REPORT OF THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION

GENERAL ASSEMBLY
OFFICIAL RECORDS : NINETEENTH SESSION
SUPPLEMENT No. 14 (A/5814)

UNITED NATIONS

(114 p.)

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New York, 1964

NOTE

Throughout the present report, references to the annexes are indicated by a letter immediately followed by a number: the letter denotes the relevant annex and the number the paragraph therein. Within each annex, references to its scientific bibliography are indicated by numbers.

Symbols of United Nations documents are composed of capital letters combined with figures. Mention of such a symbol indicates a reference to a United Nations document.

CONTENTS

ABBREVIATIONS

ABCC	Atomic Bomb Casualty Commission
FAO	Food and Agriculture Organization of the United Nations
IAEA	International Atomic Energy Agency
ICRP	International Commission on Radiological Protection
ICRU	International Commission on Radiological Units and Measurements
WHO	World Health Organization
WMO	World Meteorological Organization

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CNS	Central nervous system
DNA	Deoxyribonucleic acid
ERG	Electroretinogram
ESR	Electron spin resonance
GI	Gastro-intestinal
LET	Linear energy transfer
MPC	Maximum permissible concentration
MPRE	Minimum pure radium equivalent
OR	Observed ratio
RBE	Relative biological effectiveness
RES	Reticulo-endothelial system
RNA	Ribonucleic acid
TBR	Total body radiation
TNT	Trinitrotoluene
UV	Ultra violet

CHAPTER I

INTRODUCTION

Constitution and terms of reference of the Committee

1. The United Nations Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session on 3 December 1955, under resolution 913 (X), as a result of debates held in the First Committee from 31 October to 10 November 1955. The terms of reference of the Committee were set out in paragraph 2 of the above-mentioned resolution by which the General Assembly requested the Committee:

"(*a*) To receive and assemble in an appropriate and useful form the following radiological information furnished by States Members of the United Nations or members of the specialized agencies:

"(i) Reports on observed levels of ionizing radiation and radio-activity in the environment;

"(ii) Reports on scientific observations and experiments relevant to the effects of ionizing radiation upon man and his environment already under way or later undertaken by national scientific bodies or by authorities of national Governments;

"(*b*) To recommend uniform standards with respect to procedures for sample collection and instrumentation, and radiation counting procedures to be used in analyses of samples;

"(*c*) To compile and assemble in an integrated manner the various reports, referred to in sub-paragraph (*a*)(i) above, on observed radiological levels;

"(*d*) To review and collate national reports, referred to in sub-paragraph (*a*)(ii) above, evaluating each report to determine its usefulness for the purposes of the Committee;

"(*e*) To make yearly progress reports and to develop by 1 July 1958, or earlier if the assembled facts warrant, a summary of the reports received on radiation levels and radiation effects on man and his environment together with the evaluations provided for in sub-paragraph (*d*) above and indications of research projects which might require further study;

"(*f*) To transmit from time to time, as it deems appropriate, the documents and evaluations referred to above to the Secretary-General for publication and dissemination to States Members of the United Nations or members of the specialized agencies."

2. The Committee consists of Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, France, India, Japan, Mexico, Sweden, the Union of Soviet Socialist Republics, the United Arab Republic, the United Kingdom of Great Britain and Northern Ireland and the United States of America.

Activities of the Committee

3. Since its establishment, the Committee has held fourteen sessions. Its activities during the first eleven sessions were surveyed in the introduction to the comprehensive reports that the Committee submitted to the thirteenth and seventeenth sessions of the General Assembly in 1958[1] and 1962,[2] respectively. The 1962 comprehensive report of the Committee was noted by the General Assembly during its seventeenth session in resolution 1764 (XVII) of 21 November 1962. By that resolution, the General Assembly:

(1) Commended the United Nations Scientific Committee on the Effects of Atomic Radiation for its work and for the valuable report it had presented;

(2) Expressed its appreciation to the International Atomic Energy Agency, to the specialized agencies, to the international non-governmental and the national scientific organizations and to the individual scientists who had assisted the Scientific Committee in its work;

(3) Called particular attention to the Scientific Committee's finding that the exposure of mankind to radiation from increasing numbers of artificial sources, including the world-wide contamination of the environment with short- and long-lived radio-nuclides from weapons tests, called for the closest attention, particularly because the effects of any increase in radiation exposure might not be fully manifested for several decades in the case of somatic disease, and for many generations in the case of genetic damage;

(4) Urged all concerned to take note of the suggestions made and the views expressed in the report of the Scientific Committee;

(5) Requested the Scientific Committee to continue its assessment of radiation risks as well as its review of those studies and further investigations that should be undertaken in the interests of increasing man's knowledge of the effects of radiation, and to report to the General Assembly at the eighteenth session on its progress and on its future programme of work;

(6) Called upon the International Atomic Energy Agency, the specialized agencies, the international non-governmental and the national scientific organizations, individual scientists and the Governments of Member States to continue to co-operate fully with the Scientific Committee in carrying out its further important responsibilities;

(7) Recommended the Governments of Member States to prepare and carry out according to their means large-scale information programmes on the effects of atomic radiation;

(8) Requested the Secretary-General to continue to provide the Scientific Committee with the assistance necessary for the conduct of its work.

[1] *Official Records of the General Assembly, Thirteenth Session, Supplement No. 17* (A/3838).

[2] *Ibid., Seventeenth Session, Supplement No. 16* (A/5216).

4. As requested in that resolution, the Committee discussed its programme of work at its twelfth session, which was held at the European Office, Geneva, from 21 January to 30 January 1963, and outlined the programme in a report to the General Assembly.[3]

5. In that report, it was noted that the request of the General Assembly that the Committee continue the assessment of radiation risks involved reviewing, on the one hand, the dose contributions from different sources of radiation and, on the other, the results of biological and medical studies which would lead to a better knowledge of the genetic and somatic effects of radiation and of the way in which the frequency of their occurrence depended upon radiation dose. Any significant change in estimates of the doses received by human tissues or in the evaluation of quantitative relationships between dose and effects might require revision of the estimates of radiation risks.

6. The Committee also expressed the view that the General Assembly might be effectively kept informed of the results of the Committee's continuing deliberations if the Committee submitted, at relatively frequent but not necessarily yearly intervals, short or specialized reports on any conclusions significantly affecting the estimates of radiation risks. The Committee left open the possibility of preparing a new general review of the whole field of study encompassed by its terms of reference at a time when scientific progress would, in its opinion, so require.

7. The report was considered by the General Assembly at its eighteenth session. Following debate in the Special Political Committee, the General Assembly adopted, on 12 November 1963, resolution 1896 (XVIII) by which the Scientific Committee was requested to continue its programme and its co-ordinating activities to increase the knowledge of the levels and effects of atomic radiation from all sources.

8. In response to that request, the Committee at its thirteenth session reviewed the information on environmental contamination and on induction of malignancies as a preliminary to the present report, which was completed and adopted on 10 July 1964 during the fourteenth session of the Committee.

9. At its fourteenth session, the Committee also discussed how it should continue its work of assessment of radiation risks from all sources and asked that arrangements be made to hold one session in 1965. The Committee expressed the hope that it would find it possible to submit a further substantive report to the General Assembly in 1966.

Sources of information

10. The reports received by the Committee between 10 March 1962 and 3 July 1964, inclusive, from States Members of the United Nations and members of the specialized agencies and of the International Atomic Energy Agency, as well as from these agencies themselves, are listed in annex C. Reports submitted prior to 10 March 1962 were listed in the 1958 and 1962 reports of the Committee. These reports were supplemented by a number of other publications available in the scientific literature and also by unpublished personal communications from individual scientists.

[3] *Ibid., Eighteenth Session, Annexes,* agenda item 31, document A/5406.

Organization of the work of the Committee

11. Discussion and evaluation of the information received by the Committee, as in the past, took place in the course of informal meetings among groups of specialists set up by the Committee, their general conclusions being subsequently reviewed by the full Committee. According to the Committee's established practice, no detailed record of its technical discussions was taken.

12. Mr. D. J. Beninson of Argentina and Mr. M. E. A. El-Kharadly of the United Arab Republic served as Chairman and Vice-Chairman, respectively, during the twelfth and thirteenth sessions of the Committee. At the thirteenth session, Mr. D. J. Stevens of Australia and Mr. A. R. Gopal-Ayengar of India were elected Chairman and Vice-Chairman, respectively, to serve during the fourteenth and fifteenth sessions. The names of scientists who have attended sessions of the Committee from the twelfth to the fourteenth sessions, inclusive, as members of national delegations, are listed in appendix I.

Scientific assistance

13. As in the past, the Committee was assisted by a small scientific staff and by consultants appointed by the Secretary-General. Scientific staff and consultants were responsible for preliminary review and evaluation of the scientific information received by the Committee or published in the technical literature.

14. While the responsibility for the report rests entirely with the Committee, the Committee wishes to acknowledge the help and advice received from those scientists whose names are listed in appendix II. The Committee owes much to their co-operation and goodwill.

Relations with United Nations agencies and other organizations

15. The Committee has been gratified by the assistance that it has received during its sessions from the International Labour Organisation (ILO), the Food and Agriculture Organization of the United Nations (FAO), the World Health Organization (WHO), the World Meteorological Organization (WMO) among the specialized agencies of the United Nations and from the International Atomic Energy Agency (IAEA), as well as from the International Commission on Radiological Protection (ICRP) and the International Commission on Radiological Units and Measurements (ICRU) among the non-governmental organizations.

16. In response to a request of the Committee, FAO assembled data on the contamination of the food chain that were used in the preparation of the present report, and WMO assisted in the evaluation of problems of transport and distribution of radio-active debris by convening a group of leading meteorologists who took part in the discussions on atmospheric contamination that were held during the thirteenth session of the Committee.

17. As noted in the 1962 report, at its eleventh session the Committee had given consideration, at the request of the Secretary-General of WMO, to a draft plan proposed by that Organization for the implementation of section II of General Assembly resolution 1629 (XVI). In response to a further request of the Secretary-General of WMO, the Committee considered at its

twelfth session a revised plan prepared by that Organization following the recommendations previously made by the Committee. As a result of its deliberations, the Committee adopted a statement that was transmitted to the Secretary-General of WMO and which contained a number of recommendations on those aspects of the plan which fell within the terms of reference of the Committee.

Scope and purpose of the report

18. The present report makes no attempt to cover the whole field of radiation effects as did the 1958 and 1962 reports of the Committee. Rather, the report confines itself to two subjects only: the contamination of the environment by nuclear explosions and the possibility of quantitatively assessing the risk of induction of malignancies by radiation in man.

19. Regarding the former subject, the Committee felt it appropriate to make a detailed review of the information available as at June 1964, which would complete the survey made in the Committee's 1962 report. After the adoption of that report, atmospheric contamination from nuclear explosions had continued on a large scale until the end of 1962, thus calling for a revision of the estimates of doses and risks from radio-active debris. Besides, the results of a number of new studies had been published since March 1962 which had to some extent clarified many of the problems left unsolved in the 1962 report. In making estimates of risks from environmental contamination, the present report will therefore take into account not only the amount of radio-active material that is now present in the environment but also the new knowledge of mechanisms of distribution in the environment and of uptake in the food chains and eventually in body tissues. Since only those aspects of the subject that have been significantly altered by new advances since 1962 are reviewed in the present document, the reader is referred to the 1962 report for the necessary background information.

20. With regard to the induction of malignancies, though no radical change in our knowledge has taken place since 1962, recent information makes it possible to give a sounder basis to certain risk estimates that the Committee had already obtained in the past, to confirm some that had been adumbrated and to propose new ones. In the case of the induction of malignancies also, the reader is referred to the 1962 report for a discussion of many details of radiation carcinogenesis that will not be dwelt upon in the present report.

21. As in earlier reports of the Committee, the main text of this report is accompanied by technical annexes in which the scientific information available to the Committee is discussed in some detail. The Committee wishes to emphasize, as it did in the past, that its conclusions, being based on the scientific evidence presently available, cannot be considered as final and will require revision as scientific knowledge progresses.

CHAPTER II

RADIO-ACTIVE CONTAMINATION OF THE ENVIRONMENT BY NUCLEAR TESTS

1. The nuclear explosions carried out between September 1961 and December 1962 sharply increased the radio-active contamination of the environment and consequently the doses of radiation that human populations will receive. However, the Committee notes that after the cessation of nuclear test explosions in the atmosphere, in outer space and under water, and in view of the propitious circumstances prevailing, further contribution from these sources to the radio-active contamination of the environment has ceased. Information on the amounts of various radio-nuclides and on the rates at which they deposit on the earth's surface and enter the food chain is necessary in order to compute the doses to human tissues. Since the cessation of atmospheric tests in December 1962, the Committee has been able to collect sufficient information to enable it to up-date adequately the estimates of the resulting radiation doses.

2. Almost all of the fission products from the 1961-1962 explosions have been introduced into the stratosphere. The strontium-90 from these tests increased the stratospheric inventory at the end of 1962 by about 5 megacuries over the level in mid-1961 (A32-34).[4]

3. The rate of transfer from the stratosphere to ground level depends upon the altitude to which the products rise in the atmosphere and the latitude at which the explosions occur. For example, the mean residence time of material in the stratosphere above 100 km exceeds five years while in the lower stratosphere it is less than one year (A16-19). Assessment of the experimental data has led the Committee to adopt an over-all mean residence time for the composite stratospheric fission products of two years (A20). While this time is shorter than that used in the 1962 report, the predicted deposition of strontium-90 and caesium-137 is not appreciably altered by the change in the mean residence time.

4. The fall-out rate of long-lived radio-activity in 1962 was three times that for the period 1960-1961 and during the year 1963 the fall-out exceeded that in any previous year (A36-38). The Committee envisages that in 1964 the fall-out rate may be some two-thirds of that during 1963 and will continue to decrease progressively in future years.

5. Short-lived fission products have decayed to negligible levels during 1963 so that no further dose will be incurred from them after 1964 (A56-59).

6. Radio-active materials which have been deposited on the surface of the earth constitute sources of both external and internal radiation to the population. Whereas their contribution to the external dose depends on the gamma radiation which they emit, the magnitude of the internal dose is determined mainly by the extent to which different nuclides are transferred through food chains to man.

7. Strontium-90 and caesium-137 are the most important fission products from nuclear explosions that contaminate man's diet. The mechanisms which control the transfer of strontium-90 through food chains into man's diet were discussed extensively in the 1962 report.[5] Information which has been obtained since that time does not necessitate the modification of the basis for assessment. During 1962 dietary contamination in the northern hemisphere was somewhat greater than in 1959, which, up to that time, had been the year when highest levels were observed. In 1963, dietary levels in the northern hemisphere were at least twice those in 1962 (A80). In the southern hemisphere, dietary contamination increased in 1962 and 1963, though to a smaller extent, and the levels remained considerably lower than those in the northern hemisphere (A81).

8. Recent evidence on the transfer of caesium-137 through food chains has led to an improved basis for evaluating radiation doses from caesium-137 within the human body (A134, 135, 178-180). It is now apparent that doses from caesium-137 were somewhat over-estimated in the 1962 report. Between 1961 and 1963, the changes in levels of caesium-137 in diet were broadly similar to those in levels of strontium-90 (A117).

9. It has been found that, under certain local ecological conditions, the transfer of caesium-137 to man is enhanced, leading to the highest body contents yet measured. Thus, in arctic regions, the levels of caesium-137 in the flesh of reindeer and caribou are high on account of the accumulation of this nuclide in the vegetation on which the animals graze (A118). The body content of caesium-137 in small groups of local inhabitants who live almost exclusively on the meat of reindeer and caribou has on occasions exceeded the world average by a factor of more than 100 (A128).

10. Short-lived radio-nuclides have been measured in the environment, in food and in the human body more consistently since the end of 1961 than during earlier series of tests. As a consequence, doses delivered by those nuclides are now more accurately known. Iodine-131 has received particular attention (A136-146) because its absorption by infants from fresh milk leads to the irradiation of their thyroid glands. Adults receive much lower doses owing to the larger size of their thyroid glands, and their lower consumption of fresh milk.

11. In most areas of the temperate zone in the northern hemisphere, the average dose to the thyroid glands of children who were brought up on fresh milk was about 0.1 rad in 1961 (A, table XXX); similar doses were received in 1962, whereas in 1963 the doses were negligible (A182, 183). In the southern hemisphere, doses were considerably lower. In 1962, the concentration of iodine-131 in milk produced in some limited areas within a few hundred kilometres of testing grounds were

[4] Throughout the present report, references to the annexes are indicated by a letter immediately followed by a number. Thus A32-34 refers to paragraphs 32 to 34 of annex A.

[5] *Official Records of the General Assembly, Seventeenth Session, Supplement No. 16* (A/5216), chapter V, paragraphs 60-69.

ten times higher than the average; doses to the thyroid were correspondingly higher (A138).

12. The Committee has again reviewed the problem of the doses due to carbon-14, a radio-nuclide with a half-life of about 5,700 years, which is formed from atmospheric nitrogen both naturally, by the continuous interaction of cosmic rays, and artificially, by neutrons released from nuclear explosions. The atmospheric content of artificial carbon-14 has been increased about threefold by testing in 1961-1962. By July 1963, the artificial carbon-14 concentration in ground level air rose to 90 per cent of the natural carbon-14 concentration in the northern hemisphere (A, table XV). With time, artificial carbon-14 will tend to become uniform throughout the atmosphere and to be progressively absorbed by the oceans. Thus, by the year 2000, the artificial carbon-14 concentration in the atmosphere will fall to some 3 per cent of the natural carbon-14 concentration (A71).

13. As in its 1962 report,[6] the Committee has based its evaluation of comparative risks due to past nuclear explosions on dose commitments to the gonads, to the cells lining bone surfaces and to the bone marrow—those tissues whose irradiation may give rise to hereditary defects, bone tumours and leukaemias, respectively. The dose commitment is the total dose that will be delivered, as an average for the world population, to the relevant tissues during the complete decay of radio-active material introduced into the environment. Doses included in the dose commitments may be delivered over a very long period of time. The dose commitments due to all tests before January 1963 are summarized in table I.

14. In the present report dose commitments are expressed in rads.[7] For radiations resulting from nuclear explosions, rads, as used here, and rems, as defined in the 1962 report,[8] are numerically equivalent. In this report, doses from natural radiation also are expressed in rads and therefore are numerically slightly smaller than in the 1962 report where they were expressed in rems. They are 99, 96 and 95 millirads per year to gonads, cells lining bone surfaces and bone marrow, respectively.

15. Comparative risk estimates can be made by reference to doses from natural sources of radiation. One inherent difficulty in such comparisons arises from the arbitrary period over which the natural radiation dose must be integrated. In principle, several alternatives are possible:

(1) The dose commitment could be compared with the natural radiation dose delivered over a period of time equal to that over which a substantial part of the dose commitment is delivered. This comparison could be misleading in the sense that exposures from future nuclear tests might overlap this period.

[6] A/5216, chapter VI.

[7] The rad is the unit of absorbed dose; A/5216, chapter II, paragraph 23.

[8] A/5216, chapter II, paragraph 26; the rem has recently been given a new definition by the International Commission on Radiological Units and Measurements.

TABLE I. DOSE COMMITMENTS FROM NUCLEAR EXPLOSIONS [a]

Tissue	*Source of radiation*	*Dose commitments (mrad)*		*Paragraph of annex A*
		For period of testing 1954–1960 (estimates from 1962 report)	*For period of testing 1954–1962 (new estimates)*	
Gonads	External, short-lived [b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Cs^{137} [b]	8	13	179
	C^{14}	5[c]	13[c]	187
	TOTAL	40	76	
Cells lining bone surfaces	External, short-lived [b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Sr^{90}	67	174	173
	Cs^{137} [b]	14	13	179
	C^{14}	8[c]	20[c]	187
	Sr^{89}	0.15	0.30	176
	TOTAL	116	257	
Bone marrow	External, short-lived [b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Sr^{90}	33	87	174
	Cs^{137} [b]	10	13	179
	C^{14}	5[c]	13[c]	187
	Sr^{89}	0.07	0.15	176
	TOTAL	75	163	

[a] In the 1962 report, these doses were reported in mrems. As explained in paragraph 191 of annex A, the doses in the present report are all given in mrads.

[b] The dose commitments from short-lived nuclides and from internal Cs^{137} have been calculated on a slightly different basis in this report (paragraphs 162, 178 of annex A) as compared to the 1962 report.

[c] For C^{14} it seems to be appropriate to include only the dose which is accumulated up to the year 2000, at which time the doses from the other nuclides will have essentially been delivered in full. The *total* dose commitments from C^{14} from tests up to 1960 for the gonads, cells lining bone surfaces and bone marrow are 48, 80 and 48 mrads, respectively. For all tests up to the end of 1962, the dose commitments from C^{14} are 180, 290 and 180 mrads, respectively.

(2) As in the 1962 report,[9] a comparison could also be made with the natural radiation dose delivered during the period of testing, with the justification that it is the commitment incurred during this period which is relevant, irrespective of the radiation source. However, the latter comparison may also be considered unsatisfactory because the period is not easy to define.

(3) A direct comparison between dose commitments (millirads) and annual dose rates from natural radiation (millirad/year) is hardly justified.

(4) An alternative approach that was also used in the 1962 report[10] and is followed here is to express the dose commitments in terms of the period of time during which natural radiation would have to be doubled to give a dose increase equal to the dose commitment.

16. For all tests carried out before January 1963, these periods amount to approximately 9 months for the gonads, 32 months for cells lining bone surfaces and 20 months for the bone marrow. These periods are not directly comparable with the periods given in the 1962 report because they only take into account that part of the dose commitment from carbon-14 which is delivered before the year A.D. 2000. In addition, the periods given in the 1962 report related to tests during the years 1954-1961 and involved an assumption of testing practice for the year 1961.

[9] A/5216, chapter VI.
[10] A/5216, chapter VI, paragraph 17.

CHAPTER III

RADIATION CARCINOGENESIS IN MAN

1. Among the major problems discussed in the 1958 and 1962 reports was that of obtaining estimates of absolute risk of induction of a number of effects by irradiation at doses and dose rates such as those delivered by natural sources and by fall-out from nuclear testing. In the 1958 report, the estimates of absolute risks that were presented in terms of expected frequencies of given effects per unit dose were tentative and largely hypothetical, and in many cases involved hardly justifiable assumptions in applying the observed results of high doses and dose rates to low doses and dose rates and to different conditions of exposure. For these reasons, in the 1962 report the Committee confined itself to estimating comparative risks. Having again reviewed the available information relating radiation to cancer induction in man, the Committee sees no possibility of changing this procedure at the present time.

2. Data published since 1962 have, however, led the Committee to believe that it is possible, for a few tissues only and mainly in the high dose range, to make estimates of risk (B20)[11] (expressed for example as number of cases per year per rad per million exposed individuals) that are valid within the observed range of doses and the given conditions of irradiation. Furthermore, and especially when the doses studied lie within the range over which the frequency of the effect increases rapidly with rising dose, it is unlikely that the risk per unit dose at very low doses will be any greater than that at high doses and it is likely to be much less. Thus, the estimated risk per unit dose will in most cases represent an upper limit for effects at very low doses (B18, 19).

3. New possibilities of analysing the increased incidence of leukaemias as a function of dose among the survivors of the explosions at Hiroshima and Nagasaki have been offered by a study of a sample of survivors who had been divided in groups according to the estimated doses that they had received. The estimate was made according to distance from the hypocentre and extent of shielding from radiation (B25-30). The accuracy of the dose estimates is difficult to assess, as they might well be affected by some systematic error, in particular that due to our limited knowledge of the relative importance of neutrons and gamma rays delivered during the explosions. The estimates of the doses are, however, almost certainly not in error by a factor greater than two or three.

4. Taking the dose estimates at face value, the average yearly incidence of radiation-induced leukaemia, as determined over a period of nine years, from 1950 to 1958, shows approximate proportionality with the dose in the range from about 100 rads to 900 rads. The rate of increase with dose is between 1 and 2 cases per year per rad per million exposed individuals (B30). It is not known for how long a period of time the increased incidence of leukaemia among survivors will last. There is some indication that the excess has been slightly subsiding during the 1960's.

5. This estimate of absolute risk can only be applied with caution to the population at large. The surviving population has been heavily selected by the lethal effect of the irradiation itself so that the survivors may not necessarily be representative of the irradiated population with respect to sensitivity to radiation carcinogenesis.

6. The estimate obtained from the A-bomb survivors is consistent with that determined, between 300 and 1,500 rads, from a completely different survey of subjects irradiated therapeutically for ankylosing spondylitis (B40-55). In this survey doses were fractionated and are known with greater accuracy, but the number of cases of leukaemia that were observed is very small. Besides, there is no way of knowing to what extent the disease itself for which the patients had been treated, or other means of therapy to which they had been exposed, might have been responsible for the increased incidence of leukaemia. An estimate obtained from this survey alone would therefore only apply to spondylitic patients.

7. The 1962 report dealt briefly with data on induction of malignancies in children irradiated *in utero*. The data were at that time considered as controversial. More recent reports have confirmed a higher incidence of malignancies, including leukaemias, in children irradiated *in utero* (diagnostic irradiation, sometimes repeated) (B62-73). Though precise dose estimates are not available, there is reason to believe that the doses were of the order of a few rads. Risk estimates based on this assumption suggest that the risk of leukaemia per unit dose might be several times higher in children irradiated *in utero* than in adults (B72). These surveys have provided the important suggestion that under certain conditions low radiation doses, of the order of a few rads, can induce malignancy. As in the case of ankylosing spondylitis, there is the possibility that the sample of irradiated children may not be representative of the whole population of children (B73).

8. The 1962 report also discussed data from the Hiroshima tumour registry on the relationship between distance from the hypocentre and over-all incidence of tumours. Further data from the Hiroshima and also from the Nagasaki tumour registry have now been reviewed by the Committee. While these data still indicate a diminishing incidence with distance from the hypocentre, this relationship is now less clear-cut than that derived from earlier reports and does not lend itself to quantitative analysis. Another recent study among Japanese survivors, based on a restricted but more precisely defined population sample, though showing the increased mortality from leukaemia, gave no clear evidence that radiation affected mortality from any other cause of death between 5 and 14 years after the irradiation, though

[11] Throughout the present report, references to the annexes are indicated by a letter immediately followed by a number. Thus B20 refers to paragraph 20 of annex B.

there was some indication of an increased incidence of other malignancies (B175-180).

9. The Committee has reviewed recent surveys on the induction of thyroid carcinoma as a result of irradiation of the thyroid region for therapeutic purposes during childhood (B105-119). The irradiation was often fractionated. As in all instances of therapeutic irradiation, it is not possible to distinguish between the effect of the irradiation and the effect of the conditions for which radiation was administered. The accuracy of the estimates of doses of radiation to the thyroid is not high, but is sufficient to allow some conclusions to be drawn about the relationship between dose and incidence of thyroid carcinoma.

10. As in the case of leukaemia, the incidence of thyroid cancer shows approximate proportionality in a range of doses between 100 and 300 rads, and leads to a risk estimate of about one case per year per rad per million exposed individuals, averaged over a period of approximately sixteen years following irradiation (B117). The period of risk may, however, be somewhat longer. Higher incidence of thyroid tumours has also been reported among adult survivors of atomic explosions (B90-100). The incidence is related to distance from the hypocentre but information is not adequate to provide quantitative assessments of risk.

11. The Committee has reviewed evidence bearing on risk estimates for certain other malignancies; namely, bone tumours in persons contaminated with radium (B130-145), liver tumours in persons who had received thorium compounds for diagnostic purposes (B146-151), skin cancer from external irradiation (B126-129), and lung tumours in miners exposed to radio-active dusts (B152-174). Inadequacies of sampling and dosimetry, longer latent periods and possibly lower likelihood of induction, make unreliable the quantitative assessments based on the information now available. However, the Committee considers that for some tumours, besides leukaemias and thyroid tumours, it might be possible in time to collect enough information to make additional estimates of risk practicable, and that investigations aimed at recording significant quantitative relationships between doses and observed incidence of any specific malignancy in man should be strongly encouraged and supported.

12. It is not to be expected, however, that such estimates will become available for all, or even for many, types of human tissue. The only data suitable for determination of over-all risks of radiation-induced malignancy are those derived from whole body exposure with substantial doses, as in Hiroshima and Nagasaki. The continuation of the latter studies is therefore of great importance. It is still too soon after the exposure of these populations for all possible malignancies to have developed, but present data suggest that leukaemia may well be the predominant type of malignancy produced and that the over-all risk of all malignancies is unlikely to exceed by any large factor that given above for leukaemia (B179, 180).

13. It is important that no opportunity should be lost of exploring the possibilities for undertaking significant studies in exposed human population groups and of pursuing such studies when sound epidemiological techniques can be applied. On the other hand, the usefulness of such data in estimating the effects of very low doses must depend on progress in our understanding of the fundamental mechanisms of carcinogenesis, the mode of action of radiation, and its interaction with other carcinogenic agents in the environment.

ANNEXES

ANNEX A

RADIO-ACTIVE CONTAMINATION OF THE ENVIRONMENT BY NUCLEAR TESTS

CONTENTS

Introduction

1. The purpose of the present annex is to evaluate the information on radio-active environmental contamination from nuclear explosions that became available to the Committee since its 1962 report[a] to the General Assembly[1,b] and to obtain revised estimates of the amount of radiation due to environmental contamination that is received by human populations. An updating is required because further contamination took place after the adoption of the 1962 report, although, following the cessation of atmospheric tests in December 1962, the levels of radio-activity due to short-lived nuclides have decreased substantially through 1963. In addition, improvements in knowledge of the mechanisms involved in the transfer of radio-active material from its production to man's environment deserves attention. The reader is referred to annex F of the 1962 report for those many aspects of the problem of environmental contamination on which little progress has been made since the publication of that report.

[a] *Official Records of the General Assembly, Seventeenth Session, Supplement No. 16* (A/5216); hereinafter referred to as the "1962 report".

[b] Superscripts refer to the corresponding entries in the bibliography at the end of the present annex.

I. Movement of artificial radio-nuclides in the earth's atmosphere

2. The major part of all fission products produced by nuclear explosions up to the end of 1962 was released into the stratosphere. Estimates of future deposition rates require a knowledge of the fission product inventory in the stratosphere as well as of the mechanisms by which it is brought down to the ground. Since the 1962 report a considerable amount of new data on the movement of debris in the atmosphere has been reported. This makes it advisable to review the main features of the processes involved, with special emphasis on recent advances.

RADIO-ACTIVE AEROSOLS

3. After a nuclear explosion, the fission products contained within the fireball are initially present in the form of vapour. As it rises and expands, the hot cloud cools by radiative heat losses, by adiabatic cooling, and by mixing with cooler air, causing the fission products to condense and form an aerosol of fine particles. Since most of the fission product activity injected during 1961 and 1962 was from high yield explosions, the greater part of the fission product debris that was formed was carried up well into the stratosphere.[2]

4. The partitioning of the fission product debris between local, tropospheric and stratospheric fall-out has been discussed in the 1962 report. Most of the radio-active debris produced during 1961 and 1962 was injected into the stratosphere, and this debris, together with that present in the stratosphere from previous tests, was the main source of subsequent world-wide contamination. Local fall-out is important only near the site of tests, while tropospheric fall-out will be deposited within a month or so after tests.

5. Radio-activity created by nuclear tests may be in either gaseous or particulate form. In the high atmosphere, above about 100 km, even very small particles will possess large settling speeds.[3,4] On the other hand, at high altitudes, gaseous substances are subjected to larger molecular diffusion rates than are particulates. In the lower troposphere, particulates are rapidly removed during precipitation. Shortly after a test, particles containing high radio-activity have been observed in ground level air and in fall-out.[29,35] But most measurements in the stratosphere below about 20 km, made many months after the cessation of nuclear tests, suggest that most radio-active particles are submicron in size and thus have negligible settling speeds.[5] The particles, in contrast to gases, may be removed by impaction or settling after coagulation with other aerosols.

6. Other work has shown that there is a correlation between the activity and the sulphate content of samples collected in the stratosphere.[6] This suggests coagulation, or perhaps that sulphate builds up on radio-active particles, which then grow in size. Storebø considered theoretically the growth of particle sizes during the rise of a nuclear cloud and found that terminal sizes may be sufficient for gravitational settling to be of some importance, in comparison with movements due to air exchange.[3] The measured particle size distribution in the stratosphere indicates that the bulk of the debris will be transported at these altitudes largely by air movements. However, in the lower troposphere an important growth in the size of the particles resulting from agglomeration may enhance significantly the rate of aerosol deposition.

Transport within the atmosphere

7. To understand the movement of radio-active debris within the atmosphere, a thorough knowledge of the general circulation of the earth's atmosphere is required. Such a knowledge is necessary to predict the spatial and temporal distribution of future fall-out, as caused by the injection of debris into the stratosphere at different latitudes, altitudes and times. At the present time our understanding of air movements within the stratosphere is incomplete in certain respects, particularly of those at high altitudes. However, some basic features of this motion are now fairly well established.

8. One marked feature of atmospheric circulation is the system of westerly jet streams situated in mid-latitudes at altitudes of about 10 km (figure 1). Velocities of 100-300 km per hour are usual in these regions. In middle and higher latitudes air is carried around the globe in a week or so and in one to two months in tropical regions.[7] Since in the stratosphere these times are short compared to transfer times in the meridional and vertical directions, the debris may be considered to be zonally well mixed so that, several months after a test, it will be uniformly distributed around a circle of latitude.[7] In the troposphere, vertical motions are rapid, but in the lower stratosphere these vertical motions, and hence the vertical transports, are much smaller.[7,8]

9. Fission products injected into the stratosphere during tests have been used extensively to trace air motions.[9,10] In particular, many surveys have been made of the Sr^{90} concentration in stratospheric air,[11] and the activities of W^{185} and Rh^{102} which were injected into the stratosphere during 1958 have also been monitored.[12,13,14] In addition, the distribution of the naturally occurring radio-nuclides Be^{7}, Pb^{210}, P^{32} and C^{14} have also been studied, as well as that of ozone and water vapour.[15,16] These studies have all contributed greatly to our understanding of air movements within the stratosphere.

Movement within the stratosphere

10. Measurements of ozone and water vapour concentrations in the stratosphere show that there is a poleward and downward transfer of material during winter and in the early spring months.[16,17,18] The distribution of ozone in the lower stratosphere suggests that there may be an upward motion of air in the equatorial regions of the lower stratosphere.[16] From the movement of W^{185} from equatorial to polar regions during 1958 Feely and Spar concluded that large-scale eddy diffusion was mainly responsible for this poleward transfer.[19]

11. Eddy diffusion would also explain the movement into equatorial regions of Sr^{90} and Mn^{54} injected into the north polar stratosphere in late 1961. However, Newell argues that the transfer along sloping surfaces (figure 1) must be due to both eddy mixing and mean meridional motions.[20] By studying the time trends of Rh^{102} in the stratosphere at high latitudes, Telegadas and List found that the debris descended from 20 km to 14 km between December 1959 and March 1960 and then remained stationary during the summer of the northern hemisphere.[21] A similar rate of descent was noted for Cd^{109} in the southern hemisphere between April and August 1963.[380] These observations suggest that downward motions during winter are mainly responsible for the vertical transport of fission products in the polar stratosphere.

12. It may be concluded that debris injected into the equatorial regions of the stratosphere below 30 km will move polewards and downwards into each hemisphere during the winter months. Material injected into the lower polar regions does not seem to move upwards to any great extent, but some of it moves into equatorial regions of the lower stratosphere. A number of models of stratospheric circulation have been proposed, but at the present time none of them is completely adequate to predict the transport of fission products.[9,31,412] At the moment it seems that both advective and diffusive processes are important in stratospheric circulation. The stratospheric transfers described above are shown in figure 1.

13. Very few data concerning the movement of air above 30 km are available. Rh^{102} was injected into the stratosphere by an explosion at about 43 km over Johnston Island during August 1958.[23] The radio-active cloud was estimated to have risen to at least 100 km. Concentrations of Rh^{102} at an altitude of 20 km in the northern hemisphere showed a large increase during the period October 1959-February 1960.[12,21] In the southern hemisphere the major increase in concentration occurred during the winters of 1959 and 1960, the concentration in this hemisphere being much the same as in the northern hemisphere after mid-1960. During 1960 and 1961 the concentrations at these altitudes remained fairly constant, probably being replenished from above.

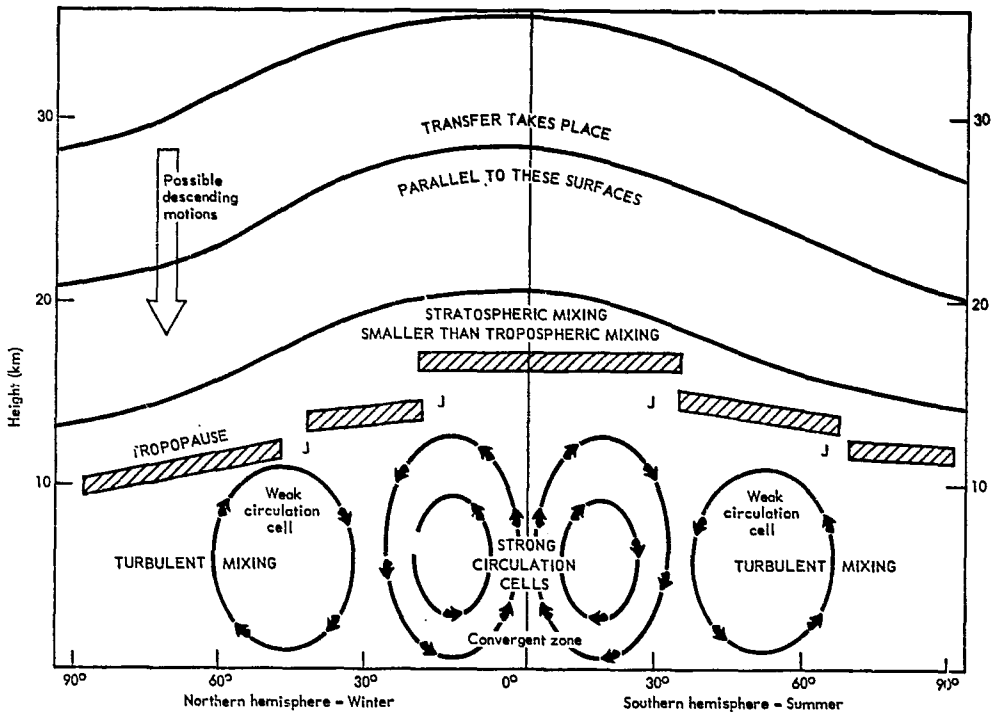


Figure 1. Schematic cross section displaying characteristics of meridional transport ("J" locates typical jet stream positions)

14. Recent stratospheric measurements of Cd^{109} activity, about 0.25 MCi of which was released above 400 km over Johnston Island at 17°N latitude in July 1962, show that some of this isotope moved down to 30 km after five or six months.[22, 24, 380] Not only did Cd^{109} appear earlier in the lower stratosphere of the southern hemisphere than in the northern hemisphere, but it was present there in greater quantities until August 1963—at least in mid-latitudes. At altitudes between 40 and 400 km, gravitational settling and, in the case of charged particles, electro-magnetic effects are expected to influence the descent of fission product debris.[53] Measurements of Rh^{102} originating from the high altitude rocket explosion (Orange), which was carried out during August 1958 in equatorial regions, indicate that Rh^{102} spread to the polar regions in both hemispheres and later descended to lower altitudes.[25, 26, 381]

15. There are many mechanisms which might account for the transfer of particulate radio-activity from the stratosphere to the troposphere. Machta compared the likely transfer of Sr^{90} by each mechanism with the subsequent observed Sr^{90} fall-out in the northern hemisphere in early 1960 and 1961.[34] The results can be summarized as follows:

(*a*) Heavy particles can settle through the tropopause, but the bulk of the radio-activity in the lower stratosphere is contained in particles too small for gravitational settling to contribute significantly to the downward transport through the tropopause.

(*b*) Vertical mixing through the tropopause and horizontal exchange through the tropopause gap (figure 1) could each account for the transport out of the stratosphere and thus explain the subsequent fall-out. The calculation assumes that a coefficient of diffusion represents the proportionality factor between the flux of Sr^{90} and the gradient of Sr^{90}. This diffusion coefficient describes various complex meteorological processes only grossly. There is also considerable uncertainty regarding the magnitude of the coefficient. The equality between diffusive transfer and subsequent fall-out is not necessarily convincing evidence of the reality or magnitude of this transport mechanism.

(*c*) The tropopause in temperate and polar regions rises to higher altitudes in the late winter and in the spring. If it is assumed that this process transfers stratospheric air into the troposphere, then its associated Sr^{90} may significantly contribute to subsequent fall-out. In 1960 and 1961 this mechanism could account for no more than one-quarter of the observed fall-out.

(*d*) Certain models of atmospheric circulation, such as the Brewer-Dobson model, imply descending motion through the tropopause in certain areas. Some of these models postulate subsiding movements whose downward transport of Sr^{90} can contribute part or all of the observed fall-out. But, as of this time, the reality of the model as well as the sense and magnitude of the vertical currents must be considered as questionable.

(*e*) Danielsen measured the radio-activity in certain thin layers of air at tropospheric altitudes. He demonstrated that these active layers represented parcels of stratospheric air extended into the troposphere.[32, 33] This process, a folding of the tropopause, is shown schematically in figure 2. After the "tongue" of stratospheric air with its high concentration of radio-activity is brought across the tropopause, diffusive mixing incorporates it into the troposphere. A quantitative estimate of the amount of Sr^{90} transferred into the troposphere made by Machta, using Staley's estimates of the frequency, intensity and areal coverage of the extrusion, suggests that this process could account for not more than about one-third of the observed stratospheric fall-out.[30, 34]

16. To predict the future deposition of long-lived fission products, the concept of mean residence time is useful. It is defined as the average time spent by fission products in the stratosphere before being transferred to the troposphere. Such a definition in no way implies that the material is well mixed within the stratosphere or that the hold-up during the transfer to the troposphere occurs

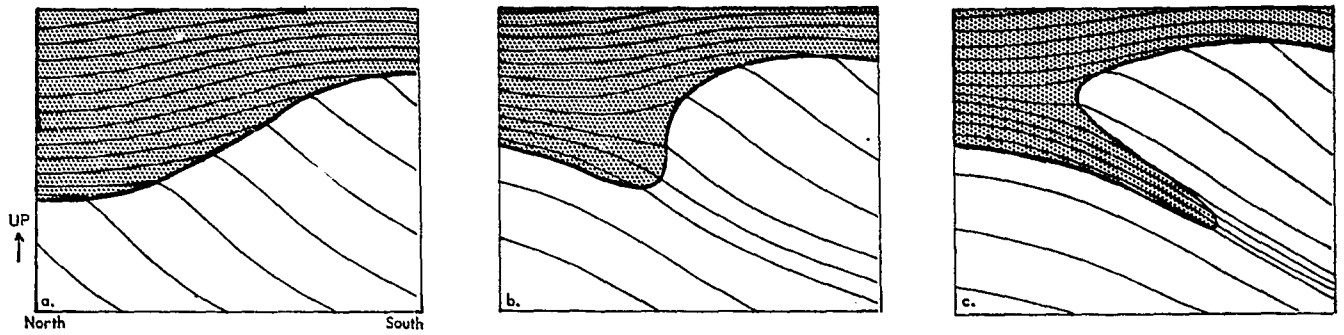


Figure 2. a, b, c. Vertical cross section, north to the left, showing in successive stages the steepening and folding of the tropopause. The thin lines are potential temperature isotherms. Air parcels tend to move along such isotherms during transit times of a few days[33]

at the tropopause.[7] Such conditions, which are needed to validate the use of first order kinetics, are not applicable to the stratosphere-troposphere transfer. However, the concept of a mean residence time is used to predict future fall-out.

17. Mean residence times may be computed for the transfer of stratospheric debris to the troposphere from published stratospheric inventories and annual deposits of Sr^{90} and W^{185} Three techniques were employed to produce the estimates in table I, as noted below the table. Each technique is subject to uncertainties. Method *b*, in particular, can yield meaningless residence times if the stratospheric distribution changes between measurement periods. The mean residence times for one hemisphere are unreliable if there is a significant transfer between hemispheres. Despite this, all three methods are consistent in suggesting longer mean residence times for the southern rather than for the northern hemisphere.

18. A periodicity of two years or of twenty-six months has been found in many meteorological parameters of the lower stratosphere.[38] This cycle appears in the ozone content of the atmosphere over Australia[37] and in the Be^7 concentration of the stratosphere of the southern hemisphere.[408] It is possible that fall-out may be partially modulated by the same cycle, at least in the southern hemisphere.

19. The mean stratospheric residence time of radioactive debris produced by an individual explosion will depend on the altitude, latitude, and possibly the time of injection. Thus, fission products in the lower polar stratosphere may have a mean residence time of six months or less, while debris from medium altitude explosions have mean residence times of perhaps two to three years.[39] At much higher altitudes, over 100 km, as illustrated by the Rh^{102} experiment, the residence time increases to five or ten years.[12,381,401] Theoretical models of transport and diffusion can qualitatively reproduce these differences. One such preliminary model by Bolin,[7] created to fit the observed ozone observations, produces reasonably plausible fall-out patterns as well. Ultimately, it is likely that predictions from such meteorologically consistent models will form the basis for fall-out forecasts.

20. The stratospheric distribution in January 1964 is roughly the same as in 1960 or early 1961. It is therefore considered reasonable to apply the observed mean residence times found during 1960 and 1961 to the stratospheric inventory in 1964 for the predictions in this report. Table I summarizes the mean residence times found in the 1960-1961 period. An average global value of two years is chosen for purposes of predicting Sr^{90}, Cs^{137} and C^{14} contamination after 1963. It is likely that, as the concentrations in the stratosphere become more uniform because of mixing, the mean residence time may tend to increase. The use of a constant rather than an increasing mean residence time will slightly over-estimate the doses due to Sr^{90} and Cs^{137}.

Movement within the troposphere

21. Once the radio-active debris enters the troposphere it is mixed fairly rapidly within the hemisphere of entry. Within the troposphere mixing by eddy diffusion and convection is much more rapid than in the stratosphere. As will be shown later, the Sr^{89}/Sr^{90} ratio in monthly precipitation was fairly constant in the different latitude bands of the northern hemisphere, between September and December 1961.[27,376] Between 10° and 70°N the meridional mixing rate is rapid compared to the half-life of Sr^{89}, 50.5 days. As in the stratosphere, the fission product debris is mixed zonally quite rapidly. At latitudes of 40°N the air takes some twelve days to move around the earth in a westerly direction.[40] In the meridional plane there are two circulation cells within each hemisphere as shown in figure 1. In the tropical regions this cell is well developed with air rising in equatorial regions and descending into the 20°-30°N latitude region. At higher latitudes there is a weaker circulation cell with descending air at latitudes of 40°-50° and rising air at higher latitudes, while at middle and higher latitudes large scale eddies give rise to rapid meridional transport.

22. The gross beta activity of ground level air is measured at many stations throughout the world. These measurements, although of importance for surveillance purposes and also for meteorological studies, are of little value in estimating radiation doses from fission products. The activities in air of individual fission products have been reported from a number of countries.[41-47] Of particular interest are the activities in air measured at stations around the 80th meridian west. Figure 3 shows the mean bi-monthly Sr^{90} activities in the northern and southern hemisphere stations during the years 1958-1963.[28,378,379] During 1960 and part of 1961 when there was little testing, the activities of Sr^{90} in air in the two hemispheres tended to equalize. Sr^{90} activity in the northern hemisphere showed large peaks in the spring of each year, but in the southern hemisphere these peaks were not so marked.

23. A rapid rise in the Sr^{90} activity of air occurred in the northern hemisphere in late 1961 after testing was resumed. In the southern hemisphere the rise was slight until April 1962, when equatorial tests were resumed in the Pacific. Lockhart and Bleichrodt[41,50] have reported the detection of debris from these Pacific tests in mid-latitudes of the northern hemisphere, and others have

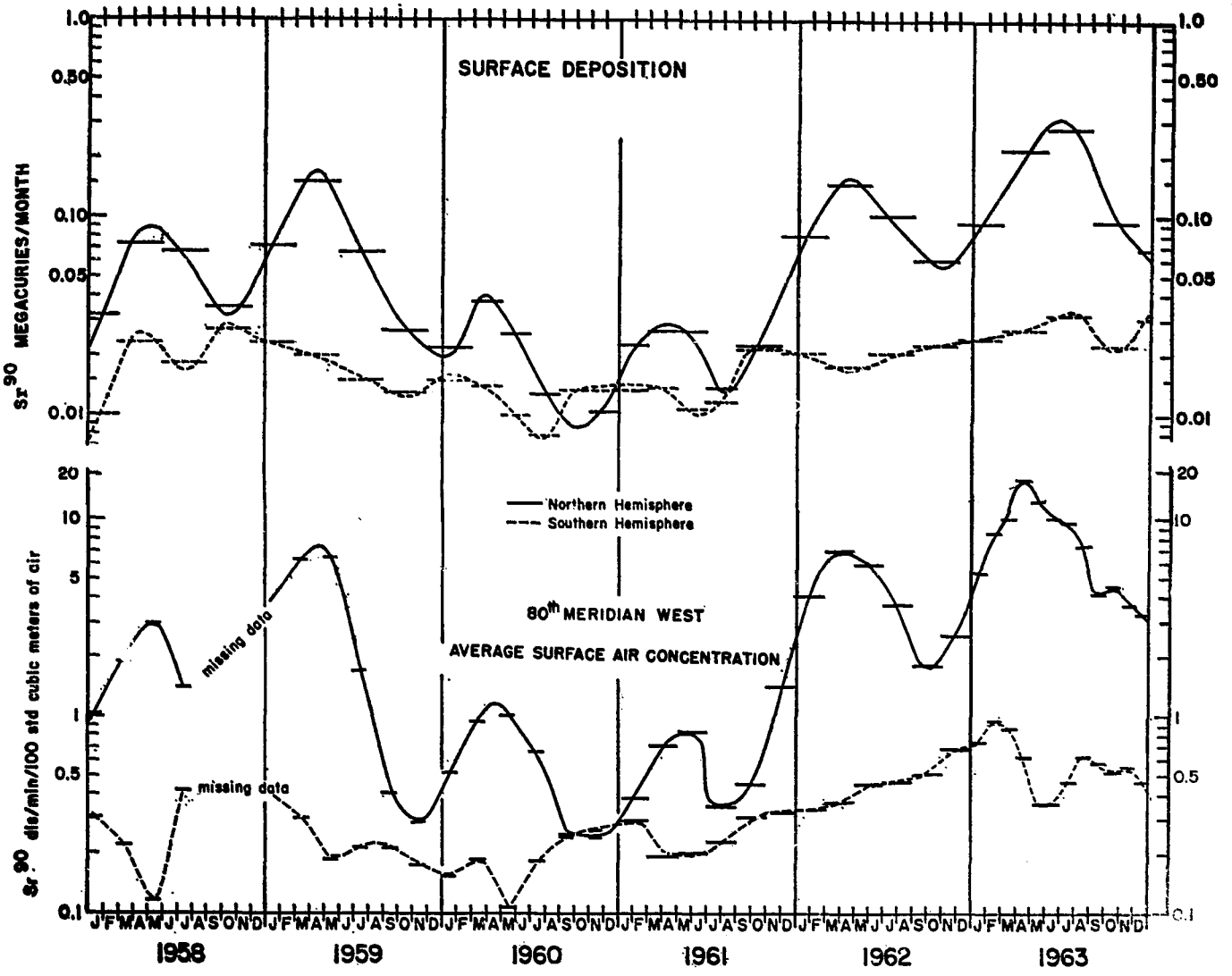


Figure 3. Strontium-90 deposition rate and its concentration in surface air[28, 378, 379]

reported the detection in May-June 1962 of Ba^{140} activity in rain at Westwood, New Jersey, United States.[48–50] Similar evidence has been noted in measurements made in the United Kingdom.[45] In 1962 the average Sr^{90} air activity in the northern hemisphere reached a peak of 0.03 pCi/m³ and a peak of 0.1 pCi/m³ in 1963. Measurements of total beta measurements in air within smaller regions of the earth's surface, e.g. in Norway, the United Kingdom and in the United States, indicated that the average activity in surface air did not vary greatly from place to place, in spite of large differences in rainfall.[45, 51, 52]

24. In the troposphere, the exchange of particulate radio-activity across the meteorological equator is impeded for two reasons. The first, convergence of low altitude air currents (figure 1), tends to keep air in the same hemisphere and also retards the exchange of gases. The second reason is the scavenging of particulates by showery precipitation in the convergent zone. A north-south profile of Sr^{89} along the 80th meridian west between September and December 1961 shows (figure 4) a sharp decrease in the convergent zone near the geographical equator.[41] This short-lived activity originated from explosions in temperate or high latitudes of the northern hemisphere. By contrast, figure 4 also displays the distribution of Sr^{90} whose origin is mainly the stratosphere of each hemisphere. The north-south distribution of Sr^{89} during and following nuclear explosions as shown in figure 4 is confirmed by profiles reported by Krasnopevtsev along 170°E over the Pacific Ocean (the profile for Zr^{95} being also shown in figure 4) and by Labeyrie and Lambert in the eastern Atlantic Ocean.[47, 55]

25. In the troposphere, the mean exchange time between hemispheres, defined as the mean time spent in the northern hemisphere by a molecule of air before transfer to the southern hemisphere, and *vice versa,* has been estimated by using various gaseous tracers.[54, 56–58, 409, 410] These estimates are listed in table II. The exchange times shown in table II are fairly consistent, except that based upon tritiated methane, and indicate that the exchange time is about 1.5 years.

26. Stewart estimated a thirty-day mean residence time for fission products injected into the troposphere.[59, 60] Evidence now suggests that in the lower, rain-bearing, layers of the atmosphere particulates reside for a period of the order of five days or less.[62] But for particulates located well above the rain-bearing layers, the residence time may be as high as forty days.[15, 60]

Mechanisms of deposition

27. After entering the troposphere from above, fission products are transported down to the level of the rain-

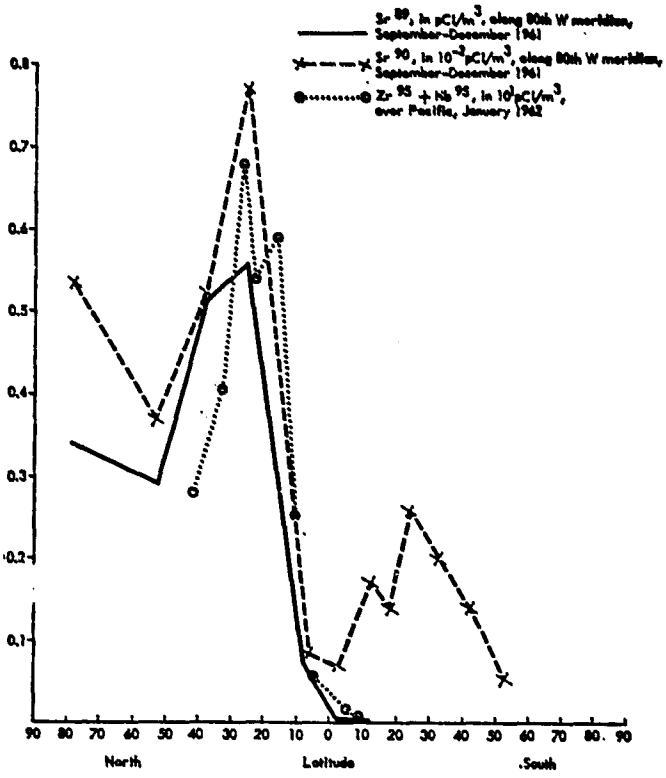


Figure 4. Latitudinal variation of the concentration of fission products in surface air[43, 47]

bearing clouds mainly by turbulent mixing. This downward movement is enhanced over anti-cyclonic systems and opposed over cyclonic systems.[32] Below this level the radio-active particles are rapidly washed out by precipitation and deposited upon the earth.[65] In addition, dry removal of fission products takes place through several mechanisms. Dry removal by sedimentation requires particles to be larger than about 5 microns and is important only in local fall-out. Dry deposition of world-wide fall-out makes an important contribution to the total fall-out only in areas of low rainfall.

28. The fission products can enter rain-water by processes within the cloud, the so-called rain-out, or by pick-up by raindrops below the cloud, the so-called wash-out. For aerosols of small particle size the wash-out process is relatively quite slow so that rain-out is probably the most important wet deposition process.[9, 63] The small contribution of wash-out processes to total deposition probably accounts for the fact that the activity of fission products in ground level air does not seem to be greatly influenced by precipitation rates.[51, 52] The rain-out of fission products may be enhanced by the presence of natural aerosols in the lower atmosphere, and it has been suggested that higher concentrations of sodium chloride in the maritime atmosphere may enhance the deposition rate over oceans.[63]

29. Experience indicates that time-averaged Sr^{90} deposition is roughly proportional to the amount of precipitation.[67] The relationship is only approximately valid when widely separate stations are compared, as for example all stations in a latitude band. The relationship becomes better when the stations are limited to the same general climatic region and becomes very good when the stations are close to one another. An example of the latter appears in figure 5 where the cumulative Sr^{90} soil deposition in Clallam County, Washington, United States in 1960 is plotted against precipitation.[66] In this case, the linear relationship between deposited activity and precipitation confirms the almost constant specific activity in rain at all these sites.

30. One method of calculating the dry fall-out in a region having places with variable amounts of precipitation depends on obtaining a relationship between deposition and the amount of precipitation, and extrapolating the relationship to zero precipitation. Thus, if the straight line in figure 5 is extended to zero rainfall, a dry deposit of 7 mCi/km² is obtained in 1960 on the West Coast of the United States. A similar analysis of the cumulative Sr^{90} deposit in Norway during 1959 yields about 5 mCi/km² due to dry deposition.[69] This indirectly observed amount of fall-out computed by extrapolation of fall-out precipitation curves to zero precipitation may express the maximum dry fall-out. Miyake argues that the fall-out precipitation relationship departs from a straight line towards lower fall-out at very low precipitation amounts.[61] It appears likely that the amount of dry deposition and the specific activity of Sr^{90} in precipitation will vary with climatic conditions and the air concentration. Many factors affect dry deposition such as micro-turbulence and the extent of vegetation cover.[64]

II. Inventory and deposition of artificial radio-nuclides

Strontium-90

31. Inventory estimates for individual nuclides would ideally be based on a knowledge of the amounts injected into the atmosphere. These can be deduced from the yields of the explosions and such estimates of yields have been published (table III)[70] but their reliability cannot be assessed on the basis of the information available to the Committee. In the present report, therefore, as in the

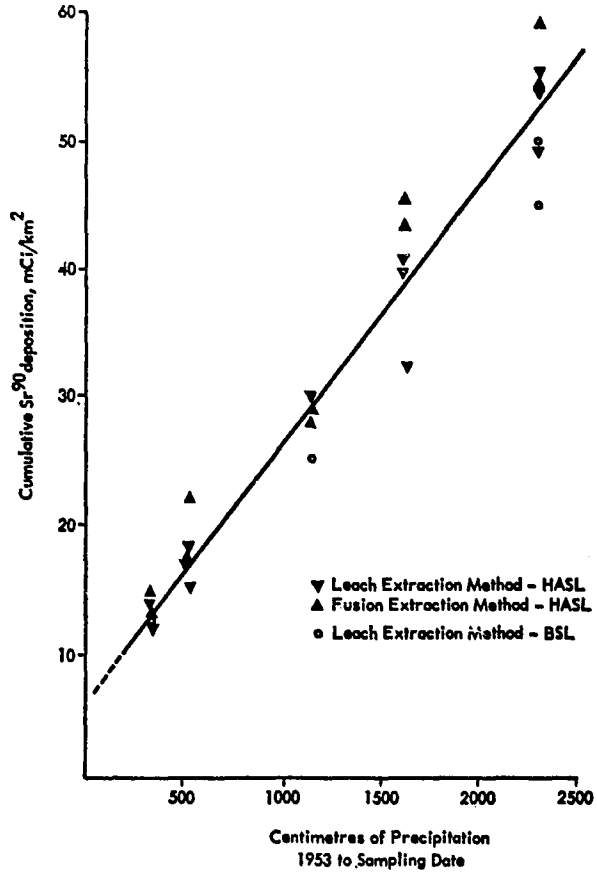


Figure 5. Strontium-90 deposition determined from soil analysis in 1960 as a function of precipitation[66]

1962 report, inventories will be estimated from actual measurements.

Atmosphere

32. To estimate the Sr^{90} inventory in the stratosphere, a considerable number of air samples have been collected by aircraft between the tropopause and 21 km.[11, 14, 71, 72] Between 21 and 30 km inventories were established through balloon sampling at two sites only, over San Angelo, Texas, United States, and over Mildura, Australia.[73] Estimates of the Sr^{90} inventory above 20 km must be regarded as somewhat approximate. In estimating the stratospheric inventory from these spot measurements, it is assumed that there is fairly rapid zonal circulation in the stratosphere and hence that the specific activity depends solely upon latitude and altitude at any one time.[23] Sr^{90} activities in the stratosphere at different altitudes and latitudes during 1961 and 1963, are shown in figures 6, 7, 8 and 9.[22, 23, 34] Isolines are used for integration purposes in estimating the total inventory. For the balloon data, only the crudest integration is possible.

33. Figure 6 shows that the Sr^{90} concentrations in the troposphere are several orders of magnitude less than in the stratosphere. To estimate the tropospheric inventory of Sr^{90}, an average value of activity is assumed for each hemisphere. The large concentration gradients present in the stratosphere can cause uncertainties in the inventory estimates. Another possible source of error is the sparsity of data available above 21 km. This is particularly significant in estimating the 1963 inventory, as it is believed that concentrations were considerable above 21 km at latitudes higher than 31°N.[22] It should be noted, however, that there is only 4 per cent of the atmosphere above 21 km, and only 1 per cent above 30 km.

34. To check on zonal uniformity within the stratosphere, Cs^{137} activities measured over the United Kingdom were compared with Sr^{90} activities measured over Canada and the United States, using the ratio 1.7 for conversion. Although this comparison did not conclusively show that there was no zonal variation, no systematic difference was noted.[23] It has been estimated that the over-all error in the stratospheric inventory of Sr^{90}

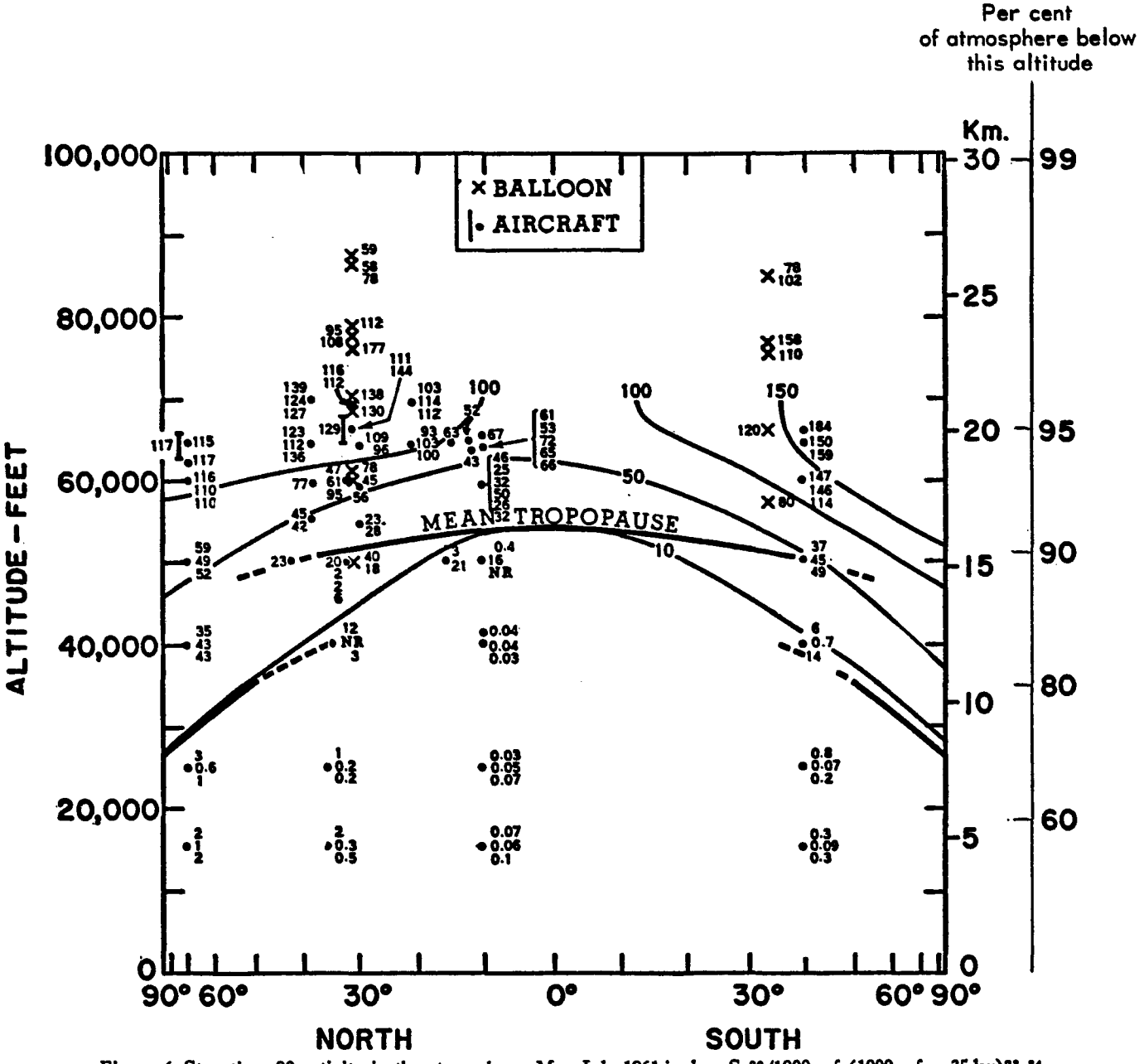


Figure 6. Strontium-90 activity in the atmosphere, May-July 1961 in dpm Sr^{90}/1000 scf (1000 scf = 35 kg)[23, 34]

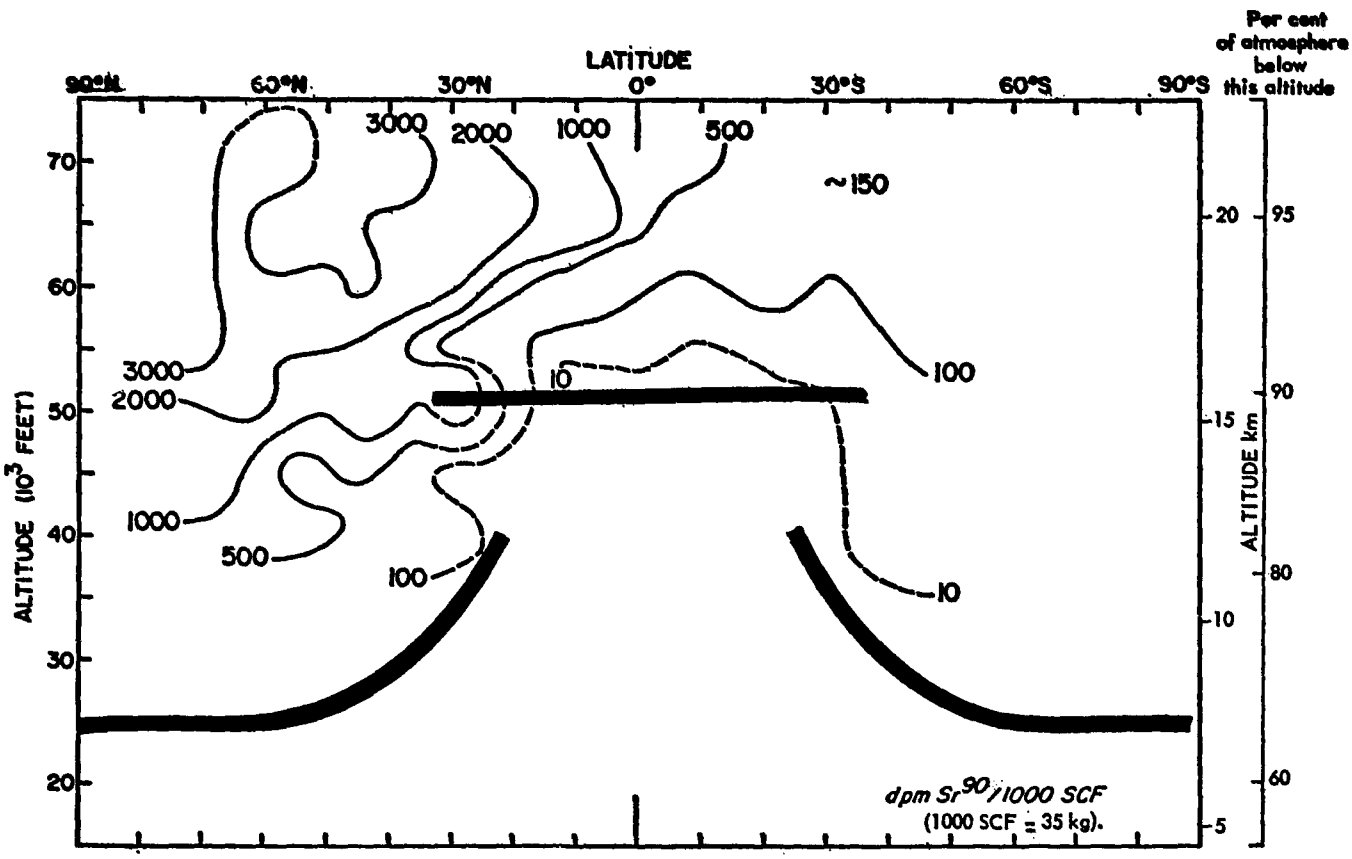


Figure 7. Strontium-90 activity in the atmosphere, December 1962-February 1963[23, 34]

is ± 50 per cent at a probability level of 90 per cent.[23] Table IV shows the global Sr^{90} inventory for different years up to January 1964.[22, 23, 34, 377]

Measurements in precipitation and soils

35. Much information has been published on Sr^{90} fall-out and the Committee has received much data from many countries throughout the world.[26, 42, 45, 46, 74–104] As an example, the monthly Sr^{90} deposition at New York City is shown in figure 10.[75] Figure 11 shows the cumulative Sr^{90} deposit at a number of sites.[45, 59, 68, 69, 75, 76, 89, 96] The monthly and annual average Sr^{90} fall-out for different regions of the Soviet Union during 1961, 1962 and 1963 are shown in table V.[81, 418] The deposition measurements from the network operated by the United Kingdom Atomic Energy Authority[45] are consistent with the results shown in table VI and obtained from the more extensive network operated by the United States Atomic Energy Commission. Figure 12 shows the 1962 latitude distribution of Sr^{90} deposition.[27]

36. The annual deposition in each 10° latitude band between 50°S and 80°N during 1961, 1962 and 1963 is shown in table VI.[27, 105, 376, 419] Few data are available for the higher latitudes, but the deposition there does not contribute appreciably to the dose commitment since the population density at these high latitudes is small. The totals were computed by averaging the annual deposition at the fall-out stations in each latitude band. Telegadas[28, 407] has compared the results of this calculation with those obtained by multiplying the specific activity of Sr^{90} in rain by the average annual rainfall for the latitude band. He found little difference between the results of the two methods of estimation.

37. The annual Sr^{90} depositions in each hemisphere for 1961, 1962 and 1963 are shown in table VII, together with the cumulative deposit up to December 1963. The latter figures were computed by adding in the cumulative values for 1960 from the 1962 report and correcting for decay. In 1963, 2.5 MCi of Sr^{90} were deposited upon the earth's surface, the highest annual deposit ever recorded.

38. Figure 3 shows the Sr^{90} fall-out rates in each hemisphere, together with Sr^{90} air concentrations for the years 1953-1962.[28, 378, 379] A strong correlation between Sr^{90} deposition and air concentrations is apparent. The Sr^{90} deposition rate in the northern hemisphere has been consistently higher, sometimes by one order of magnitude, than that in the southern hemisphere, and only during 1960 and early 1961 did the rates in both hemispheres tend to equalize.

39. Estimates of the cumulative deposition of Sr^{90} have in the past been largely based upon the measured Sr^{90} content of soil samples taken to sufficient depth to ensure that most of the accumulated Sr^{90} is recovered.[68, 106] The results of a new global survey of Sr^{90}, compiled in 1963, have become available since the adoption of the 1962 report.[103] The Sr^{90} analysis of soils at 96 sites has now been completed[103] and the cumulative deposits at these sites are displayed on a world map in figure 13. The cumulative deposit of Sr^{90} in each hemisphere has also been estimated using the Sr^{90} monthly deposition rates at stations of the United States global fall-out network.[27, 105, 379] The cumulative deposit of Sr^{90} in each hemisphere so obtained is plotted in figure 14 for the years 1954-1962.

40. Estimates of cumulative deposits of Sr^{90} in latitude bands, obtained from the 1963 soil survey, are compared in table VIII with estimates based upon measurements in precipitation for the years 1961-1963 combined with soil data for 1960. The two sets of estimates agree reasonably well, except in the latitude bands 10°-20°N and

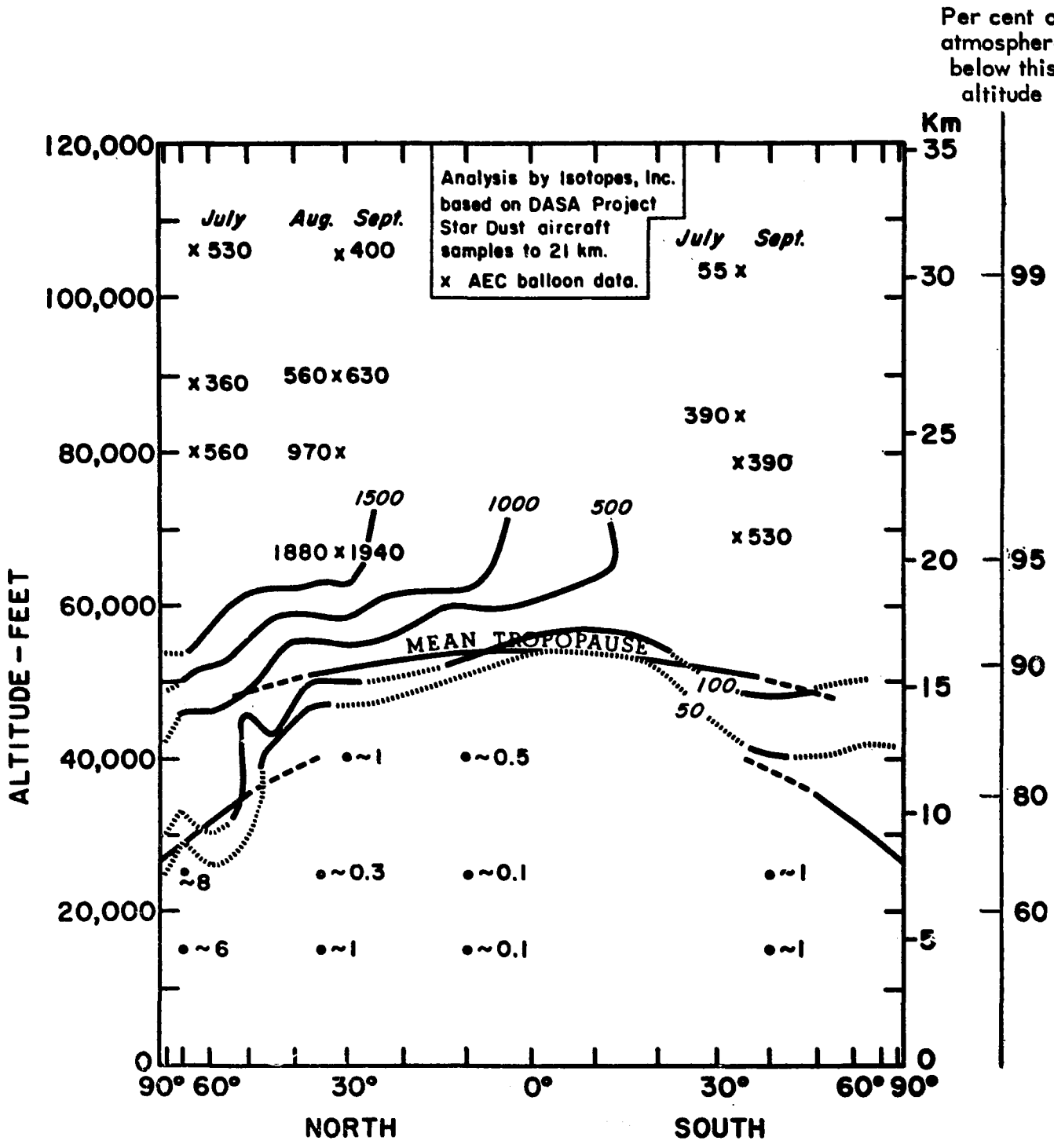


Figure 8. Strontium-90 activity in the atmosphere, September 1963 in dpm Sr^{90}/1000 scf (1000 scf = 35 kg)[23, 34]

20°-30°N, where the number of soil sampling sites was quite small. This conclusion is in agreement with the results of the 1960 Sr^{90} soil survey, which showed that, in the 20°-70°N latitude band, estimates of deposition obtained from precipitation data did not differ significantly from those obtained from soil data. For the calculation of doses, the mean value 9.6 MCi is used.

41. The extent to which results for the world-wide network of soil sampling sites correctly represent the global deposition cannot be precisely assessed. However, a detailed analysis of possible causes of error[23] suggests that uncertainties in this regard are likely to be small relative to those in the assessment of other parameters. There may be a systematic bias in the Sr^{90} global deposition estimate which could in particular be caused by the geographical distribution of the sampling sites. Possible inequality between fall-out rates over oceans and continents could also give rise to a corresponding systematic error.

Rivers and lakes

42. The Sr^{90} and Cs^{137} concentrations in the rivers of several countries have been reported.[52, 107-109, 383] Measurements on waters of Lake Grosetvann in Norway during 1958 and 1959 gave Sr^{90} concentrations about 10 per cent of those in precipitation. During early and late winter, however, with the melting of snow, the Sr^{90} concentrations of the inflowing waters increased substan-

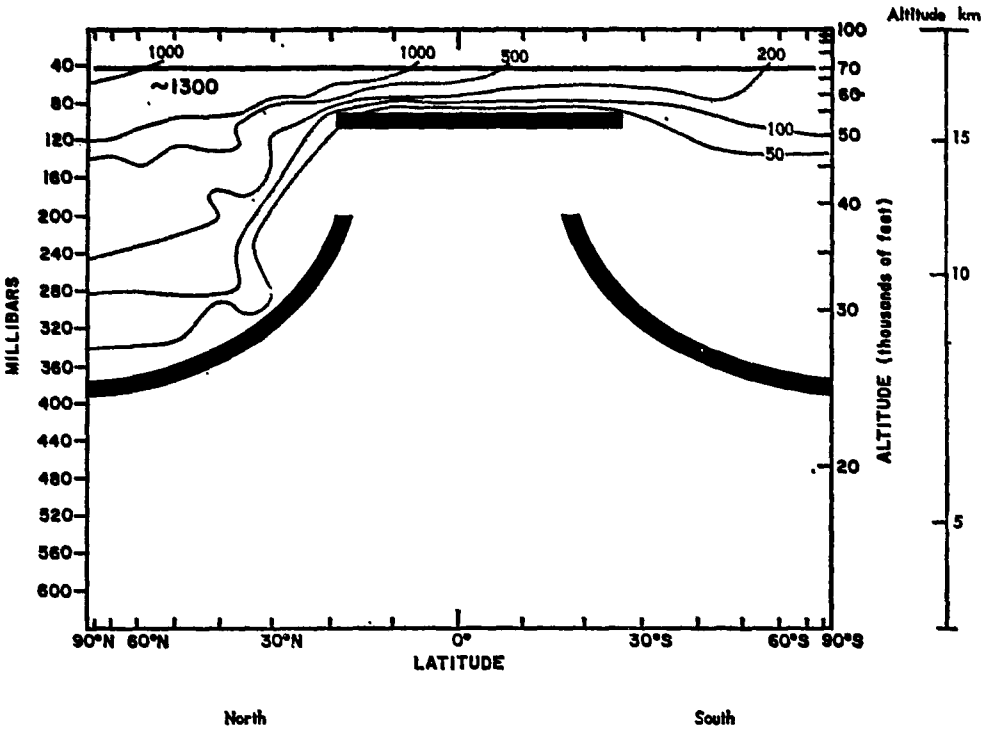


Figure 9. Strontium-90 activity in the atmosphere, January 1964 in dpm Sr^{90}/1000 scf (1000 scf = 35 kg)[22]

tially, indicating that during these periods surface water was exchanging less of its Sr^{90} with soil before moving into the lake. A similar result was noticed in river waters of the Soviet Union, where the Sr^{90} activities measured during flooding were 5-10 times greater than during the low water period.[108] Average values for the Sr^{90} concentrations in 45 rivers of the European part of the Soviet Union were 0.5 pCi/l during the second half of 1961 and 0.9 pCi/l during the first three quarters of 1962. The average values in 37 rivers of the Asian part of the Soviet Union were 0.6 pCi/l and 0.8 pCi/l, respectively.

43. Both this study in the Soviet Union and a similar study on Sr^{90} in the river waters of Japan indicate that some 5-10 per cent of freshly deposited Sr^{90} is carried off in river waters, but the removal rate of the cumulative deposit of Sr^{90} is much smaller, being in the range 0.2-1.5 per cent per year.[107,403] It can be concluded that most of the Sr^{90} that is taken up by the soil will remain there until it decays.

Oceans

44. Oceans cover some 60 per cent of the earth's

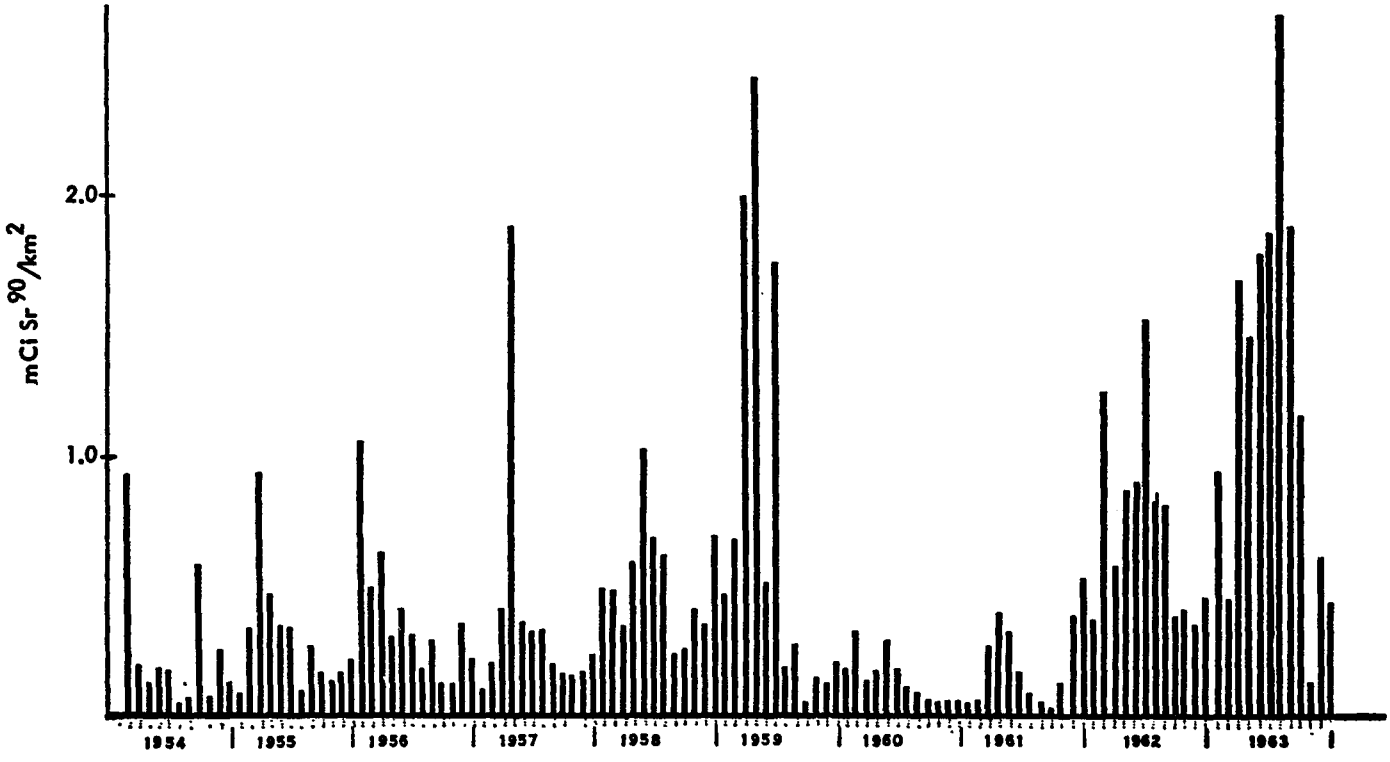


Figure 10. Monthly strontium-90 deposition in New York City[75]

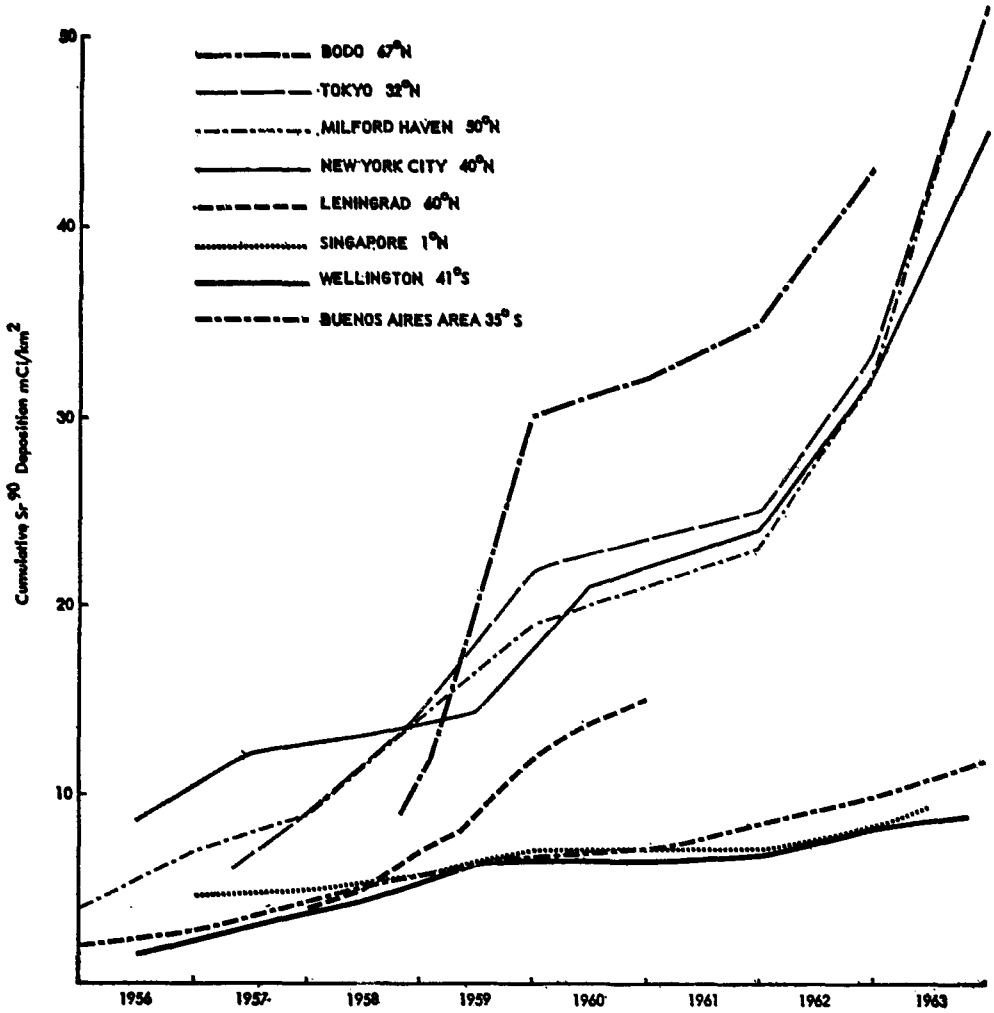


Figure 11. Increase with time of the cumulative deposition of strontium-90[27, 45, 68, 69, 89, 96, 426, 429]

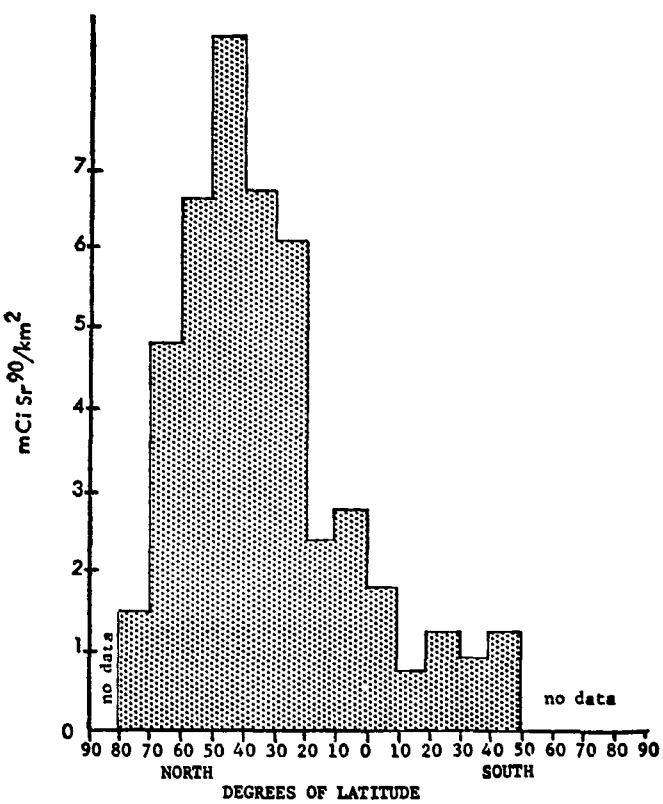
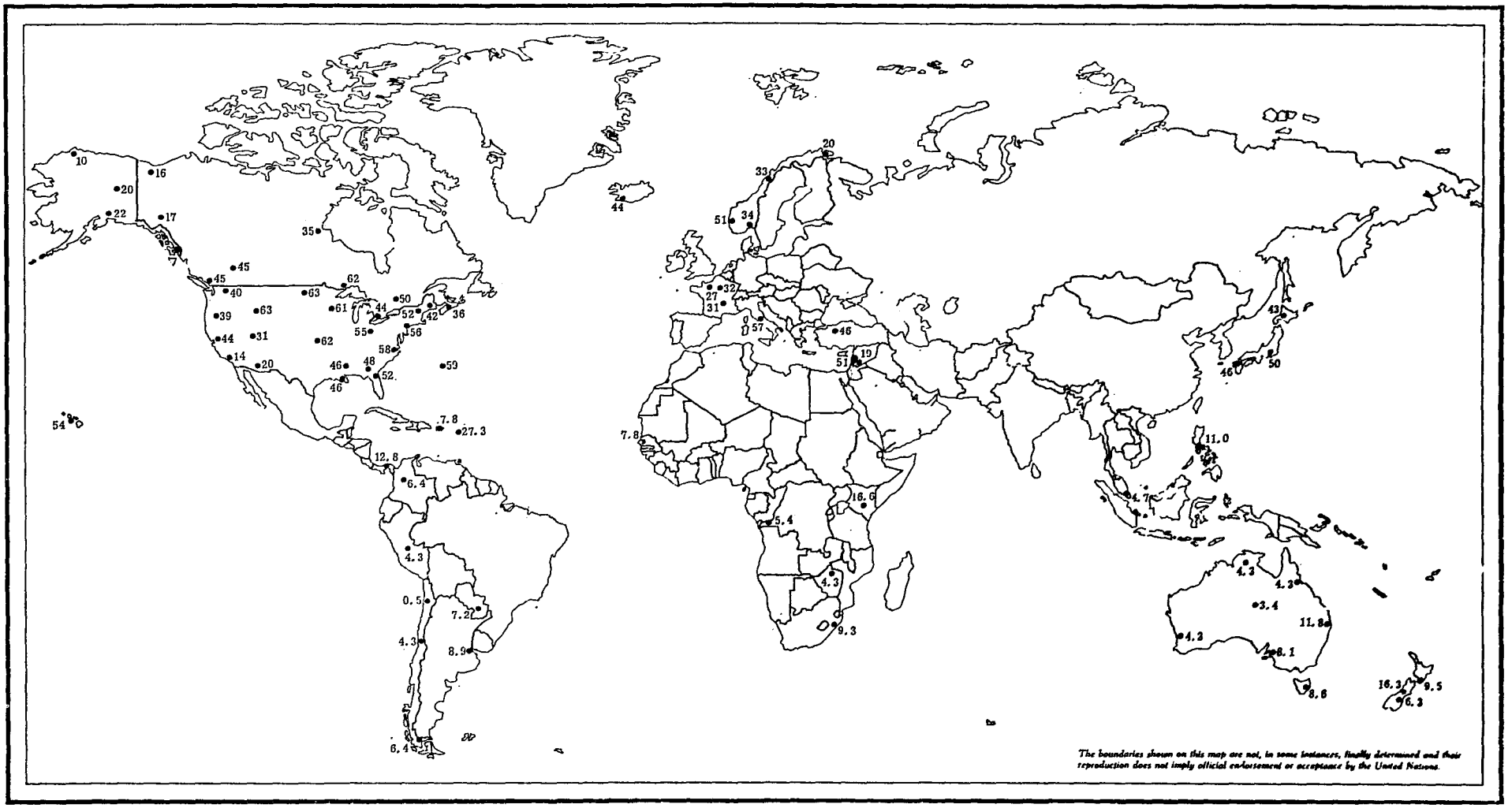


Figure 12. Total strontium-90 deposition during 1962 versus latitude band[27]

surface in the northern hemisphere and about 85 per cent in the southern hemisphere. Since predictions of the future fall-out of Sr^{90} over land are based upon the atmospheric inventory, any difference between the fall-out rates over oceans and over land could cause a corresponding error in future fall-out predictions.

45. Some measured concentrations of Sr^{90} in the Pacific, Atlantic and Indian Oceans during the last five years appear to be too high to be accounted for by the same fall-out rates that are measured over the land.[110] Bowen and Sugihara in 1957 and 1958 found that Sr^{90} was well mixed in the top 100 metres of the Atlantic Ocean.[111] Between 100 and 400 metres, there was a steep gradient in concentration with appreciable concentrations at depths greater than 1,000 metres. Later measurements appeared to confirm the presence of Sr^{90} activity at these great depths.[112, 117] Some profiles of Sr^{90} concentrations in the Atlantic Ocean in 1960 and in 1961 are shown in figure 15. Shvedov, using some of Bowen's results, estimated that the cumulative Sr^{90} fall-out over the Atlantic in the 30°-40°N latitude band was between 28 and 42 mCi/km² in 1957 and in 1958, against an estimated 10 mCi/km² from measurements over land.[113] It has been reported that the Sr^{90} concentrations in the deep waters of the western Pacific were even higher than those measured in the Atlantic.[404]

46. Rocco and Broeker, however, reported profile measurements for Sr^{90} and Cs^{137} in the Atlantic and Pacific Oceans that showed little activity below 300 metres.[114] At levels below 1,000 metres a considerable



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SEPTEMBER 1964

Figure 13. Cumulative deposition of strontium-90 determined from soil samples collected in 1963, mCi/km^2 [103]

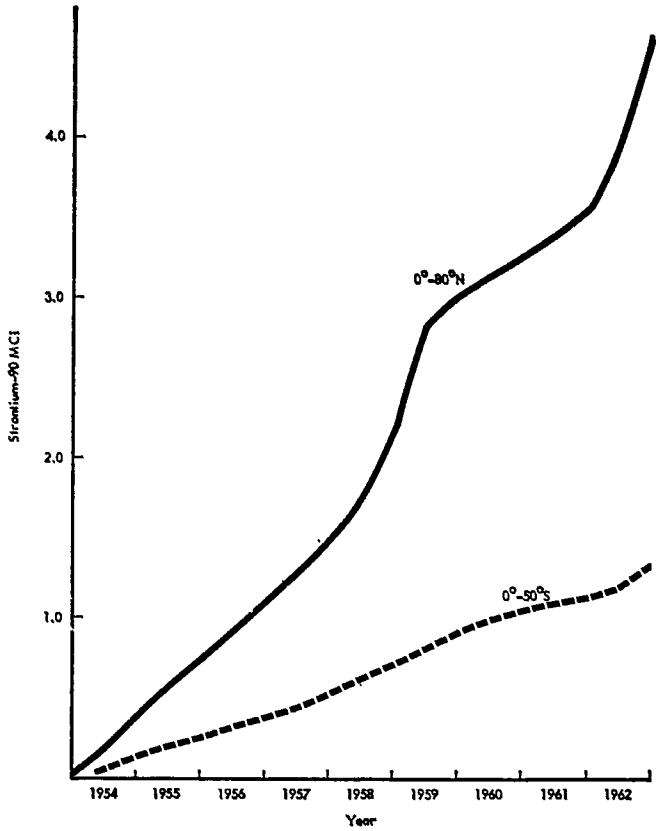


Figure 14. Cumulative deposition of strontium-90 (based upon measurements at the United States fall-out collection network stations. Before 1958 the number of collection stations was relatively small)[27,104]

increase in the concentration of these nuclides was observed, but these measurements were not considered to be sufficiently reliable by the authors. Only in the Antarctic Ocean did they consistently detect significant activities of Sr^{90} and Cs^{137} at greater depths. These latter profiles were more compatible with the ocean circulation patterns suggested by C^{14} measurements.[115,116] Integration of the Sr^{90} concentrations to a depth of 1,000 metres yields deposits of 11 mCi/km² for the Caribbean Sea at 18°N; 6 mCi/km² for the eastern Pacific at 0°S, and 10 mCi/km² for the Atlantic at 20°S. These figures do not show any marked increase over continental fall-out at similar latitudes. These workers also obtained a mean Cs^{137}/Sr^{90} ratio in ocean waters of 1.6, in agreement with fall-out and stratospheric air measurements.

47. Many measurements of the Sr^{90} concentration of surface ocean waters have been reported.[113,115,117-120,411] The surface concentrations of the Pacific Ocean in 1961 were much more uniform than in 1958, indicating considerable horizontal mixing. In all cases, the variation of concentrations with latitude in surface waters was much less marked than in air and precipitation.[117] In the western Pacific between 1957 and 1959, average concentrations of 1.7 pCi of Sr^{90} per litre were reported in surface waters.[411]

48. Indirect evidence on the differences between oceanic and continental fall-out rates may be obtained from the relative deposition over islands compared with that over continents. The Sr^{90} fall-out measured in 1962 at nine stations on small islands in the Atlantic and Pacific Oceans was compared with that measured in the same year at nine continental stations.[27] All these stations are situated in the latitude band 0°-40°N and form part of the United States world-wide Sr^{90} network. In addition the Sr^{90} cumulative deposition in soils was compared at a set of paired stations, one of each pair being near the ocean, the other continental.[420] At the same latitude, no significant difference between the fall-out at island and continental stations was apparent, in spite of the fact that the averag· rainfall at the island stations was almost three times as high as that at continental stations.

49. There are several additional pieces of contradictory evidence concerning excess fall-out over the oceans. Measurements in the Mediterranean Sea and in the Gulf of Gascony indicate a larger deposition of Zr^{95} over the sea than over adjacent areas.[421] Similar results have been obtained in the Black Sea for Sr^{90} fall-out.[120] On the other hand, O'Brien, using C^{14} as a tracer for stratospheric Sr^{90}, found agreement between pot determined Sr^{90} global fall-out and that leaving the stratosphere.[400] Thus, his findings in 1960, 1961 and 1963 required no excess Sr^{90} deposition over the ocean compared with adjacent land stations.

50. To summarize, much evidence seems to indicate that the fall-out rate of Sr^{90} over the ocean is about the same as that over land surfaces, but on the other hand a considerable number of measurements of appreciable activities of Sr^{90} in deep waters have been reported that are not compatible with present estimates of deposition. At the moment, there is no adequate explanation for these differences, and further work is needed to clarify the issue. For the purposes of dose estimation, the fall-out

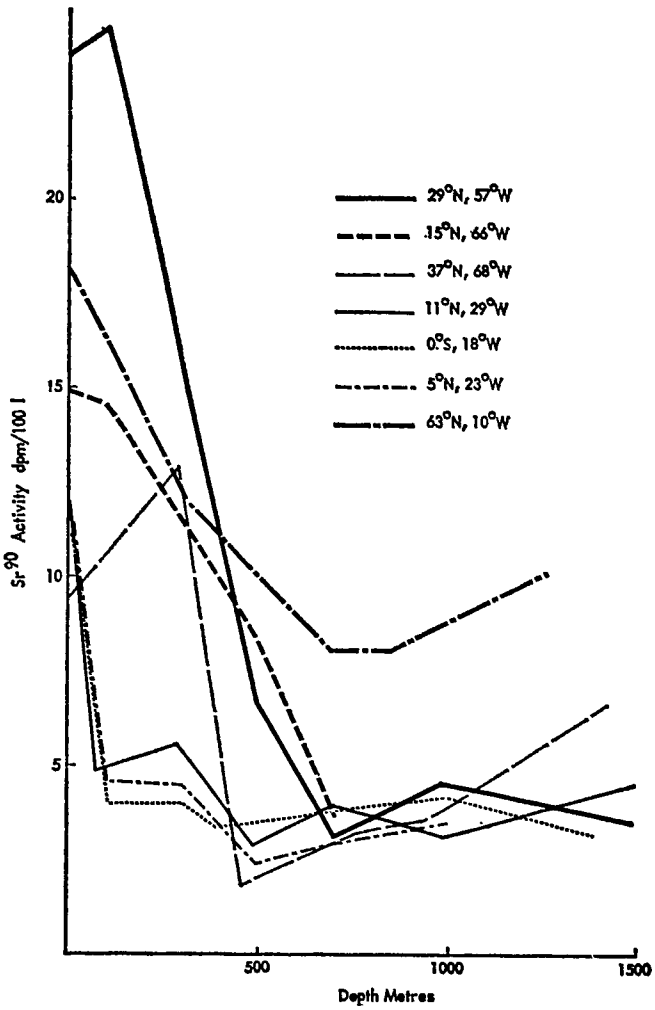


Figure 15. Strontium-90 concentrations in waters of the Atlantic Ocean during the period 1960-1961[112]

rate over the oceans will be assumed to be the same as that over the land since the use of this assumption will not underestimate the doses.

Caesium-137

51. Cs^{137} has not been measured in precipitation throughout the world on such an extensive scale as Sr^{90}, although the number of stations now reporting data on Cs^{137} is greater than before 1960. Because of this lack of world coverage, in the 1962 report the world-wide deposition of Cs^{137} was estimated by applying the mean Cs^{137}/Sr^{90} ratios for the various years under investigation to the global Sr^{90} deposition figures. This procedure is again used here.

52. The half-lives of Sr^{90} and Cs^{137}, 28 years and 30 years, respectively, are so similar that any change in the value of the Cs^{137}/Sr^{90} activity ratio due to radio-active decay over a period of ten years or less can be neglected. The initial Cs^{137}/Sr^{90} ratio estimated from fission yields varies between 1.0 and 3.0 for different fissile materials and for different neutron energies.[121] Measurements of Sr^{90} and Cs^{137} in precipitation indicated that there was also some variation in the ratio with geographical location.[45,89] Large numbers of measurements have been used below in order to obtain reliable values for the mean Cs^{137}/Sr^{90} ratio.

53. Figure 16 shows the mean quarterly Cs^{137}/Sr^{90} ratios for rain collected in the northern hemisphere at 20 stations of the United Kingdom precipitation collection network during the period January 1961-May 1963.[45] In this figure the Cs^{137}/Sr^{90} ratios of stratospheric air measured by balloon sampling over San Angelo, Texas, United States, from January 1961 to November 1963 are also shown.[73] It is evident that the ratio has diminished significantly since September 1961, possibly for the reasons mentioned in the preceding paragraphs. The mean values of the Cs^{137}/Sr^{90} ratios before and after September 1961 have been computed at stations where sufficient data were available. Table IX shows the mean Cs^{137}/Sr^{90} ratios in precipitation at the stations mentioned above, in stratospheric air above San Angelo, and in air measured in the northern hemisphere by aircraft. The measured reduction in the ratio since the resumption of tests is statistically significant.

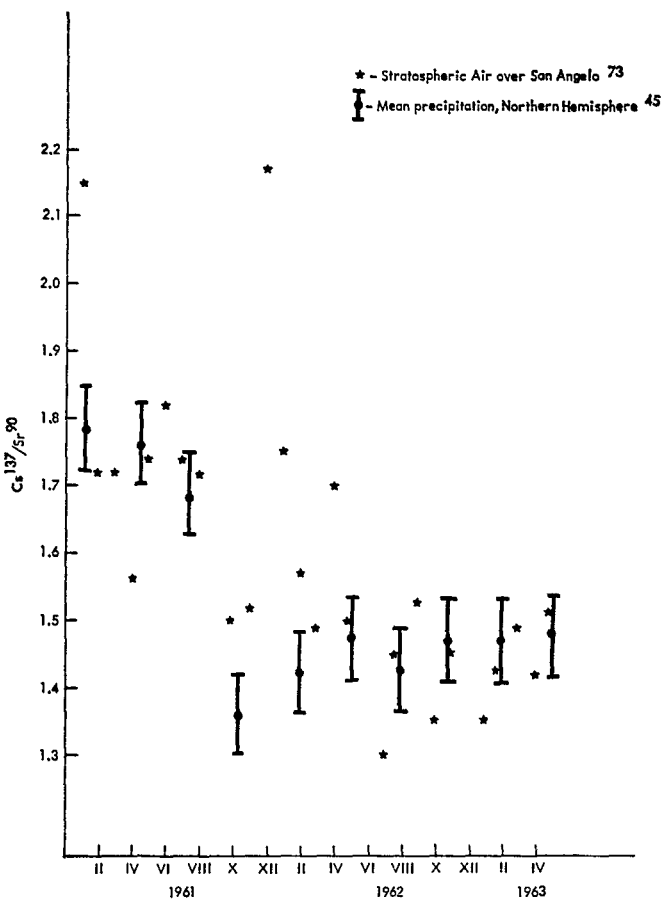


Figure 16. Cs^{137}/Sr^{90} activity ratio in stratospheric air and in precipitation

54. To estimate the total deposition of Cs^{137}, the Cs^{137}/Sr^{90} ratio has been taken as 1.7 through 1961 and as 1.5 for the period 1962-1963. The Sr^{90} deposition values shown in tables VII and VIII have been multiplied by these ratios, and the estimates of Cs^{137} deposition are shown in table X.

Krypton-85

55. The concentration of Kr^{85} in the atmosphere has been increasing steadily since 1954 [122,123,405] (figure 17) and by 1962 was 7 pCi/m³ in the air of the northern hemisphere. From the known fission yield of Kr^{85} it appears that only a fraction of this activity has been contributed by tests, the remainder having presumably been released into the troposphere by nuclear plants. This is borne out by the fact that the concentration of Kr^{85} in air, unlike that of other fission products, shows neither seasonal fluctuations nor any correlation with tests.[122] Since Kr^{85} is a noble gas, almost all of it remains in the atmosphere and so measurements of its concentration in the air of both hemispheres could be used to study exchange mechanisms in the troposphere. The present concentration of Kr^{85} in the atmosphere is not sufficient to give rise to any significant dose to populations.

Short-lived fission products

Strontium-89

56. The deposition of Sr^{89} has, for some six years now, been measured at a large number of stations throughout the world.[27] Since the half-life of Sr^{89} is 50 days, its fall-out rate is a fairly good index of the amount of short-lived activity being deposited. Figure 18 shows the monthly mean Sr^{89}/Sr^{90} ratio for different latitudinal bands during the period September-December 1961.[27]

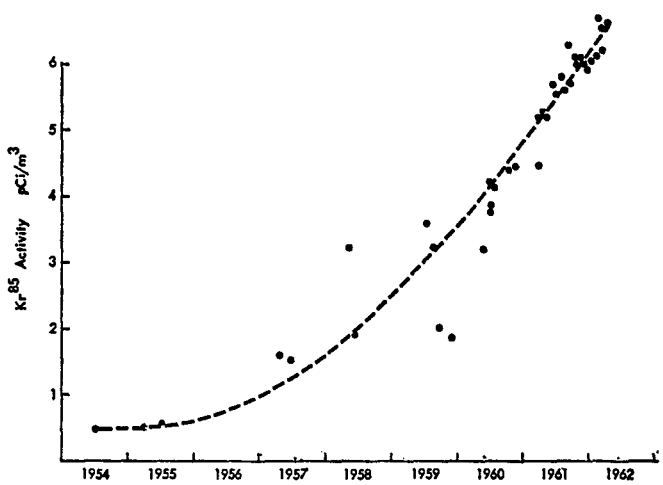


Figure 17. Krypton-85 activity in northern hemisphere air[122,123,405]

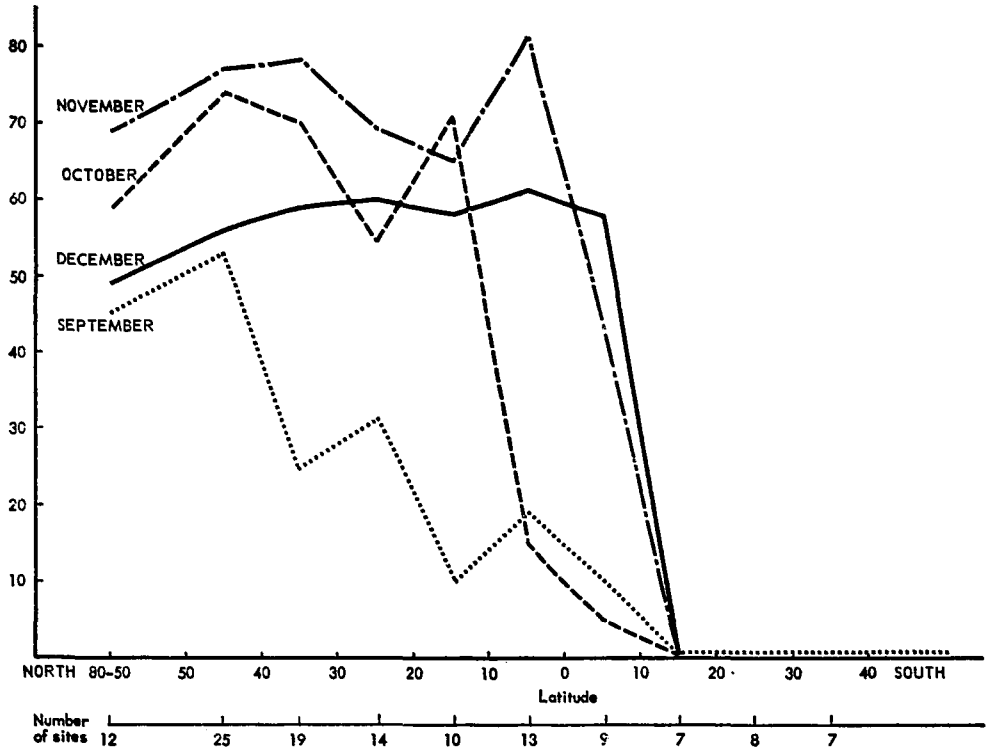


Figure 18. Average Sr^{89}/Sr^{90} ratio in precipitation in each 10° latitude band, 1961[27]

Even by December 1961 the Sr^{89}/Sr^{90} ratio was almost constant with latitude, indicating that the mixing along meridians within the northern hemisphere was relatively rapid.

57. The Sr^{89} fall-out totals in different latitudinal bands for the period September-December 1961, and for 1962 and 1963, are shown in table XI.[27, 124, 376] The deposition was at a maximum in the 30°-50°N band in 1962, about half as much being deposited in the tropical regions and even less at high latitudes. The monthly Sr^{89} deposition in different regions of the northern hemisphere, as well as the southern hemisphere averages, are plotted in figure 19.[75] It is apparent that the Sr^{89} fall-out rates for these three regions of the northern hemisphere were somewhat similar, indicating that the debris was zonally well mixed and that it was largely of stratospheric origin.

58. The Sr^{89} monthly deposition at mid-latitudes in the northern hemisphere reached a peak value of about 25 mCi/km² during January 1962 and passed through a minimum of some 3 mCi/km² during August 1962. The deposition rose again during the last part of 1962 and reached a peak of 20 mCi/km² during April 1963. Present measurements indicate that the deposit of Sr^{89} in 1964 will be practically zero. In the southern hemisphere the deposition rate for Sr^{89}, also shown in figure 19, was small until May 1962 when it increased to about 2 mCi/km² per month and remained approximately at that level during the rest of the year. Included in table XI are the annual deposits of Sr^{89} for each hemisphere during 1961 and 1962. During 1962, the deposition of Sr^{89} in the southern hemisphere was only one-sixth of that in the northern hemisphere.

Other short-lived fission products

59. A number of fission products with half-lives ranging between the 8 days of I^{131} and the 244 days of Ce^{144} are deposited in significant quantities upon the earth's surface. Monthly deposits of short-lived fission products at Milford Haven and Chilton in the United Kingdom are shown in table XII.[45] A number of these nuclides, Zr^{95} + Nb^{95} in particular, contribute significantly to the external gamma dose commitment due to nuclear tests. Although I^{131} is measured very extensively in milk supplies, it has not been measured in precipitation at many sites. However, the deposition rate of Ba^{140} is a reasonably good index of I^{131} deposition. The activity of the short-lived fission products, Sr^{89}, Zr^{95}, Ru^{103}, Ru^{106}, I^{131}, Ba^{140}, Ce^{141} and Ce^{144}, deposited during 1962, are shown in table XIII.[45, 75] The importance of dry deposition of short-lived fission products has been shown by measurements in the United Kingdom.[45] Apart from Sr^{89}, few measurements have been reported on the deposition of short-lived fission products in the southern hemisphere.

Carbon-14

Inventory

60. C^{14}, with a half-life of about 5,700 years, has always been present in the earth's atmosphere where it is produced by the action of cosmic ray neutrons on nitrogen atoms, both in the stratosphere and in the upper troposphere. The C^{14} in the atmosphere is observed almost exclusively as CO_2.[406] Before nuclear testing, the atmospheric content of C^{14} was about 40×10^{27} atoms. The normal distribution of stable carbon and of C^{14} among the different reservoirs of exchangeable carbon, prior to the industrial and atom bomb effects, is shown in table XIV. The specific activity of C^{14} in standard wood (wood grown during 1890) of 14.5 dpm/g C has been used to estimate the C^{14} content of each reservoir.[125] The C^{14} activity in the deep oceans and in humus is assumed to be 84 per cent of standard wood.[115] The natural production rate of C^{14} can be calculated by dividing the inventory (2170×10^{27} atoms, table XIV) by the mean life of C^{14} (8,300 years). The resultant production rate is 2.6×10^{26} atoms per year, which corresponds to 1.6 atoms cm^{-2} sec^{-1}.

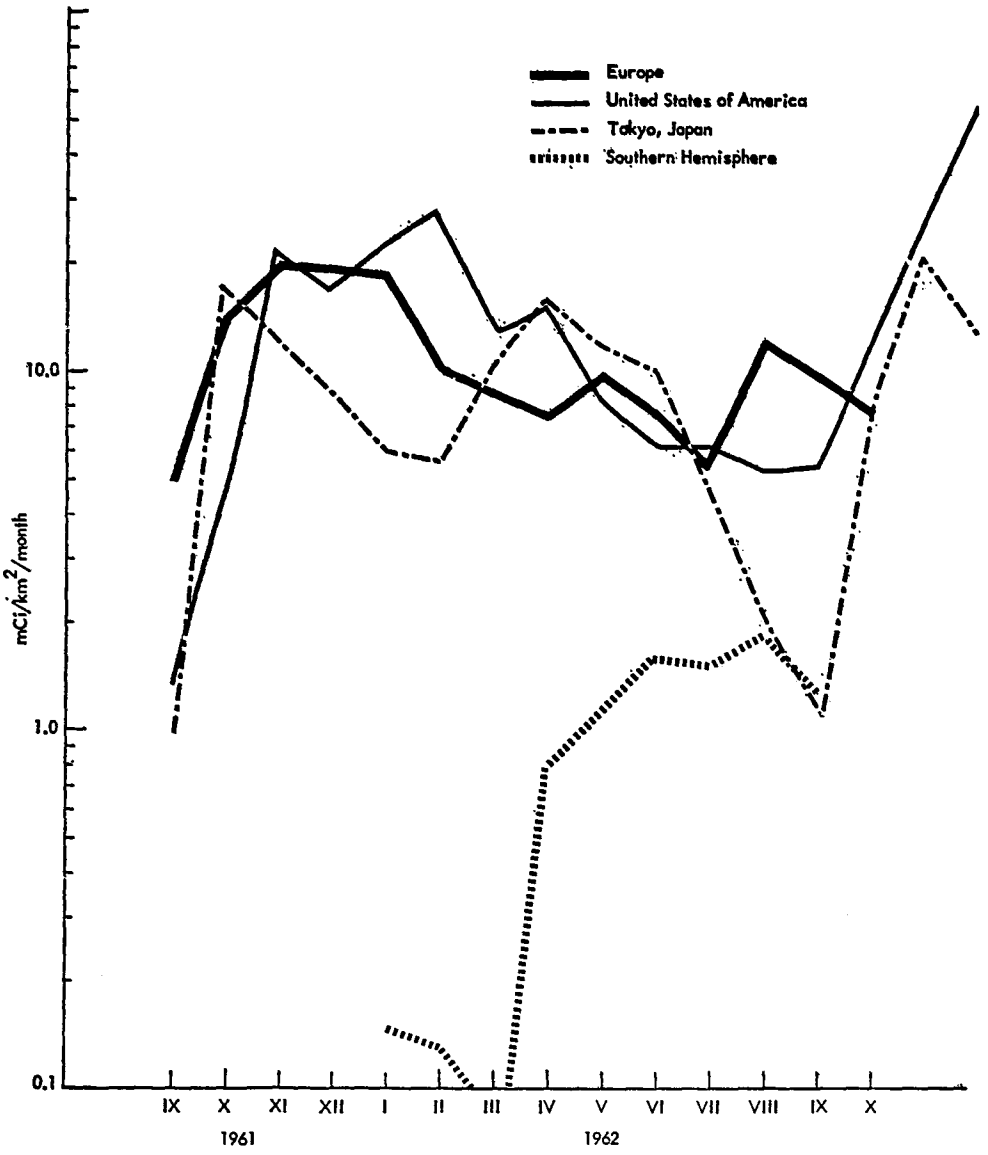


Figure 19. Average monthly strontium-89 deposition[75, 89]

61. Lal and Peters estimated the production rate, when computed from the C^{14} inventory, to be 1.6 atoms cm^{-2} sec^{-1} (when corrected for the new half-life of C^{14}) and 1.8 atoms cm^{-2} sec^{-1} when derived from cosmic ray data.[15, 139] Another estimate of the C^{14} production rate using cosmic ray data is 2.5 ± 0.5 atoms cm^{-2} sec^{-1}.[422] For purposes of risk assessment the lower figure for C^{14} production, 1.6 atoms cm^{-2} sec^{-1}, will be used here, as it will not under-estimate the dose commitment. As this production rate is about 25 per cent lower than that used in the 1962 report, its use will result in a correspondingly higher estimate of the dose commitment from C^{14} produced by tests.

62. Since 1954, large amounts of C^{14} have been produced during nuclear tests. Neutrons produced during nuclear explosions in the atmosphere react with atmospheric nitrogen, producing C^{14}. With underground explosions C^{14} production is essentially zero, while for surface or near-surface explosions the production can be assumed to be about half that due to air explosions. When explosions occur at very high altitude, a fraction of the released neutrons escapes into space and the C^{14} production is correspondingly reduced.

63. Extensive surveys have been made of the C^{14} activity in stratospheric air in both hemispheres, using aircraft and balloons for sampling.[23, 34, 127] From these measurements, the stratospheric C^{14} inventory has been estimated in a manner similar to that used to estimate the Sr^{90} inventory. The estimates of stratospheric inventory of artificial C^{14} at different times between July 1957 and January 1964 are shown in table XV, together with the estimated distribution of artificial C^{14} in other reservoirs.[34, 426]

64. The excess activity of C^{14} in the troposphere as a percentage of the activity prior to tests is shown in figure 20, based on measurements made at a number of laboratories in different parts of the world.[71, 128-137, 141] As figure 20 shows, the C^{14} activity of atmospheric carbon dioxide began to rise appreciably above normal in 1956. Between 1956 and 1958 the level rose almost linearly to a peak in 1959. During 1960 and 1961 the tropospheric C^{14} activity remained fairly constant at about 22 per cent above normal.

65. From 1959 to 1961 there were small annual fluctuations in the tropospheric C^{14} activity.[141] These fluctuations were particularly noticeable in the northern hemisphere and were probably due to the fact that most of the artificial C^{14} was transferred from the stratosphere to the troposphere during the spring months, causing a peak activity in the northern hemisphere, while a sub-

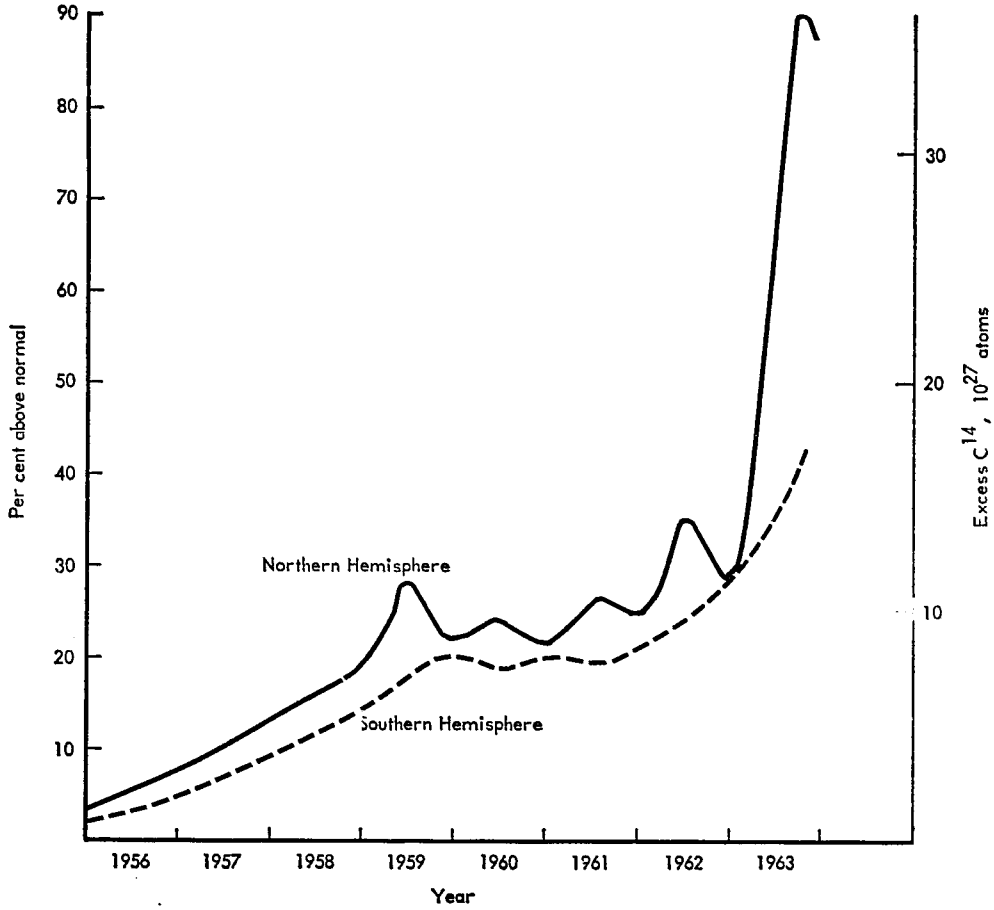


Figure 20. Tropospheric inventory of artificial carbon-14[71,128-137,141]

sequent transfer of some of this activity to the troposphere of the southern hemisphere caused a lowering of the northern hemisphere activity during the fall and winter. This explanation is supported by the fact that the fluctuations in the northern and southern hemispheres are out of phase by approximately six months. It will also be noticed in figure 20 that the peak C^{14} activity in the northern hemisphere in the years 1959-1961 occurred approximately three months later than the peak in the Sr^{90} fall-out rate, as shown in figure 3. The probable explanation is that C^{14} is removed so slowly from the troposphere that the mean tropospheric level tends to be proportional to the integrated amount transferred from the stratosphere.

66. Between 1955 and 1959 the tropospheric C^{14} activity in the southern hemisphere was some 4 per cent lower than that in the northern hemisphere (figure 20) probably because most artificially produced C^{14} had been injected into the northern stratosphere. However, the difference between the hemispheric levels became smaller during 1960-1961 (approximately 2 per cent difference). It has been estimated that the carbon dioxide exchange with oceans in the southern hemisphere is twice as great as in the northern hemisphere, which probably accounts for the residual differences in levels between the hemispheres.[138]

Circulation of carbon-14

67. The C^{14} in the atmosphere exchanges over a number of years with carbon present as bicarbonate in the surface layers of the oceans and with carbon in the biosphere. The carbon then exchanges more slowly with that dissolved as bicarbonate in the deep oceans. This last reservoir is by far the largest, as can be seen from table XIV, and most C^{14} decays while residing there.

68. Measurements of C^{14} in the northern troposphere from 1959 to 1961 have shown a definite variation with latitude.[141] This C^{14} variation and the annual fluctuations have been used to estimate the transfer of C^{14} along meridians, using a linear diffusion model.[141] Using the diffusion rate so obtained and assuming a two-compartment model for the mixing of northern and southern tropospheres, an exchange time between hemispheres of less than one year was estimated. The order of magnitude of the exchange time is borne out by the rapid fall in the northern hemisphere activity during late 1959.

Future levels of carbon-14

69. The future levels of artificial C^{14} in the atmosphere have been estimated on the basis of a four-compartment model representing C^{14} exchange between the different carbon reservoirs. This model is shown in figure 21. Many such models have been proposed in the literature, but for the purposes of predicting future atmospheric levels, there is little to be gained by using more than four compartments.[126,140] A four-compartment model makes more realistic predictions during the first few decades after injection than does a two-compartment model, as was used in the 1962 report. In the present four-compartment model, the biospheric carbon has been lumped in with the atmospheric reservoir, and the humus carbon with the deep ocean, since the atmosphere-biosphere exchange is fairly rapid and the atmosphere-humus exchange is very slow.[140] The move-

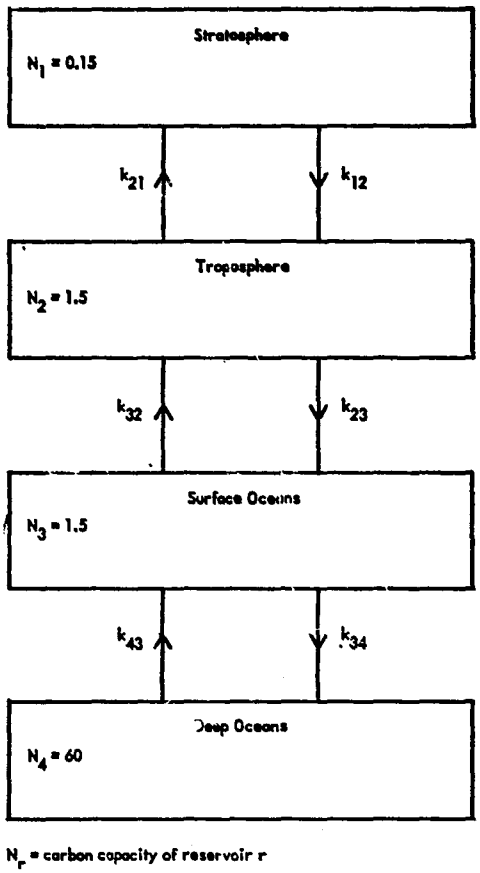


Figure 21. A four-compartment model of carbon-14 exchange

ment of C^{14} between these reservoirs can be described by the set of four equations,

$$N_r\left(\frac{dA_r}{dt} + \lambda A_r\right) = k_{r-1,\,r}\,(A_{r-1} - A_r) - k_{r,\,r+1}\,(A_r - A_{r+1})$$
$$\text{with } r = 1, 2, 3, 4,$$

where

A_r = the C^{14} concentration in reservoir r,

$k_{r,\,r+1}$ = C^{14} transfer coefficient between reservoirs r and $r + 1$, and $k_{01} = k_{45} = 0$,

N_r = the carbon capacity of the rth reservoir,

λ = decay constant of $C^{14} = 0.00012\ y^{-1}$.

70. It is assumed that fractionation effects between reservoirs are small and can be neglected in predicting future levels. These equations have been solved for A_2, the tropospheric concentration, for an initial injection of $C \times 10^{27}$ atoms of C^{14} into the stratosphere at time zero. The method is essentially the same as that used by Plesset and Latter.[140] Based upon the data in table XIV, the capacities of the C^{14} reservoirs are, in units of atmospheric capacity, 0.15, 1.5, 1.5 and 60 for N_1, N_2, N_3, N_4, respectively. The mean residence time of C^{14} in the stratosphere is assumed to be 2.0 years. The transfer coefficients, computed from the steady-state equations derived by Plesset and Latter[140] are, $k_{12} = 0.075$, $k_{23} = 0.27$ and $k_{34} = 0.082$, in atmospheric units per year. The solution giving the excess C^{14} of the troposphere in per cent above normal, after t years, is

$$A_2 = 0.83C\,[0.047e^{-0.00012t} + 1.15e^{-0.026t} + 1.34e^{-0.35t} - 2.54e^{-0.59t}]$$

71. Estimates of the bomb-produced C^{14} inventories in different carbon reservoirs for the years 1957-1963 are given in table XV. The increase due to testing in 1961 and in 1962 was about threefold. If 25×10^{27} out of a total estimated excess of 65×10^{27} C^{14} atoms are assumed to have been injected into the stratosphere in 1958 and the remaining 40×10^{27} atoms in 1962, the predicted future level of C^{14} in the troposphere, A_2, is given for t years after 1964, by

$$A_2 = [2.5e^{-0.00012t} + 64e^{-0.026t} + 26e^{-0.35t} - 26e^{-0.59t}]$$

per cent above normal. This equation predicts a peak excess C^{14} activity of about 70 per cent in 1964 or 1965; the level will then fall to some 60 per cent in seven or eight years' time. Between 1970 and 2040, the level will gradually fall to some 3 per cent and will remain below this level while the excess C^{14} decays radio-actively.

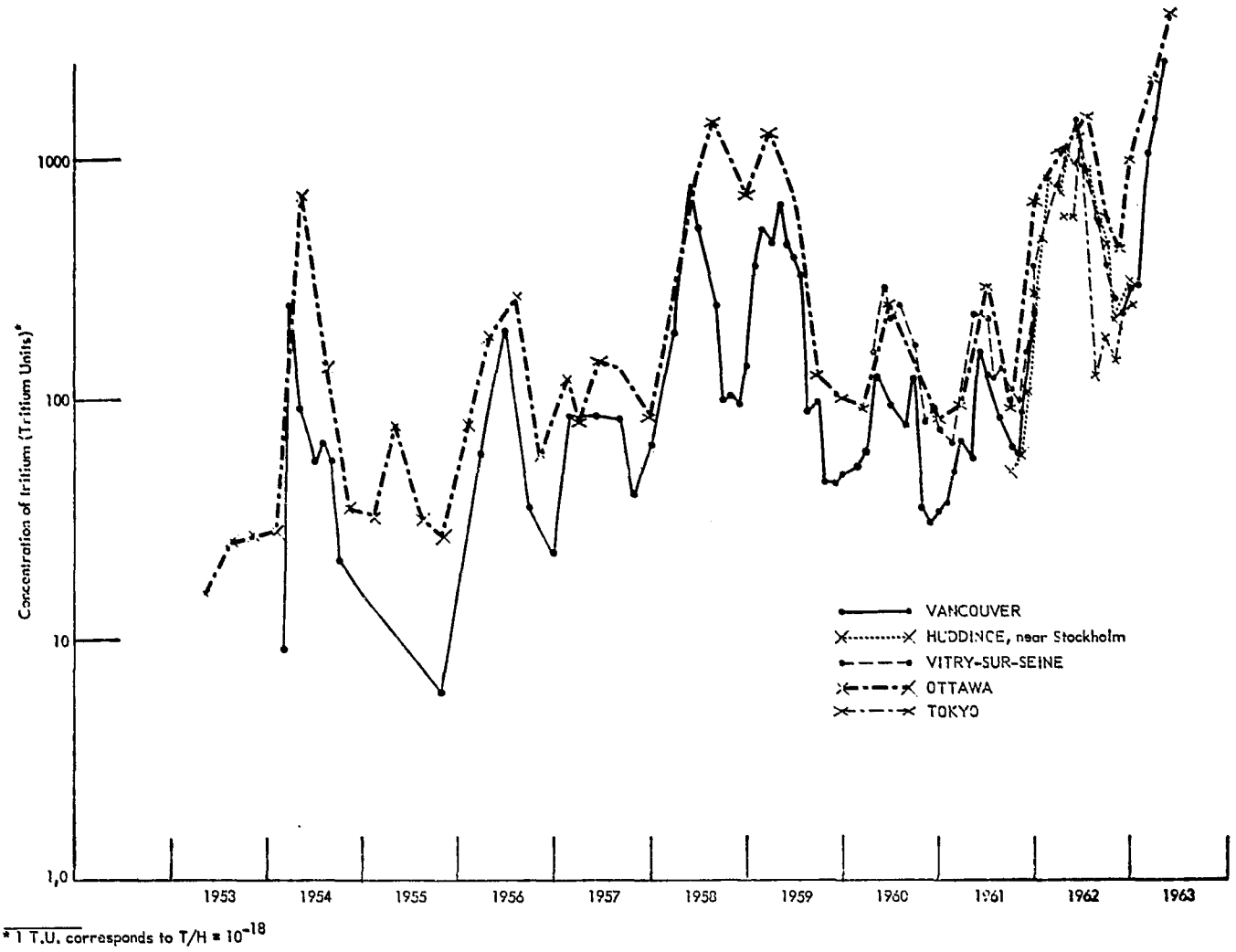
Other artificial radio-nuclides

72. Tritium (half-life 12.5 years) is not a fission product but large quantities of it have been released into the atmosphere by thermonuclear weapons during tests. During 1952 and 1953, before large-scale contamination by tests, typical concentrations of tritium in rain-water were 1-10 tritium units.[c,153] This "natural" tritium is produced by the action of cosmic rays upon the earth's atmosphere. Since thermonuclear weapons tests began, tritium concentrations in rain-water have increased greatly.[146-156,424] Concentrations of tritium in rain-water between 1953 and 1963 in Canada, France and Sweden are shown in figure 22.[143-146,153,156,384,424,425] Peak activities as high as 10,000 T.U. were recorded in Canada in mid-1963.[143] The average tritium concentration in waters of the Pacific Ocean during 1960 and 1961 was 8 T.U.[150] In the southern hemisphere, tritium activities measured in rain-water were much less than in the northern hemisphere, being in the range 5-20 T.U. during 1958 and 1960.[152]

73. Be^7 which has a 54-day half-life is produced naturally in the atmosphere, largely by the action of cosmic ray protons on oxygen and argon, and there is also the possibility of some production in nuclear explosions. Be^7 has been used as a natural tracer to study the movements of air in the stratosphere. Concentrations of both Sr^{90} and Be^7 in rain at Rijswijk during 1961 and 1962,[157] and also in air and rain at other sites, showed marked maxima during the spring months.[158-161] The Be^7 maxima in both years were of similar magnitude, unlike Sr^{90} which showed a much higher activity in 1962, indicating that most of the Be^7 deposited during 1961 and 1962 was of natural origin. Be^7 has also been measured fairly extensively in the stratosphere, and measurements during the period June 1960-May 1961 indicated that the average Be^7/Sr^{90} ratio in the lower stratosphere was 15.[34,157] Using this ratio, it can be estimated that at Rijswijk the deposition rate of Be^7 of stratospheric origin was 12 mCi/km² per year, whereas the deposition rate of tropospheric-produced Be^7 was 28 mCi/km² per year.

74. Measurements of Be^7 and Sr^{89} in stratospheric air over the Netherlands in 1962 suggest that some Be^7 was produced by nuclear tests in late 1961.[158] Be^7 activities above 5 pCi/kg of air showed a linear increase with the Sr^{89} concentration, the mean ratio of excess Be^7 activity to Sr^{89} activity being 0.04. Activities of Be^7 measured in stratospheric air samples collected by aircraft over the United States are shown in figure 23.[34] It is apparent that there has been an increase in Be^7 concentrations in

[c] 1 tritium unit (T.U.) corresponds to $T/H = 10^{-18}$.



* 1 T.U. corresponds to $T/H = 10^{-18}$

Figure 22. Concentration of tritium in precipitation[143-146, 153, 156, 424, 425]

the northern hemisphere where, during the period September 1961-December 1962, concentrations were often markedly greater than expected equilibrium concentrations. This evidence also suggests that Be^7 was produced artificially during tests in 1961 and in 1962. Various nuclear reactions involving Li^6 have been suggested as giving rise to Be^7.[158] By multiplying the above Be^7/Sr^{89} ratio of 0.04 by the figure for Sr^{89} deposition given in table XI, it is estimated that about 1 MCi of bomb-produced Be^7 was deposited in the northern hemisphere during 1962. This is equivalent to an average Be^7 deposition in the northern hemisphere of 4 mCi/km².

75. A number of other nuclides, which have either been deliberately added to nuclear devices or have been produced as a result of neutron activation during the explosion, have been measured in the atmosphere and in precipitation.[23, 73, 142] During tests in 1958, W^{181}, W^{185} and Rh^{102} with half-lives of 145 days, 74 days and 210 days, respectively, were injected into the equatorial stratosphere. Cd^{109} and Cd^{113} with half-lives of 1.6 years and 14 years, respectively, were added in 1962. These radio-nuclides have been measured in air and in precipitation, and the study of their movement in the stratosphere has contributed significantly to the understanding of its circulation.[14, 23, 73, 142] However, the contribution to the dose in humans from these radio-nuclides is insignificant.

76. During tests in 1961 and in 1962, considerable quantities of Mn^{54} (310 days), Fe^{55} (980 days), Sb^{124} (60 days), Y^{88} (104 days) and Co^{58} (72 days) were injected into the stratosphere. The activities of these radio-nuclides in the stratosphere during 1962 are shown in figure 24 and are compared with those of Sr^{90} and Ce^{144}.[13] During the period July 1962-June 1963, 128 mCi/km² of Fe^{55} were deposited at Westwood, New Jersey, United States.[142] There is no evidence at the present time to indicate that doses from these radio-nuclides are of any significance.

77. There is evidence that nuclear explosions have added Na^{22},[36] and possibly Pb^{210},[34, 417] to the existing naturally occurring background of these isotopes in the atmosphere, especially during 1961 and 1962. Various plutonium isotopes from nuclear tests have been observed in the atmosphere and in fall-out.[45, 401, 415, 416, 428] In addition, the possible burn-up of an isotopic nuclear power source for a space satellite in the stratosphere during April 1964 may have added to the burden of Pu^{238} in the atmosphere.[413, 414] However, there is no evidence that the contribution to the dose in humans from these radio-nuclides is significant.

III. Contamination of food and human tissues

Strontium-90 and strontium-89

78. The levels of Sr^{90} in foodstuffs have increased since the resumption of nuclear tests in 1961. The levels of Sr^{90} in milk rose in the northern hemisphere in 1962, the mean yearly values being generally higher by a factor

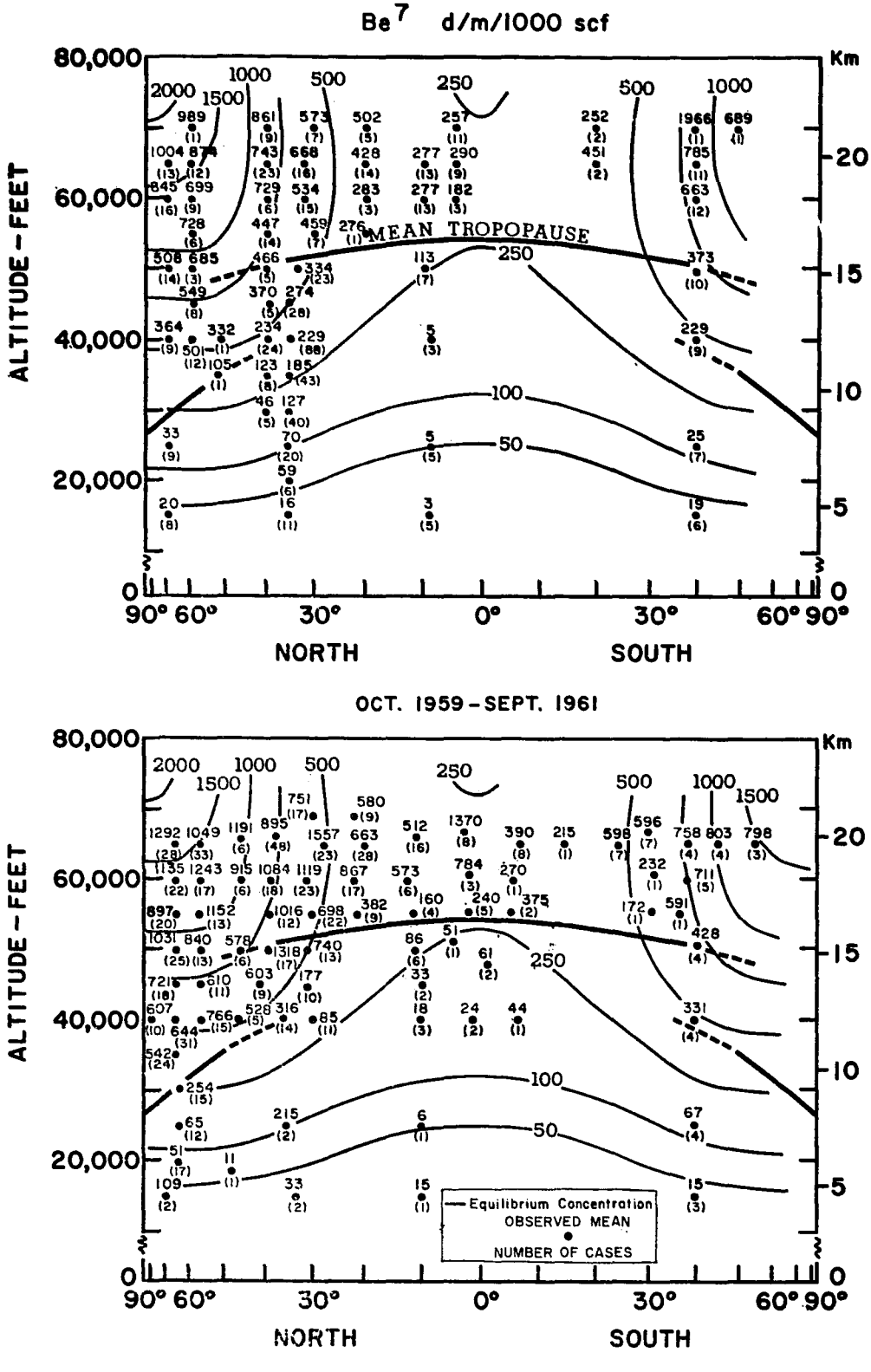


Figure 23. Beryllium-7 activity in the atmosphere[84]

of 2 than they were in 1961. The rise continued in 1963, and the available data show that in 1963 the yearly average was twice as high as it was in 1962 (table XVI). In the southern hemisphere the levels of Sr^{90} in milk rose much less in 1962 and 1963, as indicated by values from Argentina, Australia and New Zealand.

79. The available data on milk contamination by Sr^{89} are summarized in table XVII. During testing periods, high levels were measured in milk from the northern hemisphere. Because of the short half-life of Sr^{89}, milk levels fell sharply by the end of 1963.

80. In countries of the northern temperate zone the rise of the Sr^{90}/Ca ratio in total diet was similar to that in milk, average values for 1962 being higher than in 1961 by 70-100 per cent (table XVIII). The available data for 1963 (Denmark, United Kingdom, United States)

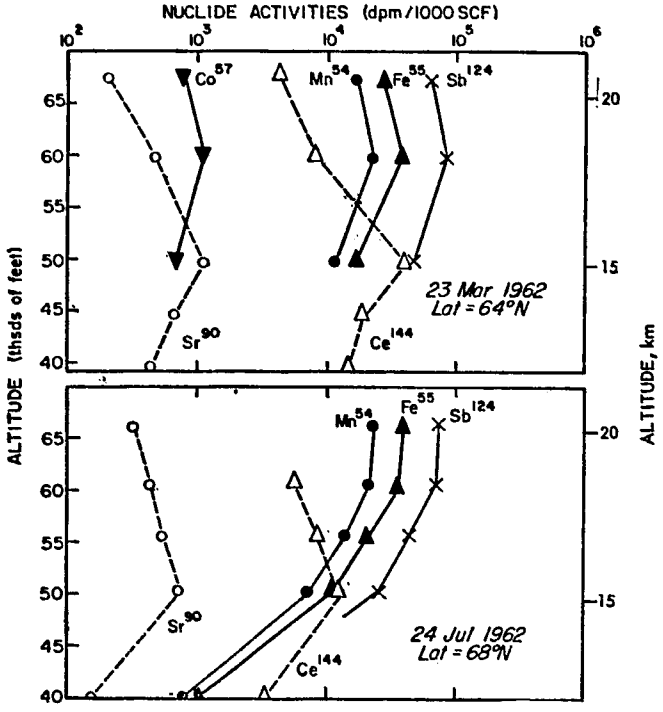


Figure 24. Vertical profiles of cobalt-57, iron-55, manganese-54 and antimony-124 activities (corrected to 15 October 1961) and of strontium-90 and cerium-144 activities (on collection date)[13]

show that the ratio was again doubled when compared with the available data for 1962.

81. Dietary information from regions other than North America and Europe is scanty. The Sr^{90}/Ca ratio in total diet in the United Arab Republic in 1961 and in 1962 was close to the lowest values reported from the northern temperate zone (Europe, United States). In 1963, however, the levels rose only slightly in the United Arab Republic and were on the average lower by a factor of 2 than most of the values reported from the northern temperate zone. Levels in Australia and Argentina were much lower, and only a slight increase in the period 1961-1963 was noted (tables XVI and XVIII).

82. The average Sr^{90}/Ca ratio in the diet in Japan (table XVIII) is close to the levels reported from the United States and Europe in spite of the entirely different composition of the average diet.[172]

83. The relationship between fall-out rate and cumulative deposit of Sr^{90} on the one hand, and milk concentrations of the isotope on the other, has recently been discussed[173,174] and has been expressed by the equation:

$$C = p_d F_d + p_r F_r,$$

where

C = yearly average Sr^{90}/Ca ratio in milk,

F_d = total accumulated deposit of Sr^{90} in soil in mCi/km^2,

F_r = yearly fall-out rate of Sr^{90} in mCi/km^2 in given year,

p_d and p_r are corresponding proportionality factors also called "soil" and "rate" factors.

The values of the soil factors calculated for England and Wales[173] and for the New York and San Francisco areas[174] tend to be lower than the value of 0.3 accepted for the world-wide situation in the 1962 report. On the other hand, it has been shown that values for both the soil and the rate factors may vary widely with local meteorological and agricultural conditions.[175,176] As the soil factor that had been adopted in the 1962 report does not tend to under-estimate the exposure, no change in its value seems justified.

84. Levels, however, vary even within countries. Thus, the yearly mean Sr^{90}/Ca ratio in milk in different regions of the United States differed considerably from the average of the results reported by all the stations.[162,163] The highest and the lowest yearly regional means in 1963 differed by factors of 2 and 6, respectively, from the arithmetical mean of the network.[164] In New Zealand in 1962, the yearly means for local stations differed by a factor of 6.[295] The average dietary levels as found in the Tri-City Study[165] varied systematically by a factor of 2 between New York and San Francisco. A similar degree of variation between average Sr^{90}/Ca ratios in diets from different regions of the country was found in Austria[166] and a smaller one in Denmark[97,167] and in the United Kingdom.[168,169,280] Data on the geographical variation of Sr^{90}/Ca ratios in total diet in Japan indicate values systematically higher than average in the western and northern parts of the country.[170]

85. It should be mentioned, however, that the range of variation observed between regions involving an appreciable fraction of the population of a country does not necessarily include the much higher values of Sr^{90}/Ca ratio found in milk and other dietary products of local origin in some places with particularly high rainfall and with special agricultural conditions. Such situations were investigated in the United Kingdom.[168,169,280] The concentrations of Sr^{90} in milk found there reached levels differing by a factor as high as 8 from the country-wide mean. These locations are usually confined to single farms and are not representative of the region as a whole, so that it is highly improbable that any large fraction of the population could be exposed to dietary contamination of this origin.[171] Broadly similar situations appear to exist in a number of cool regions in north temperate latitudes, e.g. the Faroe Islands[97,179] and northern Finland.[178]

86. The results of surveys in both Australia and New Zealand indicate that the ratio of Sr^{90}/Ca in milk relative to deposition is higher than it is in Europe and North America. In contrast to the majority of areas in Europe and North America, in Australia and New Zealand cattle derive the major part of their entire diet from grazing throughout the year, and it seems likely that this is one of the factors responsible for relatively high Sr^{90}/Ca ratios in milk.

87. Though information is scanty at the present time, it should be noted that in some areas potable water may contribute appreciably to the intake of Sr^{90}. According to observations made in Japan during 1962,[271] monthly determinations of Sr^{90} concentration in potable rainwater from twelve locations showed an average value of 4.4 pCi/l, while an average value of 0.2 pCi/l was observed in city water collected from twenty-five locations. An increase in the Sr^{90}/Ca ratio in diet of about 40 per cent was estimated to be due to this source.

88. The information available on the Sr^{90}/Ca ratio in the diet of infants is limited. Direct determinations of Sr^{90} and calcium in babies' food preparations in the United States indicate that on the average the Sr^{90}/Ca ratio in infants' diet in that country is essentially the same as in the average diet of adults.[181] The Sr^{90}/Ca ratios obtained in the course of a dietary survey limited

to children and adolescents in the United States[182, 366] showed a range of values similar to that observed in adult surveys in the same country.

89. In Argentina during the years 1961-1963,[265] average levels of Sr^{90}/Ca in babies' food preparations were almost the same as in milk. Indirect estimates, taking account of the more important role of milk as a source of calcium and Sr^{90} in children's diet,[168, 169, 183] also indicate that the Sr^{90}/Ca ratios in the average diets of infants and young children are unlikely to exceed that estimated for adult diets. It must be emphasized, however, that, although much information is available for areas where the intake of calcium is relatively high and milk is an important component of the diet, little information is available for countries in which other types of diet are consumed.

90. In the 1962 report, the ratio

$$\frac{Sr^{90}/Ca \text{ total diet}}{Sr^{90}/Ca \text{ milk}}$$

was used to predict Sr^{90}/Ca ratios in diet from those in milk in areas where insufficient information was available to estimate the dietary intake directly. Although this method is not used in the present report, it is of interest to note that the ratios in the years 1960-1963 (table XIX) do not differ substantially from those given in the 1962 report. However, some remarks are necessary:

(*a*) In Japan, where the diet has a very low milk content, the ratio was 2 in 1962, somewhat lower than that given for 1960 in the 1962 report.

(*b*) In Poland, where the average calcium intake is high and where cereals, whole-meal bread and potatoes contribute a relatively high proportion of calcium and Sr^{90} to the average diet,[183, 184] the ratio is higher than in countries where milk is the main source of calcium. A similar situation might exist in a large part of eastern and south-eastern Europe.

91. In the 1962 report, it was concluded that the Sr^{90}/Ca ratio in bone was largely determined by the Sr^{90}/Ca ratio in diet, and that, averaged over a period of several years, the ratio in bone would be about one-quarter of that in the diet from which the bone mineral had been derived (i.e., the OR was 0.25[d]). Recent investigations, which lend further support to this conclusion, have added appreciably to our understanding of the manner in which the metabolism of the two elements changes with age.

92. *Adults.* A value of 0.25 for adults appears to be reasonably acceptable, though somewhat lower values have been reported.[189, 193] The best agreement between calculated and observed Sr^{90} levels in the adult skeleton is obtained by assuming that 2.0-3.5 per cent of bone calcium is replaced every year by that supplied by the diet.[201, 202] Assuming, for the sake of simplification, a single compartment situation, the replacement rate in long bones (e.g. femur) should fall somewhere between 1.2 and 4.0 per cent per year,[187, 188, 193] most probably at the lower end of the range. In spongeous bone (vertebral bodies) several independent estimates yielded values close to 8-9 per cent per year.[187, 188, 193, 210] Good agreement was found between dietary intake and bone levels when interpreted in terms of a power-function model, using parameters obtained from experimental kinetic studies in man with Sr^{85} as a tracer.[202, 203]

93. *Infants.* Earlier indications that there is little discrimination between strontium and calcium in the very young have been supported by recent investigations in which ratios of stable strontium to calcium or Sr^{90}/Ca ratios have been measured in diet and in bone.[193, 194] Detailed investigations in Argentina, employing both methods, indicated that at the age of 2 or 3 months the ratio of strontium to calcium in bone was about 0.8 of that in diet, the value decreasing to less than half this figure at the age of 9 or 10 months, and to about 0.25 in the second year of life.[265]

94. In the early weeks of life the consequence of small discrimination between Sr^{90} and calcium in their transfer from diet to bone is in part offset by the low Sr^{90}/Ca ratio in the foetus as a result of discrimination at the placental barrier. Moreover, when infants are breast-fed the Sr^{90}/Ca ratio in their intake is appreciably less than that in the diet of older age groups.

95. The rate of turnover of minerals in the bones of the young is a major factor affecting the radiation dose received from the Sr^{90} that is deposited in the early months of life. The best agreement between the Sr^{90}/Ca ratios observed in bones of infants and young children, and those predicted from measured dietary levels by giving various values to the relevant parameters (OR and yearly replacement rate of bone mineral), was obtained when a yearly replacement of about 50 per cent of bone mineral in 0-1 year old infants was assumed.[200] However, this figure is uncertain, and its true value might lie anywhere between 30 and 70 per cent. An almost complete replacement of skeletal mineral in the first and second year of life had been postulated by Bryant and Loutit.[187, 193]

96. This high turnover rate will result in a rapid equilibration of the bone mineral with that in diet and consequently will lead to a relatively uniform distribution of Sr^{90} throughout the skeleton. It means also that the Sr^{90} accumulated in the early months of life, when little discrimination occurs, will have little, if any, effect on the total amount of Sr^{90} present in the second year of life or later, when the discriminating mechanism between Sr^{90} and calcium is operative in the same manner as in adults.

97. The over-all effect of changing discrimination on the exposure of infants to Sr^{90} has recently been examined in the United Kingdom, and the average Sr^{90}/Ca ratio in bone during the first year of life was found to be 0.25-0.3 of that in milk.[199] Since in the United Kingdom the Sr^{90}/Ca ratio in milk is close to that in the mixed diet and since the OR for adults is about 0.25, these results show that the lower discrimination between Sr^{90} and calcium in the first few months of life need not be taken into account in assessing the radiation dose from Sr^{90} over periods of a year or longer. This conclusion is further supported by the observation that the ratios between Sr^{90}/Ca in infant bone and milk in several countries in 1962 give an average value of 0.25, as shown in table XXI.

98. Evidence has been obtained that apart from its content of calcium, other components of diet may affect discrimination between strontium and calcium.[192, 196] However, the relatively constant relationship between dietary and bone levels in different countries, as shown in the 1962 report, suggests that in practice any such effect is small.

[d] Observed ratio sample/precursor $= \frac{\text{Sr/Ca of sample}}{\text{Sr/Ca of precursor}}$.[264]

This ratio is meaningful when the sample (e.g. bone or total body) is in a state of metabolic equilibrium with the precursor (e.g. diet).

99. The information on Sr^{90}/Ca ratio in human bones in the years 1961-1963 is summarized in table XX. The majority of data comes from Europe, North America and Australia. No data are available from Africa and Central America. Very limited information was obtained from South America and Asia.

100. In view of the strong age effect in the 0-4 year age group and of the varying proportion of bone samples of each age within the group, whenever possible data have been arranged in five groups covering yearly intervals from about 1 month up to 5 years of age, in addition to the group of new- and stillborn children.

101. When sufficiently detailed information is available (Australia, Denmark, Norway, Poland, United Kingdom), it can be seen that the Sr^{90}/Ca ratio in bone was highest either in the 1-month to 1-year group or in the 1-year group ($>$ 12 to $<$ 24 months) and always lower by 40-50 per cent in still- and newborns (figure 25). The lower Sr^{90}/Ca ratio in bones of still- and newborns is due to the discrimination by the placenta which favours the passage of calcium from mother to foetus by a factor of about 2 in comparison with strontium.[191, 193, 204] In older groups, the Sr^{90}/Ca ratio diminishes gradually, as was already apparent in the 1957-1960 data, until it reaches a plateau at above 20-30 years of age, indicating that the skeletal turnover rate becomes independent of age.

102. *Time trends.* Results from the United Kingdom and the Federal Republic of Germany indicate that the average levels in new- and stillborns decreased in 1961 and in the first half of 1962 (figure 26). A sharp increase was noted in the second half of 1962 when Sr^{90} from recent test series entered the diet.

103. After a fall in 1960 and in 1961, the levels in infants and 1-year old children rose again in 1962. The main increase was observed in the second half of 1962 and in the first half of 1963, as is apparent from figure 27, based on the British data. When compared on a yearly basis, however, the increase over the 1961 level observed in different countries in 1962 varied from almost 0 to about 100 per cent (table XX). The highest group means for Sr^{90} at this age were reported from New York City and Denmark, amounting in both cases to 3.8 pCi/g Ca. The 1963 data that are available (United States, United Kingdom, Soviet Union) indicate a marked increase of Sr^{90}/Ca ratios in infant bones over the levels of 1962 (table XX).

104. In the 2-4 and 5-19 year age groups, varying but usually small increases in levels from 1961 to 1962 can be seen where a sufficient number of samples are available (United Kingdom, New York City). From 1961 to 1962, the increases ranged from almost 0 (Canada, Japan) to about 60 per cent (San Juan, P.R., United States). In 1963 a marked increase occurred in the 2-4 year age group as compared with 1962, judging from the data that are available for comparison. A somewhat lower relative increase was noted in the 5-19 year age group, but the data are still sparse.

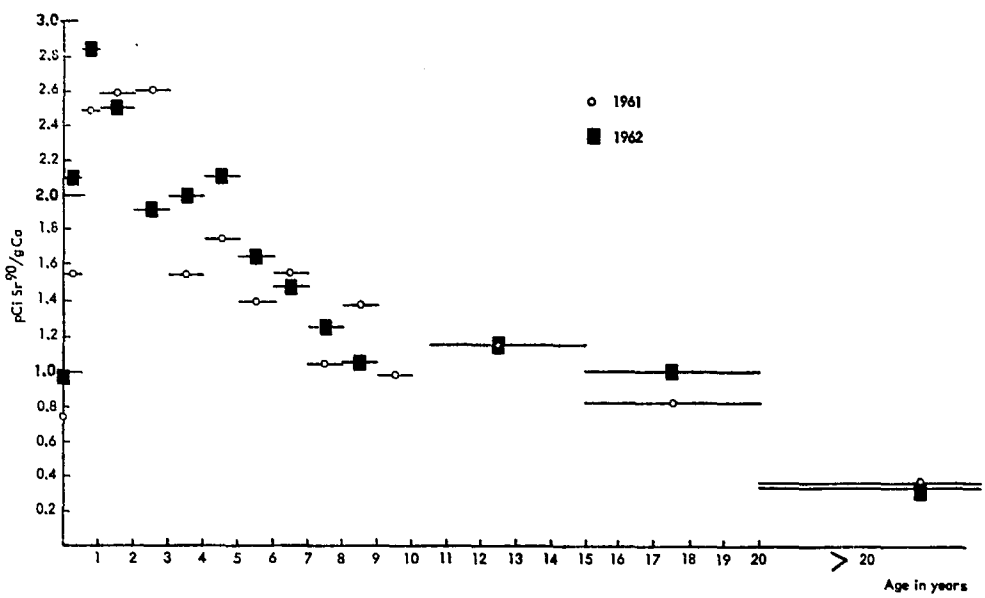


Figure 25. Age distribution of Sr^{90}/Ca ratio in human bones (United Kingdom)[329-331, 373]

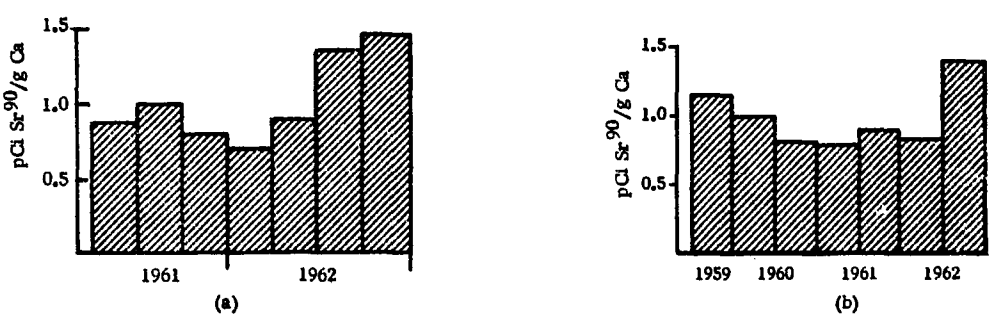


Figure 26. Sr^{90}/Ca ratio in bones of still- and newborn children in the period 1959-1962
(*a*) Stillborns in the Federal Republic of Germany in consecutive quarters of 1961-1962[204, 324]
(*b*) Still- and newborns in the United Kingdom in consecutive half-year periods of 1959-1962[331]

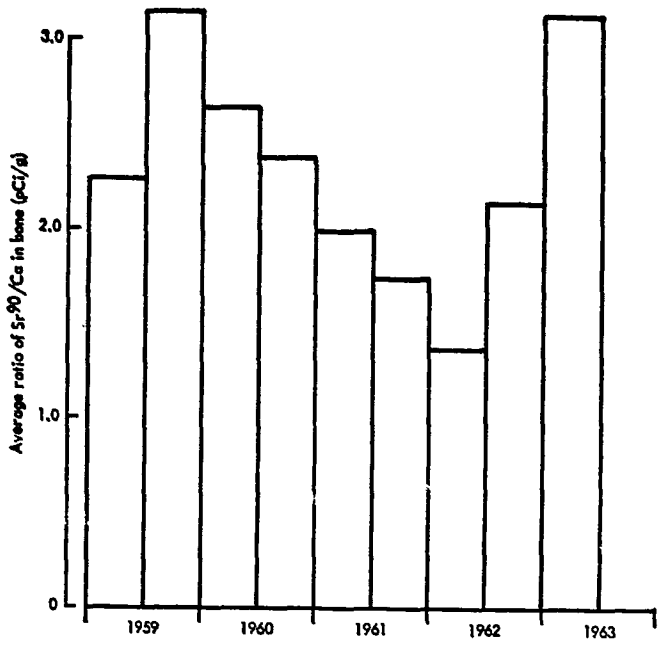


Figure 27. Average ratios of Sr^{90}/Ca in bones of infants, between the ages of 7 days and 1 year, in the United Kingdom (1959-1963)

105. Above 20 years of age, slight increases occurred in some countries (Canada, Poland, United States) from 1961 to 1962, while in others levels were almost steady (Australia, Japan, United Kingdom). A slight increase in the skeletal content of Sr^{90} over this period should be expected on the basis of the current concepts on bone turnover in adults.[187, 201-203, 205] However, biological variability, possible sampling bias and analytical errors may obscure this tendency, especially when only a limited number of samples is available. Data from 1963 are too limited to permit final conclusion.

106. *Skeletal distribution.* In contrast to children (paragraph 96), the distribution of Sr^{90} in the adult skeleton is not uniform. The highest Sr^{90}/Ca ratios are measured in predominantly spongeous bones (vertebrae, ribs) and the lowest in mostly ivory ones (e.g. femur shaft). Normalization procedures were developed in the years 1958-1959[206] which made it possible to compare Sr^{90} concentrations in different bones. Since it is expected that normalization factors will change with time, the use of the same values over extended periods is not justified. Because of these differences, the Sr^{90}/Ca ratios in bones for adults have been given in table XX in their original form, together with the type of bone analysed. For the same reason, comparisons of bone Sr^{90}/Ca ratios for adults will be made only for vertebrae when sufficiently large numbers of samples are available.

107. *Geographical variations.* The average levels of Sr^{90} in human bone from different regions of large countries, e.g. the Soviet Union (nine areas in Europe and Asia)[399] and the United States (New York City, San Francisco and Chicago),[320, 321, 322] do not differ from the mean by more than a factor of 2. In 1961 the average concentration of Sr^{90} in vertebrae of adults from the northern hemisphere (arithmetical unweighted mean of values given in table XX) was 0.8 pCi/g Ca as compared with 0.6 in Australia. In 1962 the corresponding values were 1.0 and 0.6 pCi/g Ca, though these values are not strictly comparable, as the bones from the northern hemisphere were not necessarily collected in the same areas. Nonetheless, they show that the Sr^{90} level in adult bones from Australia is lower than in those from the northern hemisphere (mainly the 30°-60°N latitude).

108. A similar pattern is observed for children's bones. The arithmetic means* for the northern hemisphere in the 0-1 year group were 1.7 and 2.4 pCi/g Ca in 1961 and in 1962, respectively. In Australia, the corresponding values were 1.1 and 1.4. As has already been mentioned for milk, the difference in bone levels between Australia and the northern temperate zone is much less than the corresponding difference in fall-out rate and accumulated deposit of Sr^{90}.

109. Comparison of diet and bone levels supports the method of calculation of dose commitment from diets of various types. The extent to which dietary and bone data in infants support the accepted value of OR (0.25) has been discussed in paragraph 97.

110. The Sr^{90}/Ca ratio in bone in adults cannot be directly compared with the corresponding dietary ratios to derive the OR value because the adult skeleton is not in equilibrium with the diet. However, the validity of dietary estimates can be evaluated by comparing the Sr^{90}/Ca ratios in diet with corresponding estimates of Sr^{90}/Ca ratios in adult bone. Such comparisons are shown in table XXII. Despite differences in dietary levels and in methods of estimation, the values given in this table are reasonably constant. It may be concluded, therefore, that the diet estimates as given in table XVIII form a satisfactory basis for calculation of dose commitment. Again, the values observed in the period 1961-1963 are close to those calculated from fall-out rate and deposit of Sr^{90} by means of the proportionality factors for diets of different types as used in the 1962 report.

Caesium-137

Food chain mechanisms

111. When the 1962 report was issued, only limited quantitative information was available on the mechanisms by which Cs^{137} was transferred along the food chain to man. It was thought that concentrations of this fission product in milk were dependent mainly on the current rates of fall-out as a result of direct deposition of the debris on plants with subsequent foliar and plant-base absorption. From the behaviour of Cs^{137} levels in humans during the period 1959-1961, it had been suggested that Cs^{137} levels in the total diet as well as in man also followed the current fall-out rate.[207] This was supported by observations showing very limited availability for root absorption of caesium atoms which, once mixed with soil minerals, become progressively and almost irreversibly bound by clays.[208, 209]

112. However, the concentrations of Cs^{137} in milk do not follow closely the current fall-out rates everywhere. In some areas, levels of Cs^{137} in milk remained higher in 1960 and in 1961 than would have been expected if they had been proportional to the current deposition that had greatly diminished in that period.[212, 213] In Scandinavia the ratio of Cs^{137} and Sr^{90} concentrations in milk remained almost constant over the period 1958-1960.[214] Since the levels of Cs^{137} in milk follow relatively closely the actual contamination of the fodder because of the rapid turnover of caesium in cows,[215] the constancy of the ratios can only be explained by assuming that, as in the case of Sr^{90}, the absorption of previously deposited Cs^{137} plays a significant role among the mechanisms responsible for the transfer of Cs^{137} to milk.

* Omitting locations where less than 5 samples were measured.

113. That caesium became fixed almost completely in all soils was disproved by Frederiksson[217] who investigated a large series of tropical soils from South America; in those with a low content of micaceous clays, no evidence of appreciable fixation was found. A high level of organic matter in soil can enhance the absorption of caesium by plants.[218] It was suggested therefore that in some permanent pastures a high content of organic material in the upper layer of the soil might reduce the binding of Cs^{137} by clays, thus prolonging its availability to plant roots. The potassium content of the soil is also an important factor, absorption being greatest when the concentration of that ion in the soil is low.[208]

114. The relationship between the concentration of Cs^{137} in milk and the pattern of fall-out varies between different areas, depending not only on soil factors and on the extent to which the deposit is retained by vegetation, but also on the fraction of the diet of animals which comes from concentrated foods, grain or hay produced in the previous year. It has been found in the United Kingdom that the average country-wide concentration of Cs^{137} in milk in any given year could be correlated to the deposit of Cs^{137} over the current and previous two years.[219] The relationship can be expressed by the empirical formula:

$$C = p'_r F_r + p'_{2c} F_{2c},$$

where

C = average country-wide concentration of Cs^{137} in milk in pCi/l in given year,

F_r = fall-out rate of Cs^{137} in given year in mCi/km^2,

F_{2c} = total Cs^{137} accumulated over the previous two years in mCi/km^2,

p'_r and p'_{2c} = proportionality factors for Cs^{137} fall-out over current year and for total deposit over the previous two years in (pCi/l)/(mCi/km^2).

The country-wide average values for p'_r and p'_{2c} were estimated to be 3.6 and 0.65, respectively, and relatively similar values were obtained when the average levels of milk for a number of stations in the United States were examined. Evidence of the effects of climate and agricultural factors on the magnitude of the proportionality factors was, however, obtained by comparing regions of high and low rainfall in the United Kingdom. The expected levels of Cs^{137} in milk calculated on this basis agreed closely with the observed values, whereas the agreement was less good when the total accumulated activity of Cs^{137} per unit area was used instead of the fall-out deposit over the previous two years.

115. In Sweden a generally similar method of analysis has been found to be applicable, but the values for both proportionality factors are greater and the relationship between observed and calculated values is somewhat improved if the deposit in the previous year only is used to derive the second proportionality factor.[266]

116. The relationships between monthly levels of Cs^{137} in milk and the recent deposit of fall-out have been examined in the Midwest of the United States.[220] A good correlation was found between the concentration of Cs^{137} in milk in any one month during the grazing season and the deposit in the previous four months; similarly, for times of the year when animals were fed on stored food a relationship was established with the deposit at the time the fodder was grown. This finding is not inconsistent with the use of the equation given in paragraph 114, since the small magnitude of p'_{2c} compared to p'_r is compatible with the levels of Cs^{137} in milk being largely determined by recent fall-out. In Argentina it appears that the contribution of Cs^{137} deposited in the previous year is extremely small, since a close linear correlation has been established between the levels of Cs^{137} in milk and in rain-water during the years 1960-1963.[394]

Caesium-137 levels in foods

117. Cs^{137} concentrations in milk in 1961 were generally slightly lower than in 1960. The levels in the northern hemisphere rose sharply in the spring of 1962 (table XXIII), and the average yearly concentrations in that year were higher by a factor of 3 than they were in 1961. Such data as are available indicate that the average yearly concentrations of 1962 were again doubled in 1963. In the southern hemisphere (Argentina) the level of Cs^{137} in milk rose only slightly over the period 1960-1962, and absolute values were lower than those from the northern hemisphere.

118. Considerable attention has been given to the situation in some arctic regions where the levels of Cs^{137} in food and man may exceed the average for northern temperate latitudes by factors of more than 100.[178, 225, 226, 243, 244, 368-372, 398] These situations are attributable both to dietary factors and to local conditions which enhance the transfer of Cs^{137} through food chains. The most striking of these conditions are the high levels of Cs^{137} in lichens and other native vegetation on which reindeer and caribou graze. These high levels are not due to unusually high rates of fall-out but to the accumulation of Cs^{137} by these slowly growing plants.[177, 227, 229, 398] In northern Europe and Asia the highest body burdens of Cs^{137} have been found in reindeer breeders for whom reindeer meat constitutes the major part of the diet. In North America the situation is similar for Eskimos who consume caribou meat in large quantities. Considerable variations in the dietary intake of Cs^{137} occur within these areas mainly because of variations in dietary habits, and it is not possible to estimate the number of persons who are exposed to the highest levels. However, the reindeer breeding groups in Finland, Norway and Sweden constitute only a small fraction of the total Lapp population, which amounts to about 35,000 people.

119. In the subarctic regions of these countries there is also a substantial non-Lapp population which shows Cs^{137} uptakes considerably higher than the country-wide average. Dietary levels of Cs^{137} appreciably above the average have also been reported in the Faroe Islands,[98, 179] the west coast of Norway[222, 223, 224] and on a very local scale in other countries. These situations are attributable to high levels of Cs^{137} in pastures grazed by cattle.

Metabolism of caesium in man

120. Although the retention of a single administration of Cs^{137} is best described by an equation with two exponential terms, one of these, with a short half-period, contributes only a negligible fraction of the dose and can therefore be ignored.[230-232] The biological half-life of the long-term component of retention, as determined in apparently normal adults by means of whole body counting techniques, can be estimated to be on the average about 100 days (table XXV). The absorption of tracer amounts of caesium from the gastro-intestinal tract in man is close to 100 per cent.[231, 233]

121. Two studies of the biological half-life of caesium in children [226, 234] led to values of about 44 and 38 days, thus at least partially accounting for the concentrations of Cs^{137}/gK and Cs^{137}/kg body weight being lower in children than in adults. The information on the turnover

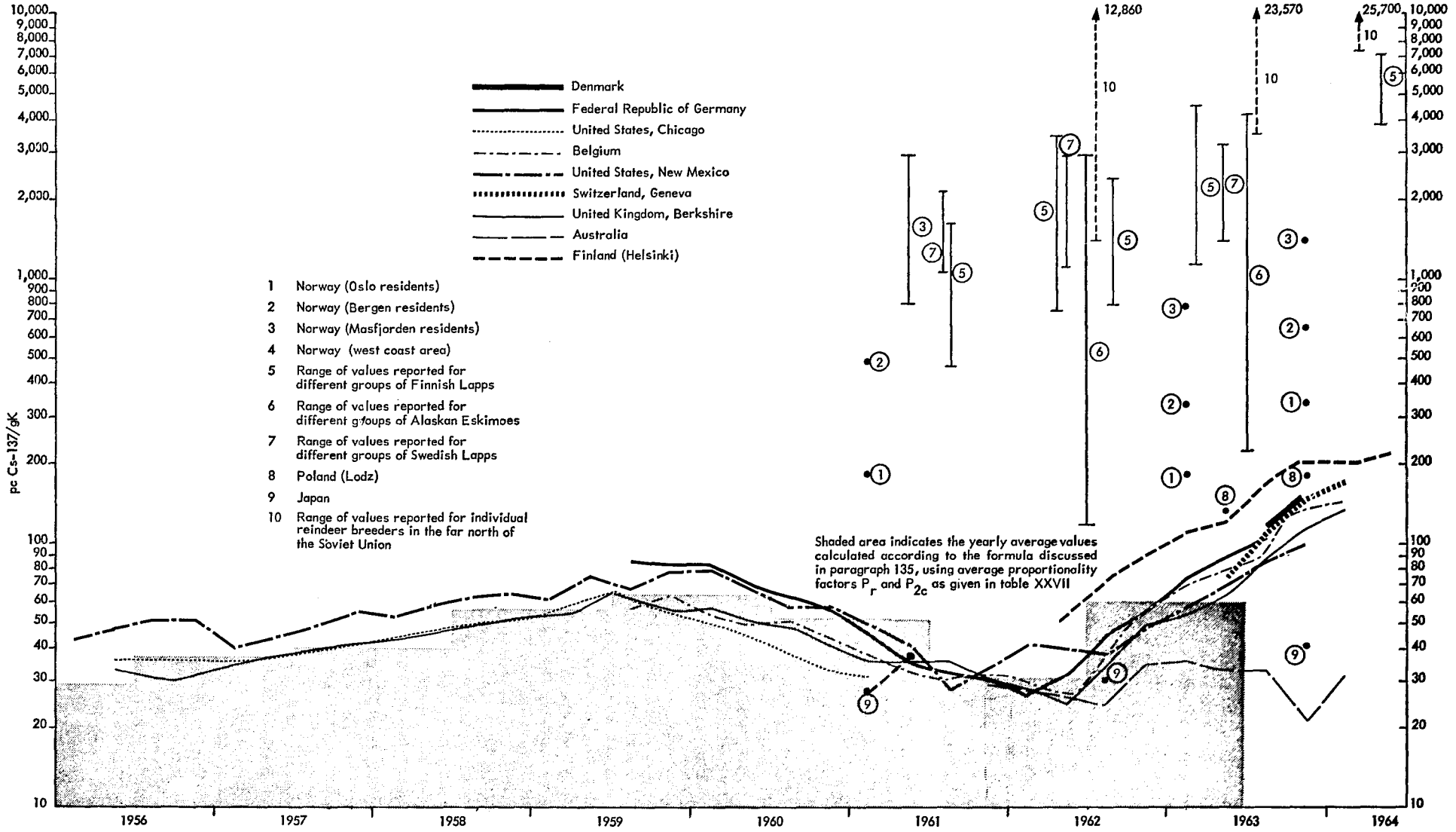


Figure 28. Caesium-137 levels in man during the period 1956 to early 1964

rate of caesium in early infancy is limited to two studies, giving values for half-life of 9.6, 6.6 days,[234] 21 and 25 days[375] in the four subjects investigated.

122. In the 1962 report it was assumed that concentrations of Cs^{137} per gramme fresh bone tissue were higher than in muscles by a factor of 2.2. Further investigations[236, 240] did not confirm this difference.

Observed levels in man

123. Extensive investigations showed[235] that the concentration of Cs^{137}/kg body weight was about 50 per cent higher in adult males than in females. This is apparently due to the higher average proportion of fat tissue in the female body. It is known that concentrations of potassium and caesium in this tissue are very low as opposed to muscles, parenchymatous organs, etc. When expressed in pCi/gK, Cs^{137} levels in males are only 10-15 per cent higher than in females.

124. All available data on Cs^{137} levels in the human body are collected in table XXVI. Values are expressed in pCi/gK because:

(*a*) Age and sex differences are minimal.[235] This is important when the sex-ratio and the age composition of the group investigated are not known.

(*b*) Values correlate closely with Cs^{137} concentration per unit of lean body mass which seems to be a more important parameter for dosimetric purposes than the whole body mass.

Use of the Cs^{137}/gK ratio naturally does not imply that the metabolism of these elements is as closely linked as that of calcium and strontium.

125. The levels from different locations in the northern hemisphere, as shown in table XXVI and figure 28, are remarkably close, with the exception of the relatively isolated and sparsely populated subarctic regions and of the coastal areas of Norway (see paragraphs 118-119). Regional differences within countries have not been extensively described except in the study of Anderson *et al.*[220] in the United States, who showed only slight differences in Cs^{137} body levels between regions with very different deposition rates and milk levels of Cs^{137}.[164]

126. After a peak in 1959 and in 1960, the Cs^{137} content in man declined to the lowest levels in late 1961 and then began to rise in the first half of 1962, reflecting the increased fall-out rates and dietary levels in 1962 and 1963. In those areas of the northern hemisphere where surveys were performed systematically, the levels in late 1963 were 2 to 4 times higher than the average levels in 1961.

127. Data from the Far East, the Middle East and the Pacific region are too scanty to permit meaningful comparisons with the northern temperate zone. Limited data from Japan, collected by means of the whole body counting technique,[241, 242] show values lower than in Europe and North America. This observation is consistent with the corresponding differences in the average intake of Cs^{137} as given in table XXIV. In the southern hemisphere data are available only from Australia.[817] Late in 1961 and in early 1962 the levels were fairly close to those observed in the northern temperate zone. In 1963 and early 1964, however, only a very slight increase occurred, so that body levels of Cs^{137} in Australia were lower by a factor of 3-6 than those reported at that time from the northern hemisphere.

128. As has already been noted and discussed in paragraph 118, exceptionally high levels of Cs^{137} in food and in population were observed in some areas. In some groups of Finnish and Swedish Lapps[226, 243, 244] average body burdens in 1961 reached levels 40 to 60 times higher than the average for northern temperate regions, with some individuals showing body concentrations up to 150 times higher. The average levels for groups of different occupational and dietary habits were essentially proportional to their estimated intake which, in turn, largely depended upon consumption of reindeer meat highly contaminated with Cs^{137}. It has been calculated,[226] and later confirmed by direct measurements,[178] that, because of the increased meat consumption in winter, levels in reindeer-breeding Lapps would have doubled in the spring of 1962, even without further contamination of the environment. Comparably high body burdens of Cs^{137} were found in 1962 among Alaskan Eskimos,[225] one group of whom showed an average of 3,000 pCi/gK. From April 1962 to April 1963 the average increase in Cs^{137}/K ratio in Swedish Lapps from Jokkmokk amounted to about 30-40 per cent.[371] A similar increase was observed in Alaskan Eskimos during summer periods in 1962 and 1963.[368] In early 1964 one group of Lapps reached the average level of 7,000 pCi/gK,[369] and in some individuals in subarctic regions the total body burden in 1963 and in 1964 exceeded the value of 3.5 microcurie of Cs^{137} (table XXVI).[398]

129. Values intermediate between those reported from subarctic regions and the averages for the northern temperate zone were observed in other regions of Scandinavia. Thus in the first quarter of 1961[222] and of 1963, 480 and 332 pCi/gK were measured in Bergen. In Masfjorden[222] where most of the food consumed is locally produced, an average concentration of about 1,400 pCi/gK was observed in 1963. Levels in Oslo[224] in 1961, in 1963 and in 1964 were lower, but still higher by a factor of 2-3 than in other locations in central and western Europe and in continental United States.

Relation of caesium-137 in fall-out and diet with body burden in man

130. Because the biological half-life of caesium in man is of the order of $\sim$ 100 days (table XXV), changes in dietary intake are fairly rapidly reflected in the levels of this nuclide in the body. To obtain close agreement between observed and predicted body burdens of Cs^{137}, detailed information about the intake of the nuclide over short intervals is necessary. In general, however, such information is lacking, so that it is unavoidable to use average levels over longer periods of time, e.g. yearly, to study correlations between dietary levels or fall-out pattern and body concentrations.

131. Even so, allowance should be made for the lag between dietary and body values. When average yearly levels of Cs^{137} in the body (New Mexico)[220] are divided by average milk levels determined on a 12-month basis, the most constant ratio is obtained by allowing for a 9-month lag (e.g. January-December 1961 average body values are compared with average milk levels computed over the March 1960-March 1961 period).

132. An empirical relationship between levels of Cs^{137} in man and the fall-out pattern, similar to that found for milk in the United Kingdom (paragraph 114), was found by Bartlett and Mercer using data from Berkshire, United Kingdom.[219] The agreement between observed and predicted values was closer than when proportionality with current yearly fall-out deposition and accumulated deposit of Cs^{137} was assumed.

133. As was pointed out in paragraph 125 and shown in figure 28, concentrations of Cs^{137} in man in the northern temperate zone (30°-60° N), excluding Norway where special ecological mechanisms operate, are very close, probably as a consequence of the extensive redistribution of marketed foodstuffs within and between most countries. As the fall-out deposition is highest in the northern temperate zone between 30°-60° N, it seems reasonable to assume that, apart from the exceptional situations mentioned in paragraphs 118-119, 128-129, which involve only a small percentage of the world population, the levels of Cs^{137} in man in this band represent an upper limit of the expected real world-wide average.

134. Because of the geographical uniformity of Cs^{137} levels in man in the latitudinal band from 30°-60° N, the average Cs^{137} deposition in this band, as derived from the Sr^{90} deposition over the period 1953-1963,[104,180] will be used here to estimate the empirical relationship between fall-out and body burden in man. The yearly average body burden will be assumed to be directly proportional to the current fall-out and to the amount deposited over the two previous years. This relationship can be expressed by the formula,

$$Q = P_r F_r + P_{2c} F_{2c},$$

where

Q = yearly average concentration of Cs^{137} in man in pCi Cs^{137}/gK,

F_r = fall-out rate of Cs^{137} in a given 12-month period in mCi/km²,

F_{2c} = total Cs^{137} accumulated over the previous two years in mCi/km²,

P_r and P_{2c} = proportionality factors in (pCi Cs^{137}/gK)/(mCi/km²).

135. The proportionality factors P_r and P_{2c} estimated for Belgium, the Federal Republic of Germany, Berkshire (United Kingdom), and New Mexico (United States) are given in table XXVII. When average factors (arithmetic means of the local factors) are used to calculate the average concentrations of Cs^{137} in man in the latitudinal band 30°-60° N, these concentrations are consistent with the observed values, as is shown by figure 28, where observed values do not differ from the predicted average by more than some ± 20-30 per cent. When yearly mean body concentrations of Cs^{137} are linearly related to the current rate of fall-out only, or to both the current rate and the total cumulative deposit, very poor agreement is obtained between results predicted on the basis of the relationships so established and those observed.

Iodine-131

Iodine-131 in food

136. I^{131} was detected in air, rain and milk in late 1961 and in mid-1962. Levels rose sharply in September and October 1961 and declined to detection limits in January 1962. A second peak followed in most countries in the period August-December 1962.[97,168,169,245-249] However, in southern Italy where for climatic reasons the grazing period for cattle, unlike that in central and northern Europe, extends into winter, I^{131} was detected as late as February 1963.[247] Typical concentration profiles of I^{131} in milk from several countries in Europe, North America and Japan are presented in figures 29-31.

137. Detailed reports on the I^{131} content in milk are limited to twelve countries of the northern hemisphere

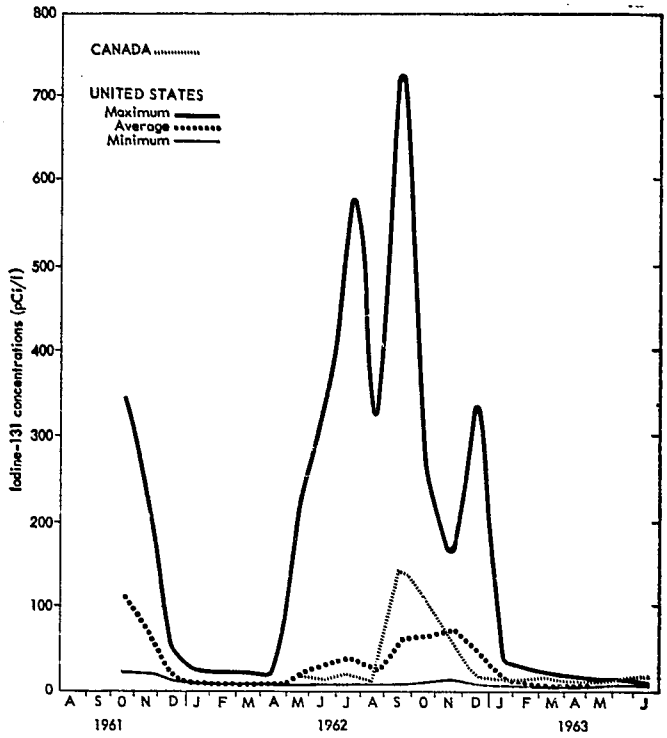


Figure 29. Iodine-131 in milk in North America, 1961-1963[245]

in 1961 and to fourteen in 1962 (table XXX). Values are expressed in pCi d/l (picocuries × days per litre) as a time integral of the concentration. In the southern hemisphere I^{131} was not detected in 1961 but was detected in 1962 (Argentina, Australia). These data indicate that levels of I^{131} in milk were lower by a factor of 10 in Australia, and by a factor of 2-3 in Argentina, compared to those observed in the northern temperate zone. It must be mentioned that average I^{131} concentrations in milk from different countries, even in similar geographical latitudes, vary by a factor of 3-4. Within large countries, such as the United States, the yearly average concentrations reported from different regions differ by a factor of 10.[164] It is easily understood that the deposition of short-lived isotopes from fall-out is more dependent upon transitory meteorological conditions over short periods of time than is the case with such long-lived fission products as Cs^{137} and Sr^{90}.

138. In the State of Utah (United States), high concentrations of I^{131} in milk were detected for several weeks starting 12 July 1962.[250] Assuming a daily intake of one litre of milk, the average total intake of I^{131} during this period was estimated at 58,000 pCi with a maximum value of 800,000 pCi. Available evidence[251-253] points to the local fall-out from test explosions in the nearby Nevada test site as the source of massive contamination of pastures with I^{131}.

139. It has been established that the main path of transfer of I^{131} to the urban population in the United Kingdom and in the United States was through milk and its fresh products (e.g. cottage cheese), the role played by other foodstuffs being negligible.[248,249,254] Only a few per cent, if any, of the measured thyroid burden could be due to inhalation of I^{131} contained in the air.

140. In Japan, however, because of dietary habits (average per capita consumption of milk in adults being only 0.05 l/day), the major contribution of I^{131} was from fresh leafy vegetables.[255] It has been calculated that in 1961 the maximum possible intake with air and milk may

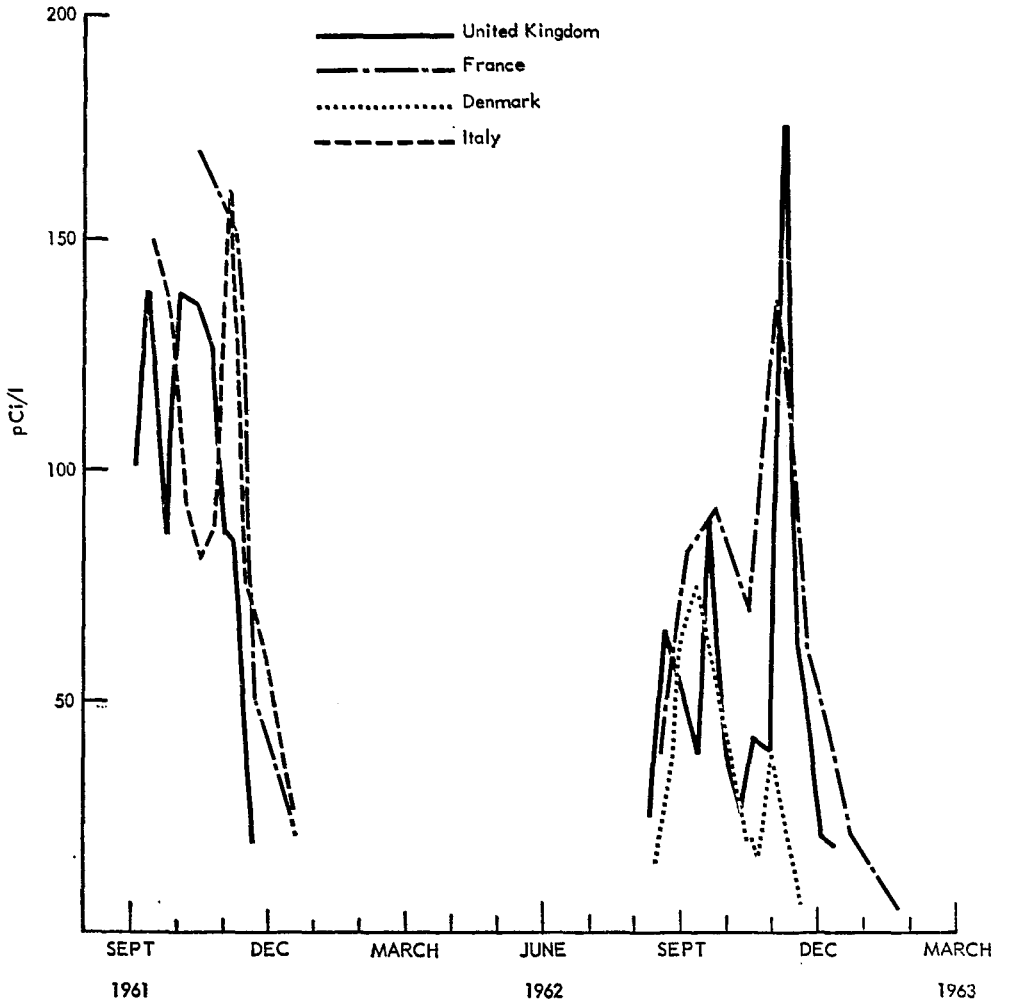


Figure 30. Iodine-131 in milk in several European countries, 1961-1963

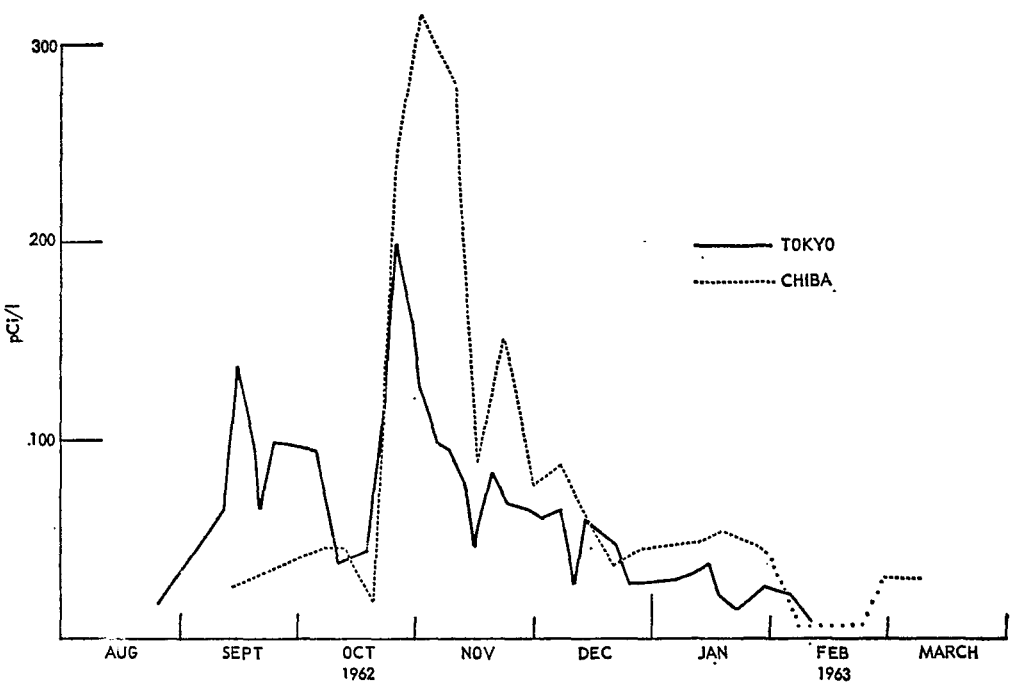


Figure 31. Iodine-131 in milk in Japan, 1962-1963

have accounted for 27 and 12 per cent, respectively, of the I^{131} detected in Japanese thyroids, the rest coming from vegetables. It has also been shown in Germany that unprocessed drinking water from cisterns can substantially add to the intake of I^{131} in periods of fresh fall-out.[256]

Iodine-131 in human thyroids

141. I^{131} concentrations in the thyroids can be determined in living subjects or *post mortem* and can also be estimated indirectly from the levels of food contamination. The various methods have been discussed by Eisenbud *et al.*[249]

142. Surveys by *in vitro* counting of thyroids taken at *post mortem* examinations were made on hospital patients and on victims of accidents dying in the period when I^{131} was present in the milk.[248, 255, 256] Results of *in vitro* measurements are presented in table XXVIII. The conclusion reached by Eisenbud *et al.*[249] was that, most probably for dietary reasons, hospital patients were not representative of the population as a whole. On the other hand, results obtained from accident victims seemed to be biased by socio-economical factors, as revealed by the comparison with results of *in vivo* counting of persons with controlled consumption of milk. This study indicated that the results from *in vitro* counting in accident victims might appreciably under-estimate concentrations as compared with the population averages, expected on the basis of milk consumption.

143. Measurements made in foetuses at different stages of prenatal life[249] showed that specific activities were higher in foetal than in maternal thyroids by a factor of 1.3-8.2 (5 measurements). In a 12-weeks-old foetus, a concentration as high as 630 pCi/g of thyroid was observed. Because of the age-dependence of thyroid weight, of milk consumption and of I^{131} uptake by the gland, the most critical age in post-natal life from the dose point of view has been estimated to be around 7 months,[257] or between 6 months and 2 years of age.[258]

144. Sensitive low-level gamma spectrometric techniques have been developed and used for *in vivo* measurements of fall-out of I^{131} in human thyroids.[259, 260] Measurements showed wide individual variations of I^{131} levels in human thyroids so that large numbers of properly sampled subjects of different ages must be examined to obtain results valid for the population at large. This is hardly possible in the case of rapidly changing fall-out situations. In comparison with the *in vitro* counting, an additional uncertainty in dose evaluation is introduced by geometrical factors (detector-gland) which play a critical role in this type of measurements, and by uncertainties regarding the weights of individual thyroids. The levels of I^{131} in thyroids, obtained by this method in the Federal Republic of Germany, New York City, Boston, and the State of Utah are given in table XXIX.

145. Indirect estimates of thyroid burdens can be obtained when results of representative and frequent sampling of milk are available.[240, 261] The average dose can be calculated when the following factors are known:

(*a*) Average consumption of milk in groups of population at different ages.

(*b*) Iodine uptake in thyroid as a function of age.

(*c*) Mass of thyroid as a function of age.

(*d*) Biological half-life of I^{131} in thyroid.

The average total intake of I^{131} with milk in different countries in 1961 and in 1962, if one litre is consumed daily, is given in columns 3 and 4 of table XXX. The total intake in any given group of population may be derived by multiplying these figures by the average consumption of milk in litres per day. As data on milk consumption in specific age groups are lacking, the average consumption of milk by infants and young children has been assumed to be about 0.7 litre per day. For lack of data, no assumption has been made with respect to adults, though it can be stated that their I^{131} intake is generally much lower than in children.

146. A calculation based on the levels of I^{131} in milk appears to be the most satisfactory means of estimating thyroid doses to various sections of the population. Moreover, it is applicable where direct measurements of I^{131} in human thyroids are not available.

IV. Doses from environmental and internal contamination allowance for the distribution of fall-out and population

147. For the purpose of this report "dose commitment" is defined as the integral over infinite time of the average dose rate in a given tissue for the world's population, as the result of a specific practice, e.g. a given series of nuclear explosions. The actual exposures may occur over many years after the practice and may be received by individuals not born at the time of the period of practice. On the basis of a linear dose-effect relationship with no dose threshold and no dose-rate effect, and assuming a stable population, the expected number of late somatic injuries and hereditary defects would be the same for a practice with a given dose commitment as for a practice which would result in an instantaneous dose of the same magnitude to all members of the population.

148. Since we are concerned in this report with average doses to populations, it is necessary to weigh the average fall-out deposition according to population distribution. It is convenient to define a population factor Z, defined according to the equation,

$$\overline{F}_N = Z \times \overline{F}_A,$$

where $\overline{F}_A$ is the mean deposition in the area concerned and $\overline{F}_N$ is the mean deposition weighted by population in the same area. $\overline{F}_N$ is computed from the formula,

$$\overline{F}_N = \frac{\Sigma N_i F_i}{\Sigma N_i}$$

The population factor Z could be computed for the whole world, for one hemisphere, or for any other local area of interest.

149. The population factor may be expressed as the sum of several partial population factors. For example, by breaking the area into three smaller areas, there are three partial factors:

$$Z = Z_1 + Z_2 + Z_3.$$

Z_1 is defined as $Z_1 = \dfrac{\Sigma N_a F_a}{F_A (N_1 + N_2 + N_3)}$,

where the summation is taken over area 1. For the other two factors Z_2 and Z_3, the summation is taken over areas 2 and 3, respectively. For estimating the internal dose commitment from Sr^{90}, the world is divided into three areas according to three basic diet types, and three partial population factors are used.

150. The local deposition F_i varies considerably from place to place, and this can be described by a "geographical factor" G_i, defined as

$$G_i = F_i/\overline{F}_a,$$

where $\overline{F}_a$ is the mean global deposition. Such a factor can be used to estimate doses in local areas. In the 1962

report a curve showing the variation of G_i with latitude was given. It is of interest to note that the global average of the local geographical factor G_i weighted by population equals the global population factor Z.

151. Since some 90 per cent of the world's population resides in the northern hemisphere, the estimated global Z factor will depend upon the fraction of fall-out deposited in each hemisphere. When the debris is largely deposited in the northern hemisphere, the Z factor will be about 2, but will be approximately 1.2 for equal fall-out in both hemispheres. Using mean deposition data from the northern and the southern hemispheres, separate estimates of mean deposition weighted by population for each hemisphere have been computed.

152. Table XXXI shows the estimated world population distribution[f] and the relevant data on Sr^{90} and Sr^{89} deposition. The data from table XXXI have been used to calculate the Z factors for both hemispheres according to the formula given above. A summary of these calculations, together with the Z factors obtained for various years, is shown in table XXXII. It can be seen from table XXXII that in the northern hemisphere the factors were fairly constant from 1960 to 1962 with a maximum variation of only 8 per cent compared with 35 per cent for the global Z factors during the same period. The slight variation in the northern hemisphere was probably due to the tropospheric fall-out in 1962, while in 1961 the newly deposited Sr^{90} was largely of stratospheric origin. It is remarkable that in the northern hemisphere the same numerical factors are obtained for Sr^{89} as for Sr^{90}.

153. In the southern hemisphere the Sr^{90} fall-out distributions gave fairly constant population factors of approximately 1.00, but the factor obtained for Sr^{89} deposition was higher and amounted to 1.45 in 1962. This is because considerable amounts of Sr^{89} diffused into the southern hemisphere from the northern troposphere and were deposited in the 0°–10° S latitude band where about half the population of the southern hemisphere lives.

154. In the case of Sr^{90} and Sr^{89}, the dose commitments are computed by dividing the world population into three groups according to dietary habits. Since C^{14} is uniformly distributed in the troposphere, no allowance for population distribution is necessary. To compute the dose commitments due to other nuclides, two population factors are used, 1.2 for the northern hemisphere, and 1.0 for the southern hemisphere.

155. In calculating dose commitments, the total radiation emitted by deposited radio-nuclides must be considered. Data in tables VII and X represent the cumulative levels of Sr^{90} and Cs^{137} on the ground at a particular time allowing for radio-active decay. To compute dose commitments, the concept of "integrated deposition", i.e., the total deposition uncorrected for decay and weathering losses, has been introduced. The integrated deposits of Sr^{90} and Cs^{137} up to December 1963 are shown in table XXXIII, together with the predicted future deposit.

External doses

Measured dose rates in air

156. Direct measurements of air doses have been reported from Japan, Sweden and the United Kingdom.[351–354] The annual doses for the years 1961-1963 are shown in table XXXIV. The Japanese measurements were made with scintillation counters that were calibrated against an ionization chamber. The counter at Tokyo was situated 7 m above the roof of a three-storied concrete building but was calibrated against an air equivalent ionization chamber situated 1 m above the ground. All the other measurements were made by using ionization chambers. These instruments measure the total gamma dose rate in the air. The dose rate from fall-out is obtained by subtracting the contribution from cosmic rays and from naturally occurring gamma emitters. The measurements at Leeds were made at 10 feet above ground and the results corrected to 1 metre.[355,356] The averages for each area of Sweden are based on measurements made at several stations.[352]

157. The gamma dose rate has also been estimated from measured deposits of gamma-emitting fission products. Gustafson used deposition figures based upon soil analyses to estimate the gamma dose rates at Argonne, Illinois, United States.[357–359] Collins estimated the external dose rates from Zr^{95}, Ru^{106}, Cs^{137} and Ce^{144} in the United States at Westwood (New Jersey), Pittsburgh (Pennsylvania) and Richmond (California), using deposition figures based upon measurements in precipitation.[360–362] Since these three radio-nuclides account for at least 80 per cent of the external dose rate, the dose rates so obtained are meaningful. Dose-rate estimates based upon deposition data have been plotted in figure 32.

Short-lived fission products

158. Estimates of dose commitments to the world population could be obtained by using average global deposition estimates for short-lived fission products, together with the appropriate dose-rate factors. However, the deposition of short-lived fission products are not measured at most collection stations. To estimate the deposition of the short-lived fission products, the northern hemisphere deposition of Sr^{89} is used, together with estimates of the ratios,

$$R_j = \frac{\text{Annual northern hemisphere deposit of short-lived radio-nuclide}}{\text{Annual northern hemisphere deposit of } Sr^{89}},$$

determined for each fission product in each year. The ratios R_j have been estimated from the local ratios computed at seven stations in the northern hemisphere. The mean of the ratios at these stations is used as an estimate of the R_j's for individual nuclides. The local ratios and the corresponding average ratios for 1962 are shown in table XXXV.[27,45,75,76,91]

159. It must be noted that the stations lie in a narrow latitude band but that this particular latitude region is highly populated. Although the ratios have no fundamental significance, if the fission product debris is well mixed within the hemisphere, they should not vary greatly from place to place during periods of steady testing except perhaps for ratio involving the very short-lived radio-nuclides I^{131}, Ba^{140} and Ce^{131}. After testing is completed, the annual ratios will change owing to radio-active decay.

[f] The absolute figures apply to 1951. Absolute figures would naturally be different now but are not available by latitudinal band. The relative magnitude, however, is unlikely to have changed.

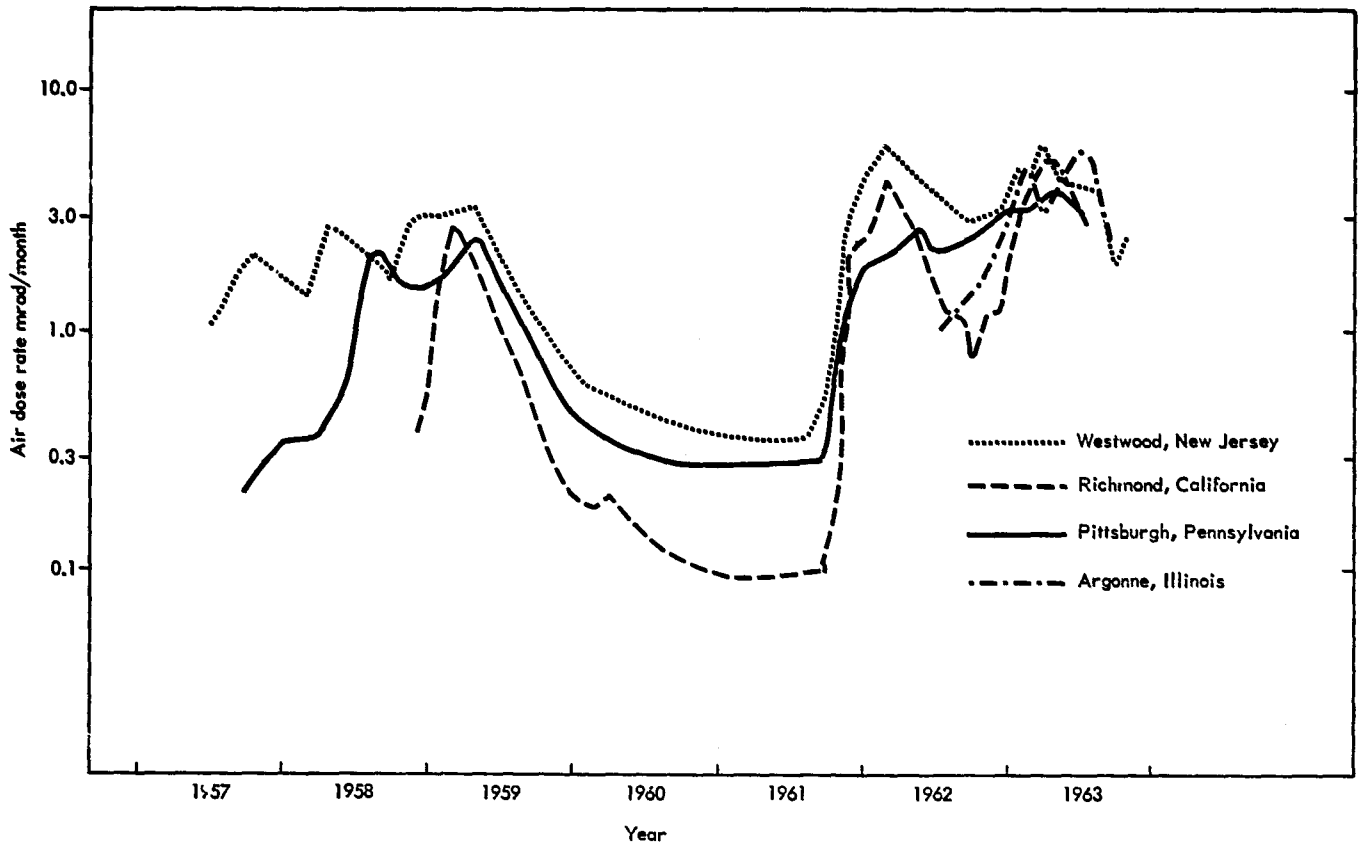


Figure 32. Gamma ray air dose rate, at one metre above ground, from fall-out as estimated from fission product deposition[357, 362]

160. Available measurements of short-lived isotopes in the southern hemisphere are insufficient to obtain reliable estimates of the ratios in that hemisphere. But since only 10 per cent of the world population lives in the southern hemisphere and since less than 20 per cent of the Sr^{89} global fall-out is deposited there, the contribution of short-lived fission products deposited in this hemisphere to the world dose can only be a few per cent.

161. The external gamma dose commitment is computed for each nuclide by using the formula

$$D_j = K_j \times B_j \times Z \times T_j \times R_j \times \overline{F}_{89},$$

where $K_j \times B_j$ = gamma-ray dose constant, including build-up factor for fission product j (mrad/y per mCi/km²); Z = population factor; T_j = mean life on the ground of fission product j; R_j = ratio defined in paragraph 158 for fission product j; $\overline{F}_{89}$ = average annual northern hemisphere deposit of Sr^{89} (mCi/km²). The details of the air dose calculations for the fission products Zr^{95}, Ru^{103}, Ru^{106}, I^{131}, Ba^{140}, Ce^{141} and Ce^{144} are shown in table XXXVI. The ratios R_j for 1962 are taken from table XXXV and the ratios for 1961 are estimated in a similar manner. Since complete deposition data for some short-lived fission products in 1963 were not available, the doses from Ru^{106} and Ce^{144} have been computed by using the average Ru^{106}/Ce^{144} and Ce^{144}/Sr^{90} ratios, together with the 1963 estimates of Sr^{90} deposition.

162. The total measured air dose in the years 1961-1963 from Cs^{137} and short-lived fission products weighted by population was 54 mrads. By deducting the contribution from Cs^{137} for this same period, namely 15 mrads, the contribution from the short-lived fission products is 39 mrads. This figure is in reasonable agreement with the computed figure of 46 mrads that can be inferred from table XXXVI after deduction of the dose to be delivered after 1963. It should be noted that the dose commitment for each of the years does not correspond exactly to the annual doses, since some part of the dose commitment is received in subsequent years. Allowance must be made for doses received in 1964 from material deposited in 1963 and also from material still to be deposited. These additional doses are estimated to be about 10 mrads, making a total dose commitment of 49 mrads for testing in 1961 and 1962. The dose commitment for short-lived isotopes due to testing up to 1960 was given in the 1962 report as 55 mrads. The total air dose commitment for all tests to December 1963 is therefore 104 mrads.

163. Shielding by buildings and screening by the human body were considered in the 1962 report. A shielding factor of 0.2 was adopted as a world average. Assuming that seventeen hours per day on average were spent indoors, the over-all dose-reduction factor due to shielding was taken to be 0.4. The same value will be used in the present report, together with a body screening factor for the gonads and bone marrow of 0.6, as was also adopted in the 1962 report. The combined shielding and screening factor (0.2), applied to the air dose commitment, yields a tissue dose commitment from short-lived fission products of 21 mrads.

Caesium-137

164. The external doses from Cs^{137} are computed by using a combined dose-rate constant and build-up factor ($K_j \times B_j$) of 0.12 mrad/y per mCi/km² of Cs^{137}. The Cs^{137} data in table X, corrected for the fourteen-year effective mean life of Cs^{137} on the ground due to decay and weathering,[382] are used to compute the annual doses in the period 1961-1963. The Cs^{137} air doses weighted by population in these years were 4, 5 and 6 mrads, respectively.

165. The air dose commitment from Cs^{137} can be estimated from the predicted total integrated deposit of Cs^{137} as shown in table XXXIII. This figure, the same as that in the 1962 report, is justified by results of measurements of the dose rates made in the United Kingdom over plots of soil contaminated with Cs^{137}.[363] The estimate of air dose commitment from Cs^{137} contributed by all tests up to the end of 1962 is 143 mrads. Using a combined shielding and screening factor of 0.2, the dose to gonads and bone marrow is 29 mrads.

Internal doses

Strontium-90

166. *Integrated dietary levels.* The estimates of mean Sr^{90}/Ca ratios in diets are obtained from the relationship,

$$C(t) = p_d F_d(t) + p_r F_r(t), \text{ pCi } Sr^{90}/\text{g Ca},$$

where $F_d(t)$ is the cumulative mean deposit of Sr^{90} (mCi/km²) and $F_r(t)$ the mean annual deposition rate (mCi/km²/y) at time t. p_d and p_r are the proportionality factors discussed in paragraph 83. As in the 1962 report, the world population is divided into three groups (table XXXVII) according to their dietary habits, and proportionality factors have been computed for each group. The same factors are used in this report, although there is some evidence that the rate factor (p_r) for the Japanese diet which is of type III may be somewhat less than the average value for other diets of that type.[427] The values of p_d used are probably over-estimates rather than under-estimates.

167. The mean integrated level of Sr^{90} in diet is given by

$$\int_0^\infty C(t)dt = p_d \int_0^\infty F_d(t)dt + p_r \int_0^\infty F_r(t)dt.$$

If the effective mean life of Sr^{90} in the soil is T_m years, it can be shown that

$$\int_0^\infty F_d(t)dt = T_m \int_0^\infty F_r(t)dt = T_m F,$$

where F is the integrated amount of Sr^{90} deposited on the ground (mCi/km²). The mean integrated level in diet is therefore

$$\int_0^\infty C(t)dt = (p_d T_m + p_r)F, \text{ pCi years of } Sr^{90} \text{ per g Ca.}$$

168. For a 2 per cent annual loss of Sr^{90} from the soil through leaching and removal by crops, the effective mean life of Sr^{90} in the soil is twenty-one years.[402] The expected total integrated deposit of Sr^{90} between latitudes of 50° S and 80° N is 14.2 MCi (table XXXIII). This is equivalent to an average deposit (F) of 31.7 mCi/km² and leads to estimated integrated dietary levels of 310, 480 and 530 pCi years of Sr^{90} per g Ca, for diet types I, II and III, respectively.

169. The composite integrated dietary level of Sr^{90} weighted by population is obtained, as in the 1962 report, by adding the integrated levels in the three dietary types multiplied by the respective partial population factors 0.7, 0.5 and 0.7. These coefficients reflect the fact that most of the world's population resides in the latitude bands of the northern hemisphere where the deposition of Sr^{90} is about twice the global average. The composite integrated global level in diet is 830 pCi years of Sr^{90} per g Ca.

170. *Dose commitment.* The dose commitment from Sr^{90} is computed as in the 1962 report. The dose dD delivered to the bone over the balance of the lifespan by Sr^{90} from an intake due to exposure to environmental contamination between times t and $t + dt$ at age u years was shown by Lindell[365] to be

$$dD = \theta c(t)dt\alpha(u)\int_u^m \frac{1}{B(\tau)}\exp[-k_1(\tau - u)]d\tau \text{ (mrad)},$$

where θ = dose-rate constant in bone (mrad/y per pCi Sr^{90}/g Ca); $c(t) = OR \times C(t) = Sr^{90}$/Ca ratio in bone mineral deposited at time t (a bone/diet OR of 0.25 is used here); $\alpha(u)$ = rate of calcium uptake by bone at age u years; $B(\tau)$ = total weight of calcium in bone at age τ years; k_1 turnover rate of Sr^{90} in bone; m = life expectancy. This equation can be written

$$dD = \theta c(t)\, dt\, F_m(u).$$

171. The lifetime dose increment $d\overline{D}$ averaged over the whole population, assuming uniform age distribution at the time of Sr^{90} uptake, is given by

$$d\overline{D} = \theta c(t)dt \frac{1}{m}\int_0^m F_m(u)du = \theta c(t)dt\overline{F}_m.$$

The dose commitment to the population contributed by a finite period of testing is then given by

$$\overline{D} = \theta\overline{F}_m \int_{-\infty}^{+\infty} c(t)dt.$$

Lindell computed the dose-increment factor $\overline{F}_m$ for different assumed Sr^{90} turnover rates in bone and for different life expectancies.[365] The values of $\overline{F}_m$ so obtained have been plotted in figure 33, which shows that $\overline{F}_m$ is not strongly dependent on the turnover rate nor on life expectancies higher than twenty years.

172. The reason for the small variation of $\overline{F}_m$ is the compensating effect of changes in turnover and life expectancy. For example, a more rapid turnover rate will reduce the retention of Sr^{90} taken up by a child but will increase the uptake and exposure of adults. Similarly, with shorter life expectancies, a larger proportion of the population will be in the younger age group with a high uptake of Sr^{90}, but the total lifetime exposure will be reduced. As in the 1962 report, a dose-increment factor of 0.6 is here being used.

173. As in the 1962 report, dose-rate factors (θ) of 2.7, 1.4 and 0.7 mrad/y per pCi Sr^{90}/g Ca are used for computing the doses to bone, cells lining bone surfaces, and bone marrow, respectively. The estimates of dose commitment contributed by Sr^{90} from all tests up to the end of 1962 are therefore 336 mrads to bone, 174 mrads

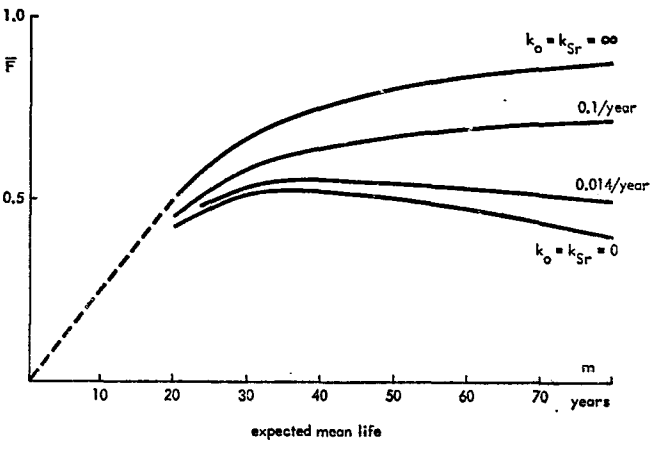


Figure 33. The population average of the dose increment factor for various biological turnover rates as a function of the expected mean life[365]

to cells lining bone surfaces and 87 mrads to bone marrow. Some 90 per cent of these doses will have been delivered by the year 2000.

174. In addition to the dose commitments, the annual dose to new bone is also of interest and can be calculated from the dietary contamination by using a discrimination factor of 0.25 (paragraphs 91-97). While new bone constitutes the whole skeleton during the first year of life, the freshly deposited bone mineral is only a small fraction of the total skeleton in adults. The composite world average level of Sr^{90} in diet during 1961, 1962 and 1963 can be calculated from the cumulative and the annual Sr^{90} deposits for each of these years (table VII). The yearly dietary levels weighted by population were 17, 26 and 38 pCi Sr^{90}/g Ca in these years. The resulting dose in 1963 was 25 mrads in new bone. This cannot be compared directly with doses to other tissues for which dose commitments are calculated.

Strontium-89 and barium-140

175. Since the metabolism of Sr^{89} is the same as that of Sr^{90}, Sr^{89} doses can be calculated from the mean Sr^{89}/Sr^{90} ratios in milk by using the appropriate dose-rate factors. The Sr^{89} dose-rate factors for doses to bone and bone marrow are, respectively, 1.5 and 0.33 mrad/y per pCi Sr^{90}/g Ca in bone. The mean dose-increment factor $\overline{F}$ for Sr^{89} is 0.005. These values are the same as in the 1962 report. The average Sr^{89}/Sr^{90} ratios in milk, as estimated from the data in tables XVI and XVII, for the northern hemisphere in 1961, 1962 and 1963, were 2.6, 4.5 and 1.8, respectively. Using these ratios as typical for total diet (this will tend to over-estimate doses), together with the mean diet levels of Sr^{90} for 1961, 1962 and 1963 as given in paragraph 174, the annual diet levels of Sr^{89} weighted by population are computed to be 44, 117 and 68 pCi Sr^{89}/g Ca for these years.

176. The dose commitments computed by using the last formula in paragraph 171 with the relative dose-rate factors for Sr^{89} deposited in 1961, 1962 and 1963 are 0.43 and 0.09 mrad to bone and to bone marrow, respectively. During 1961 and 1962, some 7 MCi Sr^{90} were injected into the atmosphere, while the total amount injected up to 1960 had been 5 MCi. The dose commitments due to Sr^{89} from all tests up to the end of 1962 can therefore be estimated to be

$$\frac{12}{7} \times 0.22 = 0.74$$

to bone and 0.15 mrad to bone marrow.

177. As for Sr^{90}, in addition to the dose commitments, the annual doses from Sr^{89} in new bone can be calculated. The average annual doses in 1961 and in 1962 were about one-third of the average doses to new bone from Sr^{90} during these years. This estimate is based on a diet of fresh milk and will therefore over-estimate the average dose from Sr^{89}. It should also be noted that the exposure to Sr^{90} is continuing while the Sr^{89} doses are limited to a few years.

Caesium-137

178. The dose commitment from internal irradiation due to Cs^{137} is calculated on the assumption that the yearly average Cs^{137}/K ratio in the body (Q_i) can be related to the deposition of Cs^{137} by the formula given in paragraph 134 and by using the average proportionality factors P_r and P_{2c} given in table XXVII. The total integrated body burden in the northern hemisphere, Q_n, will be

$$Q_n = \sum_{i=1}^{\infty} Q_i = (P_r + 2P_{2c}) \sum_{i=1}^{\infty} f_i = (P_r + 2P_{2c})\, F_{cc},$$

where F_{cc} is the total expected deposit of Cs^{137} (mCi/km²) in the northern hemisphere. The total integrated body burden for the southern hemisphere, Q_s, is computed in a similar manner. Weighting these Q_n and Q_s values by population and total expected deposition in each hemisphere, a weighted average world-wide integrated body burden of Cs^{137} of 640 pCi/y/gK, due to all tests performed so far, is obtained.

179. As discussed in paragraph 122, for purposes of dosimetry it can be assumed that the distribution of Cs^{137} in the body is uniform. The dose rate from one pCi Cs^{137}/g tissue maintained at constant level is assumed to be 10 mrad/y, or, assuming an average body content of 140 gK, 0.02 mrad/y per pCi/gK, as given in the 1962 report. The calculated dose commitment due to internal irradiation by Cs^{137} is therefore 13 mrads for all tests performed up to the end of 1962.

180. The assumptions used here in the calculation of the dose commitment from internally deposited Cs^{137} differ from those used in the 1962 report. The resulting figures are therefore not directly comparable. It is believed, however, that the present estimate based upon recent information is more satisfactory.

Iodine-131

181. The total accumulated radiation dose to the thyroid (D) can be expressed by the formula

$$D = \frac{K \times I \times F \times T,}{m}$$

where K = dose-rate factor in mrad/d per pCi/g tissue; F = fraction of ingested I^{131} reaching the thyroid; I = total integrated intake of I^{131} in picocuries over any given period of time; T = mean effective time of I^{131} storage in the gland; m = mass of the thyroid. To calculate the accumulated thyroid dose to the population of infants and young children (table XXX, columns 6-8), the following values for the parameters of the above equation have been assumed:

K = 0.010 mrad/d per pCi/g; F = 0.3; I = product of values given in columns 3-5 of table XXX and assumed consumption of 0.7 l/d milk; T = 11 days[341]; m = 2g in the first two years of age.

The thyroid doses given in table XXX, based upon a daily consumption of 0.7 l of milk, apply to the highest exposed population group, i.e., those young children who during their first year of life are brought up on fresh milk.

182. It should be pointed out that slightly lower thyroid masses[342] and lower values for fractional uptake of I^{131} than those assumed above[250, 257, 343-345] have been reported in recent studies. Furthermore, it should be noted that there are three sources of milk in infants' diet: human milk, fresh milk from animals such as goats and cows, and dried or evaporated (stored) milk. Only fresh milk of animal origin contributes I^{131} to this diet. In the United States it has been shown that about 50 per cent of the infants consume fresh milk,[216] and a similar figure probably holds for much of Europe.[364] For these reasons, the figures in table XXX are substantially greater than the average total dose to the thyroids of infants.

183. For lack of adequate data on milk consumption in adults, doses from I^{131} have not been calculated in this age group. It can be noted, however, that they should have been one or two orders of magnitude lower than

the doses in infants since the mass of the gland is approximately 10 times larger in adults than in children and because the average consumption of milk is probably much lower.

Radio-nuclides in the respiratory and gastro-intestinal tracts

184. Only a few determinations of Pu^{239} and fission products in the respiratory tracts of a very few individuals have been published. The fragmentary information available is summarized in table XXXVIII. The average dose from all insoluble nuclides over the whole respiratory system as estimated by using the data tabulated by the International Commission on Radiological Protection,[341] was of the order of a few millirads per year in 1962 and 1963, assuming that the concentrations of the nuclides as given in table XXXVIII were maintained over the whole period. As the measurements were mostly done in periods of peak concentration of debris in the air, the effective mean concentration in the period 1961-1963 and the corresponding mean doses in the organ would be lower than those in the table. Where nuclides were determined separately in lungs and tracheo-bronchial lymph nodes, the concentrations in the latter were higher by an order of magnitude, implying a correspondingly higher dose rate.

185. No data on the total ingested activity of artificial origin during the period 1961-1963 are available at present. As noted in the 1962 report, it is likely that the dose to the intestines from fission products in the gastro-intestinal tract is negligible.

Carbon-14

186. The natural production rate of C^{14}, as was shown in paragraph 60, is 2.6×10^{26} atoms per year. The radiation dose rate due to this natural C^{14} is 1.64 mrad/y to the bone, 1.15 mrad/y to cells lining bone surfaces, and 0.71 mrad/y to bone marrow and soft tissue. These quantities are the same as used in the 1962 report.

187. As shown in the 1962 report, the dose commitment D_∞ is given by

$$D_\infty = \gamma_o \frac{Q}{B},$$

where γ_o is the dose rate due to natural C^{14}; B is the natural production rate of C^{14}; and Q is the total artificial C^{14} inventory. As shown in table XV, the inventory of artificial C^{14} at the end of 1963 was about 65×10^{27} atoms. The dose commitment due to testing up to the end of 1963 is therefore 410 mrads to bone cells, 290 mrads to cells lining bone surfaces, and 180 mrads to soft tissue and bone marrow.

188. The dose received by year $1964 + t$

$$D_t = 10^{-2}\gamma_o \left[220 + \int_o^t f(t)dt\right],$$

where $f(t)$ represents the air activity of excess C^{14} in per cent above the natural level at time t years after 1964. The dose is given by

$$D_t = 10^{-2}\gamma_o \left[220 + \int_o^t (2.5e^{-0.00012t} + 64e^{-0.026t} + 26e^{-0.35t} - 26e^{-0.59t})dt\right].$$

For $t = 36$, that is by the year 2000, the dose received will be about 7 per cent of the dose commitment.

V. Summary

189. In this annex, most of the doses are expressed as dose commitments. This concept has been used because it would permit the calculation of the number of injuries expected in the future as a result of any given test series if the population size to whom the dose commitment applied and if the appropriate proportionality factors characterizing a linear dose-effect relationship with no threshold were valid and known. The number of injuries would then simply be the product of population size, proportionality factor and dose commitment.

190. As discussed in the 1962 report, when the proportionality factor is not known, an alternative to the calculation of the total number of injuries is through the evaluation of comparative risks by reference to doses from natural sources of radiation.

191. In the 1962 report, doses and dose commitments were expressed in rems. Since that time, the rem has been re-defined by the ICRU and is no longer an appropriate unit for the purposes of the Committee. In the present report dose commitments are expressed in rads. For radiations resulting from nuclear explosions, rad, as used here, and rem, as defined in the 1962 report, are numerically equivalent. In this report, doses from natural radiation also are expressed in rads, and will therefore be numerically slightly smaller than in the 1962 report where they were expressed in rems. They are 99, 96 and 95 millirads to gonads, cells lining bone surfaces and bone marrow, respectively.

192. The inherent difficulty in comparing dose commitments from nuclear tests with doses from natural sources of radiation, arises from the arbitrary period over which the natural radiation dose must be integrated. In principle, several alternatives are possible:

(1) The dose commitment could be compared with the natural radiation dose delivered over a period of time equal to that over which a substantial part of the dose commitment is delivered. This comparison could be misleading in the sense that exposures from future nuclear tests might overlap this period.

(2) As in the 1962 report, a comparison could also be made with the natural radiation dose delivered during the period of testing, with the justification that it is the commitment incurred during this period which is relevant, irrespective of the radiation source. However, this comparison may also be considered unsatisfactory because the period is not easy to define.

(3) A direct comparison between dose commitments (millirads) and annual dose rates from natural radiation (millirad/year) is hardly justified.

(4) An alternative approach, which was also used in the 1962 report and is followed here, is to express the dose commitments in terms of the period of time during which natural radiation would have to be doubled to give a dose increase equal to the dose commitment.

193. The dose commitments to the world population, due to radio-activity released into the atmosphere as a consequence of nuclear explosions carried out to the end of 1962, when such tests ceased, are summarized in table XXXIX. The dose commitments are given for specific tissues for the most important of the radio-active substances released into the environment through nuclear tests. The dose commitments computed in the 1962 report for the period of testing 1954-1960 are tabulated for comparison. For C^{14} it has seemed appropriate to include only the dose which is accumulated up to the year 2000, at which time the doses from the other nuclides are essentially delivered in full. The *total* dose commit-

ments for C^{14} which will be delivered over thousands of years are given in a footnote to the table.

194. If the northern and southern hemispheres are considered separately, it is found that the dose commitments for the northern hemisphere (excluding the contribution from C^{14} after the year 2000) are slightly larger than those for the whole world population. On the other hand, the dose commitments for the southern hemisphere are much smaller than the world average (amounting to 20 per cent).

195. For all tests carried out before January 1963, the periods of time during which natural radiation would have to be doubled to give a dose increase equal to the dose commitment to the world population amount to approximately 9 months for the gonads, 32 months for the cells lining bone surfaces and 20 months for the bone marrow. These periods are not directly comparable with the periods given in the 1962 report because they only take into account that part of the dose from artificial C^{14} which is delivered before the year A.D. 2000. In addition, the periods given in the 1962 report related to the test period 1954-1961 and involved an assumption regarding the testing practice in 1961.

TABLE I. SOME ESTIMATES OF MEAN STRATOSPHERIC RESIDENCE TIMES COMPUTED BY ALTERNATIVE METHODS

Nuclide	*Year of measurement*	*Hemisphere*	*Mean residence time, T_m, in years*	*Method of calculation*	*Reference*
W^{185}	1959–1960	Northern	0.6	[a]	23
Sr^{90}	1960	Northern	2.0	[b]	23, 27
Sr^{90}	1961	Northern	1.2	[b]	23, 27
Sr^{90}	1963	Northern	2.1	[b]	23, 419
Sr^{90}	1959, 1961	Northern	1.4	[c]	27
W^{185}	1959–1960	Southern	1.8	[a]	23
Sr^{90}	1959	Southern	2.3	[b]	23, 27
Sr^{90}	1960	Southern	2.8	[b]	23, 27
Sr^{90}	1961	Southern	2.0	[b]	23, 27
Sr^{90}	1959, 1961	Global	2.0	[c]	27
Sr^{90}	1960	Global	2.5	[b]	23, 27
Sr^{90}	1961	Global	1.8	[b]	23, 27
Sr^{90}	1963	Global	2.0	[b]	23, 419

[a] Calculated from $T_m = 1/\ln(I_1/I_2)$, where I_1 is the stratospheric inventory for May 1959 and I_2 for May 1960.
[b] Calculated from $T_m = 1/[\ln I/(I-F)]$ where I is the stratospheric inventory in January and F the subsequent annual deposit.
[c] Calculated from $T_m = 1/\ln(F_1/F_2)$, where F_1 and F_2 are the total deposits in 1959 and 1961.

TABLE II. ESTIMATES OF THE MEAN TROPOSPHERIC EXCHANGE TIME BETWEEN HEMISPHERES

Author	*Tracer used*	*Exchange time years*
Fergusson[56]	Dilution of C^{14} in atmosphere by burning of fossil fuels	< 1.8
Vogel and Münnich[57]	Bomb-produced C^{14}	> 1–2
Münnich and Vogel[141]	Bomb-produced C^{14}	< 1
Bolin and Keeling[54,58]	Fossil fuel CO_2	> 0.9
Junge[58] using data of Bishop *et al.*[410]	Tritiated methane	3.4

TABLE III. APPROXIMATE FISSION AND TOTAL YIELDS (MEGATONS) OF NUCLEAR WEAPONS TESTS CONDUCTED IN THE ATMOSPHERE BY ALL NATIONS [70]

Years	*Fission yield*		*Total yield*	
	Air	*Surface*	*Air*	*Surface*
1945–51	0.02	0.5	0.2	0.6
1952–54	1	37	1	59
1955–56	5.6	7.5	11	17
1957–58	31	9	57	28
1959–60				
1961	25		120	
1962	76		217	
TOTAL	140	54	406	105

TABLE IV. GLOBAL Sr^{90} INVENTORY [23, 28, 420]
(*Megacuries*)

	May 1960	*May 1961*	*April 1962*	*January 1963*	*September 1963 (prelim.)*	*January 1964 (prelim.)*
Stratosphere						
Northern hemisphere						
To 21 km	0.25	0.22	1.11	4.51	2.61	2.7
21–30 km	0.25	0.12	0.13	1.21	1.22	0.7
Southern hemisphere						
To 21 km	0.25	0.26	0.21	0.42	0.70	0.4
21–30 km	0.19	0.12	0.05	0.10	0.43	0.2
TOTAL, stratosphere	0.94	0.72	1.50	6.24	4.96	4.0
Troposphere	0.03	0.03	0.16	0.32	0.20	0.30
TOTAL, atmosphere	1.0	0.8	1.7	6.6	5.2	4.3
World-wide deposition[a]	5.0	5.2	5.8	6.7	8.6	9.0
TOTAL	6.0	6.0	7.5	13.3	13.8	13.0

[a] Estimates from soil samples have been increased by 15 per cent over those given in reference 23 to account for incomplete radio-chemical extraction. [105] These figures are an independent assessment of the world-wide Sr^{90} deposition and are not the ones used in tables VII and VIII.

TABLE V. AVERAGE MONTHLY AND ANNUAL Sr^{90} DEPOSITION IN THE USSR [81, 83, 418]

Region of sampling	*1961 mCi/km² per month*	*1962 mCi/km² per month*				*1962*	*1963 mCi/km² per month*				*1963*
	4th quarter	*1st quarter*	*2nd quarter*	*3rd quarter*	*4th quarter*	*Annual total mCi/km²*	*1st quarter*	*2nd quarter*	*3rd quarter*	*4th quarter*	*Annual total mCi/km²*
Murmansk region	0.22	0.29	2.1	1.3	0.70						
Leningrad region	0.23	0.22	2.4	0.93	0.34		0.3	1.5	2.3	0.22	13.0
Moscow region	0.15	0.33	1.7								
Kiev				0.40	0.40		1.8	1.6	3.0	0.6	21.0
Krasnojarsk territory		0.11					0.5	3.0	2.3	0.4	18.6
South Sakhalin	0.24	0.19	1.0	1.0	1.1		1.7	2.9	3.0	0.9	25.8
Latitude regions of European and mid-Asian USSR											
60–70°N							0.7	2.7	2.6	0.5	19.5
50–60°N							1.1	3.4	2.9	0.7	24.3
40–50°N							1.1	3.3	2.9	0.8	24.3
37–40°N							1.2	2.2	1.1	0.6	15.3
Average for USSR mCi/km²		0.23	1.5	0.8	0.7	9.6	1.1	3.0	2.8	0.7	22.5

TABLE VI. LATITUDINAL DISTRIBUTION OF Sr^{90} ESTIMATED FROM MONTHLY FALL-OUT COLLECTIONS [27, 124, 376, 419]

Latitude band	*1961 mCi/km²*	*1962 mCi/km²*	*1963 mCi/km²*	*Latitude band*	*1961 mCi/km²*	*1962 mCi/km²*	*1963 mCi/km²*
80–70°N	[a]	1.5	4.0	10–0°N	0.7	2.7	3.7
70–60°N	[a]	4.8	10.6				
60–50°N	1.4	6.6	14.9	0–10°S	0.58	1.8	1.2
50–40°N	2.0	8.6	16.3	10–20°S	0.53	0.74	0.78
40–30°N	1.6	6.7	10.9	20–30°S	0.87	1.2	1.1
30–20°N	1.4	6.0	11.0	30–40°S	0.96	0.91	1.5
20–10°N	0.6	2.4	4.5	40–50°S	0.72	1.3	1.5

[a] Insufficient data available.

TABLE VII. ANNUAL AND CUMULATIVE DEPOSITION OF STRONTIUM-90 [27, 104, 376, 379, 419]

Period	Northern Hemisphere 0–80°N		Southern Hemisphere 0–50°S		Total 50°S–80°N
	MCi	mCi/km²	MCi	mCi/km²	MCi
1961	0.31	1.2	0.15	0.77	0.46
1962	1.3	5.2	0.23	1.2	1.5
1963	2.3	9.1	0.23	1.2	2.5
Cumulative total to 31 December 1963[a]	7.9	31	1.5	7 7	9.4

[a] Corrected for decay.

TABLE VIII. CUMULATIVE Sr^{90} DEPOSITION BY LATITUDE BAND AS ESTIMATED FROM SOIL DATA AND MONTHLY FALL-OUT MEASUREMENTS [27, 104, 124, 376, 407, 419]

Latitude	(Preliminary) Estimated from analysis of soil samples collected between June 1963 and March 1964[a]		Estimated from 1960 soil data plus precipitation up to December 1963[a]
	mCi/km²	MCi	MCi
70–80°N	21	0.2	0.15
60–70°N	32	0.6	0.6
50–60°N	51	1.3	1.1
40–50°N	58	2.0	1.6
30–40°N	47	1.7	1.6
20–30°N	40	1.6	1.7
10–20°N	24	1.0	0.7
0–10°N	8	0.3	0.5
Northern Hemisphere		8.7	8.0
0–10°S	5	0.2	0.3
10–20°S	5	0.2	0.2
20–30°S	7	0.2	0.3
30–40°S	9	0.3	0.3
40–50°S	7	0.2	0.3
Southern Hemisphere		1.1	1.4
TOTAL		9.8	9.4

[a] All the results from soil analysis have been increased 15 per cent to compensate for incomplete chemical extraction.

TABLE IX. AVERAGE Cs^{137}/Sr^{90} RATIOS IN STRATOSPHERIC AIR AND IN PRECIPITATION

	Before September 1961	After September 1961
Mean for precipitation collected at 20 sites in northern hemisphere[45, 76]	January–August 1961 1.74±0.05[a] (36 cases)	September 1961–June 1962 1.42±0.04 (84 cases)
In stratospheric air over San Angelo, Tex.,[73] by balloon sampling[b]	January–August 1961 1.77±0.06 (8 cases)	January 1962–May 1963 1.51±0.03 (21 cases)
In stratospheric air from aircraft sampling[11, 408]	January 1959–March 1960 1.71±0.03 (217 cases)	March 1962–October 1963 1.59±0.02 (114 cases)

[a] Standard error of the mean.
[b] Each monthly ratio was based upon samples collected at several altitudes.

TABLE X. DEPOSITION OF Cs^{137} AS COMPUTED FROM Sr^{90} DATA GIVEN IN TABLES VII AND VIII

Period	Northern Hemisphere 0–80°N		Southern Hemisphere 0–50°S		Total 50°S–80°N
	MCi	mCi/km²	MCi	mCi/km²	MCi
1961	0.5	2.0	0.25	1.3	0.78
1962	1.9	7.5	0.30	1.5	2.2
1963	3.5	14	0.30	1.5	3.8
Cumulative to 31 December 1963[a]	13.2	52	2.5	13	15.7

[a] Corrected for decay.

TABLE XI. DEPOSITION OF STRONTIUM-89 [27, 124, 276]

Latitude band	1961		1962		1963	
	mCi/km²	MCi	mCi/km²	MCi	mCi/km²	MCi
80–70°N	[a]	[a]	13	0.16	5.4	0.06
70–60°N	[a]	[a]	90	1.7	70	1.3
60–50°N	[a]	[a]	105	2.7	81	2.1
50–40°N	58	1.8	135	4.2	103	3.3
40–30°N	44	1.6	135	4.9	93	3.4
30–20°N	33	1.3	107	4.3	80	3.2
20–10°N	15	0.65	61	2.6	27	1.2
10–0°N	16	0.73	49	2.2	28	1.2
0–80°N	31	6.1	95	24	63	15.8
0–10°S	13	0.59	29	1.3	12	0.52
10–20°S	0.0	0.0	16	0.69	1.4	0.06
20–30°S	0.0	0.0	15	0.60	2.9	0.12
30–40°S	0.0	0.0	9.2	0.33	2.7	0.10
40–50°S	0.0	0.0	7.7	0.24	1.8	0.06
0–50°S			16	3.2	4.4	0.86

[a] No data available.

TABLE XII. DEPOSITION OF Cs^{137}, Zr^{95}, Ba^{140} AND Ce^{144} (mCi/km²) AT MILFORD HAVEN AND CHILTON, UNITED KINGDOM, AND OF I^{131} AT CHILTON [45]

Date	Milford Haven					Chilton					
	Rain cms	Cs^{137}	Zr^{95}	Ba^{140}	Ce^{144}	Rain cms	Cs^{137}	Zr^{95}	Ba^{140}	Ce^{144}	I^{131}
1962											
January	12.1	1.2	25	5.1	21	10.6	0.91	26	3.2	9.8	[a]
February	2.3	2.3	6.8	0.3	5.2	0.9	0.15	2.8	0.14	2.2	[a]
March	9.6	2.1	26	0.5	17	3.3	0.54	7.8	0.10	6.9	[a]
April	5.4	1.1	4.3	[a]	8.8	4.1	0.95	8.7	[a]	10.7	[a]
May	6.6	1.7	3.0	[a]	16	4.3	0.80	5.3	[a]	10.3	[a]
June	3.4	1.0	4.2	[a]	12	0.5	0.27	1.3	[a]	3.8	[a]
July	4.5	1.2	3.2	[a]	7.5	2.8	0.81	3.1	[a]	6.7	[a]
August	11.4	1.5	5.5	3.5	18	11.3	1.3	4.4	1.2	14	[a]
September	13.7	1.2	7.1	18	17	8.1	0.77	6.6	13	6.7	40
October	3.3	0.38	6.2	13	5.5	3.5	0.29	3.4	8.4	3.3	8.4
November	8.68	1.0	14	33	14	8.3	0.73	13	34	14	7.5
December	8.75	1.2	20	35	23	5.2	0.51	20	21	12	12
1963											
January	2.3	0.4	7.9	3.7	6.8	2.8	0.62	13	8.5	15	1.2
February	5.5	1.6	16	4.5	21	1.0	0.57	5.8	1.0	8.3	[a]
March	12.8	4.5	27	2.6	54	10.0	3.4	27	1.5	37	[a]
April	10.9	3.9	24	0.1	46	5.6	2.9	15	[a]	32	[a]
May	4.8	4.1	15	[a]	52	2.2	1.5	5.0	[a]	19	[a]
June	9.6	4.9	19	[a]	50	5.7	3.1	11	[a]	40	[a]
July	5.2	2.0	4.8	[a]	26	4.4	2.1	4.2	[a]	25	[a]

[a] Not detected.

TABLE XIII. SUM OF MONTHLY DEPOSITS (mCi/km²) OF SHORT-LIVED ISOTOPES DURING 1962

Collection station	Sr^{89}	Zr^{95}	Ru^{103}	Ru^{106}	I^{131}	Ba^{140}	Ce^{141}	Ce^{144}
United States								
Westwood, N.J.[27]	240	390					160	305
Pittsburgh, Pa.[27]	190	290					360	290
Richmond, Cal.[27]	90	110		41			53	73
Houston, Tex.[27]	180	340				330	220	300
United Kingdom								
Milford Haven[45]	161	125				108		165
Chilton[45]	63	103			68	81		100
Italy								
Ispra[91]	131	270	190	190			110	350

TABLE XIV. DISTRIBUTION OF CARBON AND NATURAL C^{14} BETWEEN EXCHANGEABLE RESERVOIRS [126]

Carbon reservoir	*Mass of carbon* (g/cm^2)[a]	*Natural C^{14} content* (10^{27} *atoms*)
Atmosphere	0.12	40
Biosphere (terrestrial)	0.06	19
Humus	0.20	55
Surface waters of ocean (above thermocline)	0.18	55
Remainder of ocean	7.5	2,000
TOTAL	8.1	2,170

[a] Grammes per square centimetre of the earth's surface.

TABLE XV. DISTRIBUTION OF EXCESS C^{14} BETWEEN RESERVOIRS AND TOTAL INVENTORY [1, 22, 34, 42]
(10^{27} *atoms*)

Reservoir	*July 1957*	*July 1958*	*July 1959*	*November 1960*	*May–June 1961*	*March–April 1963*	*July 1963*	*January 1964*
Stratosphere	7.4	8.4	12.0[a]	6.4	8.1	23.6	26.7	22.0
Troposphere	2.5	4.0	6.7	10.5	11.8	24.5	28.4	26.0
Ocean[a]	0.5	1.2	2.2	5.1	6.2	12.1	13.4	15.4
Biosphere[a]	0.1	0.2	0.4					
TOTAL	10.5	13.8	20.0	21.4	25.3	60.2	68.5	63.4

[a] Computed. In the case of ocean uptake, it is based on an annual 20 per cent oceanic uptake of the tropospheric C^{14} content.

TABLE XVI. Sr^{90} TO CALCIUM RATIO IN MILK

The values are given in pCi/g Ca and represent yearly averages unless otherwise indicated

Types of study: A—Systematic widespread survey
B—Systematic local survey
C—Irregular sampling

Region, area or country	*Latitude*	*1961*	*1962*	*1963*	*Type of study*	*Reference*
NORTH AMERICA	>15°N					
Canada	40–55°N	8.4[a]	19.4[a]	27.8[a]	A	262, 263
United States	25–48°N	7±2[b]	11±4[b]	19±6[b]	A	164
Alaska	62°N	7	9	18	A	164
New York City, N.Y.		6.7	12	26	B	165
Chicago, Ill.		4.3	7.0	14.1	C	165
San Francisco, Cal.		1.7	3.5	10.2	C	165
Mexico	~15–30°N		0.9	0.9[c]	B	267
EUROPE	>30°N					
Austria	47–49°N	10.3	16.9		A	268, 386
Belgium	~50°N	4.2[d]			A	79
Czechoslovakia	48–51°N	4.6[e]	8.4[f]		C	269
Denmark	55–60°N	4.1	10.1[g], 8.1[h]	23.8[g], 24.1[h]	A	97, 167, 179
Faroes	60–70°N		68	131	A	98, 179
Federal Republic of Germany	43–55°N			17.4[s]	*	387
Finland	60–67°N	5.6[h]	13.0[h]	22.7[h]	A	270
France	42–50°N	7.9	16.8	26.7[i]	A	282
Ireland	52–55°N		21.5[j]	23.7[k]	A	169, 272
Italy	37–47°N	5.7[l]	12.4[l]		A	42, 100, 101, 273
Netherlands	52–54°N	4.4	9.3[ss]	24.5[ss]	A	274, 275, 388
Norway	58–70°N	12.1[a]	18.5[a]		A	88, 276
Poland	50–55°N	5.8[h, n]	7.8[h, o]		A	183, 184
Sweden	55–70°N			26	C	277
Switzerland	~47°N	7.7–18.9[p]	16.7[a]		B	278, 279
United Kingdom	50–60°N	5.9[h]	11.7[h]	25.6[h]	A	168, 169, 280
USSR						
Moscow region	~55°N	4.4	6.4[m]		B	281
Ryazan region	~55°N	6.1[r]			C	
NEAR EAST	>30°N					
Israel			4.8[u]	8.7[m]	C	283
ASIA						
India, Bombay	20°N	1.4	2.6		B	284, 285
FAR EAST						
Japan	30–50°N		8.8	14.9	AC	186, 286, 287, 288, 289
PACIFIC						
United States, Hawaii	21°N	4	4	9	B	164
CENTRAL AMERICA						
United States, Puerto Rico	18°N	3	9	12	B	164
SOUTH AMERICA						
Argentina (littoral area)	~36°S	2.5	3.3	3.7	B	265
AFRICA						
United Arab Republic	20–30°N	4.6[x]	7.7[y]	16.0[y]	A	290, 291, 292
OCEANIA						
Australia	10–40°S	4.4[a]	5.9[a]	5.8[yy]	A	293, 294, 395
New Zealand	35–47°S	4.9[a]	6.1[a]	7.1[a]	A	295, 296, 389

* Type unknown.
[a] Unweighted mean for all stations.
[b] Country-wide mean weighted by population.
[c] February–July only.
[d] July–December only.
[e] Limited sampling over 9 months.
[f] Limited sampling over 4 months of 1962.
[g] Dried milk country-wide mean weighted by production.
[h] Fresh milk country-wide mean weighted by production.
[i] January–August, unweighted mean for 7 collecting stations.
[j] April–December only.
[k] September 1962–September 1963.
[l] Mean weighted by population.
[m] January–June only.
[n] August 1960–July 1961.
[o] August 1961–July 1962.
[p] Range of values for lowland and mountain regions, respectively.
[r] January–May only.
[u] April–December only.
[x] Delta region, July–December only.
[y] Average for delta region and Upper Egypt.
[yy] January–February only.
[s] January–September only.
[ss] Country-wide mean weighted by consumption.

TABLE XVII. Sr^{89} IN MILK IN THE YEARS 1961, 1962 AND 1963

The values represent yearly means in pCi/g Ca unless otherwise indicated

Type of study: A—Systematic widespread survey
B—Systematic local survey
C—Irregular sampling

Region, area or country	*Latitude*	*61*	*1962*	*1963*	*Type of study*	*Reference*
NORTH AMERICA						
Canada	40–55°N		106[b, c]	32[b]	A	262, 263
United States	25–48°N	9 ± 6[a]	38 ± 34[a]	37 ± 21[a]	A	164
Alaska	62°N	14	45	25	B	164
EUROPE						
Denmark	50–60°N	18[b]	48[b]	56[b]	A	97, 167, 179
Faroes	60–70°N		333[c]	352	A	98, 179
Italy	37–47°N		63[a]		A	100, 101
Netherlands	52–54°N		44[b]	32[b, f]	A	246
United Kingdom	50–60°N	140[d, c]	60[c]	45[c]	A	168, 169, 280
CENTRAL AMERICA						
United States, Puerto Rico	18°N	13	61	59	B	164
PACIFIC						
United States, Hawaii	21°N	2	24	15	B	164

[a] Country-wide mean weighted by population ± standard deviation.
[b] Unweighted mean for all sampling stations.
[c] April–December only.
[d] October–December only.
[e] Country-wide mean weighted by production.
[f] January–June.

TABLE XVIII. Sr^{90} AND CALCIUM IN TOTAL DIET AND ITS COMPONENTS

Ca in grammes per day in brackets. Sr^{90} in pCi per day. Sr^{90}/Ca ratio in total diet in pCi/g Ca. (column 10)

Types of study: A—Widespread survey of individual foodstuffs
B—Systematic widespread diet analyses
C—Regular local sampling
D—Irregular sampling

Region, area or country	*Latitude*	*Year*	*Milk and milk products*	*Cereals*	*Fruits and leafy vegetables*	*Root vegetables*	*Miscellaneous*	*Type of study*	*Total diet in pCi/g Ca*	*Reference*
NORTH AMERICA										
Greenland	> 60°N	1962	3.2	4.6	0.5[a]	0.2[b]	1.5–6.6[c]	A	6.6–9.9[c]	99
United States	25–49°N		(0.64)	(0.16)	(0.10)	(0.04)	(0.11)			
New York City, N.Y.		1961	4.8	1.8	1.1	1.0	1.4	C	9.7	165
		1962	6.7	2.5	1.6	1.1	1.7	C	12.8	165
		1963	18.4	6.6	2.6	1.3	2.8	C	30.0	165
Chicago, Ill.		1961	2.6	1.9	1.1	0.7	0.9	C	7.1	165
		1962	4.3	2.8	1.2	0.8	1.4	C	10.7	165
		1963	8.4	6.4	2.0	1.1	1.5	C	19.2	165
San Francisco, Cal.		1961	1.1	0.8	0.4	0.5	0.7	C	3.3	165
		1962	2.4	1.5	0.4	0.5	0.8	C	5.2	165
		1963	6.9	3.9	0.9	0.7	1.6	C	12.6	165
United States (institutional diet sampling)[t]		1961						B	7 5–11[v]	366
		1962						B	10 5–20[v]	366
		1963						B	22 9–35[v]	366
United States, Alaska (institutional diet sampling)	62°N	1961						C	7	366
		1962						C	12	366
		1963						C	24	366

TABLE XVIII. Sr^{90} AND CALCIUM IN TOTAL DIET AND ITS COMPONENTS *(continued)*

Ca in grammes per day in brackets. Sr^{90} in pCi per day. Sr^{90}/Ca ratio in total diet in pCi/g Ca. (column 10)

Types of study: A—Widespread survey of individual foodstuffs
B—Systematic widespread diet analyses
C—Regular local sampling
D—Irregular sampling

Region, area or country	*Latitude*	*Year*	*Milk and milk products*	*Cereals*	*Fruits and leafy vegetables*	*Root vegetables*	*Miscellaneous*	*Type of study*	*Total diet in pCi/g Ca*	*Reference*
EUROPE	> 30°N									
Austria			(0.52)	(0.08)	(0.05)	(0.02)	(0.04)			
		1961	5.9	2.6	0.8	0.5		A	13.8	268
		1962						A	26.7[d]	297, 386
									32.7[e]	
		1963								
Denmark		1961	3.1	3.8	1.5[a]	0.5[b]	0.4	A, B	5.9	167
		1962	7.6	7.3	3.1[a]	0.5[b]	1.1	A	12.4	97
		1963	17.9	28.0	4.4[a]	0.9[b]	2.0	A	31.3	179
								B	30.8[f]	
Faroes		1962	27.8	7.5	1.5[a]	2.7[b]	4.8	A	26.9	98
		1963	54.0	27.8	2.2[a]	5.0[b]	7.1	A	58.5	179
Federal Republic of Germany		1962						C	19.8[g]	298
		1963								
France[u]	42–50°N		(0.54)	(0.07)	(0.09)	(0.04)				
		1961	3.9	1.6	0.9	0.4[b]		A	9.2	374
		1962	8.1	1.8	2.1	0.5[b]		A	16.9	374
Poland										
Towns			(0.43)	(0.16)	(0.04)	(0.03)	(0.04)			
		1961[h]	2.5	8.1	0.9	1.2[b]	0.4	A	18.2	184
		1962[i]	3.1	7.0	0.8	1.3[b]	0.4	A	17.8	183
		1963								
Rural areas			(0.88)	(0.22)	(0.07)	(0.04)	(0.02)			
		1961[h]	5.1	10.7	1.3	1.6[b]	0.1	A	15.4	184
		1962[i]	6.2	9.5	1.4	1.6[b]	0.1	A	15.2	183
		1963								
United Kingdom			(0.59)	(0.05)	(0.06)	(0.02)	(0.36)[k]			
		1961	3.4	1.0	0.6	0.6	1.0[j]	A	6.2	168
		1962	6.6	0.9	1.1	0.6	1.5[j]	A	9.9	169
		1963	14.6	3.4	2.2	1.3	3.2[j]	A	22.8	280
FAR EAST	30–45°N									
Japan		1961		2.7	3.0[l]	2.2	0.6[m]	D[n]	18.5	170, 221
				(0.05)	(0.14)	(0.08)	(0.20)			
		1962		1.3	5.6[l]	2.6	1.1[m]	D[o]	17.8	170, 221
				(0.07)	(0.28)	(0.05)	(0.20)			
		1963		1.7	9.4[l]	3.5	0.8[m]	D[p]	24.6	170, 221
				(0.05)	(0.31)	(0.07)	(0.20)			
AFRICA										
United Arab Republic (delta region)	< 30°N	1961						C[r]	5.6	290
		1962						C	10.8	291
		1963						B	13.7[s]	292
SOUTH AMERICA										
Argentina (littoral area)	~ 35°S		(0.45)	(0.02)	(0.11)	(0.02)	(0.06)			
		1963	1.7	0.5	1.2	0.4	0.5	B	6.5	265
OCEANIA										
United States, Hawaii (institutional diet sampling)	21°N	1961						C	8	366
		1962						C	10	366
		1963						C	16	366
Australia	10–40°S		(0.64)	(0.05)	(0.07)	(0.01)	(0.05)			
		1961	2.8	0.4	0.4	0.1	0.2	A	4.7	293
		1962	3.8	0.3	0.5	0.2	0.1	A	5.9	294

[a] Fruits and vegetables.
[b] Potatoes only.
[c] Depending on the source of drinking water.
[d] Towns.
[e] Country.
[f] Country-wide mean weighted by population.
[g] July–October sampling of daily diet in one of Munich's hospitals.
[h] August 1960–July 1961.
[i] August 1961–July 1962.
[j] Including drinking water and tea.
[k] Including *cretа praeparata*.
[l] Including seaweeds.
[m] Including fish and shellfish, dairy products, eggs and meat.
[n] October 1961 in Tokyo only.
[o] Four sampling-series, at four locations each.
[p] Three sampling-series, at four locations each, January–June only.
[r] Second half of 1961 only.
[s] Average for Upper and Lower Egypt.
[t] Widespread dietary survey in 21 boarding schools in the United States based on composite diets of children and adolescents of 6–18 years of age.
[u] Representative of the southeastern region of France.
[v] Range of yearly averages reported for individual schools.

TABLE XIX. RELATIONSHIP BETWEEN RATIO OF STRONTIUM-90 TO CALCIUM IN MIXED DIET TO THAT IN MILK

Country, area or location	1960	1961	1962	1963	Reference
NORTH AMERICA					
United States (Tri-city study)					
New York City, N. Y.	1.5	1.4	1.3	1.3	165, 245, 309, 310
Chicago, Ill.	1.4	1.9	1.8	1.5	
San Francisco, Cal.	1.7	1.9	1.7	1.2	
United States (institutional sampling project)		1.5	1.2	1.4	182, 245, 309, 310
EUROPE					
United Kingdom	1.0	1.0	0.9	0.9	169, 280
Denmark	1.7	1.4	1.4		97, 167
Poland		2.5[a]	2.2[a]		183, 184
		3.2[b]	2.7[b]		
Austria		1.3			268
FAR EAST					
Japan	3.0		~2.0		1, 185, 186
AFRICA					
United Arab Republic (delta region)		1.2	1.4		290, 291
AUSTRALIA		1.2	1.1		293, 294

[a] Rural regions.
[b] Urban regions.

TABLE XX. Sr^{90} IN HUMAN BONE
pCi Sr^{90}/g calcium
(Number of samples in parentheses)

Region, country or area	Year	Newborn and/or stillborn	0–1 year	1 year	2 years	3 years	4 years	5–19 years	>19 years	Bone type studied (adults)	References
NORTH AMERICA											
30–50°N											
Canada	1961		2.11 (1)	———		1.99 (5)	———	1.86 (6)	1.00 (87)	Vertebrae	318, 319
	1962	1.25 (4)	2.20 (5)	———		3.24 (7)	———	1.61 (8)	1.21 (36)	Vertebrae	
United States											
New York City, N.Y.	1961		3.43 (8)	2.67 (5)	2.34 (2)	2.05 (5)	1.74 (4)	1.24 (35)	0.83 (7)	Vertebrae	320, 321, 322
	1962		3.81 (16)	3.05 (8)	2.68 (4)	2.61 (10)	1.93 (3)	1.84 (26)	1.00 (14)		
	1963 January–June			3.49 (1)	2.44 (3)	1.54 (1)	2.22 (1)	1.82 (16)	1.55 (2)	Vertebrae	
	1963 July–December		6.81 (10)	9.84 (1)	5.03 (3)	3.41 (2)		2.41 (10)	1.55 (21)	Vertebrae	
Chicago, Ill.	1961		1.40 (1)			2.26 (1)	2.08 (3)	1.15 (9)	0.55 (39)	Vertebrae	320, 321, 322
	1962			2.32 (1)		0.68 (1)	1.06 (1)	1.38 (10)	0.83 (50)		
	1963 January–June								0.74 (2)	Vertebrae	
	1963 July–December		3.51 (2)		3.70 (2)	2.76 (3)		2.42 (2)	1.19 (10)	Vertebrae	
San Francisco, Cal.	1961		0.49 (10)	0.79 (3)		1.33 (4)	2.15 (2)	0.92 (11)	0.48 (45)	Vertebrae	320, 321, 322
	1962		1.07 (26)	1.27 (2)	1.02 (6)	1.18 (5)	1.15 (4)	0.89 (19)	0.73 (9)	Vertebrae	
	1963 January–June		1.31 (11)	2.72 (2)	1.40 (6)	1.13 (5)	1.64 (2)	1.04 (11)	0.70 (5)	Vertebrae	
	1963 July–December		2.43 (21)		3.51 (1)		1.72 (3)	1.64 (16)	1.02 (17)		
CENTRAL AMERICA											
10–20°N											
United States											
San Juan, P. R.	1961							0.98 (23)	0.79 (28)	Vertebrae	320, 321
	1962							1.36 (33)	1.36 (13)	Vertebrae	

TABLE XX. Sr^{90} IN HUMAN BONE (*continued*)
pCi Sr^{90}/g calcium
(Number of samples in parentheses)

Region, country or area	*Year*	*Newborn and/or stillborn*	*0–1 year*	*1 year*	*2 years*	*3 years*	*4 years*	*5–19 years*	*> 19 years*	*Bone type studied (adults)*	*References*
SOUTH AMERICA											
> 20°S											
Argentina (littoral area).	1961		0.71 (17)	0.77 (6)	0.75 (4)					Vertebrae	265
	1962		0.83 (7)	0.66 (7)	0.76 (2)					Vertebrae	
	1963	0.77 (23)								Vertebrae	
EUROPE											
45–70°N											
Czechoslovakia.....	1961		3.2 (3)	3.7 (1)		2.3 (1)	2.5 (1)	1.6 (1)			323
Denmark.....	1961							1.04 (19)	0.82 (45)	Vertebrae	97, 167, 179
	1962		3.80 (9)	2.68 (1)	1.66 (2)		1.61 (1)	0.94 (20)	0.81 (78)	Vertebrae	
	1963	2.80 (11)			4.23 (18)			2.17 (13)	1.53 (20)	Vertebrae	
Federal Republic of Germany.....	1961	0.88 (136)			1.23 (35)			1.12 (17)	0.38 (30)	Tibiae	204, 324
	1962	1.16 (118)			1.87 (25)						
Norway.....	1961	1.43 (11)	1.91 (27)		2.16 (17)			1.59 (25)	1.18 (14)	Vertebrae	325
	1962	0.99 (9)	3.60 (6)		1.83 (3)			1.99 (17)	1.05 (5)	Vertebrae	
	1963	1.80 (3)	7.1 (1)					3.7 (2)		Vertebrae	
Poland.....	1961		1.77 (58)	2.59 (12)	2.19 (8)		1.92 (5)	2.04 (28)	1.18 (191)	Vertebrae	326, 327
	1962	1.54 (26)	1.96 (29)	2.20 (3)	4.00 (1)			1.78 (9)	1.40 (160)	Vertebrae	
Switzerland.....	1961	1.10 (12)							0.80 (39)	Vertebrae and sternum	278, 279
	1962								0.58 (16)	Ribs	
	1962								0.92 (21)	Vertebrae	
	1962								0.68	Ribs	
United Kingdom.....	1961[a]	0.81 (282)	1.67 (105)	2.60 (24)	2.54 (18)	1.71 (7)	1.67 (12)	1.10 (68)	0.33 (25)	Femora	328, 329, 330, 331, 373
	1961[b]	0.69 (9)			1.83 (47)			1.00 (30)	0.68 (48)	Vertebrae	
	1962	0.99 (230)	2.00 (132)	2.38 (22)	2.55 (10)	1.75 (3)	2.37 (6)	1.19 (44)	0.32 (11)	Femora	
	1963 January–June	1.5 (56)	3.3 (73)	3.7 (10)	4.0 (6)	2.0 (8)	2.1 (8)	1.4 (35)	0.5 (9)	Femora	
USSR[c].....	1961	1.41 (13)	1.17 (7)		1.41 (2)			1.09 (6)	0.52[d] (112)	Different (normalized)	399
	1962	1.62 (62)	2.13 (52)		2.51 (14)			1.77 (1,661)	0.85[d] (2,071)	Different (normalized)	399
	1963		5.00 (6)		4.23 (7)			1.88 (1,567)	1.05[d] (4,142)	Different (normalized)	399
FAR EAST											
30–45°N											
Japan.....	1961	1.68 (2)			1.36 (9)			1.38 (51)	0.43 (92)	Ribs	332
	1962	0.88[c] (5)		1.86 (19)				1.38 (36)	0.44 (124)	Ribs	
	1963	1.35 (17)	1.68 (26)		1.43 (12)			1.40 (44)			
OCEANIA											
Australia											
20–40°S.....	1961	0.64 (226)	1.10 (273)	1.52 (34)	1.15 (24)	1.15 (27)	0.75 (12)	0.68 (191)	0.56 (879)	Vertebrae	293, 294
	1962	0.69 (203)	1.36 (234)	2.04 (27)	1.06 (21)	1.20 (11)	1.15 (12)	0.81 (234)	0.55 (751)	Vertebrae	

[a] AERE, Scottish and Cambridge results combined.
[b] West London survey.
[c] Including 4 foetuses.
[d] Skeletal averages obtained from different bones. In 1961 normalized according to Kulp and Schulert,[396] in 1962 and 1963 according to factors developed by the authors.[400]
[e] Average values for 9 areas in European and Asian territory of the USSR.

TABLE XXI. RATIOS OF Sr^{90}/Ca IN INFANTS' BONE TO THAT IN MILK FOR THE YEAR 1962

Country or location	Bone Sr^{90}/Ca / Milk Sr^{90}/Ca	Country or location	Bone Sr^{90}/Ca / Milk Sr^{90}/Ca
Argentina	0.25	Poland	0.25
Australia	0.24	United Kingdom	0.17
Canada	0.11	United States	
Denmark	0.42	New York	0.32
Japan	0.19	San Francisco	0.30
Norway	0.20		

TABLE XXII. COMPARISON OF Sr^{90} IN DIET AND ADULT BONES (VERTEBRAE) IN THE PERIOD 1961–1962
(Number of bone samples in parentheses) [a]

Country or location	Sr^{90}/Ca diet / Sr^{90}/Ca bones 1961-1962	Country or location	Sr^{90}/Ca diet / Sr^{90}/Ca bones 1961-1962
Australia	9.4 (1630)	United States	
Denmark	11.1 (123)	New York City, N. Y.	12.2 (21)
Poland	13.9 (351)	Chicago, Ill.	13.0 (89)
United Kingdom	10.2[a] (48)	San Francisco, Cal.	7.1 (22)

[a] 1961 only.

TABLE XXIII. Cs^{137} IN MILK

The values are given in pCi/l and represent yearly averages unless otherwise indicated

Type of study: A—Systematic widespread survey
B—Systematic local survey
C—Irregular sampling

Region, area or country	*Latitude*	*1961*	*1962*	*1963*	*Type of study*	*Reference*
NORTH AMERICA						
Canada	40–55°N		81.4[a]	172[a]	A	262, 263
United States	25–48°N	10 ± 11[b]	43 ± 18[b]	111 ± 37[b]	A	164
United States, Alaska	62°N	10	37	117	B	164
EUROPE						
Austria	47–49°N	49[c]	100[c]		A	268, 386
Denmark	55–60°N	14[a]	43[c,d] 35[c,e]	107[c,d] 112[c,e]	A	97, 167, 179
Faroes	60–70°N		535[f]	974[f]	A	98, 179
Federal Republic of Germany	43–55°N			127[r]	*	387
Finland	60–67°N			210[g]	C	299
France	42–50°N	25[c,d]	67[c,d]	220[h]	A	428
Ireland	50–55°N		77[i]		A	169
Italy	37–47°N	32[b]	80[b]		A	42, 100, 101, 273
Netherlands	52–54°N		54[b]	155[b]	A	246, 275, 388
Norway	58–70°N		75[c,k]	150[c,k] 444[c,l]	A	223, 367
Sweden	55–70°N	30	75[m]	185[m]	A,B (1961)	214, 277
Switzerland, Geneva	~46°N			103[n]	B	300
United Kingdom	50–60°N	21[c]	62[c]	135[c]	A	168, 169, 280
CENTRAL AMERICA						
United States Puerto Rico	18°N	5	40	88	B	164
OCEANIA						
United States, Hawaii	21°N	10	27	73	B	164
Australia	10–40°S			30[b]	A	397
New Zealand	35–47°S			56 ± 10[a,s]	A	295, 296, 389
FAR EAST						
Japan	30–50°N		57	119	A[o]	186, 286–289
ASIA						
India, Bombay	19°N	9.0	8.8		B	284, 285
AFRICA						
United Arab Republic	20–30°N		12.0		A	291
SOUTH AMERICA						
Argentina (littoral area)	35–55°S	17	19	13	B	394

* Type unknown.
[a] Unweighted mean for all milksheds surveyed.
[b] Country-wide mean weighted by population ± standard deviation.
[c] Country-wide mean weighted by production.
[d] Dried milk.
[e] Fresh milk.
[f] Locally produced (average consumed in 1962—412 pCi/l).
[g] Non-weighted average of 3 milksheds for the period October 1962–July 1963.
[h] Described as "laits départementaux".
[i] April–December only.
[k] February–March only.
[l] March 1963–February 1964. 31 stations regularly surveyed.
[m] Country-wide mean weighted by consumption.
[n] Milk supply of Geneva, June–December only.
[o] Non-systematic sampling.
[r] January–September only.
[s] July–December only.

TABLE XXIV. Cs^{137} IN TOTAL DIET

Yearly average values are given in pCi/day unless otherwise indicated

Type of study: A—Widespread survey of individual foodstuffs
B—Systematic composite diet analyses
C—Irregular sampling

Region, area or country	*Latitude*	*Year*	*Milk and dairy products*	*Meat*	*Miscellaneous*	*Total*	*Type of study*	*Reference*
NORTH AMERICA								
Greenland	60–80°N	1962	9	43	16–21[a]	68–73	A	99
United States	25–49°N							
New York City, N. Y.		1963[b]	82	2	72	156	C	165
Chicago, Ill.		1963[c]	42	23	51	116	C	165
San Francisco, Cal.		1963[d]	36	26	116	278	C	165
United States (institutional diet sampling)[m]		1961				28 (11–134)[n]	B	366
		1962				49 (11–100)[n]	B	366
		1963				140 (45–270)[n]	B	366
United States, Alaska (institutional diet sampling)	62°N	1961				46	B	366
		1962				42	B	366
		1963				140	B	366
EUROPE								
Denmark	50–60°N	1962	20	27	19	66	A	97
		1963	50	94	114	258	A	179
Faroes	60–70°N	1962	166	401	84	651	A	98
Finland, Lapland	> 68°N							
Males		1961	210	4,330	460[g]	5,000	A	226
Females		1961	140	1,440	320[h]	1,900	A	226
Sweden	~ 55–70°N	1962[e]	95	50	70	215	A	214
		1964[f]	90	75	100	265	A	277
United Kingdom	50–60°N	1961	9	11	11	31	A	280
		1962	26	40	16	82	A	280
		1963	57	70	40	162	A	280
AFRICA								
United Arab Republic (delta region)	< 30°N	1962				~ 34	C	291, 393
FAR EAST								
Japan	30–50°N	1961[i]	12[j]		53	65	C	170, 221
		1962[k]	17[j]		61	78	C	170, 221
		1963[l]	20[j]		67	87	C	170, 221
OCEANIA								
United States, Hawaii (institutional diet sampling)	21°N	1961				32	B	366
		1962				39	B	366
		1963				95	B	366
SOUTH AMERICA								
Argentina	~ 35°S	1961	11.6	20.2	8.6	~ 40	B	301

[a] Depending on the source of drinking water.
[b] November 1963.
[c] October 1963.
[d] December 1963.
[e] Late summer of 1962.
[f] January 1964.
[g] Including 420 pCi/d with fish.
[h] Including 310 pCi/d with fish.
[i] Data for Tokyo, October 1961 only.
[j] Dairy products, eggs, meat, fish and shellfish.
[k] Data for 1962 include 4 series of sampling at 4 locations in Japan.
[l] Data for 1963 include 3 series of sampling at 4 locations during the period January–July.
[m] Widespread dietary survey in 21 boarding schools in the USA, based on composite diets of children and adolescents of 6–18 years of age.
[n] Range of yearly averages reported for individual schools.

TABLE XXV. BIOLOGICAL HALF-LIFE OF CAESIUM IN ADULT MAN AS DETERMINED BY WHOLE BODY COUNTING TECHNIQUES
(*Long-term component of retention*)

Author	*Number of persons studied*	*Half-life in days (range in parentheses)*
Liden[344]	1	74
Richmond[322]	4	135 (110–147)
Rundo[320]	10	89 (58–129)
Rundo[320]	4	~119 (109–149)
McNeil[323]	3	115
Miller[324]	2	110
Miller[325]	2	88 (82, 95)
Oberhausen[326]	1	144
Rowe[327]	1	150
Van Dilla *et al.*[328]	3	~140
Taylor *et al.*[329]	4	109 (79–123)
Colard[316]	2	99
Liden[275]	8	72 (32–92)
Häsänen *et al.*[270]	6	63 (42–93)

TABLE XXVI. CAESIUM-137 IN MAN

All values are group averages in pCi Cs^{137}/gK. Figures in parentheses indicate number of measurements performed

Region, country or area	Latitude	1960 III	1960 IV	1961 I	1961 II	1961 III	1961 IV	1962 I	1962 II	1962 III	1962 IV	1963 I	1963 II	1963 III	1963 IV	1964 I	1964 II	Reference
SUBARCTIC REGIONS																		
United States, Alaska (Eskimoes)	> 65°N								136–3,200[a, y] (532)				240–4,500[a, y]					225, 368
Finland (Lapps)...	> 65°N					470–1,630[a] (140)			750–3,580[a] (89)	800–2,420[a] (40)		1,190–4,420[a] (48)					3,700–7,000[a] (39)	226, 369
Sweden (Lapps)...	> 65°N				820[b] (4) 3,025[n] (2)	980–2,140[a] (81)			1,070–2,960[a] (108)				1,380–3,120[a] (218)					226, 243, 244, 371
USSR[x].....	Far North								1,430–12,860[x, y] (20)				3,570–23,570[x, y] (60)			7,140–25,700[x, y] (30)		398
Greenland.....	> 60°N	45 (1)																311
NORTH AMERICA																		
United States[o].....	30–50°N	46 (147)	46 (164)	31 (170)	32 (75)	22 (61)	24 (80)	28 (104)			45[d] (155)							311, 312
United States, New Mexico....	~35°N	58	58	53	42	29	46	47 (74)	44 (69)	40 (163)	49 (33)	58 (72)	72 (86)	92 (80)	95[q]			220, 313
Canada.....	> 45°N		55 (1)				26 (2)											311
CENTRAL AMERICA and CARIBBEAN[c]...	10–20°N	58 (1)	58 (4)	37 (5)	20 (3)													311
EUROPE																		
Europe[e].....	40–60°N	66 (36)	65 (40)	52 (32)	49 (11)	41 (5)	36 (14)	36 (7)										311
Denmark.....	55–60°N													114 (31)	150[aa] (22)			179
Norway.....	~60°N			180[s] (15) 480[f] (6)								332[f] (11) (11) 784[g] (10) 180[s] (25)			651[v] (169) 341[s] (25) 1,404[g] (9)	353[s] (24)		222, 224, 244
Sweden.....	~60°N				55[s] (87)											183 (67)		243, 244, 372
Finland[b].....	~60°N					44 (11)			50	75 (10)		112 (49)	120 (49)	175 (49)	200 (49)	204 (24)	218 (49)	226, 369, 370
United Kingdom[bb].	50–55°N	49 (14)	42 (15)	37 (15)		36 (12)	31 (14)	28 (10)	25 (25)	35 (12)	50 (15)	54 (10)	64 (12)	89 (11)	115 (13)	135 (13)		314, 340
Federal Republic of Germany.....	47–55°N	64 (982)	57 (1,299)	45 (488)	36 (610)	33 (1,305)	31 (1,634)	28 (1,181)	32 (837)	45 (834)	54 (954)	74[i] (211)	89 (469)	104[i] (187)				311, 315
Poland.....	50–55°N												133[k] (23)		181[r] (23)			233

EUROPE (*continued*)																
Belgium.....	~50°N	51 (49)	44 (60)	38 (55)	33 (94)	——31—— (279)		——29—— (303)		——48—— (495)		——73—— (507)		——117—— (529)	143[p] (233)	316
Switzerland[t].....	~47°N												74 (5)	107 (5)	151 (5)	171[u] (5)	300
ASIA																
Asia[e].....	Not specified	32 (1)														311
FAR EAST																
Far East[e].....	Not specified	44 (27)	42 (38)	30 (28)	19 (3)	31 (2)	19 (9)	37 (3)								311
Japan.....	30–45°N			28 (12)	37 (11)		41[m,n] (21)	61[m] (38)	39[m] (62)	41[m] (57) 30 (15)	55[m] (54)	64[m] (68)	59[m] (68)	66[m] (74)	79[m] (65)	104[m] (66)	239, 241, 242, 311, 385
NEAR EAST[e].....	Not specified	33 (1)	62 (2)	49 (2)												311
AFRICA																
Country unspecified[e].....	Not specified		43 (5)	26 (1)		20 (4)										311
AUSTRALIA[e].....	Not specified				36 (1)		30 (5)	28 (24)	27 (23)	25 (10)	35 (7)	36 (24)	33 (19)	33 (22)	21 (21)	32 (29)	317

[aa] October only.
[bb] Data from Harwell, Berks., only.
[a] Average values for different groups of local population (Lapps only).
[b] Local farmers.
[c] Pooled data reported by the Walter Reed Army Institute of Research.
[d] This data for 155 persons reported from Los Angeles.
[e] Oslo residents.
[f] Bergen residents.
[g] Masfjorden residents.
[h] Helsinki residents.
[i] March only.
[j] July only.
[k] April only. Residents of Lodz.
[m] Estimated from radio-chemical analysis of muscles according to Yamagata.
[n] Reindeer breeders.
[p] January and February.
[q] October only.
[r] November only. Residents of Lodz.
[s] Stockholm residents.
[t] Residents of Geneva.
[u] January only.
[v] People from western Norway. Average value for Masfjorden was 1404 (778–2147) pCi/gK.
[x] Range of concentrations observed in individual reindeer breeders in regions of Murmansk, Magadan, districts of Nenec, Yamal-Nenec and Taimyr, and autonomous Soviet Socialist Republic of Komi.
[y] Approximate values calculated only for adults from total body burden (nanocurie) assuming 140 g of potassium in the body.
[z] 12–16-year old boys from Oslo.

TABLE XXVII. PROPORTIONALITY FACTORS RELATING Cs^{137}/K RATIO IN THE HUMAN BODY TO THE AVERAGE FALL-OUT OF Cs^{137} IN LATITUDINAL BAND 30°–60°N

P_r —current fall-out rate factor in the 12-month period ending 1 July of given year
P_{24}—factor for deposition over previous 24 months
Both factors in pCi Cs^{137}/gK per mCi/km²

Country	P_r	P_{24}
Belgium	2.25	2.42
Federal Republic of Germany	1.94	3.35
United Kingdom, Berkshire	1.54	2.90
United States, New Mexico	1.79	3.47
Average	$1.88 \simeq 1.9$	$3.04 \simeq 3.0$

TABLE XXVIII. I^{131} CONTENT OF HUMAN THYROIDS AS DETERMINED BY *post mortem in vitro* COUNTING
pCi/g fresh tissue
(Number of samples in parentheses)

Location and subject of study	*1961*			*1962*				*Reference*
	October	*November*	*December*	*September*	*October*	*November*	*December*	
New York City, foetal thyroids, prenatal age from 3 months to full term				199[a] 10.5–630.0 (4)	29.0 0–61.0 (7)	4.7 0–13.1 (4)	20.0 0–40.5 (2)	249
New York City, accident cases	~11.5[b] (20)		2.2 (26)		5.8 (82)			248, 249
New York City, adult hospital patients	2.3 (4)				1.6 ± 1.5 (7)			248, 249
New York City, children, hospital and accidental deaths					26 ± 17 (5)			249
Tokyo, hospital patients			5.6 (52)					255

[a] Data starting 20 September. Negative results from preceding period omitted.

[b] Calculated from reported data on total activity in gland, and from average weight of thyroid in adults given in the same reference.

TABLE XXIX. I^{131} IN THYROIDS
In vivo *counting technique*
(Number of subjects in parentheses)

Location and age	*Period of measurements*	*Average milk consumption in litres per day*	*I^{131} in thyroids pCi*	*Reference*
UNITED STATES				
New York City, N. Y. Adults	November 1961	0.25–1.0	57 ± 33 (6)	248
New York City, N. Y. Adults	November 1961	Very low	4.3 ± 4.9 (3)	248
New York City, N. Y. Below 18 years	November 1961	~1.0	83 ± 29 (16)	248
UNITED KINGDOM				
London, Sutton Adults	November 1961	~0.25	250[a] (20)	254
UNITED STATES				
Utah Adults	1–6 August 1962	1.3	5,900 3,700–11,000 (14)	250
Utah Adults	22–31 August 1962	1.3	3,600 700–10,900 (12)	250
Boston, Mass. 4–17 years	September 1962	Not specified	30–56[b]	260
Boston, Mass. 4–17 years	October 1962	Not specified	71–124[b]	260
Boston, Mass. 4–17 years	November 1962	Not specified	75–175[b]	260
New York City, N. Y. Adults	October 1962	0.8	300 ± 130 (11)	249
FEDERAL REPUBLIC OF GERMANY				
Adults (municipal water supply system)	September-October 1962	< 0.5	< 50 < 30–170 (16)	256
Adults (water supply from cisterns)	September 1962	~1.0	~300 < 30–700 (18)	256
Infants fed fresh cow's milk	October 1962		64 10–100 (5)	256

[a] Peak level. Integral activity in thyroids from September 1961 to January 1962 = 15 nanocurie days.

[b] Range of means reported from measurements on group of children, performed on different days of the period.

TABLE XXX. I^{131} IN MILK AND THYROID DOSES

Region, country or area	Latitude	Time integral of I^{131} concentration in milk in pCi d/l (picocurie × days/litre)			Calculated radiation doses in infants consuming 0.7 litre of fresh milk per day (millirads)			Reference
		1961	1962	1963	1961	1962	1963	
NORTH AMERICA								
Canada	~40–55°N	~6,100[a]	7,350		70	85		302, 303
United States	25–48°N	8,220[b]	10,820[b]	1,700[b]	95	130	20	164
United States, Alaska	62°N	21,500	37,800	1,800	250	440	21	164
EUROPE								
Denmark	55–60°N		3,160			36		97
Federal Republic of Germany	43–55°N	7,350	6,160[k]		85	71		256
France	42–50°N	12,250	9,630		140	110		282, 390
Ireland (northern Dublin zone)	52–55°N		6,070[c]			70		169
Italy	37–47°N	8,600[e]	9,100[d]		100	110		247, 304
Netherlands	52–54°N	9,520	5,180		110	60		274, 391, 392
Norway	58–70°N	20,000[j]			230			276
Sweden	55–70°N		8,600			100		214
United Kingdom	50–60°N	8,100	6,890		94	80		305, 306
FAR EAST								
Japan	30–50°N							
Chiba			13,300[f]			150		185, 255
Tokyo		5,000[h]	10,500[g]		58	120		185, 255
CENTRAL AMERICA								
United States, Puerto Rico	18°N	2,750	4,560	2,950	32	53	34	164
PACIFIC								
United States, Hawaii	21°N	2,130	4,410	2,220	25	51	26	164
SOUTH AMERICA								
Argentina, Buenos Aires	35°S	*	4,450			51		307
OCEANIA								
Australia	10–40°S		950[i]			11		308

* Not detected.
[a] Ontario area only.
[b] Country-wide average for children 0–2 years old weighted by population.
[c] September 17–December 17.
[d] September 1962–February 1963.
[e] October–December.
[f] September 1962–March 1963.
[g] August 1962–February 1963.
[h] Estimated for October–December 1961.
[i] May–November 1962 average weighted by population.
[j] September–November 1961.
[k] January–October 1962.

TABLE XXXI. LATITUDINAL POPULATION AND FALL-OUT DISTRIBUTION [27, 104, 124, 378]

Latitude	Area (10^6 km²)	Population (millions)	Sr^{90} deposition mCi/km²				Sr^{89} deposition mCi/km²		
			Cumulative to 1960	1961	1962	1963	1961	1962	1963
60–70°N	19	10	10	1.4	4.8	10.6	39	90	70
50–60°N	26	270	9.7	1.4	6.6	15	38	105	81
40–50°N	32	400	17	2.0	8.6	16	58	135	103
30–40°N	37	530	23	1.6	6.7	11	44	135	93
20–30°N	40	570	26	1.4	6.1	11	33	107	80
10–20°N	43	190	6.4	0.6	2.4	4.5	15	61	27
0–10°N	44	90	4.3	0.7	2.7	3.7	17	49	28
0–10°S	44	95	5.3	0.6	1.8	1.2		29	12
10–20°S	43	38	2.7	0.5	0.7	0.8		16	1.4
20–30°S	40	34	4.0	0.9	1.2	1.1		15	2.9
30–40°S	37	32	4.8	1.0	0.9	1.5		9	2.7
40–50°S	32	3	5.2	0.7	1.3	1.5		8	1.8

TABLE XXXII. ESTIMATES OF THE Z FACTORS

	Cumulative to 1960	Sr^{90} 1961	Sr^{90} 1962	Sr^{90} 1963	Sr^{90} 1961	Sr^{90} 1962	Sr^{90} 1963
NORTHERN HEMISPHERE							
Mean deposition $\overline{F}$, mCi/km²..	17.6	1.2	5.3	9.7	33	95	62
$\Sigma N_i F_i$.....	42,540	2,970	12,970	23,700	80,130	231,400	165,900
Z_N.....	1.17	1.17	1.20	1.18	1.18	1.18	1.27
SOUTHERN HEMISPHERE							
Mean deposition $\overline{F}$, mCi/km²..	4.3	0.8	1.2	1.2		16	4.4
$\Sigma N_i F_i$.....	907	137	270	233		4,180	1,380
Z_S.....	1.07	0.94	1.13	0.97		1.30	1.54
GLOBAL							
Mean deposition $\overline{F}$, mCi/km²..	11	1.0	3.4	5.6	16	58	37
$\Sigma N_i F_i$.....	43,000	3,240	13,200	24,000	80,130	274,000	167,000
Z_T.....	1.75	1.45	1.74	1.82	2.16	1.78	2.00

TABLE XXXIII. TOTAL INTEGRATED DEPOSITS OF Sr^{90} AND Cs^{137} UP TO DECEMBER 1963, AND PREDICTED FUTURE DEPOSITS
(*Megacuries*)

	0–80°N		0–50°S		50°S–80°N	
	Sr^{90}	Cs^{137}	Sr^{90}	Cs^{137}	Sr^{90}	Cs^{137}
Integrated deposit to December 1963	8.6	14.1	1.7	2.7	10.3	16.8
Expected future deposit, based upon stratospheric inventory, January 1964.....	3.0	4.5	0.9	1.3	3.9	5.8
Expected total integrated deposit....	11.6	18.6	2.6	4.0	14.2	22.6

TABLE XXXIV. MEASURED ANNUAL AIR DOSES (mrad/y) FROM FISSION PRODUCTS [851-855]

Country	1961	1962	1963
Japan			
Tokyo.....	8.2	31	23
Chiba.....		40	40
Yokosuka.....			26
United Kingdom			
Leeds.....	4	19	20
Grove.....	5	27	31
Sweden[a].....	5	17	21
Region A.....	4	12	18
Region B.....	5	17	23
Region C.....	6	16	23
Region D.....	3	24	18
Average dose in northern hemisphere weighted by population[a].....	5.2	26	23

[a] These doses are corrected for the latitudinal distribution of fall-out, using the Sr^{89} deposition data in table XXXI.

TABLE XXXV. RATIOS OF THE ANNUAL DEPOSITS OF SHORT-LIVED FISSION PRODUCTS TO THE ANNUAL DEPOSITS OF Sr^{89} DURING 1962 [27, 45, 75, 76, 91]

Collection station	$\frac{Zr^{95}}{Sr^{89}}$	$\frac{Ru^{103}}{Sr^{89}}$	$\frac{Ru^{106}}{Sr^{89}}$	$\frac{I^{131}}{Sr^{89}}$	$\frac{Ba^{140}}{Sr^{89}}$	$\frac{Ce^{141}}{Sr^{89}}$	$\frac{Ce^{144}}{Sr^{89}}$
United States							
Westwood, N. J.	1.6					0.7	1.3
Pittsburgh, Pa.	1.5		0.72			1.9	1.5
Richmond, Cal.	1.2		0.45			0.6	0.8
Houston, Tex.	1.9				1.8	1.2	1.7
United Kingdom							
Milford Haven	0.78				0.67		1.0
Chilton	1.6			1.1	1.3		1.6
Italy							
Ispra	2.1	1.4	1.4			0.84	2.7
1962 average ratio R_j	1.5	1.4	0.9	1.1	1.3	1.0	1.5

TABLE XXXVI. ESTIMATED AIR DOSE COMMITMENTS FROM SHORT-LIVED RADIO-NUCLIDES [27, 45, 75, 76, 91, 358]

Radio-nuclide	Zr^{95}	Ru^{103}	Ru^{106}	I^{131}	Ba^{140}	Ce^{141}	Ce^{144}	Dose commitments (mrad)
Gamma ray dose constant, $K_j \times B_j$ mrad/y per mCi/km²	0.30[a]	0.062	0.03	0.05	0.39[a]	0.008	0.004	
Mean life years, T_m	0.26	0.16	1.46	0.03	0.05	0.13	1.15	
$T_m \times K_j \times B_j \times G_n$	0.094	0.012	0.053	0.0019	0.024	0.0012	0.0055	
1961								
Annual deposit								
Annual Sr^{89} deposit	1.7	0.8	0.2	0.8	1.8	1.6	0.5	7.4
Dose commitment mrad	5.1	0.32	0.3	0.05	1.4	0.06	0.16	
1962								
Annual deposit								
Annual Sr^{89} deposit	1.5	1.4	0.9	1.1	1.3	1.0	1.5	27.2
Dose commitment mrad	14.4	1.7	4.9	2.1	3.2	0.1	0.8	
1963								
Annual deposit								
Annual Sr^{89} deposit	1.5	0.4	$\frac{Ru^{106}}{Ce^{144}} = 0.7$		$\frac{Ce^{144}}{Sr^{90}} = 18$			15.5
Dose commitment mrad	8.5	0.3	5.8				0.9	

[a] The gamma-ray dose constants include the dose due to daughter radio-nuclides such as Nb^{95} and La^{140}. In the case of $Zr^{95} + Nb^{95}$ transient equilibrium is assumed.

TABLE XXXVII. PROPORTIONALITY FACTORS USED FOR PREDICTING FUTURE Sr^{90}/Ca RATIOS IN TOTAL DIET

Diet type	Area	p_a (pCi/g per mCi/km²)	p_r (pCi/g for one mCi/km² per year)
I	North America Europe Oceania	0.4	1.3
II	Near East India South America Africa	0.6	2.6
III	Asia and Far East	0.7	2.0

TABLE XXXVIII. RADIO-NUCLIDES IN RESPIRATORY TRACT AND ESTIMATES OF DOSES

Author	Period of observation	Isotope	Lungs	Lymph nodes	Dose rate mrad/y	Reference
Krey *et al.*	1958–1959	Pu^{239}	$1.3–11.9 \times 10^{-3}$ dpm/g tissue	$11–65 \times 10^{-3}$ dpm/g tissue	Lungs: 0.06–0.5 Lymph nodes: 1.1–6.4	346
Osborne *et al.*	Spring 1962	Pu^{239}	0.16 pCi/person		0.016	347
		$Zr^{95} + Nb^{95}$	250 pCi/person		2.4	
Rundo	January 1962	$Zr^{95} + Nb^{95}$	300 pCi/person		2.9	348
Schonfeld (quoted)	February 1962	$Zr^{95} + Nb^{95}$	200 pCi/person		2.0	
Liebscher *et al.*	1960	$Ce^{144} + Pr^{144}$	0.03 pCi/g tissue	0.7 pCi/g tissue	Lungs: 0.7 Lymph nodes: 17	349
Wegst *et al.*	March 1963	Ce^{141} $Ce^{144} + Pr^{144}$ Ru^{103}	156 pCi[a]		1–7	350
		$Ru^{106} + Rh^{106}$	106 pCi		0.5–3	
		$Zr^{95} + Nb^{95}$	173 pCi		3.4	

[a] In one lung cleared of bronchial lymph nodes.

TABLE XXXIX. DOSE COMMITMENTS FROM NUCLEAR EXPLOSIONS [a]

Tissue	Source of radiation	Dose commitments (mrad): For period of testing 1954–1960 (estimates from 1962 report)	Dose commitments (mrad): For period of testing 1954–1962 (new estimates)	Paragraph
Gonads	External, short-lived[b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Cs^{137}[b]	8	13	179
	C^{14}	5[c]	13[c]	187
	TOTAL	40	76	
Cells lining bone surfaces	External, short-lived[b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Sr^{90}	67	174	173
	Cs^{137}[b]	14	13	179
	C^{14}	8[c]	20[c]	187
	Sr^{89}	0.15	0.30	176
	TOTAL	116	257	
Bone marrow	External, short-lived[b]	11	21	163
	Cs^{137}	16	29	165
	Internal, Sr^{90}	33	87	174
	Cs^{137}[b]	10	13	179
	C^{14}	5[c]	13[c]	187
	Sr^{89}	0.07	0.15	176
	TOTAL	75	163	

[a] In the 1962 report, these doses were reported in mrems. As explained in paragraph 191, the doses in the present report are all given in mrads.

[b] The dose commitments from short-lived nuclides and from internal Cs^{137} have been calculated on a slightly different basis in this report (paragraphs 162, 178) as compared to the 1962 report.

[c] For C^{14} it seems to be appropriate to include only the dose which is accumulated up to year 2000, at which time the doses from the other nuclides will have essentially been delivered in full. The *total* dose commitments from C^{14} from tests up to 1960 for the gonads, cells lining bone surfaces and bone marrow are 48, 80 and 48 mrads, respectively. For all tests up to the end of 1962, the dose commitments from C^{14} are 180, 290 and 180 mrads, respectively.

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ANNEX B

RADIATION CARCINOGENESIS IN MAN

CONTENTS

I. Introduction

1. The purpose of this survey is to review data on the risk of induction of cancer by ionizing radiations in man, with emphasis on information either new or not discussed in detail in the 1962 report of the Committee[a] to the General Assembly.[1,b]

2. Most of the information available on tumour induction by radiation in man and experimental animals comes from studies of the effects of high doses of radiation, that is, doses of hundreds of rads and greater. From these data it is known that ionizing radiation in high doses may cause or contribute to the induction of cancer in widely diverse types of mammalian tissues, though the susceptibility of the different tissues varies greatly with genetic and physiological factors.

3. Few data are available at low doses of radiation (doses of the order of 10 rads and less), and the extent to which radiation has a general carcinogenic effect at low doses is a matter of speculation.

4. The mechanisms of carcinogenesis in general are not well understood. However, the evidence is that the neoplastic change occurs at the cellular level and is frequently associated with observable modifications in cell structure (particularly chromosomal constitution) and function. A variety of carcinogens, including chemicals and viruses as well as ionizing radiations, produce structural and functional changes which appear to be similar in nature.

5. Among the mechanisms likely to be of importance, which include systemic as well as local factors, are (*a*) direct cellular injuries, including changes in genes and chromosomes; (*b*) promotion of growth and development of cells with malignant potential through local injurious effects on related cellular systems and stroma; (*c*) complex perturbations of cellular and tissue homeostasis; (*d*) systemic influences and effects on distant tissues and cells which may result in altered immunological mechanisms, endocrine disturbances, and changes in metabolism and nutrition.

6. Radio-biological investigations suggest that relatively large doses of radiation are usually required to cause severe systemic effects or produce observable changes in tissue structure and function. Such high-dose effects may play a major role in the production of the neoplasms that are superimposed on chronic radiation-induced tissue changes. However, in so far as cancer may result from low doses of ionizing radiation, the major mechanisms would presumably be more consistent with the production of malignancy as a result of mutations, of chromosomal injuries or possibly of changes enhancing the susceptibility of cells to transformation by viruses.

7. Radiation-induced tumours are indistinguishable in general from cancers arising from other causes. Furthermore, as noted above, there may be common basic mechanisms. These two considerations affect both the type of statistical data that can be obtained in clinical

[a] *Official Records of the General Assembly, Seventeenth Session, Supplement No. 16* (A/5216); hereinafter referred to as the "1962 report".

[b] Superscripts refer to the corresponding entries in the bibliography at the end of the present annex.

and experimental studies and the interpretation of such data.

8. When similar basic mechanisms are involved in the production of radiation-induced and other cancers, the effect of radiation may be either (*a*) to advance in time the control curve of age distribution of tumours or (*b*) to multiply by some factor the age-dependent incidence. When similar mechanisms are not involved, the effect of radiation may be to produce an additional incidence unrelated to the control age incidence. The yield of tumours, in any given interval after the exposure, will be determined by which of these processes or by what combination of them operates and by possible changes in survival time occasioned by the irradiation.

9. Most of the data available in man, and even in experimental animals, are confined to a limited period of time following irradiation. Any attempt to estimate the total lifetime incidence of tumours is likely to involve extrapolation.

10. In making risk estimates for human populations, the difficulties of epidemiological studies must be borne in mind. There is the possibility of large variation in susceptibility in the population because of differences in genetic, physiological and environmental factors. Thus the dose-effect relationship observed in a study of a whole population does not necessarily apply to individuals or to subgroups of the population. When isolated risk estimates are derived from irradiated subjects highly selected for one reason or another and are applied to the general population, this should be done with great caution.

11. In many instances it has been necessary to perform dose determinations long after the irradiation, and since it may be impossible to reproduce the conditions existing at the time of the original irradiation, many of the dose estimates necessarily have a wide margin of uncertainty.

12. In addition, there are problems of terminology and statement of dose. When the body is irradiated non-uniformly (because of limited field size, limited penetration of the radiation, or non-uniform distribution of radioactive nuclides within the body) the irradiation cannot be unambiguously specified by a single number but only in terms of dose distribution. To provide a basis for a particular risk estimate, one must, however, select a single value of a quantity that characterizes the irradiation, even though such a value may be of limited accuracy or significance.

13. The physical quantities most commonly determined for external irradiation are either kerma or exposure in free air,[2] since measurements are frequently performed with devices of minimal mass and without the use of phantoms. Because of the complexity of the physical interactions, there may be a considerable difference between the quantities thus determined and the absorbed dose in the tissues of interest.

14. All empirical risk estimates are valid only for the conditions of irradiation under which they were obtained. Thus, a risk estimate valid for a certain dose delivered in a single exposure probably is not valid for the same dose delivered over a long period of time.

15. For estimation of risk as a function of dose one requires ideally a dose-effect curve established over a wide range of doses. However, in man, information is scanty and when available refers only to a limited dose range. Within this range and within the limitations described above estimates of risk can be made. Outside this dose range it is necessary to make assumptions as to the nature of the underlying dose-effect relationship.

16. One such assumption relates to "threshold dose". For certain types of radiation-induced cancer there may be a threshold dose, but this can be established only from an understanding of the mechanisms. Even statistical studies of cancer incidence in large irradiated populations cannot unequivocally establish the existence of a threshold. In general, therefore, for the estimation of risk at low doses it has to be accepted that there is a finite risk of cancer induction, however small, even at the lowest doses.

17. In most cases in which extrapolation to low doses has been attempted a linear relationship between dose and effect has been assumed. A discussion of the use of the linear hypothesis can be found in the 1962 report. It should be noted that the assumption of linearity is the only one which allows the use of mean doses in estimating risks. In general, the assumption of a linear dose-incidence relationship at low-dose levels is likely to result in an over-estimate of the degree of risk.

18. This is, in general, likely to be true because most known dose-response curves tend to be sigmoid or linear at their lower ends, and because, on theoretical grounds, single-event changes may be expected to predominate at very low doses. Assurance that linear extrapolations are likely to over-estimate the risk is greatest when observation includes the region of rapidly increasing response with increasing dose.

19. An estimate based on the relative increases over the "natural" incidence will be influenced to a large extent by the natural incidence, which in some cases (for example, thyroid or bone cancer) is very low. Absolute risk estimates are a measure of the susceptibilities of different tissues to the induction of cancer by radiation.

20. In this report risk estimates of cancer induction will be presented either in relative terms, as increase relative to natural incidence, or in absolute terms, expressed as the number of cases per unit time and unit dose in a population of given size, for example as number $\times 10^{-6}$ per year per rad. It must be emphasized that these estimates of risk are reliable only in the range of doses, usually high, for which information is available. The use of the estimate at doses outside the observed range may be very much in error, and in the low dose range where a linear extrapolation to zero dose is used, it can in most cases only be taken as an indication of the upper limit of risk.

II. Leukaemia

Leukaemia in Japanese A-bomb survivors

21. The most informative data that are available on the relationship between radiation exposure and leukaemia in man are those obtained from the studies of the survivors of the atomic bombing in Hiroshima and Nagasaki. Even in these studies there are great uncertainties in dosimetry, in the size and characteristics of exposed populations, and in the leukaemia incidence data. Furthermore, the incidence is being observed in a population of survivors, therefore of individuals heavily selected at least at the high dose levels.

22. Some studies have been done on an "open population" in which there may be little accounting for migra-

tion of persons both non-exposed and exposed. To attempt to circumvent the uncertainties that arise in the follow-up of such an "open population", the Atomic Bomb Casualty Commission (ABCC) created the Master Sample, a "closed population" matched by age and sex in segments of the population proximally exposed (0-2,000 metres from hypocentre), distally exposed (2,000-10,000 metres), and non-exposed (beyond 10,000 metres).

23. Watanabe[6] has reported the incidence of leukaemia among Hiroshima survivors ("open population"), including all leukaemia cases for the seventeen years 1946-1962. Recomputations of these data to obtain the leukaemia incidence in the total population, in the non-exposed population (beyond 5,000 metres, presumably including post-detonation entrants), and in the exposed population (within 5,000 metres) are given in table I and figure 1. Estimates of relative risk are given in table II.

24. In table I and figure 1 it can be seen that there was a sharp increase in the reported leukaemia incidence in the exposed (within 5,000 metres) after 1946, reaching a peak eleven times higher than in the non-exposed (beyond 5,000 metres) in 1951. From 1952 to 1959 the incidence in the exposed fluctuated below this peak; from 1960 to 1962 it fluctuated within a still lower range.

25. Brill *et al.*[7] have summarized and compared previously reported findings on leukaemia in atomic bomb survivors in Hiroshima and Nagasaki, as obtained from the ABCC Master Sample or "closed population", up to 1958. Table III presents incidence by distance from the hypocentre for the 89 confirmed leukaemia cases in Hiroshima and the 60 confirmed leukaemia cases in Nagasaki during the twelve-year period 1947-1958.

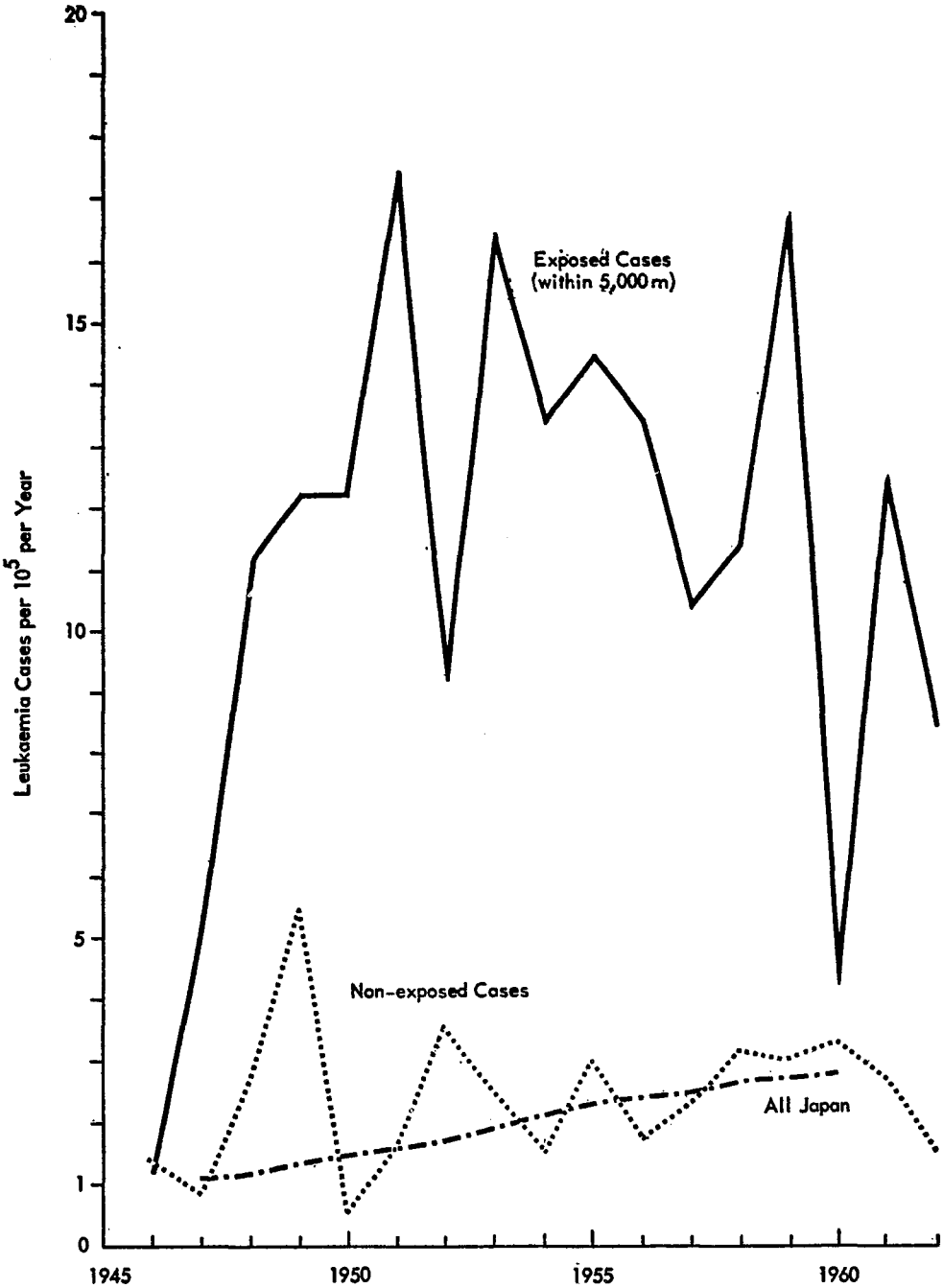


Figure 1. Leukaemia cases in Hiroshima, 1946-1962 (modified from Watanabe[6])

26. The incidence of leukaemia was greatly increased among survivors exposed within 1,500 metres from the hypocentre. Between 2,000 and 10,000 metres the average yearly incidence in Hiroshima ($28/10^6$/y) was regarded by Brill *et al.* as not significantly greater than that expected in Japan where leukaemia rates are reported to be $20\text{-}30/10^6$/y. Nor is the incidence significantly different from the average yearly incidence in the non-exposed Hiroshima population during the same period of time, as deduced from Watanabe's figures. The elevated leukaemia incidence ($37/10^6$/y) in those exposed between 2,000 and 10,000 metres in Nagasaki (table III) is significantly ($P < 0.05$) higher than expected on the basis of the frequency obtaining in the Japanese population.

27. Brill *et al.*[7] also analysed relations between radiation dose corrected for shielding (among those persons for whom knowledge permitted individual dose corrections) and average incidence of confirmed leukaemia during the nine-year period 1950-1958 in the Master Sample in Hiroshima and Nagasaki. Figures 2 and 3 provide a general guide to neutron and gamma radiation doses according to distance from hypocentre in Hiroshima and Nagasaki. Figures for attenuation of dose by shielding were based on the average attenuation factors observed during test explosions in the Nevada Desert.[8] For individuals who had been located in the open no attenuation factor for shielding was applied. For those partly shielded a 15 per cent attenuation of the air dose was assumed for gamma radiation and 25 per cent for neutrons. For persons inside houses of light Japanese style construction an attenuation of 30 per cent of the air dose was assumed for gamma radiation and of 50 per cent for neutrons. Persons located in other shielding categories were not taken into account because the corresponding attenuation factors were not known. This resulted in further reduction of the sample and of the number of leukaemia cases. Gamma and neutron estimates were added in a 1:1 ratio, assuming an RBE of 1 for neutrons. If the RBE were in actual fact higher than 1, then using a 1:1 ratio, as is done here, would yield a higher estimate of risk of leukaemia per unit dose, at least in Hiroshima.

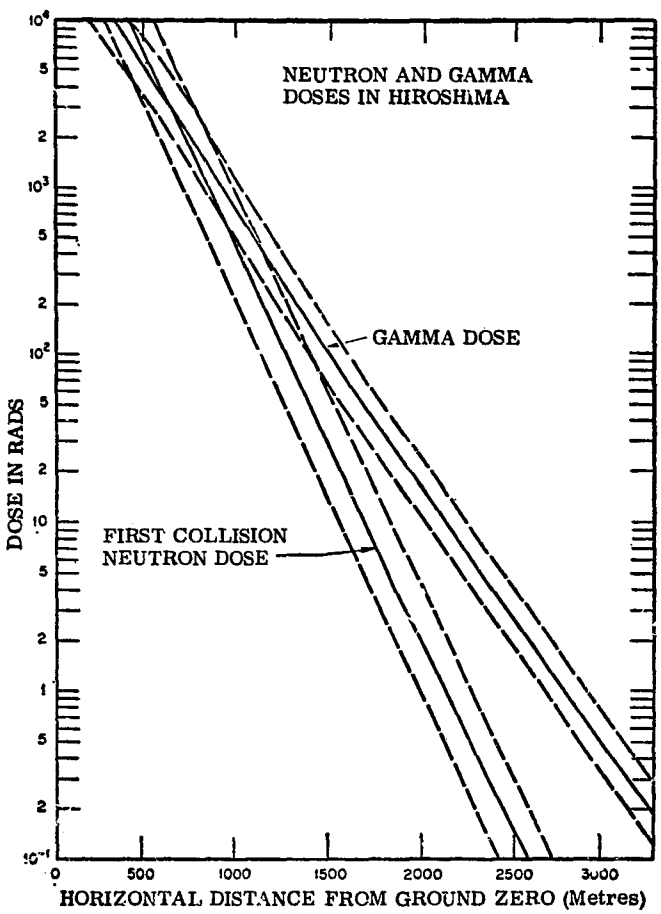


Figure 2. Estimates of neutron and gamma air doses in Hiroshima as a function of horizontal distance from ground zero (modified from York[3])

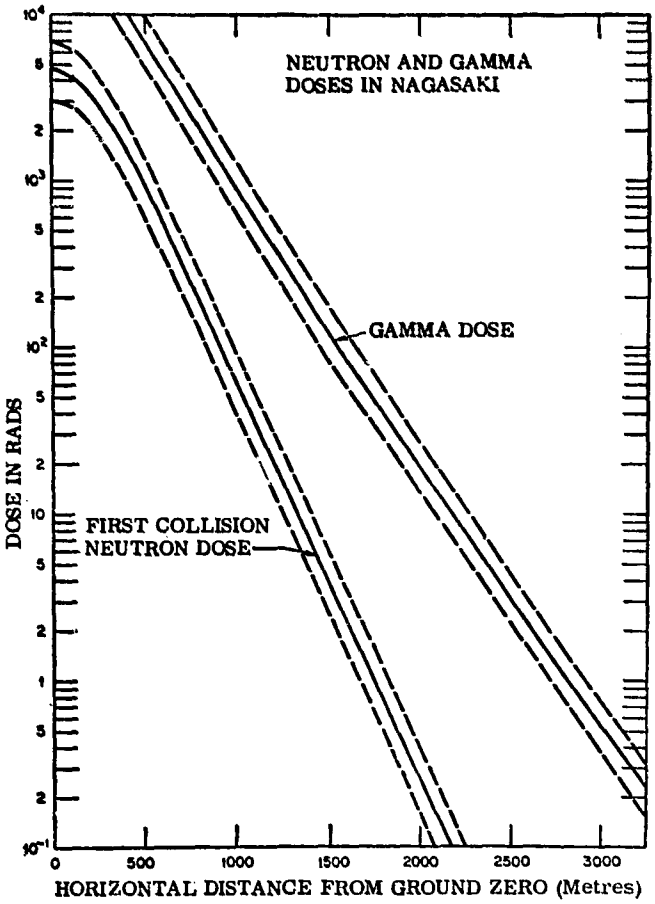


Figure 3. Estimates of neutron and gamma air doses in Nagasaki as a function of horizontal distance from ground zero (modified from York[3])

28. Table IV presents data on confirmed leukaemia incidence according to absorbed dose as obtained by these procedures for 51 cases in Hiroshima and 25 cases in Nagasaki during the nine-year period 1950-1958. The yearly leukaemia incidence according to dose is shown graphically in figure 4.

29. For Hiroshima the relation of dose to annual leukaemia incidence in the nine-year period 1950-1958 can be described by a straight line over the dose range of about 100-900 rads. Between 10 and 100 rads the incidences in each dose group, though consistent with the same straight line, do not differ significantly from one another.

30. The data indicate that at least in the range between 100 and 900 rads the average rate of increase of the incidence with dose was 1.1 cases/10^6/y/rad at Hiroshima and 1.6 cases/10^6/y/rad at Nagasaki, or between 1 and 2 cases/10^6/y/rad in both cities. Since in Nagasaki the exposed population was smaller and the cases of leukaemia fewer, the estimate for that city is statistically less reliable. However, the major cause of uncer-

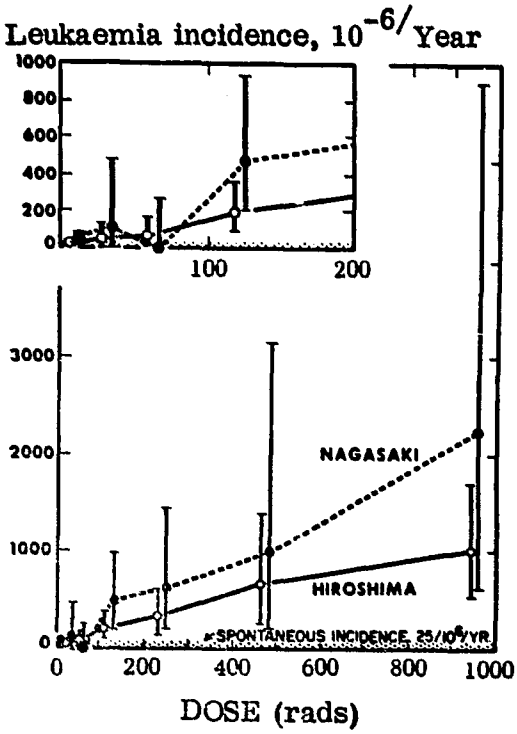


Figure 4. Average incidence of confirmed leukaemia in the master sample, proper plus reserve, 1950-1958, light shielding, by relative radiation dose (modified from Brill *et al.*[7])

tainty affecting both estimates probably lies in the limitations of the dosimetry rather than in the inherent variability of the data.

31. In both Hiroshima and Nagasaki taken together there is moderate correlation between year of onset of leukaemia and distance from the hypocentre. In both cities the group with symptoms of radiation sickness was closer to the hypocentre and had a significantly increased leukaemia incidence over the group without radiation symptoms.

32. The predilection of the various types of leukaemia for specific age groups does not appear to have been markedly altered as a result of irradiation. The rarity of chronic lymphocytic leukaemia in Japan was confirmed. The types of leukaemia observed to be increased most in survivors under 1,500 metres were chronic granulocytic and acute leukaemia. The acute leukaemias occurred predominantly in people under ten years of age at the time of exposure. There is normally a strong predilection for this type in the young. In the Japanese survivors, although chronic granulocytic leukaemia has occurred predominantly in the middle age groups, it has also been seen with increased frequency in children. Acute leukaemias—including granulocytic, myelomonocytic and unclassified types—also have occurred with increased frequency.

33. The incidence of leukaemia of various haematologic types has varied systematically in relation to age at the time of irradiation,[7] so that the age-incidence curves for the irradiated and control populations are nearly parallel.[135] Because the natural incidence of certain types varies with age by as much as an order of magnitude or more, the numbers of cases of induced leukaemia of these types have varied correspondingly with age at the time of irradiation. A given amount of radiation may thus be inferred to have increased the probability of the disease by a given percentage of the natural age-adjusted rate rather than by a given number of additional cases, a distinction of practical as well as theoretical importance.[135]

Leukaemia in early post-detonation entrants in Hiroshima

34. The data from the report of Watanabe[6] on leukaemia cases developing between 1950 and 1962 (thirteen years) in persons entering Hiroshima soon after the detonation have been recomputed and presented in table V. Although the incidence was higher than expected from data on non-exposed persons, the difference between the earliest entrants and later entrants was not statistically significant.

35. Estimates of radiation exposure from fall-out and from neutron-induced radio-activity have been published[4,5] (figure 5), but their reliability cannot be assessed.

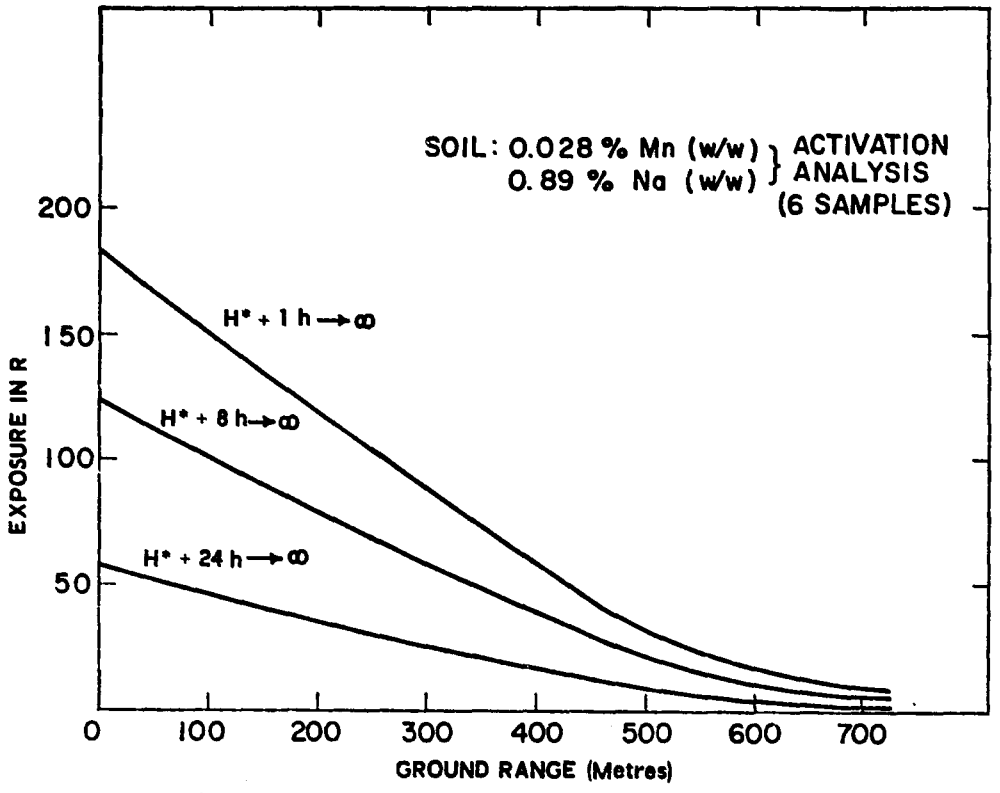
36. The locations of the early post-detonation entrants during the first days after entry are not known. In view of the extensive destruction and the long-lasting fire storm near the hypocentre which broke out within a few hours of the explosion, it has been considered probable that very few persons might have been close to the hypocentre for substantial periods of time during the first day after detonation.

Leukaemia in American radiologists

37. Earlier studies, already discussed in the 1962 report, are summarized in table VI. Since the 1962 report Lewis[9] has reviewed the 425 death certificates of registered specialists in radiology dying between the ages of thirty-five and seventy-four during the fourteen-year period 1948-1961. In table VII the observed numbers of deaths for which the main cause was leukaemia or a related disease are compared with the corresponding expected numbers calculated by applying to the number of living radiologists at risk the death rates (standardized with respect to sex, race, age and year of death) due to each relevant cause. The estimated number of male radiologists, thirty-five to seventy-four years of age, increased from 2,167 in 1948 to 4,713 in 1961; in the fourteen-year period the number of man years at risk at these ages was 47,348. The mortality ratio for leukaemia was 3.0, with 12 cases observed among the radiologists as compared with 4.02 cases expected. The average annual incidence of death from leukaemia in radiologists during these fourteen years was $253/10^6$/y, as compared with an expected incidence of $85/10^6$/y, giving an excess incidence of $168/10^6$/y. About 4.4 of the twelve leukaemia deaths in the group of radiologists were expected to be of the lymphocytic type (predominantly chronic), but only 1 case of lymphocytic leukaemia was observed, and it was of the acute type. Therefore, all 12 cases of leukaemia in the radiologists were of types known to be increased by irradiation.

38. Based on the risk estimates from Hiroshima, the excess leukaemia incidence observed in the radiologists would result from a single acute whole body exposure of about 100 rads. Little is known about the magnitude and distribution of the dose received by the early radiologists, but the evidence available indicates that doses far in excess of 100 rads were received chronically over periods of up to forty years, and this would suggest that long-term radiation exposure is less effective than short-term exposure in inducing leukaemia.

39. For Hodgkin's disease, lymphosarcoma, and lymphoblastoma, the observed numbers of deaths among



*H = Time of detonation.

Figure 5. Neutron-induced ground activity—Hiroshima (modified from Borg and Conard[6])

radiologists were in reasonable agreement with the expected numbers (table VII). On the other hand, in the population at risk there were five deaths from multiple myeloma where one was expected, and four deaths from aplastic anaemia where 0.2 was expected. While the increased incidence of aplastic anaemia was not surprising, the association of multiple myeloma with radiation had not been substantiated before.

Leukaemia in ankylosing spondylitis patients

40. There have been no new data on leukaemia incidence in relation to dose in patients X-irradiated therapeutically for presumed ankylosing spondylitis since Court Brown and Doll[12,13] in England studied the case records of 13,352 patients treated in 81 treatment centres from 1 January 1935 to 31 December 1954. Only cases of leukaemia found before 1955 were considered in the analysis. This study was discussed in the 1962 report. However, for purposes of comparison with studies of dose-incidence relations in other irradiated populations, it is discussed here also in somewhat modified form.

41. In the evaluation of the dose-incidence relationship, the cases used included 32 with an established diagnosis ("A" series) and 5 probable cases ("B" series). Since only 1 certain case of leukaemia was recognized among the 2,065 irradiated women (total incidence $48/10^5$), the analysis was confined to men, and the population at risk was defined as the 11,287 males in the study series.

42. The patients had been subjected to total cumulative exposures ranging from 112 R to more than 3,000 R in from one course of fractionated exposures (over about a month) to eight courses of therapy with separations by as many as eight years. The majority of patients given only one course had been treated since 1950, and most of the patients given multiple courses had been followed for a longer period. Young men were given, on the average, more courses of treatment than older men. The patients included boys of fourteen years of age to men well over fifty-five years of age.

43. In over 90 per cent of the patients the radiation fields were confined to limited areas of the body which included the affected joints. Fields with generous margins were used over the spine, sometimes including the pelvis and other joint areas. Details of X-ray exposure were obtained from a sample of approximately one in every six of the patients (1,878 men). Both exposures to spinal bone marrow (roentgens) and whole body integral exposures (megagramme-roentgens) were estimated. The spinal exposure was estimated in two different forms: as mean exposure to the marrow throughout the whole length of the spine, and as maximum exposure at a point in the spinal marrow. Finally, the mean spinal marrow exposure was estimated separately for those patients receiving only spinal irradiation. Eighteen of the 37 leukaemia cases were patients receiving only spinal irradiation.

44. Since the patients did not receive all their radiation treatment on one occasion, and since the proportions of patients given various exposures did not remain constant throughout the period under review, it is not possible to measure the incidence of leukaemia in relation to the size of the exposure simply by relating the numbers of patients with leukaemia following a given exposure to the estimated total numbers of patients given the same exposure. Court Brown and Doll met this difficulty by estimating the population at risk for each level of exposure from the sum of the number of years the patients survived after receiving an individual exposure and before receiving additional radiation treatment which would place them in the next, arbitrarily defined, exposure category.

45. The accuracy of this method depends much on the limits of latent period for the radiation-induced leukaemia. On the basis of 10 cases receiving only one course of treatment it appeared that the latent period would be seldom less than two years, more frequently of the order of three to five years, and less commonly, longer. However, in their analysis the authors postulated that leukaemia was equally likely to appear at any time after irradiation with the exception of the first year. Since many patients were treated during the last few years of the study, the study may be incomplete with respect to recording the total incidence of leukaemia.

46. The incidence of leukaemia in irradiated spondylitics increased with age from $11/10^6$/y in those less than twenty-five years of age to $56/10^6$/y in those fifty-five years of age or older, consistent with the age-dependent increase in incidence of comparable forms of leukaemia in the general population of England and Wales.[136] The excess of leukaemia, which was of the order of a tenfold increase in the "natural" age-adjusted rate, was present irrespective of the age at which radio-therapy was started but was slightly greater when treatment was begun late in life. For a given exposure, therefore, at least five times as many cases of leukaemia were induced in the elderly group as in the young group, although the natural age-dependent rate was increased by a similar, or only slightly different, percentage in both groups. The greater increase in the elderly cannot be ascribed to differences in exposure because there was a tendency to give older patients less radiation treatment rather than more.

47. A significant excess of deaths from all types of leukaemia occurred in the irradiated spondylitics, as compared with the expected numbers in a non-spondylitic, non-irradiated population, with ratios of 4.6 for lymphocytic, 6.1 for myelocytic, 11.8 for monocytic, and 52.4 for other and unspecified leukaemia.

48. Since adequate data are not available on the incidence of leukaemia in male spondylitics not treated by irradiation nor in non-spondylitics receiving spinal irradiation, the expected rates of leukaemia used in this study were those for the general male population of Britain standardized for age.

49. A certain possibility of error exists in the use of this control population in view of the fact that an association between leukaemia and rheumatic diseases has been observed.[15] Also, the existence of hereditary factors in ankylosing spondylitis and rheumatoid arthritis has been reported.[14] Another cause of concern is the possibility that some of the other forms of therapy used for ankylosing spondylitis may also be leukaemogenic. Agents such as antipyrine, gold salts, and phenylbutazone have been known to depress the bone marrow activity.[16] Bean[17] has reported the development of leukaemia in six non-irradiated patients who had received phenylbutazone. Three of these had ankylosing spondylitis.

50. In the survey of Court Brown and Doll[13] the case records did not provide enough detail for a special study of the use of drugs. These authors pointed out that butazolidine could hardly have played any part in the development of the cases of aplastic anaemia found, as it was not in general use until 1952. The authors indicated that gold had been used for many years, as had many types of analgesics, including amidopyrine. That drugs were largely responsible for the development of leukaemia did not seem reasonable to the authors, but decisive data were not provided in their report.

51. The numbers of men receiving various exposures of radiation to spine alone or spine plus extraspinal regions, given in terms of mean exposures to spinal marrow, and the numbers of man years at risk at twelve levels of estimated exposures, are given in table VIII. The exposures are the total exposures received less those received in the twelve months preceding the diagnosis of leukaemia. The data on the leukaemia patients are summarized in table IX, along with the crude and standardized incidence rates.

52. The annual leukaemia incidence increased from $0.5/10^6$/y in the standard control population to $72/10^6$/y following an exposure of 2,250 R or more. Below 500 R 2 of the 4 cases of leukaemia were lymphatic.

53. The mean exposure received by the spinal marrow was believed by Court Brown and Doll to be the most satisfactory measure of radiation exposure under the conditions of the investigations. However, they pointed out that it failed to take into account the considerable exposure to the extraspinal marrow in some of the patients. Therefore, they considered separately the patients who received treatments to the spine and sacro-iliac regions only. These data are given in table X.

54. Patients whose treatment was initially confined to the spine and sacro-iliac joints but who subsequently received extraspinal (including wide field) irradiation were included in the group only for that part of their life between initial treatment and the first extraspinal treatment. Although this improved the accuracy of dose estimates, the data became limited in value because of the consequent reduction in the number of leukaemia cases.

55. A regression line fitted through the incidences observed at exposures in the range between approximately 300 and 1,500 R to the spine, has a slope of $0.5/10^6$/y/R. Extrapolation of the line below 300 R is not warranted. It will be recalled that the straight line describing the relation between dose and incidence of leukaemia in the Hiroshima population of survivors to the A-bomb explosion has a slope between 1 and $2/10^6$/y/rad in the range from 100 to 900 rads. The characteristics of the irradiated population and the conditions of irradiation in Hiroshima were very different from those obtaining in the spondylitics. Not only had the Hiroshima survivors received an instantaneous burst of radiation while the spondylitics had received several courses of treatment, but most of the Hiroshima survivors had presumably been exposed to whole body irradiation, whereas in the spondylitics only parts of the body (probably involving one-third to one-half of the bone marrow) were irradiated. The similarity of the two slopes suggests that a common risk estimate may apply to both populations.

Leukaemia in children irradiated therapeutically for "enlarged thymus"

56. There have been a number of epidemiologic studies of the incidence of leukaemia in children who received therapeutic X-irradiation during childhood for benign conditions.[18-26] These studies have been discussed in the 1962 report. Among these studies, those by Hempelmann and his colleagues are the largest and the only ones which have shown an increased incidence of leukaemia.

57. The most recent reports on the Hempelmann survey are those by Pifer *et al.*[27] and by Toyooka *et al.*[28,29] The numbers of leukaemia cases observed, of cases expected in infants treated with X-rays for "thymic enlargement", and of sibling controls in both Series (I and II), are given in table XI. All children were con-

sidered to have been at risk from birth until 31 December 1960, or until death.

58. In Series I most of the children were treated between 1926 and 1946, and treatment techniques and reasons for treatment varied greatly. In Series II the children were treated between 1940 and 1957, and treatment methods were more uniform. Many of the children irradiated in the earlier years were given relatively large doses to large areas, and posterior as well as anterior positioning of ports was often involved. The children more recently irradiated were usually given smaller doses to relatively small areas, with anterior positioning of ports. In Series II the X-ray energies, the numbers of treatments and the total period of treatment were usually less than in Series I.

59. The 6 cases of leukaemia observed in Series I included four lymphocytic and two acute undifferentiated types. The data do not make it possible to study the regression of incidence with dose.

60. These data are inadequate to distinguish between the influence of (*a*) the irradiation and (*b*) the condition, so-called "thymic enlargement", or the underlying basis for the diagnosis which served as the indication for therapy. Conti *et al.*[22] reported no cases of leukaemia in a group of 1,564 children and 96 per cent with no evidence at treatment of the condition called "thymic enlargement". Eighty-eight per cent of the children were treated through relatively small ports with exposures of 75 to 300 R, averaging 150 R, to the thymic region, and 12 per cent of them received 200 to 450 R. One case was expected. These children (90 per cent) were studied from eleven to eighteen years after the therapy.

61. Murray *et al.*[21] found 2 cases of leukaemia in seventy-five children treated for pertussis, 1 case in 1,073 persons treated to the head and neck mainly for lymphoid hyperplasia of the nasopharynx, and no leukaemia deaths in 2,460 children treated with superficial X-rays for benign skin lesions.

Leukaemia in children irradiated *in utero*

62. Several retrospective studies of children irradiated *in utero* have been carried out and their results are summarized in table XII.

63. In the retrospective study by Stewart *et al.*[30-32] of the incidence (mothers' memory) of pre-natal abdominal diagnostic irradiation among mothers of 780 children dying under ten years of age of leukaemia (during the years 1953-1955) and mothers of 1,638 control live children matched for age, sex, and location, there was a relative risk for leukaemia of about 1.83. Excessive maternal age was also related to an increased risk of leukaemia and an increased incidence of Down's syndrome. The incidence of *post-natal* diagnostic or therapeutic exposure was almost twice as high in the leukaemic children as in the control group for the first three years of life, and the frequency of *post-natal* acute pulmonary infections and severe injuries was also significantly increased in children who subsequently died of leukaemia.

64. The controls in these retrospective studies varied considerably. Stewart *et al.*[32] used live children matched for age, sex and location. Ford *et al.*[33] used children dying under ten years of age of causes other than cancer during the same period of time as the treated children. Here, the information about radiation exposure during the last trimester of pregnancy was obtained from obstetricians and hospital records. Kaplan[34] employed two control groups—closest siblings and most habitual playmates. Polhemus and Koch[35] used children attending the same hospital during the same period (consecutive surgical admissions) matched for age, socio-economic and geographic factors. In Kjeldsberg's study[36] the controls were healthy children born at a time different from that of the leukaemic children and therefore not experiencing the same likelihood of antenatal X-ray examination. In the study by Murray *et al.*[21] there were three control groups—children dying of non-malignant disease during the same period, siblings, and live siblings of the deceased controls. Finally, Lewis[37] used as controls all of the children born at the hospital in which the leukaemic children had died.

65. Court Brown *et al.*,[38] in an extensive prospective study of women who were irradiated during pregnancy between 1945 and 1956, found that among 39,166 live-born children who had received antenatal irradiation, nine had died of leukaemia before the end of 1958. The expected number was 10.5. None of the children of the 750 women irradiated in the first three months of pregnancy had developed leukaemia during an average follow-up of over six years. No correlation was found between incidence of leukaemia and the amount of antenatal exposure.

66. MacMahon[39,40] designed an extensive prospective study to examine this problem by a method which utilized objective evidence of intra-uterine X-ray irradiation. The study population consisted of 734,243 children born in, and discharged alive from, any of thirty-seven large maternity hospitals in the north-east United States in the years 1947-1954. The frequency of intra-uterine X-ray irradiation in the population was estimated by review of the records of a 1 per cent systematic sample. Abdominal or pelvic X-rays were recorded in 370 (10.6 per cent) of the 7,242 single pregnancies in the sample. Children dying of leukaemia before the end of 1960 were identified by review of death and birth certificates. After correction for indirect associations with birth order and other complicating variables, it was estimated that leukaemia mortality was about 40 per cent higher in the X-rayed than in the non-X-rayed members of the study population, giving a relative risk of 1.4, with 95 per cent confidence limits 1.0 and 2.0.

67. No estimates of dose were obtained in any of these studies. However, in MacMahon's study, X-ray cases were divided into three categories ranked in order of probable (relative) dose, according to the probable numbers of films involved in the diagnostic procedures. Although there was a minor trend toward higher cancer risk in the more heavily exposed categories, it was far from significant. It was shown that neither the higher risk of leukaemia in first births nor the higher frequency of pre-natal X-ray in first births explained the association between X-ray irradiation and increased leukaemia incidence. It remains to be demonstrated whether or not the association between irradiation and leukaemia explains the high frequency of leukaemia in first births. The ratio of mortality in first-born to that in later-born was as great in the separate X-ray categories (1.5 in the X-rayed, 1.3 in the non-X-rayed) as in the combined data, 1.4. The birth-order differential was also as great among children in specific relative dose categories (pelvimetry) as in the total.

68. The report[39] which included deaths to the end of 1960 of children born during the years 1947-1954, dealt with children predominantly over five years of age and showed a higher frequency of intra-uterine X-irradiation in cases of leukaemia and cancer deaths, as compared

with the 1 per cent sample of the population. It is of interest that an earlier report,[40] which included only deaths prior to 1958, showed little or no difference in frequency of intra-uterine irradiation between the two groups. It is possible that differences in young children have been similarly under-estimated in some other studies as a result of insufficient follow-up time for a significant portion of the population. The small relative risk and the occurrence of the peak risk after five years of age are two characteristics of the association which may go far towards reconciling this view with the negative results reported in a number of surveys.

69. Wise[41] has studied first-born children who had died of leukaemia during the period 1953-1955. In a group of 306 such cases ten birth cohorts were represented (1945-1954), and in nine of these the mean age at death of cases with a history of pre-natal irradiation was four months greater than the mean age at death of the non-X-rayed cases. Only two cohorts had been followed from birth to one year of age (1953-1954), and only one had been followed from birth to two years of age (1953).

70. Stewart and Hewitt,[42] studying 628 children born between 1952 and 1956 who died of leukaemia and lymphosarcoma before the age of five years, found that the percentage of X-rayed cases varied significantly with age at death and increased from age zero to age four. Assuming that none of the leukaemia cases dying within about fourteen months after exposure, i.e., before the age of one year were radio-genic, these authors also suggested that radio-genic cases had a different age distribution than spontaneous cases, and that, over this age range, the radio-genic cases had a tendency to develop later than "spontaneous" cases. The proportion of controls X-rayed (8.3 per cent) was almost the same as that for the youngest—"spontaneous"—leukaemia cases (8.5 per cent), and according to this hypothesis the "extra" X-rayed cases in the older age groups are attributable to the intra-uterine exposures.

71. According to MacMahon[43] the probability of death from leukaemia for white children in the United States up to the age of ten years is 46 per 10^5 persons. Assuming that 10 per cent of these children were X-rayed *in utero*, and that the X-rayed have a 40 per cent higher risk than the non-X-rayed, it follows that the over-all risk of $46/10^5$ is made up of a risk of $62/10^5$ for the 10 per cent X-rayed and $44/10^5$ for the 90 per cent not X-rayed. The difference, $18/10^5$, is the excess risk in the irradiated children. This would be compatible with extrapolated estimates from post-natal irradiated groups of 2 cases per million per rad per year only if the foetal dose received was 9 rads. This is probably at least twice the average dose received.

72. While individual studies of the incidence of leukaemia in children irradiated *in utero* have yielded different risk estimates, these are of different reliability on purely statistical grounds as indicated by their confidence limits. As discussed later in this annex, it has been shown[108] that there is no inconsistency in the findings of eleven surveys, five of which involved small samples with large sampling variability and gave estimates of relative risks less than one. The joint maximum likelihood estimate of the relative risk from all these surveys was in fact found to be 1.4 with 95 per cent confidence limits 1.2 and 1.6.

73. Although accurate estimates of doses are not available, it seems difficult to avoid the conclusion that irradiation of foetal tissue gives rise to a greater risk per unit dose than post-natal irradiation, possibly by a factor as high as 5. As in all cases of irradiation for medical reasons, there is no way to separate the leukaemogenic effect of radiation from other possible aetiologic factors connected with the reasons that had prompted the irradiation.

Leukaemia in other groups medically irradiated from external sources

74. Simon *et al.*[44] found, in 71,582 patients treated by radium therapy for carcinoma of the cervix in large treatment centres throughout the world, an incidence of between 6.2 and 11.6 cases of leukaemia per 10^5 persons. Comparing this to the British death rate of $5.8/10^5$ for leukaemia in women fifty-five years old and the incidence of $9.0/10^5$ for a similar group in the United States, they concluded that the incidence was not increased by radium treatment. However, since 25 per cent of the patients did not survive one year and since 60 per cent did not survive five years, the population at risk was greatly reduced before the greatest incidence of radiation-related leukaemia, if it occurred, would have been expected.

75. Faber[45,46], in a study of cases of leukaemia reported in the Danish Cancer Registry during the years 1940 to 1954, found that 34.8 per cent of the 442 acute leukaemia cases, 32.6 per cent of the 307 chronic granulocytic leukaemics, 17.7 per cent of the 861 chronic lymphocytic leukaemics, and 21.3 per cent of the 395 non-leukaemic controls used, had a history of diagnostic or therapeutic X-irradiation. The period between irradiation and development of the disease ranged from ten to sixty months for the acute cases and from ten to one hundred and forty months for the chronic granulocytic cases with no notable peak in the distribution. The relative risks and their 95 per cent confidence limits are: 2.0 (1.7-2.7) for acute leukaemia, 1.8 (1.3-2.5) for chronic granulocytic leukaemia, 0.8 (0.6-1.1) for chronic lymphocytic leukaemia. From the limits it can be concluded that only for acute and chronic granulocytic types was there a significant increase in risk. The doses received and the reasons of irradiation are not known.

76. Neumann[47] reported that from 1954 to 1960, 10 fatal cases of leukaemia were recorded in Stuttgart among tuberculosis patients over fourteen years of age in a sample equivalent to 91,549 person-years, and 5.86 cases were expected in a corresponding sample of the general population of Stuttgart in the same period. This gives a relative risk for leukaemia of 10/5.86 or 1.7 for the tuberculous patients who presumably were exposed more frequently to chest roentgenography than were control subjects. However, this does not differ significantly from unity, and in the ten tuberculous patients who died of leukaemia the exposure was slightly lower than that for the whole sample of tuberculous subjects.

77. Stewart *et al.*[48] in the United Kingdom made a retrospective survey of possible association between exposure to diagnostic or therapeutic X-irradiation and the subsequent development of leukaemia in adults. The frequency of irradiation appeared to be the same in the so-called L group (512 lymphatic leukaemia and lymphosarcoma cases), in a group of 951 cases with tumours of various sites, and in a group of 974 apparently healthy controls. In the so-called M group (511 myeloid and monocytic leukaemia cases) the frequency of irradiation of chest or abdomen was higher and that of the limbs not different from controls. To obtain risk estimates these authors compared frequency of trunk irradiation in their M series with the frequency of trunk

irradiation among all other groups pooled together (standard group), disregarding limb irradiation because the frequency of limb irradiation was not higher than in control groups.

78. In the standard group during the last ten years prior to the survey, 29 patients reported therapeutic irradiation, 1,025 reported diagnostic irradiation, and 1,323 reported no exposure. In the M group (myeloid and monocytic leukaemia) the corresponding figures were: 24 with therapeutic irradiation, 243 with diagnostic irradiation, and 244 with no exposure. The relative risks of leukaemia were 4.5 (95 per cent confidence limits: 8.0-2.5) for therapeutic irradiation of trunk and 1.2 (1.4-1.0) for diagnostic irradiation of trunk.

79. Gunz and Atkinson,[49] in a retrospective study in New Zealand, found that 47 of 590 leukaemia patients had received prior X-ray and/or radio-isotope (various) therapy for a variety of malignant or benign conditions, as compared with 38 of 712 non-diseased controls, giving a relative risk of 1.5 (1.0-1.9). They also found that 7 of 122 cases of myelomatosis (5.7 per cent) had previously been irradiated therapeutically (relative risk 1.1, limits 1.7-0.7). Prior radiation therapy was found in 25 of the 355 acute leukaemia cases (relative risk of 1.3, limits 2.1-0.8), 9 of the 78 chronic granulocytic leukaemias (relative risk 2.3, limits 5.0-1.0), and 13 of the 157 chronic lymphocytic cases (relative risk 1.5, limits 2.9-0.8).

80. In the same study the frequency of diagnostic irradiations received in the previous ten years was also investigated. Significant differences in the frequency of diagnostic irradiation among the various groups were apparent. No conclusion can be drawn with regard to the relative risks involved because the effect of diagnostic doses cannot be separated from that of the presumably much higher therapeutic doses received by a fraction of the same sample.

81. While therefore there is evidence that, as expected, therapeutic irradiation in adults may give rise to a detectable increase of risk of leukaemia, the question of the effect of diagnostic irradiation remains open. The results of Stewart *et al.*[48] are open to question as they barely reach statistical significance, because in that study leukaemics showed not only higher frequency of irradiation but also a different distribution, compared to controls, of diseases that might themselves lead to an increased risk of leukaemia, and because irradiations of the limbs, though they must have involved substantial doses to the bone marrow, were disregarded. More intensive investigations and better dosimetry are required before meaningful estimates of risk from diagnostic irradiation of adults can be obtained.

Leukaemia after I^{131} therapy

82. Pochin,[50] in a study of an estimated 59,000 patients (estimated 221,900 patient-years at risk) treated for thyrotoxicosis with radio-iodine for the twenty years preceding mid-1960 in the main clinics of the United Kingdom, Canada and Austria, and in a number of clinics in the United States, collected 8 published cases and 10 others, as well as 1 case of lymphosarcoma. He estimated from the appropriate national leukaemia rates an expected number of 21 $\pm$ 5 cases of leukaemia and concluded that presently available evidence, while giving no support to the possibility of leukaemia induction, neither excluded such induction nor made it possible to set any upper limit to the actual frequency. The average follow-up time in this study was about 3.8 years after therapy.

83. Wald *et al.*,[16] in their 1962 review, listed 2 additional cases of leukaemia and 1 of lymphoblastoma. Of the total of 20 cases there were 19 in which the type of leukaemia was known and 15 were acute. This proportion was higher than anticipated in a normal population of the same age and sex distribution. About 20 per cent only of the patients treated were males, but over 70 per cent of the cases of leukaemia occurred in males. Eight of the 20 cases were diagnosed in less than two years after therapy, and only 5 were diagnosed more than four years after therapy. The mean age of this population was higher than that of hyperthyroid patients in general, probably reflecting the conservative use of I^{131} in the young.

84. Werner *et al.*[51] in 1961 further analysed the data for the patients in the United States (collected by Werner and included in Pochin's report). In this series there were 10 leukaemia cases among 32,000 thyrotoxicosis patients treated with I^{131} with an average follow-up time of 4.44 years. An incidence of 13.8 cases was expected. There were significantly fewer chronic cases (3 as opposed to 9 expected). For males there was a significant excess of acute cases (6 to 1), while for females there was a significant deficit of total cases (2 to 10) and of chronic cases (1 to 6).

85. No information is available on the incidence of leukaemia in thyrotoxic patients not treated with radiation which would make it possible to distinguish the influence of the disease itself on leukaemia incidence from that of I^{131}.

Leukaemia in polycythemia vera patients treated with P^{32}

86. Wald *et al.*[16] in 1962 reviewed 1,238 cases of polycythemia vera cases treated with P^{32}. Among these there had been 41 deaths (3.3 per cent) from acute leukaemia. Since nearly 75 per cent of the patients in the combined series reviewed (observation periods ranging from seven to eighteen years) were still alive at the time the reports were made, complete ascertainment of leukaemia incidence has yet to be made. In the absence of adequate information on leukaemia incidence in polycythemia vera patients not treated with radiation, it is not possible to distinguish the influence of P^{32} from that of the disease. Prior to the advent of radio-phosphorus, as well as since, X-ray therapy has also been used frequently. There is some evidence among patients not treated with radiation suggesting that the condition of polycythemia may predispose to leukaemia or may be a condition closely related to leukaemia.

87. The general concept of myelo-proliferative diseases developed by Dameshek[52] would suggest that the stimulus producing an increased incidence of one form of this general type of disorder, e.g. myeloid leukaemia, might well be capable of the initiation of closely related clinical entities such as polycythemia vera, erythremic myelosis, and myelo-sclerosis. Yamazaki *et al.*[53] found 18 cases of polycythemia vera among Hiroshima atomic bomb survivors (22 per cent of the 81 cases reported in all of Japan since 1950). It should also be recalled that Lewis[9] found an increased incidence of multiple myeloma in United States radiologists.

Leukaemia and environmental radiation exposure

88. Craig and Seidman[54] have reported that the incidence of leukaemia in the one hundred and sixty-three

metropolitan areas of the United States bears no demonstrable relationship to differences in the amounts of cosmic radiations received.

89. Court Brown and Doll,[55] in a study of the four principal cities of Scotland, five rural areas, and the rest of Scotland found that, from 1939 to 1956, leukaemia mortality was highest in Aberdeen (mainly excess of acute and chronic myeloid) and in Edinburgh (mainly excess of chronic lymphocytic leukaemia). The average gamma radiation background in Aberdeen was 90 mrads per year and in Edinburgh 57 mrads per year. It was thought that the excess leukaemia mortality found was partly the result of better case finding in the large cities.

III. Thyroid neoplasms

Thyroid carcinoma in Japanese A-bomb survivors

90. Socolow *et al.*[56] have reviewed the cases of carcinoma of the thyroid that were detected through routine medical examination, between 1 July 1958 and 1 July 1961, in matched groups of exposed and non-exposed subjects included in the long-term medical investigations (Adult Health Study) of the Atomic Bomb Casualty Commission (ABCC). The sample used in the Adult Health Study consisted of four components balanced for age and sex: group 1 (proximal), exposed within 2,000 metres of the hypocentre (reported acute radiation symptoms); group 2 (proximal), exposed within 2,000 metres (reported no acute radiation symptoms); group 3 (distal) exposed 3,000 to 3,499 metres; and group 4 (non-exposed), beyond 10,000 metres or not in the city at the time of the bombing. The review by Socolow *et al.* includes 10,730 subjects in Hiroshima and 4,589 in Nagasaki.

91. During the three-year period of this study 355 patients were found to have enlarged thyroid glands in Hiroshima and Nagasaki. Biopsies were recommended in 114 cases in Hiroshima and 17 in Nagasaki, corresponding to 37 and 38 per cent, respectively, of patients with thyroid enlargement. Seventy biopsies were performed, 64 in Hiroshima and 6 in Nagasaki. While there was a random distribution of three types of thyroid enlargement among the four component groups in Nagasaki, single nodules were diagnosed more often among patients of exposure group 1 in Hiroshima. Biopsy was recommended in about 80 per cent of cases with single nodules, 50 per cent with multi-nodular glands and 3 per cent of cases with diffuse goitre. That there was some degree of uniformity of sampling is suggested by the fact that the percentages of patients with single nodules of the thyroid who were biopsied were fairly constant (range 42-45 per cent) within the four exposure groups in Hiroshima. A similar analysis cannot be made in Nagasaki in view of the smaller number of cases and fewer biopsies performed.

92. Twenty-one patients with histologically confirmed thyroid carcinomas were seen. Seventeen cases were diagnosed as a result of a routine Adult Health Study medical examination and 4 cases were diagnosed elsewhere just before the inception of the current study in July 1958.

93. In Hiroshima, exposure group 1 contained 10 of the 18 thyroid cancer cases seen in that city, a finding which is moderately significant ($0.05>P>0.02$). When the figures for both cities are combined, 10 of the 21 cases fall within group 1, a distribution of doubtful significance ($0.10>P>0.05$). However, if only the 17 cases diagnosed as a result of the Adult Health Study examinations are considered, a different distribution is seen. All 4 cases diagnosed at other hospitals are in the proximal exposed group, and their elimination from the analysis results in a distribution that does not depart significantly from chance ($P>0.30$). Fourteen of the 21 cases occurred within 1,399 metres, a significant increase compared with those at greater distances.

94. According to Socolow *et al.*,[56] although the overall incidence of thyroid cancer in the Adult Health Study may not depart greatly from that cited by others for Japan, the age distribution differed significantly in that the cases in the A-bomb survivors were younger. Of the 21 cases, 8 were diagnosed in the group under the age of thirty-five years. At the time of exposure these patients ranged in age from six to twenty years. The latent period after exposure can only be defined as less than thirteen or fifteen years owing to the fact that all cases in this study were diagnosed between 1956 and 1961. Among cases occurring in the younger age groups, over 80 per cent were exposed within 1,400 metres, whereas in older age groups fewer than 50 per cent were similarly exposed.

95. From the available information it is difficult to define a pertinent population base for these cases or to evaluate the general significance of identified cases. However, if the findings were representative of the incidence in the total exposed and non-exposed populations in this study, the incidence of thyroid carcinoma in the exposed would be 19/14,970 or 0.13 per cent, and in the non-exposed 2/4,992 or 0.04 per cent.

96. In 1964 Zeldis *et al.*[57] reviewed thyroid lesions in autopsy and surgical pathology specimens in Hiroshima A-bomb survivors. From 1 January 1948 to 30 December 1960, thyroid specimens were available in 1,253 of a total of 1,535 adult autopsy cases examined at ABCC. For the same period, a total of 342 surgical thyroid specimens, representing 301 cases, were assembled from all pathology departments in Hiroshima. However, for analysis of the surgical thyroid specimens, it proved impossible to define a pertinent population base for the entire series or to evaluate the general significance of identified cases in the face of the known occult character of many thyroid lesions. Therefore, analysis was made simply of diagnoses in those 70 of the surgical cases which fell into two exposure groups (within 1,399 metres and between 1,400 and 1,999 metres). Table XIII gives the incidence of thyroid cancer and thyroid adenoma in the autopsies and in the 70 surgical thyroid specimens according to distance from hypocentre.

97. The incidence of thyroid carcinoma in the total of 1,253 autopsy cases, in the entire exposed group, or in the group exposed between 2,000 and 9,000 metres, is about 3 per cent, or only about 1.15 times the incidence in the non-exposed. The incidence in the non-exposed is 1.7 times that in the group exposed between 1,400 and 1,999 metres. The incidence in the group exposed within 1,400 metres is about twice that in the non-exposed group. The incidence of thyroid adenoma is more than doubled in the most proximally exposed group and slightly increased in other exposure groups.

98. In the surgical specimens, the incidence of thyroid carcinoma in the group exposed within 1,400 metres was 3.75 times that in the 1,400-1,999 metres group. However, the incidence of thyroid adenoma was 2.6 times greater in the latter group than in the former group.

99. The increased incidence of thyroid carcinoma in the autopsy cases in the group exposed within 1,400

metres was found not to be statistically significant ($P = 0.07$), but the increased incidence in the thyroid cancers in the surgical specimen groups exposed within 1,400 metres was found to be statistically significant.[57]

100. Taken together, both surveys suggest that the incidence of thyroid carcinoma has been increasing in the irradiated population of Hiroshima and Nagasaki, the incidence varying inversely with distance from the hypocentre. Difficulties of ascertainment, due to the fact that the incidence of carcinoma of the thyroid is difficult to record, and the fact that the latent periods are long, make it difficult to set up the surveys that would be necessary to obtain information on the dose-effect relationship and therefore on the risk of induction.

Thyroid neoplasms in patients therapeutically irradiated from external sources

101. Takahashi *et al.*,[58] in a retrospective survey of human cancer in relation to medical exposure in certain hospitals up to 1962 in Japan, found 638 cases of thyroid cancer, of which 29 (4.55 per cent) had histories of therapeutic irradiation with the thyroid within the radiation beam. In control groups of similar age distribution, 9 of 1,535 (0.59 per cent) received irradiation to this neck region. The difference was significant at the 1 per cent level.

102. However, the control group in this study is insufficiently characterized to permit adequate interpretation of the results with respect to the influence of factors other than radiation in the results. The study does not distinguish between (*a*) effects of radiation, and (*b*) effects of the conditions which prompted the irradiation, on the occurrence of neoplasms after irradiation. Furthermore, it is not clear how far back in time the survey of irradiation went. It should be noted also that the data indicate an average dose of 14 rads in the controls.

103. Neglecting these reservations, it is possible to compare the proportion of irradiated subjects among patients developing thyroid cancer and among healthy controls. The relative risk estimate so obtained amounts to 8.4 with 95 per cent confidence limits 14.8 and 4.8, respectively. Dividing the estimate by the excess radiation (160 R) received on the average by thyroid cancer patients compared to healthy controls gives a relative risk per R of approximately 5 per cent. In table XIV are given the proportions of patients with and without thyroid cancer, according to the estimated exposure.

104. In many prospective surveys[18,23-29,60,61] and in numerous retrospective surveys,[63-73] therapeutic irradiation of children has been associated with a subsequent increase in incidence of thyroid tumours. In some prospective studies[22,62] this association has not been found. A number of surveys of thyroid cancer incidence following therapeutic irradiation for benign conditions are summarized in table XVI.

105. In 1962 Hanford *et al.*[59] reported the results of a study of 458 patients who had received therapeutic irradiation for non-malignant disease of the cervical region (thyroid included). Of these patients, 431 patients comprised three main treatment groups, including 43 treated as infants for "enlarged thymus", 92 children and adults treated for toxic goitre, and 296 children and adults treated for tuberculous adenitis. Eight cases of thyroid cancer were found where ~ 0.1 case was expected, 7 of them among the 162 tuberculous adenitis patients followed ten years or longer after treatment, and 1 case among the toxic goitre patients.

106. Data on ages at irradiation and at operation for thyroid cancer, and approximate doses, for the 7 cancer cases in the tuberculous adenitis group are given in table XV. Five of the 7 cases were four to eighteen years of age at the time of irradiation, and the others were twenty-three and thirty-four years of age, respectively. Five of these 7 cancer cases were among 54 patients who had received exposures between 500 R and 1,000 R, and 2 of the cases were among 66 patients who had received exposures of 1,000 R or more. The one patient who developed thyroid cancer after irradiation for toxic goitre had received an exposure of about 2,000 R at sixteen years of age.

107. In the prospective survey by De Lawter and Winship[74] of adult patients X-irradiated for hyperthyroidism or other benign diseases of thyroid, there was no evidence of thyroid cancer in any of the 222 patients followed for an average of 22.5 years.

108. In a retrospective survey of 286 cases of thyroid cancer, Winship and Rosvoll[70] found that 80 per cent of the 286 cancer cases had received prior therapeutic irradiation to head or neck during infancy or childhood, mostly for "enlarged thymus", others for hypertrophic tonsils and adenoids, and some for other benign diseases. The exposures ranged from 180 R to 6,000 R with an average of 600 R. The latent periods averaged 8.6 years, were often less than five years, and in 12 cases there were thyroid tumours at birth which later proved to be carcinoma.

109. In the prospective survey of Pifer *et al.*[27] (see tables XI and XVI and paragraphs 57 and 58 for description of survey) of infants X-irradiated therapeutically for "thymic enlargement", 8 cases of thyroid cancer were found in Series I among patients followed from thirteen to thirty-four years, as compared with 0.09 case expected on the basis of the "natural" incidence. The relative risk was 88.9. In Series I, 21 cases of thyroid adenoma were found, and 0.9 was expected, with a ratio of 23.3. The mean exposure for Series I was 329 R.

110. In Series II, with patients followed only from three to twenty years, only 1 case of thyroid carcinoma was found, and 0.01 case was expected. The mean exposure for Series II was 126 R.

111. The 9 cases of thyroid carcinoma in the two series combined were associated with cumulative exposures ranging from 156 R to 1,092 R, with a mean of 598 R. Seven of these cases had been irradiated with combined anterior and posterior port arrangements, 1 anteriorly only, and in 1 case the port arrangement had not been ascertained. The 21 cases of thyroid adenoma were associated with cumulative exposures ranging from 144 R to 756 R, with a mean of 372 R. The percentages of these adenoma cases in the various exposure positions were similar to those for the carcinoma cases.

112. Toyooka *et al.*,[28] in analysing the data of Pifer *et al.*,[27] found that the incidence of thyroid tumours was considerably higher when a dose was given half anteriorly and half posteriorly than when the same dose was given from an anterior approach only. Including additional thyroid neoplasms found between 1960 and 1964, it was found that 29 of the total of 34 thyroid tumour cases (malignant and benign) occurred among 472 children treated by combined anterior and posterior

approaches, whereas only 5 of the tumour cases developed in the 2,111 children treated anteriorly only.

113. Attempts to demonstrate a dose dependence for these major positional groups were unsuccessful. A decision cannot now be made as to whether the high tumour incidence in children treated anteriorly and posteriorly was a consequence of port position, of statistical variation, or of other factors. The possibility has been demonstrated that the posterior approach, with its attendant difficulties, could have resulted in exposure of larger regions of the neck and head, including the pituitary gland, than was the case when the anterior approach alone was used. In this connexion, there is uncertainty in this and other surveys of children irradiated for "thymic enlargement" as to the extent of the inclusion of the thyroid gland in the radiation beam in many of the patients. Of importance in this study are the facts that many of the older children (irradiated in earlier years) were given relatively large doses to relatively large areas, often with posterior as well as anterior position of ports, and with greater X-ray energies, numbers of treatments and total periods of treatment, as compared with younger children (more recently irradiated).

114. Toyooka *et al.*,[29] in a report of clinical aspects of these thyroid tumour cases, indicated an equal sex distribution of adenomas, but a greater frequency of carcinomas in females. The mean exposure in carcinoma cases was almost three times that for the entire group, and the mean exposure for adenoma cases was almost double that for the whole group. The average latent periods were 16.3 years for carcinomas and 18.2 years for adenomas. There was no relation between latent period and exposure.

115. Saenger *et al.*[24] reported a prospective study of 1,644 patients under sixteen years of age who had received X-ray therapy to the head, neck or chest during the period 1932-1950 for benign conditions, and of 3,777 untreated siblings. Irradiation for "thymic enlargement" accounted for 33 per cent and irradiation for cervical adenitis for 26.9 per cent. Twenty-one per cent of the sample received radiation therapy for two or more presumably unrelated conditions. Exposures ranged from $<$ 50 R to 5,000 R, with 62 per cent receiving less than 400 R and 72.5 per cent receiving less than 600 R. Eleven cases of thyroid cancer were found among previously treated cases who were under twenty-three years of age at the time of cancer development, and 0.12 case was expected, giving a relative risk of about 91.

116. No one of the surveys on the incidence of thyroid cancer among irradiated subjects makes it possible to obtain dose-effect regression. Because of the small number of cases in any individual survey only one point of the dose-response curve can be determined from each of them. Crude risk estimates, however, can be obtained for a number of surveys by determining the slope of the straight line connecting the origin with the points obtained when the observed incidences (cases per man year) are plotted against exposures. The risk estimates are summarized in table XVI.

117. A single straight line can be fitted through the incidences observed in surveys of children when plotted against exposures. Its slope indicates that the rate of increase of the incidence with exposure is 0.9 cases/10^6/y/R. Taking into account the statistical uncertainty of the data and the probably larger uncertainties of the dosimetry, it appears that the joint risk estimate may be between 0.5 and 1.5 cases/10^6/y/R. The estimate is based on an average follow-up time of about sixteen years and is valid for acute exposure of children only in an estimated exposure range of 100 to 300 roentgens.

118. Analogous joint estimates had been obtained by Beach and Dolphin[138] through a different method of estimation and on the basis of more limited and partly different data. Their risk estimate (35 cases/10^6/rad) is a lifetime risk estimate. Assuming that the period of risk for cancer of the thyroid is between fifteen and twenty years, Beach and Dolphin's estimate is slightly higher than the one given in the previous paragraph. Differences in method and in material can easily account for the discrepancy.

119. The validity of an estimate of risk such as is given here is limited, like all empirical estimates, to the range of exposures from which it has been derived—in the present case between 100 and 300 R. It also suffers from the fact that the results were obtained in children irradiated for medical reasons and may therefore be different from the estimate that would apply to a random sample of the population. Finally, the uncertainties in dosimetry are high. Doses have probably been overestimated, thus slightly lowering the risk estimate.

Thyroid neoplasms in patients given I^{131}

120. In 1962 Sheline *et al.*[75] reported on a group of 256 patients with diffuse hyperthyroidism (non-nodular thyrotoxicosis) treated with I^{131} between 1946 and 1953. Eight patients (3.12 per cent) developed multiple nodules in the thyroid glands between five and eleven years after therapy and then had surgery. The nodules occurred in six of eighteen patients (33.3 per cent) under 20 years of age at the time of treatment, and in two of 238 adult patients (0.84 per cent). The ages at time of treatment in these tumour cases ranged from 2.66 to 29 years. One of the children showed evidence of thyroid carcinoma of low grade malignancy.

121. Aside from the above case, no association between I^{131} treatment for thyrotoxicosis and thyroid cancer has been reported.

IV. Neck tumours (excluding thyroid) after therapeutic irradiation

122. Takahashi *et al.*,[58] in their retrospective survey of human cancer in relation to medical exposure in Japan, found 906 cases of cancer of the neck (excluding thyroid cancer), namely, cancer of the pharynx, larynx, root of tongue, cervical oesophagus, parotid gland, etc. Of these, eleven (1.21 per cent) had histories of therapeutic irradiation of the neck, compared with a control irradiation frequency of 8 out of 1,770 (0.45 per cent). The difference was significant at the 5 per cent level and gave an over-all relative risk of 2.7.

123. In table XVII are presented the relative risk values for neck cancer at various ranges of estimated exposure. The relative risk is only 1.22 at the 500-2,000 R range and increases to 4.41 for the 6,000-8,000 R range. The risk of radiation-induced cancers of the neck other than thyroid cancers seems to be less than that for radiation-induced thyroid cancer.

124. These data suggest that relatively large exposures are required to cause substantial increase in incidence of neck cancers other than thyroid cancer. This is in accord with the observation by Goolden[76] in his review of pharyngeal and laryngeal cancers following radiotherapy (which in 75 per cent of patients was given for thyrotoxicosis) that few cases did not show signs of

severe radiation damage to the skin or subcutaneous tissues long preceding the appearance of the cancer. It should be noted that the severity of skin changes depends on a number of radiological and temporal factors.

125. Pifer *et al.*,[27] in their study of 1,451 children treated for thymic enlargement between 1926 and 1946 with exposures averaging 329 R found 3 cases of salivary gland cancer up to 1960, compared with 0.05 case expected, and compared with 0.09 case expected in 2,073 sibling controls and none observed.

V. Skin tumours after therapeutic irradiation

126. Takahashi *et al.*,[58] in their survey of human cancer in relation to medical exposure in Japan noted the increase in relative risk of skin cancer with the exposure received (table XVIII). As in the case of thyroid cancer, though the rising trend is apparent, limits are very wide owing to the paucity of cases.

127. The over-all relative risk was 6.0, with limits 16.3 and 2.2. With an average excess radiation of 240 R in the cancer group the relative risk per roentgen is 2.6 per cent. Most instances of radio-therapy probably involved fractionated exposures and these risk estimates may not apply to single exposures.

128. Clinical and occupational experience suggests that the risk of cancer of the skin is low and that radiation-induced skin lesions usually precede the development of cancer. It may well be, however, that, as the observations are not based on well designed surveys, radiation-induced cancers are recognized as such mainly because of the existence of previous lesions.

129. Two studies have shown the occurrence of skin cancer in 10 per cent[77] and 28 per cent,[78] respectively, of patients with chronic radiation dermatitis. Since the latent period may vary from three to as long as forty-eight years or more,[79] those percentages may reflect incomplete ascertainment. The dependence of the frequency and seriousness of radiation dermatitis on dose has not been studied quantitatively and would in any case complicate the establishment of dose-effect relationships for cancer of the skin. No increase in incidence of skin cancer has been noted yet among the survivors of Hiroshima and Nagasaki.

VI. Bone tumours

Bone tumours after therapeutic irradiation

130. Most of the few cases of bone sarcoma that have been reported after radiation therapy have arisen in areas of bone containing previously either a benign tumour or chronic osteomyelitis.[80] In some instances, however, the site of origin was normal prior to irradiation. Reports of radiation-induced osteogenic sarcoma have in some cases indicated substantial radiation damage of bone and/or marrow persisting before cancer development, while in other cases this has not been detected. The lowest exposure thought to have caused an osteogenic sarcoma[81] is, at the present time, about 3,000 R. Most other reported cases have received larger exposures, usually between 4,000 and 7,500 R, sometimes greater.

131. However, Pifer *et al.*[27] found 9 osteochondroma cases (1.2 expected) up to 1960 in 1,451 (Series I) children treated for thymic enlargement (from 1926 to 1946) with exposures averaging 329 R, and 2 cases of osteochondroma (0.26 expected) up to 1960 in 1,358 (Series II) children so treated (from 1940 to 1957) with exposures in the same range but with a mean of 126 R. The relative risks are 7.5 for Series I and 7.7 for Series II, representing increases per R in the vicinity of 2.0 and 5.3 per cent, respectively. However, it should be noted that all of the 6 cases of osteochondroma given both anterior and posterior treatments (Series I) occurred within the tissues in the primary X-ray beam, whereas 4 of the 5 cases of osteochondroma observed in children irradiated anteriorly (Series I and II) arose in tissues outside the primary beam. In the sibling control groups, 1 case was observed in Series I (1.53 expected), and no case was found in Series II (0.66 case expected).

Bone tumours in persons with radium body burdens

132. Hasterlik *et al.*[82,83] have extended and confirmed the results of earlier surveys which indicated a strong correlation between the frequency of development of bone tumours and the skeletal content of radium. They reported a study of 264 persons sought out and measured for radium content in the Chicago area who had been formerly employed in the radium watch dial industry (219) or as radium chemists (4), or who had received radium as a form of medical therapy (41). With respect to watch dial painters, this study concerned itself almost exclusively with those women whose occupational history antedated 1925. Detailed radiographic studies of the entire skeleton were completed in 236 of the 264 persons. Of the 264 persons measured for radium, there were 23 (8.7 per cent) with a body burden of < 0.001 μCi Ra^{226}, 36 (13.6 per cent) with 0.001—0.01 μCi, 102 (38.4 per cent) with 0.01 — 0.1 μCi, 62 (23.5 per cent) with 0.1 — 1.0 μCi, and 41 (15.5 per cent) with > 1 μCi. The frequencies of different body burdens and the number of cases of bone malignancy are given in table XIX.

133. Three of the 61 radiographed persons (including 43 dial painters) in the 0.1 to 1.0 μCi range (4.9 per cent) revealed malignant neoplasms of or associated with the skeleton. Of the 40 radiographed persons (including 19 dial painters) containing above 1 μCi, 14 (35 per cent) had such malignant neoplasms. Of the 17 neoplasms, 12 were bone sarcomas (0.45 to 6.8 μCi), and the others were epithelial and other neoplasms of mastoid and paranasal sinuses (0.89 to 4.7 μCi). The bone sarcoma case at 0.45 μCi, however, was found to be a person with an exceptionally rapid metabolism and therefore a higher original burden than this figure would indicate.[84]

134. Analyses of these data suffer from the fact that the determinations of radium content of the body have been done at least thirty-six years after the acquisition of the radio-active material. At the present time, the extrapolation of these burdens of radium to radium burdens at early times is difficult and carries large factors of uncertainty. The dial painters may, in addition, have been exposed to substantial gamma doses over the period of their employment at this work. Furthermore, there is great uncertainty as to the relevant radiation dose and its target in terms of production of osteosarcoma, e.g. integration of radiation doses to whole skeleton, to some unit volume of bone, etc. The radiation doses to different units or components of bone vary greatly.

135. However, tentative and very rough estimates of relationships between incidence and body burden have been drawn by Hasterlik[82] who analyzed data in two body burden ranges. In the range of 1 to 10 μCi there was

a total of 41 persons with about 1,300 man years irradiation experience and 14 cases of malignancy. The risk of malignancy per man year of exposure in this group is therefore about 1.06×10^{-2}. In the group of 62 persons (2,200 man years irradiation experience) in the 0.1 to 1.0 μCi body burden range, with 3 cancer cases, the risk is about 1.34×10^{-3}.

136. On the basis of the assumed initial intake of radium, it can be calculated that the risks of development of bone tumours in the two groups are 22 and 33×10^{-6} cases per year per microcurie intake, respectively. The selection of midpoints in the two content ranges for these calculations involves certain assumptions concerning the distribution of patients with different body burdens within the two ranges.

137. Assuming as the Committee did in the past that the cells lining bone surfaces are those that give rise to malignancies when irradiated, crude estimates of risk per unit dose can be obtained from these data and may be taken to be about 4 cases/10^6/y/rad, or a figure of the same order of magnitude as that for leukaemia and thyroid cancer following irradiation from external sources. However, comparison of the figures may be quite misleading in this case, since it is not known how much of the dose that is delivered over several decades is, in fact, responsible for the induction. Also, the doses are average values to the cells lining bone surfaces and do not take account of the highly inhomogeneous distribution of the absorbed dose. Furthermore, the estimate of risk of bone tumour is based on experience within two broad dose ranges only and therefore gives no information on the shape of the dose incidence relationship nor on the effects of lower doses. It may be noted that the general incidence of primary bone tumours is reported to be about 10 cases/10^6/y and may, in fact, be somewhat lower.

138. In these studies[82] only 2 of the 264 patients showed (by X-ray spectroscopy) evidence of the presence of mesothorium as well as of Ra^{226}. This situation is in contrast to the patients studied by other groups in the United States, since the dial painters in other groups ingested paints containing varying mixtures of Ra^{226}, radio-thorium, and/or mesothorium. In the study by Hasterlik *et al.*, most of the dial painters were unselected cases but several of the other subjects were not.

139. Aside from the study described above, Hasterlik *et al.*[83] have found 2 cases (death certificates) of acute myeloid leukaemia in radium patients, one of which was confirmed by their study of the original blood smear made shortly before the patient's death in 1931.

140. Barrer *et al.*[127] have given a preliminary summary of the data from the first 150 cases studied in a large survey in the State of New Jersey, United States. Three (2 per cent) developed osteogenic sarcomas, associated with body burdens of 0.6, 0.9, and 1.67 μCi Ra^{226}. There was 1 case of chronic myelogenous leukaemia associated with a body burden of < 0.0042 μCi Ra^{226}. Of 190 death certificates for deceased radium cases, 64 (33.7 per cent) made mention of malignancies, of which 16 cases were of bone and periosteum (8.4 per cent) and 3 were tumours of the nose and of the paranasal sinuses (1.5 per cent).

141. At the Massachusetts Institute of Technology[85] there have been studies of persons with skeletal burdens of radium or radium-mesothorium mixtures resulting from occupation (dial painters, chemists, physicists), from parenteral injection of radium solutions and from ingestion of radium or radium-mesothorium mixtures.

142. Because many of these patients are exposed to substantial amounts of mesothorium (Ra^{228}) the total radiation from decay of the mesothorium chain has been estimated and the equivalent in terms of radiation from Ra^{226} has been used, the data being expressed in μCi minimum pure radium equivalent (MPRE).

143. In December 1963 Evans *et al.*[124] reported on 237 subjects (of the more than 350 under study) who had had radiological examinations. Of these, 76 cases showed radiological abnormalities associated with body burdens ranging from 0.23 to 44.0 μCi minimum pure radium equivalent (MPRE). Excluding dental abnormalities, which were almost universal in these 76 cases, there were 18 persons with no evidence of abnormality or disease (including body burdens as high as about 3 μCi MPRE), 19 cases of spontaneous fracture (0.6 to 20.0 μCi), 23 cases of osteogenic sarcoma (0.6 to 24.0 μCi), 8 cases of cancer of the paranasal sinus or mastoid (1.0 to 10.0 μCi), and 3 cases of osteomyelitis of mandible or maxilla (15.0 to 44.0 μCi).

144. More recently, Maletskos *et al.*[128] summarized the results of the same survey covering 361 subjects (88 males and 273 females) with skeletal burdens of Ra^{226} and Ra^{228}. Two-thirds of the subjects were dial painters, some of whom had received Ra^{226} alone and others a mixture of Ra^{226} and Ra^{228}. The remainder were persons contaminated as a result of laboratory work or by ingestion or injection of the radio-active materials. Most of the cases studied earlier (before 1957) came to attention because of the symptoms they developed, while the majority of cases studied more recently were investigated after being found by search. There were 299 found by search and 62 by symptoms. The main exposure period of the dial painters was from 1918 to 1925, although a few started as early as 1915. Fourteen of the cases had originally been examined by Martland.[129-131]

145. With regard to the data of Maletskos *et al.*,[128] one of the authors has stated:[86] "At the present stage of the statistical study of these data . . . , no clinically significant signs or symptoms are seen with residual or terminal body burdens of <0.5 μCi Ra (MPRE). With higher residual body burdens, beginning in the neighbourhood of 1 μCi Ra and extending to about 25 μCi Ra, the fraction of the total number of cases which involve either of these types of malignancies (osteogenic sarcomas and carcinomas of the paranasal sinuses and mastoids) amounts to roughly ¼." This statement need not be taken to be in conflict with reports of radiographically observed changes in fine structure of bone such as appear in table XXII associated with burdens below 0.1 μCi. Data on the percentages of subjects in various body burden ranges showing osteogenic sarcomas or cancers of paranasal sinuses or mastoid are not yet available for publication.

VII. Thorium-related neoplasms

146. Thorium dioxide in a colloidal suspension, known as Thorotrast, was widely used in diagnostic radiology between 1928 and 1945, primarily for the visualization of the liver, spleen, cerebral arteries and cavities of the body. The thorium dioxide content of Thorotrast is usually about 25 per cent by weight, but may vary considerably. After intracavitary or extravascular injection, Thorotrast remains largely at the place of injection and may be carcinogenic there. After intravascular injection, the thorium dioxide particles are deposited in phagocytic cells of the reticulo-endo-

thelial system, and by this means they are concentrated in liver and spleen. Immediately a redistribution begins which continues slowly for years and results in change of position of the particles in the organs of concentration and in increasing amounts in the connective tissues of the body.

147. In 1962 Dahlgren[88] reviewed the literature on tumours following administration of Thorotrast. He listed 68 cases of malignant tumours that had been reported, including 3 sarcomas at the site of extravascularly deposited Thorotrast (volume injected 20 ml in 1 case, unknown in 2 cases) with latent periods of six to twenty-five years; 26 carcinomas and sarcomas in a variety of organs (kidney, breast, eyelid, maxillary sinus, bronchi, peritoneum, ovary, seminal vesicle) after deposition of Thorotrast in natural cavities (volume injected known in 4 cases, 10, 24, 30 and 30 ml, respectively) with latent periods ranging from ten to thirty-five years; 33 malignant tumours (sarcomas and carcinomas) of liver and bile-ducts after systemic injection of Thorotrast (volume injected known in 12 cases: 3 cases at 20, 34 and 70 ml, respectively; 5 cases at 75 ml; and 4 cases at 80 ml) with latent periods ranging from three to twenty-four years; and 6 cases of malignant tumours in various organs (colon, lung, spleen, kidney) after systemic injection of Thorotrast (only one known injected volume, 15 ml) with latent periods ranging from thirteen to twenty-four years. The mean latent period for the total cases is 17.7 years, with mean latent periods in the four subgroups above ranging from 16.7 to 18.3 years.

148. In 1963 Blomberg *et al.*[89] reported a study of patients who had received Thorotrast injections in cerebral angiography during the years 1932-1947. They found 6 cases of primary malignant liver tumours (5 hepatic cancers and 1 haemangio-endothelioma) in 908 patients (about 0.66 per cent). Information on the amount of Thorotrast injected was available for 436 of the patients and for 3 of the 6 cases of liver cancer. The 413 patients who had received less than 30 ml showed no tumours of liver, while 18 patients receiving between 30 and 40 ml showed 1 case of liver cancer and 2 among 5 patients receiving more than 40 ml had liver cancers. Of the remaining 3 patients who had received more than 40 ml, 2 died during the first year after the injection and the fifth patient was not traced. The latent periods for the 6 liver cancer cases found in this study ranged from nineteen to twenty-seven years, and the longest observation period was twenty-nine years. Blomberg *et al.*[89] indicate that the order of magnitude of the mean alpha radiation doses to the liver and the spleen after intravascular injection may be hundreds of rads per year.

149. According to Looney,[91] the accumulated mean radiation dose to the liver in Thorotrast-injected patients who developed Kupffer cell sarcoma of the liver (mean latent period 15 $\pm$ 7 years) was of the order of 1,000 to 1,500 rads. The estimated accumulated dose to the liver of one patient who developed an hepatic tumour only three years after 20 ml of Thorotrast was about 100 rads.

150. However, it should be emphasized that there are enormous difficulties involved in attempts to determine dose parameters and the doses relevant to cancer incidence for thorium dioxide. After intravascular injection, the distribution of the material, and therefore of the radiation as well, is extremely non-uniform and changes with time. The radiation, consisting of alpha-, beta-, and gamma radiation in the ratio 90:9:1,[132] is confined largely to a very short radius from the particles of origin and is absorbed to variable degrees by the particles or particulate agglomerations of origin themselves. It should also be mentioned that irritation of tissues by the particles, apart from effects of radiation, cannot be discounted as a factor in the pathogenesis of tumours. Data from experimental mammals,[133] using non-radio-active zirconium hydroxide in colloidal suspension, give some support to that view.

151. According to Wald *et al.*,[16] various case reports and follow-up studies have resulted in the accumulation of about ten instances of leukaemia following thorium injection. In 6 cases for which detailed data were available, the latent period was 12 $\pm$ 7 years. A causal relationship has not been established between leukaemia and Thorotrast injection.

VIII. Lung cancer in miners

152. A review of earlier studies of lung cancer related to radiation in miners has been published by Stewart and Simpson.[92]

153. In 1926 Rostoski *et al.*[93] reported that in miners (pitchblende) in the Schneeberg region of Saxony, dying between 1921 and 1926, about 50 per cent had carcinoma of the lung, with the majority originating in large bronchi, while the incidence of lung carcinoma in the control groups was not noteworthy.

154. Pirchan and Sikl[94] found that about 50 per cent of the miners (pitchblende) of Jachymov, dying in 1929 and 1930, also had carcinoma of the lung. Observations of these miners up to 1939 confirmed earlier findings.

155. Although these mines contain several potentially carcinogenic materials including arsenic and cobalt, these elements are also found in many mines where there is no particularly high incidence of carcinoma of the lung. Radium itself, as a constituent of the airborne dust in the mines, has been suspect, but measurements of the radium content of the lungs of deceased miners have shown no substantial difference from that in the average human of middle age. Silicosis and silicotuberculosis have been considered as contributing causes since at Jachymov nearly half the miners died of either silicosis, tuberculosis, or a combination of the two, and pneumoconiosis was often found at autopsy in the Schneeberg miners as well.

156. The opinion is now generally held that airborne radon in the mines may probably be the most essential factor in the production of lung cancer. The Jachymov and Schneeberg mines have radio-active air and radio-active ore in common.

157. According to Bale[95] the major portion of the radiation dose from Rn^{222} stems from the decay of the short-lived daughters (RaA through RaC') that are carried by the atmosphere in varying degrees of equilibrium with the radon parent and are trapped in the lungs. Radon itself and the daughter products of its decay while in the lungs contribute only about one part in a thousand of the total dose to the bronchial epithelium.

158. Chamberlain and Dyson[96] concluded from experiments that the major portion of the radiation dose to the trachea and large bronchi was attributable to the fraction of RaA preformed in the inhaled atmosphere and unattached to dust or condensation nuclei.

159. Evans and Goodman[97] summarized data on the radon content of the air in the Schneeberg and Jachymov

mines and concluded that the average radon concentration to which the miners had been exposed was about 2.9×10^{-9} Ci/l. They concluded also that prolonged breathing of an atmosphere containing about 10^{-9} Ci/l of radon may have been responsible for the increase in the incidence of lung cancer observed in the Schneeberg and Jachymov miners.

160. In 1945 Mitchell[98] considered it possible that the average radon concentration was higher in the years before the hazard was recognized and regarded a level of about 1.5×10^{-8} Ci/l as a reasonable estimate of average concentration.

161. Assuming a linear relationship between concentration of Rn^{222} and duration of exposure and the lung cancer incidence, and using the Schneeberg and Jachymov experience, Evans and Goodman[97] in 1940, and Mitchell[98] in 1945 suggested MPC (maximum permissible concentration) values for human exposure to airborne Rn^{222} and daughters of 10^{-11} Ci/l and 5×10^{-11} Ci/l, respectively. From the literature Mitchell concluded that the approximate 50 per cent incidence of lung carcinoma in the Jachymov miners was associated with employment of the order of ten years and exposure during working hours to a concentration of radon not less than 2.5×10^{-8} Ci/l. To reduce the incidence of lung carcinoma to about that of the community as a whole, i.e., to the order of 0.1 per cent for ten years, the concentration of radon in the air would have to be reduced to 5×10^{-11} Ci/l.

162. Sikl[99] in 1950 stated that in his experience the average duration of exposure associated with carcinoma of the lung was seventeen years, the shortest thirteen years.

163. Jacoe[100] in 1953 in his search for airborne radon in tunnels and non-uranium mines in Colorado, mostly in areas of little air movement, found a range of concentration from zero (instrumental) to one reading of 2.1×10^{-9} Ci/l. Most of the samples were in the range of 5×10^{-10} to 10^{-11} Ci/l.

164. Harris[101] in his examination of atmospheres in zinc, iron and talc mines in New York, where ventilation was low, found radon concentrations ranging from 4×10^{-11} to 10^{-12} Ci/l.

165. Oosthuizen *et al.*[102] in their measurements of radon concentrations in the air of the gold mines of the East, Central and West Rand, South Africa, found in areas where the uranium content of the ore was too low for economic extraction, average radon atmospheric concentrations in the range from 2.5×10^{-11} Ci/l, to 5×10^{-11} Ci/l, with measured values as high as 2×10^{-10} to 3×10^{-10} Ci/l. In mining areas where the extraction of uranium was economic, radon concentrations in the mines were in the range from 10^{-10} to 5×10^{-10} Ci/l. These authors reported that the incidence of lung cancer in a large group of underground workers was investigated in two independent surveys, and in each the incidence was similar to that observed in a comparable age group of the population at large.

166. Yourt[103] found in dead-end drifts in a number of non-uranium hard-rock mines (gold mines) in Northern Ontario radon concentrations with a median in the range of 1.2×10^{-11} to 1.5×10^{-11} Ci/l.

167. No gross excess of lung cancer has been noted in the miners employed in non-uranium mines in Colorado, New York State or northern Ontario. However, there apparently has not been a study carried out to detect small significant increases in incidence.

168. Earlier calculations of radiation dose received by the bronchi of the Jachymov miners were made by Mitchell,[98] who concluded that the epithelial cells of the main bronchi received about 0.13 rad of alpha radiation in eight hours of exposure, and by Evans,[104] who calculated that the total dose to the bronchus during the duration of underground exposure (average seventeen years) amounted to about 600 rads.

169. Later, Bale,[95] taking into account the short-lived daughter products preformed in the radon-containing atmosphere, calculated the dose rate to the epithelial layer of bronchial tissue from 10^{-10} Ci Rn^{222}/l, 10^{-10} Ci RaA/l and 5×10^{-11} Ci RaC'/l as being 1.0 rad/40-hour week. In comparison with previous calculations of the dose from the same radon concentration, this value is about 2,000 times that predicted by Evans and Goodman[97] in 1940, about 300 times that predicted by Mitchell[98] in 1945, and about 100 times that predicted by Evans[104] in 1950.

170. De Villiers and Windish[105] reported that 23 of the 51 deaths (45 per cent) among miners with one or more years of underground experience in the fluorspar mining community of St. Lawrence, Newfoundland, during the ten-year period 1952-1961 were due to primary lung cancer, chiefly near the hilum of the lungs. A shift in the average age at death of the lung cancer cases to involve younger age groups, and an association between age at entry into risk and age at death, were also observed. The number of deaths expected as a result of malignant neoplasms of trachea, bronchus and lung among the total of 71 deaths from all causes in all St. Lawrence miners during 1952-1960, based on 157 such deaths among 15,264 deaths (less those among St. Lawrence miners) from all causes in males of Newfoundland, was 0.73. The 21 observed cases among the 71 miners dying of all causes was 28.8 times this expected number. The ratios for the four ten-year age groups between 25 and 64, inclusive, were 43.2 (25 to 34 years), 10.6 (35 to 44 years), 16.0 (45 to 54 years), and 8.0 (55 to 64 years). Considerable numbers of deaths from tuberculosis and pneumoconiosis were also found.

171. The most outstanding environmental finding in the mines at St. Lawrence has been the discovery of concentrations of radon and daughters in air, well in excess of suggested MPC levels. On the basis of these concentrations and other considerations, it was suggested that underground workers were probably exposed to an average potential alpha energy to complete decay of between 2.5 and 10 times the previously suggested working level of 1.3×10^5 Mev per litre of air for a forty-hour working week.[134] These levels were measured in mines in which no radio-active ore bodies had been found. The mine water was regarded as the source of the radon.

172. The more important findings relating to St. Lawrence, Jachymov and Schneeberg, and to the uranium mines of South Africa and the United States, are compared in table XX which gives data on radio-activity in various mines and the associated incidence of lung cancer. The incidence of lung cancer at St. Lawrence as a percentage of miner deaths ranges between 33.3 per cent (23 of 69 underground miner deaths 1933-1961) and 45.1 per cent (23 of 51 underground miner deaths 1952-1961).

173. Wagoner *et al.*[125] reported recently on a study of the cancer mortality pattern of a group of United States uranium miners and millers for the thirteen-year period 1950-1962 and compared their age-race-cause-

specific mortality experience with that of the general male population of the Colorado Plateau area. Among white uranium millers, total- and cause-specific mortality did not differ significantly from that expected. Among white uranium miners, 218 deaths were observed as compared with 148.7 expected ($P < 0.01$). Categories in which death significantly exceeded that expected were: (*a*) respiratory neoplasms among uranium miners with five or more years underground experience (11 observed as compared with 1.1 expected or a relative risk of 10); (*b*) "all other causes", in the same group of miners, a reflection of pulmonary fibrosis and its complications; and (*c*) accidents, particularly in mines, regardless of type of employment or duration of underground mining experience. The tenfold increase in respiratory cancer was not attributable to age, smoking, heredity, urbanization, self-selection, diagnostic accuracy, or prior hard-rock mining or other ore constituent, including silica dust. The evidence presently available implicates airborne radiation in the genesis of this increase in respiratory cancer.

174. The data available in miners from Jachymov, Schneeberg, the Colorado Plateau and the St. Lawrence region strongly suggest that cancer of the lung can be induced by inhaled radon and its daughters. Risk estimates cannot, however, be obtained from these groups because only the relative frequency of lung cancer among all cases of death is known, rather than the mortality from lung cancer in the population of miners.

IX. Over-all incidence of neoplasms after total-body irradiation

Japanese A-bomb survivors

175. The 1962 report discussed the report of Harada and Ishida[106] on the incidence of neoplasms among survivors of the Hiroshima A-bomb during the period April 1957-December 1958. The data were obtained from the tumour registry and were not based on the Atom Bomb Casualty Commission (ABCC) closed sample. The incidence of neoplasms varied inversely with distance from hypocentre. The "doubling dose" for cancer incidence was estimated as having been received at about 1,300 metres from hypocentre, where the dose was approximately 400 rads.[8]

176. Studies of the mortality of A-bomb survivors recently reported[107] show that in a subsample of 20,000 persons (ABCC Medical Subsample, Selection I, 1950-1958) there is thus far no evidence of a higher general mortality in the more heavily irradiated groups. When mortality from specific causes was studied, the well-known leukaemogenic properties of radiation were clearly reflected, but for no other causes were radiation effects seen. In this study the non-exposed group was deemed unsatisfactory as a control, at least for the periods in question, since it was characterized by abnormally low mortality in relation to both the exposed survivors and the Japanese population generally, and deaths from tuberculosis and cancer were notably deficient. Therefore, the control of this study relied on comparison of persons exposed at different distances from the hypocentre.

177. Zeldis *et al.*[57] reported recently on the Hiroshima and Nagasaki tumour registry study of cancer incidence covering a further twelve months in Hiroshima in addition to the twenty months already reported by Harada and Ishida[106] (May 1957-December 1959), and on the first thirty-six months in Nagasaki (April 1958-March 1960). Neoplasms diagnosed prior to the beginning of these studies were eliminated, and analyses were based on the more restricted, but known, Life-Span Study population rather than on the total city population.

178. In table XXI are shown crude incidence rates and age-sex-adjusted rates of malignancies (excluding leukaemia) per 100,000 persons. The indicated excess of malignancies in the most proximally exposed group in Hiroshima is not so large as that previously reported on a city-wide basis by Harada and Ishida,[106] but the gradient of incidence with exposure distance is statistically significant. In Nagasaki, with a considerably smaller number of collected cases, variations with exposure distance are not significant. For reasons not understood, the incidence in the non-exposed group in Nagasaki (particularly in males) is significantly greater than in exposed groups.

179. Figures now available[137] allow some comparison to be made between the number of cases of leukaemia and that of all other forms of cancer which may have been induced by radiation. During the years 1950-1959, 36 more deaths from leukaemia (standard error 6.4) occurred amongst the groups of people who had been exposed within 1,400 m of the hypocentre at Hiroshima and Nagasaki than was to be expected from the mortality amongst those exposed to lower doses of radiation at between 1,400 and 2,000 m. During the same period, 30 more deaths from other forms of malignant disease (standard error 13.6) occurred in the former group than were expected on the basis of mortality in the latter group. In this comparison, therefore, the number of deaths from all malignancies other than leukaemia, in excess of those in the comparison population, was 0.8 (standard error 0.4) times the excess number of deaths from leukaemia occurring in the same population and during the same period.

180. It seems probable that the mean latency for radiation-induced leukaemia is less than that for other forms of radiation-induced malignancy for which information is available and, therefore, that within the period of this survey (extending to thirteen years after irradiation) the proportion detected of all deaths that would finally occur would be greater for leukaemia than for other malignancies. For this reason the value of the ratio may be expected to rise somewhat in coming years and cannot yet be assessed accurately. Moreover, these data are based on mortality statistics and not on morbidity records, and so may not accurately reflect even the present incidence of disease. It already seems evident, however, that leukaemia (in acute forms and the chronic myeloid form), which normally accounts for only 2 per cent of all deaths from malignant disease, is increased in its incidence by radiation received under these circumstances, by a much larger factor than is the total of all other forms of malignant disease.

Populations exposed to high levels of environmental radiation

181. Gianferrari *et al.*[111] made a survey of births, deaths and other relevant variables in some communities of the Cervo Valley, Italy, where the background radiation is higher than normal (average total exposure of gamma radiation 15 R per thirty years), and in a nearby area similar geographically, socially and economically except for one-fifth (3 R per thirty years) of the background radiation level. The average uranium content (as U_3O_8) in various alimentary sources in the respective low background and high background areas was:

soil 1.0, 20.8 mg/kg; drinking water 0.06, 0.24 μg/l; vegetables 0.3 17.7 mg/kg; and fodder 0.4, 18.4 mg/kg. In the high radiation area the observed proportion of deaths from cancer was higher than expected in every age group. However, the increase was statistically significant only in the sixty-one to eighty-year age group.

CHILDREN EXPOSED *in utero* TO DIAGNOSTIC IRRADIATION

182. In the first reports of the retrospective study by Stewart *et al.*[30,31] of the relationship of pre-natal diagnostic X-ray exposure to subsequent development of leukaemia and other malignancies in children dying under ten years of age, it was found that more of the mothers of children dying of cancer had received abdominal X-ray examination during the relevant pregnancy than had mothers of control (living) children. Of the twenty mothers X-rayed in the first half of pregnancy, eighteen were mothers of children dying of cancer and only two were controls. The ratio of foetal irradiation in cancer cases to controls was 1.7 for deaths up to the age of four years and 2.5 for deaths from five to nine years of age.

183. The frequency of viral infections and threatened abortions was also significantly higher among the mothers of the dead children. X-ray exposures in infancy, acute pulmonary infections and severe injuries were three *post-natal* events significantly increased in children who subsequently died of leukaemia. Excessive maternal age was related to increased incidence of Down's syndrome and increased risk of leukaemia.

184. More recently, in an extension of the survey, Stewart[32] concluded that most childhood cancers and leukaemias were pre-zygotically determined, that the recent increase in incidence of childhood leukaemia was due to the pre-zygotic form, and that the maximum incidence of that form occurred earlier than the maximum incidence of pre-natal leukaemias.

185. MacMahon's prospective study[39] of this problem utilized a study population of 73,243 children born in the years 1947-1954 and the frequency of intra-uterine X-ray exposure estimated from a 1 per cent systematic sample. Abdominal or pelvic X-rays were recorded in 770 (10.6 per cent) of the 7,242 single pregnancies in the sample, and a total of 584 children born in the study sample who subsequently died of cancer before the end of 1960 were identified. There were 85 (15.3 per cent) of the 556 cancer deaths born of "single pregnancies" which had maternal abdominal or pelvic X-ray recorded, as compared with 770 of the 7,242 (10.6 per cent) single pregnancies in the whole 1 per cent samples ($P < 0.05$). After correction for indirect associations with birth order and other complicating variables, it was estimated that cancer mortality was about 40 per cent higher in the X-rayed than in the non-X-rayed members of the study population. This relationship held for each of the three major diagnostic categories: leukaemia, neoplasms of the central nervous system, and other neoplasms.

186. The excess cancer mortality in the X-rayed group was most marked at ages five to seven years, at which time the relative risk was 2.0. The excess risk was apparently exhausted by age eight years. A trend toward higher mortality in the more heavily exposed children was small and not statistically significant. No significant variation with stage of pregnancy at exposure was evident.

187. The most important determinant of the amount of pre-natal X-ray exposure appeared to be birth order. First births had three times as many exposures, and greater doses, than later births. There was little change with birth order after the first birth.

188. In MacMahon's study the ratio of 556 cancer deaths in 7,242 sample births indicates a total cancer mortality rate of $76.8/10^5$ single live births. Since in both the zero to four and the five to nine-year age groups for United States children born in 1950, leukaemia accounts for about half of all deaths, he regarded it as likely that the probabilities of dying from any neoplastic disease during these age intervals were approximately double the values for leukaemia. Applying these probabilities to the population of his study, he estimated that the study population would be expected to yield 388 cancer deaths in the first five years of life and 246 in the second five years (total 634). The observed numbers were 352 and 197, respectively (total 549). Eighteen other cancer deaths occurred at age ten years or older.

189. A deficit of 20 per cent of the expected deaths for the five to nine-year age group can be accounted for to some extent because the population has so far been followed for only three-fourths of the person-years necessary for complete ascertainment.

190. Using an indirect method of standardization, MacMahon calculated that the cancer mortality rate in the X-rayed population, adjusted for the several variables, was 10.31 per 10,000 live births. Using this value, he obtained a relative risk for all cancers of 10.31/7.8 or 1.42, compared to that of 1.52 without adjustment.

191. MacMahon, in estimating relative risk for malignancy from Stewart's data, excluding twins and taking into account Stewart's figure of 1.16 as the measure of bias in mothers reporting, and including all abdominal X-ray during the relevant pregnancy, derived a figure of 1.65, not very different from that derived in his study.

192. Stewart's data, however, indicate no appreciable decline in relative risk even at the highest ages included, whereas MacMahon's data show no increased risk after the age of seven years.

193. Recently, MacMahon and Hutchison[108] reviewed eleven published studies (to September 1, 1962) on the question of relative risk of malignant disease in children from exposure to X-ray *in utero*. The relative risks in five of these studies[21,36–38,109] were less than one, and in six studies[31,33–35,39,110] were greater than one. However, in view of the great overlap in the confidence limits of all eleven studies, they tested the possibility that all studies were consistent with a single risk value and found the maximum likelihood estimate of this common risk value by computing the weighted mean of the eleven observed relative risks. They concluded that there was no inconsistency in the findings of the eleven studies, and that the five studies reporting relative risks less than one all involved small samples with large expected sampling variability. The maximum likelihood estimate of the risk involved is 1.40, and the true value may be expected to be within the range of 1.20 to 1.64 ($P = 0.05$).

TABLE I. INCIDENCE OF LEUKAEMIA IN HIROSHIMA (1946–1962)
(Computed from data of Watanabe[6])

Onset year	Total population Hiroshima: Persons number	Total population Hiroshima: Cases No.	Total population Hiroshima: Cases 10^{-5}	Non-exposed population Hiroshima[a]: Persons number	Non-exposed population Hiroshima[a]: Cases No.	Non-exposed population Hiroshima[a]: Cases 10^{-5}	Exposed population Hiroshima (within 5 000 metres): Persons number	Exposed population Hiroshima (within 5 000 metres): Cases No.	Exposed population Hiroshima (within 5 000 metres): Cases 10^{-5}	Excess Cases 10^{-5}	Leukaemia deaths all Japan Cases 10^{-5}
1946.....	171,204	2	1.17	72,135	1	1.38	99,069	1	1.01	0.00	—
1947.....	222,434	6	2.70	123,607	1	0.81	98,827	5	5.06	4.25	1.07
1948.....	246,134	15	6.09	147,548	4	2.71	98,586	11	11.16	8.45	1.19
1949.....	262,832	21	7.99	164,498	9	5.47	98,334	12	12.20	6.73	1.37
1950.....	285,712[b]	13	4.55	187,610[b]	1	0.53	98,102[b]	12	12.23	11.70	1.47
1951.....	297,758	20	6.75	199,898	3	1.50	97,860	17	17.37	15.87	1.58
1952.....	321,973	17	5.28	224,355	8	3.56	97,618	9	9.22	5.66	1.67
1953.....	339,432	22	6.48	242,055	6	2.48	97,377	16	16.43	13.95	1.91
1954.....	361,367	17	4.70	264,232	4	1.51	97,135	13	13.38	11.87	2.12
1955.....	360,808	22	6.10	263,915	8	3.03	96,893	14	14.45	11.42	2.28
1956.....	382,011	18	4.71	285,360	5	1.75	96,651	13	13.45	11.70	2.41
1957.....	396,730	17	4.29	300,321	7	2.33	96,409	10	10.37	8.04	2.44
1958.....	412,707	21	5.09	316,539	10	3.16	96,168	11	11.44	8.28	2.65
1959.....	426,564	26	6.10	330,638	10	3.02	95,926	16	16.84	13.82	2.67
1960.....	431,285[b]	15	3.48	335,601[b]	11	3.28	95,684[b]	4	4.18	0.90	2.80
1961.....	459,301	22	4.79	363,859	10	2.75	95,442	12	12.63	9.88	—
1962.....	479,379	14	2.92	384,000	6	1.56	95,379	8	8.42	6.86	—
	TOTALS	288	83.19	TOTALS	104	40.83	TOTALS	184	189.84	149.38	

[a] Non-exposed population presumably includes early entrants after detonation.
[b] Figure from census for year indicated.

TABLE II. RISK OF LEUKAEMIA IN HIROSHIMA A-BOMB SURVIVORS[6]
(1946–1962, open population)

Metres from hypocentre	Estimated dose range[a] (rad)	Exposed population	Leukaemia cases	Cases 10^{-5} exposed	Ratio to expected incidence[b]
0–1,500	> 10,000–200	11,839	127	1,072	26.0
1,500–5,000	200– < 1	87,230	57	65	1.6
0–5,000	> 10,000– < 1	99,069	184	186	4.5

[a] Without correction for attenuation by shielding.
[b] The expected incidence is that observed in the period 1946–1962 in the population beyond 5,000 m., namely 41 × 10^{-5}.

TABLE III. AVERAGE INCIDENCE OF CONFIRMED LEUKAEMIA IN RESIDENTS OF HIROSHIMA AND NAGASAKI IN 12 YEARS (1947–1958) BY CITY OF EXPOSURE AND DISTANCE FROM HYPOCENTRE. ABCC MASTER SAMPLE
(Modified from Brill et al.[7])

Distance (metres) dose (rad)[a]	Hiroshima: No. leukaemia cases	Hiroshima: Man-y at risk 1947-1958	Hiroshima: Cases 10^{-6} Man-y at risk	Nagasaki: No. leukaemia cases	Nagasaki: Man-y at risk 1947–1958	Nagasaki: Cases 10^{-6} Man-y at risk
0–999 m. (1,400– > 10,000)	20	14,638	1,366	3	5,330	563
1,000–1,499 m. (200–1,400)	39	126,446	308	20	37,758	530
1,500–1,999 m. (30–200)	9	214,629	42	3	44,197	68
2,000–9,999 m. (< 1–30)	21	747,827	28	34	925,653	37
TOTAL	89	1,103,540	81	60	1,012,938	59

[a] Uncorrected for shielding.

TABLE IV. LEUKAEMIA INCIDENCE IN ABCC MASTER SAMPLE IN 9 YEARS (1950–1958) BY RADIATION DOSE (CORRECTED FOR LIGHT SHIELDING) IN HIROSHIMA AND NAGASAKI
(*Modified from Brill* et al.[7])

	Hiroshima				*Nagasaki*			
Dose in rads	*No. leukaemia cases*	*Man-y at risk 1950–1958*	*Cases 10^{-6} Man-y at risk*	*Ratio to expected incidence*[a]	*No. leukaemia cases*	*Man-y at risk 1950–1958*	*Cases 10^{-6} Man-y at risk*	*Ratio to expected incidence*[a]
> 1,280	5	3,204	1,561	78	0	387	0	—
641–1,280	10	9,999	1,000	50	3	1,341	2,237	112
321–640	5	7,623	656	33	2	2,043	979	49
161–320	7	21,888	320	16	4	6,408	624	31
81–160	7	37,278	188	9	6	12,681	473	24
41–80	3	48,798	61	3	0	11,565	0	—
21–40	2	48,402	41	2	1	9,981	100	5
0–20[b]	12	547,839	22	1	9	217,782	41	2
TOTAL	51	725,031	70	3.5	25	262,188	95	4.8

[a] The expected incidence is the estimated yearly incidence in Japan in the period 1950–1958, namely 2×10^{-6}.
[b] Includes A-bomb survivors exposed between 2,000 and 10,000 metres.

TABLE V. INCIDENCE OF LEUKAEMIA (1950–1962) AMONG EARLY ENTRANTS INTO HIROSHIMA AFTER A-BOMB EXPLOSION
(*Modified from Watanabe*[6])

	Cases entered within 3 days	*Cases entered 3–7 days after*	*Cases entered 7–14 days after*	*Cases entered within 2 weeks*
Population	25,799	11,001	7,326	44,126
No. of leukaemias developed	27	5	7	39
Incidence per 100,000 per year[a]	8.05	3.50	7.35	6.79

[a] Average yearly incidence of leukaemia for 13 years (1950–1962) in non-exposed population (beyond 5,000 metres) in Hiroshima, computed from data of Watanabe,[6] was 2.34×10^{-5}.

TABLE VI. INCIDENCE OF DEATH FROM LEUKAEMIA IN PHYSICIANS [16]

Occupation	*Time*	*Place*	*Total deaths*[a]	*Leukaemia deaths*	*Incidence*	*Ratio of incidences radiologists: all physicians*	*General population incidence*	*Reference*
Radiologist	1929–43	USA	175	8	4.57%			March[114]
Non-radiologist	1929–43	USA	50,160	221	0.44%	10.3:1		
All physicians	1933–42	USA	26,788	143	0.53%		0.39%	Henshaw and Hawkins[116]
Radiologist	1938–42	USA	95	5	5.30%			Dublin and Spiegelman[117]
All specialists	1938–42	USA	2,029	19	0.94%	10.6:1		
All physicians	1938–42	USA	12,419	62	0.50%			
Radiologist	1944–48	USA	124	6	4.84%			March[115]
Non-radiologist	1944–48	USA	15,637	113	0.72%	6.7:1		
All physicians	1947–51	USA	11,481	133	1.20%		0.52%	Peller and Pick[118]
Radiologist	1949–58	USA	296	11	3.71%			Cronkite[121]
All physicians	1949–58	USA	23,393	221	0.77%	4.8:1		
Radiologist	1897–56	G. Brit. Eire	463	3	0.65%			Court Brown[113]
Radiologist	1938–42	USA	205	8	3.90%			Ulrich[119]
Non-radiologist	1938–42	USA	34,626	158	0.44%			
Radiologist	1952–55	USA			3.57%	3.6:1		Melville in Schwartz and Upton[112]
Non-radiologist	1952–55	USA			1.00%			
Radiologist	1930–54	USA			3.65%		0.39%	Warren[120]
Non-radiologist with X-ray	1930–54	USA			2.33%		(1950)	
Non-radiologist without X-ray	1930–54	USA			0.63%			

[a] With known diagnosis.

TABLE VII. MORTALITY AMONG RADIOLOGISTS: DEATHS ATTRIBUTED TO CANCERS OF THE LYMPHATIC AND BLOOD-FORMING TISSUES AND FROM APLASTIC ANAEMIA. ONLY DEATHS OCCURRING BETWEEN THE AGES OF 35 AND 74, INCLUSIVE, IN THE 14-YEAR PERIOD, 1948 TO 1961, ARE INCLUDED [9]

International code rubric	Principal disease	Number of deaths: Observed	Number of deaths: Expected	P[a]	Mortality ratio (M.R.)	95% Confidence M.R.
200	Lymphosarcoma	4[b]	2.4	> .05	1.7	0.5 to 4.3
201	Hodgkin's disease	1	1.6	> .05	0.6	0.02 to 3.5
202, 205	Lymphoblastoma	1	0.38	> .05	2.6	0.07 to 14.6
203	Multiple myeloma	5	1.01	.004	5.0	1.6 to 11.6
204	Leukaemia	12	4.02	.001	3.0	1.5 to 5.2
292.4	Aplastic anaemia	4	0.23	.0001	17.0	4.7 to 44.5

[a] Probability that the observed number of deaths, or a larger number, would occur by chance.
[b] Includes two deaths from lymphosarcoma, one from reticulum cell sarcoma, and one from malignant lymphoma.

TABLE VIII. NUMBER OF MEN RECEIVING THERAPEUTIC RADIATION TO THE SPINAL MARROW BY 31/12/54, AND MAN-YEARS AT RISK FOLLOWING EACH LEVEL OF EXPOSURE THROUGHOUT THE PERIOD OF OBSERVATION: STUDY SERIES

Mean exposure to spinal marrow (roentgen)	No. of men exposed by 31/12/54	No. of man-years at risk following exposure	Mean exposure to spinal marrow (roentgen)	No. of men exposed by 31/12/54	No. of man-years at risk following exposure
Less than 250	1,153	8,184	1,750–1,999	305	1,550
250–499	1,708	10,339	2,000–2,249	172	939
500–749	1,912	10,126	2,250–2,499	118	509
750–999	2,268	11,654	2,500–2,749	44	283
1,000–1,249	2,124	10,632	2,750 or more[a]	45	151
1,250–1,499	938	5,098			
1,500–1,749	500	2,437	TOTAL	11,287	61,902

[a] Average exposure—3,043 R.

TABLE IX. THE NUMBERS OF PATIENTS WHO DEVELOPED LEUKAEMIA, AND THE CRUDE AND STANDARDIZED INCIDENCE RATES, AFTER DIFFERENT MEAN EXPOSURES TO THE SPINAL MARROW: MALE "A" AND "B" CASES, EXCLUDING CO-EXISTENT CASES [12]

	Mean exposure to spinal marrow (R)													
	0[a]	Less than 250	250–499	500–749	750–999	1,000–1,249	1,250–1,499	1,500–1,749	1,750–1,999	2,000–2,249	2,250–2,499	2,500–2,749	2,750 or more	Total
No. of men developing leukaemia														
"A" cases	—	1	2	6	3	7	2	3	1	2	3	1	1	32
"A" and "B" cases	—	1	3	6	4	8	3	3	1	2	4	1	1	37
Crude incidence per 10,000 men per year														
"A" and "B" cases	0.49	2.16		4.59		6.99			12.18			63.65		5.98
Standardized incidence per 10,000 men per year														
"A" and "B" cases	0.49	1.98		4.66		7.21			14.44			72.16		5.98

[a] The rate given for "zero" therapeutic exposure is the corresponding rate among men of the same age-distribution and observed over the same period, calculated from the mortality from leukaemia experienced by the whole male population of Britain.

TABLE X. THE INCIDENCE OF LEUKAEMIA AFTER DIFFERENT MEAN EXPOSURES TO THE SPINAL MARROW: MALE "A" AND "B" CASES GIVEN ONLY SPINAL IRRADIATION, EXCLUDING COEXISTENT CASES [18]

	Mean exposure to spinal marrow (R)									
	0	*Less than 250*	*250–499*	*500–749*	*750–999*	*1,000–1,249*	*1,250–1,499*	*1,500–1,749*	*1,750–1,999*	*2,000 or more*[a]	*Total*
No. of man-years at risk following exposure	—	5,404	7,673	6,573	8,262	7,411	2,782	897	566	679	40,247
No. of men developing leukaemia											
"A" cases	—	0	2	4	3	4	0	2	1	1	17
"A" and "B" cases	—	0	2	4	3	5	0	2	1	1	18
Crude incidence per 10,000 men per year											
"A" and "B" cases	0.49		1.53		4.72		6.75[b]		8.12[c]		4.47
Standardized incidence per 10,000 men per year											
"A" and "B" cases	0.49		1.44		4.83		6.82[b]		8.70[c]		4.47

[a] Average exposure, 2,290 R.

[b] For the group receiving 1,000–1,499 R the crude incidence is 4.91; standardized incidence 5.06. For the group receiving 1,000–1,749 R the crude incidence is 6.31; standardized incidence 6.82.

[c] For the group receiving 1,500 R or more the crude incidence is 18.68; standardized incidence 19.86. For the group receiving 1,750 R or more the crude incidence is 16.07; standardized incidence 16.82.

TABLE XI. OBSERVED AND EXPECTED NUMBERS OF LEUKAEMIA CASES IN CHILDREN IRRADIATED FOR "THYMIC ENLARGEMENT"
(*Modified from Pifer* et al.[27])

Series group	*Series I*		*Series II*	
	Treated	*Siblings*	*Treated*	*Siblings*
Number of persons	1,451	2,073	1,358	2,256
Average age (yrs.) at observation	18.5	—	8.1	—
Leukaemia cases (total)	6	0	0	1
Cases expected[a] (local)	0.96	1.27	0.51	0.87
Ratio obs./expected	6.25	—	—	1.15
Mean exposure (R) for cases	336 (150–684)	—	—	—
Mean exposure for series	329	—	126	—
Cases < 200 R	4	—	0	—
Cases > 200 R	2	—	0	—

[a] Cases expected on the basis of records for upstate New York, the area in which the children were treated.

TABLE XII. RELATIVE LEUKAEMIA RISK IN RETROSPECTIVE STUDIES OF CHILDREN DYING OF LEUKAEMIA AFTER DIAGNOSTIC IRRADIATION *in utero*

Reference	*Age (years) of leukaemics at death*	*Years of death for leukaemics*	*Percentage of mothers receiving abdominal irradiation during pregnancy*		*Relative risk, 95% limits in brackets*
			Leukaemics	*Controls*	
Stewart[32]	< 10	1953–55	96/780 (12.3%)	117/1,638 (7.1%)	1.8 (2.4–1.4)
Ford *et al.*[33]	< 10	1951–55	20/70 (28.6%)	48/247 (19.4%)	1.7 (2.9–0.8)
Kaplan[34]	?	1955–56	37/150 (24.7%)	24/150 (16.0%)	1.7 (3.7–1.0)
Kaplan[34]	?	1955–56	34/125 (27.2%)	27/125 (21.6%)	1.4 (2.5–0.7)
Polhemus and Koch[35]	?	1950–57	72/251 (28.7%)	58/251 (23.1%)	1.3 (2.0–0.9)
Kjeldsberg[36]	?	1946–56	5/55 (9.1%)	8/55 (14.5%)	0.6 (2.0–0.2)
Murray *et al.*[21]	< 20	1940–57	3/65 (4.6%)	3/65 (4.6%)	1.0 (12.0–0.6)
Murray *et al.*[21]	< 20	1940–57	3/65 (4.6%)	7/93 (7.5%)	0.6 (2.4–0.1)
Murray *et al.*[21]	< 20	1940–57	3/65 (4.6%)	2/82 (2.4%)	1.9 (40.0–1.1)

TABLE XIII. THYROID TUMOURS IN AUTOPSIES AND SURGICAL THYROID SPECIMENS IN JAPANESE A-BOMB SURVIVORS ACCORDING TO DISTANCE FROM HYPOCENTRE
(*Hiroshima ABCC, 1948–1960*[57])

	Metres from hypocentre			
	< 1,400	*1,400–1,999*	*2,000–9,999*	*Non-exposed*
Number of autopsies	124	188	397	544
No. and per cent carcinoma	7 (5.6%)	3 (1.6%)	12 (3.0%)	15 (2.7%)
No. and per cent adenoma	9 (7.2%)	7 (3.7%)	18 (4.5%)	15 (2.7%)
Number of surgical specimens	35	35	—	—
No. and per cent carcinoma	15 (42.8%)	4 (11.4%)	—	—
No. and per cent adenoma	5 (11.1%)	10 (28.6%)	—	—

TABLE XIV. RELATIVE RISKS FOR THYROID CANCER AT VARIOUS EXPOSURE LEVELS AFTER THERAPEUTIC IRRADIATION (EXTERNAL SOURCES) TO NECK REGION
(*Computed from data of Takahashi* et al.[58])

Estimated exposure (roentgen)	*Proportion of thyroid cancer cases* %	*Proportion of controls* %	*Relative risk, 95% limits in brackets*
0	95.45 (609)	99.43 (4,044)	—
500–2,000	0.94 (6)	0.25 (10)	4.0 (1.2–13.2)
2,000–4,000	2.04 (13)	0.25 (10)	8.6 (4.3–15.4)
4,000–6,000	0.78 (5)	0.05 (2)	16.6 (3.1–89.0)
6,000–8,000	0.47 (3)	0.02 (1)	19.9 (2.0–200.0)
8,000–10,000	0.16 (1)	—	—
> 10,000	0.16 (1)	—	—

TABLE XV. AGES AT IRRADIATION AND AT OPERATION, LATENT INTERVAL, AND THYROID EXPOSURE IN 7 CASES OF THYROID CANCER FOLLOWING RADIO-THERAPY FOR TUBERCULOUS ADENITIS [59]

Cases	*Age at irradiation years*	*Age at cancer operation years*	*Interval years*	*Approximate exposure (roentgen)*
1	4	17	13	1,500 (rt. lobe) 1,000 (isthmus)
2	9	27	18	700 (both lobes)
3	15	26	11	500–700 (rt. lobe)
4	18	45	27[a]	500 + (over 8 years)
5	18	42	24	650 (rt. lobe)
6	23	40	17	1,000 (each lobe)
7	34	44	10	700 (rt. lobe)
Means	17.3	34.4	17.1	

[a] From first radiation treatment.

TABLE XVI. THYROID CANCER FOLLOWING THERAPEUTIC IRRADIATION

Author	Age at irradiation	Man-y at risk	Average exposure (roentgen)	Cases	Cases× 10^{-6}/y	Risk estimate ($c \times 10^{-6}$/y/R)
Conti[28]	Children	21,896	168	0	0	0.0 (0.0–1.1)[a]
De Lawter[74]	Adults	5,000	2,100	0	0	0.0 (0.0–0.03)
Hanford[59]	Children, Adults	5,711	900[b]	8	1,400	1.6 (0.7–3.1)
Latourette[32]	Children	15,130	214	1	66	0.3 (0.01–1.7)
Pifer[37], Series I	Children	26,843	329	8	298	0.9 (0.4–1.8)
Pifer[37], Series II	Children	11,000	126	1	91	0.7 (0.01–4.0)
Saenger[34]	Children	24,871	330	11	442	1.3 (0.9–2.3)
Simpson[60,c]	Children	18,829	520[d]	10	531	1.0 (0.5–1.9)

[a] In brackets, approximate 95% confidence limits of the estimate.
[b] Mean exposure to largest group (65%).
[c] Partly overlaps Pifer's Series I.
[d] Mean exposure to cases developing cancer.

TABLE XVII. RELATIVE RISKS FOR NECK CANCER (EXCLUDING THYROID) AT VARIOUS EXPOSURE LEVELS AFTER THERAPEUTIC IRRADIATION (EXTERNAL SOURCES) TO NECK REGION
(*Computed from data of Takahashi* et al.[58])

Estimated exposures (roentgen)	Proportion of cancer cases %	Proportion of controls %	Relative risk, 95% limits in brackets
0	98.79 (895)	99.43 (4,044)	—
500–2,000	0.33 (3)	0.25 (10)	1.5 (0.4–16.0)
2,000–4,000	0.66 (6)	0.25 (10)	2.7 (0.9–7.0)
4,000–6,000	0.11 (1)	0.05 (2)	2.2 (0.2–24.0)
6,000–8,000	0.11 (1)	0.02 (1)	4.5 (0.3–74.0)

TABLE XVIII. RELATIVE RISKS FOR SKIN CANCER AT VARIOUS EXPOSURE LEVELS AFTER THERAPEUTIC IRRADIATION (EXTERNAL SOURCES)
(*Computed from data of Takahashi* et al.[58])

Estimated exposures (roentgen)	Proportion of cancer cases %	Proportion of controls %	Relative risk, 95% limits in brackets
0	95.45 (294)	99.43 (4,044)	—
500–2,000	0.97 (3)	0.25 (10)	4.1 (1.2–9.6)
2,000–4,000	0.97 (3)	0.25 (10)	4.1 (1.2–9.6)
4,000–6,000	0.65 (2)	0.05 (2)	13.7 (1.8–100.0)
6,000–8,000	0.65 (2)	0.02 (1)	27.4 (2.5–300.0)
8,000–10,000	0.97 (3)	—	—
> 10,000	0.32 (1)	—	—

TABLE XIX. LONG-TERM EFFECTS OF RADIUM DEPOSITION IN MAN (CORRELATION OF CLINICAL AND RADIOGRAPHIC FINDINGS WITH CURRENT BODY BURDENS OF Ra^{226})
Hasterlik et al.[82]

Body content (μCi)	*Number radiographed*	*Radiographic changes in skeleton attributable to radium deposition*					
		None	*Minimal*	*Mild*	*Moderate*	*Advanced*	*Malignant*
<0.001	17	14	3	0	0	0	
0.001–0.01	28	28	0	0	0	0	
0.01–0.1	90	80	8	1	1	0	
0.1–1.0	61	25	13	9	9[a]	5[b]	3
> 1.0	40	1	2	5	4	28[c]	14
TOTAL	236	148	26	15	14	33	17

[a] MsTh present in two cases.
[b] Severe tooth changes only in one case.
[c] Based on film taken elsewhere in two cases.

TABLE XX. COMPARISON OF ST. LAWRENCE, JACHYMOV AND SCHNEEBERG, COLORADO PLATEAU AND SOUTH AFRICA DATA [105]

	Fluorspar Mines		*Uranium Mines*			
	St. Lawrence		*Jachymov & Schneeberg*		*Colorado Plateau Working mines*	*South Africa Working mines*
	Non-working areas	*Working areas*	*Abandoned mine*	*Working mine*		
Radon (picocuries per litre)						
Average				2,900		
Range	270–25,000[a]	5–1,510	?–59,000	?–18,000	70–59,000	25–500
Radon daughters (multiples of 1.3×10^8 Mev per litre per working week)						
Average	53	2.5–10[b]				
Range	4.2–193	0–12				
Gamma radiation (mR/h)	0.03–0.50					
Incidence of lung cancer as % of miner deaths	33 (1933–1961)		43 (1875–1939)[c]			
	45 (1952–1961)		52 (1921–1939)[c]		11.4[d]	3.5[c]
Duration of underground exposure (years)						
Average and range	12.5 (5.5–21.3)		17 (13–23)[f]		7, 8, 9, 10, 12	17.3 (3–30)[h]
Induction period (years)						
Average and range	19.1 (11.5–25.0)		25 (15–43)[f]			
Age at death (years)			50 (40–67)[f]			
Average and range	46.8 (33.56)		55 (37–69)[g]			58.2 (45–73)

[a] Calculated on basis of highest radon daughter concentration found.
[b] Estimated.
[c] After Lorenz (1944).[122]
[d] Miners with 3 or more years underground experience—5 cases—Archer and others (1962).[123]
[c] Based on an autopsy series—23 cases among 650 autopsies—Oosthuizen and others (1958).[102]
[f] Nine cases—Pirchan and Sikl (1932).[94]
[g] Thirteen cases—Rostoski and others (1926),[93] as quoted by Lorenz (1944).[122]
[h] Based on 14 of 23 cases reported by Oosthuizen and others (1958).[102]

TABLE XXI. INCIDENCE OF MALIGNANT NEOPLASMS EXCLUDING LEUKAEMIA, HIROSHIMA AND NAGASAKI TUMOUR REGISTRIES, 1957–1959 (SEXES AND AGES COMBINED) [57]

	Distance from hypocentre in metres				
	500–1,499	*1,500–2,499*	*2,500–9,999*	*Non-exposed*	*Total*
Hiroshima (32 months)					
No. of cases	79	183	151	120	533
Crude rate per 10^5	978.1	829.2	773.4	625.1	774.0
Age- and sex-adjusted rate	953.3	819.7	758.6	650.1	774.0
Nagasaki (30 months)					
No. of cases	19	47	29	36	131
Crude rate per 10^5	635.9	608.1	475.5	697.1	596.0
Age- and sex-adjusted rate	846.4	667.1	600	915.2	735.4

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ANNEX C

LIST OF REPORTS RECEIVED BY THE COMMITTEE

1. This annex lists reports received by the Committee from Governments and agencies of the United Nations.

2. All those reports are included of which a sufficient number of copies for distribution in the A/AC.82/G/L. document series were received between 10 March 1962 and 3 July 1964, inclusive.

3. The first 750 reports received by the Committee are listed in annex I of its first comprehensive report[a] and annex J of the second.[b]

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[a] *Official Records of the General Assembly, Thirteenth Session, Supplement No. 17* (A/3838).

[b] *Ibid., Seventeenth Session, Supplement No. 16* (A/5216).

Document No.	Country and Title
A/AC.82/G/L.	
	BRAZIL
751	A survey on artificial radio-active contamination in Brazil (1958-1962).
	SWEDEN
752	The increase of gamma radiation from the ground during September 1st-November 30th 1961.
753	Some observations of variations of the natural background radiation.
754	Distribution and excretion of radiocesium in goats, pigs and hens.
755	Report on observations made at Kiruna Geophysical Observatory during the series of nuclear weapon tests carried out at Novaja Zemlja between 10 Sept. and 4 Nov. 1961.
756	Determination of strontium-90 and stable strontium in bones from sheep, ewes and fetuses.
757	Cesium-137 deposition in Sweden 1958-1960 and the 1961 spring maximum in air.
758	The radio-activity in air and precipitation during autumn 1961.
759	Internal doses from mixed fission products.
760	A fall-out model II. Some quantitative properties.
761	Behaviour of yttrium-91 and some lanthanons towards serum proteins in paper electrophoresis, density gradient electrophoresis and gel filtration.
	UNITED KINGDOM
762	Radioactivity in milk. Interim report, Dec. 1961.
	JAPAN
763	Japanese dietary habits and the fall-out problem.
764	Environmental contamination with short-lived radio-nuclides in Japan in 1961.
	UNITED STATES OF AMERICA
765	Radiological Health Data, Volume III, Number 2, February 1962.
766	Health and Safety Laboratory fall-out program quarterly summary report, April 1, 1962. HASL-122.
	INTERNATIONAL ATOMIC ENERGY AGENCY
767	Survey of radio-activity in food consumed in Austria. Report on second sampling period — January-June 1961.
	SWEDEN
768	The effect of radiostrontium on mouse testes.

Document No.	Country and Title
A/AC.82/G/L.	
	NORWAY
769	Assay of strontium-90 in human bone in Norway 1956-1961.
	ITALY
770	Data on environmental radioactivity, collected in Italy (January-June 1961).
	UNITED KINGDOM
771	Interim report on radio-activity in diet. ARCRL-7.
	UNITED STATES OF AMERICA
772	Atmospheric radio-activity at Washington, D.C., 1950-1961. NRL-5764.
773	Radiological Health Data, Volume III, Number 4, April 1962.
774	Radiological Health Data, Volume III, Number 3, March 1962.
775	Radio-active fall-out from nuclear weapons tests, Nov. 15-17, 1961. Book 1.
776	Radio-active fall-out from nuclear weapons tests, Nov. 15-17, 1961. Book 2.
777	Health implications of fall-out from nuclear weapons testing through 1961.
778	Radiological Health Data, Volume III, Number 5, May 1962.
	UNITED KINGDOM
779	Assay of strontium-90 in human bone in the United Kingdom. Results for 1961, part I.
	SWEDEN
780	Doses to the gastrointestinal tract from ingestion of fission products in drinking water or food.
781	Internal doses from fission products in milk.
782	Simulation of an atmospheric transport problem.
783	Studies in reversal autoradiography.
784	Strontium-90 induced bone and bone-marrow changes.
785	Sr^{90} induced osteosarcomas.
786	Uptake and retention of strontium-90 in mouse tissues studied by whole animal autoradiography and impulse counting.
787	II. Uptake and retention of strontium 90 in strontium-90-induced osteosarcomas.
788	III. Dissemination of metastases from a strontium-90-induced transplanted osteosarcoma investigated by whole-body autoradiography.
789	Effects of radiostrontium on the blood and haematopoietic tissues of mice.
790	Histogenesis of Sr^{90}-induced osteosarcomas.

Document No.	Country and Title
A/AC.82/G/L.	
	UNITED STATES OF AMERICA
791	Measurement of bone marrow and gonadal dose from X-ray examinations of the pelvis, hip and spine as a function of field size, tube kilovoltage and added filtration.
792	Strontium-90 in man and his environment —Volume I: Summary.
793	Fall-out program quarterly summary report, July 1, 1962. HASL-127.
794	Survey of fall-out operations, July 1, 1962. HASL-128.
	BELGIUM
795	La retombée radioactive à Mol. Rapport d'avancement — ler semestre 1961.
796	Evolution du ^{90}Sr dans les sols et les végétaux: analyse des résultats obtenus en 1959.
	UNITED STATES OF AMERICA
797	Radiological Health Data, Volume III, Number 6, June 1962.
	ISRAEL
798	Early radio-active fall-out in Israel following the 1961 nuclear weapon tests.
	UNITED STATES OF AMERICA
799	Terrestrial and freshwater radioecology: A selected bibliography.
800	Radiological Health Data, Volume III, Number 8, August 1962.
	SWITZERLAND
801	5. Bericht der Eidgenössischen Kommission zur Überwachung der Radioaktivität zuhanden des Bundesrates für das Jahr 1961.
801/ Corr. 1	Corrigendum to above document.
	DENMARK
802	Environmental radio-activity in Denmark, 1961.
	UNITED STATES OF AMERICA
803	Health and Safety Laboratory fall-out program quarterly summary report, October 1, 1962. HASL-131.
804	Radiological Health Data, Volume III, Number 9, September 1962.
805	Radiation standards including fall-out. Summary analysis of Hearings held on June 4, 5, 6, and 7, 1962, before the Subcommittee on Research, Development, and Radiation of the Joint Committee on Atomic Energy, Congress of the United States, September 1962.
	UNITED KINGDOM
806	Annual report, 1961-1962. ARCRL-8.
	UNITED STATES OF AMERICA
807	Medical survey of Rongelap people seven years after exposure to fall-out. BNL-727 T-260.

Document No.	Country and Title
A/AC.82/G/L.	
	ARGENTINA
808	Contaminación por I^{131} debida al fall-out.
	ITALY
809	Data on environmental radio-activity collected in Italy (July-December 1961). BIO/06/62.
	NORWAY
810	Fall-out in Norwegian milk in 1961.
	UNITED STATES OF AMERICA
811	Radiological Health Data, Volume III, Number 11, November 1962.
812	Radiological Health Data, Volume III, Number 10, October 1962.
813	Radiation standards, including fall-out. Hearings before the Subcommittee on Research, Development, and Radiation of the Joint Committee on Atomic Energy, Congress of the United States, 87th Congress, 2nd session. Parts 1 and 2 (Appendix).
814	Fallout program quarterly summary report, January 1, 1963. HASL-132.
	BELGIUM
815	La retombée radioactive à Mol. Rapport d'avancement — second semestre 1961.
	SWEDEN
816	Distribution of radioruthenium in mice.
	UNITED STATES OF AMERICA
817	Radiological Health Data, Volume III, Number 12, December 1962.
	BRAZIL
818	Environmental radio-activity in high background areas of Brazil.
	UNITED KINGDOM
819	Assay of strontium-90 in human bone in the United Kingdom. Results for 1961, part II.
	UNITED STATES OF AMERICA
820	Radiological Health Data, Volume IV, Number 1, January 1963.
821	Factors influencing strontium-90 in milk from the Brainerd, Minn. milkshed.
822	Strontium-90 on the earth's surface II. Summary and interpretation of a world-wide soil sampling program: 1960-1961 results.
	UNITED KINGDOM
823	Radio-active fall-out in air and rain results to the middle of 1962.
	UNITED STATES OF AMERICA
824	Bioenvironmental features of the Ogotoruk Creek area, Cape Thompson, Alaska. TID-17226.
825	Radiological Health Data, Volume IV, Number 2, February 1963.

Document No.	Country and Title
A/AC.82/G/L.	
	SWEDEN
826	Distribution of plutonium in mice. An autoradiographic study.
827	Radioaktivitestsmätningar pa livsmedel 1962.
	UNITED STATES OF AMERICA
828	Radionuclides and ionizing radiation in ornithology. TID-17762.
	UNITED KINGDOM
829	Interim report on radio-activity in milk. ARCRL-9.
	SWEDEN
830	Genetic effects of strontium-90 injected into male mice.
	UNITED STATES OF AMERICA
831	Health and Safety Laboratory fallout program quarterly summary report, April 1, 1963. HASL-135.
832	Intercalibration of some systems employed in monitoring fission products in the atmosphere. NRL-5850.
833	Atmospheric radio-activity and fall-out research. TID-12616 (Rev. 1).
	SWITZERLAND
834	Messung des Natürlichen Strahlenpegels in der Schweiz.
	UNITED STATES OF AMERICA
835	Rapid methods for estimating fission product concentrations in milk.
836	Radiological Health Data, Volume IV, Number 3, March 1963.
	ITALY
837	Data on environmental radio-activity collected in Italy (January-June 1962). BIO/26/62.
	SOUTH AFRICA
838	Radio-active fall-out, its dispersion, deposition over South Africa and biological significance.
	UNITED STATES OF AMERICA
839	Radiological Health Data, Volume IV, Number 4, April 1963.
840	Radiological Health Data, Volume IV, Number 5, May 1963.
841	Studies of the fate of certain radionuclides in estuarine and other aquatic environments.
842	Health and Safety Laboratory fallout program quarterly summary report, July 1, 1962. HASL-138.
843	Estimates and evaluation of fall-out in the United States from nuclear weapons testing conducted through 1962.

Document No.	Country and Title
A/AC.82/G/L.	
	UNITED KINGDOM
844	Assay of strontium-90 in human bone in the United Kingdom, results for 1962, Part I with some further results for 1961.
	INDIA
845	Estimates of biospheric contamination and radiation dose from nuclear explosions up to April 1962. AEET/AM/27.
846	Gamma activity of the food samples in India after the autumn 1961 tests of nuclear weapons. AEET/AM/32.
	USSR
847	Определение возраста продуктов деления по измерению суммарной площади фотопика $Zr^{95}+Nb^{95}$ со временем.
	UNITED STATES OF AMERICA
848	Review of the army food irradiation program. Hearings before the Joint Committee on Atomic Energy, Congress of the United States, 88th Congress, May 13, 1963.
849	Fall-out, radiation standards, and countermeasures. Hearings before the Subcommittee on Research, Development, and Radiation of the Joint Committee on Atomic Energy, Congress of the United States, 88th Congress, 1st session, June 3, 4, and 6, 1963. Part I.
850	Radiological Health Data, Volume IV, Number 7, July 1963.
851	Radiological Health Data, Volume IV, Number 6, June 1963.
852	The treatment of radiation injury. NAS-NRC Publ. 1134.
853	Radiological Health Data, Volume IV, Number 8, August 1963.
	CANADA
854	Iodine-131 content of Canadian milk during 1961. AECL-1797.
	UNITED KINGDOM
855	Radio-activity in drinking water in the United Kingdom—1962 results.
	UNITED STATES OF AMERICA
856	Health and Safety Laboratory fall-out program quarterly summary report. HASL-140.
	SWEDEN
857	Genetic effects of strontium-90 on immature germ-cells in mice.
	UNITED KINGDOM
858	Annual report 1962-1963. ARCRL-10.
	UNITED STATES OF AMERICA
859	Radiological Health Data, Volume IV, Number 9, September 1963.

Document No.	Country and Title
A/AC.82/G/L.	
	UNITED KINGDOM
860	The weathering of caesium-137 in soil. AERE-R-4241.
	UNITED STATES OF AMERICA
861	Mathematical programming models for selection of diets to minimize weighted radionuclide intake.
862	Terrestrial and freshwater radioecology. A selected bibliography (Suppl. 1). TID-3910 (Suppl. 1).
	INDIA
863	Levels of cerium-141 and cerium-144 in surface fallout samples collected at Bombay during the monsoon months of 1962.
	UNITED KINGDOM
864	Radio-active fall-out: Short-lived fission products in air and rain, August 1962-April 1963. AERE-R-4384.
865	Assay of strontium-90 in human bone in the United Kingdom, results for 1962, part II.
	AUSTRALIA
866	Iodine-131 levels in milk in Australia during period May-November 1962.
	DENMARK
867	Genetically significant radiation doses in diagnostic radiology.
	UNITED STATES OF AMERICA
868	Radiological Health Data, Volume IV, Number 10, October 1963.
869	Radiological Health Data, Volume IV, Number 11, November 1963.
	WORLD HEALTH ORGANIZATION
870	Radiation hazards in perspective (3rd report of the Expert Committee on Radiation).
	UNITED STATES OF AMERICA
871	Health and Safety Laboratory fallout program quarterly summary report, January 1, 1964. HASL-142.
872	Iodine-131 in fresh milk and human thyroids following a single deposition of nuclear test fall-out. TID-19266.
	INDIA
873	Estimation of zirconium-95 deposited by fallout during September 1961 to February 1962. AEET/AM/31.
	UNITED KINGDOM
874	Radio-active fall-out in air and rain. Results to the middle of 1963. AERE-R-4392.
	AUSTRALIA
875	Strontium-90 in the Australian environment during 1961.

Document No.	Country and Title
A/AC.82/G/L.	
	FRANCE
876	Doses d'exposition au cours d'examens radiologiques per-opératoires. SCPRI-55.
877	Surveillance de la radioactivité sur le territoire français de 1961 à 1963. SCPRI-98.
878	Radioactivité des eaux de la Durance.
879	Accroissement du tritium atmosphérique dû aux explosions thermonucléaires.
880	Mesures de la radioactivité naturelle dans la région parisienne.
881	Mesure de la radioactivité d'origine artificielle dans l'hémisphère Sud.
882	De la dynamique des transports des aérosols radioactifs au-dessus de la France.
883	Mesure de l'activité de l'air due au krypton 85.
884	Essai d'analyse de la radioactivité de l'éponge "Hercinia variabilis".
885	Identification directe des nuclides radioactifs dans l'eau de mer par spectrographie gamma.
886	Spectrométrie gamma *in situ* des eaux de la Méditerranée occidentale.
	UNITED STATES OF AMERICA
887	Radiological Health Data, Volume V, Number 1, January 1964.
888	Fall-out, radiation standards, and countermeasures. Hearings before the Subcommittee on Research, Development and Radiation of the Joint Committee on Atomic Energy, Congress of the United States, 88th Congress, 1st session, August 20, 21, 22, and 27, 1963. Part 2.
	NORWAY
889	A study of Sr^{90} and Cs^{137} in Norway 1957-1958. Intern rapport K-253.
	ITALY
890	Data on environmental radio-activity collected in Italy (July-December 1962). BIO/03/63.
	UNITED STATES OF AMERICA
891	Neoplasms in children treated with X-rays for thymic enlargement. I. Neoplasms and mortality.
891/ Add. 1	Neoplasms in children treated with X-rays for thymic enlargement. II. Tumor incidence as a function of radiation factors.
891/ Add. 2	Neoplasms in children treated with X-rays for thymic enlargement. III. Clinical description of cases.
892	Distribution and retention of Cs^{137} after accidental inhalation. UAC-8383.

Document No.	Country and Title
A/AC.82/G/L.	
	UNITED STATES OF AMERICA (*continued*)
892/ Corr. 1	Correction to above report.
893	Radiological Health Data, Volume IV, Number 12, December 1963.
	SWITZERLAND
894	6. Bericht der Eidg. Kommission zur Uberwachung der Radioaktivität, zuhanden des Bundesrates für das Jahr 1962.
	CZECHOSLOVAKIA
895	Prediction of individual differences in postirradiation regeneration of the lymphatic tissue of the thymus.
	USSR
896	Стронций-90 в воде пресных водоемов Советского Союза в 1961-1962 годах.
897	Содержание цезия-137 в поверхностных водах Атлантического океана и его морей в 1961 году.
898	Изучение распределения изотопов церия, рутения, циркония и ниобия в процессе разложения минеральными кислотами проб радиоактивных выпадений и атмосферных осадков.
899	Определение стронция-90 в водах Атлантического океана в 1961 году.
900	Поведение цезия-137 в ходе анализа проб атмосферных осадков.
901	Аккумуляция радиоактивного стронция пресноводными растениями.
902	Выпадение Ce^{141}, Ce^{144}, $Zr^{95}+Nb^{95}$ и Sr^{90} на территории СССР во второй половине 1962 года.
903	Радиохимическая методика массового контроля содержания стронция-90 в пресных водоемах.
904	Стронций-90 в поверхностных водах Индийского океана в 1960-1961 годах.
905	Сравнительный анализ наблюдаемых планетарных распределений содержания озона и некоторых радиоактивных изотопов в атмосфере.
906	Концентрация суммарной радиоактивности продуктов деления, а также Ce^{141}, Ce^{144}, $Zr^{95}+Nb^{95}$ и Cs^{137} в приземном слое воздуха в подмосковье в 1960-1962 годах.
907	Стронций-90 в водах Тихого океана.
908	Некоторые данные по концентрации радиоактивных продуктов над Индийским и Тихим океанами в 1961-1962 годах.
909	Содержание стронция-90 в Черном море в 1959-1961 годах.
910	Содержание стронция-90 в молоке отдельных районов СССР в 1960-1961 годах и в первой половине 1962 года.

Document No.	Country and Title
A/AC.82/G/L.	
	USSR (*continued*)
911	Природная радиоактивность океанской воды.
912	Искусственная радиоактивность приземного слоя атмосферы на о. Хейса земли Франца-Иосифа в 1960-1961 годах.
913	О загрязнении растительности стронцием-90 и цезием-137 в 1961 году.
914	Радиоактивные выпадения близ Симеиза в 1960-1961 годах.
915	Исследование состава рутения-103 и рутения-106 в смеси продуктов деления методом бета-гамма-совпадений сцинтилляционных спектрометров.
916	Выпадение стронция-90 на поверхность территории СССР в четвертом квартале 1961 года и первой половине 1962 года.
917	Статистика появления «горячих» частиц в приземном слое воздуха в Подмосковье в 1961-1962 годах.
918	Стронций-90 в воде бассейна р. Дон.
919	Вопросы глобального радиоактивного загрязнения вод Мирового океана.
	INDIA
920	Active particles in fallout from nuclear weapon tests. AEET/AM/34.
	JAPAN
921	Radio-activity survey data in Japan, No. 1.
922	Deposition of Sr^{90} and Cs^{137} in Tokyo through the end of July 1963.
923	Leukaemia and ionizing radiation in Japan. An epidemiological survey.
924	X-ray microanalysis of highly radioactive fall-out particles.
925	Fractionation phenomena in highly radioactive fall-out particles.
926	The increase in induced mutation frequency after fractionated irradiation of gonial cells of the silkworm.
	UNITED KINGDOM
927	Interim report: Radio-activity in milk, 1963. ARCRL-11.
	UNITED STATES OF AMERICA
928	Radiological Health Data, Volume V, Number 2, February 1964.
	INDIA
929	Deposition of iodine-131 from the nuclear weapon tests in the Pacific during 1962. AEET/AM/35.
930	Airborne cerium-141 and cerium-144 at Bombay during November 1961-February 1963. AEET/AM/36.

Document No.	*Country and Title*
A/AC.82/G/L.	
	MEXICO
931	Determinación del estroncio 90 en leches y aguas de lluvia.
	UNITED ARAB REPUBLIC
932	Fall-out and radio-active content of the food chain in U.A.R. during the year 1962.
933	Fall-out and radio-active content of certain food items in U.A.R. during the first quarter of 1963.
	SWEDEN
934	A review of measurements of radioactivity in food, especially dairy milk, and a presentation of the 1963 data on Cs^{137} and Sr^{90}.
	UNITED STATES OF AMERICA
935	Radiological Health Data, Volume V, Number 3, March 1964.
936	Fall-out program quarterly summary report, April 1, 1964. HASL-144.
937	Radiological Health Data, Volume V, Number 4, April 1964.
	UNITED KINGDOM
938	Assay of strontium-90 in human bone in the United Kingdom. Results for 1963, part I.

Document No.	*Country and Title*
A/AC.82/G/L.	
	UNITED STATES OF AMERICA
939	Environmental radiation measurements in the South-eastern, Central and Western United States, 1962-1963. HASL-145.
	SWEDEN
940	The radio-activity of watches.
941	Physics, chemistry and meteorology of fall-out.
	UNITED STATES OF AMERICA
942	Radiological Health Data, Volume V, Number 5, May 1964.
	SWEDEN
943	Protective effect of cysteamine at fractionated irradiation.
944	The behavior of certain lanthanons in rats.
945	Note on the increase of gamma radiation from the ground during September 1st-November 30th, 1961.
946	Some observations of variations of the natural background radiation.
947	Gamma radiation from the ground in Sweden during 1960-1963.
	UNITED ARAB REPUBLIC
948	Fall-out and radio-active content of food chain in U.A.R. during the year 1963.

APPENDIX I

LIST OF SCIENTIFIC EXPERTS, MEMBERS OF NATIONAL DELEGATIONS

The scientific experts who took part in the preparation of the present report while attending Committee sessions as members of national delegations are listed below.

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