age following an acute exposure.

Leukemia mortality also shows a marked reduction of risk with both increasing age at exposure and time after exposure. When appropriate temporal parameters are included, a relative risk model fits the leukemia data as well as an absolute risk model. Contrary to earlier speculations, leukemia mortality in the Life Span Study population of A-bomb survivors has not returned to baseline rates. Therefore, a lifetime risk plateau is assumed in the model. In keeping with some recent RERF studies of somatic mutations in lymphocytes, the committee found that dependence of relative risk on age of exposure, E, changed at age 20, about the same age at which the thymus stops producing T cells. There does not appear to be any effect of age at exposure within the two age groups,  $E \leq 20$  and  $E > 20$  years, but there is a different time pattern for the expression of leukemia within each age group.

$$f(d) = d + \alpha_2 d^2$$

$$g(\beta) = \begin{cases} \exp[\beta_1 + \beta_2 I(15 < T \leq 25) + \beta_3 I(T > 25)] & \text{if } E \leq 20 \\ \exp[\beta_1 + \beta_4 I(T \leq 25) + \beta_5 I(25 < T \leq 30) + \beta_3 I(T > 30)] & \text{if } E > 20 \end{cases}$$

where  $\alpha_2 = 1.120(1.082)$ ;  $\beta_1$  is  $3.442(1.016)$ ;  $\beta_2$  is  $-2.478(1.101)$ ;  $\beta_3$  is  $-4.857(1.343)$ ;  $\beta_4$  is  $-2.492(0.870)$  and  $\beta_5$  is  $-3.218(1.115)$ .

These equations are formally equivalent to the leukemia risk model given in the BEIR V report<sup>(1)</sup> but, at the suggestion of committee member Dr. Duncan Thomas, the excess risk in the period when the risk is highest, 2-15 years after exposure, rather than 30 years, is used as a baseline. Expressing the BEIR V leukemia model in this new format has several advantages. The estimates of the parameter values are more stable and inclusion of the coefficient of the dose term,  $\beta_1$ , in the exponential provides a better description of the skewed distribution of its possible values while also minimizing the correlation between the linear and quadratic terms. Although the step functions in the leukemia model may not be appealing on biological grounds, this model fits the A-bomb survivor data considerably better than the leukemia risk model in the BEIR III report.<sup>(2)</sup> In this regard, it should be noted that the committee used a number of diagnostic tests to examine the degree of correspondence between a given model and the data on which it is based, see Annex 4F of the BEIR V report.<sup>(1)</sup>

#### The Committee's Estimates of Lifetime Risk

The committee's risk estimates take into account only the excess number of deaths due to cancer, i.e. the difference between the number of deaths in an exposed and an unexposed lifetable population. In contrast, the 1988 UNSCEAR report<sup>(6)</sup> and the BEIR III report<sup>(2)</sup> calculate the lifetime excess risk somewhat differently in that the difference in cause specific death rates

between an exposed and unexposed population is applied to persons alive at a given age in an exposed lifetable population. In this way, the detriment due to fatal radiogenic cancers that occur earlier than nonradiogenic cancers is also taken into account. Both the excess death and the early death method are correct; the best measure of the detriment is probably somewhere between the two estimates they provide. Vaeth and Pierce<sup>(6)</sup> have pointed out that to a good first approximation the numerical difference in lifetime risk for these two methods is: early deaths equal excess deaths divided by  $1 - p_0$ , where  $p_0$  is probability of death due to cancer in an unexposed population having the same age distribution. For the 1980 U.S. stationary population used as a baseline in the BEIR V analyses,  $p_0$ , averaged over sex, is 0.202.

The BEIR V committee considered three exposure regimes in their lifetable calculations of lifetime risk: acute exposure, lifetime exposure from age 0 to 100 and continuous exposure from 18 to 65 years of age, Table 2. These calculations assume the same dose equivalent to all organs. The 90% confidence intervals for the point estimates of excess mortality in Table 2 were obtained by Monte Carlo sampling of the probability distributions of the parameters in the committee's equations for relative risk and over 10,000 successive lifetable calculations, one for each set of parameters. The confidence intervals for leukemia in Table 2 (and Table 3, below) are based on the renormalized model discussed above. The statistical uncertainty of the lifetime risks includes only that part of the uncertainty due to sampling variation. Other sources of uncertainty considered by the committee were: model misspecification, dosimetry errors, populations differences, and the possible effect of sex where this factor was not part of the model. It was judged that these factors would increase the 90% confidence intervals by a

factor of about 1.4.

#### Dose Rate Effects

The risk estimates for solid cancers presented in Table 2 do not include a dose-rate effectiveness factor (DREF). In some cases, depending on the LET of the radiation, use of such a factor may be necessary. The committee did not believe a DREF was justifiable for acute doses of either high or low LET radiation. However, for exposure to low LET radiations at low dose rates, the committee thought that the risks listed in Table 2 would be lower. Unfortunately, they were only able to quantify the DREF for low LET radiation as being in the range of 2 to 10. It is their belief that at this time there is no good scientific basis for selecting a particular value for solid cancers in humans. It should be noted that in the BEIR III report,<sup>(2)</sup> all of the linear quadratic-based risk estimates for solid cancers contained an implicit dose rate reduction factor of 2.5. This single factor accounts for most of the differences between the BEIR III committee's estimates of cancer risk based on relative risk and those of the BEIR V committee. Because the BEIR V committee's linear quadratic equation for leukemia risk implicitly includes a dose rate reduction factor of 2, the committee thought that no additional dose rate reduction factor was applicable to the risks estimates for leukemia.

#### Lifetime Risk by Age of Exposure and Type of Cancer

Even though the committee's models show less variation of the risk with age at exposure than constant relative risk models, considerable differences between age groups remain evident. This is illustrated in Table 3 for the acute exposures. As indicated in this table there is still considerable

statistical uncertainty in the risk estimates for those exposed at less than 20 years of age.

The division of the risk of cancer mortality between types of cancer for exposure from age 18 to age 65 is shown in Table 4. Because children are not included in this group, the relative prevalence of the various types of cancer differs from that given in the BEIR V report<sup>(1)</sup> for exposures occurring at all ages. The estimated risks for acute occupational exposures between the ages of 18 and 65 are large; a 10 mSv dose equivalent per year (one fifth of the legal limit) throughout a 47 year working life increases the baseline cancer risk by about 14% for U.S. males and about 17% for U.S. females.

When the difference between excess death and early death are taken into account, the BEIR V committee's risk estimates for acute exposures are not much different from those given in the 1988 UNSCEAR report<sup>(7)</sup> which are based on a constant relative risk model. Early deaths for the UNSCEAR model yields 1,070 deaths per  $10^4$  person Sv for the 1982 Japanese population, while for the 1980 U.S. stationary population, BEIR V yields 1,000 early deaths per  $10^4$  person Sv. In contrast, the current ICRP estimate is ten times smaller, 100 deaths per  $10^4$  person Sv.<sup>(8)</sup> It is apparent that the ongoing reevaluation of radiation risks by the ICRP is well warranted.

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Footnote on Page 2) Diskettes of the data from Life Span Study 9<sup>(4)</sup> as provided by RERF to the BEIR V committee are available from the Editorial Office, Radiation Effect Research Foundation, Hiroshima, 732, Japan (FAX 082-263-7279)

TABLE 1 Effect of Excluding High Dose Group on the Fitted Dose Response Relationship. Source: BEIR V(1)

Exclusion (Sv)	Linear Dose Coefficient per Sv	Test for Adding a Quadratic Term <sup>a</sup>
Leukemia		
None	0.58	.08
>5	0.76	1.76
>4	0.48	2.14
>3	0.25	1.45
>2	0.05	1.49
Solid Cancers		
None	0.78	-2.04
>5	0.82	-1.88
>4	0.97	-0.41
>3	0.98	-0.31
>2	1.14	0.66

<sup>a</sup>Signed square roots of Score Statistics for a test of the null hypothesis (no effect). These statistics are asymptotically distributed as standard normal deviates.



TABLE 2 Excess Cancer Mortality - Lifetime Risk per 100,000 Exposed<sup>(a)</sup>  
Source: BEIR V<sup>(1)</sup>

All Organ Dose Equivalent	Males		Females	
	Leukemia	Solid Cancers	Leukemia	Solid Cancers
0.1 Sv	110	660	80	730
All ages	(60-350) <sup>b</sup>	(420-1,040)	(50-230)	(550-1,020)
Acute				
1.0 mSv/yr	70	450	60	540
All ages	(20-230)	(320-830)	(20-180)	(430-800)
continuous				
10.0 mSv/yr	400	2,480	310	2,760
Age 18-65	(150-1,200)	(1,670-4,560)	(110-980)	(2,120-4,190)

<sup>a</sup>These risk estimates do not include a dose rate reduction factor, see text.

<sup>b</sup>90% confidence interval

TABLE 3 Excess Risk Estimates and 90% Confidence Intervals with the Preferred Models. (0.1 Sv acute exposure to 100,000 persons of each age and sex). Source: BEIR V<sup>(1)</sup>

Males			
Age at Exposure	Leukemia	All Solid Cancers	Breast Cancer
5	111 (33-655) <sup>a</sup>	1165 (673-1956)	
15	109 (35-640)	1035 (642-1775)	
25	36 (16-215)	885 (534-1442)	
35	62 (33-221)	504 (272-947)	
45	108 (60-315)	492 (257-883)	
55	166 (90-399)	450 (217-815)	
65	191 (97-436)	290 (137-572)	
75	165 (84-380)	93 (38-233)	
85	96 (49-221)	14 (5-44)	
Females <sup>b</sup>			
5	75 (24-455)	1457 (1001-2114)	129 (28-440)
15	72 (24-434)	1494 (1051-2095)	295 (112-652)
25	29 (14-179)	1149 (809-1527)	52 (10-144)
35	46 (26-182)	511 (315-819)	43 (9-132)
45	73 (42-214)	468 (277-776)	20 (-3-108)
55	117 (66-292)	388 (221-616)	6 (-8-71)
65	146 (76-333)	240 (134-426)	0 (-6-38)
75	127 (64-291)	100 (53-197)	-1 (-3-13)
85	73 (37-168)	17 (7-40)	-1 (-2-2)

<sup>a</sup>(5%, 95%) 200 replications.

<sup>b</sup>Unpublished committee calculations

TABLE 4 Excess cancer Mortality by Site for 100,000 persons exposed between age 18 and 65.<sup>a,b</sup>

10 mSv per year to all organs

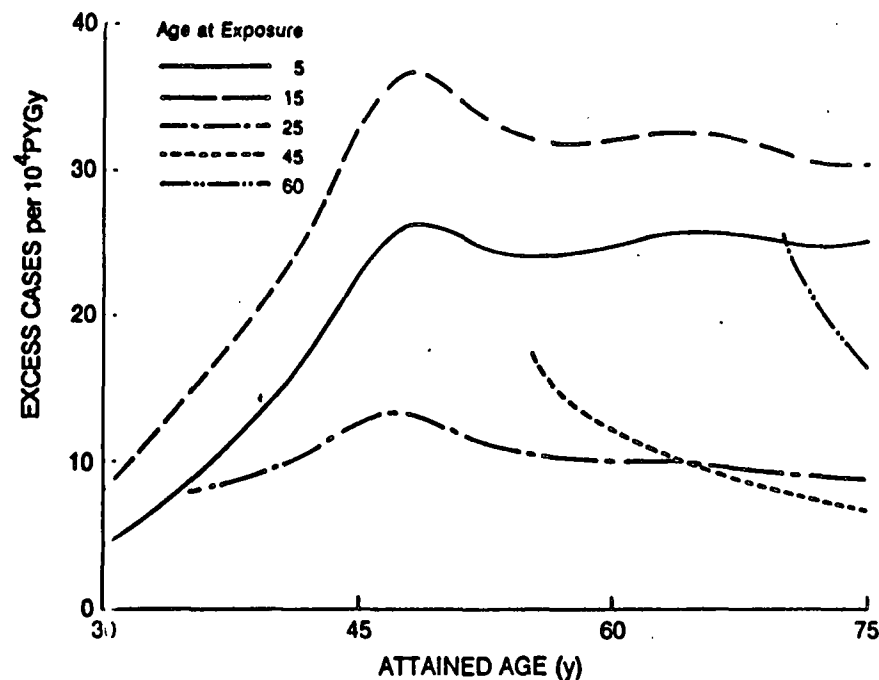
	Leukemia	Respiratory	Digestive	All Other	Breast
Males	394	1120	468	844	--
Females	306	935	945	713	132

<sup>a</sup>These risk estimates do not include a dose rate reduction factor.

<sup>b</sup>Unpublished committee calculations.

FIGURE 1. The projected incidence by age at exposure and attained age of radiogenic breast cancer following an acute dose of low LET radiation. Incidence decrease with attained age for women exposed after age 45. Source: BEIR V (1)

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