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ICRP Risk Estimates—An Alternative View

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ABSTRACT

For 58 years ICRP has served as the international source of information on risks of exposure to ionizing radiation and has provided recommendations for radiation protection. In general its publications have served a very useful purpose of reducing unnecessary radiation exposure but in some respects ICRP has delayed action to reduce excessive exposure, has underestimated radiation risks and has recommended radiation exposure levels that are much too high. For decades it showed concern to reduce exposure of doctors and nurses but ignored the principal source of population exposure, namely, patient exposure. Beginning in 1960 we became aware of two serious radiation exposure problems (occupational exposure in uranium mines and population exposure from testing of nuclear weapons). One might have expected ICRP to be the first to try to reduce these exposures but it was conspicuous by its silence. In 1958 ICRP set limits of exposure for radiation workers and members of the public. Nineteen years later (1977) when it was realized that the risk of radiation induced cancer was ten to thirty times what it was perceived to be in 1958, ICRP might have been expected to recommend a major reduction in permissible exposure levels, but to the dismay of some of us, it increased them. It was also a great disappointment when in 1977, levels of MPC or radionuclides in air, water and food were increased for a large fraction of the more dangerous radionuclides. The reactor accident at Chernobyl calls for a number of new ICRP recommendations. When can we expect them?

The International Commission on Radiological Protection, ICRP, has been in existence for almost 60 years, beginning under the name, International X-ray and Radium Protection Committee (IXRPC) in 1928 when it was formed as

a committee of the Second International Congress of Radiology, ICR.[1] This Committee operated with seven members for nine years until 1937 and during this period formulated recommendations on protection from ionizing radiation that were based on earlier recommendations published by the British X-ray and Radium Protection Committee in 1921. During this period a principal concern was protection of the radiologist and his staff. The International X-ray and Radium Protection Committee of the ICR ceased to function during the Second World War years, 1937–1950, and was reorganized with new members and in most respects as a new organization with the name, International Commission on Radiological Protection, ICRP, in 1950.

During the latter part of the doldrum period of IXRPC, 1943 to 1950, there were many publications dealing with protection from ionizing radiation by health physicists and radiobiologists working on the nuclear weapons programmes at Harwell, England, Chalk River, Canada and the US National Laboratories but most of these were in-house classified reports until a few years after the war ended. A large fraction of the members of the ICRP Main Commission and its Committees in the revival period of ICRP (1950–1960) were associated with these laboratories and were joined on the ICRP by medical doctors from these and other countries.

Through the years the ICRP has served as the principal source of information on risks of exposure to ionizing radiation and since 1950 has provided extensive recommendations that have been of assistance to the countries of the world in setting their radiation protection standards, rules and regulations. Some countries have accepted the ICRP recommendations without question as though they were Gospel truth or infallible. Perhaps in most cases they were wise in this reliance, but in some respects I believe ICRP has not met their expectations or justified unqualified acceptance of its recommendations. For this reason I believe it might be helpful to look at what we might consider have been some of the shortcomings of the ICRP, and call attention in a constructive vein to some cases where it was at fault for ignoring radiation exposure problems and to others where it made bad recommendations. Others on this programme are scheduled to provide balance to this topic by enumerating some of the successful accomplishments of ICRP so I leave this discussion to them. Some of the early mistakes of ICRP were reflections of the misconceptions of the science of the times and the fault of ICRP was that perhaps it should have been a bit more ahead of its time. The early publications of the International X-ray and Radium Protection Committee[1] were in various journals, mostly the *British Journal of Radiology*. The ICRP now has in preparation its fiftieth handbook; the first of these, ICRP-1, was published in 1959.

ICRP's greatest mistake in the early period resulted from the false belief of many of its members that low-level exposure is harmless and to many the term tolerance dose connoted a safe dose well below the threshold at which any

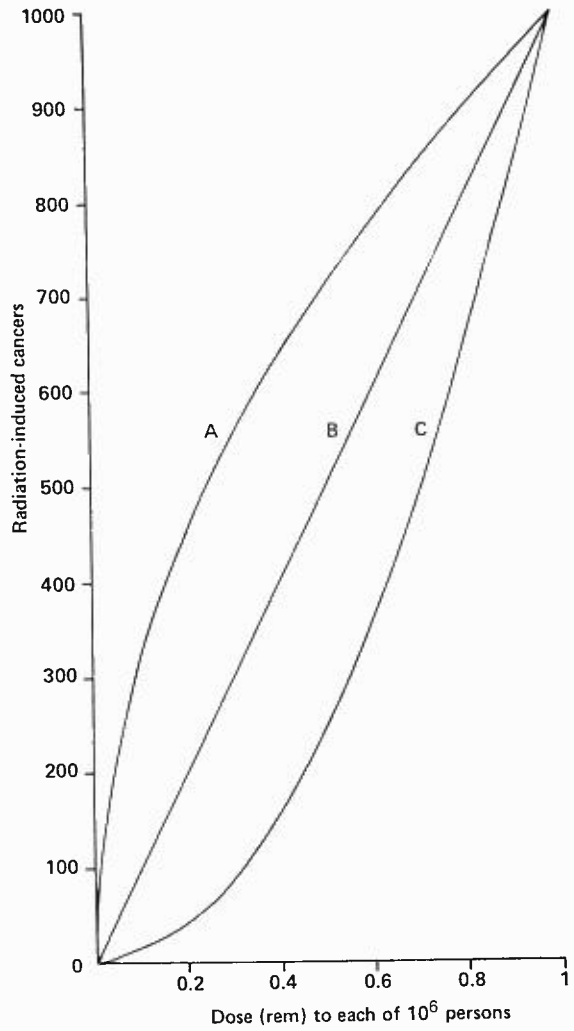


Figure 1 Plot of equation $C = \sigma N D^n$

C = cancer deaths from dose D
D = dose (rems) to each person
N = number of persons = 10^6 in this case
σ = fatal cancers/person rem
$n = 1$ for linear hypothesis, Curve B
$n > 1$ for threshold hypothesis
$= 2$ in Curve C
$n < 1$ for supralinear hypothesis
$= \frac{1}{2}$ in Curve A

harm would ever result. During the years that followed animals and human studies indicated this to be a bad assumption and so the threshold hypothesis was discarded in favour of the linear hypothesis although some of the ICRP publications left the reader with the impression this certainly was a most conservative assumption that without doubt greatly overestimated the risk. Today ICRP is at another crossroad pointing clearly in the direction of the supra-linear hypothesis, which not only disclaims a safety factor associated with the application of the linear hypothesis, but asserts that it under-estimates the cancer risk. The three hypotheses can be expressed in a simplified form by the equation, $C = \sigma D^n$, in which C is the radiation-induced cancer incidence or mortality, σ is a constant referred to as the cancer coefficient and D is the dose in rems. If n is greater than 1, we have the threshold hypothesis; if it is equal to 1, we have the linear hypothesis and if it is less than 1, we have the supralinear hypothesis. In many cases of cancer induction, $n = \frac{1}{2}$ or the cancer risk increases with the square root of the dose so that more cancers are produced per rem at low doses than at high doses. Figure 1 illustrates the three hypotheses. Although the ICRP and other agencies frequently are forced to mention results of epidemiological studies where the cancer risk versus the dose of ionizing radiation conforms best with the supralinear theory, they seem to do so reluctantly implying that something most certainly is wrong with such data. For example, the US General Accounting Office[2] formed an expert committee to evaluate the risks of low-level exposure and it concluded, after examining studies of cancer incidence among patients with ankylosing spondylitis who had developed cancer following x-ray medical treatments, that

> Both mixed models tested did much better than the linear model and the unusual square root-cubic model did the best of all. Since the cubic term is negligible at low doses, this last model has a faster than linear growth in leukemia risk for very low doses of X-rays.

The ICRP, UNSCEAR, BEIR-III and other groups are quick to devalue or criticize studies which lend support to the supralinear hypothesis such as the *in utero* studies of Alice Stewart, the tinea capitis studies of B. Modan *et al.* or the Hanford radiation worker study of T. F. Mancuso, A. Stewart and G. Kneale, but they wait for years until they are forced to acknowledge the more obvious and serious flaws in their inspired, irrefutable hallmark, the study of survivors of the atomic bombing of Hiroshima and Nagasaki. They invent all sorts of explanations as to why the former studies are not reliable or admissible in determining σ, the cancer coefficient (σ = cancer deaths per person rem) but fail to recognize the shortcomings of the Japanese study. Some reasons why the study of survivors of bombings of Hiroshima and Nagasaki underestimate cancer risk are:

(1) The total dose estimates were too high, thus σ was underestimated

(2) The neutron dose, especially at Hiroshima, was lower than estimated, thus σ was underestimated

(3) Some evaluations use the low dose group as controls. On the supra-linear hypothesis this could greatly underestimate σ.

(4) It was not a normal population.[3] The bomb survivors had been exposed to fire, blast, deprivation, psychological damage and severe damage to their immune systems so the weaker persons with less resilience died of a variety of common diseases. On the other hand, those of superior stamina or the 'healthy survivors' had a lower than normal death rate five years after the bombing when the epidemiological studies got underway. Thus, as explained by A. M. Stewart,[4] the two effects tended to neutralize each other and when the survivor study began, the death rate appeared normal except for cancer but the population most certainly was far from normal. Thus the cancer incidence was suppressed.

(5) There still is an above normal cancer rate among the survivors and this will continue to increase the value of σ.

In spite of very limited knowledge about the long-term hazards of exposure to low levels of ionizing radiation in the early period (1950–60), I believe in many respects the relative level of excellence as measured by the quality of performance of the Main Commission of ICRP was higher than since then, especially if one takes into account the fact that during this early post-war period ICRP was a pioneer breaking new ground. Certainly much of this credit or blame depends on the stature of the thirteen members comprising the Main Commission of ICRP. I believe it would be difficult to contemplate finding men of less bias and higher qualifications, for example, than Sir Ernest Rock Carling, W. Binks and M. V. Mayneord of the UK, A. J. Cipriani of Canada, R. M. Sievert of Sweden, and G. Failla and H. J. Muller of the US.

Perhaps one of the greatest weaknesses of ICRP stems from its process of nominating and electing members to the Main Commission; however, I must be quick to say it is difficult to think of a perfect solution. The nomination and election processes are flawed because they invite bias and appointment of members who have a conflict of interest and tempt some to make this a lifetime profession assuring them wide political recognition as an authority on radiation protection. In the first place, ICRP functions under the auspices of the International Congress of Radiology, ICR. Possible conflict comes here from the fact that ICRP is set up supposedly to reduce non-beneficial radiation exposure, yet the greater the number of radiation diagnostic procedures and the more routine and assembly-line style in which medical X-rays are administered, the greater the demand for radiology. In many cases this leads to X-rays that are not necessary[5] and to administrative rather than medical requirements for X-rays. On the other hand, when ICRP began in 1928, radiologists comprised the segment of the population with the largest exposure

to ionizing radiation and the greatest number with reported radiation injuries. They knew more about its uses, its measurement and its control than any other group. More importantly, the ICR was the first and only international professional organization sufficiently concerned to form such a protection committee and finance its operation. Some of the national society affiliates that comprise the ICR, however, have done more to increase unnecessary patient exposure to X-rays than to minimize it. For example, some of us worked for many years to do away with the mass chest X-ray programme in the schools in the US but we only got negative support of our national radiological societies or the ICR. In this programme buses with photo-fluorometric X-ray equipment would pull up to a school each year and the children were marched through to have a chest X-ray. It would have been better had they instead been branded with a sizzling Texas branding iron because measurements made by my group at Oak Ridge National Laboratory of a number of these devices in use indicated surface doses per X-ray ranging between 2000 and 3000 mR while the average chest X-ray dose at my facility (Oak Ridge National Laboratory) was only 15 mR. Finally, years after the US Surgeon General repeatedly urged a discontinuance of these programmes and after he indicated they had not been finding cases of TB, these programmes were done away with in the US.

Another example of the low priority the American radiological societies and the American College of Radiology, ACR, have given to radiation protection is their reluctance to endorse and failure to make use of the Ten-Day-Rule. This ICRP Rule stated that diagnostic X-rays to the pelvic and abdominal region of women in the child-bearing age should be delayed in most cases and given during the 10-day interval following the beginning of menstruation unless such delay would be harmful to the woman. Dr Muller and I had worked long and hard for ICRP to adopt this Rule and we were delighted when it was adopted by ICRP at the 1962 London meeting. Our delight, however, was short-lived and somewhat impaired when we returned to the US and read in the Bulletin of the ACR that this was a bad Rule and it had been unsuccessfully opposed by two of the members of ICRP, L. S. Taylor and R. S. Stone. It is true this Rule adds to the complexities of operating a radiology department like a factory assembly line and means rescheduling of many X-rays but I believe the unborn child deserves this extra inconvenience and consideration. I have been very disappointed in recent years that ICRP has weakened its stand on the Ten-Day-Rule.

ICRP has not taken full advantage of the findings of Alice Stewart and G. Kneale[6] in their Oxford Studies of *in utero* exposure. It is not to ICRP credit that the permissible likely occupational exposure of pregnant women was decreased from the 1962 value of 1866 mrem[7] only to 1708 mrem[8] in 1977.* I believe this 1708 mrem is far too high. This would correspond to about six

*In 1962 ICRP-6 permitted exposure of 1300 mrem in 13 weeks or an average of 866 mrem in first two months of pregnancy and 1000 mrem in the last seven months or a total of 1866 mrem. In 1977 ICRP-26 permits exposure of 5000 mrem in a year or 833 mrem in first two months and exposure at the rate of 0.3×5000 mrem/year for the last seven months or a total of $833 + 875 = 1708$ mrem. Both ICRP-6 and ICRP-26 do not actually prohibit the woman receiving exposure of 5000 mrem during the first two months of pregnancy.

of the typical pelvimetries delivered during the period of the Oxford Study[9] and ten times the normal risk that the child will die of cancer in early childhood. It is probably true very few mothers would be so calloused as to willingly allow this likely occupational exposure of 1708 mrem to their unborn children but many radiation workers are not aware of the serious warning given us by the Stewart data and certainly many nuclear industries would just as soon this information were not publicized. Even worse, there is nothing in the ICRP recommendations to deter the nuclear industry from allowing the young woman to receive the full 5000 mrem during the two months before pregnancy is recognized (i.e. three times the above risk estimates).

One of the weaknesses of ICRP is in their rules of turnover of membership on the thirteen member ICRP Main Commission. The rules specify not less than two or more than four members shall be changed at each meeting of ICR (every three years) and there is no restriction regarding one's tenure on the Main Commission. Several members have been on the Commission more than twenty years and the average turnover has been 3.7 members every three years. I believe it would be a big improvement to change the rules to require a turnover of not less than four or more than five every three years and have a maximum tenure of nine years. Selection of new members is made every three years by the thirteen member Main Commission from nominations submitted to it by National Delegations to the ICR and by the thirteen member Main Commission members themselves. This has resulted in a self-perpetuated body. I am confident there are several ways in which this election process could be improved. The ICRP has a number of active committees which it appoints from time to time and these usually comprise fifty or more persons in addition to the thirteen members on the Main Commission. Perhaps they too should submit nominations for the Main Commission membership and they, plus the thirteen members of the Main Commission, could vote every three years on the membership. Only Committee members on committees that have been active during the three-year period should have a vote. I am sure such a change would not solve all the membership problems but I believe it would place more qualified persons on the Main Commission to respond to needed or current projects of the Commission. It would more likely result in having certain disciplines properly represented. It would bring in highly qualified scientists from countries seldom, if ever, represented and hopefully it would lessen the chance of special interest groups such as from radiology or the nuclear energy industry having excessive influence. It might lessen the number who have a conflict of interest in reference to current projects of the ICRP or bring to the

top of the agenda new areas where ICRP should operate. I believe there have been two groups excessively represented on the Main Commission of ICRP that have a strong interest in depreciating the harmful effects of low exposure to ionizing radiation. These are persons wishing no restrictions on dose from excessive use of diagnostic X-rays and those with the nuclear establishment (employees of National Laboratories and, with industry and government agencies, responsible for promoting the development of nuclear weapons or supporting nuclear power). These groups need representation but I would like to see them counterbalanced by persons such as, for example, Drs Alice Stewart, J. Rotblat, B. Modan and Frank von Hippel, just to name a few of many who are well qualified.

I believe since ICRP has been considered by many as the most reliable and the ultimate authority on radiation protection for sixty years, its failure to address and try to correct a situation of high, unnecessary radiation exposure must be considered a public disservice. I will mention a few of these faults of omission in the following as typical examples:

(1) In the first period of operation of ICRP, X-ray technicians were instructed in their training classes and in their textbooks[10] to give larger X-ray doses to black people. The General Electric Company's X-ray department recommended in their technique charts for X-ray technologists that they give higher doses to blacks and the textbook, *X-Ray Technology*, by C. A. Jacobs and D. E. Hagen recommended doses to blacks that were higher by 40 to 60 per cent. Why did ICRP remain silent?

(2) The excessive doses delivered in the mass chest X-ray programme to millions of children went on for many years. The dose per X-ray could have been reduced by a factor of 200 by the use of better equipment but dollars were more important than children's lives. Why was ICRP silent on this issue?

(3) During the period of 1960–65 there were many papers published indicating the serious risk of lung cancer among underground uranium miners in the Colorado Plateau region of the US. There were several US Congressional hearings in which many scientists testified—some for reducing the maximum permissible working level in the mines while others urged the level not be reduced. It was no surprise that the US Atomic Energy Commission (USAEC) opposed any reduction in the permissible working level (WL) but I was disappointed that the US Public Health Service and the US Federal Radiation Council (USFRC) joined with the USAEC in opposing any reduction. Table 1[11] indicates the number of men employed in uranium mining in 1954–66 and the high percentage of underground mines operating at very high working levels in 1956–9. Note that for these years only 18 to 28 per cent were operating

Table 1

Estimates of the number of mines producing uranium ore during the calendar year as reported by the industry to the US Bureau of Mines (1954–64) and AEC (1965–66)*[a]

Year	Underground mines	Open pit mines
1954	450	50
1955	600	75
1956	700	100
1957	850	125
1958	850	200
1959	801	165
1960	703	166
1961	497	122
1962	545	139
1963	573	162
1964	471	106
1965	562	74
1966	533	88

Number of men employed in uranium mines[a]

Year	Underground[b] mines	Open pit mines
1954	916	53
1955	1376	293
1956	1770	584
1957	2430	574
1958	2796	1175
1959	3996	1259
1960	4908	1499
1961	4182	1047
1962	4174	1074
1963	3510	886
1964	3249	726
1965	2900	700
1966	2545	359

Estimated distribution of mines by Working Level ranges from 1956 to 1959

Year	Number mines measured	<1.0 WL (%)	1.0–2.9 WL (%)	3.0–10.0 WL (%)	>10.0 WL (%)	Total (%)
1956	108	19	25	33	23	100
1957	158	20	26	28	26	100
1958	53	28	21	36	15	100
1959	237	18	26	28	28	100

[a]Published by the US Federal Radiation Council as report on, *Guidance for Control of Radiation Hazards in Uranium Mining*, Report No. 8 (revised), Sept. 1967.
[b]Excludes above-ground employees who may occasionally go underground.

at a level less than 1 WL ($\sim 10^{-7}$ μCi/cc of RN-222) while ICRP-2 handbook (1959) gave 3×10^{-8} μCi/cc (~ 0.3 WL) as the maximum permissible concentration (MPC) for Rn-222. Figure 2[11] indicates how the cancer risk increased with working level months (WLM). Fortunately after all this discouragement an honest government official turned up in Washington, Secretary of Labor, Mr Wirtz, and he unilaterally set the level at 0.3 WL or $0.3 \times 12 \simeq 4$ WLM (working level months per year). But where was ICRP all this time? It was not until 1977 that ICRP-24,

Radiation Protection in Uranium and Other Mines[12] was published. Surely it should not have taken twenty years for ICRP to decide this was a very serious radiation problem and come to our assistance? One might have expected this to be one of ICRP's first handbooks, warning of the risks of Rn-222 and its daughter products in underground mines. This

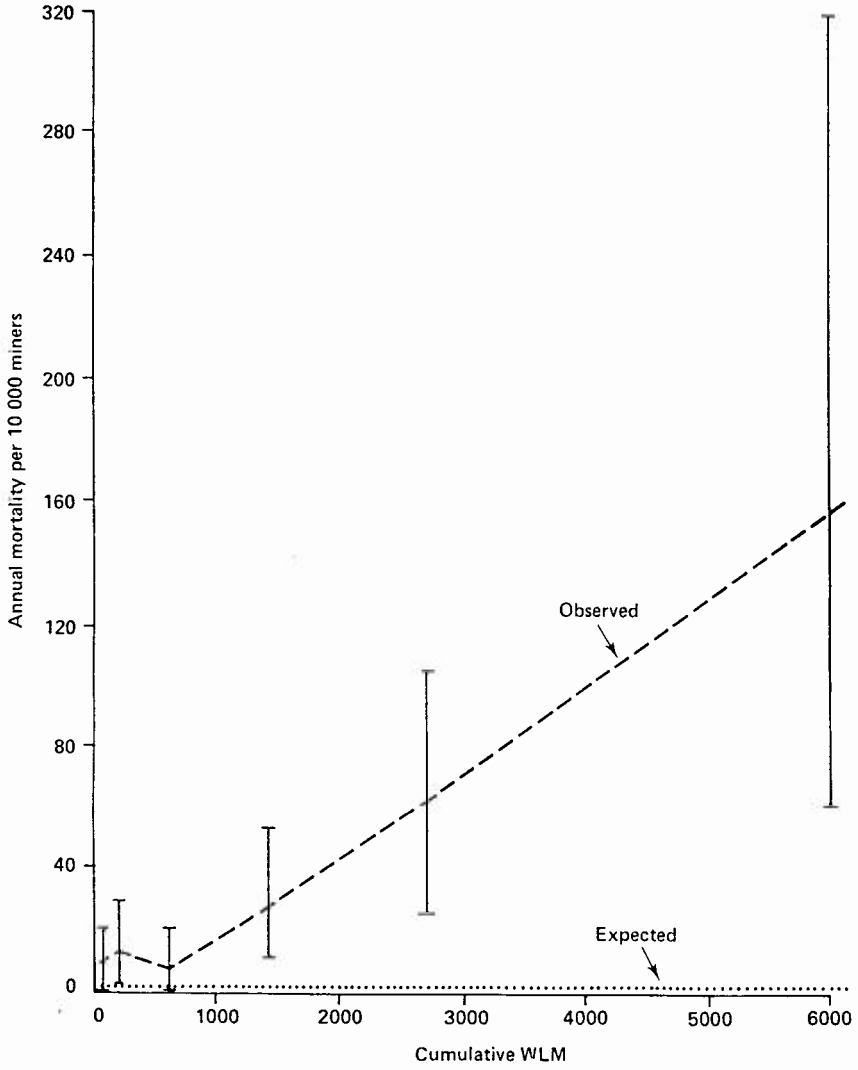


Figure 2 Observed and expected annual lung cancer mortality per 10 000 miners and 95 per cent confidence limits in relation to exposure. (From report of the US Federal Radiation Council titled, '*Guidance for the Control of Radiation Hazards in Uranium Mining*, No. 8 (revised), Sept. 1967.

hazard had first been recognized over 500 years ago when miners in the Schneeberg cobalt mines of Saxony and the Joachimsthal pitchblende miners of Bohemia were dying of a miners' disease, now known as radiation-induced cancer. It is good to have ICRP come in and give support years after a battle is won but it would have been so helpful to have its support to expedite and help the battle earlier.

(4) In the discussion above it was mentioned that ICRP operating under support of the ICR showed great interest in preventing excessive occupational exposure to the radiologists and their staff but dragged its feet in publishing the first comprehensive reports on protection of the patient.

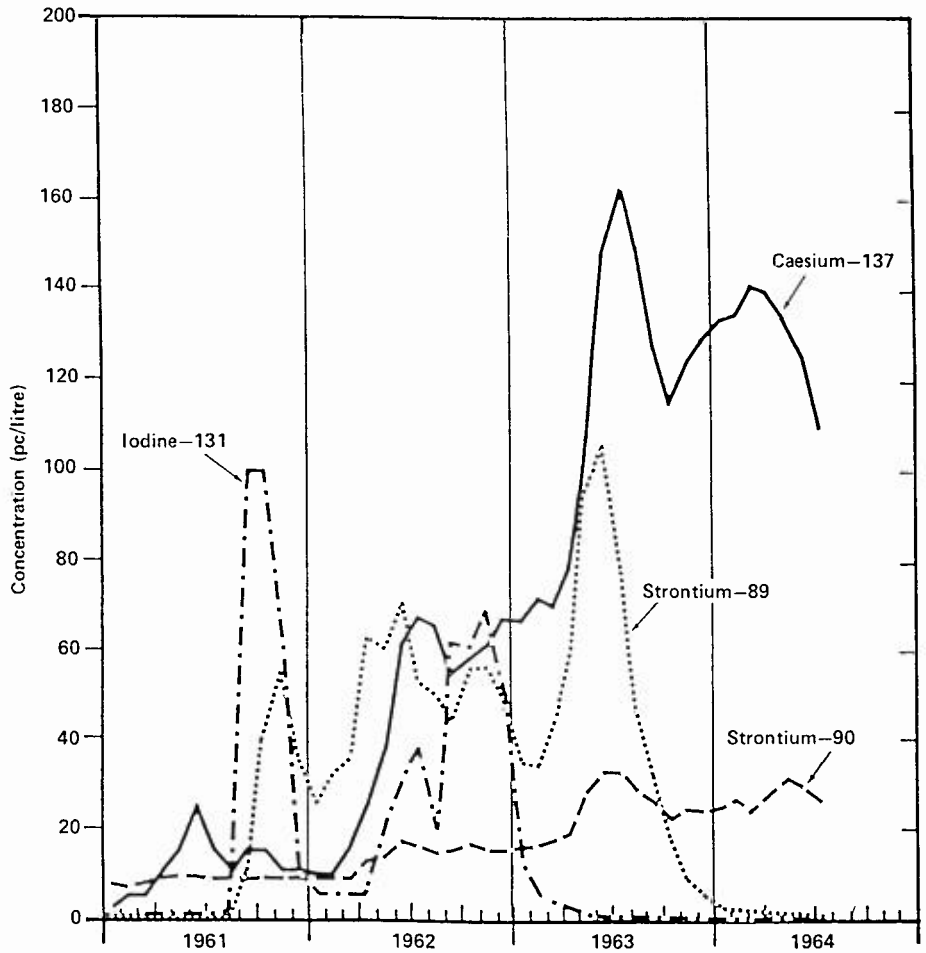


Figure 3 Average concentration of radionuclides in milk samples from Public Health Service pasturized milk network.

These reports finally appeared in 1970[13] and 1982[14] which were 42 and 54 years, respectively, after ICRP began. These are very useful handbooks but far more is needed on this subject.

(5) One of the most frustrating experiences many of us faced over a period of years was the large dose delivered both locally and world wide during atmospheric testing of nuclear weapons. Figure 3 indicates the seriousness of this problem in the US as attested by fallout levels of I-131, Cs-137, Sr-90 and Sr-89 in the pasteurized milk samples collected from 100 sampling stations in the US Public Health Service Network program[15] and Table 2[16] indicates the estimated dose in the 'wet' areas of the US. If one assumes there were 150 million people in the wet areas receiving the whole-body dose of 130 mrem over 70 years and that three quarters of this dose is effective, this corresponds to ~15 000 cancer deaths when using a cancer dose coefficient of $\sigma = 10^{-3}$ cancer deaths per rem (i.e. $0.130 \times 150 \times 10^6 \times 3/4 \times 10^{-3} = 14\,625$). Those conducting these tests tried to make the problem seem small, first by using a cancer coefficient σ that was too small by an order of magnitude, i.e. $\sigma = 10^{-4}$, by comparing the dose with that from natural background, i.e. ~ 100 mrem per year, and by comparisons with the natural cancer death rate of 20 per cent. In other words, 15 000 cancers on top of 30 million was considered 'negligible'. To me this is absurd. It is like telling a mother whose child is dying of radiation induced cancer not to worry because 30 million other people in the wet area of the US will die naturally of cancer.

Table 2 Estimated radiation 70-year dose commitment in the wet areas of the United States from nuclear weapon testing in 1962 and from all testing through 1962[a]

Tissue or organ	70-year dose commitment from 1962 testing (mrem)	70-year dose commitment from all testing through 1962 (mrem)
Whole body and reproductive cells		
Caesium-137 external	10	
Caesium-137 internal	10	
Short-lived nuclides	18	
Carbon-14	18	
Total	56	130
Bone		
Strontium-90	180	
Strontium-89	39	
Whole body	56	
Total	275	465
Bone marrow		
Strontium-90	60	
Strontium-89	13	
Whole body	56	
Total	129	215

[a]Values given in report, *Estimate and Evaluation of Fallout in the United States from Nuclear Weapons Testing Conducted through 1962*, Federal Radiation Council, Report No. 4, May 1963.

When a nation or an industry decides to go ahead with a programme that costs lives, I consider it has reached a conclusion on the value of a human life. Some years ago our Nuclear Regulatory Commission, NRC, made this decision when it set the value of a rem at $1000, i.e. industry was justified in spending $1000 to prevent one person-rem. If $\sigma = 10^{-3}$ cancer deaths per rem, this corresponds to $1000 $\div$ 10^{-3} = a million dollars per life. Others have set the value of a life much lower. For example, two members of the ICRP Main Commission, Mr H. J. Dunster[17] and Dr A. S. McLean (Dr A. S. McLean was a member of the Main Commission of ICRP from 1973 to 1977 and Mr H. J. Dunster has been a member since 1977), published the value of 10 to 25 dollars per man-rem or $10 000 to $25 000 per life.

The average world-wide annual whole-body dose commitment from weapons fallout is about 5 mrem per year[18] to the year 2000. This will cause a projected 750 000 cancer deaths (5×10^{-3} rem $\times$ 10^{-3}c/person rem $\times$ 5×10^9 persons $\times$ 3/4 $\times$ effective lifespan of 40 y = 750 000 cancer deaths from 1960 to the year 2000). Again I consider this very significant even though it is only 0.075 per cent of the total cancer deaths in the world population. The fact that other agencies such as the United Nations Scientific Committee on Atomic Radiation (UNSCEAR) addressed the fallout question in no wise obviates the obligation of ICRP, the recognized world authority on effects of radiation on man, from letting its voice be heard by taking up this issue and doing what it can to stop the deaths of 750 000 people. I regret ICRP was silent on this issue.

As I view it, the question of whether an organization like ICRP should set a higher value on a human life or whether it should go against the politically expedient stream, stick its neck out and ask for trouble by trying to put a stop to testing of nuclear weapons in the atmosphere is one of morality. Now an even greater question stands out. Should ICRP, like Physicians for Social Responsibility or the International Physicians for Prevention of Nuclear War, join the battle and help prevent the horrible suffering following World War III in considerable measure due to mass radiation of the world population? Perhaps this question will be answered with the word NO or in the typical way government agencies resolve issues. I quote from a letter uncovered via the US Freedom of Information Act. It was from Dr P. C. Tompkins, then Executive Director of the US Federal Radiation Council to Dr Haworth, Chairman of the US Atomic Energy Commission, dated 25 September 1962. It states in

part,

> If any reasonable agreement on this subject can be reached among Agencies, the basic approach to the report would be to start with a simple, straightforward statement of conclusions. It would then be a straightforward matter to select the key scientific consultants whose opinions should be sought in order to substantiate the validity of the conclusions or recommended appropriate modifications.

I hope ICRP will not operate like Government Agencies! I do believe, however, this was the basis on which the US Atomic Energy Commission decided the so-called particle problem (high dose near a small radioactive particle) was not a problem. I was never satisfied that the decision makers took proper account of studies such as those of H. Lisco *et al.*[19] where they observed a high incidence of cancer at the sites of injection of Pu-239 and other radionuclides under the skin of animals.

(6) An ICRP report on major radiation accidents, problems encountered and in what manner they were handled is long overdue. In 1984 ICRP published[20] a report on major radiation accidents but it is very brief, superficial and fails to address the emergency situations that have been experienced in major radiation accidents in many places. There have been four fatal criticality accidents in the US (Los Alamos, NM on 21 August 1945, 21 May 1946 and 30 Dec, 1958 and Wood River Junction, RI on 24 July 1964) and the SL-1 Reactor explosion at Idaho Falls, ID, 3 January 1961. One person was killed in each of the four criticality accidents and three were killed in the SL-1 accident. Only this SL-1 accident resulted in environmental contamination but because of its isolation in a desert environment, contamination beyond the plant-site and into the public domain was minimal. There were several bad accidents at the Rocky Flats Plant[22] not far from Denver, CO and these resulted in widespread environmental contamination from Pu-239. Recently information was uncovered indicating that on 2 December 1949, 5500 Ci of I-131 were released at the Hanford, WA plant to test meteorological patterns and adequacy of instruments in case fission products were used as an adjunct to chemical warfare. Also there had been hundreds of thousands of Curies released into the air and into the Columbia River during operations of the plant in weapons production during the war. The Savannah River, SC plant[23] had a number of releases of radioactive material into the environment. All of these accidents could provide a wealth of useful information on what to do and not to do and this information is just waiting for an organization like ICRP to bring it together. There have been also many non-fatal criticality accidents like the one at Y-12 in Oak Ridge, TN on 16 June 1958 in which five workers

got over 200 rem doses and there was the mild explosion at Mol, Belgium on 30 December 1965 where a worker received 550 rem. The Vinca, Yugoslavia accident on 15 October 1958 resulted in four persons receiving over 400 rem and one of these died after 32 days. I consider it a shame that much valuable information about these accidents has not been brought together and put into print. At present it only lingers in the minds of a few persons still living.

So far as I know until Chernobyl there had been only three major reactor accidents (SL-1 reactor, 3 January 1961, Windscale reactor No. 1 on 8 October 1957 and Three Mile Island Reactor no. 2 on 28 March 1979). There was a massive explosion[24,25] apparently in improperly buried nuclear waste in the Ural Mountains in 1957, but I have only fragmentary information on this. Possibly the Russian member on the ICRP Commission could provide details of this accident for this long awaited ICRP handbook. Some of the information I have in mind could have been of assistance at the time of the Chernobyl accident. In the following I list a few personal experiences that suggest the kind of information that should be given in this ICRP handbook that ought to have been written many years ago. They are as follows:

(a) At the SL-1 accident the men who ran up the stairs of the reactor building on a rescue attempt were in a thick cloud of dust and in radiation field of hundreds of rem/h. They certainly received very large doses to the nasopharyngeal and tracheobronchial regions of the lungs and to the gastrointestinal tract from the inhalation of large dust particles. It is unfortunate that faecal as well as urine blood and sputum samples were not collected and analysed after this exposure. The autopsy data on the three bodies (of those caught in the blast and eventually recovered from the debris in the reactor building) provided extremely useful information on the nature and cause of the accident.

(b) Following the explosion of the chemical process tank at Oak Ridge National Laboratory in 1965, it was very important that we approach the scene of the accident with operating neutron dose meters in hand. Although not known at the time, it turned out later there was enough plutonium in this tank for many critical assemblies. At any moment the liquid could have settled into a critical configuration.

In this same explosion a large amount of plutonium was blown out over an adjacent building and onto a road. Within four hours after the accident we had tarred the road and sprayed the adjacent building with paint. Later the road was taken up piece-by-piece, placed in plastic containers and sent to the official burial ground. The building later was disassembled piece-by-piece, placed in burial containers and properly buried. I have seen pictures from Chernobyl where they are washing

down the buildings and roads with water. This is opposite of what I would recommend except for contamination by short-lived radionuclides.

(c) In the Y-12 accident mentioned above I had all sorts of meters as I entered this building and homed-in on the criticality assembly with my operating instruments in hand but I failed to have with me a much needed instrument—a simple flashlight to see and to read the meters—for the electricity was now cut off in this labyrinth of a windowless building. In major accidents, important but simple things often are lacking. For example, gasoline pumps may not operate because the electrical power is knocked out. After the Y-12 accident I had the plant doctor collect 5 cc of blood from each of the highly exposed persons, mix it with heparin to prevent coagulation, and we measured the P-32 and Na-24 in the blood to determine the fast and thermal neutron dose (i.e. $^{32}S(n,p)^{32}P$ and $^{23}Na(n,\gamma)^{24}Na$). Differential blood counts were made from time to time and studies of chromosal aberrations were carried out.

(d) When I visited Windscale a couple of days after the accident, I was told of two major problems: (1) they did not get their light aircraft airborne for aerial surveys soon enough and (2) utter confusion at times could have been avoided if they had had a well equipped communication centre ready and waiting for them at the time of the accident. Neither of these two things were available at the Three Mile Accident or the Chernobyl accident. Maybe if ICRP will prepare this handbook on accidents, emergency personnel will be better prepared for the next major reactor accident?

(e) Perhaps ICRP in this proposed handbook could give guidance on how to put out a fire in graphite, uranium or zirconium? We had a fire at the back of our graphite reactor at Oak Ridge National Laboratory about the time we had a visit from Sir John Cockcroft in the late 1940s and he was impressed with the necessity of filters in the cooling air from a reactor before the cloud of dust and smoke went up the stack. But alas when he returned to the UK, the Windscale stack was already half built. But nothing could stop a great scientist. An immense filter house was built halfway up the stack. This became known as 'Cockcroft's folly' but it partly saved the day during the graphite fire at Windscale on 8 October 1957.

Early at Oak Ridge and at Windscale and recently at Chernobyl water was used with much trepidation to extinguish the fires but it put them out. However, at Chernobyl the water probably reacted with the hot metals and graphite to produce large amounts of hydrogen. While I was at Windscale during the time of the accident early in October 1957, I was puzzled that even though the filters near the top of the stack were saturated with water, they had held up most of the Sr-89 and Sr-90, Cs-134 and Cs-137 and the I-131, I-132, I-133, I-134 and I-135. How could this be? Then I was told that dense fumes from Bi-209 which was

stored in the burning part of the reactor had acted as condensation nuclei and made even water-saturated filters relatively efficient. I knew of course that the Bi-209 was in the reactor to produce Po-210 for the neutron trigger then used in our atomic bombs,* so I kept this a dark secret in my mind until a few years ago when information was declassified and released that Po-210 was one of the Windscale fallout products in 1957. Incidentally, about ten years ago Dr A. Stewart and I had just given lectures at a meeting in London and in the question period H. J. Dunster (now a member of ICRP) criticized Dr Stewart for having said an alpha-emitter was discharged with the fission products. In the discussion I tried in a weak way to come to Dr Stewart's rescue, but my lips were sealed because of security. Po-210, an alpha-emitter, was discharged along with the beta- and gamma-emitting fission products during the Windscale accident.

(7) One subject which I believe should be carefully followed by ICRP and on which it could make very useful recommendations, for example, is that of the person-rem per year at the various nuclear plants. This should be addressed both in terms of person-rem per year per plant and person-rem per year per 1000 MWe. It is astonishing to note that some nuclear power facilities consistently have a better record than others in this regard by more than an order of magnitude.[26,27]

(8) A final example of where ICRP, in my estimation, has been somewhat negligent is in meeting the need of an in-depth treatment of the environmental releases of radionuclides of greatest concern in the nuclear industry. Here we think of H-3, C-14, Sr-89 and -90, I-131, Cs-134 and -137, noble gas etc. Such a publication might help to answer many recurrent questions such as: What are the genetic risks of these radionuclides? Was ICRP justified in reducing the quality factor of the low energy beta radiation of H-3 from 1.8 to 1.0 when theory suggests the value of 2 is more appropriate? Why do some nuclear power plants discharge routinely into the environment a hundred times the curies of fission products released by the average power plant? Should additional efforts be made to reduce the large routine release of noble gases and H-3 by a nuclear power plant? Should power plants monitor the release of C-14?

In the foregoing I have discussed what I consider are some of the weaknesses of omission of ICRP and given a few typical examples. Perhaps there are as many weaknesses of commission by ICRP but in the following I will discuss only one, namely the ICRP-26 handbook and how it has resulted in an increase

*$^{209}Bi(n + \gamma)^{210}Bi \xrightarrow[5d]{\beta} {}^{210}Po$ (138d) and $^{210}Po(\alpha, Be)$ neutrons

in values of maximum permissible concentration (MPC) for many of the more common and more dangerous radionuclides such as Sr-89 and Sr-90, I-131 and Pu-238, Pu-239 and Pu-240. The values now recommended by ICRP are higher than we developed in 1959 for ICRP-2 when I was chairman of the Internal Dose Committee of ICRP. This increase might be justified were the risk of radiation-induced cancer much less than we perceived it to be almost thirty years ago but just the contrary is the case; today the cancer coefficient is known to be at least an order of magnitude greater than it was perceived to be in 1959.

During the last few years that I was an active member of the Main Commission of ICRP, we discussed an inconsistency in our basic internal dose standard, namely, the values of MPC were based on concentrations in air, water and food that at the end of an occupational exposure period of fifty years would result in dose rates of 5 rem/y to total body, gonads and active (red) bone marrow, 30 rem/y to bone, thyroid and skin and 15 rem/y to any other body organs that were the critical body organs (usually the organ with the greatest concentration of the radionuclide). In short, our Internal Dose Committee was criticized for using the same dose rate limit for gonads and active marrow as for whole body because, were the whole body exposed to 5 rem/y, the gonads and active bone marrow also would be exposed to 5 rem/y. Partial-body exposure was known to be less harmful than whole-body exposure so the permissible dose rate of the whole body should not be the same as that to the gonads and active bone marrow. It seemed to me the solution was very simple, namely reduce the whole-body dose rate to 2.5 rem/year. However, some members felt this would be a hardship to the nuclear industry and we should keep the limiting whole body dose at 5 rem/y for the nuclear worker exposure both to internal and external sources of radiation. I took the view that the external dose limit of 5 rem/y as well as the internal dose limit was too high and both should be reduced to be more in conformance with our realization that the cancer risk from radiation was greater than we thought it to be when these limits were first set. Unfortunately, in 1977, some years after I had been moved to the status of an emeritus member, ICRP-26 was adopted in which the limiting dose rates after fifty years of occupational exposure were set at 5 rem/y to whole body, 20 rem/y to gonads (an increase by a factor of 4), 42 rem/y to active bone marrow (an increase by a factor of 8.3), 42 rem/y to lungs (an increase by a factor of 2.8), 50 rem/y to thyroid and bone surfaces (an increase by a factor of 1.7) and 33 rem/y to breasts (an increase by a factor of 2.2).

While I was still an active member of ICRP (not yet moved to the status of an emeritus member) we also discussed the possibility that with the coming of the computer age we should be more sophisticated and calculate the MPC not just on the basis of the dose to a critical body organ from the radionuclide that was in this organ but also from what was in all the body organs as they irradiated the critical body organ (now called the target organ). I am pleased to say that ICRP-26 and ICRP-30 followed this suggestion and this has resulted in an improvement over ICRP-2. Had it not been for this latter change, all MPC values now provided by ICRP-26 and ICRP-30 would be increased. Table 3 indicates some of these changes for a few of the important radionuclides. I do not see justifications for any of these values being greater than they were in 1959 when ICRP-2 was published. It should be mentioned also that in ICRP-30[28] the term (MPC) in air has been changed to (DAC) or Derived Air Concentration and values of (MPC) for water for some unknown reason are no longer given. Instead, values of (ALI) or Annual Limit on Intake are given. Changing from the curie to the becquerel (= 1 disintegration per second) was bad enough (because we already have the unit hertz with the same dimensions) but now the ALI makes it difficult for us in the US to make direct use of ICRP-30 since we must keep in mind that

$$(\mathrm{MPC})_w = 10^{-10}\mathrm{ALI\ (Bq)}\ \mu\mathrm{Ci/cc\ of\ water}$$

$$(\mathrm{MPC})_a = \frac{\mathrm{DAC(Bq/m^3)}}{3.7 \times 10^{10}}\ \mu\mathrm{Ci/cc\ of\ air}$$

In this list of 46 radionuclides in Table 3 there are 36 cases for radionuclides in air where the $(\mathrm{MPC})_a$ has been increased and ten cases where these is a decrease. For radionuclides in water (or most foods) there are 35 cases where the $(\mathrm{MPC})_w$ is increased and 19 cases where they are lower. I believe all values should be lower. This same ratio of increased to decreased MPC values is maintained approximately in the other 200 radionuclides listed in ICRP-2 but not shown in Table 3.

It was of interest to me to note that when ICRP set up a table of weighting factors in ICRP-26[8] to obtain the new limiting dose rates (as given above) following fifty years of occupational exposure (or the limiting committed dose from a year's exposure) to eliminate the above-mentioned long recognized inconsistency, it did the equivalent of jumping from the frying-pan into the fire, i.e. it made an even more inconsistent move. Using their chosen weighting factors the limiting dose rate for thyroid and bone surfaces turned out to be 167 rem/y. Although all limiting occupational exposure levels are set to limit stochastic* damage and in particular radiation-induced cancer, ICRP was now faced with the fact that 167 rem/y could be expected to result in non-stochastic forms of damage among radiation workers. This of course could not be tolerated so ICRP reached up and adopted the figure of 50 rem/y out of thin air with no justification in terms of cancer induction.

*Stochastic forms of damage are those like cancer that have no threshold and the damage once it develops is not a function of the magnitude of the dose that caused it. Examples are cancer and genetic mutations. Non-stochastic forms of damage do not show up unless a threshold dose is exceeded and the larger the dose that caused them the more severe the effect. Examples are radiation-induced erythema, epilation, cataracts and radiation sickness.

Table 3 Comparison of ICRP-30 with ICRP-2 values

Radio-nuclide	Half-life	ICRP-30 DAC-(Bq/m³) MPC_a ($\mu Ci/cc)_a$	ICRP-2 MPC_a $(\mu Ci/cc)_a$	ICRP-30[a] ALI (Bq) MPC_w $(\mu Ci/cc)_w$	ICRP-2 MPC_w $(\mu Ci/cc)_w$
H-3(H_2O)	12.26y	8×10^5		3×10^9	
		(2.2×10^{-5})	(5×10^{-6})	(0.3)	(0.1)
C-14(CO_2)	5730y	3×10^6		9×10^7	
		(8.1×10^{-5})	(4×10^{-6})	(9×10^{-3})	(0.02)
Na-24	14.96h	8×10^4		10^8	
		(2.2×10^{-6})	(10^{-7})	(0.01)	(8×10^{-4})
P-32	14.28d	6×10^3		2×10^7	
		(1.6×10^{-7})	(7×10^{-8})	(2×10^{-3})	(5×10^{-4})
S-35	87.9h	3×10^4		4×10^8, 2×10^8	
		(8.1×10^{-7})	(3×10^{-7})	(0.04, 0.02)	(2×10^{-3})
Cl-36	3.1×10^5y	4×10^3		6×10^7	
		(1.1×10^{-7})	$(2 \times 10^{-8}$	(6×10^{-3})	(2×10^{-3})
Ca-45	165d	1×10^4		6×10^7	
		(2.7×10^{-7})	(3×10^{-8})	(6×10^{-3})	(3×10^{-4})
Cr-51	27.8d	3×10^5		10^9	
		(8.1×10^{-6})	(2×10^{-6})	0.1	(0.05)
Mn-54	303d	1×10^4		7×10^7	
		(2.7×10^{-7})	(4×10^{-8})	(7×10^{-3})	(3×10^{-3})
Fe-55	2.6y	6×10^4		3×10^8	
		(1.6×10^{-1})	(9×10^{-7})	(0.03)	(0.02)
Fe-59	45.6d	8×10^3		3×10^7	
		(2.2×10^{-7})	(5×10^{-8})	(3×10^{-3})	(2×10^{-3})
Co-58	71.3d	1×10^4		6×10^7, 5×10^7	
		(2.7×10^{-7})	(5×10^{-8})	$(6 \times 10^{-3}, 5 \times 10^{-3})$	(3×10^{-3})
Co-60	5.26y	500		2×10^7, 7×10^6	
		(1.4×10^{-8})	(9×10^{-9})	$(2 \times 10^{-3}, 7 \times 10^{-4})$	(10^{-3})
Ni-59	8×10^4y	3×10^4		9×10^8	
		(8.1×10^{-7})	(5×10^{-7})	(0.09)	(6×10^{-3})
Cu-64	12.8h	3×10^5		4×10^8	
		(8.1×10^{-6})	(10^{-6})	(0.04)	(6×10^{-3})
Zn-65	245d	4×10^3		10^7	
		(1.1×10^{-7})	(6×10^{-8})	(10^{-3})	(3×10^{-3})
Sr-89	52.7d	2×10^3		2×10^7	
		(5.4×10^{-8})	(3×10^{-8})	(2×10^{-3})	(3×10^{-4})
Sr-90	27.7y	60	$[10^{-9}]^b$	10^6, 2×10^7	$[10^{-5}]^b$
		(1.6×10^{-9})	(3×10^{-10})	$(10^{-4}, 2 \times 10^{-3})$	(4×10^{-6})
Mo-99	66.7h	2×10^4		6×10^7, 4×10^7	
		(5.4×10^{-7})	(2×10^{-7})	$(6 \times 10^{-3}, 4 \times 10^{-3})$	(10^{-3})
Ru-106	368d	200		7×10^6	
		(5.4×10^{-9})	(6×10^{-9})	(7×10^{-4})	(3×10^{-4})
Te-127m	109d	4×10^3		2×10^7, 10^7	
		(1.1×10^{-7})	(4×10^{-8})	$(2 \times 10^{-3}, 10^{-3})$	(2×10^{-3})
I-126	2.6h	500		8×10^5	
		(1.4×10^{-8})	(8×10^{-9})	(8×10^{-5})	(5×10^{-5})
I-129	1.7×10^7y	100		2×10^5	
		(2.7×10^{-9})	(2×10^{-9})	(2×10^{-5})	(10^{-5})
I-131	8.05d	700		10^6	
		$(1.9 \times 10^{-8}$	(9×10^{-9})	(10^{-4})	(6×10^{-5})

Table 3 *Continued*

Radio-nuclide	Half-life	ICRP-30 DAC-(Bq/m³) MPC_a ($\mu Ci/cc)_a$	ICRP-2 MPC_a $(\mu Ci/cc)_a$	ICRP-30[a] ALI (Bq) MPC_w $(\mu Ci/cc)_w$	ICRP-2 MPC_w $(\mu Ci/cc)_w$
I-132	2.26h	1×10^5		10^8	
		(2.7×10^{-6})	(2×10^{-7})	(0.01)	(2×10^{-3})
I-133	20.3h	4×10^3		5×10^6	
		(1.1×10^{-7})	(3×10^{-8})	(5×10^{-4})	(2×10^{-4})
I-135	6.68h	2×10^4		3×10^7	
		(5.4×10^{-7})	(10^{-7})	(3×10^{-3})	(7×10^{-4})
Xe-133	5.27d	4×10^6		—	
		(1.1×10^{-4})	(10^{-5})	—	—
Cs-134	2.046y	2×10^3		3×10^6	
		(5.4×10^{-8})	(10^{-8})	(3×10^{-4})	(3×10^{-4})
Cs-137	30.0y	2×10^3		4×10^6	
		(5.4×10^{-8})	(10^{-8})	(4×10^{-4})	(4×10^{-4})
Ba-140	12.8d	2×10^4		2×10^7	
		(5.4×10^{-7})	(4×10^{-8})	(2×10^{-3})	(7×10^{-4})
Ce-144	284d	200		8×10^6	
		(5.4×10^{-9})	(6×10^{-9})	(8×10^{-4})	(3×10^{-4})
Ir-192	74.2d	3×10^3		4×10^7	
		(8.1×10^{-8})	(3×10^{-8})	(4×10^{-3})	(10^{-3})
Po-210	138.4d	10		10^5	
		(2.7×10^{-10})	(2×10^{-10})	10^{-5}	(2×10^{-5})
Ra-226	1602y	10		7×10^4	
		(2.7×10^{-10})	(3×10^{-11})	(7×10^{-6})	(4×10^{-7})
Th-232	1.41×10^{10}y	4×10^{-2}		3×10^4	
		(1.1×10^{-12})	(2×10^{-12})	(3×10^{-6})	(5×10^{-5})
U-234	2.47×10^5y	6×10^{-1}		4×10^5, 7×10^6	
		(1.6×10^{-11})	(10^{-10})	$(4 \times 10^{-5}, 7 \times 10^{-4})$	(9×10^{-4})
U-235	7.1×10^8y	6×10^{-1}		5×10^5, 7×10^6	
		(1.6×10^{-11})	(10^{-10})	$(5 \times 10^{-5}, 7 \times 10^{-4})$	(8×10^{-4})
U-238	4.51×10^9y	7×10^{-1}		5×10^5, 8×10^6	
		(1.9×10^{-11})	(7×10^{-11})	$(5 \times 10^{-5}, 8 \times 10^{-4})$	(10^{-3})
Np-237	2.14×10^6y	9×10^{-2}		3×10^3	
		(2.4×10^{-12})	(4×10^{-12})	(3×10^{-7})	(9×10^{-5})
Pu-238	86.4y	3×10^{-1}		3×10^5, 3×10^6	
		(8.1×10^{-12})	(2×10^{-12})	$(3 \times 10^{-5}, 3 \times 10^{-4})$	(10^{-4})
Pu-239	24390y	2×10^{-1}		2×10^5, 2×10^6	
		(5.4×10^{-12})	(2×10^{-12})	$(2 \times 10^{-5}, 2 \times 10^{-4})$	(10^{-4})
Pu-240	6580y	2×10^{-1}		2×10^5, 2×10^6	
		(5.4×10^{-12})	(2×10^{-12})	$(2 \times 10^{-5}, 2 \times 10^{-4})$	(10^{-4})
Am-241	458y	8×10^{-2}		5×10^4	
		(2.2×10^{-12})	(6×10^{-12})	$(5 \times 10^{-6}$	(10^{-4})
Am-243	7.95×10^3y	8×10^{-2}		5×10^4	
		(2.2×10^{-12})	(6×10^{-12})	(5×10^{-6})	(10^{-4})
Cm-244	17.6y	2×10^{-1}		9×10^4	
		(5.4×10^{-12})	(9×10^{-12})	(9×10^{-6})	(2×10^{-4})

[a] Class W only
[b] Value given in ICRP-6 (1962).

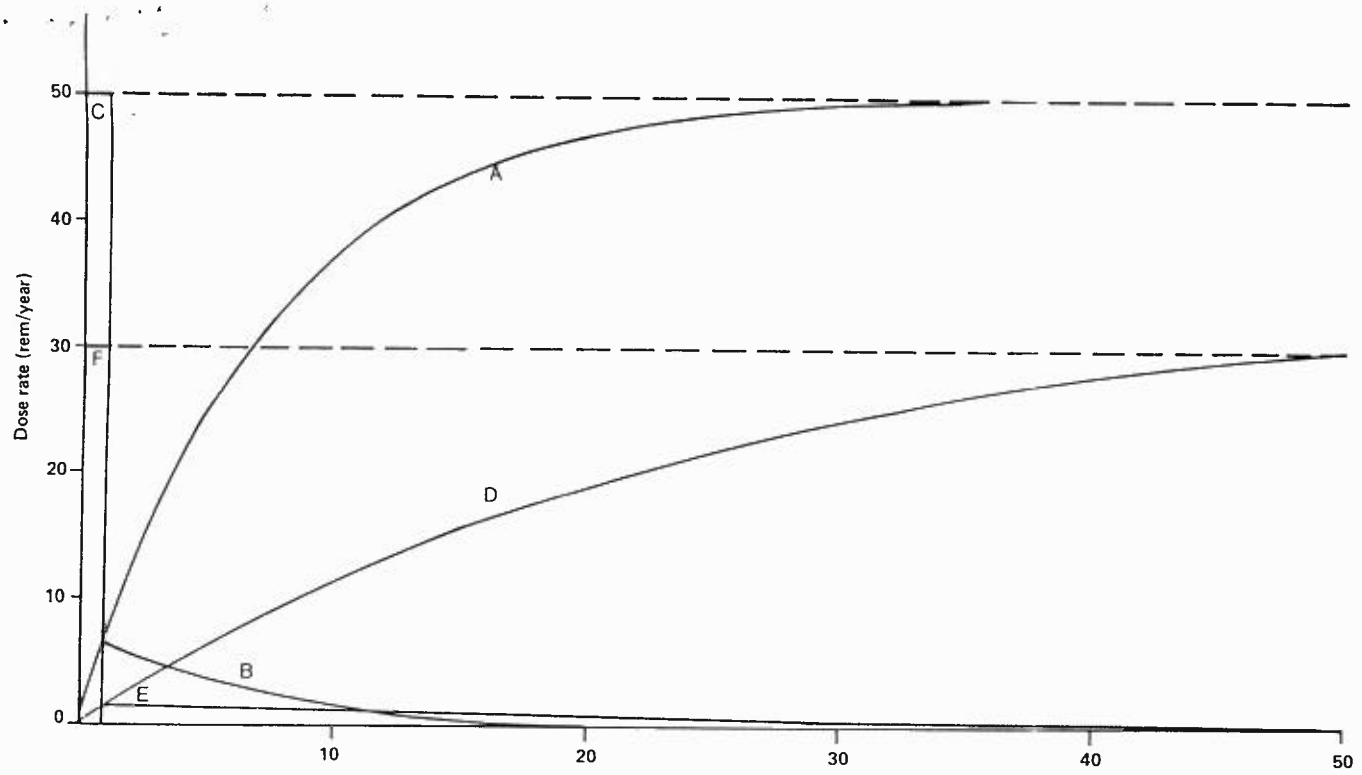


Figure 4 Time of occupational exposure (years).

A Exposure to Sr-90 at ICRP-30 level of DAC = 60 Bq/m^3 (1.62×10^{-9} μCi/cc)
D Exposure to Sr-90 at LCRP-2 level of 3.1×10^{-10} μCi/cc
Area under curve E = area in rectangle F = 30 rem
Area under curve B = area in rectangle C = 50 rem

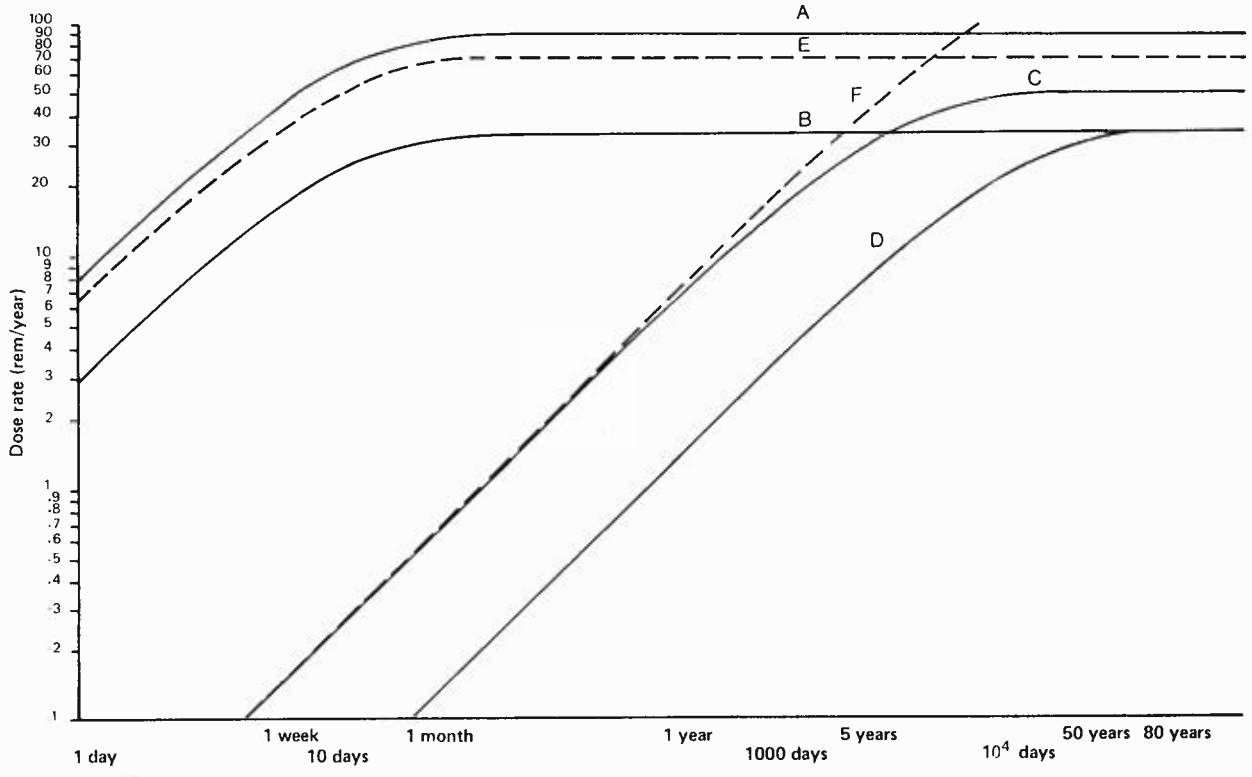


Figure 5 Period of occupational exposure.

A Exposure to I-131 at ICRP-30 level of DAC = 700 Bq/m^3 (1.9×10^{-8} μCi/cc)
B Exposure to I-131 at ICRP-2 level of 9×10^{-9} μCi/cc
C Exposure to Sr-90 at ICRP-30 level of DAC = 60 Bq/m^3 (1.62×10^{-9} μCi/cc)
D Exposure to Sr-90 at ICRP-2 level of 3.1×10^{-10} μCi/cc
E Exposure to I-131 at DAC = 700 Bq/m^3 but using values of $\overline{EQN}$, f_a, m and T from ICRP-2
F Exposure to Sr-90 at DAC = 60 Bq/m^3 but using values of $\overline{EQN}$, f_a, m and T from ICRP-2

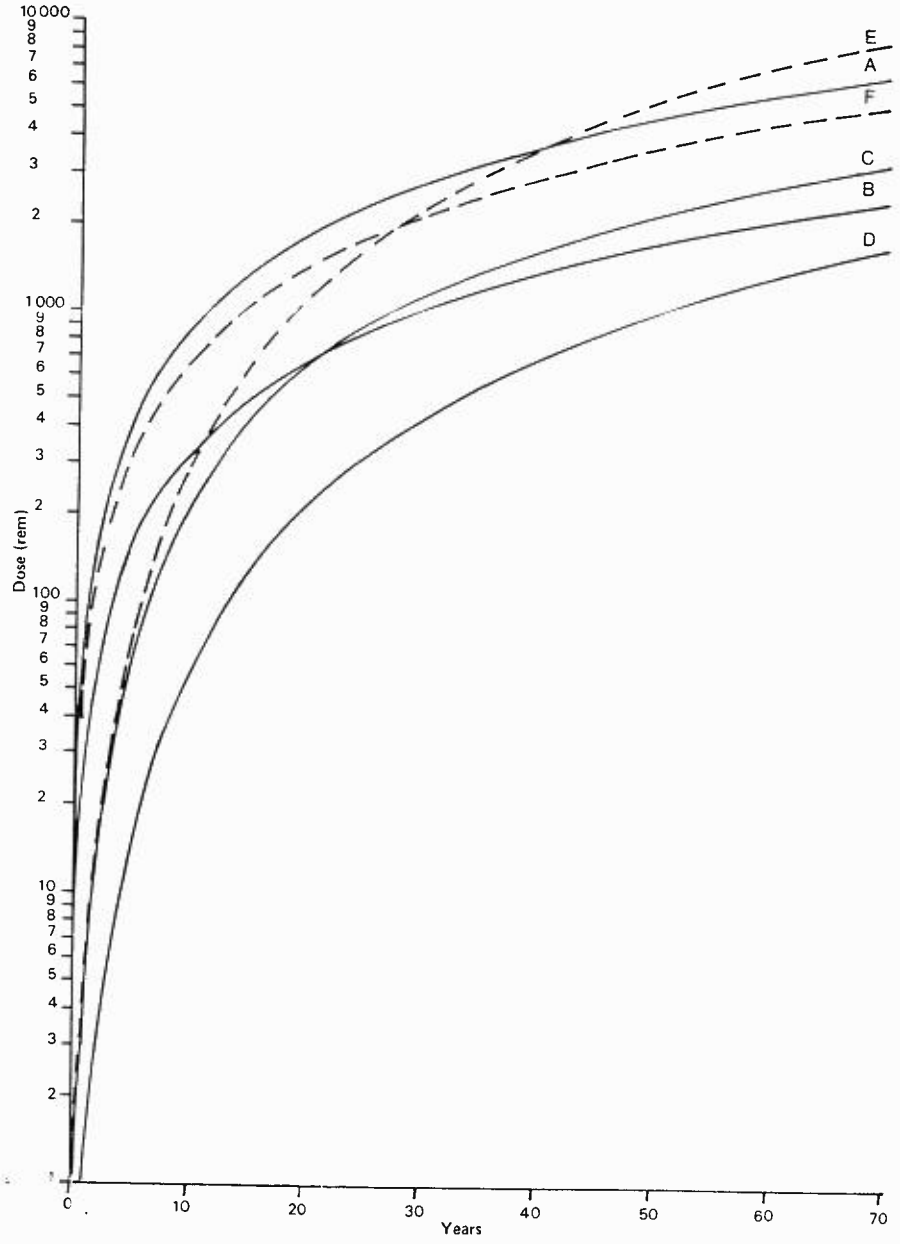


Figure 6 Period of occupational exposure (years).

A Accumulated dose from exposure to I-131 at ICRP-30 level of DAC = 700 Bq/m^3 (1.9×10^{-8} μCi/cc)

In the above it is pointed out that ICRP has now increased the limiting dose rate to all individual body organs except when the whole body itself is the critical body organ, i.e. the radionuclide is distributed rather uniformly in the body such as would be the case for H-3 taken in as HTO. The dose rates listed above are those to which the body organ of the radiation worker would be subjected were he to be exposed at the MPC of one of these radionuclides in air, water or food for fifty years at 40 hours per week and 50 weeks per year. This limiting dose rate, after fifty years as given by ICRP-30 for example, is 50 rem/y to bone surfaces and to the thyroid. Actually in these cases ICRP sets what it calls the limiting committed effective dose equivalent, CEDE, [8,29] during a year at 50 rem. That is, a worker, for example, can take in Sr-90 during a year in any manner provided the dose from this one year intake is no more than 50 rem when the bone surface dose is integrated thereafter over fifty years. In Appendix I it is shown that intake for a work year at the MPC will deliver a critical body organ dose integrated over fifty years that is equal to this CEDE and this CEDE is equal to the dose rate that would be reached in this organ after fifty years of occupational exposure multiplied by one year, i.e. it is numerically equal to the dose rate reached in this organ following fifty years' exposure at the MPC. This is shown in Figure 4 for the case of occupational exposure to Sr-90. Curve A shows the increasing dose rate to bone surfaces of the radiation worker working in a constant work environment 40 hours per week, 50 weeks per year for fifty years when the air concentration is maintained at the present ICRP-30 DAC of 60 Bq/m^3 (1.62×10^{-9} μCi/cc) of Sr-90. In this case I took the ICRP value of $f_a = 0.01$ as the fraction of Sr-90 going to bone surfaces and $m = 120$ g as the mass of these surface tissues. ICRP-30 did not give a separate value for the fraction of Sr-90 going from blood to bone surfaces so I interpreted the 0.01 value to be the product of the fraction to blood by the fraction from blood to bone surfaces. A value was not given in ICRP-30 for the biological half-life so I back calculated to get $T = T_b T_r/(T_b + T_r) = 4.961$ years in order that the fifty-year integration of dose rate from a year's intake would be 50 rem as required by ICRP-30. I used the $\overline{\text{EQN}}$ 1.1 MeV per disintegration of Sr-90 plus its daughter Y-90. It should be emphasized that ICRP-2 set $N = 5$ or $\overline{\text{EQN}} = 5.5$ MeV per disintegration and since I see no justification for ICRP having set $N = 1$ in ICRP-30, I believe

B Accumulated dose from exposure to I-131 at ICRP-2 level of 9×10^{-9} μCi/cc
C Accumulated dose from exposure to Sr-90 at ICRP-30 level of DAC = 60 Bq/m^3 (1.62×10^{-9} μCi/cc)
D Accumulated dose from exposure to Sr-90 at ICRP-2 level of 3.1×10^{-10} μCi/cc
E Accumulated dose from exposure to Sr-90 at DAC = 60 Bq/m^3 but using values of $\overline{\text{EQN}}$, f_a, m and T as given in ICRP-2
F Accumulated dose from exposure to I-131 at DAC = 700 Bq/m^3 but using values of $\overline{\text{EQN}}$, f_a, m and T as given in ICRP-2

the dose rate values of curve A in Figure 4 are under-estimated at least by a factor of 5. The equations for curves A and D are derived in Appendix II. It is noted that both curves pass through the dose rate limit at fifty years—Curve A at 50 rem/year and Curve D at 30 rem/year. Curve A with a shorter half-life ($T = 4.96$y) reaches its equilibrium at 50 rem/y in about forty years whereas Curve D with a longer half-life ($T = 17.53$y) passes through 30 rem/y (at 86 per cent of equilibrium) at fifty years but as shown by Curve D in Figure 5 it would not reach its equilibrium level of 34.8 rem/y until about 150 years. As indicated by Curve C in Figure 6, exposure at the new DAC of 60 Bq/m^3 (1.62×10^{-9} μCi/cc) for Sr-90 for fifty years would result in an average bone surface dose of 2140 rem but, as indicated by Curve E, when applying the values of EQN *m* and *T* as given in ICRP-2 this would result in a total bone dose of 5130 rem. Figures 5 and 6 indicate similar increases in thyroid dose rate and dose for I-131 except that the equilibrium dose rate in Curve A of Figure 5 is 91 rem/y instead of the ICRP-30 limit of 50 rem/y. No reason is given in ICRP-30 for this apparent discrepancy. Figure 6 indicates the 50-year accumulated dose by ICRP-30 specifications is 4570 rem (Curve A) and by ICRP-2 specifications is 1680 rem (Curve B). I see no justification for any of these increases.

CONCLUSION

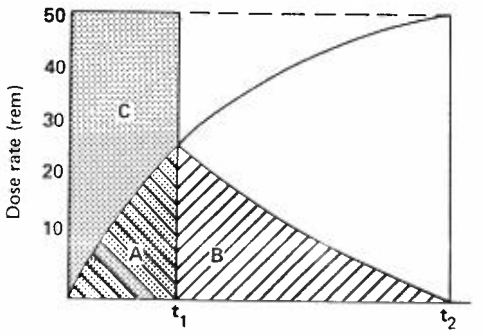
In conclusion I wish to re-emphasize that I have limited this discussion to criticisms of the work of the Main Commission of ICRP in its accomplishments and lack of them over the past sixty years. In this I am pointing the finger at myself as well as to others because I was one of this thirteen member body for about twenty years. Some of the committees of ICRP have done an excellent job. My principal criticisms of the Main Commission are that in many cases it has not responded to important situations of high exposure to ionizing radiation or has been unnecessarily slow in response and it has increased permissible exposure levels at a time when they should have been reduced.

REFERENCES

1. L. S. Taylor, *Radiation Protection Standards*, CRC Press, Cleveland, Ohio, US.
2. Report to the Congress of the United States, *Problems in Assessing the Cancer Risks of Low-Level Ionizing Radiation Exposure*, Vol. 2, EMD-81-1, 2 January 1982.
3. M. Kaku and J. Trainer, 'Nuclear power: both sides', Chapter 2, *Underestimating the Risks* by K. Z. Morgan, W. W. Norton & Co., New York and London.
4. A. M. Stewart, Delayed effects of A-bomb radiation: a review of recent mortality rates and risk estimates for five-year survivors, *J. of Epidemiology and Community Health*, **36**, 80–86 (1982).

5. McClenahan, Wasted X-rays, *Radiology*, **96**, 453 (Aug. 1970).
6. A. M. Stewart and G. Kneale, Radiation dose effects in relation to obstetric X-rays and childhood cancer, *Lancet*, 1185, 6 June 1970.
7. ICRP-6, *Recommendations of ICRP*, 1962.
8. ICRP-26, *Recommendations of ICRP*, 1977.
9. G. W. Kneale, and A. M. Stewart, Prenatal X-rays and cancer: further tests of data from the Oxford Survey of Childhood Cancers, *Health Phys.*, **51**, 3, 369 (1986).
10. Nader, R., Wake up America, unsafe X-rays. *Ladies Home Journal*, **85**, 126, (1968).
11. U.S. Federal Radiation Council, *Guidance for the Control of Radiation Hazards in Uranium Mining*, Report No. 8 (revised), Sept. 1967, US Supt of Documents, US Govt Printing Office, Washington DC 20402, US.
12. ICRP-24 *Radiation Protection in Uranium and Other Mines*, 1977.
13. ICRP-16, *Protection of the Patient in X-Ray Diagnosis*, 1970.
14. ICRP-34, *Protection of the Patient in Diagnostic Radiology*, 1982.
15. US Federal Radiation Council, *Revised Fallout Estimates for 1964–1965 and Verification of the 1963 Predictions*, Report No. 6, October 1964, US Supt of Documents, US Govt Printing Office, Washington, DC 20402, US.
16. US Federal Radiation Council, *Estimates and Evaluation of Fallout in the United States from Nuclear Weapons Testing Conducted through 1962*, Report No. 4, May 1963, US Supt of Documents, US Govt Printing Office, Washington, DC 20402, US.
17. ICRP-22, *Implications of Commission Recommendations That Doses Be Kept as Low as Readily Achievable*, 1973.
18. BEIR-III Report, *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation: 1980*, by Committee on the Biological Effects of Ionizing Radiations, National Research Council.
19. H. Lisco, M. P. Finkel and A. M. Brues, 'Carcinogenic Properties of Radioactive Fission Products and Plutonium, 'Thirty-Second Annual Meeting of the Radiological Soc. of North Amer., 2 Dec. 1946.
20. ICRP-40, *Protection of the Public in the Event of Major Radiation Accidents: Principles for Planning*, 1984.
21. K. Z. Morgan and J. E. Turner, *Principles of Radiation Protection*, R. E. Krieger Pub. Co., 1973.
22. C. J. Johnson, Cancer incidence in an area contaminated with radionuclides near a nuclear installation, *Ambio*, **10**, 176 (1981).
23. B. Franke and R. Alvarez, External gamma radiation around the Savannah River plant, *Ambio*, **14**, 2, 104 (1983).
24. Z. Medvedev, Facts behind the Soviet nuclear disaster, *New Scientist*, 30 June 1977.
25. Z. Medvedev, Winged messengers of disaster, *New Scientist*, November 1977.
26. D. W. Moeller, L. C. Sunt and W. R. Casto, Personnel overexposures at commercial nuclear power plants, *Nuclear Safety*, **22**, 4, 498 (July–Aug 1981).
27. R. E. Kasperson, Closing the protection gap: setting health standards for nuclear power workers, *Environment*, **24**, 10, 14 (Dec 1982).
28. ICRP-30, *Limits for Intakes of Radionuclides by Workers*, Part 1, Vol 2, No. 3/4, 1979; Part 1, Vol 3, No. 1–4, 1979; Part 2, Vol. 4, No. 3/4, 1980; Part 2, Vol 5, No. 1–6, 1981; Part 3, Vol. 6, No. 2/3, 1981; Part 3, Vol. 7, No. 1–3, 1982.
29. ICRP-42, *A Compilation of the Major Concepts and Quantities in Use by ICRP*, 1984.

APPENDIX I



To show that area A + area B = area C.

$$R = \frac{1.87 \times 10^4 \text{P } \overline{\text{EQN}} \ (1 - e^{-\lambda t})}{\lambda m} = C_1 \frac{(1 - e^{-\lambda t})}{\lambda} \text{ rem/y}$$

$$A = \int_0^t R \, dt = \frac{C_1}{\lambda^2} (\lambda t_1 + e^{-\lambda t_1} - 1)$$

$$B = \frac{C_1(1 - e^{-\lambda t_1})}{\lambda} \int_0^{t_2} e^{-\lambda t} \, dt = \frac{C_1(1 - e^{-\lambda t_2})}{\lambda^2} (1 - e^{-\lambda t_1})$$

$$= \frac{C_1}{\lambda^2} (1 - e^{-\lambda t_2} - e^{-\lambda t_1} + e^{-\lambda t_1} e^{-\lambda t_2})$$

$$A + B = \frac{C_1}{\lambda^2} (\lambda t_1 - e^{-\lambda t_2} + e^{-\lambda t_1} e^{-\lambda t_2}) \qquad (1)$$

$$t_1 R_{(t_1 - t_2)} = \frac{C_1 [1 - e^{-\lambda (t_1 + t_2)}] t_1}{\lambda} = \frac{C_1}{\lambda^2} (\lambda t_1 - \lambda t_1 e^{-\lambda t_1} e^{-\lambda t_2}) \qquad (2)$$

and

$$A + B = t_1 R_{(t_1 + t_2)} \text{ if } -e^{-\lambda t_2} + e^{-\lambda t_1} e^{-\lambda t_2} = -\lambda t_1 e^{-\lambda t_1} e^{-\lambda t_2}$$

multiplying by $e^{\lambda t_2}$ we have $-1 + e^{-\lambda t_1} = -\lambda t_1 e^{-\lambda t_1}$ and $e^{\lambda t_1} = 1 + \lambda t_1$.

Expanding by Maclaurin's Series,

$$e^{\lambda t_1} = 1 + \lambda t_1 + \frac{\lambda^2 t_1^2}{2} + \frac{\lambda^3 t_1^3}{6} + \frac{\lambda^4 t_1^4}{24} + \cdots$$

If $\lambda t_1 \ll 1$, $e^{t_1} = 1 + \lambda t_1$.

For

$$\text{Sr-90 } \lambda t_1 = \frac{0.693}{T} = \frac{0.693}{17.53} = 0.04 \quad \text{and} \quad 1 + \lambda t_1 = 1.04$$

while

$$\frac{\lambda^2 t_1^2}{2} \frac{\lambda^3 t_1^3}{6} \frac{\lambda^4 t^4}{24} = 3.6 \times 10^{-6} + 3.2 \times 10^{-9} + 2.1 \times 10^{-2} \text{ or } \ll 1.04.$$

Or for Sr-90, equation (1) above gives

$$A + B = \frac{6.9905}{(0.1397)^2} = (0.1397 - 0.001 + 0.0009) = 50 \text{ rem}$$

and from equation (2)

$$t_1 R_{(t_1 + t_2)} = \frac{6.9905}{(0.1397)^2} (0.1397 - 0.00013) = 49.99 \text{ rem.}$$

For I-131 (using ICRP-2 values),

$$A + B = \frac{1120}{(33.28)^2} (33.28 - 0 + 0) = 33.7 \text{ rem and } t_1 R_{(t_1 + t_2)}$$

$$= \frac{1120}{(33.28)^2} (33.28 - 0) = 33.7.$$

APPENDIX II

Equations used

$$R\left(\frac{\text{rem}}{\text{y}}\right) = q f_2 (\mu\text{Ci}) \, 3.7 \times 10^4 \left(\frac{\text{dis}}{\text{S } \mu\text{Ci}}\right) 3600 \times 24 \times 365 \left(\frac{\text{S}}{\text{y}}\right) \overline{\text{EQN}} \left(\frac{\text{MeV}}{\text{dis}} \cdot \frac{\text{rem}}{\text{rad}}\right)$$

$$\times 1.602 \times 10^{-6} \left(\frac{\text{erg}}{\text{MeV}}\right) \frac{1}{m} \left(\frac{1}{\text{g}}\right) \frac{1}{100} \left(\frac{\text{g rad}}{\text{erg}}\right)$$

$$= 1.869 \times 10^4 \frac{q f_2 \times \overline{\text{EQN}}}{m}$$

$$P\left(\frac{\mu\text{Ci}}{\text{y}}\right) = 6.9 \times 10^6 f_a \left(\frac{\text{cc}}{\text{d}}\right) 365 \left(\frac{\text{d}}{\text{y}}\right) (\text{MPC})_a \left(\frac{\mu\text{Ci}}{\text{cc}}\right) = 2.518 \times 10^9 f_a (\text{MPC})_a$$

$$\frac{dq}{dt} + \lambda q = P \quad \text{or} \quad q(\mu\text{Ci}) = \frac{P}{\lambda} (1 - e^{-\lambda t})$$

$$R\left(\frac{\text{rem}}{\text{y}}\right) = \frac{(\text{MPC})_a \overline{\text{EQN}} \, f_a (1 - e^{-\lambda t})}{2.124 \times 10^{-14} m \lambda}$$

$$D(\text{rem}) = \int_0^t R \, dt = \frac{(\text{MPC})_a \, \overline{\text{EQN}} \, f_a}{2.124 \times 10^{-14} \, m \lambda^2} \times (\lambda t + e^{-\lambda t} - 1)$$

Using values from ICRP-2

$$R_{\text{for Sr-90}} = \frac{3.1 \times 10^{-10} \times 5.5 \times 0.12\ (1 - e^{-0.0395t})}{2.124 \times 10^{-14} \times 7 \times 10^{3} \times 0.0395}$$

$$= 34.8(1 - e^{0.0395t})_{\text{rem/y}} \text{ to bone}$$

$$R_{\text{for I-131}} = \frac{9 \times 10^{-9} \times 0.23 \times 0.23\ (1 - e^{-33.28t})}{2.124 \times 10^{-14} \times 20 \times 33.28}$$

$$= 33.67(1 - e^{-33.28t})_{\text{rem/y}} \text{ to thyroid}$$

$$D_{\text{for Sr-90}} = 881(0.0395t + e^{-0.0395t} - 1) \text{ rem to bone surfaces}$$

$$D_{\text{for I-131}} = 1.01(33.28t + e^{-33.28t} - 1) \text{ rem to thyroid}$$

Using values from ICRP-30

$$R_{\text{for Sr-90}} = \frac{1.62 \times 10^{-9} \times 1.1 \times 0.01\ (1 - e^{-0.140t})}{2.124 \times 10^{-14} \times 120 \times 0.140}$$

$$= 50\ (1 - e^{-0.14t})_{\text{rem/y}} \text{ to bone}$$

$$R_{\text{for I-131}} = \frac{1.9 \times 10^{-8} \times 0.23 \times 0.3\ (1 - e^{-33.73t})}{2.124 \times 10^{-14} \times 20 \times 33.73}$$

$$= 91\ (1 - e^{-33.73t})_{\text{rem/y}} \text{ to thyroid}$$

$$D_{\text{for Sr-90}} = 357(0.14t + e^{-0.14t} - 1) \text{ rem to bone surfaces}$$

$$D_{\text{for I-131}} = 2.71(33.73t + e^{-33.73t} - 1) \text{ rem to thyroid}$$