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

GENERAL ASSEMBLY

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Chapter I

INTRODUCTION

Constitution and terms of reference of the Committee

1. The 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 is composed 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 sixteen sessions. Its activities during the first fourteen sessions were surveyed in the introductions to the reports that the Committee submitted to the General Assembly in 1958, 1962 and 1964.[1]

4. The Committee held its fifteenth session at the European Office of the United Nations in Geneva from 15 to 23 November 1965. During that session the Committee discussed new information on natural radiation sources and on radio-active contamination of the environment as well as on certain biological effects of ionizing radiation on the basis of reviews prepared in the Secretariat.

5. The Committee also adopted its annual progress report to the General Assembly (A/6123). In that report the Committee expressed its intention of preparing for submission to the General Assembly at its twenty-first session a substantive report dealing with such estimates of risk as might result from consideration of the subjects mentioned in paragraph 4 of this chapter.

6. The General Assembly considered both the 1964 and 1965 reports of the Committee during its twentieth session. By resolution 2078 (XX) of 18 December 1965, the General Assembly: (1) took note of the reports of the United Nations Scientific Committee on the Effects of Atomic Radiation on the work of its thirteenth, fourteenth and fifteenth sessions; (2) commended the Scientific Committee for its valuable contributions to wider knowledge and understanding of the effects and levels of atomic radiation during the ten years of the Committee's existence; (3) requested the Scientific Committee to continue its programme, including its co-ordinating activities, to increase the knowledge of the levels and effects of atomic radiation from all sources; (4) commended the World Meteorological Organization for its work in carrying forward the scheme for monitoring and reporting levels of atmospheric radio-activity; (5) acknowledged with appreciation the assistance rendered to the Scientific Committee by the World Meteorological Organization, the Food and Agriculture Organization of the United Nations and the International Atomic Energy Agency; (6) recommended that all parties concerned continue their co-operation with the Scientific Committee; (7) noted the intention of the Scientific Committee to submit a report to the General Assembly at its twenty-first session; (8) requested the Secretary-General to continue to provide the Scientific Committee with the assistance necessary for the conduct of its work and the dissemination of its findings to the public.

[1] *Official Records of the General Assembly, Thirteenth Session, Supplement No. 17* (A/3838); *ibid., Seventeenth Session, Supplement No. 16* (A/5216); *ibid., Nineteenth Session, Supplement No. 14* (A/5814). Hereafter these documents will be referred to as the 1958, 1962 and 1964 reports, respectively.

7. The sixteenth session of the Committee was held at Headquarters from 6 to 17 June 1966. At that session the Committee adopted the present report to the General Assembly. It also considered the problem of the effects of ionizing radiation on the central nervous system. The Committee decided that it should at its future meetings consider in detail the effects of ionizing radiation on the nervous system, biological indicators of irradiation of man, and the principles, procedures and parameters used by it in estimating doses to the population from global radio-active contamination of the environment. In its consideration of the last topic, the Committee proposed to review the requests it had made in the past to States Members of the United Nations or members of the specialized agencies and of the IAEA for data on levels of environmental contamination. The above subjects, together with a further review of reported levels of environmental global contamination, might form the substance of a report or reports to the General Assembly. The Committee also decided to request that arrangements be made for a session to be held in 1967.

Organization of the work of the Committee

8. As in the past, most of the technical discussions were held during informal meetings of groups of specialists whose conclusions were eventually reviewed by the full Committee. According to the Committee's established practice, no detailed record of its technical deliberations was taken.

9. Mr. D. J. Stevens of Australia and Dr. A. R. Gopal-Ayengar of India served as Chairman and Vice-Chairman, respectively, during the fifteenth session of the Committee. At the fifteenth session, Dr. A. R. Gopal-Ayengar of India and Dr. G. C. Butler of Canada were elected Chairman and Vice-Chairman, respectively, to serve during the sixteenth and seventeenth sessions. The names of those scientists who attended the fifteenth and/or the sixteenth sessions of the Committee as members of national delegations are listed in appendix I.

Sources of information

10. The reports received by the Committee 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, between 15 June 1964 and 7 June 1966, are listed in annex D. Reports received prior to 15 June 1964 were listed in earlier reports of the Committee to the General Assembly. The information received officially by the Committee was supplemented by, and interpreted in the light of, information available in the current scientific literature or obtained from unpublished private communications from individual scientists.

Scientific assistance

11. 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.

12. 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

13. Sessions of the Committee were attended by observers 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), and from the International Atomic Energy Agency (IAEA), as well as from the International Commission on Radiological Protection (ICRP) and the International Commission on Radiation Units and Measurements (ICRU). The Committee wishes to record its appreciation for their contribution to the discussions.

Scope and purpose of the report

14. Like the 1964 report, the present report is not intended to cover comprehensively the whole field of interest of the Committee; in particular, the report will not deal with medical irradiation nor with somatic radiation effects. The report is limited to a discussion of environmental radiation, both natural and artificial, and of the genetic risks arising from exposure to ionizing radiation. The Committee has surveyed especially those aspects of both subjects in which new advances may require a revision of its earlier assessments of radiation risks. The present report, being neither comprehensive nor self-contained, must be read in the context of the earlier reviews made by the Committee in its 1962 and 1964 reports.

15. The main text of the report is followed by technical annexes where the scientific information on which the Committee rests its conclusions is discussed in 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

ENVIRONMENTAL RADIATION

Radiation from natural sources

1. The interest of the Committee in radiation from natural sources arises from the fact that living beings have been exposed to it for a very long time at a relatively constant rate. Because of this constancy of the average dose rate from natural radiation to which human populations have been exposed, these dose rates are used by the Committee as a standard against which population doses from other sources are compared for the purpose of risk estimation. It is of importance, therefore, that the estimates of dose rates from natural radiation should be kept under review.

2. Natural radiation owes its origin to interactions of primary cosmic rays from outer space with the atmosphere, and to the radio-active decay of naturally-occurring radio-isotopes.

Cosmic rays

3. The interactions of primary cosmic rays with the atmosphere give rise to secondary rays which contribute about one-third of the external natural radiation reaching the human body. Higher contributions from both primary and secondary cosmic rays apply at very high altitudes; the resulting dose rates have been studied in connexion with the planning of supersonic transport and of space flights, but they will not be considered in the present report.

4. The major advances in the study of cosmic rays as contributors to the natural radiation to which man is exposed have been made with regard to their neutron component. Recent data on cosmic-ray neutron flux densities show that the dose-rate estimate of about two millirads per year to the world population made in the 1962 report needs revision. The Committee now believes the dose rate due to neutrons at sea level to lie between 0.3 and 1.1 millirads per year. This range reflects the uncertainties involved in measurements and the variation of neutron flux densities with latitude.

5. No change is called for in the estimate of the dose rates due to the other (so-called ionizing) components of cosmic rays—28 millirads per year—that was accepted in the 1962 report. As was mentioned in that report, dose rates approximately double every 1,500-metre increase in altitude for the first few kilometres.

6. It must be mentioned that neutron doses are more effective than doses of ionizing radiation in bringing about biological effects. To obtain estimates of risk from cosmic-ray neutrons, allowance must be made for their relative biological effectiveness. However, the necessary weighting factors applying to neutrons as compared to the other components are not known at low dose rates, although they are frequently assumed to have a value of ten. Even with such a high weighting factor, the contribution from neutrons would still be small compared to the total dose rate from natural sources.

Radiation from the earth's crust

7. Terrestrial radio-activity contributes both to natural radiation reaching the human body from outside, owing to the emission of penetrating gamma radiation, and to that arising internally from radio-active nuclides which decay within the organism with the emission of alpha, beta or gamma rays.

External irradiation

8. The Committee has reviewed the dose rates from naturally-occurring external radiation and considers that there is no reason to change its view as expressed in the 1962 report, namely that, subject to wide geographical variations, the average external dose rate from naturally-occurring radio-active nuclides to which the world population is exposed is about 50 millirads per year, allowing for the fraction of time spent indoors and outdoors.

9. In some areas, however, the soil and the underlying rocks contain abnormally high amounts of radio-active material. In some high radiation areas where sizable populations live, external dose rates up to twenty times higher than average have been reported.

Internal irradiation

10. Radio-active material in soil may either be absorbed by plants or leached into water, and so may enter the human food chain and eventually be ingested by man. Radon, a radio-active gas resulting from the radio-active disintegration of nuclides of the uranium and thorium series, escapes from soils and rocks into the atmosphere, and can thus be inhaled together with its radio-active daughters.

11. The major natural sources of internal radiation are potassium-40, which delivers relatively uniform dose rates to the whole body, and members of the uranium and thorium series which predominantly irradiate the bone and bone marrow. Carbon-14 and rubidium-87 are among other nuclides which deliver much smaller dose rates.

12. The estimates of dose rates to gonads and to bone and blood-forming cells from internally deposited radionuclides, expressed in millirads per year, are essentially the same as in the 1962 report. In that report, however, dose rates were expressed in different units to take into account the higher efficiency of alpha particles in producing biological effects when compared to gamma rays. As in the case of cosmic-ray neutrons, it seems more appropriate to express dose rates in millirads per year, since allowing for the relative biological effectiveness of alpha particles would require information that is not available now and would therefore involve largely arbitrary assumptions.

13. The Committee has re-evaluated the dose rates from naturally-occurring radio-active material to the

lung tissues. Such material reaches the lungs mainly through inhalation of the daughter products of radon. These daughter products are inhaled in particulate form and therefore tend to be deposited on the walls of alveoli and bronchi and to remain there long enough for significant doses to be delivered. The dose rates to the cells lining these cavities seem to be of the order of some hundreds of millirads per year, although no exact figure can, at present, be given. These are the highest tissue dose rates received from natural radiation. Any biological significance that these dose rates may have, however, is still unknown.

Dose-rate estimates

14. Dose rates from natural radiation are summarized in table I. They have been computed for the gonads, irradiation of which gives rise to genetic effects, for cells lining the inner surface of bone from which bone tumours may arise, and for blood-forming cells, the irradiation of which may result in leukaemias. The average dose rate in the whole body is taken as equal to that to the gonads.

15. The figures in the table must be considered as average dose rates received by the world population. It has not been possible to assess accurately the variability of the dose rates received by different population groups. Those limited populations, however, which live in subarctic regions and consume large amounts of caribou and reindeer meat or of fresh-water fish may receive somewhat higher doses to blood-forming cells and to cells lining the internal surface of bone. Similarly, populations living in the high-radiation areas of Brazil and India receive higher dose rates of external radiation from the soil.

Radiation from man-made sources

16. Nuclear tests are the main source of present world-wide radio-active contamination of the environment. Low activity wastes released from facilities using nuclear technologies for industrial, medical and research purposes contribute a negligible fraction of the doses received by human populations from artificial sources, though their significance may increase in the future as a consequence of the increased use of nuclear energy in human activity. Accidents at nuclear establishments have been only of local importance.

17. The unplanned re-entry into the atmosphere in April 1964 of a spacecraft carrying a power source containing plutonium-238 resulted in the dispersion of this radio-active material. This material is slowly descending towards the ground and has now been detected in surface air at some sampling stations in the southern hemisphere. It is expected that the average amounts of plutonium from this source that may be inhaled in the coming years will remain exceedingly small, and will give rise to negligible radiation exposures.

18. The atmospheric tests that were carried out in central Asia in 1964 and 1965, and those underground tests from which leakage of radio-active material into the atmosphere has taken place, have not contributed significantly to world-wide mean doses. A further atmospheric test took place in May 1966; although no detailed evaluation is yet possible, it appears that the quantity of fission products released was very small compared with the total quantity produced by all previous tests.

19. Results of measurements of radio-activity in the stratosphere, which constitutes the main reservoir of radio-active debris still available for world-wide deposition, and estimates of the total amount of artificial radio-activity so far deposited over the surface of the globe lead to estimates of current and expected contamination of land areas which are the same as, or only slightly lower than, those made by the Committee in its 1964 report.

20. Increasing but conflicting evidence indicates that higher amounts of radio-active debris fall into the oceans than were assumed in the past. However, this does not influence greatly the prediction of future land deposition, since only relatively small amounts of radio-active material still remain in the stratosphere. The estimate of sea deposition relative to land deposition is, in fact, mainly of interest for predictions of the fate of material located in the stratosphere. The somewhat higher radio-activity deposition over the oceans does not affect the estimates of doses due to intake of seafood, since the previous estimates were based upon direct measurements of radio-activity in food.

21. The Committee has reviewed the current information on body contents of strontium-90 and caesium-137 in the world population and on dietary levels of these radio-active nuclides, and has concluded that no change in the method of calculation of dose commitments from strontium-90 appears warranted at this time. There are, of course, still considerable uncertainties in the numerical factors used in the calculation of dose commitments.

22. New evidence indicates that the factors used to calculate the long-term contamination of diets by strontium-90 contained in the soil are probably too high and hence the dose commitments from strontium-90 listed in table II may be over-estimates. The numerical factors used in the calculation of the internal dose commitments from caesium-137 have been somewhat increased taking into account new information. As a consequence, these dose commitments are slightly higher than those given in the 1964 report.

23. With regard to external doses from artificial radio-activity deposited on to the ground, the Committee has modified its methods of calculating the external dose commitment from gamma emitters. There is no significant change in the numerical values obtained, but the new methods follow the actual processes more closely.

24. Estimates of the average dose commitments already received and to be received by the world population by the year 2000 from all tests carried out to the end of 1965 are summarized in table II. These estimates differ little from those made in 1964. The fraction of the total dose commitment which is attributable to external sources ranges from about two-thirds for gonads to one-fifth for cells lining bone surfaces.

25. Appreciable variations of dose are found in different parts of the world. A particular situation is that prevailing in the arctic and subarctic regions of Alaska (United States), Canada, the Scandinavian countries and the Soviet Union, where sizable populations consume large amounts of caribou and reindeer meat. As these animals graze over land areas and feed on lichens that derive their nutrients mainly from atmospheric dusts, their meat contains high concentrations of radio-active nuclides, particularly caesium-137. As mentioned

in paragraph 15, a similar food chain mechanism explains that these same populations are also exposed to higher levels of internal natural radiation.

Conclusions

26. The Committee has re-evaluated the contributions to the exposure of human populations from natural radiation (annex A) and from radio-active contamination of the environment by past nuclear weapon tests (annex B). Estimates of comparative risks have also been reviewed. Comparative risks are expressed, as in the 1964 report, in terms of the periods of time during which natural radiation would have to be doubled to give a dose increase equal to the total doses expected by the year 2000 from the current contamination of the environment due to past nuclear weapon tests.

27. These periods do not differ appreciably from those given in the 1964 report. Present estimates are approximately three-quarters of a year for the gonads, two and a half years for the cells lining bone surfaces and one year and a half for the bone marrow. These values present a certain degree of approximation since they are based on assumptions and measurements which may not be entirely representative of the whole world situation. They are more likely to be over- rather than under-estimates.

TABLE I. DOSE RATES DUE TO EXTERNAL AND INTERNAL IRRADIATION FROM NATURAL SOURCES IN "NORMAL" AREAS

Source of irradiation	*Dose rates (mrad/y)*			*Paragraphs in annex A*
	Gonads	*Cells lining bone surfaces*[a]	*Bone marrow*	
External irradiation				
Cosmic rays				
Ionizing component	28	28	28	48
Neutrons	0.7	0.7	0.7	49
Terrestrial radiation (including air)	50	50	50	58
Internal irradiation				
K^{40}	20	15	15	136
Rb^{87}	0.3	< 0.3	< 0.3	136
C^{14}	0.7	1.6	1.6	136
Ra^{226}	—	0.6	0.03	135–139
Ra^{228}	—	0.7	0.03	135–139
Po^{210}	0.3	2.1	0.3	135–139
Rn^{222} (dissolved in tissues)	0.3	0.3	0.3	135–139
TOTAL[b]	100	99	96	
Percentages from alpha particles and neutrons	1.3	4.4	1.4	

[a] The dose rates under this heading were actually calculated for the Haversian canals of bone. Doses to cells lining bone surfaces may be somewhat lower than those quoted here.

[b] Totals have been rounded off to two significant figures.

TABLE II. DOSE COMMITMENTS FROM NUCLEAR EXPLOSIONS

Tissue	*Source of radiation*	*Dose commitments (mrad) for period of testing 1954-1965*[a]	*Paragraphs in annex B*
Gonads	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Cs^{137}	15	145
	C^{14}[b]	13	147
	TOTAL[c]	76	
Cells lining bone surfaces	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Sr^{90}	156	143
	Cs^{137}	15	145
	C^{14}[b]	20	147
	Sr^{89}	0.3	146
	TOTAL[c]	240	

TABLE II. DOSE COMMITMENTS FROM NUCLEAR EXPLOSIONS (*continued*)

Tissue	*Source of radiation*	*Dose commitments (mrad) for period of testing 1954-1965*[a]	*Paragraphs in annex B*
Bone marrow	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Sr^{90}	78	143
	Cs^{137}	15	145
	C^{14}[b]	13	147
	Sr^{89}	0.15	146
	TOTAL[c]	150	

[a] As in its 1962 and 1964 reports, the Committee has based its evaluation of comparative risks due to past nuclear tests on dose commitments to the gonads, to the cells lining bone surfaces and to the bone marrow. The dose commitment is the total dose that will be delivered, as a world population average, to the relevant tissues during the complete decay of radioactive material introduced into the environment. Some of the doses included in the dose commitments may be delivered over a very long period of time.

[b] As in the 1964 report, only the doses accumulated up to the year 2000 are given for carbon-14; at that time, the doses from the other nuclides will have essentially been delivered in full. The *total* dose commitment to the gonads due to carbon-14 from tests up to the end of 1965 is about 180 millirads.

[c] Totals have been rounded off to two significant figures.

Chapter III

THE GENETIC RISKS OF IONIZING RADIATION

1. Radiation damage to the genetic material may take two forms: gene mutations and chromosome anomalies. Gene mutations result in an alteration of the elementary units of information that make up the genetic message received by the progeny from their parents, whereas chromosome anomalies involve the loss, duplication or rearrangement of minor or major parts of the same message. It will be recalled from the 1962 report that the elementary units of genetic information are called genes and that they are linearly arrayed along nuclear structures called chromosomes.

2. Both gene mutations and chromosome anomalies occur for reasons usually not ascertainable in populations not unduly exposed to radiation. As in the past, the Committee has reviewed information on both the spontaneous incidence of genetic changes in the general population and on the induction of those changes by radiation. The advances in genetics and cytology made in the last few years have made it possible for the Committee not only to review its earlier estimates of the risk of induction of gene mutations, but also to reconsider the risk of induction of a few chromosome anomalies.

Natural incidence of mutations in man

Gene mutations

3. Gene mutations are believed to occur at a rate of approximately one in seven gametes (mature germ cells) per generation in males and possibly at a lower rate in females (C23).[2] The great majority of these continually arising mutations are harmful in various degrees and, by failing sooner or later to be transmitted to the following generations, are eliminated from the population at a rate related to their harmfulness. Failure of transmission may occur through death of the cell carrying the mutation, through lack of fertilization, or lack of implantation of the fertilized egg in the maternal organism, all events that pass practically unnoticed. It may also occur through processes involving hardship, such as miscarriages or peri-natal mortality, as well as reduction of fertility associated with physical or mental defects of all shades of severity. There is no way to tell at present whether the elimination of mutants occurs predominantly through events of limited social consequence or by processes associated with major sufferings.

4. It is, however, possible to estimate the frequency of those mutations that give rise to various severe and well-known disabilities and which, being dominant, become manifest in the generation immediately following the one in which they have arisen. The total rate of mutations responsible for these disabilities appears to be between one and two mutations per 10,000 gametes per generation (C9). Therefore, of all the spontaneous mutations, only one in 1,000 is a dominant mutation associated with a clearly identified hereditary disability recognizable at birth. Many more, not necessarily dominant, mutations are probably associated with disabilities less easily identifiable as genetic in origin.

Chromosome anomalies

5. Chromosome anomalies consist of changes in the number or in the structure of chromosomes. Two categories of chromosomes are recognized—autosomes and sex-chromosomes. With the exception of mature germ cells, human cells contain twenty-two pairs of autosomes and one pair of sex-chromosomes. The two members of each of the twenty-two autosomal pairs are morphologically identical regardless of the sex of the subject to which the cell belongs; the sex-chromosomes in each pair, on the other hand, are identical in females but not in males.

6. The first anomaly that was described in man involved the presence of a specific extra autosome. This anomaly is associated with a severe clinical condition called Down's syndrome (mongolism). Other extra chromosomes were described subsequently. These anomalies have always been associated with grave disabilities. The frequency of children with extra autosomes is about two per 1,000 live-born children (C42).

7. Changes in the number of sex-chromosomes, including loss of a chromosome, are also known. The syndromes associated with these changes are detected in about three per 1,000 live-born children (C51). Though less severe in their effects than extra autosomes, changes in the number of sex-chromosomes are responsible for serious clinical syndromes and are usually associated with sterility.

8. Alterations of structure and numbers of chromosomes appear to occur with equal frequency, but small structural rearrangements probably escape detection because they may affect the individual only slightly and may be difficult to recognize cytologically. Two types of structural rearrangements can be easily detected in man—translocations and deletions. Both autosomes and sex-chromosomes can be affected.

9. Translocations consist of exchanges of fragments between non-identical chromosomes. One survey gave a frequency of translocations of five per 1,000 adults (C46). When the whole of the chromosomal material is present in the cell, even though arranged in a different order as a consequence of a translocation, the anomaly is called balanced, and the individual that carries it is usually normal. During the reshuffling of the chromosomes that takes place in the course of the maturation of germ cells, unbalanced translocations, characterized by deficiency or excess of chomosome material, may arise. Individuals with unbalanced translocations may live, but only with severe handicaps.

10. Deletions are losses of part of a chromosome. Those that have been identified are associated with

[2] Throughout this chapter, references to paragraphs of annex C are indicated by the letter C followed by the corresponding number.

severe syndromes. Their total frequency in the population cannot yet be estimated. One type of deletion appears to occur with frequency of at least two per 10,000 live-born children (C45).

Summary

11. Between 2 and 3 per cent of all live-born children are affected by one of the disabilities mentioned in paragraph 4 or by detectable chromosome anomalies. In addition, about 4 per cent of all pregnancies terminate in miscarriage associated with a chromosome anomaly (C53). Genetic changes occurring naturally must also be responsible for a number of other detrimental consequences, but, in the present state of our knowledge, we are unable to identify them as being genetic in origin, and their frequency is therefore difficult to estimate.

Risk of induction of genetic changes by radiation

12. Gene mutations can be induced by ionizing radiation. This has been shown experimentally in so many animal and plant species that there is no reason to doubt that they can be induced in man. On the other hand, chromosome changes have been proved to arise following irradiation in human somatic cells. The great majority of the radiation-induced genetic changes are harmful, but the damage that they entail extends over a wide range of severity. Some changes have scarcely noticeable consequences; others may be incompatible with reproduction or survival.

13. Clear evidence of genetic damage in the offspring of irradiated human subjects is, however, meagre. The only effect that has been reported is a change of the sex-ratio in the offspring of irradiated individuals. Such an effect, though probably genetic in origin, is difficult to interpret, and the observations are of little use in predicting other genetic consequences of radiation damage.

14. There is no alternative therefore to using results obtained with experimental animals in estimating rates of induction in man. The limitations of such a procedure are obvious when it is realized that animal species differ from each other in their susceptibility to the induction of genetic changes by radiation and that there is no evidence indicating whether the genetic material of man is more or less sensitive to radiation than that of other animal species. The only mammal which has been studied in some detail with respect to radiation genetics is the mouse. Results of mouse experiments must therefore form the main basis for the assessment of genetic risks in man.

15. Most of the experimental data were obtained with immature germ cells, which are also the cells that accumulate most of the genetic damage induced in germ cells. The estimates given in paragraphs 16-23 apply to acute single doses of x or gamma rays. For each of them it will be indicated whether the numerical values refer to mature germ cells (gametes) or to immature ones.

Risk of gene mutations

16. The over-all risk of induction of gene mutations, as based on rates of induction in the mouse at acute high doses, is estimated by the Committee to be two mutations per 1,000 male gametes per rad (C256). As discussed later, the rate of induction of mutations is much less when radiation is delivered at a lower dose rate. It may be recalled from chapter II that man receives from natural sources about one-tenth of a rad per year to the gonads or about three rads in a reproductive lifetime.

17. Induced mutations are similar in nature to those discussed in paragraph 3. Generally harmful, they are eliminated from the population at a rate depending upon their harmfulness, but we are unable at present to determine to what extent the elimination takes place through practically unnoticed events rather than through events that involve individual or collective hardship.

18. It would be desirable to know the risk of induction of that part of the total induced damage that is expressed through those disabilities which are easily detected and are known to occur spontaneously with a measurable frequency in human populations (paragraph 4). To obtain such an estimate it is necessary to make certain assumptions. Depending on the assumptions made, the resulting estimates differ by several orders of magnitude (C264). Observations in mice show that a number of serious skeletal abnormalities can be induced in the offspring of animals irradiated at high doses. The yield of abnormalities is not known at low doses, but the observations may in the future give a clue to a more precise estimation of risks of induction of dominant traits in man.

19. The particular importance of dominant mutations lies in the fact that, once induced, they become apparent in the offspring of the irradiated individuals, and each of these mutations will persist for a number of generations depending on the detriment to which it gives rise. It must be emphasized, however, that this category of induced mutations represents only part of the total damage due to induced gene mutations and that the elimination of perhaps a large fraction of the rest may also involve considerable hardship.

Risk of chromosome anomalies

20. Data on the induction of chromosome anomalies in mice are scantier than on the induction of gene mutations but can be supplemented by data obtained from the irradiation of human somatic cells grown outside the organism. The limitations of this latter material as a basis for estimating rates of induction in man arise from the fact that the anomalies induced in these cells may not be transmitted at cell division in the same way as if they had been induced in immature germ cells within the body.

21. Loss of a sex-chromosome can be induced in the mouse at a rate of one to four losses for 100,000 immature male germ cells per rad (C278). In man, loss of a sex-chromosome is known to be one of the most frequent among the chromosome anomalies that are associated with spontaneous miscarriages. There is no way to assess at present the rate of induction of extra sex-chromosomes or autosomes. Preliminary information indicating an increased incidence of Down's syndrome in the offspring of irradiated individuals needs to be confirmed.

22. Estimates of rates of induction of translocations in man can be obtained on the basis of experiments both with mice and with human somatic cells grown *in vitro*. The rise of the frequency of translocations is not expected to be proportional to the dose but to depend on it in a complicated manner that does not permit a simple expression of risks. It may, however, be said that the rate of induction after one rad is of the order of one translocation in every 200,000 im-

mature male germ cells (C286). At higher doses, the number of translocations induced is higher than would be expected if the frequency of induction was linearly related to the dose increase.

23. The rate of induction of those deletions that have so far been observed to occur spontaneously in man can be estimated on the basis of *in vitro* experiments on human somatic cells. The estimates, however, depend so much on the assumptions about the mechanism that brings about deletions that the figures obtained differ widely according to the particular theory which is adopted (C293, 294).

Conclusions

24. The Committee has considered genetic effects of radiation, with particular regard to recent data, and has tried to derive from them information as to the importance of genetic effects of irradiation of man.

25. A new estimate has been obtained for the spontaneous frequency of gene mutations over the whole of the hereditary material of man. An estimate has also been made of the rate of induction of gene mutations per unit of radiation dose. From these it would appear that a dose of one rad per generation would add something like one-seventieth to the total number of mutations arising spontaneously in a generation. Taking into account the various uncertainties, the range of that estimate would be very wide, but it is probably not in disagreement with the limits set in the 1962 report of between one-tenth and one one-hundredth. It is known that the great majority of all harmful mutations are expressed as small reductions of viability over intra-uterine and post-natal life, and their effects on health are detectable with difficulty in man. However, it is known that the cumulative effect of these small changes causes the major part of the damage from induced mutations. Furthermore, these changes will be expressed over many generations.

26. The proportion of one-seventieth above might also apply to hereditary diseases of man which are known to be important and which can be transmitted directly from parent to offspring, but it should be emphasized once more that these diseases contribute only a small proportion of the damage from gene mutations. There is evidence that complexly inherited characteristics, such as stature and intelligence, may be affected by induced gene mutations and that the effects would probably be adverse.

27. One-quarter of all abortions are caused by, and 1 per cent of all live-born infants suffer from, severe effects of chromosomal anomalies which arise spontaneously. It is, in our present state of knowledge, only possible to give estimates of rates of induction by high doses of radiation of chromosomal damage of types which include not more than a small proportion of the anomalies that occur naturally. The number of these that would arise after exposure to high doses can be estimated, but it is not known how many would occur following low doses, although the yield per unit dose would be much less than that expected if the yield were directly proportional to the dose. It should be noted that a large part of this type of genetic damage is not expected to persist in a population for more than one generation.

28. Part of the total impairment in the first generation offspring of irradiated parents has been studied in mice, namely, certain skeletal defects. From experiments using high doses, it is known that malformations of the skeleton do occur fairly frequently in these offspring. Whether proportional numbers of such defects would result from low doses to parents is not known.

29. The estimates arrived at in this report relate to the genetic effects of acute exposures, at high doses, of male reproductive cells in the stage (spermatogonia) that is most important in human hazards. Lower numbers of these mutations per unit dose will occur where the radiation dose is low or is spread out over a long time. It is also known that the reproductive cells of the two sexes differ in sensitivity; fewer mutations, on the average, will occur when the reproductive cells of females (oöcytes) are exposed to radiation.

30. The Committee is of the opinion that these estimates, because they are subject to many uncertainties, should not be applied in a simple and direct fashion to radiation protection. Any practical application of these numerical estimates must be made with full recognition of the qualifications set out in the above paragraphs and discussed in detail in annex C.

31. Although there are insufficient data for making satisfactory estimates of risk, it is clear that, with any increase of radiation levels on earth, the amount of genetic damage will increase with the accumulated dose. While any irradiation of the human population is genetically undesirable because of its implications for future generations, it should be pointed out that the proper use of radiation in medicine and in industry is important for the health of the individual and for the welfare of the community.

32. The limited number of estimates made, the many uncertainties as to their accuracy and the reservations which have to be attached to each of them may seem disappointing. The reasons will be clear to readers of annex C where the complications of establishing meaningful estimates are fully discussed. Although absolute measures of risk are still very uncertain and will probably remain so for some time, major advances have been made in our knowledge of the relative risks under various conditions of radiation exposure and for different biological variables such as the reproductive-cell stage. These findings are of considerable practical value. Thus, it is useful to know that the genetic hazard will be less per unit dose of radiation when the exposure is spread out in time, is delivered in small dosage, or when a long interval occurs between irradiation of the female germ cell and conception. These factors must be clearly borne in mind when making comparative risk estimates.

ANNEXES

Annex A

RADIATION FROM NATURAL SOURCES

CONTENTS

I. Introduction

1. Radiation from natural sources was examined both in the 1958[1] and in the 1962[2] reports of the Committee. Its importance lies in the fact that the human species has always been exposed to natural radiation at relatively stable average levels and that doses from natural radiation are being used by the Committee as a standard of comparison with those received from other, man-made, sources.

2. The present review is essentially an updating of the earlier ones and should be read in conjunction with them. The relative emphasis here given to individual topics—as, for instance, neutron doses and radio-activity in air—thus reflects the amount of new information which has become available since 1962 rather than their actual importance in the over-all study of natural radio-activity. Much of the new information has been the result of improvements in instrumentation which have been stimulated by the study of environmental contamination.

3. Natural radiation arises from two sources: cosmic rays, entering the atmosphere from outer space, and radio-active materials in the earth's crust. These materials were already present when the earth was formed, or, in the case of shorter-lived radio-isotopes, they are continually being produced by radio-active decay or nuclear reactions. Some radio-isotopes are produced by the interactions of secondary cosmic rays, mainly neutrons, with atmospheric gases and, to a small extent, by the interactions of the cosmic rays that reach the earth's surface.

4. Owing to the varying content of natural radio-active elements in the soil and the underlying rock, the intensity of external radiation and the levels of radio-active intake by man vary from place to place. Because of limited geographical representation and of the obvious limitation of sampling, the arithmetic means calculated from the most frequent values were accepted, but they may not be strictly representative for the whole world population.

5. It is obvious that arbitrary criteria are used to separate typical situations from those where background radiation is considered elevated either because of local abundance of radio-active material or because of special food chain mechanisms. In the present review, only those situations will be described as non-typical in which at least one of the factors contributing to the natural irradiation of man is higher than typical by one order of magnitude or more.

II. Cosmic rays

Introduction

6. Cosmic radiation consists of primary radiation entering the atmosphere from outer space and of secondary cosmic radiation produced by interactions of the primary radiation with nuclei in the atmosphere. Most measurements of the primary component have been carried out at atmospheric depths of 15 g/cm^2 or more (up to about 30 km), but extrapolations to outer space as well as direct measurements indicate that the primary component consists of highly energetic, positively charged nuclei, with protons constituting 83-89 per cent of the primary radiation, alpha particles 10-15 per cent, leaving 1-2 per cent for nuclei having $Z \geqslant 3$ and for some energetic electrons. About one positively charged particle/cm^2 $\times$ second with an average energy of 2×10^3 MeV arrives at the top of the atmosphere.[3-12]

7. This primary radiation is incident from all directions. The major portion of the primary cosmic radiation is of galactic origin. Low energy particles of solar origin in the 10 MeV range may reach the earth during large sunflares. The contribution of solar particles to the total cosmic-ray intensity in the lower atmosphere is negligible, however, if averaged over long periods. Slight annual modulations of cosmic-ray flux densities, as well as variations during magnetic solar storms, have been observed.[13-17]

8. The secondary cosmic-ray component comprises many types of radiation produced by nuclear collisions of the primary particles with nitrogen, oxygen or argon nuclei mainly in the upper atmosphere. At about sea level, this secondary radiation is usually divided into three separate groups: the muon, the nucleon, and the electron component. Some properties of the main secondary particles in cosmic radiation are summarized in table I.

9. *The muon component.* Muons (previously called μ-mesons) are the daughters of short-lived pions. The pions result from the interaction of high energy protons with atmospheric nuclei. The maximum muon flux density occurs at an atmospheric depth of about 150 g/cm^2 (usually at a height of about 12 km). Ionization due to cosmic rays detected at low altitudes is mainly due to the penetrating muons (about 70 per cent).[18, 19] The muon component still contributes about 50 per cent to the ionization from cosmic rays at an altitude of 3 km, and its relative share continues to decrease with altitude.

10. *The nucleon component.* The nucleon component consists of nuclear fragments, mainly neutrons and protons.[7, 12, 20, 21] It multiplies by cascade processes involving nuclear spallation in the atmosphere, whereby the secondary fragments knocked out of atomic nuclei possess high enough energy to produce still further fragments in subsequent nuclear interactions. As nitrogen and oxygen consist of an equal number of protons and neutrons, the initial nucleonic cascade in air consists of approximately equal numbers of protons and neutrons with high energies. Below about 500 MeV, however, ionization losses of protons in the air start competing with nuclear absorption; protons are therefore progressively removed from the cascade which, below a few hundred MeV, consists mainly of neutrons.

11. As neutron emission is the most probable de-excitation reaction when nitrogen and oxygen are excited to energies of about 8 MeV, neutrons in the range of a few MeV will be evaporated isotropically during the terminal process in nucleonic cascades.[21, 22] About 20 per cent of the energy from the incident primary radiation is transferred to nucleonic cascades. Most of it, however, is absorbed before reaching the earth's surface. Slow protons are stopped by ionizations, and slow neutrons are captured mainly in nitrogen, forming C^{14}.

12. *The electron-photon component.* The electron-photon component includes electrons, positrons and photons. They originate mainly from the electron-positron-photon cascade resulting from the decay of the extremely short-lived neutral π° meson produced when high energy primary radiation interacts with atmospheric nuclei. The spontaneous decay of muons also contributes some high energy electrons to this component.

13. When cosmic rays are measured, the neutrons are usually dealt with separately from the other components. The latter, including gamma rays, are further subdivided according to their penetration power. The "soft" component consists of the radiations that are completely absorbed in about 15 cm of lead. At low altitudes, this component includes the bulk of the electrons, gammas, and protons, as well as a few slow muons. The "hard" component, some of which is only slightly attenuated by 15 cm of lead, may be able to penetrate much thicker layers. The "hard" component at sea level consists mainly of muons and of high energy protons.

Geomagnetic effects

14. An approximation to the external geomagnetic field is obtained by assuming a magnetic dipole located 340 km off the centre of the earth, with poles pointing towards 80.1°N, 82.7°W, and 76.3°S, 121.2°E. This terrestrial magnetic field acts as a momentum selector for the primary charged particles. Consequently, the cosmic ray flux entering the upper atmosphere is latitude dependent as well as directional.[7-9]

15. The net result of these effects is that particles with the lowest energies reach the earth only in the vicinity of the geomagnetic poles, whereas those with energies in excess of about 6×10^4 MeV can reach the earth anywhere.[23-28]

16. The minimum momentum that an incident charged particle must possess in order to reach the earth's atmosphere despite the deflecting influence of its magnetic field is called the threshold rigidity. It varies with geomagnetic latitude as $\mathrm{Cos}^4\phi$ and depends on the angle of incidence of the charged particle.

17. The threshold rigidity for vertically incident charged primaries at the geomagnetic equator is about 5.7×10^7 gauss $\times$ cm corresponding to a proton energy of 1.7×10^4 MeV.[7, 27] However, the intensity of the ionization due to cosmic rays measured at sea level is only about 10 per cent higher near the geomagnetic poles than the ionization recorded at the equator. Thus, about 90 per cent of the ionization detected at sea level results from primaries which had enough momentum to arrive at the earth's geomagnetic equator. The primaries of lower momentum are relatively ineffective in producing penetrating muons.

18. The latitude variations are more pronounced in the case of the nucleonic component, as shown in figure 1. This latitude effect of neutrons (and of secondary protons) demonstrates that the nucleonic component is mainly produced by low energy primaries that are prevented by the earth's magnetic field from reaching the earth between 50°N and 50°S but that

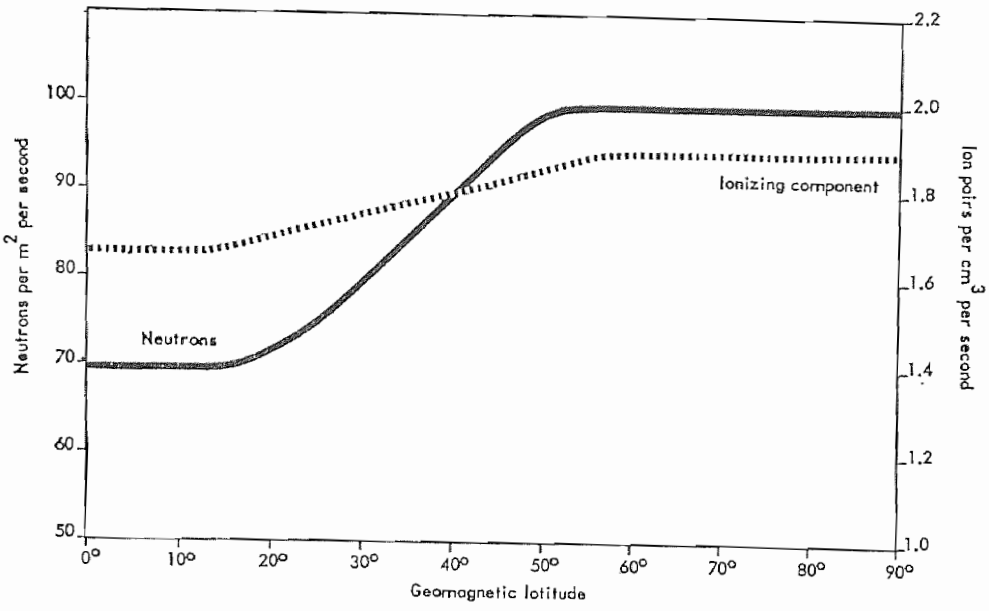

Figure 1. Geomagnetic latitude dependence of the ionizing component and neutrons at sea level[9, 27, 28]

do reach the polar regions. Pion production, on the other hand, predominates at higher energies (above a few 10^4 MeV) and therefore is much less affected by the geomagnetic field.

19. The apparent plateau region for the secondary components at high geomagnetic latitudes has been extensively studied.[4, 11, 13-17] Atmospheric attenuation due to ionization losses, and insufficient sensitivity of some experimental equipment, might partially account for it. However, there seems to be a real decline in the flux density of primary particles of galactic origin for energies less than 2×10^4 MeV per nucleon. Low energy primaries reaching the upper atmosphere are mostly of solar origin.

20. As the geomagnetic poles do not coincide with the earth poles and as the magnetic dipole representation of the geomagnetic field itself is a rather crude approximation which needs correction terms (higher magnetic moments) to account for the actual energy cut-off of primary cosmic rays, it is easy to explain the slight longitudinal effect observed in the relative intensities (up to 15 per cent) of cosmic radiation. Temporal distortions of the geomagnetic field due to solar activity are also observed.

21. It should be emphasized that the geomagnetic latitude effect revealed by the secondary cosmic radiations should be wholly attributed to the primary component. Owing to their comparatively short trajectory from the point of production in the atmosphere to ground level, no appreciable geomagnetic deviation is expected for the secondary charged particles.

Energy spectrum of primary radiation

22. Figure 2 represents the integral energy spectrum of primary protons.[6-12, 20, 29, 30] The integral energy spectrum per nucleon of the total primary radiation is similar within the experimental accuracy. The latitude-sensitive portion of cosmic radiation up to about 6×10^4 MeV—the threshold rigidity for positively charged protons from the east at the magnetic equator—is often referred to as "low energy radiation". As low energy particles are much more abundant, most of the cosmic-ray energy will reach polar regions where, therefore, most of the secondary rays will be produced. The term, "very low energy radiation", is reserved for primaries below 1.7×10^8 MeV, whose abundance undergoes seasonal changes correlated with solar events.

Cosmic rays in the atmosphere

23. Figure 3 shows the altitude variations of the main components of cosmic radiation in the atmosphere.[7-9, 26, 29] Production of the secondary particles

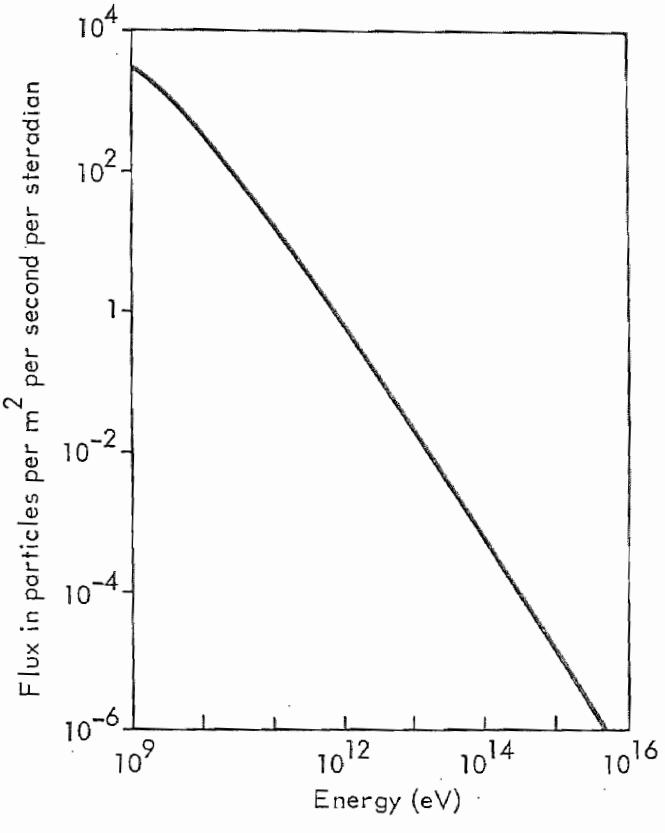

Figure 2. Integral energy spectrum of the primary cosmic-ray protons[9, 10]

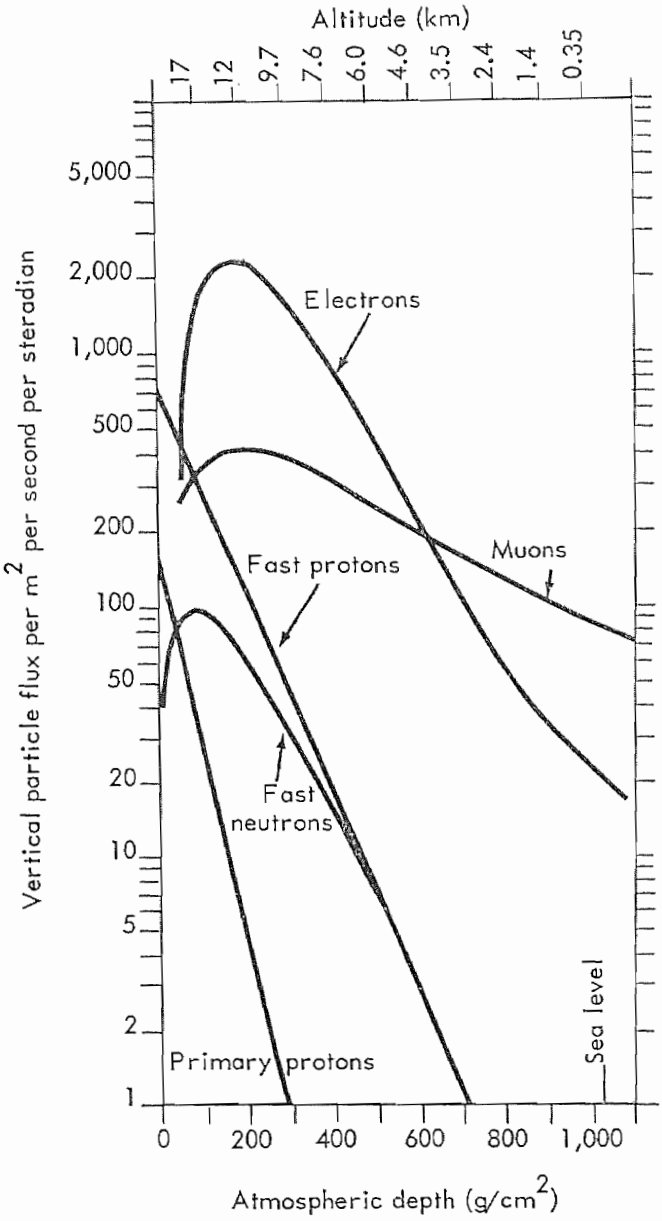

Figure 3. The vertical flux densities for the main components of cosmic radiation as a function of atmospheric depth (at geomagnetic latitude of 45°N)[7]

increases with atmospheric depth up to about 150 g/cm² (down to about 12 km) where the maximum flux density of cosmic rays is found. Below this altitude, particle loss through capture, ionization and muon decay predominates over production, and the various secondary cosmic-ray components decrease exponentially with decreasing altitude. The primary radiation carries almost all the energy above 25 km, while the muon component predominates below 3 km.

24. Atmospheric effects depending on barometric pressure and temperature are known to affect somewhat the cosmic-ray intensity at sea level, especially the muon component. The muon flux density reaching sea level depends on the thickness of the atmosphere below the point of production, and, because some muons decay in flight, will also depend on their path length in the atmosphere. These local changes are of the order of a few per cent and will not be discussed any further.

25. The vertical component of cosmic radiation is of relatively large significance at sea level, as atmospheric absorption of secondary radiation and muon decay are minimal for vertical incidence. The east-west effect being small compared to the absorption effect at various angles, the lower threshold rigidity of particles coming from the west does not compensate for the additional attenuation of the secondary radiation in the atmosphere. For radiation from the east, the higher rigidity threshold and atmospheric attenuation both operate to diminish the secondary flux arriving at sea level.

Radio-nuclides produced by cosmic rays

26. Some of the nuclear fragments resulting from spallation of atmospheric nuclei during the nucleonic cascade process are radio-active.[31-38] About 1.7 spallations/cm² × second is the global average induced by cosmic rays. They occur in atmospheric gases in proportion to their relative abundance (nitrogen : oxygen : argon ~ 76.5 : 22.5 : 1). The distribution of nuclide production rates within the atmosphere, taking into account the energy spectrum of the nucleon component of cosmic radiation, was calculated by Lal.[32, 39] Most radio-nuclides are produced in the stratosphere by low energy neutrons, their rate of production being approximately proportional to the neutron flux density and subject to its pronounced latitude dependence.

27. The main radio-nuclides produced by cosmic rays are listed in table II with some of their nuclear properties, calculated production rates and activity concentrations in the lower troposphere. C^{14} is mainly formed by the $N^{14}(n, p)C^{14}$ capture reaction with atmospheric nitrogen. About two-thirds of the neutrons produced by cosmic rays are removed through this process from the atmosphere.

28. The production rates of radio-active spallation fragments from oxygen and nitrogen (H^3, Be^7 and Be^{10}) exceed by far the production rate of fragments due to argon spallation. Cosmic rays which reach the earth's surface can interact and produce radio-active nuclides, but these have extremely low activity and are unimportant in comparison with other nuclides of natural origin.

Cosmic-ray neutrons

29. As neutrons usually elude detection when ionization chambers are used, this nuclear component of cosmic radiation should be assessed separately. Cosmic-ray neutrons are produced by two kinds of reactions.[5, 23, 28, 41-49] First, neutrons are knocked out of nuclei as a result of nuclear collision of high energy cosmic rays. These neutrons have energies from a few MeV up to more than 1,000 MeV. A larger source of neutrons in the atmosphere, however, is represented by evaporation neutrons which have an energy distribution peaked at about 1 MeV.[12, 21, 23, 43, 50, 52] The evaporation process may account for about 80 per cent of the atmospheric neutron flux density according to estimates derived by Hess *et al.*[44]

30. Neutrons produced in the atmosphere will eventually either leak out of the upper atmosphere into space or disappear through absorption. Atmospheric absorption occurs largely through $N^{14}(n, p)C^{14}$ capture reactions after the neutrons have been slowed down by elastic and inelastic collisions in some 150 g/cm² of air. A stationary condition in time is thus maintained where the neutron flux density in the atmosphere is proportional to the neutron production rate at a somewhat higher altitude.

31. Owing to the low flux density of neutrons produced by cosmic rays at sea level, most measurements concerning the neutron component were carried out at various heights in the atmosphere within the equilibrium region, below 150 g/cm^2 (up to 12 km) where the energy spectrum of neutrons is essentially constant.[28, 41-58] From these measurements, estimates of neutron fluxes at sea level were obtained through extrapolations. It is, however, difficult in making such extrapolations to allow for the actual situation that obtains at sea level because of the presence of an interphase and of backscatter with a consequent breakdown of the atmospheric equilibrium conditions.

Flux densities and energy spectra

32. A major limitation in comparing the results of different experimenters is the interdependence between the energy spectrum, which is assumed, and the absolute neutron flux density that is derived from the measurements. Each detector is sensitive in a limited energy range only, and its sensitivity is often energy dependent.[41] Thus, the interpretation of counting rates obtained through detectors of different kinds presupposes a detailed knowledge of the energy spectrum of cosmic-ray neutrons.

33. Figure 4 shows the cosmic-ray neutron energy spectrum in the equilibrium region as derived by Hess *et al.*[44, 52] They used a set of detectors sensitive to different energy ranges from thermal energies up to about 10^4 MeV. Their extrapolation to sea level yielded a flux density of 4×10^{-2} n/cm^2 $\times$ second, of which 15 per cent were between 1 and 10 MeV, 75 per cent had lower energies, and about 10 per cent were above 10 MeV.

34. Miyake *et al.*[43] obtained an energy spectrum of cosmic-ray neutrons from 1 to 15 MeV by observing recoil protons by means of a high pressure cloud chamber filled with hydrogen. Their derived energy spectrum for 1 eV to 10^4 MeV neutrons is in good agreement with figure 4, especially above 10 MeV and in the eV region.

35. Another calculation of the equilibrium spectrum of neutrons in the atmosphere up to energies of 20 MeV was performed by Newkirk.[51] This spectrum also differs from Hess' spectrum mainly in the energy range of 0.1 to 4 MeV, and thus agrees better with other measurements.[53, 55]

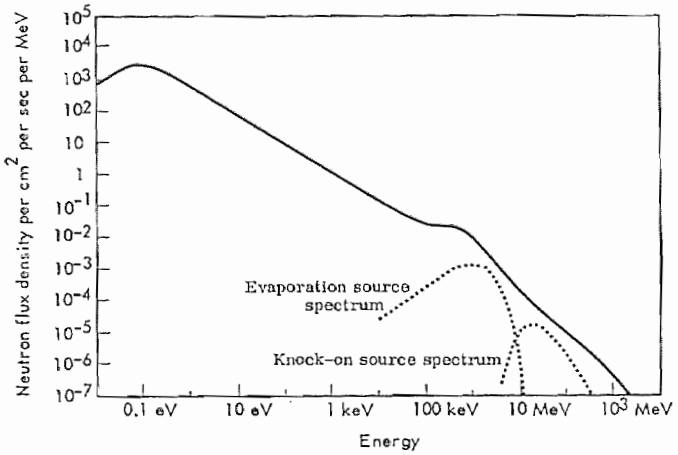

Figure 4. The equilibrium neutron flux density versus energy at sea level (44°N)[44] (the assumed spectra of the two neutron sources are also shown)

36. Subsequent workers arrived at lower estimates for the total neutron flux density.[49, 55-59] The lower values might partly be due to the special care taken to minimize neutron production in the instruments themselves and in the surrounding materials. In particular, lightweight detection equipment flown with balloons was used instead of heavier aircraft-borne detectors.

37. Kastner *et al.*[60] using a liquid scintillator, reported a flux density of 4.5×10^{-3} n/cm^2 $\times$ second for neutrons with energies from 1 to 10 MeV as measured directly at an altitude of 180 metres. They suggested a total neutron flux density at sea level (41°N) of about 10^{-2} n/cm^2 $\times$ second, 15-25 per cent of which are in the energy range 1 to 10 MeV, and about 70 per cent have lower energies.

38. It seems reasonable to follow the suggestion of Kastner *et al.*[60] and to assume the value of 10^{-2} n/cm^2 $\times$ second as the total neutron flux density at sea level. A lower value of 0.54×10^{-2} n/cm^2 $\times$ second at sea level as given by Haynes,[58] was recently used as a basis for dose estimates.[61]

Cosmic-ray dose rates[a]

39. In the 1962 report,[2] the dose rate to soft tissue due to the ionizing component of cosmic rays at sea level at middle latitude was estimated as 28 mrad/year. Recent reported values generally agree with this figure.[18, 19, 62-66] Table III lists the reported values in ion pairs/cm^3 $\times$ second as well as in mrad/year.

40. Recent surveys[18] confirmed the altitude dependence of the ionizing component as discussed in the 1962 report. Muons are the main contributors to absorbed dose at low altitudes (some 80 per cent of the

[a] *The "dose" concept.* The interaction between radiation and matter results in a variety of processes that include absorption, scattering and the production of secondary radiations. Hence, when the human body is exposed to radiation, the distribution of absorbed energy is usually not uniform. Although such quantities as the surface dose, the maximum dose and the average dose can therefore differ appreciably depending on the nature, energy and direction of the incident radiation, it is frequently desirable to utilize a single numerical index to specify the degree of irradiation. This is usually termed the "dose" and expressed in the unit "rad" or its submultiples (mrad, etc.). The usual meaning is the one that will be adopted here when the term "dose" is employed without further specification. It corresponds to

(*a*) The kinetic energy of the secondary charged particles produced per unit mass of soft tissue of approximate composition $(H_{40} C_5 O_8 N)_n$, in the case of indirectly ionizing radiations (neutrons, photons and other uncharged particles);

(*b*) The energy deposited per unit mass in matter of the same composition as given in (*a*), in the case of directly ionizing radiations (all charged particles).

These quantities are obtained when the absorbed energy is computed from the appropriate interaction cross sections for the incident radiation spectrum. They will also be obtained within a few per cent, if the absorbed dose is determined in a small tissue-equivalent detector having a wall of sufficient thickness to establish secondary charged particle equilibrium. In most instances, the "dose" thus determined will differ by less than a factor of two from the maximum dose in a human phantom.

It is recognized that the above definitions do not conform with recently recommended usage.[69] In particular, (*a*) defines tissue kerma rather than the absorbed dose.[68] This departure from rigorous terminology is necessitated by the fact that most of the literature sources use this approach and by the absence of an acceptable term for (*b*).

The relative biological effectiveness (RBE) of any radiation depends not only on its type and quality but also on the effect under study and on other factors, such as dose, dose rate and the value of physiological parameters. This is because, even when equal amounts of different radiations are absorbed in any tissue, their effect is usually different as a result of differences in the microscopic distribution of the absorbed energy.[67]

ionizing component according to Lillicrap,[10] 70 per cent according to Lowder and Beck[18]).

Neutron dose rates

41. On the basis of Hess' energy spectrum[44, 52] and the usual factor to convert flux densities to dose rates,[61, 70, 71] 10^{-2}n/cm^2 × second is assumed to correspond to a dose rate of 0.7 mrad/year. However, this value is considered to be an over-estimate, since the spectrum derived by Newkirk[51] gives a lower dose rate for the same total flux density of neutrons. As most of the other experimental values for flux densities range from 0.4 to 1.5 × 10^{-2}n/cm × second,[53, 55, 57, 58, 60] one obtains a range from 0.3 mrad/year to 1.1 mrad/year for the neutron dose rate at sea level.

42. Neutron flux densities, and to a lesser extent the energy spectrum, are latitude dependent. Thus, assuming 0.7 mrad/year at 41°N, slightly higher dose rates will be encountered near polar regions,[57, 61] whereas at the equator the neutron dose rate might be about 30 per cent lower or approximately 0.5 mrad/year.

43. *Correction factors.* A number of factors need to be considered when estimating radiation dose rates to human tissue (outdoors or in buildings) from an established neutron flux density in air with a known energy distribution. Owing to the abundance of hydrogen atoms in the human body, neutrons produced in the atmosphere lose their energy faster in traversing 1 g/cm^2 of tissue than in traversing 1 g/cm^2 of air.[72-76]

44. Practically all neutrons entering the human body with energies below 5 MeV lose their energies by elastic collisions with hydrogen and are finally absorbed by $H(n,\gamma)D$ or $N^{14}(n,p)C^{14}$ reactions. The neutron dose decreases rapidly with increasing depth[71, 75] and at 10 cm beneath the surface the dose is about one-third to one-tenth of that at the skin, depending on the energy and isotropy of the neutrons.

45. As neutrons lose on the average about half of their energy in the first collision with a hydrogen nucleus, neutrons of higher energy (above 5 MeV) lose significantly more of their energy in the human body than in the same mass of air (or other non-hydrogenous materials). However, neutrons above 5 MeV are relatively rare in the equilibrium spectrum of air, thus limiting this additional contribution to human tissue dose to 20 per cent at most of the neutron dose.

46. Neutron production in the human body must also be considered. One may assume that the cross sections per atom for the production of evaporation neutrons in the body (oxygen, carbon) are comparable with those in air (oxygen, nitrogen). The neutron component which is responsible for most of this terminal stage in the nuclear cascade leading to evaporation neutrons has a significantly shorter mean free path in the body than in air. More neutrons will therefore reach this final stage of evaporation interactions in the body than in an equivalent mass of air, thus tending to raise the average tissue dose received from neutrons.

47. As to the shielding by buildings and the computation of neutron dose indoors, tissue equivalent material might be as likely to add somewhat to the dose as to shield part of it, because of the scattering effects and the possibility of higher production rates in these materials as mentioned above. Heavy construction materials, such as lead and iron, tend to add to the neutron background.[22, 77-79] Thus, it seems unjustified to allow for the shielding effect on neutron doses unless detailed knowledge on the composition of the building materials is available.

CONCLUSIONS

48. The contribution of the ionizing component of cosmic rays at sea level was estimated in the 1962 report as 28 mrad/year at middle latitudes, which is also the best value agreed upon lately.[18, 63, 65] This dose rate of ionizing radiation is mainly delivered by muons[19] and is subject to the slight variations due to latitude effects which may lower the dose rate by about 10 per cent near the equator. The total dose rate approximately doubles for every 1,500-metre increase in altitude for the first few kilometres.[2, 18, 64] The relative importance of the nucleonic component as compared to the muon component increases with altitude. These facts will be of importance in assessing doses received at high altitudes, including space flights and other high altitude flights.[61]

49. Revised data on cosmic-ray neutron flux densities indicate that, without allowing for shielding by building structures and for screening by body tissues, the corresponding absorbed dose is 0.7 mrad/year (range 0.3-1.1 mrad/year) in temperate and polar regions, while in equatorial regions it is likely to be around 0.5 mrad/year. This value may be compared with the dose rate of 2 mrad/year which can be inferred from the estimate given in the 1962 report.

III. Terrestrial radio-activity

SOIL

50. Practically all the natural enviromental radiation of terrestrial origin is due to radio-nuclides of the uranium and thorium series and to K^{40}. Typical abundance figures for the accessible lithosphere are 2.8 parts per million of uranium and about eleven parts per million of thorium. The nuclides belonging to these two radio-active series and their more important properties are listed in tables IV and V. The U^{235} series is of lesser importance but is listed for completeness in table VI. A schematic representation of these three decay series is given in figure 5.

51. There are other primordial radio-nuclides in the earth's crust. Of these, the greatest contributor to terrestrial radiation is K^{40}, which makes up about 0.01 per cent of natural potassium. Certain properties of this nuclide are listed in table VII, along with the properties of selected primordial radio-nuclides which are of lesser importance because of low abundance, long half-life or weak radiations.

EXTERNAL RADIATION

52. External natural radiation is produced mainly by the gamma emitters of the natural radio-active series and by K^{40} in soils, rocks and construction materials, by cosmic radiation as discussed in part II, and to a small extent by atmospheric radio-activity. Outdoor and indoor terrestrial gamma dose rates measured before 1961 were summarized in tables IV, V and VI of annex E of the 1962 report.

53. Measurements of the total gamma dose obtained by means of high pressure argon or tissue equivalent ionization chambers,[80-83] were recently supplemented by gamma spectroscopy in order to assess separately the various contributions to the external radiation dose.[64, 81-89, 91, 92] Figure 6 shows a typical gamma spectrum on which the K^{40} photo-peak of 1.46 MeV and the main peaks of the uranium and thorium series are indicated.

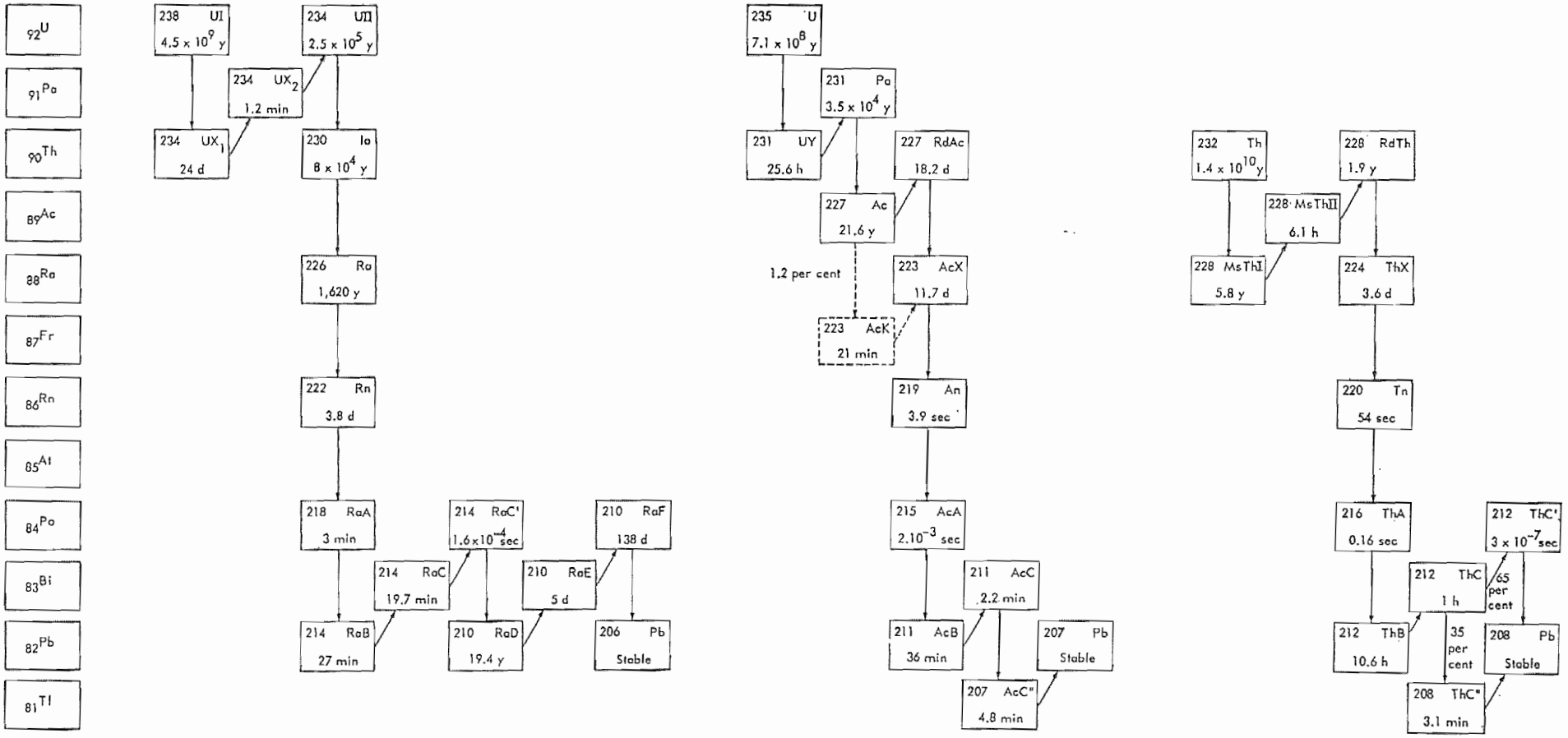

Figure 5. Decay schemes of the natural series[a] (boxes show atomic weight, historical name and half-life)

[a] Parallel decay branches of less than 1 per cent are not included.

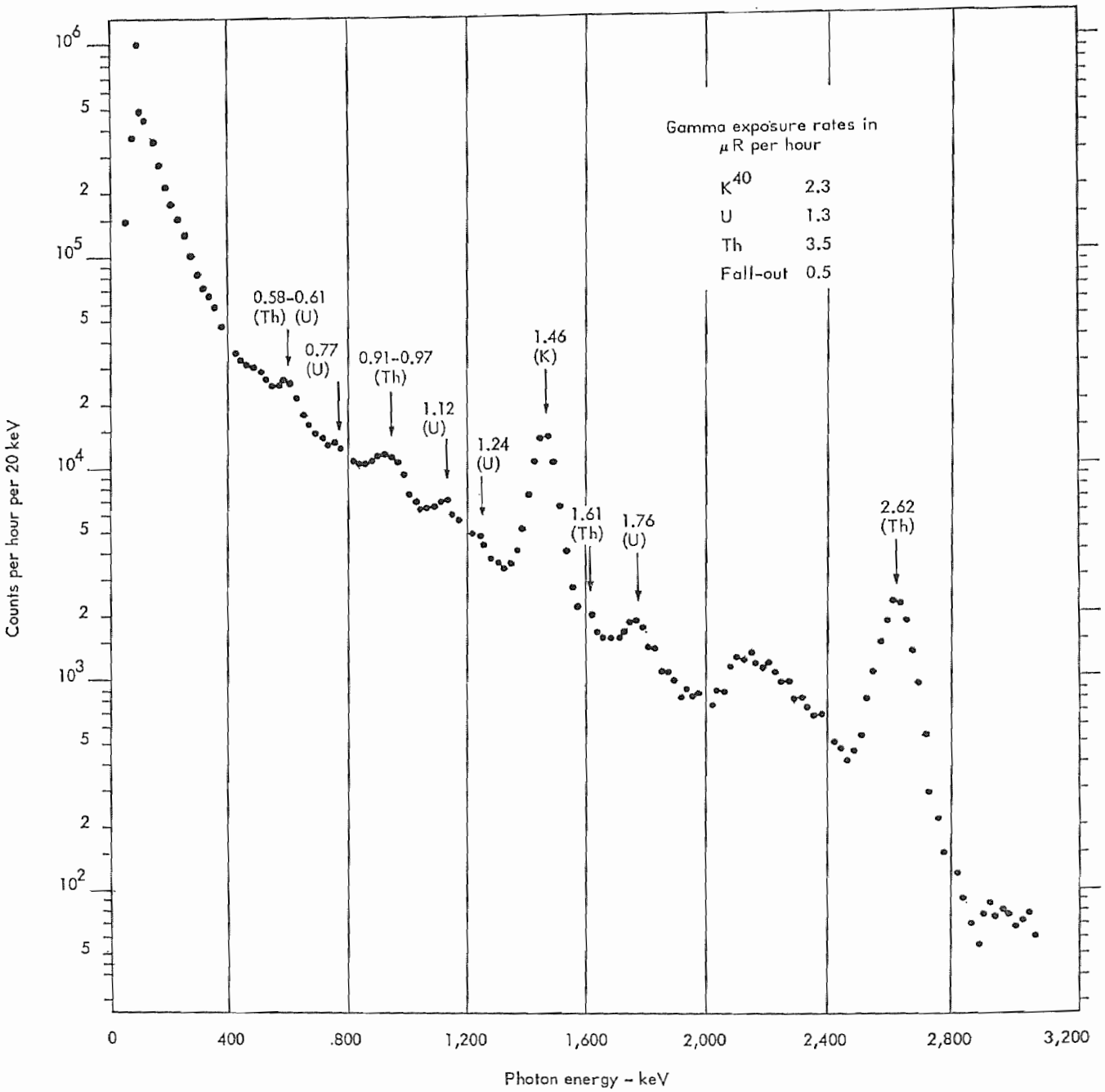

Figure 6. Environmental gamma radiation spectrum, 4 × 4″ NaI(Tl)-scintillation detector, Denver, Colorado, 12 August 1965[241]

54. The contribution of each component to the air dose, as derived from the evaluation of gamma spectra, using the 2.62 MeV photo-peak of Tl^{208} to represent the thorium series, the 1.46 MeV photo-peak for K^{40} and the 1.76 MeV photo-peak of B^{214} for the uranium series, were often compared with doses estimated on the basis of the measured abundance of Th^{232}, K^{40} and U^{238} in underlying soils or rocks.[86-88] In general, the two methods gave results consistent within 5-10 per cent for K^{40} and Th^{232}, when soil density and moisture content were taken into account.

55. No such close correlation between the dose determined from Bi^{214} and the U^{238} concentrations in underlying soils was found, however.[84, 89, 93] Nor could it be expected, since the movement of free radon out of the upper layers of the soil into the atmosphere generally reduces the contribution of the uranium series to the external dose at ground level. Dose rates can be correlated with the gamma-emitting daughters of the radon retained in soil. The concentrations of the daughters is only a fraction of the actual Ra^{226} concentration in upper soils.[93, 94] Moreover, U^{238} is not likely to be in equilibrium with Ra^{226} because of the greater tendency of uranium to leach from upper soil layers during soil formation and weathering processes.

56. An increase in water content of soil will increase the density and consequently the attenuation of gamma rays from K^{40} and the radio-active series and will thus cause temporal variations in the dose rate correlated to soil moisture. The dose rates from the uranium series may, however, be affected also in the opposite direction, since clogging of the pores in the soil impedes the escape of radon from soil air into the atmosphere, thus resulting in an increased dose rate

from radon daughters. Furthermore, during rainfall, the wash-out of short-lived gamma-emitting daughters of radon from the atmosphere will temporarily increase the external dose rate attributed to the uranium series.[84, 85, 90, 94-97]

57. The measured average external radiation dose rates one metre above soil in populated areas fall mostly in the range 30-65 mrad/year with a representative value of about 50 mrad/year.[84, 92, 97-100] Potassium and thorium each generally contribute more than one-third, while the contribution from the uranium series is usually somewhat less.

58. From the point of view of many human populations, exposure levels out of doors are more relevant than indoors. Indoor doses depend primarily on the radio-active content of construction materials and on the attenuation of outside radiation by roofs and walls. There seems to be no reason for changing the value of 50 mrad/year adopted in the 1962 report, as the few additional experimental results[101, 102] fall within the ranges quoted earlier.

59. Unusually high natural radiation areas were discussed in the 1962 report and the five major inhabited areas with increased terrestrial radiation were tabulated in table XX of annex E of that report. Further studies concerning external dose rates to the populations in these areas supplement and confirm previous data.[103-107]

60. The comments made in the 1962 report concerning the insignificance of external beta radiation to gonad or bone marrow doses must still be accepted. The external radiation dose rate from natural airborne radio-activity is usually of the order of 2 mrad/year. Since it is small compared to the dose variations discussed in paragraph 56, atmospheric radio-activity as an external radiation source will not be dealt with separately.

Neutrons

61. Neutrons in the earth's crust may be produced by interactions of soil elements with cosmic rays, by spontaneous fission of U^{238}, by (α, n) and possibly by (γ, n) reactions. While of no significance as far as doses are concerned, these neutrons, some of which escape from soils and rocks, are of interest to geologists, and may sometimes be confused with cosmic-ray neutrons.

62. An indirect estimate for the production rate of neutrons due to the interactions of cosmic rays in upper soils may be obtained by dividing the average neutron flux density at sea level by the neutrons' mean free path in air, thus neglecting the differences in atomic composition of soil relative to air. A production rate of 7×10^{-5} n/second $\times$ g soil at mid-latitudes (corresponding to about 2,000 n/year $\times$ g soil) is consistent with the extrapolated cosmic ray neutron flux density of 10^{-2} n/cm^2 $\times$ second at sea level and a mean free path of 150 g/cm^2 as discussed in paragraphs 29-38.

63. Spontaneous fission of U^{238} (half-life 8×10^{15} years, and 2.2 n/fission)[108] gives rise to a production rate of the order of only 1.4 n/year $\times$ g soil (assuming three parts per million U/g soil).

64. Gorshkov *et al.* report the experimental yield of 0.107-0.014 n/10^6 alphas for the (α, n) reaction of Po^{210} alphas on SiO_2, and a value of 0.238 n/10^6 alphas for granite.[109] The conversion factor from the Po^{210} alpha yield to the alpha yield from the uranium series in equilibrium should be at least eight and for the thorium series at least six. Upper limits were derived from Gurfinkel's[110] values for the (α, n) reaction on O^{18}, taking into account the increase in yield with alpha energies. These upper limits are twelve and fourteen for the uranium and thorium series, respectively.

65. Thus, taking three parts per million of uranium and eleven parts per million of thorium as representative of the upper earth's crust,[111] an average production rate of 13-24 n/year $\times$ g soil and of 20-50 n/year $\times$ g granite is obtained. Assuming sixty parts per million of uranium and 110 parts per million of thorium as possible upper limits of concentrations in granite, one would obtain a production rate of 450-800 n/year $\times$ g granite from (α, n) reactions which might add a few per cent to the cosmic-ray neutrons observed at elevated altitudes. The 28 n/year $\times$ g granite from spontaneous fission for this case would still remain insignificant.

Natural activity in waters

Oceans

66. The natural radio-activity of sea water[112, 113] is mainly due to K^{40} (300 pCi/litre). Rb^{87} and the uranium series contribute about 3 pCi/litre each to the total activity of oceans. Natural H^3 in the upper oceanic layers might range from 0.6 to 3 pCi/litre, while all other radio-isotopes, including those of the thorium series, contribute less than 0.2 pCi/litre to sea water activity.

Fresh waters

67. Natural radio-activity in fresh waters is due to some activity transfer from soils and the atmosphere. Thus the activity concentrations found in waters depend on the concentrations encountered in the rocks with which the waters are in contact.[36, 114] Members of a radio-active series in water are rarely in radio-active equilibrium with each other because of differences in chemical and physical properties such as solubility, sorbability, etc. K^{40} is often the main contributor to the beta activity in water.

68. As drinking water is one of the media by which natural radio-isotopes are transferred to man, the concentrations of specific nuclides, mainly belonging to the Ra^{226} chain, have been extensively measured for many years.[104, 106, 115-128] Typical concentrations in continental waters, as well as usual concentrations of Pb^{210} and Po^{210} found in rain, are given in table VIII. Tables VII and VIII in annex E of the 1962 report list some earlier values of natural radio-activity in natural waters, springs and public water supplies for various countries. Recent information accumulated since 1962 is summarized in table IX.

69. Ra^{226} concentrations in drinking water vary by orders of magnitude, though most waters show values between less than 0.1 to about 1 pCi/litre. Surface waters (lakes, rivers) usually show less activity than those derived from deep wells, especially in areas where the concentration of natural radio-active minerals in the earth's crust is higher than usual.

70. Rn^{222} concentrations in fresh water may vary from less than 1 pCi/litre up to the order of 106 pCi/litre.[2, 125-130] Levels less than 10 pCi/litre are found in lakes and rivers, and the highest concentrations observed have been reported for some spas and spring waters.[123, 128] Concentrations of 10^2-10^4 pCi/litre have been found in ground waters and even higher concentrations in deep wells (table VIII).

71. Pb^{210} in water is mainly derived from the decay of radon in the air and the resulting deposition of this nuclide with rain, for Pb^{210} concentrations in surface water seem to exceed those in water from deep wells.[117]

Pb^{210} concentrations in water do not fluctuate as strongly as Ra^{226}, and there is no correlation between the two elements nor between Pb^{210} and the sulfate, calcium or fluoride content of water. Typical concentrations are between 0.05 and 0.2 pCi/litre.

72. Information on Ra^{228} or Th^{228} concentrations in water is extremely limited.[36, 104, 119] Turner *et al.*[123] observed the presence of short-lived Th-series nuclides (Ra^{224}) in water sampled in the United Kingdom. Long-lived elements, such as Th^{228} and Ra^{228}, were present only in traces, Th^{228} constituting a very small fraction of the long-lived alpha activity in water.

The natural radio-activity of air

73. The natural radio-activity of the atmosphere is caused mainly by Rn^{222}, Rn^{220} and their radio-active decay products. The contribution of Rn^{219} or its radio-active daughters is negligible.[34, 36, 37] Radio-active nuclides produced by cosmic rays (table II) are of minor importance as sources of atmospheric radio-activity; the radio-activity in dust particles blown from the soil by wind, or from K^{40} brought into the atmosphere by the evaporation of sea-water spray is also extremely low. These sources will therefore not be discussed any further.

74. A simple mathematical model to account for radon exhalation rates from soils, and to estimate concentration profiles of the radon isotopes in the troposphere was developed by Israel.[131] His model, with some minor changes at times, has been widely followed by many authors.[37, 132-134]

75. The spreading out of Rn^{222} and Rn^{220} in the atmosphere after their exhalation from the ground is caused by turbulent diffusion and convection. The decay products of the radon gases, being isotopes of heavy metals, become readily attached to aerosol particles. At ground level, more than 99 per cent of the Pb^{214}, Bi^{214} and Pb^{212} atoms, and about 75 per cent of the shorter-lived Po^{218} atoms are carried by aerosols.[34, 36, 37, 131-137] The fraction of natural radio-activity not attached to aerosol particles increases with altitude. As concentrations of aerosols in the atmosphere are about 10^6 times higher than the concentrations of radon daughters, radio-active aerosol particles will usually contain a single radio-active atom each.

76. The effective radius of aerosols which contain the main portion of natural atmospheric radio-activity is between 50 mμ and 80 mμ.[37, 135] As aerosols are unstable and tend to increase in size as a consequence of condensation and coagulation processes, reported size distributions of activity are only valid for short-lived radio-active decay products. Long-lived isotopes like Pb^{210} and daughters are therefore expected to be attached to larger aerosols.

77. The distribution of the radio-active decay products of radon in the atmosphere is consequently controlled not only by radio-active decay and diffusion, but also by sedimentation and wash-out related to the removal of aerosols.

78. Figure 7 gives a vertical distribution of concentrations for the radon isotopes and their radio-active decay products for "normal" conditions, assuming a half removal time of twenty days for all the radon daughters.[134-136] Full radio-active equilibrium at ground level air for Rn^{222} and its short-lived radio-active daughters is not reached because of the continuous elimination of radio-active aerosols by downward diffusion and settling. However, equilibrium between

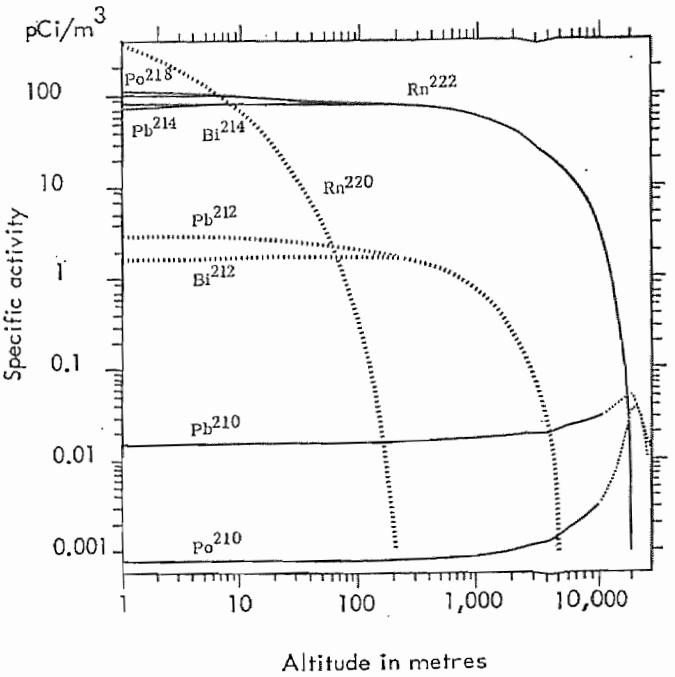

Figure 7. Vertical distribution of concentration of the radon isotopes and their radio-active decay products[134]

Rn^{222} and its short-lived daughters is approached quite closely a few metres above ground.

79. Rn^{220} is virtually never in equilibrium with its radio-active decay products. The 10.6-hour half-life of Pb^{212} greatly exceeds that of Rn^{220} so that Pb^{212} atoms after their formation can diffuse to higher altitudes than Rn^{220} (or Po^{216}). A very low Pb^{212}/Rn^{220} ratio, as expected, was measured by Fontan *et al.*[139] in ground level air. An excess of Pb^{212} over Rn^{220} is expected at somewhat higher altitudes. Owing to their short half-lives, the concentrations of Rn^{220} and of any of its decay products become insignificant, when compared to Rn^{222} concentrations, at some ten metres above ground.

80. The concentrations of Pb^{210}, the long-lived decay product of Rn^{222}, and its radio-active products Bi^{210} and Po^{210} must be extremely low in the troposphere owing to removal by precipitation. Their concentrations, however, increase gradually in the upper troposphere, and actually exceed those of their short-lived parents in the stratosphere. Concentration ratios of Po^{210} to Pb^{210} depend critically on the mean removal rate and increase rapidly above 1 km. Observed Bi^{210}/Pb^{210} and Po^{210}/Pb^{210} ratios in air and precipitation have therefore been used to determine the mean residence time of natural aerosols in the troposphere.[37, 140-145]

Horizontal distribution and variability

81. Local concentrations of the radon isotopes and their radio-active daughter products depend not only on the emanation rate of radon gases from the soil but also on a variety of local meteorological conditions. Wind direction is of special importance in coastal sites, on the sea and in the polar regions, as radon is mainly carried by continental air masses.

82. Winds reduce Rn^{222} concentration in the upper soil layers, thus enhancing the exhalation rate. All air motions near the earth-atmosphere interphase are turbulent; they assist to some extent in the extraction of radon gases from the upper soil air. These air motions,

on the other hand, may enhance the removal rate of radio-active daughters formed in the lower atmosphere by increasing their impact on the ground. They are also removed by precipitation and dry settling of aerosols.[37, 134-137, 145]

83. Diurnal, seasonal or more irregular variations of the radio-activity at a given site have been reported.[135, 146-151] During night hours, a temperature inversion usually develops near the ground, which reduces the vertical mixing of the radon gases after their exhalation from the ground, thus producing a build-up of radio-activity which reaches its maximum concentrations in the early morning hours. The rising temperature during day-time will break this temperature inversion and facilitate turbulent mixing, thus reducing radio-activity concentrations near the ground. Minimum concentration values are usually found during the afternoon. Diurnal variations may cause concentrations to change by a factor of two to three on the average, but variations exceeding a factor of ten have been reported.

84. In some locations, the average radon concentrations in the air during winter and early spring are lower than those prevailing during summer.[30, 97, 151, 152] This may be due to the known seasonal variations in escape of radon from the ground, the relatively low winter and spring values being attributable to the higher moisture content of the soil. Frozen ground reduces escape considerably, and thick layers of snow or ice stop it completely. In other locations, the maximum radon concentrations are observed during autumn and winter and minimum ones in spring and summer, in accordance with variations of atmospheric conditions, e.g. the rate of vertical turbulent exchange.[139, 153, 154]

Measured concentrations of natural radio-activity

85. The release of Rn^{222} and Rn^{220} is considerably greater from land than over water. Typical measured levels in ground level air over continents range from 30 to 300 pCi Rn^{222}/m^3 and 0.5 to 10 pCi Pb^{212}/m^3;[146-150, 152-157] over the oceans, the concentrations are between one and two orders of magnitude lower.[37, 131, 158, 159] Concentrations of the order of 2 pCi/m^3 for Rn^{222} and less than 0.3 pCi/m^3 for Pb^{212} were measured in Antarctica.[160, 161]

86. Concentrations of Rn^{222} and Rn^{220} in ground level air in various regions were summarized in table IX of annex E of the 1962 report, and in some recent publications.[36, 37, 135] Only a few determinations for Rn^{220} have been reported, based on the beta decay of Pb^{212}. The erroneous assumption of radio-active equilibrium of the Pb^{212} with the short-lived Rn^{220} in the air sampled is partly responsible for the low Rn^{220}/Rn^{222} ratios reported.[37, 131, 162, 163]

87. Radon concentrations indoors are generally higher than outdoor concentrations, depending on the construction materials, the degree of ventilation and the escape from indoor water sources. Average values of Rn^{222} concentrations indoors at various locations were reviewed in annex E of the 1962 report and summarized there in table XI.

88. On theoretical grounds, it can be expected that the concentration of radon in air in deep layers of undisturbed soils can be of the order of 10^5 pCi/m^3 air;[37, 131] therefore, such radon concentrations are to be expected in the air of caves and unventilated underground mines. Measurements made in mines, particularly uranium or thorium mines, give results consistent with this expectation.[170-173]

Long-lived decay products of radon-222

89. The general features of the circulations of Pb^{210} and Po^{210} resemble the patterns of stratospheric fall-out of artificial nuclear debris. The concentrations of Pb^{210} in ground level air are dependent on latitude and on the distribution of land masses and oceans. Lower values were found in tropical and polar regions than in temperate areas.[164] Concentrations in the southern hemisphere are lower than those in the northern hemisphere, because the land surfaces from which radon is exhaled are smaller in the southern hemisphere. The observed range of the average continental Pb^{210} concentrations vary from 0.002 to 0.016 pCi/m^3 air (excluding Antarctica). Data reported by other authors for selected locations were of the same order of magnitude.[140, 142-144, 146, 165-169] Only a small fraction of the Po^{210} in equilibrium with Pb^{210} is present in the lower troposphere.

90. Yearly deposition rates of 1.7 mCi Pb^{210}/km^2 were measured for the United Kingdom,[140] and 2.4 mCi Pb^{210}/km^2 were reported from France.[166] Under equilibrium conditions, Pb^{210} and Po^{210} activity in upper soil layers could thus amount to some 60 mCi/km^2 each.

IV. The transfer of environmental radio-activity to man

INTRODUCTION

91. Naturally-occurring radio-active nuclides enter the body mainly through food and water, inhalation being of secondary importance. The information on K^{40}, C^{14} and tritium transport through the food chain has remained unaltered since the 1962 report was issued; this subject, therefore, will not be discussed again here. Rb^{87} accompanies potassium in metabolic processes, and doses to man due to this element, which is commonly distributed in the biosphere, will be dealt with in paragraph 136.

92. As was pointed out earlier, the radio-active elements of the uranium series in soil are seldom in equilibrium because of their different chemical properties which determine the varying behaviour of these elements in the processes of weathering, soil formation, redistribution of minerals in the lithosphere, etc. Uranium is taken up by plants; however, because of its low specific activity and the low concentrations found in the biosphere, its radio-activity may be disregarded. Thorium is absorbed very poorly by the root systems.[174-177] Therefore, only radium isotopes (Ra^{226}, Ra^{228}), and possibly lead (Pb^{210}) and polonium (Po^{210}), among the long-lived nuclides of both the uranium and thorium series are taken up by plants to a degree that may be important when further transfer of radio-activity to animals and to man is considered. On the other hand, significant amounts of Po^{210} and Pb^{210} are deposited on plants from the atmosphere.

93. Ra^{226} and Ra^{228} decay, directly or indirectly, to Rn^{222} and Rn^{220}, respectively. The latter are chemically inactive gases. They leave the soil and, together with their short- and long-lived daughter products, form the most significant part of the natural radio-activity that is inhaled by man.

94. *Areas of "normal" activity.* As reported in the 1962 report, the content of Ra^{226} in soil varies considerably with geological and geochemical conditions. The levels reported from the United States, which are possibly typical,[178] are of the order of 0.09 to 0.8 pCi/g soil. No extensive measurements of Ra^{228}, or Th^{228} have recently been reported. Because of the short half-

life of Ra^{228}, it may be assumed that its concentration in soils is closely related to that of its parent, Th^{232}. From the data on the escape of radon from soil and on deposition of Pb^{210} from the atmosphere it has been postulated that equilibrium levels of this nuclide in the few upper inches of soil should be about 60 mCi/km^2 (paragraph 90).

95. *Areas of high natural radio-activity.* Those areas in the State of Kerala in India, and the States of Rio de Janeiro and Espirito Santo in Brazil, where levels of radio-activity in soil are high because of the presence of monazite sands of high thorium content, were discussed in the 1962 report. Since then, additional data have been reported on the availability of radio-active elements from these soils to plants[177, 179, 180] and will be discussed in paragraphs 110 and 111, together with the data on accessibility to plants of Ra^{226} and Ra^{228} in the high background area of volcanic intrusives in the State of Minas Gerais, Brazil.

Intake by man

96. In addition to K^{40}, Rb^{87} and C^{14}, which are not considered in this section, radium (Ra^{226} and Ra^{228}), radon (Rn^{222}), radio-lead (Pb^{210}) and polonium (Po^{210}) contribute appreciable doses to man through food. When inhaled, radon (Rn^{222} and Rn^{220}) with their short-lived decay products, and possibly radio-lead (Pb^{210}), contribute to lung doses and must also be considered.

Water

97. The relative importance of drinking water as a source of natural radio-activity in human diet was studied by numerous investigators.[117, 120, 123, 129, 165, 180-182]

98. Sanitary (usually chemical and physico-chemical) treatment of water, as commonly applied in a majority of urban centres in a number of countries, may remove large proportions—up to 90 per cent—of Ra^{226} and Pb^{210}.[117, 122] Water-softening procedures seem to be especially effective in this respect. Chemical treatment (e.g chlorination and subsequent aeration) and storage may reduce the radon level in tap water, and boiling may further remove radon from the water which is actually consumed.

Typical levels of natural radio-nuclides in water

99. Water consumption contributes only a small percentage to the total intake of the nuclide (of the order of a few to 10 per cent—table X). As indicated in the 1962 report, the waters consumed by the majority of the world population usually contain less than 0.1 pCi/litre Ra^{226}.

100. Typical concentrations of Rn^{222} in waters derived from lakes, rivers and shallow wells, which are consumed by the majority of the world's population, are indicated in table VIII.

101. The contribution of drinking water to the total intake of Pb^{210} and Po^{210} has been estimated to be of the order of 1-10 per cent for the typical diet in the United States.[165]

Areas with high concentrations of radium and radon in drinking water

102. In some areas, where Ra^{226} concentrations in water of several pCi/litre or even more are not uncommon, water may be a significant or even a dominant contributor to the total intake of Ra^{226}. Thus, in rather exceptional situations, involving populations relatively limited in size (less than 1 per cent of the population of the country as a whole in the cases investigated so far), the total intake of Ra^{226} may be twice as high as the average for the country (Cornwall in the United Kingdom),[123] or even higher by an order of magnitude (some regions of the midwestern States of the United States).[122, 182, 183] However, in the monazite area in the State of Kerala, India, where elevated intakes of radium were noted, the relative role of water as a contributor of radium was limited to about 10 per cent, because of higher concentrations of Ra^{226} both in water and foodstuffs of plant origin from local sources.[180]

103. Consumption of water with high concentrations of dissolved radon (deep wells in some areas, and spa waters which show concentrations of about 20 to 200 nCi/litre, with the highest known value of 5,000 nCi/litre)[129] is rather exceptional. In some areas, such as those found in the States of Maine and New Hamphire in the United States,[184] well waters supplying a population of the order of 100,000[130] contain radon at average concentrations of about 16 and 30 nCi/litre. Average concentrations of 1 to a few nCi/litre were found in central Sweden[129] (population of about 300,000), Cornwall[123] (population of about 300,000), the Federal Republic of Germany and elsewhere (see also 1962 report). Many factors discussed in paragraph 98 reduce the intake considerably. The possible significance of doses from radon taken in with water will be discussed in paragraph 148.

Foodstuffs

Areas of typical "normal" levels of intake

104. The total intake of Ra^{226} and the contribution of different food categories was studied in the United Kingdom,[181, 185] the United States,[180-189] and India.[180] The average estimates reported for areas with apparently normal concentrations of Ra^{226} in drinking water and foodstuffs vary between 0.7 and 5 pCi/day (table X), while smaller variations in the values of the Ra/Ca ratio have been reported.

105. In both the United States[187] and the United Kingdom[181, 185] the main sources of radium in the diet are not the same as those from which the main proportion of calcium is derived (table X). In all the circumstances investigated so far, foodstuffs of plant origin (cereals and grain products, fruits and vegetables) form a major source of dietary Ra^{226}. The relative availability for absorption in the gastro-intestinal tract of radium from water and different foodstuffs is unknown.

106. The daily intake in very low-income groups in Puerto Rico[190] is slightly less than 1 pCi/day. However, the calcium intake is also low, and thus the resulting Ra^{226}/Ca ratio in the diet is similar to that in other regions of the United States. It could be inferred from the data of Chhabra[180] that a similar picture may obtain in India—at least for the area investigated—where the intakes of both radium and calcium seem to be low.[2]

107. The intake of Ra^{228} has not been studied on a scale comparable to that of Ra^{226}, but the data derived from alpha-spectrometric measurements in different materials, including water, foodstuffs and human tissues,[191, 192] indicate that the intake of Ra^{228} is, on the average, about two to four times less than that of Ra^{226}.

108. The Pb^{210} intake has not been specifically determined, and only some indirect estimates are avail-

able[165] which show that, on the average, the total intake per day should be of the order of 1-10 pCi. This seems to be typical for most regions, with the exceptions discussed in paragraph 113. The contributions of different foodstuffs are not known, but those of plant origin probably play the dominant role.

109. The estimated usual intake of Po^{210} is of the order of several pCi per day,[193, 194] in agreement with the results of total dietary analyses.[189] The data are consistent with a faecal excretion of 1.8 pCi/day as determined by Holtzman[165] or of 1.7-6.4 pCi/day as found by Hill.[193] The data indicate that the main sources are fresh leafy vegetables and, in some circumstances, a few animal tissues (kidney, liver).

Special food chain mechanisms and areas of high natural radio-activity

110. Information on the intake of natural radio-elements in areas of high radio-activity in the soil is very limited. In Brazil, locally grown foodstuffs with higher than normal radio-activity are found in some localities near the towns of Araxa and Tapira, in the State of Minas Gerais.[179] In these areas, the concentrations of Ra^{226} and Ra^{228} in edible plants are about one to two orders of magnitude higher than normal. However, estimates of intake in this area are not yet available. Food from the monazite areas of Brazil contains only the normal amount of radio-activity from both the uranium and the thorium series. This seems to be due to the very low solubility of the minerals of which the monazite sands are composed.

111. In India, in the high activity area of Kerala State, recent studies[180] have revealed that the radium intake due to locally grown plant foodstuffs may be higher by a factor of four to five than in the Bombay area, and is still considered within the range of values typical of other countries.

112. Brazil nuts are known to accumulate the heavier alkaline earth elements, radium and barium, to an unusual extent. This effect has been observed in samples from Brazil, Malaya and Guyana;[181, 195, 196] concentrations in kernals usually range from one to five pCi Ra^{226}/gramme. Thus the quantity of Ra^{226} in one gramme of Brazil nuts is comparable to the estimated average daily intake of the population shown in table X. It is not known whether the radium from Brazil nuts is absorbed in the gastro-intestinal tract of man to the same extent as radium in other foods.

113. In the 1964 report (annex A, paragraphs 118, 128), a special food chain mechanism, transferring unusually high activities of Cs^{137} from lichens through reindeer or caribou meat to man, was discussed. The same applies to Pb^{210} and Po^{210} of natural origin, and fragmentary data are available (table XV), showing high concentrations of both Pb^{210} and Po^{210} in lichens and in the meat of reindeer and caribou, which graze in arctic regions.[193, 197] It was postulated that the dietary Pb^{210} and Po^{210} intakes of people subsisting on large amounts of caribou or reindeer meat may be elevated by an order of magnitude above the typical values observed elsewhere.[197] This is supported by still fragmentary evidence of Pb^{210} and Po^{210} in human tissues from northern Canada[198] and Finnish Lapland[199] (paragraph 133).

Air

114. *Normal concentrations.* The normal concentrations of radon and thoron in ground level atmospheric air have been discussed in paragraphs 79, 85 and 86. The usual levels to which human populations are exposed, however, are heavily influenced by indoor concentrations. Indoor concentrations are normally higher than those found outside because of the accumulation of radon and thoron from building materials in closed or poorly ventilated premises. The problem was reviewed in the 1962 report, and no recent data are available which would warrant a change in the position adopted therein. It will be maintained, therefore, that the typical average effective concentration of Rn^{222} to which the world population is exposed is about 500 pCi/m^3. Daughter products, from Po^{218} through Bi^{214}, are assumed to be present in concentrations between 100 and 500 pCi/m^3. Corresponding average concentrations of thoron daughter products (Po^{216}, Pb^{212} and Po^{212}) are based on an estimate by Jacobi[200] and have been assumed to equal about 600, 10 and 10 pCi/m^3, respectively.

115. *High concentrations.* It needs to be mentioned here that, because of their occupation in underground mines, some groups of workers are exposed to air concentrations of Rn^{222} and daughter products higher than usual (paragraph 88). The fraction of the population subjected to this type of exposure, even in industrialized countries, does not exceed a fraction of 1 per cent. The importance of the problem and the health hazards involved were discussed in the 1964 report (annex B, paragraphs 152-174) and elsewhere.[201]

116. The downward flow of cold air gives rise to increased concentrations of radon at the bottom of valleys. Rn^{222} values ten times higher than the night concentrations over flat country have been observed in valleys by Servant.[202]

LEVELS IN MAN

117. Our knowledge of concentrations of naturally-occurring radio-active potassium (K^{40}), carbon (C^{14}), tritium (H^3) and beryllium (Be^7) in human tissues and of their distribution in different organs has remained essentially unchanged since the 1962 report was issued, but substantial new information has become available on radium isotopes and nuclides of the Pb^{210}-Bi^{210}-Po^{210} chain.

Metabolic data on Ra^{226} and the Pb^{210}-Bi^{210}-Po^{210} chain

118. Several investigators have shown recently[203, 204] that about 80-85 per cent of radium in the body is contained in the skeleton, the remaining fraction being distributed approximately uniformly in soft tissue as shown in table XIII. The concentration of radium per gramme of ash from soft tissues is similar to that in the skeleton, but is lower by a factor of about twenty when calculated on a fresh weight basis.[203]

119. The concentration of Ra^{226} per gramme of body ash of man is constant throughout the entire life span, starting from the earliest period of foetal life (four months) in which the determinations were possible.[205] The rise of body calcium and radium in the process of growth is apparently parallel, and no significant discrimination between the two elements seems to take place at the placental barrier. The total body burden becomes constant in adult age and displays the organ distribution described in the foregoing paragraph.

120. Under steady-state conditions the ratio of radium to calcium in the body contrasts markedly with that in diet. The observed ratio (OR), defined as

$$\frac{\text{Ra/Ca in bone}}{\text{Ra/Ca in diet}},$$

for adults is about 0.016.[186, 190] This compares with ratios of about 0.25 for strontium and 0.08 for barium.[207] The consistency of the OR for radium under different conditions of dietary composition suggests that the content in bone is determined by the dietary ratio of the two elements.[190]

121. The content of Pb^{210} in the human body has been the subject of several studies in recent years; the results of measurements are collected in tables XII and XIII. There is apparently little correlation of Pb^{210} with the content of Ra^{226} in bone from the same places.

122. Most of the total Pb^{210} present in the body is in the skeleton.[165, 190] The distribution of Pb^{210} in the skeleton appears to be uniform within a factor of two, with slightly but significantly higher concentrations in trabecular than in compact bone. Concentrations in the bone tissue of males are somewhat (about one-third) higher than in that of females.[165]

123. The contributions of different sources of Pb^{210} to the total body burden are not known in detail. However, inhaled air and dietary intake seem to be the main contributors. The decay of radon in the body (both the atmospheric radon and that formed *in situ* from the disintegration of Ra^{226}) and the intake of Pb^{210} with water seem to account for only a few per cent of the activity present in the human body.[165]

124. The distribution of Pb^{210} in human soft tissues is not known in detail. The ratio of Po^{210} to Pb^{210} is less than unity in most of them with values close to equilibrium in the liver, and with some excess Po^{210} in the kidneys and ovaries.[193] The origin of this excess Po^{210} is still open to question. However, as pointed out by Holtzman,[208] there are kinetical reasons for supposing that by far the largest fraction of Po^{210} in the body, including this so-called "unsupported Po^{210}", comes mainly from the redistribution of that Po^{210} that is formed by radio-active decay of Pb^{210} in the body rather than from direct dietary intake.

125. Hunt *et al.*[209] showed that, despite marked differences between individual teeth and observable variations between trabecular and compact bone in the same person, the average levels of Ra^{226} and Pb^{210} in bone were closely correlated with those in teeth. The average concentrations of radium in bone and teeth were equal, whereas those for Pb^{210} were twice as high in bone. It may be concluded, therefore, that teeth analyses for Ra^{226} may be used to study average population levels in the skeleton when bone material is not available and when no significant and prolonged changes in the rate of intake are expected. On the other hand, as the length of the physical half-life of Pb^{210} ($T_{½} = 19.4$ years) is comparable with the human life span, and the mineral turnover of the skeleton may significantly differ from that of the teeth, the Pb^{210} concentration in teeth as a measure of skeletal levels demands further study.

Normal levels of natural radio-activity in man

126. Ra^{226} has repeatedly been determined in human tissues by several investigators. The results published since 1962 are collected in tables XI and XIII.

127. The Ra^{226} values as reported in table XI indicate that bone levels in most areas of normal dietary intake of this nuclide are similar, the most frequent values clustering around $1\text{-}1.5 \times 10^{-2}$ pCi/g ash, corresponding to an average of about 30 to 40 pCi Ra^{226} in the skeleton of a standard man (assuming an average of 2,800 grammes of ash in the skeleton). This value is substantially lower than the value of 60 pCi accepted in the 1962 report. It seems appropriate therefore to suggest, for the purpose of dose-rate calculation, a value of 30 pCi Ra^{226} as typical of the whole skeleton burden in areas of normal rates of intake.

128. Ra^{228} is the only important long-lived element of the thorium series that is absorbed to a significant degree from the gastro-intestinal tract. Ra^{228} decays through Ac^{228} to Th^{228} ($T_{½} = 1.9$ years), which in turn decays through a series of short-lived nuclides to the stable isotope Pb^{208}. Very small fractions of the amounts of radio-active decay products of Th^{228} found in bone leave the skeletal deposition site of Ra^{228} and Th^{228}.[210] The latter nuclide has recently been determined in bone, together with Ra^{226}. In two studies, the average ratio between activities of Th^{228} and Ra^{226} in bone ash varied between 0.25[183] and 0.4.[203] These values are consistent with results reported earlier,[211] and it seems appropriate to accept that the value of 0.3 to 0.4 is closer to the real situation than the figure of 0.7, which was assumed in the 1962 report. Thus, a natural skeletal burden of 10 pCi Th^{228} in a standard man seems typical.

129. From table XII it follows that the typical Pb^{210} burden in the skeleton of an adult is about 270 pCi. As only a small fraction of Po^{210} formed by the radio-active decay of Pb^{210} and Bi^{210} in bone is removed from its site of formation, the activities of all three nuclides in the skeleton are close to radio-active equilibrium.[165, 203, 212-214] It seems that, in contrast to the 1962 report where a ratio of Po^{210}/Pb^{210} of 0.5 was assumed, a value of about 0.9 is closer to reality.

Areas of high natural radio-activity and special food chain mechanisms

130. In some areas of the midwestern States of the United States, where concentrations of Ra^{226} in drinking water of about 1 to 10 pCi/litre are not uncommon, Ra^{226} levels in bone are three to four times higher than those typical elsewhere (table XI). The population of the area concerned is about 1,000,000.[122]

131. Preliminary data[215] from those areas of high natural radio-activity in Brazil where high activities of radium in plants of local origin were found (towns of Araxa and Tapira, State of Minas Gerais) show that the concentrations of Ra^{226} in teeth collected from the inhabitants are higher than in "normal" areas but only by a factor of two to three (table XIV). In view of the fact that a significant correlation exists between concentrations of Ra^{226} in bones and teeth[209] (paragraph 125), these observations suggest that the skeletal levels of Ra^{226} in this area of Brazil might be elevated by a similar factor. The population living in the areas of Araxa and Tapira is about 15,000.

132. The data from the monazite area in Kerala State in India are even more scanty. As shown in table XI, in five samples of human bone analyzed so far,[180, 216] the concentrations of radium were on the average higher than those typical for "normal" areas by one order of magnitude. The population residing in the monazite area of Kerala State amounts to about 80,000 people.

133. As discussed in paragraph 113, high concentrations of Pb^{210} and Po^{210} in lichens and in caribou and reindeer meat were found in arctic regions. Fragmentary data available on Pb^{210} and Po^{210} show that human beings subsisting to a significant degree on caribou meat display levels of both nuclides that differ widely from those usually found in the northern temperate zone. Thus, Hill[198] was able to demonstrate that

the average concentration of Po^{210} in placentae of Eskimo women from northern Canada who consumed large amounts of caribou, reindeer or moose meat amounted to 59 pCi/kg fresh weight, whereas in women subsisting on a diet normal for England and Canada 3.3 and 5.0 pCi/kg were found. Kauranen and Miettinen[109] reported levels of Po^{210} in the blood of reindeer-breeding Lapps which were eight times higher than those in the blood of southern Finns. A similar trend was observed for Pb^{210} in Eskimo bones, as can be inferred from table XII. The size of the populations living in the arctic regions and subsisting on reindeer or caribou meat as one of their main dietary items is difficult to evaluate but may be as high as 1,000,000.

Doses from internal irradiation by natural radio-active nuclides

134. Dose rates in millirads per year from internal sources of natural radiation to the gonads, to cells lining Haversian canals, to osteocytes and to the bone marrow contained in trabecular bone have been assembled in table XVI. The per cent contributions of alpha radiation to the total dose rates are also indicated.

135. It is realized that for the purpose of comparing risks, doses in rads ought to be weighted by appropriate RBE values. In the case of alpha emitters, however, the information on RBE is so uncertain and the values proposed by various authors so widely different that it is reasonable to consider them as still unknown, although it is possible that they may be higher than one.[67] If this were so, and if unweighted dose rates from natural sources as given in table XVI were used as a standard for obtaining comparative risks from other, man-made, sources, the comparative risks would thus be over-estimated depending on the RBE values applying to any given effect.

136. The estimates of doses from K^{40}, C^{14} and Rn^{222} and short-lived daughter products in soft tissues other than the respiratory tract are the same as those given in the 1962 report. Other values have been recalculated on the basis of different estimates of tissue concentrations and/or by introducing some changes in the method of dose calculation. Doses to gonads from Rb^{87} amount to about 0.3 mrad/year; they are certainly less in osteocytes and in bone marrow.

137. The doses of alpha radiation to osteocytes and Haversian canals from Ra^{226} and daughter products, Ra^{228} and daughter products, and Po^{210} have been calculated by the method of Spiers.[217] The assumed diameters of the cavities were 5 μ for the osteocytic lacunae and 50 μ for the Haversian canals. The values of the geometrical factors for Ra^{226} were those calculated by Charlton and Cormack,[218] assuming one-third retention of Rn^{222} and its short-lived daughter products (through Po^{214}), while those for Ra^{228} and daughters in equilibrium and for Po^{210} were those given by Stahlhofen.[208] Doses to bone from beta emission of all nuclides of these series were ignored because the percentage of energy delivered from beta decay in bone constitutes, under the conditions of equilibrium assumed, only about 2, 4 and 7 per cent of the total energy of the Ra^{226}, Ra^{228} and Pb^{210} chains, respectively.[b] The dose of beta radiation to bone marrow from these series was also ignored as it amounted to approximately 0.1 millirad per year.

138. The alpha dose rates in fresh bone from individual nuclides that result from these calculations are significantly higher than those obtained in 1962. The doses from Ra^{226} and Ra^{228} differ only slightly from previous estimates, whereas those from Po^{210} are definitely higher, both because higher concentrations have now been assumed and because a different method of dose computation has been used.

139. In the present review, the dose rate to the marrow contained in trabecular bone from Po^{210} has been assessed on the basis of the presumed concentration of the nuclide in soft tissues (~3 pCi/kg fresh weight) rather than in the mineralized bone itself as was done in the 1962 report.

140. If the total annual dose estimates from internal sources presented in table XVI are compared with the dose estimates given in the 1962 report expressed in millirads, very little difference is seen.

141. At the time when the 1962 report was adopted, it was realized that the irradiation of the respiratory tract from natural sources exceeded that of any other organ of the human body. On the other hand, only approximate calculations of average doses to different volumes of tissues in the respiratory tract were possible. Since then, however, significant progress has been made in better understanding the mechanisms by which daughter products of Rn^{222} and Rn^{220} are deposited and transported in the respiratory tract of man.

142. Thus, Altshuler *et al.*[170] have calculated the doses of alpha radiation from Po^{218} and Po^{214}. These doses account for almost the whole of the dose to alveolar, bronchiolar, bronchial and tracheal epithelia, and, in estimating them, allowance was made for the distribution of radio-activity (Po^{218}, Pb^{214}, Po^{214}) between free ions and natural aerosols of varying particle size, for the deposition of ions and particles in different parts of the respiratory tract, for the upward transport of radio-activity in the bronchial tree with the flow of mucus, and for the physical and anatomical factors involved in the penetration of alpha particles through the mucus and the bronchial epithelium.

143. A similar study has been made by Jacobi[200] for both Rn^{222} and daughter products, and Rn^{220} and daughter products by applying somewhat different physical and anatomical criteria. In both studies, it was assumed that the critical irradiated tissue was the basal layer of cells of the bronchial epithelium.

144. Both studies yielded similar results, pointing to the fact that the dose rates from Po^{218} and Po^{214} alpha particles are highest in the epithelium of segmental and lobar bronchi. With average concentrations of Po^{218}, Po^{214} and Bi^{214}, each between 100 and 500 pCi/m^3, the results of both studies indicate that the dose rate in these parts of the respiratory system is of the order of several hundreds of millirads per year, the doses to alveolar tissues and to bronchioli being lower by two and to the trachea by one order of magnitude. At average concentrations of Po^{216}, Pb^{212} and Bi^{212} of 600, 10 and 10 pCi/m^3, respectively, as are assumed for the purpose of the present annex, the irradiation from these nuclides adds only another few per cent to the values given above for doses from Po^{218} and Po^{214}.[200]

145. The quantitative agreement between the results of the two studies may be somewhat fortuitous, and further studies are necessary to provide sounder infor-

[b] Recent evidence indicates that it is the irradiation of the endosteal cells lining the inner surface of bone, rather than that of osteocytes or the cells of Haversian canals, which is of relevance to the induction of bone tumours. The doses delivered to endosteal cells will normally be less than those quoted in this report for osteocytes and will often also be less than those given for cells of the Haversian canals.

mation on several of the critical parameters, especially those of a physiological and anatomical nature.[219, 220] It must be mentioned that higher doses may be received if some uptake and retention of radio-activity by the tissues of the respiratory tract is assumed, but such a possibility is neglected at present for lack of relevant information.[170]

146. On the other hand, rapid dissociation of Po^{218}, Pb^{214} and Bi^{214} atoms (or ions) from aerosol particles deposited in the respiratory tract with subsequent direct transfer to the blood stream and other organs could reduce the dose significantly. That such a possibility exists was demonstrated by Pohl.[221] Even so, however, it is still uncertain whether the demonstrated resorption of radon decay products takes place predominantly from the alveolar regions or from the bronchial tree, which would be more significant from the dosimetric point of view. In any case, the doses to the critical cells in some areas of bronchial epithelium thus obtained seem higher by at least one order of magnitude than those to the whole respiratory organ that were accepted in the 1962 report.

147. An additional exposure to natural polonium may result from cigarette smoking.[193, 222-228] Average alpha-radiation doses to the respiratory tract of smokers from excess Po^{210} deposition is not expected to exceed about 1 mrad/year. In some areas of bronchial epithelium where concentrations up to 0.3 pCi/gramme have been detected,[228] the alpha dose due to Po^{210} might reach the level of some tens of millirads per year. The biological significance of this irradiation is unknown, but it appears most unlikely to be appreciable in view of the low doses involved.

148. The metabolism of radon ingested with water and the doses attributable to this source of radio-activity were studied by von Döbeln and Lindell[129] and by Hursh *et al.*[130] The dose per unit of activity ingested seems highest in the stomach (~20 mrad/μCi Rn^{222}), those to other organs being lower by two orders of magnitude. At normal or typical concentrations of radon in drinking water of surface origin, which are of the order of a few picocuries per litre,[123] the doses are negligible. In those areas where concentration of Rn^{222} in drinking water is of the order of nanocuries per litre, the doses to the stomach could be correspondingly higher—of the order of a few millirads per year.

V. Recapitulation of dose rates

149. Estimates of dose rates to man from natural sources are summarized in table XVII. For comparison, the estimates given in the 1962 report but expressed in millirads per year are also included. The differences between the two sets of estimates are slight. The dose rates to cells lining Haversian canals, however, include a larger contribution from alpha particles, whereas those to the gonads and bone marrow include a smaller contribution than the dose rates obtained in 1962. Dose rates to the lung tissues are not given in the table since no exact estimate is available, but those to the epithelium of segmental and lobar bronchi are believed to be of the order of several hundreds of millirads per year (paragraph 144).

TABLE I. PROPERTIES OF THE MAIN SECONDARY PARTICLES IN COSMIC RADIATION

Particle	*Electric charge*	*Rest mass*		*Mean lifetime seconds*	*Decay products*
		MeV	*Electron masses*		
n	0	940	1,839	10^3	$e^- + \nu + p$
p	+1	938	1,836	Stable	—
$\pi^{\pm}$	± 1	140	273	2.5×10^{-8}	$\mu^{\pm} + \nu$
π^0	0	135	264	2×10^{-16}	2γ
$\mu^{\pm}$	± 1	106	207	2.2×10^{-6}	$e^{\pm} + 2\nu$
$e^{\pm}$	± 1	0.511	1	Stable*	—

*A positron (e^+) is annihilated by combining with an electron, and two photons are emitted.

TABLE II. SOME RADIO-NUCLIDES PRODUCED BY COSMIC RAYS[31-40]

Radio-nuclide	*Half-life*	*Maximum energy of beta radiation in keV*	*Main mode of formation*	*Calculated atmospheric production rate (atoms/cm² × year)*	*Calculated concentration in the lower troposphere (pCi/m³)*
H-3	12.3y	18	Spallation of N^{14} or O^{16}	8×10^6	5×10^{-2}
Be-7	53d	Electron capture	Spallation of N^{14} or O^{16}	2.5×10^6	0.5
Be-10	2.7×10^6y	550	Spallation of N^{14} or O^{16}	$1.4\text{-}2.6 \times 10^6$	5×10^{-8}
C-14	5,760y	165	N^{14} (n, p) C^{14}	$50\text{-}65 \times 10^6$	1.3–1.6
Na-22 ...	2.6y	540	Spallation of A^{40}	1.8×10^3	5×10^{-5}
Si-32	700y	100	Spallation of A^{40}	$5\text{-}6 \times 10^3$	8×10^{-7}

TABLE II. SOME RADIO-NUCLIDES PRODUCED BY COSMIC RAYS[31–40] (*continued*)

Radio-nuclide	*Half-life*	*Maximum energy of beta radiation in keV*	*Main mode of formation*	*Calculated atmospheric production rate (atoms/cm² × year)*	*Calculated concentration in the lower troposphere (pCi/m³)*
P–32	14.3d	1,720	Spallation of A^{40}	2.5×10^4	1.1×10^{-2}
P–33	25d	250	Spallation of A^{40}	2.1×10^4	6×10^{-3}
S–35	87d	165	Spallation of A^{40}	4.4×10^4	6×10^{-3}
Cl–36	3.1×10^5y	710	Spallation of A^{40}	3.5×10^4	1.2×10^{-8}

TABLE III. THE IONIZING COMPONENT OF COSMIC RAYS AT SEA LEVEL

Author	*Year*	*Ion pairs/cm³ × second (STP)*	*mrad/year*
UNSCEAR[2]	1962	1.90 – 1.96	28
Shamos and Liboff[65]	1965	2.2 ± 0.06	28.5 ± 0.8
Herbst[64]	1963		28
Kastner[60]	1965	2.2 ± 0.1	29 ± 1.3
Lowder and Beck[18]	1965	2.1 ± 0.1	27.6 ± 1.3
Lillicrap[10]	1965	*	26 ± 1.5

* Energy absorbed in a water Čerenkov detector.

TABLE IV. URANIUM SERIES[a]

Isotope	*Atomic number*	*Historical name*	*Half-life*	*Alpha and/or beta ray energies (MeV)*[b]	*Gamma ray energies (MeV)*[b]
U-238	92	Uranium I	4.5×10^9 y	α4.18(77), 4.13(23)	
Th–234	90	Uranium X_1	24.1 d	β0.19(65), 0.10(35)	0.09(15), 0.06(7), 0.03(7)
Pa–234	91	Uranium X_2	1.18 min	β2.31(93), 1.45(6), 0.55(1)	1.01(2), 0.77(1), 0.04(3)
U–234	92	Uranium II	2.50×10^5 y	α4.77(72), 4.72(28)	0.05(28)
Th–230	90	Ionium	8.0×10^4 y	α4.68(76), 4.62(24)	
Ra–226	88	Radium	1,622 y	α4.78(94), 4.59(6)	0.19(4)
Rn–222	86	Radon	3.82 d	α5.48(100)	
Po–218	84	Radium A[c]	3.05 min	α6.00(100)	
Pb–214	82	Radium B[c]	26.8 min	β1.03(6), 0.66(40) 0.46(50), 0.40(4)	0.35(44), 0.29(24), 0.24(11), 0.05(2)
Bi–214	83	Radium C[c]	19.7 min	β3.18(15), 2.56(4), 1.79(8), 1.33(33), 1.03(22), 0.74(20)	2.43(2), 2.20(6), 2.12(1), 1.85(3), 1.76(19), 1.73(2), 1.51(3), 1.42(4), 1.38(7), 1.28(2), 1.24(7), 1.16(2), 1.12(20), 0.94(5), 0 81(2), 0.77(7), 0.61(45)
Po–214	84	Radium C'[c]	160×10^{-6} sec	α7.68(100)	
Pb–210	82	Radium D[c]	19.4 y	β0.06(17), 0.02(83)	0.05(4)
Bi–210	83	Radium E[c]	5.0 d	β1.16(100)	
Po–210	84	Radium F	138.4 d	α5.30(100)	
Pb–206	82	Radium G	Stable		

[a] Based on reference 230.
[b] Figures in parentheses indicate per cent yield per disintegration.
[c] Parallel decay branches of less than 1 per cent are not listed.

TABLE V. THORIUM SERIES[a]

Isotope	Atomic number	Historical name	Half-life	Alpha and/or beta ray energies (MeV)[b]	Gamma ray energies (MeV)[b]
Th-232 ...	90	Thorium	1.41×10^{10} y	α4.01(76), 3.95(24)	0.06(24)
Ra-228 ...	88	Mesothorium I	5.8 y[c]	β0.05(100)	
Ac-228 ...	89	Mesothorium II	6.13 h	β2.18(10), 1.85(9), 1.72(7), 1.13(53), 0.64(8), 0.45(13)	1.64(13), 1.59(12), 1.10, 1.04, 0.97(18), 0.91(25), 0.46(3), 0.41(2), 0.34(11), 0.23, 0.18(3), 0.13(6), 0.11, 0.10, 0.08
Th-228 ..	90	Radio-thorium	1.91 y	α5.42(72), 5.34(28)	0.08(2)
Ra-224 ...	88	Thorium X	3.64 d	α5.68(95), 5.45(5)	0.24(5)
Rn-220 ...	86	Thoron	54.5 sec	α6.28(99 +)	
Po-216 ...	84	Thorium A[d]	0.158 sec	α6.78(100)	
Pb-212 ...	82	Thorium B	10.64 h	β0.58(14), 0.34(80), 0.16(6)	0.30(5), 0.24(82), 0.18(1), 0.12(2)
Bi-212 ...	83	Thorium C	60.5 min	α(35%) 6.09(10), 6.04(25) β(65%) 2.25(56), 1.52(4), 0.74(1), 0.63(2)	(35%) 0.04(1), (65%) 2.20(2), 1.81(1), 1.61(3), 1.34(2), 1.04(2), 0.83(8), 0.73(10)
Po-212 ...	84	Thorium C′	0.30×10^{-6} sec	α8.78(100)	
Tl-208 ...	81	Thorium C″	3.1 min	β2.37(2), 1.79(47), 1.52, 1.25	2.62(100), 0.86(14), 0.76(2), 0.58(83), 0.51(25), 0.28(9), 0.25(2)
Pb-208 ...	82	Thorium D	Stable		

[a] Based on reference 230, except where otherwise indicated.
[b] Figures in parentheses indicate per cent yield per disintegration.
[c] From reference 231.
[d] Parallel decay branches of less than 1 per cent are not listed.

TABLE VI. ACTINIUM SERIES[a]

Isotope	Atomic number	Historical name	Half-life	Alpha and/or beta ray energies (MeV)[b]	Gamma ray energies (MeV)[b]
U-235	92	Actinouranium	7.13×10^{8} y	α4.59(5), 4.55(4), 4.50(1), 4.41(4), 4.39(57), 4.36(18), 4.32(3), 4.21(6)	0.204(6), 0.185(54), 0.164(5), 0.143(11), 0.110(3)
Th-231 ...	90	Uranium Y	25.64 hr	β0.30(45), 0.22(20), 0.13(20), 0.09(15)	0.095(2), 0.084(7), 0.026(12)
Pa-231 ...	91	Protoactinium	3.47×10^{4} y	α5.05(11), 5.02(23), 5.00(25), 4.97(2), 4.94(23), 4.84(1), 4.72(10), 4.68(3)	0.33(1), 0.30(2), 0.10(2), 0.06(13), 0.04(15), 0.029(90), 0.025(11), 0.02(4), 0.0165(20)
Ac-227 ...	89	Actinium	21.6 y	α(1.2%) 4.94(1), others (weak) β(98.8%) 0.046(100)	
Fr-223 ...	87	Actinium K	21 min	β1.15(100)	0.08(24), 0.05(40)
Th-227 ...	90	Radio-actinium	18.17 d	α6.04(23), 6.01(3), 5.98(24), 5.96(4), 5.87(3), 5.76(21), 5.70(19), 5.67(2)	0.33(7), 0.31(4), 0.30(5), 0.29(2), 0.28(2), 0.26(7), 0.24(13), 0.17(1), 0.11(4), 0.10(1), 0.08(5), 0.06(9), 0.05(16), 0.03(39)
Ra-223 ...	88	Actinium X	11.68 d	α5.87(1), 5.75(10), 5.71(52), 5.61(25), 5.54(9), 5.50(1), 5.43(2)	0.45(1), 0.34(7), 0.27(13), 0.15(11)
Rn-219 ...	86	Actinon[c]	3.92 sec	α6.81(80), 6.54(13), 6.42(7)	0.40(5), 0.27(9)

TABLE VI. ACTINIUM SERIES[a] (*continued*)

Isotope	*Atomic number*	*Historical name*	*Half-life*	*Alpha and/or beta ray energies (MeV)*[b]	*Gamma ray energies (MeV)*[b]
Po-215 ...	84	Actinium A	1.83×10^{-3} sec	α7.37(100)	
Pb-211 ...	82	Actinium B	36.1 min	β1.36(92), 0.95(1), 0.53(6), 0.25(1)	0.83(4), 0.70(1), 0.43(1), 0.40(4)
Bi-211 ...	83	Actinium C[c]	2.16 min	α6.62(83), 6.27(17)	0.35(14)
Tl-207 ...	81	Actinium C″	4.76 min	β1.47(100)	0.87(1)
Pb-207 ...	82	Actinium D	Stable		

[a] Compiled by W. M. Lowder from "Nuclear Data Sheets".
[b] Figures in parentheses indicate per cent yield per disintegration.
[c] Parallel decay branches of less than 1 per cent are not listed.

TABLE VII. SOME NON-SERIES PRIMORDIAL RADIO-ISOTOPES[2, 111, 229]

Isotope	*Abundance in the litosphere (parts per million)*	*Half-life (years)*	*Alpha or beta ray energies (MeV)*[a]	*Gamma ray energies (MeV)*[a]
K-40	3	1.3×10^{9}	β1.32(89)	1.46(11)
V-50	0.2	5×10^{14}	Electron capture	0.71, 1.59
Rb-87	75	4.7×10^{10}	β0.27(100)	
In-115	0.1	6×10^{14}	β0.6(100)	
La-138	0.01	1.1×10^{11}	β0.20(30)	0.81(30), 1.43(70)
Sm-147	1	1.2×10^{11}	α2.24	
Lu-176	0.01	2.1×10^{10}	β0.42(100)	0.088(100), 0.202(100), 0.309(100)

[a] Figures in parentheses indicate per cent yield per disintegration.

TABLE VIII. TYPICAL CONCENTRATIONS OF Ra^{226} AND DAUGHTERS IN CONTINENTAL WATERS (pCi/litre)[36]

	Ra^{226}	Rn^{222}	Pb^{210}	Po^{210}
Spa waters and deep wells	1–10	10^{4}–10^{5}	< 0.1[a]	~ 0.02
Ground water	0.1[a]–1	10^{2}–10^{3}	< 0.1[a]	~ 0.01
Surface water ...	< 1	10	< 0.5	—
Rain-water	—	10^{3}–10^{5} [b]	0.5–3	~ 0.5

[a] Below detection limits.
[b] As determined through presence of short-lived Rn^{222} daughters.

TABLE IX. RECENT DATA (REPORTED SINCE 1962) ON CONCENTRATION OF NATURAL RADIO-ACTIVE NUCLIDES IN WATER

(Concentration in pCi/litre)

Country or area	*Source of water*	Ra^{226}	Ra^{228}	Rn^{222}	Pb^{210}	*References*
AUSTRALIA	Surface reservoirs	0.1-0.2				232
BRAZIL, high background area of Morro di Ferro (volcanic intrusives)	Different sources	11.9 (0.12-107)				233
	Tap, wells and springs	0.3-1.6	0.2-3.2			104
BRAZIL, monazite area	Tap and wells	0.8	0.2-1.6			104
INDIA, normal areas	Tap water (Bombay)	0.02				180
	Surface waters	0.05-0.6[a]				106
	Springs and wells	0.16-0.5[a]				106
INDIA, monazite area of Kerala State	Shallow wells	0.14				180
ISRAEL	Surface waters	< 2		18-180		128
	Springs, wells and boreholes			< 2-21, 300		128
	Dead Sea (lake)	62		20		128
JAPAN	Surface waters (rivers)	0.04-1.4				119
NEW ZEALAND	Artesian wells			~ 0–1,000		234
	Surface waters			0.3		234
SWEDEN	Tap water of varying origin (deep bored wells, inclusive)			~ 1,000 (100-33,000)		129
UNITED STATES						
Illinois	Ground waters			30-300		117, 165
	Raw surface waters				0.127	15, 81, 117, 165
	Treated surface waters				0.019	15, 81, 117, 165
	Raw well water				0.05	15, 81, 117, 165
Florida	Thermal and mineral springs	0.3-3.3				121
South Carolina	Wells	1.4-2.8				235
Utah, near Great Salt Lake	Artesian wells	0.1-2.0		400-1,800		

[a] Including Ra^{223} and Ra^{224}.

TABLE X. ESTIMATES OF TOTAL INTAKE OF Ra^{226} AND OF CONTRIBUTIONS FROM DIFFERENT FOODSTUFF CATEGORIES

	UNITED STATES						*UNITED KINGDOM*[185]	*INDIA*[180]	
Category of foods	*New York, N.Y.*[187]	*Chicago, Ill.*[187]	*San Francisco, Cal.*[187]	*San Juan, P.R.*[190]	*Consumers' Union Five-city study*[188]	*Consumers' Union Teenager twenty-two-city study*[189]	*Country-wide study*	*Bombay*	*Kerala State Monazite area*
Cereals and grain products	0.56	0.76	0.51				0.17	0.41	1.48
Meat, fish, eggs	0.38	0.37	0.28				0.38		
Milk and dairy products	0.14	0.12	0.13				0.14	0.04	0.19
Green vegetables, fruits and pulses	0.81	0.56	0.48				0.32	0.17	0.81
Root vegetables	0.40	0.22	0.26				0.10	0.02[a]	0.07[a]
Water	~ 0.02	~ 0.03	~ 0.01				0.07	0.06	0.29
Total pCi/day	~ 2.3	~ 2.1	~ 1.7	~ 0.7	~ 3 (2.2–4.3)	~ 5 (2.5–6.5)	~ 1.2	~ 0.7	~ 2.8
pCi Ra^{226}/g Ca	2.2	2.0	1.6	1.3	1.9	2.5	1.1		

[a] Miscellaneous.

TABLE XI. Ra^{226} IN HUMAN BONE AS REPORTED AFTER 1962

Location of area	*pCi/g ash*	*pCi/g Ca*	*Total[a] in the skeleton (pCi)*	*References*
	NORMAL AREAS			
CENTRAL AMERICA				
United States				
Puerto Rico	0.006	0.017	17	190
EUROPE				
Federal Republic of Germany	0.013	0.040	36	203
United Kingdom	0.008–0.02			
NORTH AMERICA				
United States				
Illinois	0.012[b]		32	183
New England	0.014		39	209
New York, N.Y.	0.012	0.032	32	186
Rochester, N.Y.	0.010; 0.017		28, 48	204
San Francisco, Cal.	0.0096	0.026	27	186
	HIGH LEVEL AREAS			
ASIA				
India				
State of Kerala (monazite area)	0.096 (0.03–0.14)		~ 270	180, 216
NORTH AMERICA				
United States				
Illinois	0.037[c]		~ 100	165
Illinois	0.028[c]		78	183

[a] Skeleton of 7,000 g fresh weight yielding 2,800 g ash was assumed.
[b] In people consuming water with "normal" levels of Ra^{226}.
[c] In people consuming water with elevated Ra^{228} concentration.

TABLE XII. Pb^{210} AND Po^{210} IN HUMAN BONE

(Number of samples in brackets)

Area or location	*Pb^{210}*		*Po^{210}*		*Po^{210}/Pb^{210}*	*Total Po^{210} in the skeleton[a] (pCi)*	*References*
	pCi/g fresh bone	*pCi/g ash*	*pCi/g fresh bone*	*pCi/g ash*			
	NORMAL AREAS						
FEDERAL REPUBLIC OF GERMANY	0.032 (20)	0.11 (20)	0.031 (20)	0.13 (20)	1	290	213
	0.037		0.030		0.8	210	203
POLAND	0.040 (20)					250	237
UNITED KINGDOM	0.026 (5)					160	238
			0.017 (6)			120	239
			0.017 (9)			120	214
UNITED STATES							
Illinois (mostly)		0.150 (128)			1	410	165
Illinois		0.080 (32)				200	183
New England		0.140 (25)				360	209
Rochester, N.Y.	0.015 (18)					95	212
San Juan, P.R.		0.120 (28)				300	240
	HIGH LEVEL AREAS						
CANADA (Eskimos)	0.140 [0.08–0.71] (10)					960	193

[a] Calculated directly from Po^{210} or from data on Pb^{210}. In the latter case, if no data for Po^{210}/Pb^{210} ratio were reported the value of 0.9 was assumed. Skeleton of 7,000 g fresh weight yielding 2,800 g ash was further assumed. If the data were reported both for fresh tissue and tissue ash, the values in pCi/g ash were used to estimate the total skeletal burden.

TABLE XIII. NATURAL ALPHA EMITTING RADIO-NUCLIDES IN HUMAN SOFT TISSUES IN AREAS OF NORMAL BACKGROUND RADIO-ACTIVITY
VALUES ARE GIVEN IN pCi/kg FRESH WEIGHT

For Po^{210} the ranges include the average values reported by various authors

Tissue	*Ra^{226}*	*Th^{228}*	*Po^{210}*
Soft tissues in general	~ 0.1[203, 206]	~ 0.04[203]	~ 3[203]
Liver	~ 0.2[204]	—	11–17[116, 193, 198, 203, 214, 239]
Kidney	0.1[204]	—	5–17[193, 198, 203, 214, 239]
Gonads	—	—	3–4[193, 198, 214, 239]
Spleen	0.1[204]	—	3[203, 239, 240]
Lung	—	—	2–5[193, 198, 214, 228, 239]
Skeletal muscle	0.06[204]	—	1–6[165, 193, 203, 239]

TABLE XIV. Ra^{226} IN HUMAN TEETH FROM DIFFERENT AREAS

Values in pCi/g ash; number of samples in parentheses

Locality	*Mean*	*Range*	*References*	*Remarks*
		NORMAL AREAS		
BRAZIL				
Vitoria	0.030	0.008–0.079 (14)	215	
Rio de Janeiro	0.037	0.006–0.123 (13)	215	
Poços de Caldas	0.015	0.006–0.031 (13)	215	
UNITED STATES				
New England	0.016	0.01–0.062 (25)	209	
		AREAS OF HIGH TERRESTRIAL RADIO-ACTIVITY		
BRAZIL				
Guarapari	0.036	0.006–0.104 (23)	215	Monazite sand areas
Meaipe	0.023	0.006–0.077 (15)	215	Monazite sand areas
Araxa and Tapira	0.077	0.008–0.204 (52)	215	Areas of high radio-activity due to volcanic intrusives

TABLE XV. Po^{210} CONTENT OF VEGETABLES AND ANIMAL TISSUES[193]

Materials	*Number of samples*	*Po^{210} specific activity (pCi/kg)*	*Pb^{210}/Po^{210} activity ratio*
Grass (dried) United Kingdom	24	400–16,000	1–5
Dry lichen (*Caloplaca elegans*) United Kingdom	2	7,800; 10,000	1
Dry lichen (*Cladonia alpestris*) Lapland	3	6,600–8,100	1
Dry lichen (*Cladonia alpestris*) Canada	1	3,500	1
Edible green vegetables (United Kingdom)	5	6–90	1–3
Carrots and potatoes (United Kingdom)	2	1	—
Breads and cereals (United Kingdom)	4	1–7	—
Dried milk powder (United Kingdom)	3	2–6	—
Beef and lamb muscle (United Kingdom)	2	3; 3	—
Beef and lamb liver (United Kingdom)	3	4–100	0.7
Beef and lamb kidney (United Kingdom)	3	48–270	0.05–1
Lamb kidney (north Wales)	6	90–1,800	0.2
Reindeer (Lapland) summer killed			
Muscle	6	15–50	—
Liver	5	350–750	—
Kidney	4	110–490	—
Reindeer (Canada, North-west Territory) winter killed			
Muscle	2	200; 210	—
Liver	2	2,400; 5,600	—
Kidney	2	4,200; 2,300	—
Spleen	1	980	—
Cockles (United Kingdom, east and west coasts)	3	400–900	0.1–0.2
Crab (United Kingdom, south coast)	2	1,300; 1,400	—
Plankton (south Pacific)	1	2,000	—

TABLE XVI. INTERNAL DOSES FROM NATURAL RADIO-ACTIVITY;[a] VALUES IN MILLIRADS PER YEAR

(In parentheses, fraction in per cent of total yearly dose derived from alpha radiation)

Nuclide	Gonads	Bone		Bone marrow (trabecular bone)
		Haversian canals (50 μ diameter)	Osteocytes (50 μ diameter)	
K^{40}	20	15	15	15
Rb^{87}	0.3	< 0.3	< 0.3	< 0.3
C^{14}	0.7	1.6	1.6	1.6
Ra^{226}	—[e]	0.6[f, d]	1.4[f, d]	0.03[f]
Ra^{228}	—[e]	0.7[f, b]	1.1[f, b]	0.03[f]
Po^{210}	0.3[f]	2.1[f, c]	4.2[f, c]	0.3[f]
Rn^{222}	0.3[f]	0.3[f]	0.3[f]	0.3[f]
TOTAL	21.6	20.3	23.6	17.3
	(3)	(18)	(30)	(4)

[a] Doses of alpha radiation to tissues of respiratory tract are discussed in paragraphs 141-148. The highest doses are most probably those delivered to basal layers of bronchial epithelium in segmental and lobar bronchi. They may reach the level of some hundreds of millirads per year at assumed average concentrations of Po^{218}, Pb^{214} and Po^{214}.

[b] Accepted concentration of Ra^{228} (in equilibrium with daughter products) of 1.4×10^{-3} pCi/g fresh bone.

[c] Accepted concentration of Po^{210} of 3.3×10^{-2} pCi/g fresh bone.

[d] Accepted concentration of Ra^{226} (plus one-third of short-lived daughter products through Po^{214}) of 4.3×10^{-3} pCi/g fresh bone.

[e] Doses to gonads from Ra^{226} and daughter products and Ra^{228} and daughter products could not be estimated with reasonable accuracy. The upper limits seem to be 0.02 and 0.03 mrad/year, respectively.

[f] Doses of alpha radiation.

TABLE XVII. DOSE RATES DUE TO EXTERNAL AND INTERNAL IRRADIATION FROM NATURAL SOURCES IN "NORMAL" AREAS

(In italics, estimates given in the 1962 report)

Source of irradiation	Dose rates (mrad/y)			Paragraphs
	Gonads	Haversian canal	Bone marrow	
External irradiation				
Cosmic rays				
Ionizing component	28	28	28	48
	28	*28*	*28*	
Neutrons	0.7	0.7	0.7	49
	2.5	*2.5*	*2.5*	
Terrestrial radiation (including air)	50	50	50	58
	50	*50*	*50*	
Internal irradiation				
K^{40}	20	15	15	136
	20	*15*	*15*	
Rb^{87}	0.3	< 0.3	< 0.3	136
C^{14}	0.7	1.6	1.6	136
	0.7	*1.6*	*1.6*	
Ra^{226}	—	0.6	0.03	135–139
	0.05	*0.54*	*0.06*	
Ra^{228}	—	0.7	0.03	135–139
	0.08	*0.86*	*0.1*	
Po^{210}	0.3	2.1	0.3	135–139
	0.03	*0.36*	*0.04*	
Rn^{222} (dissolved in tissues)	0.3	0.3	0.3	135–139
	0.3	*0.03*	*0.3*	
TOTAL[a]	100	99	96	
	102	*99*	*98*	
Percentage from alpha particles and neutrons	1.3	4.4	1.4	
	3	*2.8*	*3*	

[a] Totals were rounded off to two significant figures.

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Annex B

ENVIRONMENTAL CONTAMINATION

CONTENTS

I. Airborne and deposited artificial radio-activity

INTRODUCTION

1. There have been no major weapons tests between the end of 1962 and the date of the present report (June 1966). The atmospheric tests in central Asia in October 1964 and in May 1965 and the several accidental releases from low-yield underground tests that have occurred are known to have contributed negligibly to human exposure. Data on an additional Asian test explosion in May 1966 are still not sufficient to permit complete evaluation.

2. Fall-out, however, still provides the major contribution to radio-active contamination of the environment. The purpose of the present report is to update the evaluations of fall-out from the earlier major series of tests made by the Committee in 1964[1] and of the doses to which man has been thereby committed.

3. The cessation of injections of fission products into the stratosphere since the end of 1962 has facilitated the study of the transport mechanisms of nuclear debris and of the depletion process of the stratospheric reservoir that the Committee had discussed at some length in its 1962 and 1964 reports.[1, 2] The reader is referred to these reports for a general outline of the problems of transfer and distribution of radio-activity injected into the atmosphere.

4. There have been indications that fall-out over the oceans may be greater than on equivalent land surfaces. This is a new subject and is therefore treated in some detail in this report.

5. Space activities can also be a potential source of accidental contamination, as shown by the unplanned re-entry into the atmosphere of a spacecraft carrying a power source containing Pu^{238}. As a consequence of the burning up of this device, Pu^{238} was released into the atmosphere. Its distribution will be dealt with later.

6. Local contamination took place in Spain in January 1966 when, as a result of an air collision between an aircraft carrying nuclear weapons and a refuelling plane, fissionable materials were scattered over a limited area by the unexploded devices.[3]

7. Industrial, medical and research applications of nuclear technology contribute only to a very limited extent to environmental contamination and to the attendant exposure of populations. Some gaseous wastes are discharged in limited amounts directly into the atmosphere from reactors and fuel reprocessing plants, while, as discussed in the 1962 report, some low activity liquid wastes are diluted in sea and rivers during normal operations.

8. Doses to the world population from either accidental or controlled releases of wastes have been negligible so far in comparison with those due to nuclear explosions, particularly those carried out above ground. While it is realized that, if large-scale atmospheric testing is not resumed, other sources of contamination

from the peaceful uses of atomic energy may in the future contribute comparatively more to environmental contamination, the present review will mainly be devoted to contamination from nuclear explosions.

Atmospheric injections

Tropospheric tests

9. Two fission devices were exploded above ground in central Asia on 16 October 1964 and on 14 May 1965. The debris of the first device reached Japan two days after the explosion, was detected in North America and Europe about one week later and persisted in some locations for about three weeks. Most of the debris was limited to the band from 20°N to 80°N by the time it reached 80°W.[4-21]

10. Short-lived fission products such as Sr^{89}, Zr^{95}, I^{131}, Ba-La^{140} and Ce^{141} were identified at several sampling sites and 2-10 mCi/km² of short-lived debris were deposited in the United States of America during October-December 1964.[22] Half removal times from the troposphere of five to over twenty days were reported for barium-140.[4, 17, 23] The first evidence of Ba^{140} at Gracefield, New Zealand (41°S 175°E), was found in the monthly sample of December 1964, six to ten weeks following the detonation.[24]

11. According to preliminary data, debris of the second nuclear device, detonated on 14 May 1965, followed tropospheric pathways similar to those followed by debris from the first test and with comparable activity concentrations. Traces of short-lived activity were detected in the United States and elsewhere eight to twelve days after the shot.[16, 17, 20, 25-30] Relatively high concentrations of Np^{239} and some activity due to U^{237} were observed a few days after each detonation in Japan[19, 31] as well as in high altitude samples collected by Indian aircraft.[16]

12. These two explosions were estimated to have contributed about 2 per cent of the long-lived activity in the troposphere that was measured in the monthly samples immediately following each explosion. The contribution became practically insignificant thereafter.[17]

Underground tests

13. Some accidental releases of fission products into the troposphere occurred from at least three low-yield underground tests since March 1964.[17, 20, 31-38] A relative enrichment of volatile radio-isotopes and their radio-active daughters in surface air was expected.[36, 39, 40] The contribution of these events to the radiation doses received by the world population is negligible. Doses in the proximity of the vented tests are not known with any precision.

Stratospheric injection of plutonium-238

14. The only stratospheric injection of radio-active material since December 1962 has consisted of the 17,000 Ci Pu^{238} (alpha emitter, half-life eighty-six years) from a radio-isotope power source (SNAP-9A) which burned up on 21 April 1964 above the Indian Ocean upon re-entry of a spacecraft into the atmosphere. The nuclide was probably released at an altitude of about 50 km, mainly in the form of submicron particles.[41-44] Pu^{238} from this burn-up was first detected in high altitude balloon samples collected at 33 km and 34°S in August 1964, and was also found at 28 km and 34°S in September 1964.[45, 46] In the northern hemisphere (35°N) Pu^{238} was first identified in samples collected at an altitude of 33 km in January 1965.[47] By June 1965, it was found in samples from altitudes exceeding 28 km, at 35°N and higher northern latitudes.[47]

15. By May 1965, the Pu^{238} attributable to the SNAP-9A power source had descended into the lower stratosphere of the southern hemisphere. None was detectable in the lower stratosphere of the northern hemisphere by June 1965.[47-50]

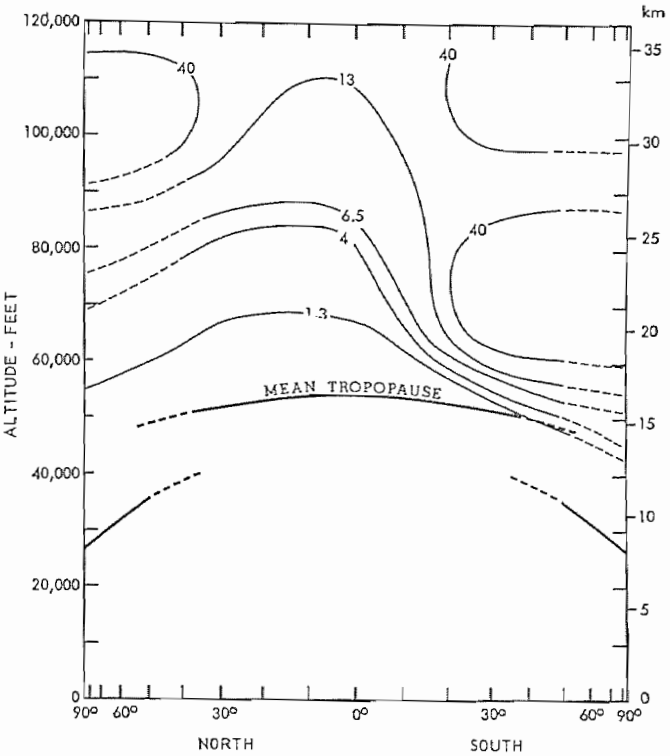


Figure 1. Global atmospheric distribution of Pu^{238}, September-November 1965[64] (pCi/1,000 kg air)

16. Figure 1 shows the global atmospheric distribution of SNAP-9A Pu^{238} a year and a half after its injection, in units of 1×10^{-3} pCi/kg air.[64] Integrating the concentration pattern obtaining one year after the injection indicates that about 10^4 Ci Pu^{238} (approximately 60 per cent of the total) were then present in the stratosphere between 22 and 36 km.[48] Part of the unaccounted for 7×10^3 Ci were probably still above 36 km in the southern hemisphere. Data available so far[44, 47, 48, 50, 51] confirm the general trend predicted by Harley[42] and Machta[43] concerning Pu^{238} concentrations expected in the atmosphere. These predictions were based on the information gathered from the follow-up of the radio-active tracers Rh^{102} and Cd^{109} that were introduced into the stratosphere by high altitude explosions at about 17°N and 43 km in August 1958, and at 17°N and 400 km in July 1962, respectively.[42, 43, 48, 50, 52]

17. Average concentrations of Pu^{238} oxide of the order of 10^{-5} pCi/m³ are expected to persist in surface air during 1965 to 1968,[50] corresponding to about one alpha disintegration due to Pu^{238} per month per m³. This is well below the detection limit of most sampling stations; however, concentrations ten times as great may be reached at some locations. Further predictions should await additional data.

Inventories

Stratosphere

18. Stratospheric sampling by aircraft between the tropopause and 21 km was continued during 1964 and 1965.[49, 51, 53-56] Higher altitudes up to 35 km were sampled by balloons.[57-60] Because of sampling limitations, the inventories obtained from these data by interpolation and integration may not be better than ± 25 per cent.[61]

19. Recent data refer mainly to C^{14}, Sr^{90} and Cs^{137} which are relevant to population dose estimates, as well as to certain tracer elements such as Cd^{109}, Mn^{54} or Pu^{238}, the movement and distribution of which might contribute to our understanding of stratospheric motions.

20. The distribution of Sr^{90} in the stratosphere during January 1964 and January 1965 is shown in figures 2 and 3. The corresponding distribution of C^{14} is shown in figures 4 and 5. Integration of the distributions show[51, 62] that the stratospheric burden of Sr^{90} decreased from about 4 ± 1 MCi in January 1964 to about 1.6 ± 0.4 MCi by January 1965, while the C^{14} excess in the stratosphere decreased from about 25 ± 5 × 10^{27} atoms to 15 ± 3 × 10^{27} within the same period.

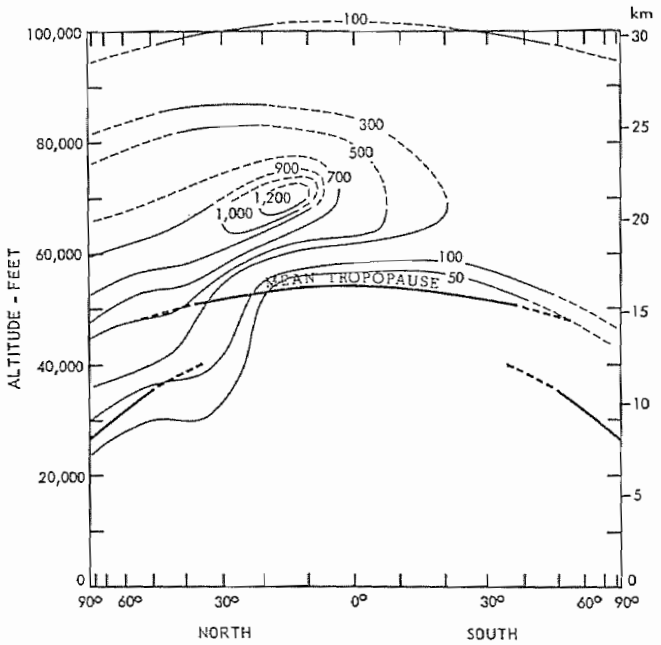


Figure 2. Distribution of Sr^{90} in the stratosphere, January 1964[64] (dpm Sr^{90}/1,000 SCF)[a]

[a] 1 dpm/1,000 SCF = 13 pCi/1,000 kg air.

21. The mean residence time of particulate radioactive debris in the stratosphere, as derived by integration of the results of the stratospheric sampling network for Sr^{90}, was approximately fourteen months during 1963 to 1964.[51, 63] The mean residence time of $C^{14}O_2$ in the stratosphere was about twenty-five months during the same period[51] (about seventeen months for the northern stratosphere alone). These differences in residence times, as well as the different distributions of these two nuclides in the stratosphere, may reflect the facts that some settling of particulates may take place in the atmosphere and that tropospheric air, rich in $C^{14}O_2$ relative to Sr^{90}, re-enters the stratosphere.[64]

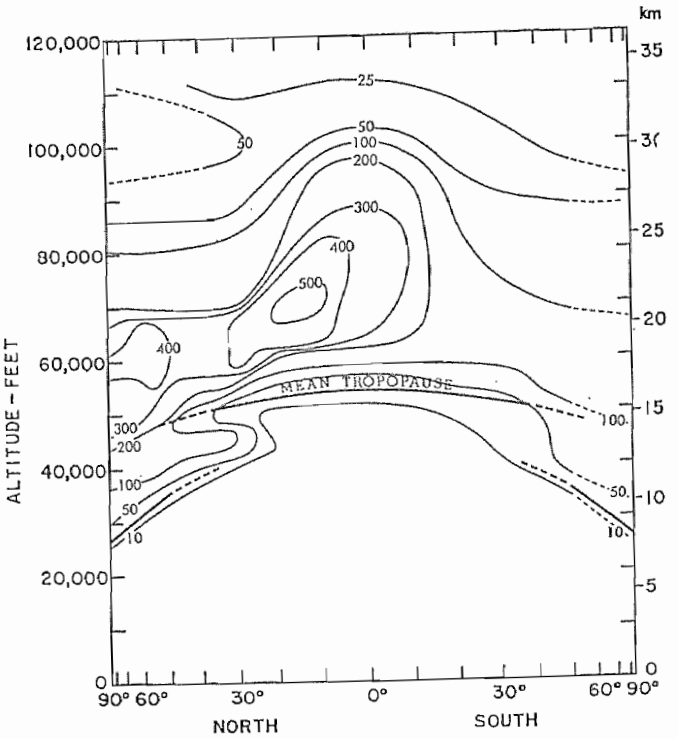


Figure 3. Distribution of Sr^{90} in the stratosphere, January 1965[62] (dpm Sr^{90}/1,000 SCF)[a]

[a] 1 dpm/1,000 SCF = 13 pCi/1,000 kg air.

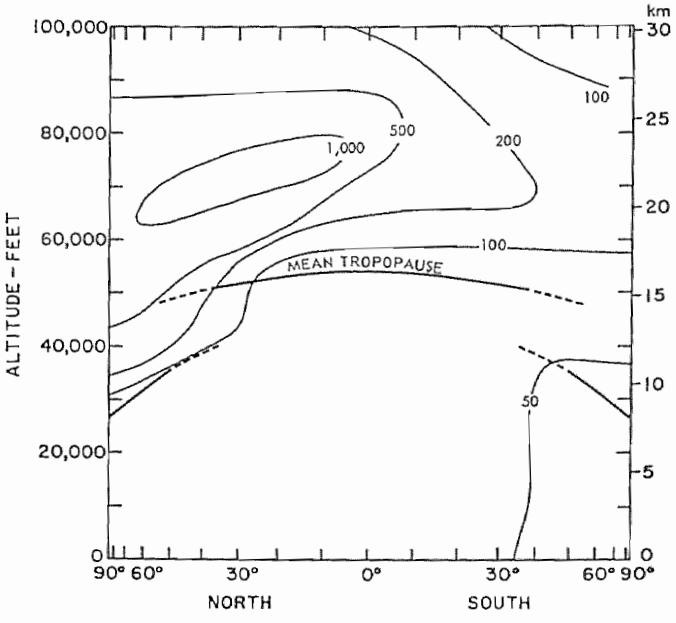


Figure 4. Distribution of C^{14} in the stratosphere, January 1964[64] (10^5 atoms of excess C^{14}/g air)

22. Provisional results since October 1964 of total gamma counts on high altitude samples, when expressed in activity per unit standard volume of air, suggested some accumulation of nuclear debris above 20 km at equatorial latitudes.[57, 58] Balloon samples taken during the period January-April 1965, at 9°N and 20 km also contained activity per unit standard volume of air about two to three times higher than samples collected at 31°, 45° and 65°N at similar altitudes.[60] No final results are available yet from the few samples taken by aircraft in the lower stratosphere at equatorial latitudes during the first half of 1965.

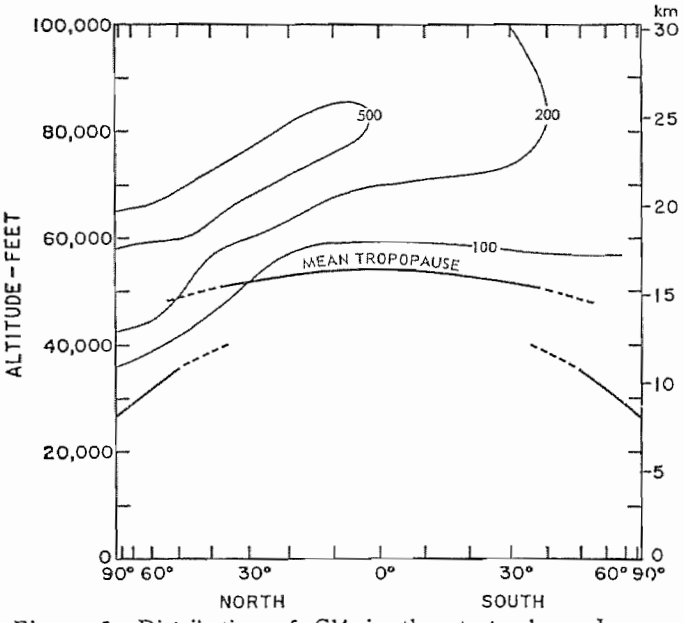


Figure 5. Distribution of C^{14} in the stratosphere, January 1965[62] (10^5 atoms of excess C^{14}/g air)

23. These findings are summarized in figure 3, which reveals two regions of high activity concentrations, one at about 10°N and the other at about 65°N, some 7 km above the tropopause. However, the maximum concentrations above equatorial regions as shown in the figure, rather than reflecting the actual stratospheric distribution of the nuclear debris, are an artifact due to the unit chosen to express concentrations (SCF^{-1}).

24. Figure 6 depicts the same data as figure 3, but expresses concentrations in terms of activity per actual volume of space rather than per unit mass (standard volume) of air.[64] The concentration gradients become steeper above altitudes where the maximum concentrations occur and less steep at lower altitudes. The apparent piling up of activity over the equator is much less pronounced. This type of presentation gives a more direct picture of the activity distribution but does not permit simple calculations of the movement and mixing of air masses. Figure 7 is the corresponding figure for C^{14} concentrations in the stratosphere using the same data as those in figure 5.

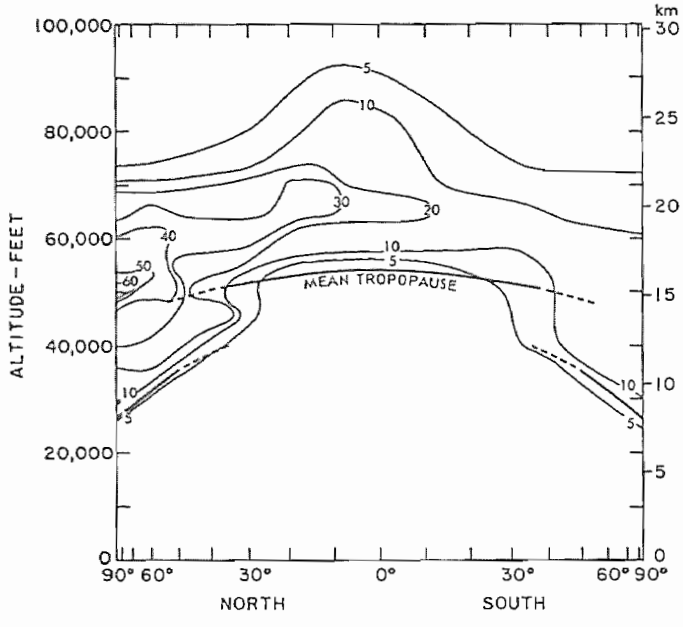


Figure 6. Distribution of Sr^{90} in the stratosphere, January 1965[64] (dpm Sr^{90}/1,000 ambient cubic feet)[a]

[a] 1 dpm/1,000 cubic feet = 15.9 pCi/1,000 m³.

Troposphere

25. *Strontium-90.* Figure 8 shows the average Sr^{90} concentrations in surface air at the United States western hemisphere stations.[1, 62, 65] Average Sr^{90} concentrations for the fourteen stations sampled in the northern hemisphere ranged from about 15 pCi/1,000 m³ to 70 pCi/1,000 m³ in 1963, from about 7 pCi/1,000 m³ to 50 pCi/1,000 m³ in 1964 and continued to decline in 1965. The decrease in Sr^{90} concentrations in the northern hemisphere during 1964 and 1965 is compatible with a fourteen-month mean residence time in the stratosphere.

26. The average concentrations in surface air at seven stations of the southern hemisphere were lower than those encountered in the northern hemisphere by a factor of ten to twenty during 1963 and ranged from about 1.5 to 4 pCi/1,000 m³. They ranged from about 2 pCi/1,000 m³ to 5 pCi/1,000 m³ in 1964 and declined during the first half of 1965. Air concentrations throughout the first half of 1965 were about five times lower than the values for the northern hemisphere.[66]

27. An isopleth representation of Sr^{90} concentrations in surface air as a function of latitude is shown in figure 9, which was derived from results at twenty western hemisphere stations for the years 1963-1965.[67] The data from Chacaltaya, Peru (16°S 68°W), were not included, because the altitude of this station (5,200 m) is such that the results cannot be considered as representative of the surface air concentrations at this latitude. An approach to symmetry between the concentration patterns in each hemisphere is evident when a six-month shift is allowed for between hemispheres.

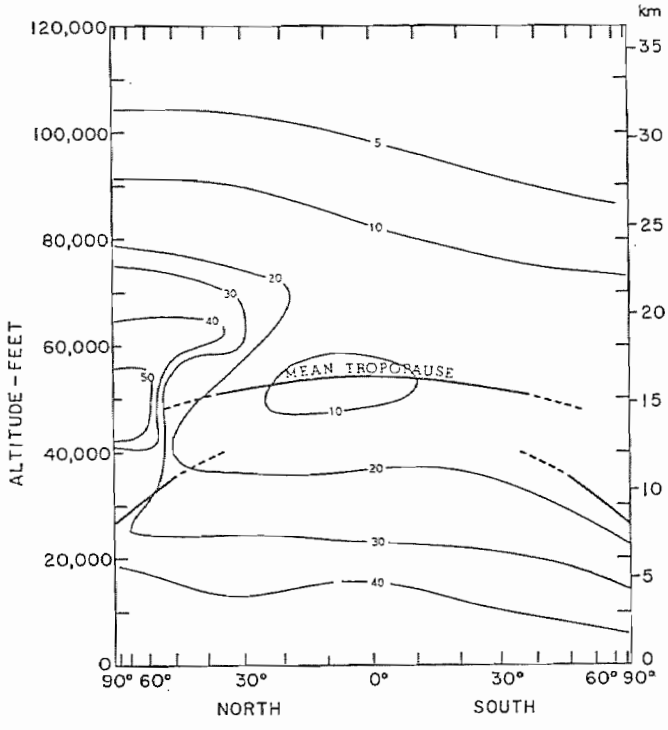


Figure 7. Distribution of excess C^{14} in the stratosphere, January 1965[64] (10^5 atoms C^{14}/0.8 litres ambient air)

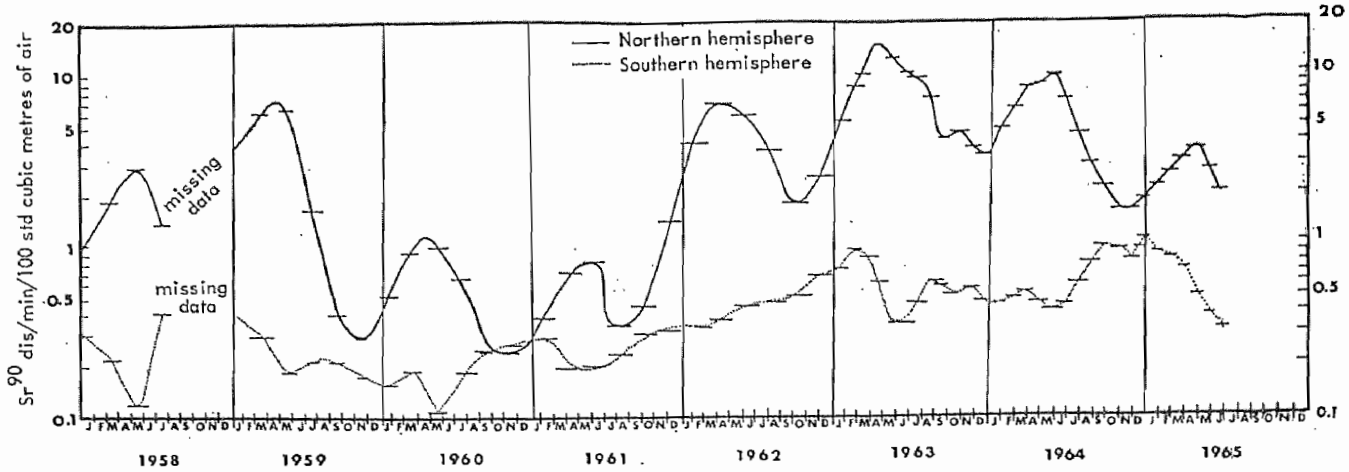


Figure 8. Average Sr^{90} concentrations in surface air (80°W), 1958-1965[65]

28. *Caesium-137.* Figure 10 shows the average monthly Cs^{137} concentrations in surface air in Norway[68, 69] and Sweden[70, 71] since January 1962. Cs^{137} concentrations in the United Kingdom[17, 72] and at Argonne, Illinois, United States,[23, 73, 74] are shown in figure 11. The spring maxima and autumn minima evident in figures 8 and 9 are also obvious in figures 10 and 11. Cs^{137} concentrations were highest at Argonne and of comparable magnitude in Norway, Sweden and the United Kingdom. A decrease in concentrations during 1964 and 1965 as compared to 1963 levels is evident. Measurements in India show similar trends.[75, 76]

29. *Carbon-14.* Excess C^{14} concentrations in the troposphere for each hemisphere are shown in figure 12 for the years 1956-1965.[62] Concentrations in both hemispheres tend to equalize, and they follow closely earlier predictions.[1, 77] Over-all average values of excess C^{14} concentration in the troposphere for 1963 were 60 per cent above the natural level of 74×10^5 atoms C^{14} per gramme of air[78] that obtained before nuclear tests were conducted. The excess of artificial C^{14} continued to increase until mid-1964. Average excess values were about 85 per cent for 1964 and 1965.[62, 78-81]

Deposition on land

30. Samples of deposited activity have been collected in the period under review at more than 200 sites scattered throughout a large part of the land area of the earth's surface.[16-19, 22, 31, 68-70, 72, 76, 82-105] Total beta activities and deposition rates of Sr^{90}, Cs^{137} and other radio-nuclides have been systematically analysed.

31. *Strontium-90.* The monthly Sr^{90} deposition in New York City since 1954[106] and in Buenos Aires since 1959[102] are given in figures 13 and 14, respectively, as examples of rates of deposition in the northern and southern hemispheres. As the rainfall throughout 1964 and 1965 was exceptionally low in the New York area, deposited Sr^{90} levels after August 1964 were somewhat lower than predicted on the basis of a normal precipitation pattern.[107]

32. An isopleth representation of global Sr^{90} deposition rates per month from 60°S to 80°N for the period 1963-1965 is shown in figure 15, based on the sampling network operated by the United States Atomic Energy Commission.[65, 86] The deposition pattern follows closely the surface air concentrations shown in figure 9. Sr^{90} peak deposition values were encountered in the period April-July 1963 between 40°N and 60°N. Comparable values were encountered during the period April-June 1964 at the same latitudes.

33. A general decrease in the yearly deposition rates for the northern hemisphere has been evident since 1963, total Sr^{90} depositions being 2.32, 1.47 and 0.69

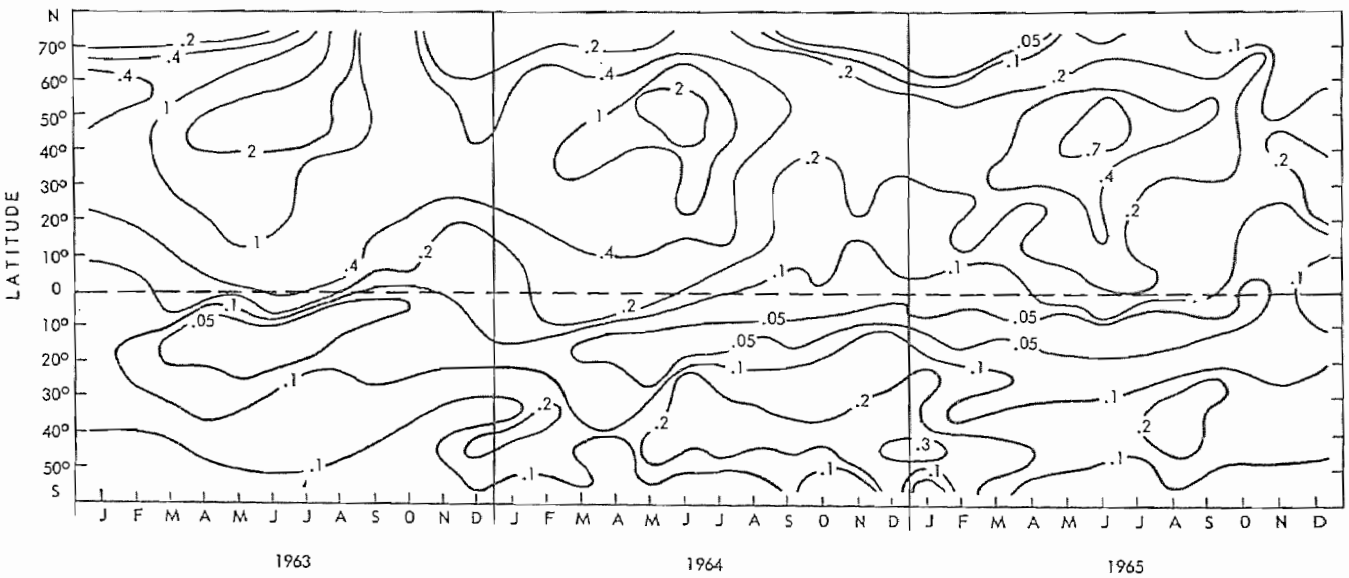


Figure 9. Isopleth representation of Sr^{90} concentrations in surface air, 1963-1965[65, 67] (dpm Sr^{90}/1,000 m^3)

as to a shift in Cs^{137}/Sr^{90} ratios towards lower values since 1963.

Deposition in the oceans

39. Deposition of Sr^{90} in the oceans is mainly of importance in relation to the inventory of Sr^{90}. The dilution of Sr^{90} and other fall-out products by the large volume of the ocean at present suggests minimal doses to man.

40. The oceans cover 70 per cent of the earth's surface, and sampling them presents a different, and,

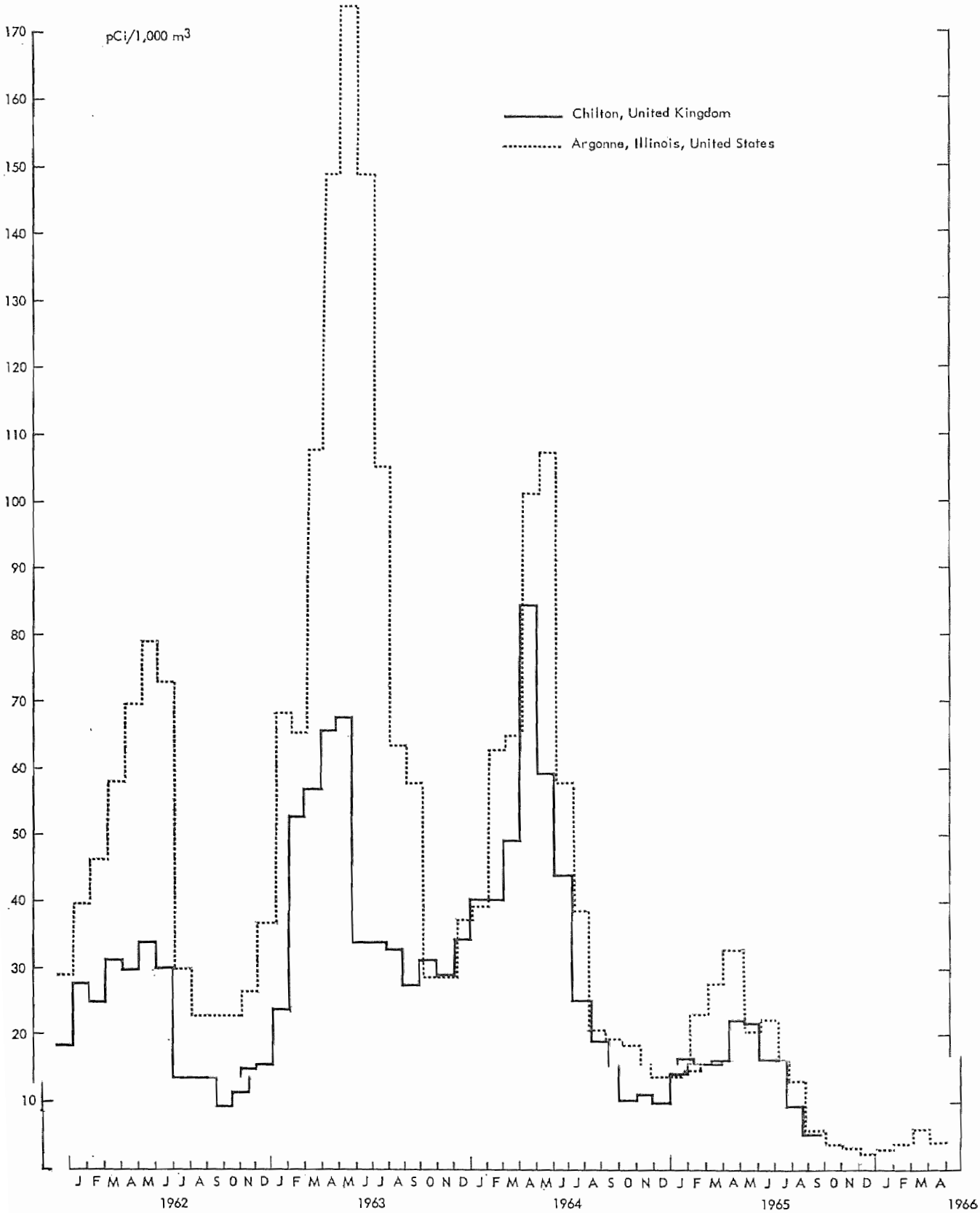
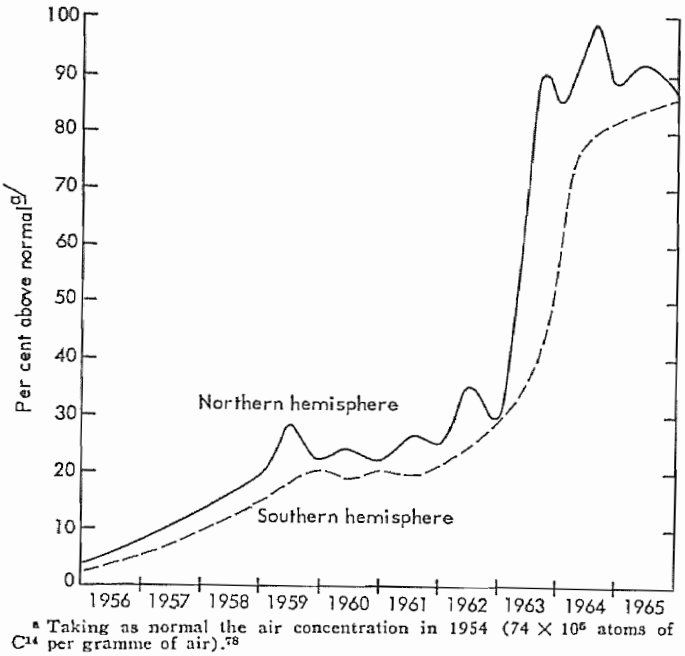


Figure 11. Monthly Cs^{137} concentrations in surface air for Chilton, United Kingdom,[17, 72] and Argonne, Illinois, United States,[23, 73, 74] 1962-1966



[a] Taking as normal the air concentration in 1954 (74×10^6 atoms of C^{14} per gramme of air).[78]

Figure 12. Tropospheric inventory of carbon-14[64]

from some points of view, a more difficult problem than sampling the continents. The depth of the oceans, averaging almost 4,000 metres, and the movement of water are major complicating factors.

41. The radio-chemical analysis of Sr^{90} in ocean water is difficult, particularly the analysis of samples from great depths, since levels are low and large amounts of reagents are required. Blank samples are frequently found to show detectable levels, often of the same order as the measured values.[111-117] This increases the uncertainty of measurement and has cast doubt on some of the results for deep water.

Concentrations of strontium-90 in surface water

42. The Sr^{90} concentration of surface water in the seas and oceans as reported by various investigators has varied greatly as a function of both geography and time. Figures 19 to 22 summarize most of the reported surface water Sr^{90} results through 1961. The variation of Sr^{90} concentrations with latitude in the oceans and certain seas for 1960 and 1961 clearly illustrates that, even under the best circumstances, as in the southern Atlantic and Indian Oceans, the values have a variability of at least a factor of two. Pacific Ocean values have even greater variability, probably reflecting "hot spots"

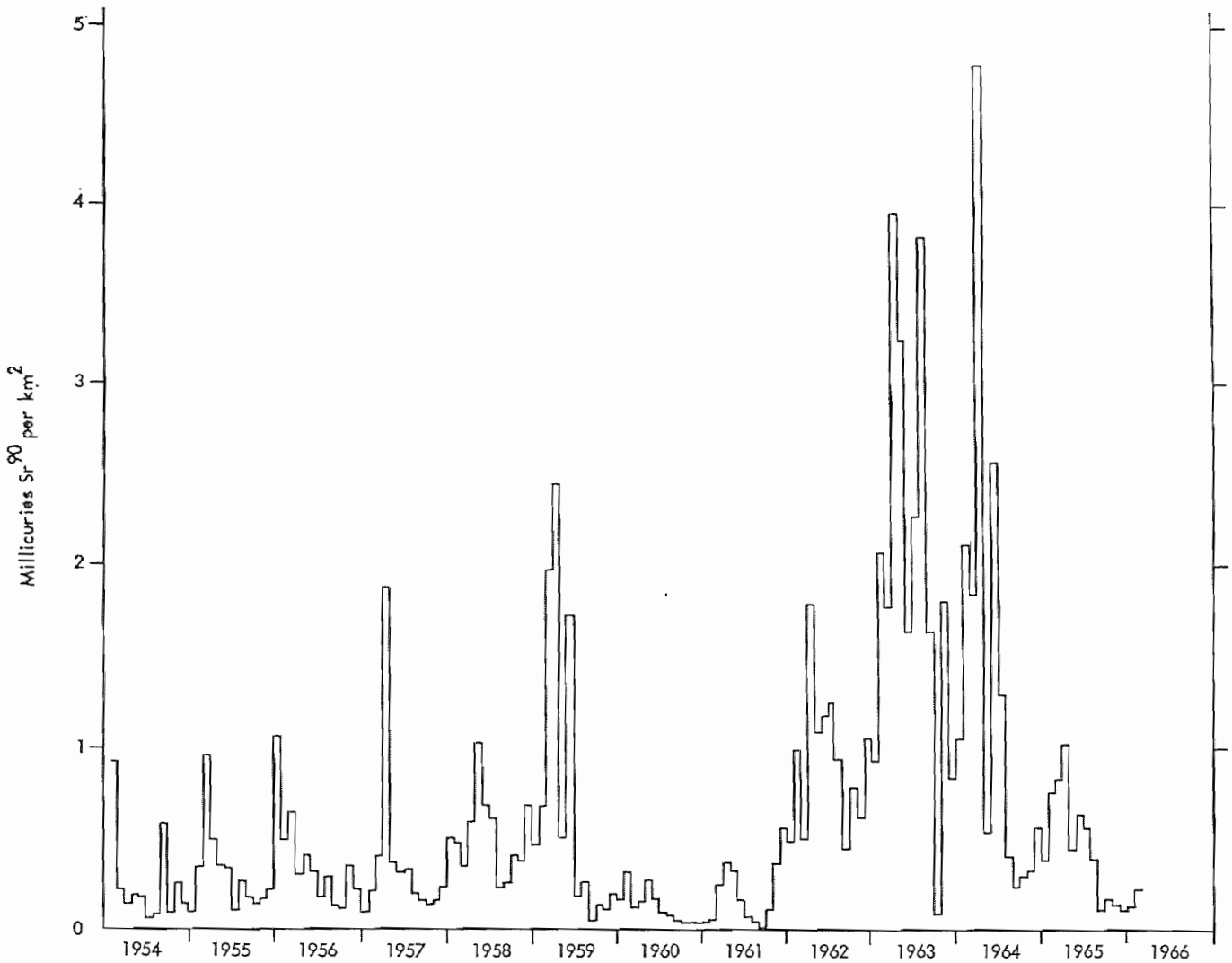


Figure 13. Monthly deposition of Sr^{90} in New York City, 1954-1966[106]

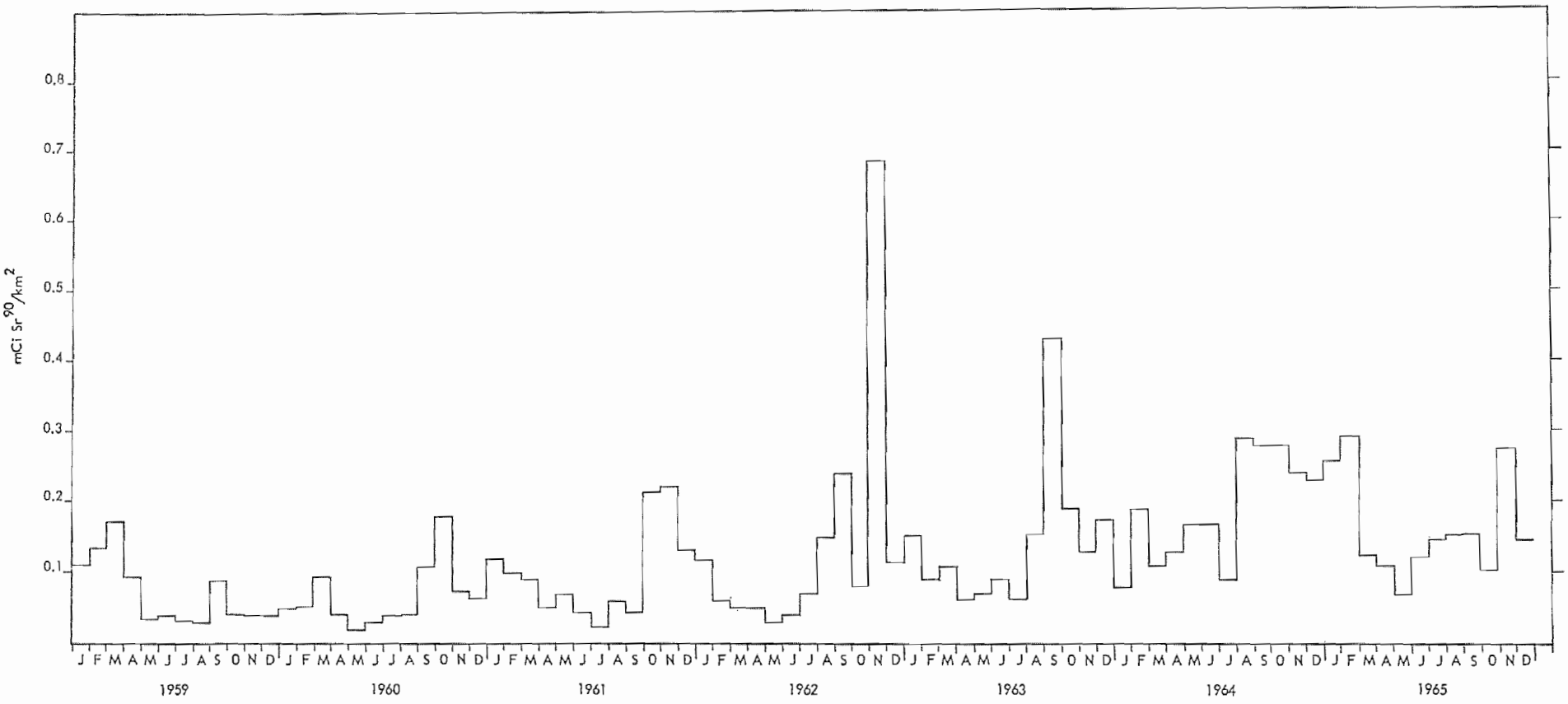


Figure 14. Monthly deposition of Sr^{90} in Buenos Aires, Argentina, 1959-1965[215]

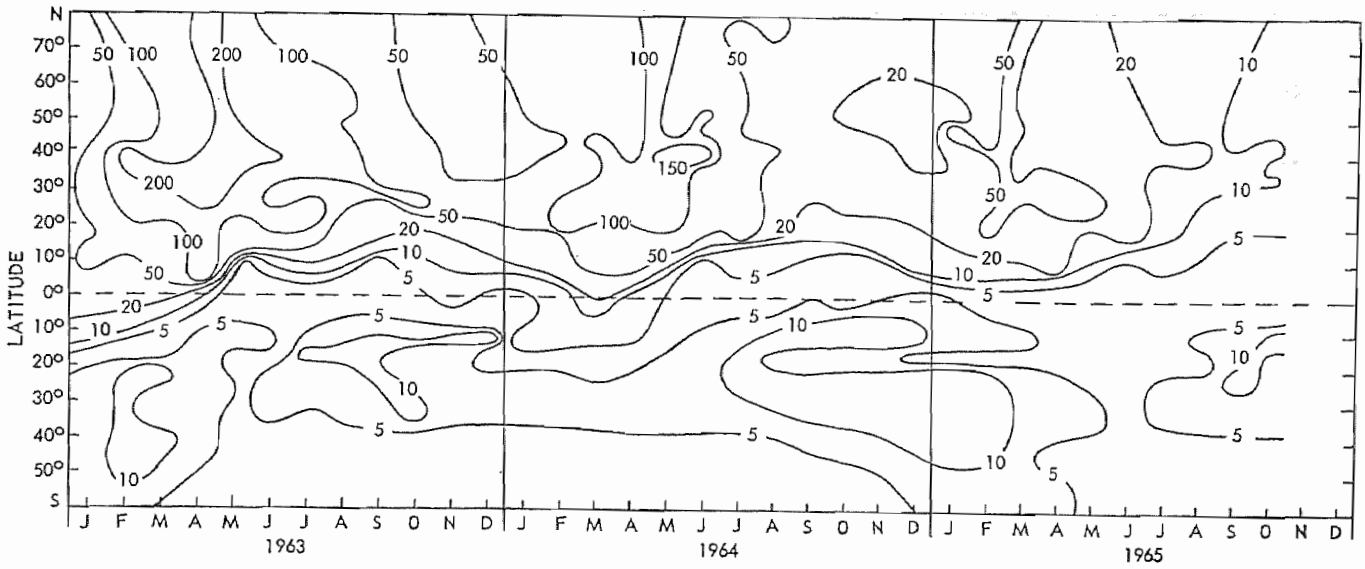


Figure 15. Sr^{90} deposition integrated according to zonal belts as a function of time, 1963-1965[65] (mCi/km² per month)

due to greater local fall-out.[325, 326, 338-341] For inventory purposes, however, only data from the north Atlantic Ocean have been considered.

43. From the data used in these graphs, Popov and Patin tabulated the average concentrations of Sr^{90} for 1960 and 1961 shown in table III. Very few measurements have been reported after 1961 except from the north Atlantic Ocean.[112, 113, 115, 118] These (table IV) indicate an increasing trend of Sr^{90} concentrations in surface water until the end of 1964. The data from the first half of 1965 suggest that the level of Sr^{90} in this region has started to decline.

44. For the western Pacific Ocean in the vicinity of Japan, surface water in the years 1963 and 1964 averaged about 0.43 pCi/litre and ranged from 0.25 to 0.53,[119] while, for the eastern region of the Pacific Ocean, concentrations from about 0.04 pCi/litre[112] in 1963 to 0.5 pCi/litre in 1965[111] were reported.

45. The now well documented latitudinal variation of deposition of Sr^{90} on land (figure 15) is not observable in surface ocean water. However, in table III and figures 19 to 21 the generally higher values for the northern hemisphere presumably reflect the fact that most nuclear tests have so far been carried out in the northern hemisphere.[120-128]

Concentrations in deep water

46. Measurements of fission product concentrations are consistent with more rapid contamination of deeper water with strontium-90[111, 113, 117, 119, 121-123, 129, 130] than some investigators believed possible.[112, 118, 131] Bowen *et al.*[113, 115, 120] found mid-depth (1,000 to 3,000 metres) concentrations of Sr^{90} in the Atlantic Ocean consistent with the flow rates of intermediate depth currents derived from hydrographic considerations. Belyaev *et al.*[130] used observed turbulent diffusion coefficients and mean current velocities to solve the complete transport equation. The calculated distribution of Sr^{90} with depth in the Atlantic Ocean was found to be in agreement with observed data.[325, 327, 338-341]

47. Oznidov and Popov[122] pointed out the slower increase of Sr^{90} concentration with time in the north Atlantic Ocean surface water compared to that on adjacent land and concluded that these data indicated intense vertical turnover of ocean water above and below the thermocline.

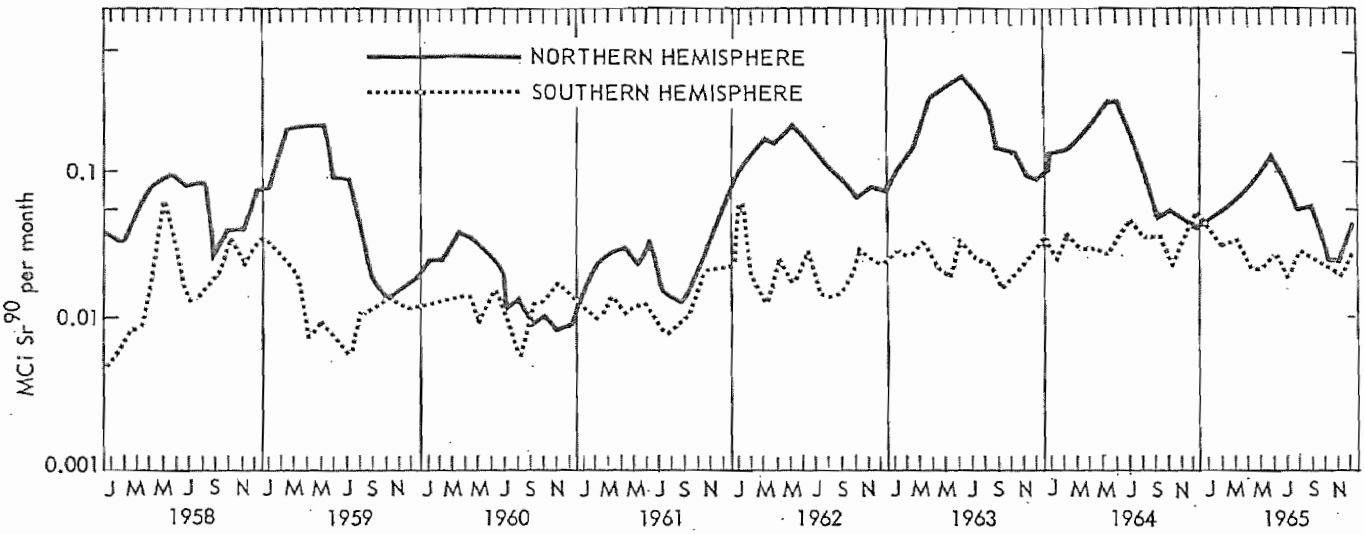


Figure 16. Hemispheric deposition rates of Sr^{90}, 1958-1965[65, 86]

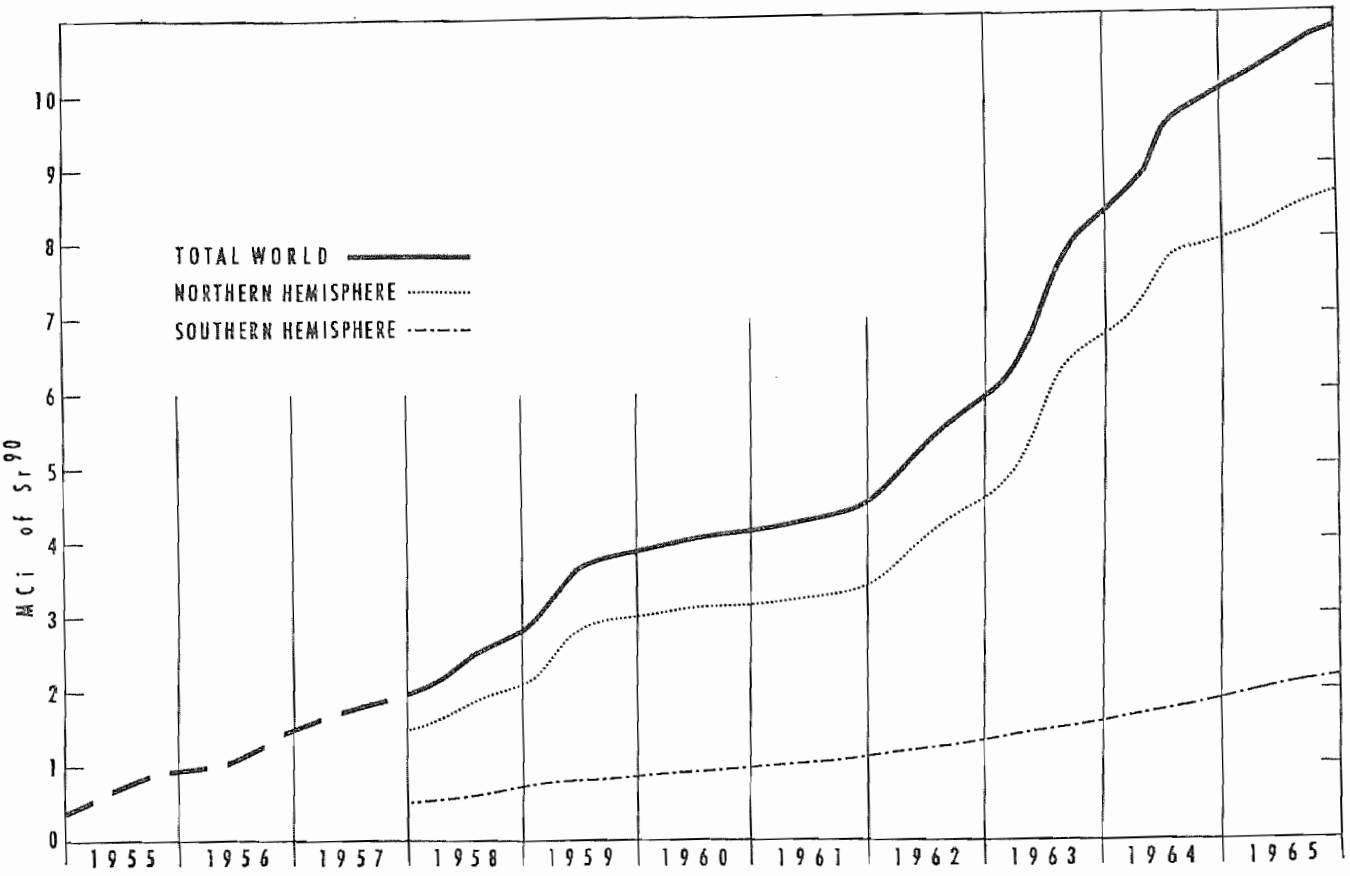


Figure 17. Cumulative Sr^{90} deposit, 1955-1965[85, 86] (corrected for radio-active decay)

48. In 1962, north Atlantic water samples at depths from 1,000 to 3,000 metres or more had Sr^{90} concentrations averaging about 0.03 pCi/litre with values ranging from 0.01 to 0.07.[113] Equatorial Atlantic samples from similar depths averaged about 0.04 pCi/litre in 1963.[113] Figure 23 illustrates average profiles of Cs^{137} and Sr^{90} for the equatorial Atlantic Ocean in 1963 and 1964.

49. Broecker[112] reported much lower values for mid-1963, generally less than 0.01 pCi/litre, for deep water from both the Atlantic and Pacific Oceans. This lack of agreement in deep water samples probably relates to the problem of corrections for blank samples.

50. The dating of deep water samples by natural pre-test radio-carbon shows a very slow rate of exchange between deep and surface water which is incompatible with the appearance of Sr^{90} in the deep ocean. Östlund,[139] moreover, found water with little or no tritium in certain areas of the deep Atlantic Ocean where Bowen detected appreciable amounts of Sr^{90}.

51. Several investigators[111, 119, 120, 132] have also reported measurements of other nuclides from atomic explosions, such as Cs^{137}, Zr^{95}, Nb^{95}, Ce^{144}, Ru^{103}, Ru^{106} and Pm^{147}, at considerable depths in the oceans. It must be recognized, however, that the mechanism of transport of some of these nuclides may be different from that of Sr^{90}.

Comparison of terrestrial and oceanic fall-out

52. Several investigators[111, 115, 120-124, 129, 133-136] found that deposition of Sr^{90} in the oceans was higher than on the adjacent land bodies, generally by a factor of between 1.5 and 5 per unit area. Karol *et al.*,[124] studying 1960-1961 data in 10° latitude bands, found ocean/land deposition ratios ranging from 1 to 8, with the average for the entire 70°N to 30°S region being about 2.

53. Integration of observed Sr^{90} values in the mixed layer down to the thermocline yields a value for accumulated Sr^{90} deposited which is approximately the same as that expected from observations on land. Accordingly, in so far as the presence of Sr^{90} in water below the thermocline can be demonstrated, the fall-out over the oceans must be greater than that measured over land.

54. Enhanced Sr^{90} deposition over the ocean compared with land areas would help to explain the disparity between the change in atmospheric content, and that in land fall-out. The annual reduction in the observed atmospheric content of Sr^{90} is found to be 1.5 times the total world-wide deposition estimated from land-based stations since 1963.[63, 64, 111, 124, 137]

55. Calculation shows that this corresponds to a deposition factor over the oceans, per unit area, of about 1.8 times that which has been estimated over land. It must be remembered that our knowledge of deposition over the entire land surface is far from complete, little information being available, for example, regarding heavily forested areas. Heavier deposition in such regions could partially explain the apparent discrepancy.

56. Chesselet *et al.*[132, 138] measured concentrations of $Zr^{95} + Nb^{95}$, Ru^{103} and Ru^{106} in waters of the western Mediterranean and the Bay of Biscay in late 1963 by *in situ* gamma spectrometry. Comparing the results of these measurements with observations made simultaneously at continental sea shore stations, they concluded that the deposition had been two to seven times greater over the oceans.

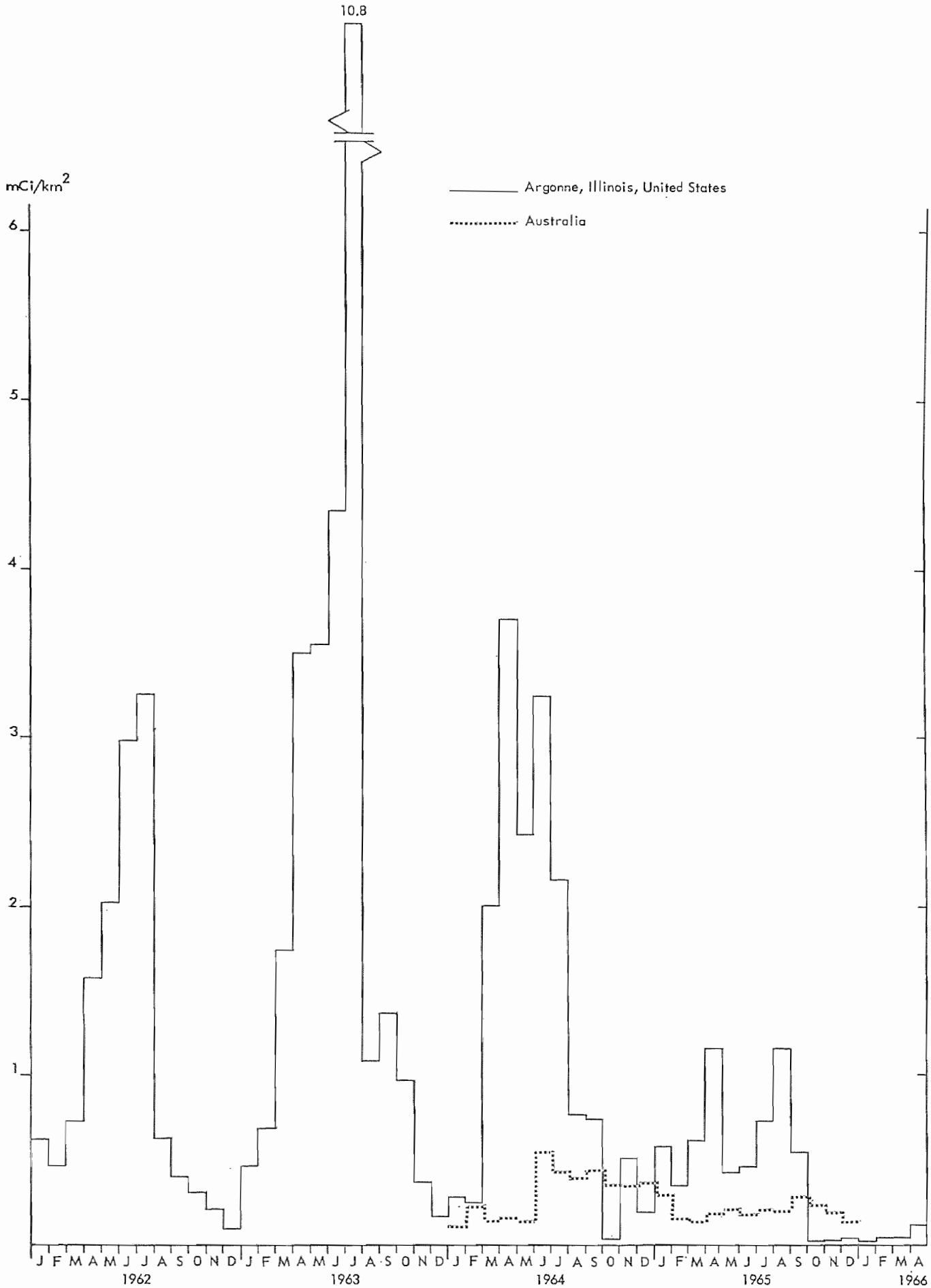


Figure 18. Monthly deposition of Cs^{137} at Argonne, Illinois, United States, for 1962-1966,[28] and the average monthly deposition of Cs^{137} in Australia for 1964-1965[104]

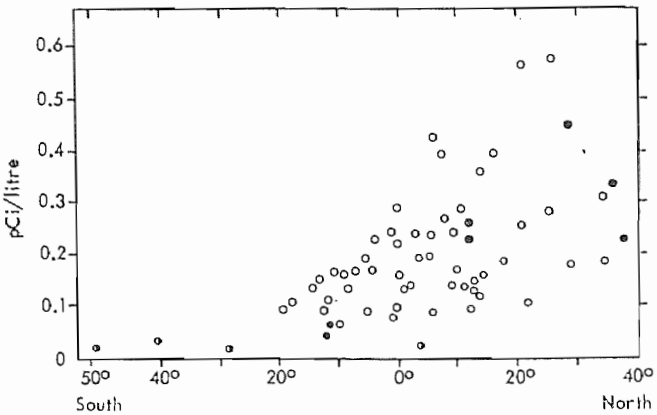


Figure 19. Sr^{90} concentration in Pacific Ocean surface water in 1960 and 1961[125, a]

[a] Different symbols indicate results of different investigators.[118, 326, 330]

showed that, in almost all latitudes tested, the concentration of artificial radioactivity is lower above the sea than above the land.

Conclusions

60. The presence of Sr^{90} in the deep waters of the oceans and seas appears to be confirmed by several investigators, although the low levels of the radioactivity may in some cases preclude the accuracy in the radio-chemical analyses that would be necessary for unambiguous interpretation. Other information, such as C^{14} and H^3 measurements, is in conflict with this.

61. On balance, it appears that more Sr^{90} has been deposited over oceans than over land per unit area. The precise amount of the excess has not yet been determined but the average ratio of ocean deposition per unit area to that measured on land may be between 1.5 and 3. The mechanisms which bring about this

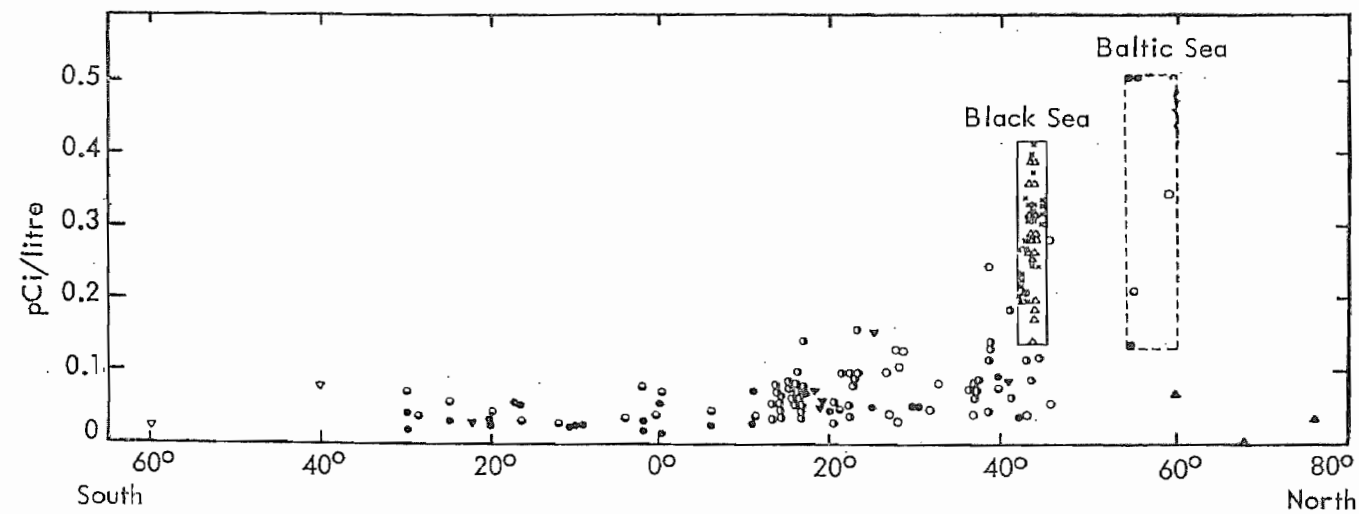


Figure 20. Sr^{90} concentration in Atlantic Ocean surface water in 1960 and 1961[125, a]

[a] Different symbols indicate results of different investigators.[318, 385, 326-324]

57. Broecker *et al.*[134] utilized the Sr^{90} concentration of shallow water on the Bahama Banks which was also dated by its content of artificial C^{14}. These parameters permitted a comparison of fall-out in that region with land sites. The data suggested that fall-out on the Bahama Banks was not systematically higher than that observed on land masses in the same latitude belt. The sensitivity of this experiment, however, could not distinguish differences of less than about a factor of two.

58. Machta *et al.*[140] studied the Sr^{90} distribution in Lake Michigan and found that the deposition over this large lake was not higher than that extrapolated from nearby land stations. The relevance of this observation will depend upon many factors such as absorption by sediments and general similarity to ocean conditions.

59. Karol *et al.*[124] have shown that neither higher concentration of Sr^{90} in oceanic rain[342, 343] nor higher precipitation are likely mechanisms of enhanced deposition over the ocean, since no increased deposition was observed on island stations, confirming the conclusion reported in the 1964 report. They suggest that the major mechanism is related to the air-sea interface, and consists of "scrubbing" of the surface air by sea-water spray and salt particles, and of irreversible absorption of the aerosol by the water surface. These conclusions were tested by comparing Sr^{90} and other radio-nuclides in surface air taken over land and sea areas. The result

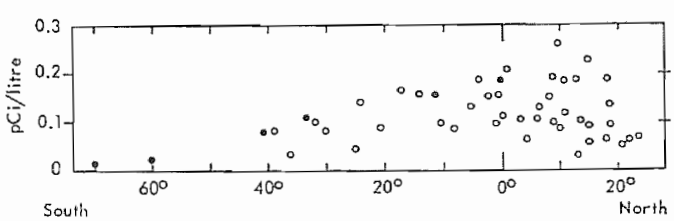


Figure 21. Sr^{90} concentration in Indian Ocean surface water in 1960 and 1961[125, a]

[a] Different symbols indicate results of different investigators.[329, 336]

enhanced oceanic deposition are not clear, but evidence suggests that it is related to phenomena at sea-air interface rather than to precipitation.

Summary

62. Table V shows the changes in the global Sr^{90} inventory since 1960. The estimate of cumulative deposition is based on the assumption that deposition per unit area is the same over oceans as that measured on land.

63. The observed stratospheric mean residence time of fourteen months for particulate radio-activity in the years 1963-1965 is shorter than the assumed value of twenty-four months used in the 1964 report. No single

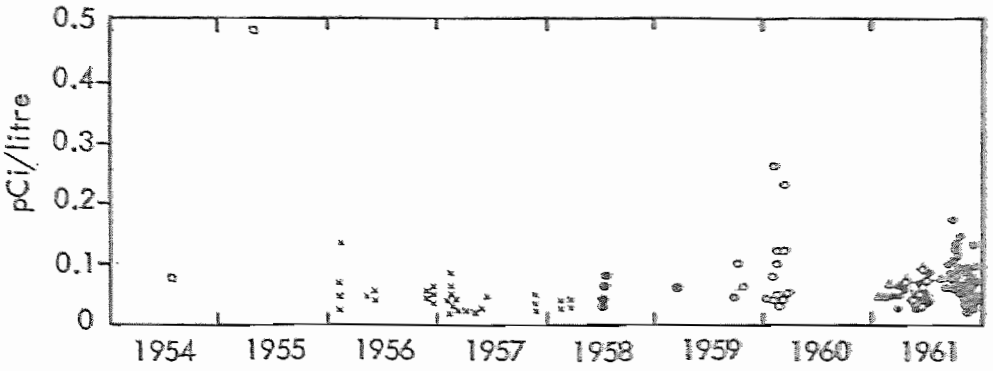


Figure 22. Sr^{90} concentration in northern hemisphere Atlantic Ocean surface water, 1954-1961[125, a]

[a] Different symbols indicate results of different investigators.[123, 125, 127-128, 135-137]

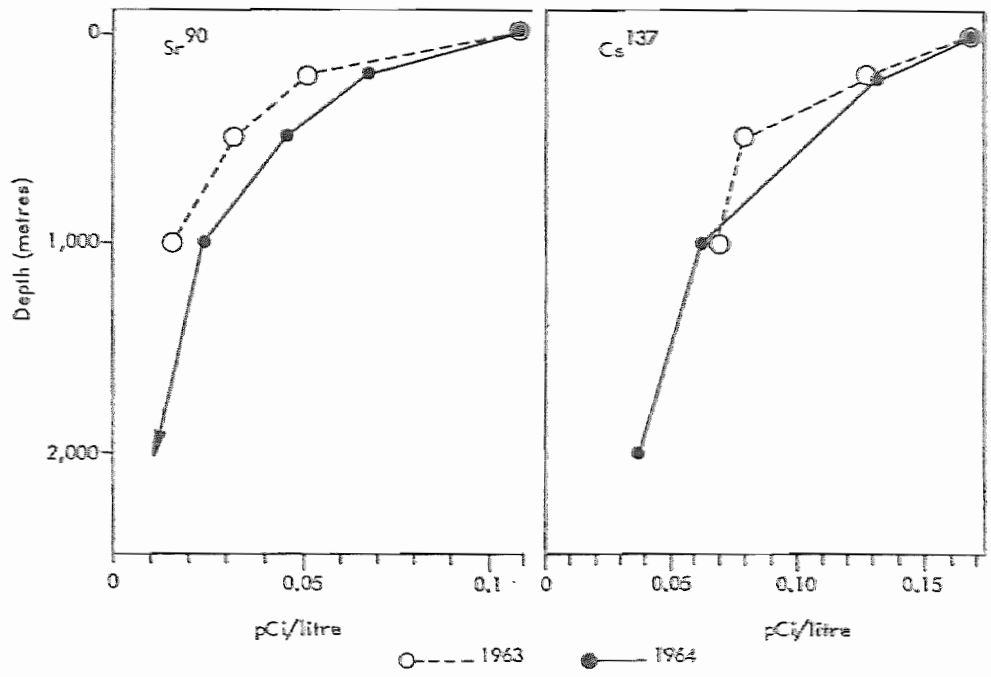


Figure 23. Vertical profiles of concentration of Sr^{90} and Cs^{137} in the equatorial Atlantic Ocean, 1963 and 1964[121]

value for the mean residence time can be applied over long periods to describe the depletion of the stratospheric reservoir. The estimates of the residence time must be adjusted to conform with the temporal changes of the distribution of the nuclear debris in the stratosphere.

64. In the absence of further large scale injections of fresh radio-activity into the atmosphere, the radio-active decay of already deposited Sr^{90} and Cs^{137} will more than offset any subsequent deposition rates starting in the current year, so that the amount present over the whole surface of the globe will begin to decrease. However, some further increases in deposited activity are expected in the southern hemisphere.

65. Strontium-90 observations in deep oceans, though in conflict with C^{14} and H^3 findings, suggest that between 1.5 and three times more Sr^{90} has been deposited per unit area over oceans than measured over land. This enhanced oceanic fall-out helps explain the disparity between changes in atmospheric content and global fall-out derived from land stations.

66. *Carbon-14 and tritium.* Table VI shows the changes in the global inventory of excess C^{14} since July 1963.[62, 64] Values in the northern stratosphere declined gradually, while little change occurred in the southern stratosphere or in the troposphere where peak values of C^{14} concentrations were reached by the end of 1964. Tropospheric C^{14} will decline steadily, since oceanic uptake rates already exceed transfer rates from the stratosphere. No measurements of tritium concentrations in the atmosphere have been reported recently.

II. Artificial radio-activity in food and tissues

Strontium-90 and strontium-89

Food chain mechanisms

67. The principal factors which control the entry of Sr^{90} into human diet were reviewed in the earlier reports of the Committee. More recent information supports the general conclusions advanced therein. However, as is shown in a subsequent section, because of the larger body of survey data which is now available, quantitative relationships between the deposition of Sr^{90}

and dietary levels can now be predicted somewhat more confidently.

Metabolism in man

68. Attempts have been made to explain in kinetic terms the discrimination between strontium and calcium in metabolic processes. The experiments of Walser and Robinson[141] and of Marcus and Wasserman[142] show that the degree of discrimination against strontium during its tubular re-absorption from the glomerular ultra-filtrate is inversely related to the efficiency of calcium re-absorption. A similar relationship holds with regard to absorption of both elements from the gastro-intestinal tract. Quantitative analysis of the data suggests that the ion transfer across the relevant biological membrane is in both cases a first order process and that a constant ratio obtains between the rates of absorption of calcium and strontium.

69. It follows from these experiments and from theoretical considerations that the body/diet OR[a] must vary slightly with the efficiency of calcium absorption in the gastro-intestinal tract and be higher when a greater fraction of calcium is absorbed. In early infancy, however, the limited discrimination between calcium and strontium is due not only to high absorption efficiency but also to the lack of difference in the absorption rates of both elements in the gut.

70. Average values, however, are little affected by individual variations and an average OR of 0.25 will still be used in this report, since:

(*a*) Recent studies,[143, 144] carried out in apparent metabolic steady-state (using stable Sr/Ca ratios in bone and diet) have yielded OR values very close to 0.25;

(*b*) Higher OR values in the first year of life[145] are offset by additional discrimination at the placental barrier during foetal life and by a very rapid turnover of bone mineral in early infancy (1964 report, annex A, paragraphs 94-97).

71. Investigations of the metabolism of strontium in children and adolescents (one to nineteen years old) are in progress. Preliminary results indicate that no changes in the over-all estimate of the bone/diet OR used to calculate dose commitments from Sr^{90} are warranted.

72. Analysis of recent data from various countries on the empirical relationship

$$\frac{Sr^{90}/Ca \text{ infant bone (0-1 year)}}{Sr^{90}/Ca \text{ milk}}$$

shows that the values reported cluster around one-quarter (tables VIII and X).

73. Marei *et al.*[146] on the basis of extensive investigations established a relationship between the Sr^{90}/Ca ratios in human teeth and in the skeleton in all age groups. The

$$\frac{Sr^{90}/Ca \text{ teeth}}{Sr^{90}/Ca \text{ skeleton}}$$

ratio was higher in infancy and childhood than in adults (table VII), and remained relatively constant over a period of two to three years. The authors concluded that teeth may be used for monitoring Sr^{90} levels in man, if the ratios between dental and skeletal values are corrected for age and changes in time.

[a] OR = observed ratio = $\frac{\text{Sr/Ca sample}}{\text{Sr/Ca precursor}}$

74. The distribution of Sr^{90} and calcium in skeletons of adults in 1963 was re-examined in a recent study.[147] The

$$\frac{Sr^{90}/Ca \text{ single bone}}{Sr^{90}/Ca \text{ total skeleton}}$$

ratios were as follows: vertebrae/skeleton = 1.7; rib/skeleton = 1.0; femur shaft/skeleton = 0.4. These results are in essential agreement with data reported earlier,[148-150] showing that changes in these ratios over the last three to four years did not exceed some 20 per cent.

Levels of strontium-90 in foods

75. The levels of Sr^{90} in milk in the period 1963-1965 are presented in table VIII. In the northern hemisphere the Sr^{90}/Ca ratios in milk in 1964 remained roughly at the level of 1963, when yearly mean values are compared. In 1965 a significant decline was observed which amounted, on the average, to about 20-30 per cent of the 1964 values (based on results of widespread, systematic studies). Time trends in milk concentrations of Sr^{90} in a few countries are shown in figure 24.

76. In the temperate zone of the southern hemisphere (Argentina, Australia, New Zealand), the absolute levels of Sr^{90} in milk were lower by a factor of three to four than those in the northern temperate zone. They were similar, however, to the levels reported from equatorial regions, reflecting roughly the latitudinal distribution of past fall-out levels in both hemispheres. The time trends in the two hemispheres were somewhat different: the increase in concentrations in the southern hemisphere continued from 1963 to 1964, but the yearly average levels remained unchanged in 1965.

77. In some areas, such as the Faroe Islands, Iceland, western Norway and the mountainous regions of many countries, the levels of Sr^{90} in milk were significantly higher than the average values typical for most of the northern temperate zone. As already indicated in previous reports, these elevated concentrations of Sr^{90} in milk are mainly due to high rainfall and poor pastures.

78. The levels of Sr^{89} in milk declined in 1964 below detection limits. In the summer of 1965, transient levels of this nuclide in milk were reported[151, 152] from some countries of the northern hemisphere following the atmospheric nuclear test conducted in May of that year. As the levels declined again below detection limits within three to four months, Sr^{89} will not be further considered in this report.

79. Information on the average Sr^{90}/Ca ratios of total diets is more limited than for milk, but seems to disclose similar time trends and geographical distribution. The available information is presented in table IX.

80. New information on Sr^{90} levels in total diet and in milk shows that the

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

ratio has remained at about 1.5 in the United States, western Europe, Argentina and Australia (tables VIII and IX). Information now available from the USSR indicates that this ratio ranged from 2 to 3 in 1963 and 1964; this result is similar to that found in Poland from 1961 to 1963. These higher ratios found in the USSR and in Poland are due to greater consumption of whole-meal cereal products.

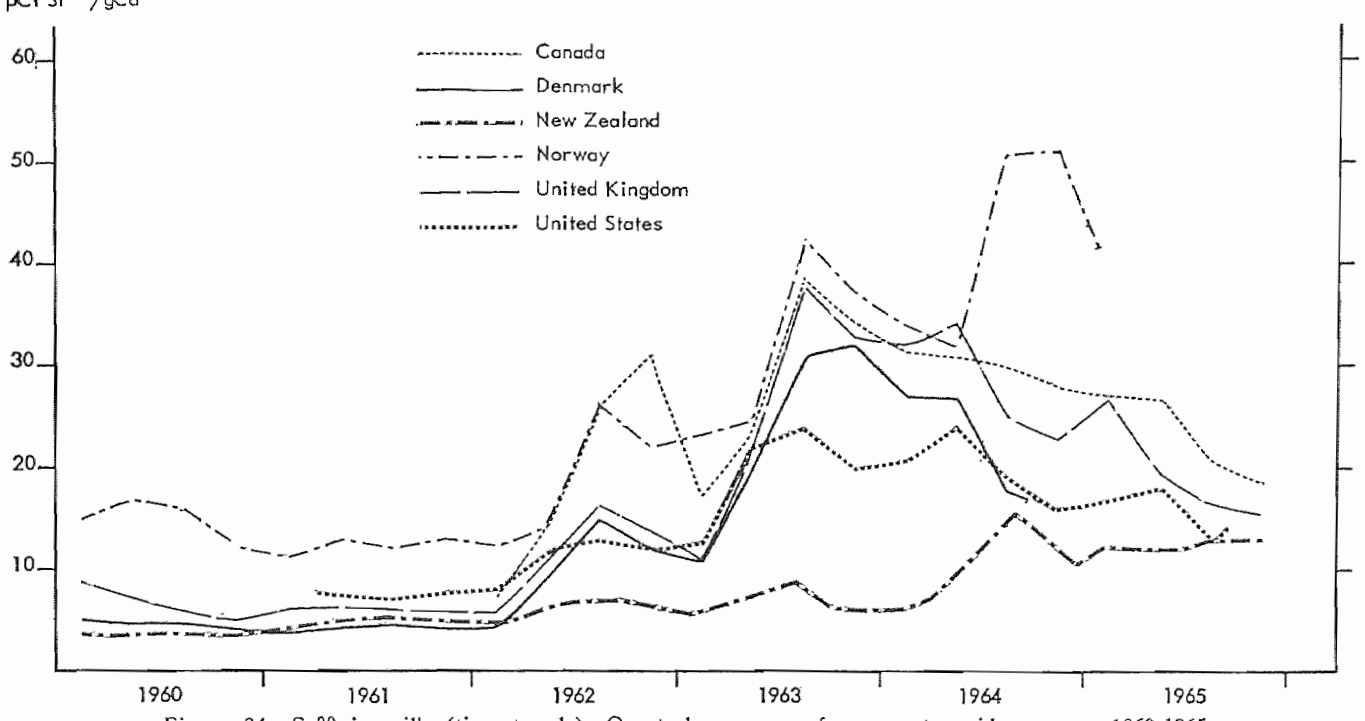


Figure 24. Sr^{90} in milk (time trends). Quarterly averages from country-wide surveys, 1960-1965

81. The relative constancy of the ratio

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

at least for a few years, supports the conclusion reached in the 1962 report that in many areas the Sr^{90}/Ca ratio in total diet can be reasonably inferred from analyses of milk.

82. Studies in Argentina,[145] Austria,[153] the United States[154] and the United Kingdom[155] have shown that the Sr^{90}/Ca ratio in the diets of infants who were not breast-fed in the first year of life falls between the values determined for milk and those for a typical adult's diet. As the ratio of Sr^{90}/Ca in human milk equals about one-tenth of the adult diet, the effective contamination of infant food depends very much on the extent of breast feeding.

Strontium-90 in human bone

83. Levels of Sr^{90} in human bone in 1963, 1964 and 1965 are presented in table X.

84. The age distribution of the Sr^{90}/Ca ratios in human bone remained essentially unaltered from that previously reviewed. The highest levels are still encountered in the zero to one or in the one-year-old age group (figure 25).

85. The data for adults are reported in table X, both in original values and as normalized skeletal averages, calculated by applying the normalization factors given in paragraph 74. In the years of high fall-out rate, the average skeletal levels in adults were lower by a factor of four to ten than the highest values in young infants. This difference is expected to diminish with decreasing dietary levels of Sr^{90}, because the skeletal contaminations in infants will change faster than in adults, and trends in these two age categories may be in opposite directions.

Geographical distribution and time trends

86. In the northern hemisphere, bone levels were higher by a factor of two to four than those reported from Argentina and Australia. There was a tendency for a marked increase in bone levels in all age groups from 1963 to 1964, the highest relative increase being in youngest infants. In the zero to one-year-old age group, the increase amounted on the average to some 30 per cent over the 1963 values, as compared to the factor of two or three by which the latter rose above the 1962 values. Although only limited results are available for 1965, it may be provisionally concluded that, in the zero to one-year-old age group, levels may have reached their peak in 1964 and started decreasing in 1965. In the one-year-old age group, bone levels in 1965 seemed either the same as or slightly higher than in 1964. As expected, levels in adults are still increasing.

87. On the basis of available data, it seems that in the southern hemisphere, in the period under review, the time trends of Sr^{90} levels in human bone were not markedly different from those in the northern temperate zone.

88. The frequency distribution of the Sr^{90}/Ca ratios in adult bone—where levels are age-independent—has been repeatedly studied and recent data[156, 157] confirmed earlier findings,[158, 159] indicating that the distribution was positively skewed and might be described as log-normal. If data from individual locations are considered, the probability that the Sr^{90}/Ca ratio in a single sample will exceed the mean value by a factor of three is of the order of 1 per cent.

Quantitative relationships between deposition of strontium-90 and dietary levels

89. In earlier reports of the Committee, fall-out rates and accumulated soil deposits of Sr^{90} were related to the concentrations of this nuclide in foodstuffs to

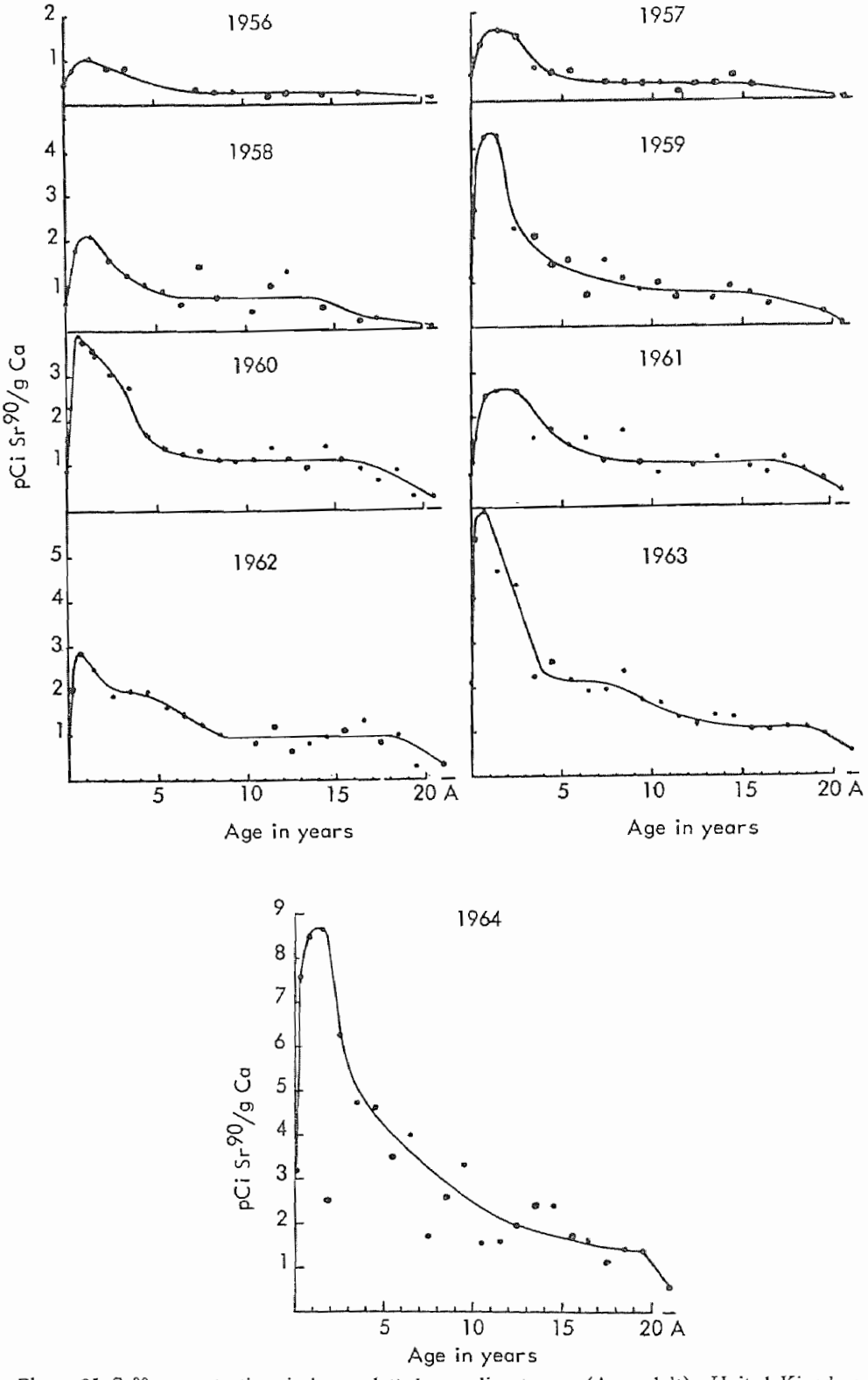


Figure 25. Sr^{90} concentrations in bone, plotted according to age (A = adult): United Kingdom results, 1956-1964[272]

predict future levels of contamination. The basic equation applied with respect to milk was:

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

where

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

F_d = total accumulated deposit of Sr^{90} in soil in mCi/km²,

F_r = yearly fall-out rate of Sr^{90} in mCi/km² in given year,

p_d and p_r = proportionality factors also called "soil" and "rate" factors.

Although data from many parts of the world remain meagre, it is now possible in a number of countries to derive soil and rate factors for milk by appropriate regression analysis.

90. In some extensive regions, factors such as stored fodder for cattle justify the introduction of a third term in the equation which takes into account the contribution to milk levels of Sr^{90} which was deposited when crops and grass were growing in the second half of the previous year. The inclusion of this "lag-rate factor" may lead to a significant improvement in the prediction of year to year trends, especially when the rate of fall-out is changing markedly.[112, 160] However, the separate evaluation of rate and lag-rate factors is not necessary for the calculation of dose-commitment.

91. An analysis of survey results from Argentina, Australia, Canada, Denmark, Germany, Japan, New Zealand, the United Kingdom and the United States indicates that the averages of the rate and soil factors for milk fall within the range of 0.8 to 1.0 and 0.2 to 0.3, respectively. These values do not differ sufficiently from those formerly estimated (0.8 and 0.3) to necessitate a revision of the proportionality factors previously adopted for milk nor for the three major types of total diets consumed by the world's population (1962 report, annex F, paragraphs 108-119).

92. In a number of countries, the annual average levels of Sr^{90} in diet, and especially in milk, have hitherto agreed satisfactorily with those predicted from the proportionality factors. While this encourages confidence in the use of this procedure, it must be borne in mind that assessments of the situation many years hence is subject to considerable uncertainty. This is so largely because absorption by plant roots is usually greater from the upper layers of soil where so far the levels of Sr^{90} have been highest. When downward penetration of Sr^{90} leads to its more uniform distribution throughout the rooting zone, lower values of the soil factors are to be expected. However, there is at present no basis for predicting how the soil factors will change with time in the majority of areas.

93. Because at the present time Sr^{90} is more concentrated in the upper layers of soil in permanent pastures than in the ploughed land on which other crops are grown, the decrease in the soil factor for milk is expected to be larger than for other foodstuffs. This should lead to a gradual increase in the ratio of

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

which will approach the relationship obtaining for stable strontium and calcium.

94. A further problem in predicting absorption from the soil in the distant future arises from uncertainties as to the fraction of the total Sr^{90} in the soil which will be removed annually by crops or lost from the rooting zone by leaching. The Committee previously assumed that those processes might decrease the content of the soil by about 2 per cent per annum, leading to a total decrease of 4.5 per cent per year when radioactive decay is taken into account. It is recognized that this figure will lead to an under-estimate of the losses in many countries, but the data are insufficient to justify its revision.

95. Because it is expected that the soil factor for total diet will decrease with time and that the rate of loss of Sr^{90} from the soil may be greater than 2 per cent per year, estimates of the integrated quantities of Sr^{90} which will enter diet over long periods, based upon the methods adopted here, should be regarded as upper limits.

CAESIUM-137

Food chain mechanisms

96. Cs^{137} which is deposited from the atmosphere is retained on vegetation in a manner similar to Sr^{90}, but the two nuclides contrast markedly in their behaviour in soil. In mineral soils which contain appreciable quantities of clay and moderate or low quantities of organic matter, the entrapment of Cs^{137} in the clay lattice structure causes it to be little absorbed by plants.[161] The process of fixation may not be complete until several years have passed; thereafter, Cs^{137} may be absorbed to not more than about one-fortieth of the extent of Sr^{90}.[162] In contrast, when soils contain much organic matter, and especially when their clay content is low, Cs^{137} enters plants considerably more freely.[163-165]

97. Soils in which organic matter is present in large concentrations throughout the entire rooting depth are mainly important in moist, temperate regions; this circumstance appears to explain, at least partially, why the average levels of Cs^{137} in diet from northern regions, e.g. Scandinavia, exceed those in lower latitudes.[161] A more transient effect of organic matters occurs commonly in the permanent pastures of temperate regions. Appreciable quantities of organic matter form in the superficial soil layers, and, until Cs^{137} has penetrated into the underlying mineral soil, it is readily absorbed by plants; enhanced uptake due to this cause has been shown to continue for one to two years in these circumstances.[161, 163] In the 1964 report, it was noted that Cs^{137} may also readily enter plants when soils are lateritic in type.

98. In its 1964 report, the Committee discussed a special food chain mechanism, whereby in some populations living in subarctic regions unusually high activities of Cs^{137} are transferred to man from lichens through reindeer or caribou meat. Results of a detailed dietary study of Finnish Lapps which became available recently[166] show that about 75 per cent of their total intake of Cs^{137} comes from reindeer meat and meat products, while fish and milk contribute about 11-12 per cent each. It has since become evident that other nuclides in fall-out, such as Na^{22} and Fe^{55}, or of natural origin (Pb^{210} and Po^{210}), also show unusually high levels because of similar mechanisms of transfer.[167-172]

99. Lichens constitute the main fodder of reindeer and caribou during the winter season. In the summer, however, almost only grass and herbaceous plants are consumed. The Cs^{137} body burden of the animals therefore shows a marked seasonal variation, particularly some years after the peak deposition, when Cs^{137} levels in the grass have decreased while the levels in the lichens remain high.

100. Lichens on which these animals graze entrap almost all the Cs^{137} deposited per unit area of ground. It has been shown that the levels in grazing animals have increased up to 1965 in proportion to the lichen levels which are closely related to accumulated deposit of Cs^{137} (table XI). The levels are expected to decrease during the coming years when the fall-out rate will not be sufficient to compensate for the Cs^{137} loss due to grazing and possible wash-out. A number of estimates of the total elimination rate have been published,[173-175] and although there is a wide divergence between these estimates, it seems likely that at least some 5-10 per cent of the Cs^{137} is eliminated annually.

101. Other mechanisms have also been shown to cause elevated levels of Cs^{137} in foodstuffs in northern

latitudes. Unusually high levels (10 or more nCi Cs^{137}/kg) have been reported in fresh water fish from some Scandinavian lakes.[176-178] This has been attributed to the low mineral content of the water which enhances the absorption of Cs^{137} and other fission products. Similarly, aquatic plants which are sometimes important in the diet of cattle in these areas contain higher levels of Cs^{137} than normal pasture species.[179]

Metabolism in man

102. Investigations of the metabolism of caesium in pregnant women and in new-born children[180] have shown that the turnover of this element becomes accelerated in the course of pregnancy, with a resulting biological half-life of the order of about thirty to seventy days as contrasted with the usual value in adults of seventy to 140 days. There seems to be no placental barrier for Cs^{137} in its movement to the foetus, for new-born children revealed practically the same Cs^{137}/weight ratio as their mothers.

103. That the Cs^{137} turnover is generally faster in infants than in adults has been further confirmed, suggesting a biological half-life in the first month of life of only seven to ten days (Wilson and Spiers, quoted by Vennart[181]), or twenty-one to twenty-five days.[180] Both the lower intake and the faster turnover of Cs^{137} in children explain that they show lower levels than adults, as has already been indicated in the 1964 report. This observation was recently confirmed by radiochemical analysis of skeletal muscles obtained from human beings of all age groups.[182] The levels of Cs^{137} per gramme of potassium were lowest in still-born and rose with age, reaching the highest values in adults.

104. Newly reported data on the biological half-life, characterizing the long-term retention of caesium in adults,[183-188] fall in line with the information already reviewed in the 1964 report (table XII). The remarkably shorter average half-life in the inhabitants of some Scandinavian countries has been tentatively explained by elevated potassium intakes[189] which may lead to faster turnover of both caesium and potassium in the body.

105. The distribution of Cs^{137} in human tissues was studied by Nay *et al.*[190] who showed that concentrations in compact bone and bone marrow were lower by a factor of twenty than in skeletal muscle. This implies a dose rate from internal Cs^{137} to bone marrow, to osteocytes and to cells lining the marrow cavities, two to three times lower than to muscular tissue. Although the data are preliminary, refer only to compact bone and may reflect a non-steady-state situation, they suggest that the dose commitment to bone marrow and bone-lining cells due to internal Cs^{137} radiation may have been somewhat over-estimated in the 1964 report. However, the evidence is not convincing enough to warrant a re-evaluation of the dose-rate factors in estimating dose commitments. The changes would in any case have a trivial effect on the total dose commitment because of the relatively low contribution of internal Cs^{137} irradiation.

106. The frequency distribution of the Cs^{137} concentration in human tissues (muscles) was studied by Ellett and Brownell[191] and appeared to be non-normal with positive skewness. By fitting a particular form of gamma function to their data, the authors estimated that the probability that an individual might exceed the population average in a given locality by a factor of three was 1.5×10^{-3}.

107. Static and dynamic distribution studies were conducted by Yamagata *et al.*[192] on caesium in human blood. When the dietary Cs^{137} levels changed slowly with time, the concentrations in blood reflected the total body contents of Cs^{137}. Whole blood samples collected from urban areas in the Far East and the Pacific region gave estimates of body burden ranging from 56 pCi/g K in Rangoon, Burma, to 110 pCi/g K in Manila, the Philippines, in April 1966.[193]

Levels of caesium-137 in food

108. Concentrations of Cs^{137} in milk in the northern hemisphere (table XIII) in 1964 were essentially the same or only slightly higher than in 1963. In 1965, average values were—with only a few exceptions—markedly lower. Time trends are presented in figure 26. In the southern hemisphere, the 1964 yearly average levels rose over those observed in 1963 and remained unchanged in 1965. However, quarterly values in New Zealand and Australia (figure 26) showed a clear decrease in concentrations at the end of 1965.

109. Time trends in the total dietary content of Cs^{137} were basically similar to those for milk, but the data are more limited, especially with regard to the 1965 levels (table XIV). There is, however, a well-established correlation between the Cs^{137} contents in beef and milk, the ratio of the concentrations

$$\frac{\text{pCi/kg meat}}{\text{pCi/l milk}}$$

averaging ~ 4[194-196] (figure 27). Since meat and milk together are the main dietary sources of Cs^{137}, it is evident that trends in dietary contamination can be adequately inferred from the monitoring of milk alone.

110. Cs^{137} levels of up to about 100 nCi/kg were measured in reindeer meat during the winter of 1964 and 1965. According to Lidén,[174] the proportionality factor of Cs^{137} in fresh lichen to that in fresh reindeer meat in northern Sweden averaged 4.9 ± 0.4.

Levels of caesium-137 in man

111. Levels of Cs^{137} in man continued to increase in the northern hemisphere until mid- or late 1964, and declined thereafter. In early 1966 the levels were reduced by about 30-40 per cent as compared to the peak values in 1964. As shown in table XV and figure 28, the ratio between the lowest (Japan) and the highest (France) values reported from the northern temperate zone was between 1 : 2 and 1 : 3 (with the exception of Norway where unusually high levels of rainfall and particular grazing conditions prevail).

112. In the southern hemisphere (southern Australia), the levels rose until late 1965 and appear to have been declining since. The highest levels reached in Australia were lower by about a factor of three when compared with the average 1964 peak levels in normal areas of the northern temperate zone (figure 28).

113. As already discussed in the 1964 report (owing to the factors mentioned in paragraph 111), the levels in Norway, in particular on the western coast, were much higher than those usually encountered in these latitudes.

114. The levels of Cs^{137} in man also rose until 1965 in the majority of those areas where this nuclide follows the lichen-reindeer-man food chain, e.g. Finnish Lapland, Swedish Lapland and the far north of the USSR. Some decrease was, however, reported in Alaska. For reasons explained in paragraph 100 it was ex-

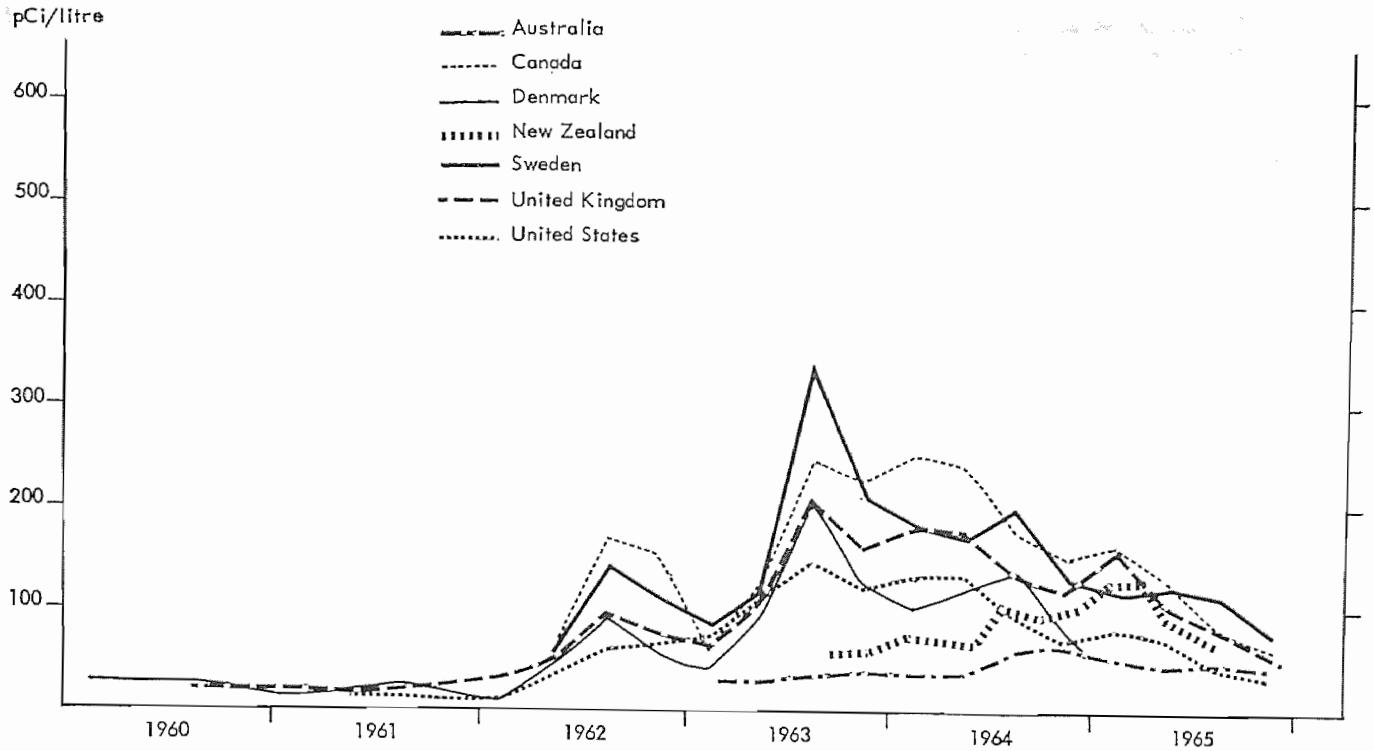


Figure 26. Cs^{137} in milk (time trends). Quarterly average values from country-wide surveys, 1960-1965

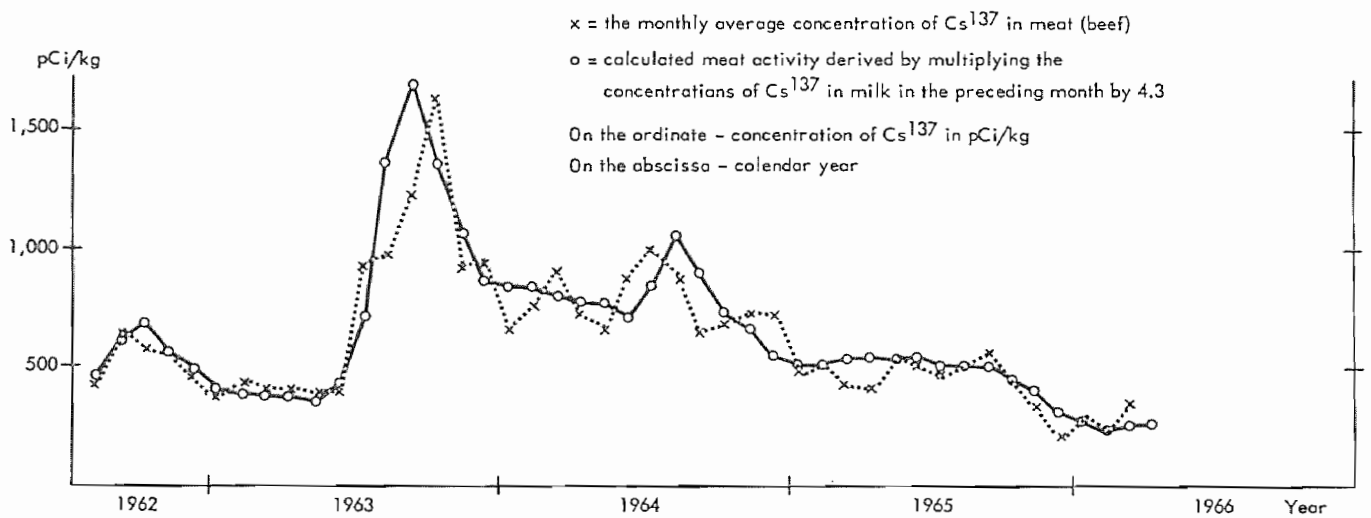


Figure 27. Relationship between concentration of Cs^{137} in beef and milk in Sweden, 1962-1966[344]

pected[173, 174] that levels in man in those areas in 1966 would be the same as, or slightly lower than, in 1965. This is supported by the data for Finnish Lapps (table XV), which show a slight decrease in Cs^{137} body levels (by 5-10 per cent) from April 1965 to March and April 1966.

Quantitative relationships between deposition of caesium-137 and contamination of milk

115. The relationships between fall-out rates and the cumulative total of Cs^{137} in soil, on the one hand, and the contamination of milk on the other, have been further studied in the United Kingdom[160] and in Sweden.[194] In the United Kingdom, it has been shown that levels of Cs^{137} in milk hitherto have depended predominantly on the current rate of deposition and on that observed in the last half of the previous year. Estimates based on tracer experiments indicate that the cumulative deposit in soil has not so far contributed more than about 1 per cent of the total Cs^{137} in milk. Thus, under conditions which have prevailed hitherto, absorption from the cumulative deposit can be ignored. In Sweden, it appears that the transfer of Cs^{137} to milk from the deposit in the current and previous years is about twice[194] that estimated in the United Kingdom.[160]

Quantitative relationships between deposition of caesium-137 and human body burden

116. In the 1964 report, an empirical relationship between rates of deposition of fall-out and Cs^{137} in man was used in calculating dose commitments. The Cs^{137}/K ratio (Q) in human beings was related to the current yearly fall-out and to the deposition over the preceding two years by a 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/km^2,

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

P_r and P_{2c} = proportionality factors.

117. The proportionality factors have been revised, using the survey results now available from Belgium, Germany, Sweden, the United Kingdom and the United States. The resulting values $P_r = 2.5$ and $P_{2c} = 4.0$ are larger than those used in the 1964 report; this is due in part to inclusion of data from Sweden,[194] where the factors are approximately twice as high as for the other countries. Since present evidence suggests that factors similar to those in Scandinavia may affect only a small fraction of the world population the above value of the factors may well lead to an over-estimate of the world situation. No allowance has been made for the expected small contribution of the cumulative deposit of Cs^{137} in the soil, as its effect should be very slight in comparison with other sources of uncertainty.

IODINE-131

118. I^{131} was detected in rain and in the air in Japan[197, 198] following the atmospheric tests carried out in central Asia in 1964 and 1965. Average levels of the I^{131} detected in milk in Japan[199, 200] between 18 October and 12 November 1964 were of the order of 100 pCi/litre and nearly the same during one week of May 1965. Only traces of I^{131} were found in milk, at the end of 1964 in Canada[151] and in the United Arab Republic,[201] and at the end of May and in early June 1965 in the United States[152] and in Israel.[26, 242] I^{131} was also observed in human thyroids (0.02-1.3 pCi/g fresh tissue)[202] and in urine[203] in October and November 1964 in Japan.

OTHER NUCLIDES

119. Fe^{55} has been detected in foodstuffs, in cattle and in human beings.[167, 172] Fe^{55} is an emitter of low energy x rays due to electron capture, and at present levels of contamination the radiation dose from this nuclide to man is insignificant.

120. Na^{22} has been measured in rain, in grass and in foodstuffs.[168, 204, 205] This nuclide was also detected by means of whole body counting in Alaskan Eskimos.[168] The sources of Na^{22} and Cs^{137} in human diet are similar. So far, the activities of the first nuclide do not exceed a few per cent of the latter.

121. Mn^{54} was detected in human food[206, 207] but no observation was reported on its presence in human beings, except for possible traces detected in liver at *post mortem*.[208] The radiation dose from both Na^{22} and Mn^{54} may be neglected at present.

122. Three recent studies[209-211] have confirmed that, as expected, the C^{14} specific activity of plant foodstuffs and milk follows with a slight delay that of tropospheric air. With the exception of adult cartilage, in which the C^{14} specific activity remained essentially unaltered since the pre-test period, the specific activity of human tissues follows the specific activity of air with a delay of the order of one to two years. Until 1964, individuals from the southern hemisphere showed little increase in C^{14} content as compared with the pre-test levels (before 1952), consistent with the small rise of C^{14} specific activity in the air in that hemisphere. Results of C^{14} measurements in biological materials have been presented in table XVI and figure 29.

RADIO-NUCLIDES IN THE RESPIRATORY TRACT

123. Data subsequent to those reviewed in the 1964 report on the contamination of the human respiratory tract by fission products from nuclear tests have been published. Thus, Rundo and Newton[212] reported levels of $Zr^{95} + Nb^{95}$ in the range 79-161 pCi in 1962 and 1963 in England, while Wrenn *et al.*[213] reported values of 210-460 pCi in the respiratory system of people deceased in the United States in the first half of 1963. Assuming a homogeneous distribution of the nuclide, these levels would lead to an annual dose rate of a few millirads at a constant level of contamination. However, the levels of $Zr^{95} + Nb^{95}$ in the air decreased in the period 1963-1965 by two orders of magnitude.

124. A study of the contamination of the human respiratory tract by Ce^{144} revealed[214] average yearly lung burdens of 105, 160, 268 and 182 pCi of this nuclide in 1961, 1962, 1963 and 1964, respectively. The authors calculated the average annual dose rate to the whole lung tissue from Ce^{144} and all other fission products over the period of investigation to be 24 mrad/year, with 10 per cent of the population exceeding the

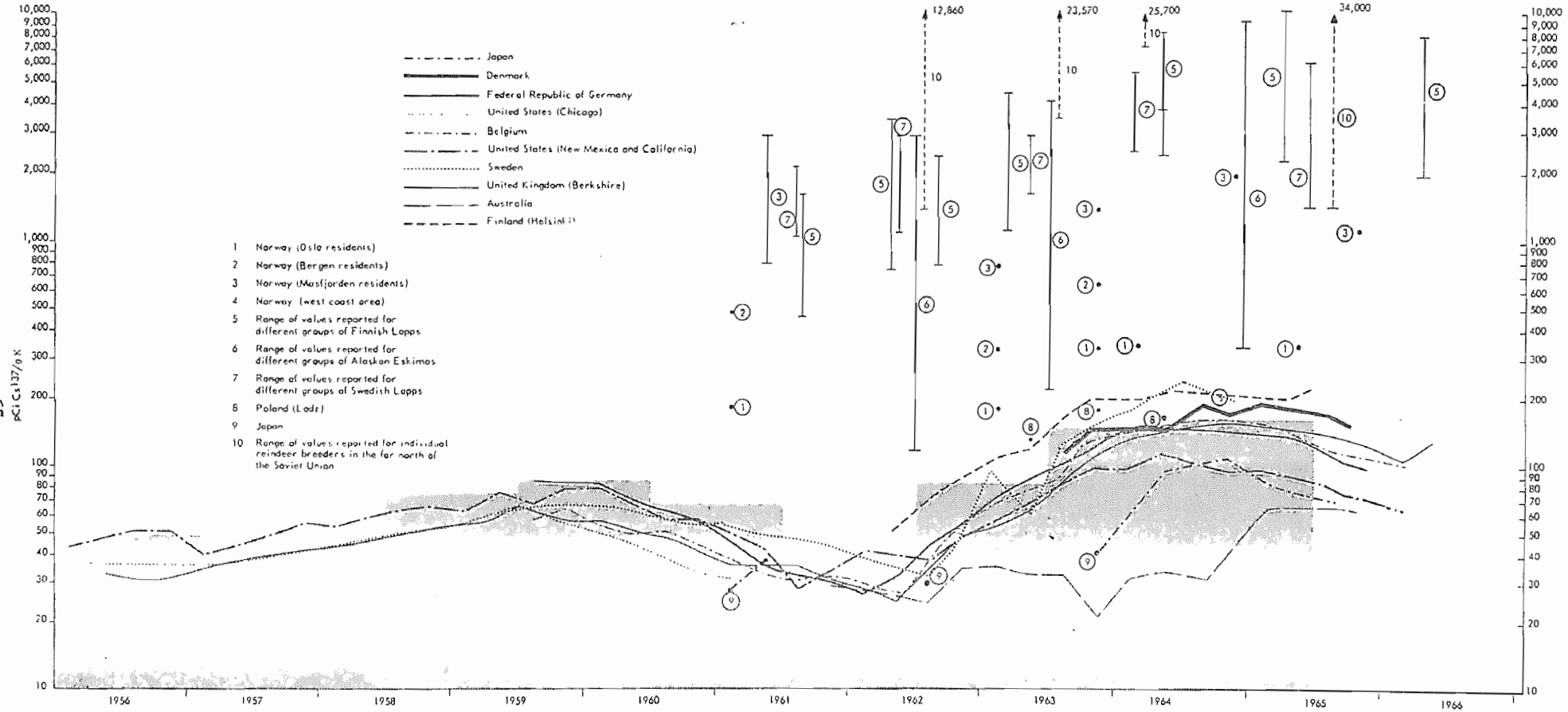


Figure 28. Cs^{137} levels in man during the period 1956 to early 1966 (shaded area indicates the yearly average values calculated according to the formula given in paragraphs 116-117, using average proportionality factors P_r and P_{90} of 2.5 and 4.0, respectively).

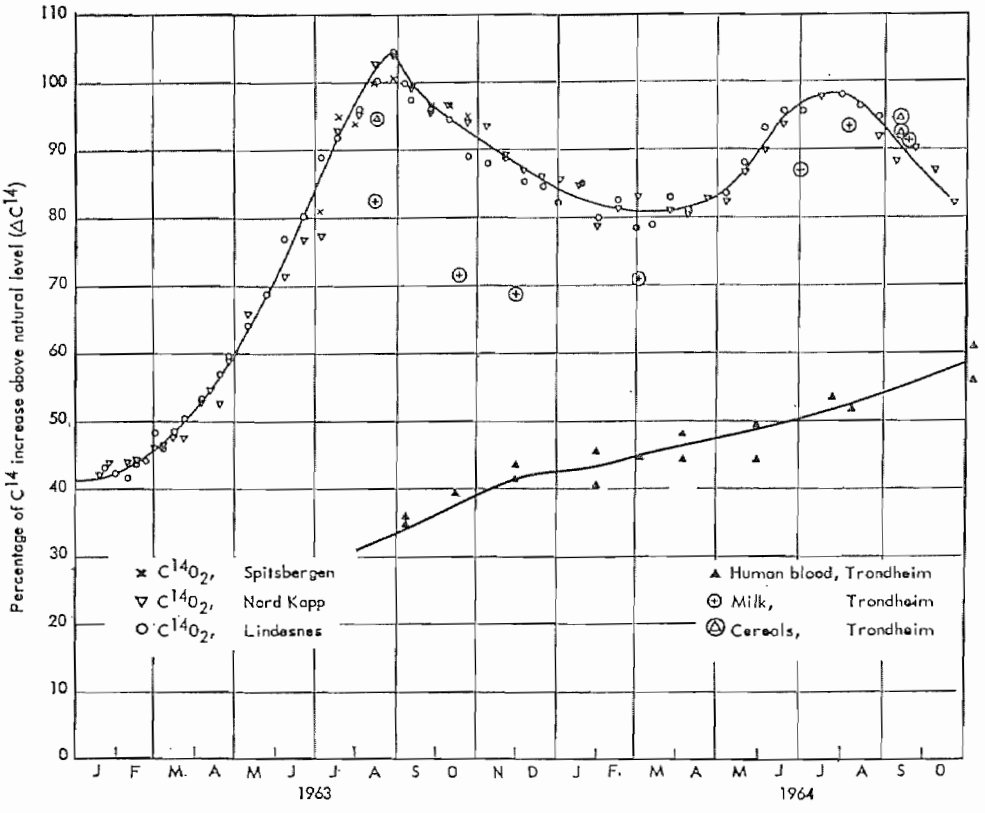


Figure 29. Time trends in C^{14} in Scandinavia, 1963 and 1964[210]

value of 43 mrad/year. In view of the author's assumptions, the dose-rate estimate may be accepted as a possible upper limit.

125. The concentrations and the doses from Ce^{144} in bronchial lymph nodes appear to be higher by one order of magnitude than those in the respiratory system as a whole.[214] The same proportionality has been obtained for the concentrations, and therefore for the doses, from other nuclides that are retained in the respiratory tract and that show similar affinity for the lymphatic tissue (e.g. Pu^{239}).

III. Doses from external and internal contamination

Introduction

126. In this annex, most of the doses are expressed as dose commitments. For the purpose of this report, the dose commitment to a given tissue is defined as the integral over infinite time of the average dose rates delivered to the world's population as a result of a specific practice, e.g. a given series of nuclear explosions. The actual exposures may occur over many years after the explosions have taken place and may be received by individuals not yet born at the time of the explosions (1964 report, annex A, paragraph 147).

127. This concept has been used because it would permit the calculation of the total expected number of individuals eventually showing certain radiation effects as a result of any given test series, if the size of the population to which the dose commitment applied and the appropriate proportionality factors characterizing a linear dose-effect relationship with no threshold were valid and known. The number of affected individuals would then simply be the product of population size, proportionality factor and dose commitment. For further details regarding the general method of computation, see paragraphs 147-155 of annex A of the 1964 report.

128. To estimate dose commitments, the world-wide cumulative deposition of Sr^{90} uncorrected for decay is obtained from the annual deposition figures up to the end of 1965 given in table I and from the cumulative deposition before 1958 assumed to be 2 MCi. The results from the United States and from the United Kingdom networks are weighted according to the number of sampling stations in each network and yield a total figure of 12 MCi. Estimates of future deposition are based on the stratospheric inventory of 0.8 MCi Sr^{90} as of January 1966 (table V) and a mean residence time of fourteen months. Taking into account, as in earlier reports of the Committee, the area of the earth

between 80°N and 50°S (448×10^6 km^2), in which over 97 per cent of the deposition takes place,[65] gives an average expected deposit of

$$\frac{12.8}{0.448} = 28.6 \text{ mCi/km}^2.$$

This figure will be used for the purpose of estimating the Sr^{90} dose commitment. The figure differs slightly from that obtained in the 1964 report (31.7 mCi/km^2), as a consequence of a revision in the estimate of the global inventory of Sr^{90}.

129. Cs^{137} deposition is derived from the deposition of Sr^{90} by means of a Cs^{137}/Sr^{90} ratio of 1.6. This ratio is representative of measurements carried out at various sites, as shown in table II. The total integrated expected deposit of Cs^{137} is therefore 20.5 MCi. The expected deposit in each hemisphere is derived from this figure by using the ratio of the estimated cumulative deposits in either hemisphere as of January 1966 (table V). This gives integrated Cs^{137} deposits of 16.3 MCi in the northern hemisphere (0°-80°N) and 4.2 MCi in the southern hemisphere (0°-50°S), corresponding to 64.5 mCi/km^2 and 21.4 mCi/km^2, respecively.

External dose rates

130. External dose rates from all deposited fission products may be determined by means of ionization chamber measurements, by converting to dose rates directly from results of gamma-ray spectroscopy obtained in the field, or by calculating the rates from radio-activity measurements in soil cores.[18, 346, 349-364] Reasonable agreement is found when results obtained by these different methods are compared.

131. Figure 30 shows the external gamma air dose as measured outdoors 1 m above ground at Grove, Berkshire, United Kingdom[346] and Chiba, Japan.[31] These doses are obtained by subtracting a constant natural background from the measured dose rates. Thus, the net fall-out dose rates include an error due to fluctuations in background. Such fluctuations may be considerable, particularly where snow cover reduces the radiation from the ground during the winter. This is illustrated by figure 31, which shows the total external exposure rate in central Sweden during the years 1962-1965.[348]

132. At Argonne, Illinois, United States, the fall-out doses have been calculated from gamma spectrometric analyses of soil cores.[23, 74, 353] In 1963, the dose rate from fall-out was as high as 30 per cent of the external natural radiation.[351, 353, 356] Its contribution decreased in 1964, owing to the decay of Zr^{95}, Ce^{144} and Ru^{106}. Dose rates during 1965, due mainly to deposited Cs^{137}, were somewhat lower than the 1964 levels.

133. Beck,[364] comparing actual field measurements of dose rates to calculations based on deposition data at Westwood, New Jersey, United States, concluded that the best agreement with regard to Cs^{137} was obtained by assuming an exponential decrease of activity with depth having a 3 cm relaxation length. Measured soil profiles always reveal some penetration of deposited fall-out depending on the type of soil, on climatic conditions and on the fall-out history.[365-369] This exponential distribution in soil would imply a gamma-ray dose-conversion factor of 0.034 mrad/year per mCi/km^2 deposited Cs^{137}. A similar study in Japan gave 0.050 mrad/year per mCi/km^2.[370] For calculation, a value of 0.040 mrad/year per mCi/km^2 will be used in this report.

134. A higher dose-conversion factor (0.12 mrad/year per mCi/km^2) was used in the 1962 and 1964 reports. Such a value was obtained on the assumption that Cs^{137} was deposited on an infinite plane. In estimating dose commitments, the effect of this higher dose-conversion factor was offset by assuming that Cs^{137} disappeared from the ground with an effective mean life of fourteen years, whereas calculations based on the exponential distribution assume disappearance by radio-active decay alone (mean life forty-three years). These two approaches, therefore, lead to about the same numerical estimate of the external dose commitment from Cs^{137}, but that discussed in the previous paragraph is probably more realistic at present and is followed here.

135. From the expected integrated deposits per unit area in each hemisphere as given in paragraph 129, a dose-rate factor of 0.04 mrad/year per mCi Cs^{137}/km^2, a physical mean life of forty-three years, and geographical factors 1.2, and 1.0 (1964 report, annex A, paragraphs 147-155) the external dose commitments from Cs^{137} in the northern and southern hemisphere are 133 and 36.8 mrads, respectively. Weighting these figures by the relative size of the populations involved (91 per cent in the northern hemisphere) gives a world-wide external dose commitment of 124 mrads. By applying the same combined reduction factor of 0.2 that was used in previous reports to allow for shielding by buildings and screening by body tissues, a final estimate of the external dose commitment from Cs^{137} of 25 mrads is obtained, as compared to 29 mrads estimated in the 1964 report.

136. In the case of short-lived radio-nuclides such as Zr^{95}, Ru^{106} and Ce^{144}, accounting for the roughness of the ground would involve reducing by a factor of about two the dose rate estimated from deposition figures on the assumption that the nuclides constitute an infinite plane source.[371] The factor of about three suggested for Cs^{137} for the reduction in dose rate over a long period of time would be too high for short-lived radio-nuclides, since these may decay before substantial redistribution to deeper layers of soil can occur.

137. In the 1962 report, dose commitments due to short-lived nuclides were estimated from actual measurements of external doses after deduction of an estimated contribution of Cs^{137}. The same approach was followed in 1964, except for that fraction of the dose commitment from short-lived nuclides that had not yet been delivered and which was estimated from deposition figures. The correction factor discussed in the previous paragraph can be applied to this fraction which amounts, therefore, to 5 instead of 10 mrads. On the other hand, the measured values of external dose rates from short-lived nuclides must now be somewhat increased in the light of the new estimates of Cs^{137} deposition, thus leading to values of 109 instead of 92 mrads. Adding the 5 mrads estimated above gives a total dose commitment of 114 mrads instead of 104 as obtained in the 1964 report. Applying the combined correction factor for shielding and screening (0.2) leads to an estimated dose commitment from short-lived radioactive nuclides of 23 mrads that has now been essentially delivered in full.

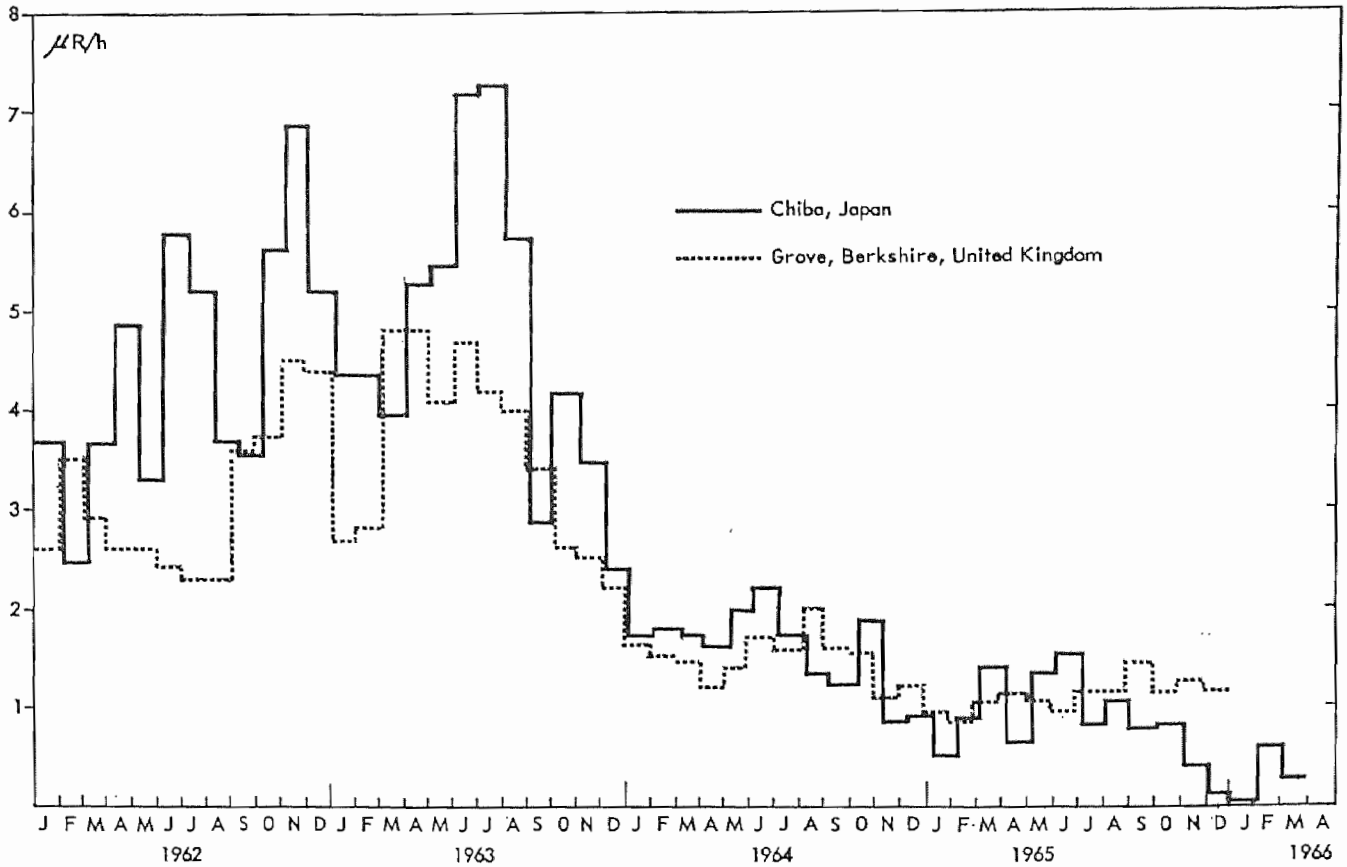


Figure 30. External gamma exposure rates due to fall-out, 1962-1966[31, 340, 347]

INTERNAL DOSES

Strontium-90

138. The dose commitment $\overline{D}$ to the world's population from Sr^{90} will be calculated by using the formula as derived by Lindell[345] and used in the 1964 report (annex A, paragraph 171):

$$\overline{D} = \theta \overline{F}_m \int_{-\infty}^{+\infty} c(t)dt \qquad (1)$$

where $\overline{D}$ = dose commitment in mrads; $\overline{F}_m$ = dose increment factor; 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).

139. As in previous reports, the integrated dietary Sr^{90}/Ca ratio is calculated by taking into account differences in the contamination patterns for three broad diet categories. Diet type I is that diet for which the predominant sources of calcium are milk or other dairy products. This diet type is generally consumed by peoples of North and South America, Europe and Oceania. Diet type II applies to the Middle East and to India where milk provides less than half of the total calcium intake. In diet type III, which is consumed predominantly in Japan and the Far East, milk provides only a minor source of the total calcium intake.

140. The estimates of mean Sr^{90}/Ca ratios in diets are obtained from the relationship,

$$C(t) = p_d F_d(t) + p_r F_r(t), \text{ pCi } Sr^{90}/\text{g Ca} \qquad (2)$$

where $F_d(t)$ = cumulative mean deposit of Sr^{90} (mCi/km^2); $F_r(t)$ = mean annual deposition rate (mCi/km^2 × y) at time t; p_d and p_r = proportionality factors discussed in paragraphs 89-91. It can be shown (1964 report, annex A, paragraph 167) that the infinite integral of the Sr^{90}/Ca ratio in the total diet can be expressed in terms of the effective mean life of Sr^{90} in the soil in years (T_m), of the proportionality factors p_d and p_r and of the integrated deposition of Sr^{90} per unit area, F, as follows:

$$\int_o^\infty C(t)dt = (p_d T_m + p_r)F, \text{ pCi years of } Sr^{90} \text{ per g Ca} \qquad (3)$$

141. The integrated Sr^{90}/Ca ratios in each diet as calculated from equation (3) must be combined by means of appropriate weighting factors to take into account differences in the size of the populations represented in each diet category and differences in fall-out deposit over the regions where the different types of diet are consumed. These weighting factors (Z) were calculated in the 1962 report (annex F III, table VIII) and are tabulated below together with the corresponding p_d and p_r factors used in equation (3).

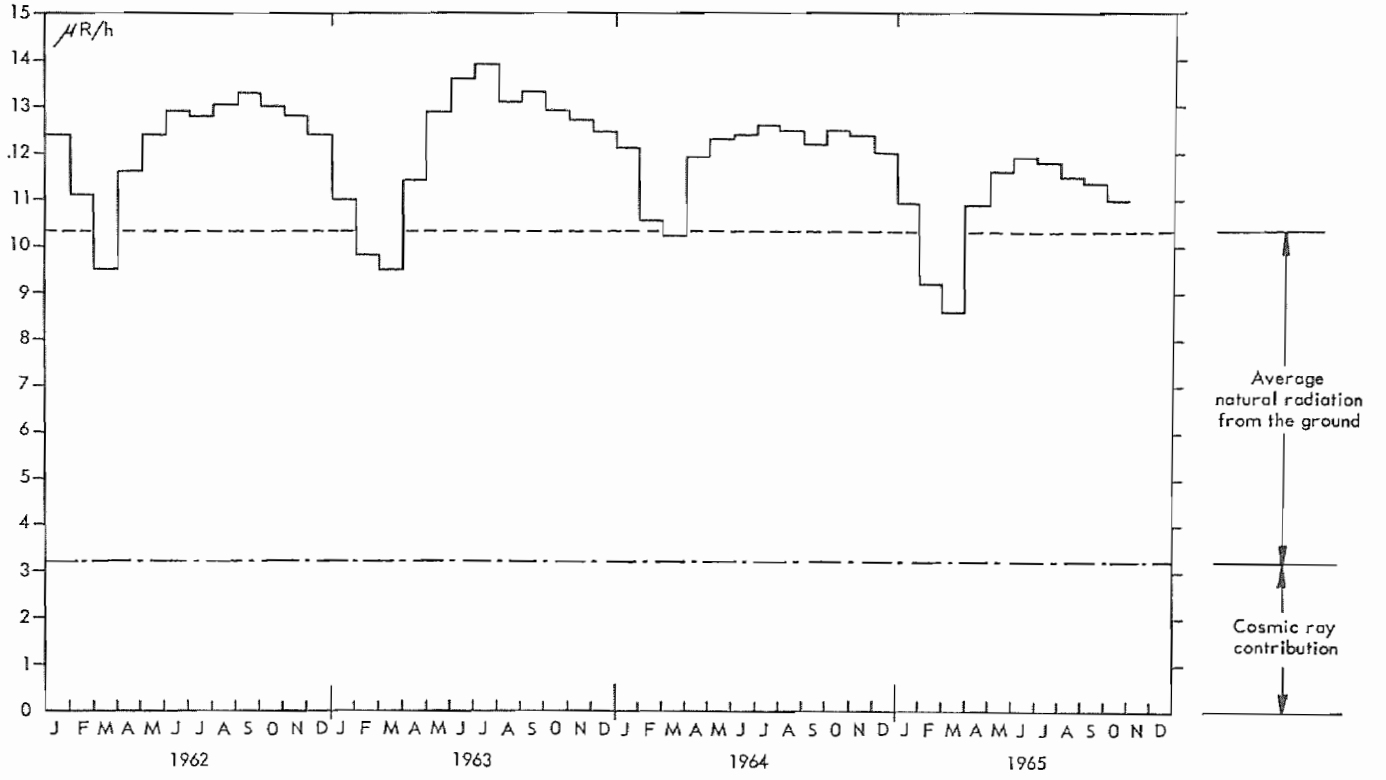


Figure 31. External exposure rates in central Sweden, 1962-1965[348]

Diet type	p_d	p_r	Z
I	0.4	1.3	0.7
II	0.6	2.6	0.5
III	0.7	2.0	0.7

142. Assuming losses of Sr^{90} from the soil of 2 per cent per year due to leaching etc. (paragraph 94), the effective mean life of Sr^{90} in the soil (T_m) is taken to be (as in the 1964 report) twenty-one years. The integrated deposition of Sr^{90} per unit area (F) is now estimated to be 28.6 mCi/km² (paragraph 128). By substituting successively in equation (3) the values of p_d and p_r for each diet type and the integrated Sr^{90} deposition, and by adding the contributions from individual diet types weighted by the Z factors, the world's average integrated dietary intake is estimated to be

$$(21 \times 0.4 + 1.3) \times 28.6 \times 0.7 + (21 \times 0.6 + 2.6) \times 28.6 \times 0.5 + (21 \times 0.7 + 2.0) \times 28.6 \times 0.7 = 746 \text{ pCi}$$

years of Sr^{90} per g Ca.

The average integrated dietary intake multiplied by the observed ratio (0.25) gives the average integrated bone level

$$\int_{-\infty}^{+\infty} c(t)\,dt = 186 \text{ pCi years of } Sr^{90} \text{ per g Ca.}$$

143. The evidence produced since the last report does not warrant changes in the parameters $\overline{F}_m$ and θ used in equation (1) relating the integrated dietary Sr^{90}/Ca level to dose commitments. The factors applied in the previous report will therefore be used again ($\overline{F}_m = 0.6$; $\theta = 1.4$ and 0.7 mrad/year per pCi Sr^{90}/g Ca for bone-lining cells and bone marrow, respectively). The dose commitments from Sr^{90} are thus

$\overline{D} = 1.4 \times 0.6 \times 186 = 156$ mrads to cells lining bone surfaces

$\overline{D} = 0.7 \times 0.6 \times 186 = 78$ mrads to bone marrow

Caesium-137

144. The method of calculation of the dose commitment from internal irradiation due to Cs^{137} is the same as used in the 1964 report of the Committee. It is assumed that the yearly average Cs^{137}/K ratios in the body (Q_t) can be related to the deposition of Cs^{137} by the formula given in paragraph 116, with proportionality factors $P_r = 2.5$ and $P_{2c} = 4.0$. The total integrated body burden for the northern hemisphere (Q_n) will therefore be:

$$Q_n = \sum_{t=1}^{\infty} Q_t = (P_r + 2P_{2c}) \sum_{t=1}^{\infty} f_t = (P_r + 2P_{2c})\, F_{cc},$$

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. The estimates of total expected deposition of Cs^{137} per unit area as obtained in paragraph 129 were 64.5 and 21.4 mCi/km² in the northern and in the southern hemisphere, respectively, giving $Q_n = 677$ and $Q_s = 225$ pCi years of Cs^{137} per g K.

145. The world-wide average integrated body burden (Q) is obtained by weighting the values for the northern and southern hemispheres by population (91 and 9 per cent, respectively) and correcting for the fall-out deposition pattern by means of the geographical factors of 1.2 and 1.0

$Q = (677 \times 0.91 \times 1.2) + (225 \times 0.09 \times 1.0) = 760$ pCi years of Cs^{137} per g K.

Using the same dose-rate factors as in the 1964 report (0.02 mrad/year per pCi Cs^{137}/g K) and assuming

approximately uniform tissue distribution of Cs^{137}, this burden corresponds to 15 mrads to gonads, bone marrow and cells lining the internal surfaces of bone.

Strontium-89, barium-140 and iodine-131

146. As there has been no appreciable deposition of Sr^{89}, Ba^{140} and I^{131} since the Committee prepared its report in 1964, there is no need to increase the estimates of doses from these nuclides which were made at that time.

Carbon-14

147. C^{14} data have so far followed quite closely the 1964 report predictions, and no re-evaluation of the corresponding dose commitment is necessary. By the year 2000, the doses delivered to bone marrow and gonads will each be 13 mrads and the dose delivered to cells lining the internal surfaces of bone will be 20 mrads, some 7 per cent of the total dose commitment due to C^{14}.

Conclusions

148. The dose commitments to be received by the world population by the year 2000 from the radio-activity released into the atmosphere as a consequence of nuclear explosions carried out to the end of 1965 are summarized in table XVII. They are given for specific tissues for the most important of the radio-active substances released into the environment through nuclear tests.

149. Differences between the present figures and the 1964 estimates are due to minor modifications in the numerical factors used in the computations. They do not reflect any basic change in approach. External radiation accounts for about two-thirds of the total dose to gonads and for only one-fifth of that to cells lining bone surfaces; in this respect, bone marrow is intermediate. The contribution of the 1964 and 1965 tests and of the burn-up of the Pu^{238} power source to the over-all dose commitments of the world population has been negligible. No information is available to assess the exposure of populations living in the proximity of the test sites.

150. The dose commitments, based as they are on a series of assumptions and on measurements which may not be entirely representative of the whole world situation, are subject to uncertainties discussed in the preceding sections of this annex. The values given in table XVII are considered more likely to be an over- rather than an under-estimate of the actual dose commitments.

151. An alternative to presenting dose commitments as such is to express them, as in the 1964 report, in terms of the periods of time during which natural radiation would have to be doubled to give a dose increase equal to the dose commitment. For all tests carried out up to the end of 1965, these periods are approximately three-quarters of a year for the gonads, two and a half years for the cells lining bone surfaces and one year and a half for the bone marrow.

Table I. World-wide deposition of Sr^{90} in megacuries[17, 63, 65, 68]

Year	Annual deposition		Cumulative deposition[a]	
	United States network	*United Kingdom network*	*United States network*	*United Kingdom network*
1957		0.66	2.0	2.1
1958	0.90	0.84	2.84	2 89
1959	1.13	1.46	3.89	4.28
1960	0.38	0.40	4.17	4.58
1961	0.46	0.56	4.52	5.02
1962	1.53	2.01	5.93	6.90
1963	2.59	2.87	8.35	9.61
1964	1.84	2.20	9.95	11.58
1965	1.0	1.1	10.7	12.4

[a] Corrected for radio-active decay.

Table II. Some average Cs^{137}/Sr^{90} ratios[a]

Type of sample	*1963*	*1964*	*1965*
Deposition			
Argentina[215]		1.7 (12)	1.7 (12)
Australia[103, 104]		1.7	
Canada[91, 99]	1.8 (144)	1.7 (288)	1.6 (288)
Finland[216]		1.6 (204)	1.6 (204)
Japan[31, 100]		1.4 (144)	1.6 (144)
New Zealand[24]		1.7 (14)	1.8 (17)
Union of Soviet Socialist Republics[217]	1.9	1.5	
United Arab Republic[105]		1.5	
United Kingdom[17, 72]		1.5 (28)	1.5 (14)
United States[22, b]		1.6 (23)	1.3 (12)

TABLE II. SOME AVERAGE Cs^{137}/Sr^{90} RATIOS[a] (*continued*)

Type of sample	*1963*	*1964*	*1965*
SURFACE AIR			
Union of Soviet Socialist Republics[217]	1.4	1.4	
United Kingdom[17, 72] Chilton, Berkshire	1.7 (12)	1.7 (12)	1.7 (6)
United States network[59, 62, 66, 218, 219]			
Northern hemisphere	1.3 (98)	1.4 (107)	1.4 (75)
Southern hemisphere	1.5 (78)	1.4 (83)	1.4 (40)
STRATOSPHERE			
Northern hemisphere (15-21 km)[59, 60, 62, 218]	1.7 (38)	1.6 (14)	1.4 (8)
Southern hemisphere (15-21 km)	1.5 (11)	1.5 (8)	1.3 (4)
Northern hemisphere (22-34 km)[49, 62, 218, 220]	1.5 (48)	1.5 (47)	1.5 (50)
64°S (22-34 km)	1.5 (35)	1.4 (37)	1.4 (23)

[a] Figures in parentheses indicate the number of samples.
[b] Based on New York City, New York and Westwood, New Jersey data only.

TABLE III. Sr^{90} IN SURFACE WATERS OF THE WORLD'S OCEANS AND SEAS, 1960-1961

(Based on reference 125)

	Average concentrations (pCi/litre)	*Number of samples*		*Average concentrations (pCi/litre)*	*Number of samples*
South China Sea	0.26	2	Baltic Sea	0.30	5
Sea of Japan	0.20	1	Red Sea	0.11	3
Sulu Sea	0.20	2	Indian Ocean	0.11	38
Pacific Ocean			Atlantic Ocean		
Northern hemisphere	0.23	42	Northern hemisphere	0.07	81
Southern hemisphere	0.08	24	Southern hemisphere	0.01	24
Black Sea	0.26	41			

TABLE IV. Sr^{90} IN NORTH ATLANTIC SURFACE WATER, 1960-1965

Sampling year	*Average value (pCi/litre)*	*Range (pCi/litre)*	*Number of samples*	*References*
1960-1961	0.07	0.03–0.14	81	125
1962	0.10	0.05–0.15	6	113, 118
1963	0.15	0.06–0.30	27	112, 115
1964	0.18	0.09–0.26	19	115
1965 (January-June)	0.14	0.07–0.19	20	115

TABLE V. GLOBAL INVENTORY OF Sr^{90} (IN MEGACURIES)[61, 62, 65, 86]

	May 1960	*May 1961*	*April 1962*	*January 1963*	*September 1963*	*January 1964*	*January 1965*	*January 1966 (estimated)*
STRATOSPHERE								
Northern hemisphere	0.50	0.34	2.1	6.2	4.1	3.1	1.10	0.45
Southern hemisphere	0.44	0.38	0.7	0.8	1.0	0.9	0.54	0.35
TOTAL stratosphere	0.94	0.72	2.8	7.0	5.1	4.0	1.64	0.80
TROPOSPHERE								
Northern hemisphere	0.05	0.04	0.03	0.24	0.19	0.24	0.08	0.06
Southern hemisphere	0.01	0.01	0.02	0.03	0.03	0.02	0.04	0.04
TOTAL troposphere	0.06	0.05	0.05	0.27	0.22	0.26	0.12	0.10
CUMULATIVE WORLD-WIDE DEPOSITION[a, b, c]								
Northern hemisphere	3.13	3.25	3.85	4.57	6.39	6.74	8.01	8.48
Southern hemisphere	0.92	1.04	1.23	1.36	1.54	1.61	1.94	2.20
TOTAL deposition	4.05	4.29	5.08	5.93	7.93	8.35	9.95	10.68
TOTAL ACCOUNTED FOR	5.05	5.06	7.93	13.20	13.25	12.61	11.71	11.58

[a] Based on continental sampling.
[b] Corrected for decay.
[c] Does not include local fall-out, estimated as 2.6 MCi in 1960.[61]

TABLE VI. GLOBAL EXCESS C^{14} INVENTORY[62, 64]

(10^{27} atoms)

	July 1963	*January 1964*	*July 1964*	*January 1965*	*July 1965*	*January 1966 (estimated)*
STRATOSPHERE						
Northern hemisphere	24.2	20.3	13.4	10.2	8.8	8.5
Southern hemisphere	5.9	5.3	5.8	5.4	5.5	5.0
TROPOSPHERE	25.3	27.1	29.9	25.9	28.1	28.5
OCEANIC UPTAKE						
Assuming 20 per cent of tropospheric content per year	13.5	17.0	18.9	21.7	24.4	27.2
Assuming 25 per cent of tropospheric content per year	16.0	19.3	24.9	26.4	29.8	33.3
TOTAL	68.9–71.4	69.7–72.0	68.0–72.0	63.2–67.9	66.8–72.2	69.2–75.3

TABLE VII. RATIOS BETWEEN CONCENTRATIONS OF Sr^{90} (Sr^{90}/Ca) IN TEETH AND IN THE SKELETON (WHOLE SKELETON AVERAGES) IN HUMANS OF DIFFERENT AGE IN 1962 AND 1964[146]

Year	*Age group*	
	5-13 years	*> 30 years*[a]
1962	0.60	0.34[b]
1964	0.47	0.30

[a] Values above thirty years were found to be independent of age.
[b] The values estimated for seven urban areas of the USSR vary between 0.33 and 0.37. They are based on 124 bone samples and 2,390 teeth samples pooled in fifty-four analyses.

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

The values are given in pCi/g Ca 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*	*1963*	*1964*	*1965*	*Type of study*	*References*
NORTHERN HEMISPHERE						
North America						
Canada	40–55°N	28[a]	31[a]	23[a]	A	151
United States						
conterminous	25–48°N	19[b]	19[b]	14[b]	A	221
Alaska	~ 62°N	17	13	14	B	221
Chicago, Ill.	~ 40°N	14	16	13	C	222, 223
New York City, N.Y.	~ 40°N	26	23	19	B	222, 223
San Francisco, Cal.	~ 40°N	10	9	9	C	222, 223
Europe						
Austria	47–49°N	25–42[d]			A	153
Belgium	~ 50°N	20[e]	27		A	224, 225
Czechoslovakia	48–51°N	21[c]	20[c]	18[c]	B	226
Denmark	55–60°N	24[f, g]	24[f, g]	17[f, g]	A	206, 207, 227
Faroes	60–70°N	131	154	115	A	227–229
Federal Republic of Germany	43–55°N	28[f]	28[f]	21[f]	A	230
Finland	60–67°N	22[g]	23[g]	18[g]	A	231
France	42–50°N	27[h]	35	29	A	232–234
Iceland	66–73°N	80[a]	65[a]	80[a]	A	235
Ireland	50–55°N	41	43		A	155
Norway	58–70°N	38[a]	50[a]	40[a]	A	69, 236
Switzerland	46–47°N	55[a]	55[a]		A	237, 238
USSR	32–70°N	30[a]	31[a]		A	239
United Kingdom	50–60°N	26[g]	28[g]	19[g]	A	155, 240

TABLE VIII. Sr^{90} TO CALCIUM RATIO IN MILK (*continued*)

The values are given in pCi/g Ca 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*	*1963*	*1964*	*1965*	*Type of study*	*References*
Near East						
Israel	~32°N	9		3.3	C	241, 242
Asia						
India	8–35°N	10[n]	8[n]	6[n]	A	243
Far East						
Japan	30–50°N	15	15	13	A	244–248
Middle America and Caribbean						
Jamaica	~13°N		16[l]	13[k]	B	221
Panama	~7°N			6[k]	B	221
United States						
Puerto Rico	18°N	12	10	8	B	221
Venezuela	5–12°N	4	6[m]	4[k]	B	221
Africa						
United Arab Republic	20–30°N	16[j]	17[l]		A	201
Oceania						
United States						
Hawaii	21°N	8	10	7	B	221
		SOUTHERN HEMISPHERE				
Oceania						
Australia	10–40°S	6[n]	8.9[n]	9.2[n]	A	103, 249
New Zealand	35–47°S	7[n]	11[n]	12[n]	A	87, 98, 250, 251
South America						
Argentina (littoral area)	35–55°S	3.7	6.2	6.5	B	215, 252

[a] Unweighted mean for all stations.
[b] Country-wide mean weighted by population.
[c] Dried milk from several factories, unweighted mean.
[d] Range of average values reported for three zones of different rainfall, covering the country.
[e] Annual average for three farms and three dairies.
[f] Dried milk country-wide mean weighted by production.
[g] Fresh milk country-wide mean weighted by production.
[h] January-August, unweighted mean for seven collecting stations.
[i] Cairo area only.
[j] Average for Delta region and Upper Egypt.
[k] January-August.
[l] April-December.
[m] January-November.
[n] Means for the European and Asian parts of the USSR.

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

Calcium in grammes per day in parentheses; Sr^{90} in pCi per day; Sr^{90}/Ca ratio in total diet in pCi/g Ca in column 10 (all values have been rounded out to two significant figures)

Types of study: A — Survey of individual foodstuffs
B — Total diet analyses
C — Widespread regular sampling
D — Local regular sampling
E — 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*	*References*
			NORTHERN HEMISPHERE							
North America										
Greenland	>60°N	1963	7.5	23	0.9	0.4	8.0	AC	26	253
		1964	7.8	69	0.7	0.4	8.0	AC	56	254
		1965								
United States	25–62°N									
Alaska (institutional diet sampling)		1963						BD	24	221
		1964						BD	37	221
		1965						BD	21[e]	221

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

Calcium in grammes per day in parentheses; Sr^{90} in pCi per day; Sr^{90}/Ca ratio in total diet in pCi/g Ca in column 10 (all values have been rounded out to two significant figures)

Types of study: A — Survey of individual foodstuffs
B — Total diet analyses
C — Widespread regular sampling
D — Local regular sampling
E — 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*	*References*
Chicago, Ill.			(0.64)	(0.16)	(0.10)	(0.04)	(0.11)			
		1963	8.4	6.4	2.0	1.1	1.5	AD	19	222
		1964	9.7	8.4	3.9	1.7	0.9	AD	25	223
		1965	7.9	5.7	3.7	1.3	0.9	AD	19	223
New York City, N. Y.		1963	18	6.6	2.6	1.3	2.8	AD	30	222
		1964	15	8.5	5.2	1.8	1.0	AD	32	223
		1965	11	6.2	4.6	1.7	1.3	AD	24	223
San Francisco, Cal.		1963	6.9	3.9	0.9	0.7	1.6	AD	13	222
		1964	5.5	4.7	1.8	0.9	0.7		14	223
		1965	5.5	2.6	1.6	1.3	0.6		11	223
United States (institutional diet sampling)		1963						BC	22 8–41	221
		1964						BC	26 14–54	221
		1965						BC	22[e] 11–43[e]	221
Europe	> 30°N									
Austria	47–49°N		(0.75)	(0.06)	(0.05)	(0.01)	(0.21)			
		1963	26	16	4.5	0.7	1.0	AC	45	153
		1964								
		1965								
Denmark	55–60°N	1963	18	28	4.4	0.9	2.0	AC	31	206
								BC	31	206
		1964	19	48	3.8	0.9	1.8	AC	43	207
								BC	39	207
		1965	13	22	2.3	0.8	1.1	AC	23	227
								BC	23	227
Faroes	60–70°N	1963	54	28	2.2	5.0	7.1	AC	58	228
		1964	63	48	1.7	5.0	8.9	AC	77	229
		1965	46	22	1.0	9.0	13.4	AC	56	227
Federal Republic of Germany	43–55°N	1963	14	9.9	3.0	1.8	0.9	AC		255
		1964	12	11	5.4	2.4		AC		255
		1965								
France	42–50°N		(0.54)	(0.07)	(0.08)	(0.05)				
		1965	15	7.2	1.8	1.3		AC	34	234
USSR (rural population)	35–70°N	1963						AC	62 46–99[d]	239
		1964							88 73–140[d]	239
		1965								
USSR (urban population)	35–70°N	1963						AC	67	239
		1964						AC	94	239
		1965								
United Kingdom	50–60°N		(0.63)	(0.04)	(0.05)	(0.03)	(0.35)[a]			
		1963	15	3.4	2.2	1.3	3.2	AC	23	256
		1964	17	4.3	1.6	1.2	4.0	AC	26	155
		1965	12	2.8	1.4	1.4	1.8	AC	18	257
Near East										
Israel			(0.37)	(0.23)	(——0.11——)		(0.17)		(0.88)	
		1965	11	4.5[c]	0.9[c]	0.5[c]	4.5[c]	AE	25	241, 242
Caribbean										
United States	18°N									
San Juan, P. R.		1963						AD	30	143
Far East										
Japan	30–50°N			(0.05)	(0.31)[b]	(0.07)	(0.20)[c]			
		1963		1.7	9.4	3.5	0.8	BE	28	258
		1964	0.9	2.1	7.2	3.0	2.5	BE	33	258
		1965	1.0	1.8	3.0	2.3	2.9	BE	25	258

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

Calcium in grammes per day in parentheses; Sr^{90} in pCi per day; Sr^{90}/Ca ratio in total diet in pCi/g Ca in column 10 (all values have been rounded out to two significant figures)

Types of study: A — Survey of individual foodstuffs
B — Total diet analyses
C — Widespread regular sampling
D — Local regular sampling
E — Irregular sampling

Region, area or country	*Latitude*	*Year*	*Milk and milk products*	*Cereals*	*Fruits and leafy vegetables*	*Root vegetables*	*Mixed leaves*	*Type of study*	*Total diet in pCi/g Ca*	*References*
Oceania										
United States										
Hawaii (institutional diet sampling)	21°N	1963						BD	14	221
		1964						BD	22	221
		1965						BD	29	221
			SOUTHERN HEMISPHERE							
Oceania										
Australia	10–40°S		(0.64)	(0.05)	(0.07)	(0.01)	(0.05)			
		1963	3.9	0.5	0.5	0.1	~0.3	AC	6.4	249
		1964							9.0	103
		1965								
South America										
Argentina (littoral area)	~35–55°S	1963	(0.45)	(0.02)	(0.11)	(0.02)	(0.09)			
			1.7	0.5	1.2	0.4	0.5	AD	6.5	252
		1964	(0.4)	(0.07)	(0.05)	(0.09)	(0.05)			
			2.3	2.1	0.9	0.8	0.4	AD	9.2	215
		1965	2.8	2.1	0.6	0.2	0.4	AD	6.7	215

[a] Including *ovis praefericata*.
[b] Including seaweeds.
[c] Including fish and shellfish, dairy products, eggs and meat.
[d] Range of average values reported for sixteen republics of the USSR.
[e] First half of the year 1965.

TABLE X. Sr^{90} IN HUMAN BONE

pCi Sr^{90} per gramme of calcium[a]
(Number of samples in parentheses)

Region, country or area	*Year*	*New-born and/ or still-born*[b]	*0-1 year*[c]	*1 year*	*2 years*	*3 years*	*4 years*	*5-19 years*	*> 19 years*	*Bone type studied (adults)*	*Average Sr/Ca ratio for adult skeleton*[c]	*References*
					NORTHERN HEMISPHERE							
North America (30–60°N)												
Canada	1963	2.3 (15)	6.0 (40)	5.4 (12)	4.8 (9)	3.4 (5)	3.1 (9)	2.6 (48)	1.7 (34)	V	1.0	259
	1964	4.3 (35)	8.4 (46)	9.5 (27)	———	5.8 (47)	———	3.8 (94)	3.1 (59)	V	1.8	260
	1965											
United States												
Chicago, Ill.	1963		3.5 (2)		3.7 (2)	2.8 (3)		2.4 (2)	1.1 (12)	V	0.65	261
	1964					2.0 (1)		3.1 (5)	1.4 (43)	V	0.85	223, 262
	1965								1.3 (16)	V	0.76	263
New York City, N. Y.	1963		6.8 (10)	6.9 (2)	3.7 (6)	2.8 (3)	2.2 (1)	2.1 (26)	1.6 (23)	V	0.94	261
	1964	6.9 (3)	7.9 (7)	4.9 (4)	6.0 (4)	3.2 (3)	4.4 (2)	3.2 (30)	2.0 (28)	V	1.2	223, 262
	1965	2.8 (6)	5.0 (5)	7.0 (3)	7.2 (2)	6.7 (6)	4.1 (2)	3.5 (39)	2.1 (16)	V	1.2	263
San Francisco, Cal.	1963		2.1 (32)	2.7 (2)	1.7 (7)	1.1 (5)	1.7 (5)	1.4 (27)	0.9 (22)	V	0.53	261
	1964	2.2 (13)	2.9 (1.5)	3.2 (8)	2.4 (4)	1.8 (2)		1.6 (24)	1.4 (14)	V	0.82	223, 262
	1965	1.6 (13)	3.3 (13)	3.8 (6)	3.1 (1)	3.0 (3)	1.8 (5)	1.7 (19)	1.2 (30)	V	0.68	263
All regions	1963		1.7 (4)	4.8 (3)	5.7 (8)	5.3 (11)	4.2 (14)	3.0 (141)	2.2 (55)	V	1.3	221
	1964		4.3 (4)	5.4 (7)	5.7 (10)	4.6 (16)	4.1 (15)	3.2 (160)	2.2 (46)	V	1.3	221
	1965		5.5 (6)	3.6 (3)	5.5 (6)	4.2 (6)	3.2 (10)	2.8 (66)	1.9 (19)	V	1.1	221
Caribbean (10–20°N)												
United States												
San Juan, P. R.	1963							2.2 (69)	1.7 (26)	V	1.0	143
Europe (45–70°N)												
Czechoslovakia	1964	4.6 (15)	7.1 (42)	8.3 (6)	3.0 (4)	3.9 (4)	4.0 (3)	3.3 (36)	1.8 (64)	V	1.1	226

TABLE X. Sr^{90} IN HUMAN BONE (*continued*)

pCi Sr^{90} per gramme of calcium[a]
(Number of samples in parentheses)

Region, country or area	*Year*	*New-born and/ or still-born*[f]	*0-1 year*[g]	*1 year*	*2 years*	*3 years*	*4 years*	*5-19 years*	*> 19 years*	*Bone type studied (adults)*	*Average Sr/Ca ratio for adult skeleton*[e]	*References*
Denmark	1963	2.8 (12)	4.2 (13)	4.9 (2)		2.3 (1)	5.1 (2)	2.2 (13)	1.2 (44)	V	0.71	206
	1964	3.8 (11)	6.5 (44)	5.9 (2)	3.9 (1)	5.1 (3)	9.1 (5)	3.9 (46)	2.4 (77)	V	1.4	207
	1965	2.7 (14)	6.1 (17)	8.5 (3)	6.5 (2)	8.4 (2)		4.1 (31)	2.7 (23)	V	1.6	227
Federal Republic of Germany	1963	2.3 (166)	4.0 (21)		2.9 (9)			1.4 (27)	0.5 (46)	T	—[d]	264, 265
	1964	3.1 (172)	4.9 (20)		6.0 (16)			2.8 (28)	0.8 (47)	T	—[d]	264, 265
	1965	2.5 (92)	6.2 (10)		5.5 (9)			2.7 (13)	1.1 (43)	T	—[d]	264
Norway	1963	2.5 (14)	6.9 (2)		5.3 (1)			3.2 (5)	1.7 (1)	V	1.0	266
	1964	4.8 (7)	8.9 (6)		9.2 (4)			4.2 (8)				266
	1965	5.7 (13)	15.0 (6)		12.0 (1)			5.3 (4)				266
Poland	1963	5.0[h] (53)	5.6[i] (55)	6.1 (3)	4.8 (2)		3.3 (2)	3.2 (23)	1.6 (66)	V	0.94	267
	1964	4.8[h] (44)	5.6[i] (49)	3.9 (3)	6.0 (4)		3.1 (1)	3.8 (10)	2.5 (56)	V	1.5	267
	1965											
Switzerland	1963								1.0 (47)	V	0.59	237, 238
	1963								0.70 (40)	R	0.70	237, 238
	1964								2.5 (15)	V	1.5	238
	1964								1.4 (4)	R	1.4	238
	1965											
USSR[b]	1963		5.0 (6)		4.2 (7)			1.9 (1,567)	1.0[c] (4,142)		1.0	156
	1964	3.5 (68)	5.9 (33)		6.9 (9)			2.9 (5,425)	1.4 (18,694)		1.4	157
	1965											
United Kingdom	1963[k]	2.4 (167)	5.2 (104)	5.1 (24)	4.1 (12)	2.2 (11)	2.3 (14)	1.6 (74)	0.6 (22)	F	—[e]	268, 269
	1963[l]	1.8 (20)	4.5 (17)				2.6[m] (11)	1.3[n] (15)	0.86 (47)	V	0.51	270
	1964[k]	3.0 (92)	8.6 (80)	9.2 (21)	6.4 (7)	4.9 (9)	4.6 (13)	2.2[n] (46)	0.5 (9)	F	—[e]	271, 272

TABLE X. Sr^{90} IN HUMAN BONE (*continued*)

pCi Sr^{90} per gramme of calcium[a]
(Number of samples in parentheses)

Region, country or area	*Year*	*New-born and/ or still-born*[f]	*0-1 year*[g]	*1 year*	*2 years*	*3 years*	*4 years*	*5-19 years*	*> 19 years*	*Bone type studied (adults)*	*Average Sr/Ca ratio for adult skeleton*[e]	*References*
	1964[l]	3.3	8.1	———	———	———	4.9[m]	——2.4——	1.5	V	0.88	270
		(15)	(12)				(14)	(13)	(44)			
	1965[k]	3.0	7.2	11.0	8.4	7.1	5.3	2.8	1.3	Diff.	—	270
	Jan.-June	(102)	(53)	(16)	(8)	(3)	(4)	(41)	(5)			
Far East (30–50°N)												
Japan	1963	1.4	———	———	2.0	———	———	1.4	0.4	R	0.4	273
		(17)			(38)			(44)	(47)			
	1964	2.0	———	———	5.1	———	———	2.8	0.9	R	0.9	274
		(36)			(14)			(58)	(39)			
	1965	2.2	3.6	8.5	11	6.0	3.6	2.5	1.0	R	1.0	275
		(12)	(8)	(2)	(1)	(1)	(1)	(27)	(20)			
SOUTHERN HEMISPHERE												
South America (~ 35°S)												
Argentina (littoral area)	1963	0.8										145
		(23)										
	1964											
	1965	1.6	2.0	2.3	2.0	——1.7——		1.5[j]				215
		(12)	(39)	(8)	(5)	(4)		(12)				
Oceania (10–40°S)												
Australia	1963	0.78	1.2	2.1	1.7	1.2	1.3	1.0	0.61	V	0.36	249
		(80)	(315)	(22)	(9)	(15)	(5)	(247)	(577)			
	1964	0.86	1.9	2.2	2.0	1.8	1.3	1.0	0.69	V	0.41	103
		(60)	(153)	(35)	(25)	(20)	(13)	(119)	(494)			
	1965	1.2	2.9	3.0	2.7	2.0	2.8	1.3	0.76	V	0.45	103
	January-June	(21)	(68)	(12)	(8)	(9)	(6)	(48)	(225)			

Note:
V—Vertebrae.
R—Ribs.
T—Tibiae.
F—Femora.

[a] All values have been rounded out to two significant figures.

[b] Average values for all areas investigated in the European and Asian territory of the USSR. Values in parentheses indicate number of individual samples collected, composed mainly of teeth in 5-19 and > 19 year groups (usually analyzed as pooled samples).

[c] Skeletal average for adults obtained by applying normalization factors determined in 1963 by Marei *et al.*, the ratios vertebrae/skeleton, ribs/skeleton, femur shaft/skeleton and teeth/skeleton being 1.7, 1.0, 0.4 and 0.3, respectively.

[d] No direct normalization factor determined; skeletal averages may be assumed to be higher by a factor of ~ 2.

[e] Vertical hemisections of femora analyzed; no recent normalization factors available.

[f] Still-born children and children of the age between zero and fourteen or zero and thirty days.

[g] Age between two weeks and one year or one month and one year.

[h] Age between zero and two months.

[i] Age between two and twelve months.

[j] Age between five and ten years.

[k] Country-wide survey, including England, Wales and Scotland.

[l] Limited survey in West London area.

[m] One to nine years of age.

[n] Ten to nineteen years of age.

TABLE XI. DEPOSITION OF Cs^{137} IN FINLAND AND Cs^{137} CONTENTS OF LICHEN AND REINDEER MEAT, 1961-65[178]

Year	Deposition Cs^{137} mCi/km²		Lichen			Reindeer meat		
	Annual	Cumulative[a]	Date	nCi/kg dry weight (mean ± σ)	Number of samples	Date	nCi/kg fresh weight (mean ± σ)	Number of samples
1960		24.0[b, c]						
1961	1.3[c]	24.7	8/61	16 ± 3.5	(5)	10/61	15.8	(2)
1962	10.6[c]	34.7	7/62	22 ± 3	(15)	2/62	17.2	(2)
1963	19.4[c]	53.3	7/63	37 ± 0.5	(3)	3/63	48.0	(12)
1964						3/64	50.0 ± 4	(60)
	10.2	63.3	7/64	64 ± 8	(3)	12/64	60.1 ± 5	(6)
1965						4/65	72.3 ± 9	(4)
	4.3	66.1	8/65	56 ± 3	(3)	12/65	55.3 ± 9	(4)

[a] Corrected for decay to the end of each year.
[b] For the 1960 value (end of year) that of Sr^{90} for Leningrad (15 mCi/km²) is used.
[c] Calculated from Sr^{90} data using Cs^{137}/Sr^{90} ratio of 1.6.

TABLE XII. THE VALUES OF BIOLOGICAL HALF-LIFE OF CAESIUM IN ADULT MAN

Author	Locality	Number of cases studied	Average half-life in days (range in parentheses)	References
Melandri	Bologna, Italy	1	165	183
Oberhausen	Landstuhl, Germany	1	144	276
Van Dilla	Los Alamos, N. Mex., U.S.A.	4	128 (113–150)	184
Richmond	Los Alamos, N. Mex., U.S.A.	4	126 (110–146)	277
McNeill	Chalk River, Canada	3	115 (110–119)	278
Taylor	London, U.K.	4	109 (79–123)	279
Jeanmaire	Paris, France	1	100	188
Jordan	Bethesda, Md., U.S.A.	3	99 (76–126)	187
Colard	Mol, Belgium	2	99	280
Rundo	Harwell, Berks., U.K.	14	98 (58–149)	281
Miller	Chicago, Ill., U.S.A.	4	94 (82–100)	282, 283
Hesp	Windscale, West Cumberland, U.K.	2	92 (68–116)	185
Naversten	Lund, Sweden	6	77 (63–89)	186
Häsänen	Helsinki, Finland	6	63 (42–93)	284

TABLE XIII. Cs^{137} IN MILK

Values are given in pCi/litre and represent yearly averages unless otherwise indicated

Type of study: A—Widespread systematic survey
B—Local systematic survey
C—Irregular sampling

Region, area or country	1963	1964	1965	Type of study	References
NORTHERN HEMISPHERE					
North America					
Canada (40–55°N)	170[a]	210[a]	110[a]	A	151
United States (25–48°N) (conterminous)	110[b]	110[b]	57[b]	A	221
United States Alaska (62°N)	120	120	57	B	221
Europe					
Austria (47–49°N)	160–290[e]			A	153
Belgium (~ 50°N)	300[g]	110[a]		B	224, 225
Denmark (55–60°N)	110[d]	110[d]	56[d]	A	206, 207, 227

TABLE XIII. Cs^{137} IN MILK (*continued*)

Values are given in pCi/litre and represent yearly averages unless otherwise indicated

Type of study: A — Widespread systematic survey
B — Local systematic survey
C — Irregular sampling

Region, area or country	*1963*	*1964*	*1965*	*Type of study*	*References*
Faroes (60–70°N)	790[h, r]	1,400[h, r]	1,100[h, r]	A	227–229
Federal Republic of Germany (43–55°N)	130[d]	110[d]	110[d]	A	230
Finland (60–67°N)	240[i]	250[i]	190[i]	A	285
France (42–50°N)	240[j]	220[j]	130[j]	A	232–234
Iceland (66–73°N)	580[r]	870[r]	750[r]	A	235
Ireland (50–55°N)	190	170		A	155
Italy (37–47°N)	160[b]	200		A	286, 287
Norway (58–70°N)	540[a, e, r]	600[a, e, r]	520[a, e, r]	A	288
	330[a, f, r]	430[a, f, r]	360[a, f, r]	A	69, 236
Sweden (55–70°N)	180[l]	172[l]	110[l]	A	289
Switzerland Geneva (~46°N)	210[m]	180		B	237, 238
United Kingdom (50–60°N)	130[d]	150[d]	98[d]	A	155, 240, 256
USSR (35–70°N)	210[a]	160[a]		C	239
Middle America, South America and Caribbean					
Jamaica (~13°N)		350[p]	280[k]	B	152
Panama (~7°N)			40[k]	C	152
United States Puerto Rico (18°N)	88	70	42	B	221
Venezuela (5–12°N)	22[p]	26	21[k]	B	152
Asia					
India (8–35°N)	29[a]	15[a]	11[a]	A(C)	243
Near East					
Israel (~32°N)	35[a]		25[a]	C	241, 242
Far East					
Japan (30-50°N)	120	95	59[k]	C[o]	244–248, 290
Oceania					
United States Hawaii (21°N)	73	77	51	B	221
SOUTHERN HEMISPHERE					
Oceania					
Australia (10-40°S)	33[a]	49[a]	47[a]	A	291, 292
New Zealand (35–47°S)	56[a, n]	90[a]	96[a]	A	98, 250
South America					
Argentina (littoral area, 35-55°S)	13	20	20	B	102, 215

[a] Unweighted mean for all milksheds surveyed.
[b] Country-wide mean weighted by population.
[c] Average values for three zones of different yearly rainfall, covering the whole country.
[d] Country-wide mean weighted by production.
[e] Unweighted mean from the survey of thirty milksheds.
[f] Unweighted mean from the survey of ten milksheds.
[g] Simple average of values obtained from three farms and three dairies.
[h] Locally produced
[i] Non-weighted average of three milksheds for the period October 1962-July 1963.
[j] Unweighted mean of representative samples from all departments of France.
[k] January-August only.
[l] Country-wide mean weighted by consumption.
[m] June-December only.
[n] July-December only.
[o] Non-systematic widespread sampling.
[p] April-December only.
[r] Unusually high levels due to heavy rainfall and particular grazing conditions.

TABLE XIV. Cs^{137} IN TOTAL DIET

Yearly average values are given in pCi/day unless otherwise indicated (all values have been rounded out to two significant figures)

Type of study: A—Survey of individual foodstuffs
B—Total diet analyses
C—Widespread sampling
D—Local sampling
E—Irregular sampling

Region, area or country	*Year*	*Milk and milk products*	*Meat*	*Miscellaneous*	*Total*	*Type of study*	*Remarks*	*References*
North America								
Greenland (> 60°N)	1963	24	180[a]	95	300	AC	Area of poor grazing conditions and high rainfall	253
	1964	24	310[a]	230	560	AC		254
	1965							
United States								
Chicago, Ill. (42°N)	1963[b]	58	34	58	150	AD		293
	1963[c]	88	5	6	99	AD		294
	1964[b]	69	43	90	200	AD		293
	1964[c]	120	9	13	140	AD		294
	1965[b, d]	51	38	64	150	AD		294, 295
	1965[b, d]	82	5	12	99	AD		294, 295
United States conterminous (25–48°N) (institutional diet sampling)	1963				140 (14–270)[f]	BC		221
	1964				150 (70–300)[f]	BC		221
	1965				100 (40–220)[f, k]	BC		221
Alaska (62°N) (local institutional diet sampling)..	1963				140	BD		221
	1964				160	BD		221
	1965				65[k]	BD		221
Europe								
Denmark (50–60°N)	1963	50	94	130	270	AC		206
					(250)	BC		206
	1964	53	91	180	320	AC		207
					(320)	BC		207
	1965	26	64	100	190	AC		227
					(240)	BC		227
Faroes (60–70°N)	1963	280	570	170	1,000	AC	Area of high rainfall and poor grazing conditions	228
	1964	430	210	250	890	AC		229
	1965	340	290	250	880	AC		227
Federal Republic of Germany (43–55°N)	1963	50	60	90	200	AC		255
	1964	62	110	88	260	AC		255
	1965				150	AC		296
Sweden (55–70°N)	1964	90	75	100	260[g]	AC		194
USSR (rural population) (35–70°N)	1963				250 (130–450)[j]	AC		239
	1964				330 (190–550)[j]	AC		239
	1965							
USSR (urban population)	1963				210	AC		239
	1964				290	AC		239
	1965							
United Kingdom (50–60°N) ..	1963	57	70	51	180	AC		256
	1964	66	59	36	160	AC		155
Near East								
Israel (~ 32°N)	1965				92	AE		242

TABLE XIV. Cs^{137} IN TOTAL DIET (*continued*)

Yearly average values are given in pCi/day unless otherwise indicated (all values have been rounded out to two significant figures)

Type of study: A—Survey of individual foodstuffs
B—Total diet analyses
C—Widespread sampling
D—Local sampling
E—Irregular sampling

Region, area or country	*Year*	*Milk and milk products*	*Meat*	*Miscellaneous*	*Total*	*Type of study*	*Remarks*	*References*
Far East								
Japan	1963				65	BC		297, 298
	1964				64	BC		299, 300
	1965				41	BC		299, 300
Oceania								
United States								
Hawaii (institutional diet sampling) (21°N)	1963				97	BD		221
	1964				120	BD		221
	1965				120[k]	BD		221

[a] Including eggs and fish.
[b] Average adults' diet.
[c] Infants' diet during the first year of life.
[d] January and April sampling only.
[e] Widespread dietary survey in twenty-one boarding schools in the United States, based on composite diets of children and adolescents of six to eighteen years of age.
[f] Range of yearly averages reported for individual schools.
[g] January 1964 sampling.
[h] Dairy products, eggs, meat, fish and shellfish.
[i] Data for 1963 include three series of sampling at four locations in the period January-July.
[j] Range of values calculated for daily average intake by the rural population of sixteen republics of the Soviet Union.
[k] January-June only.

TABLE XV. CAESIUM-137 IN MAN

All values are group averages in pCi Cs^{137}/g K unless otherwise indicated

(Figures in parentheses indicate the number of measurements performed)

Region, country or area of residence	1963 I	1963 II	1963 III	1963 IV	1964 I	1964 II	1964 III	1964 IV	1965 I	1965 II	1965 III	1965 IV	1966 I	References
NORTHERN HEMISPHERE														
North America														
Canada (northern) (H)	~ 1,000[b, c]				~ 1,400–2,800[b, c]				~ 2,800–9,000[b, c]					301, 302
Canada														
Ottawa										170 (63)				303
United States														
Alaska (Eskimos) (H)	240–4,500[a, b]				360–9,100[a, b]				310–7,500[a, b]					304–306
Alaska (Eskimos) Anaktuvuk Pass (H)					3,400[b] (50)		8,200[b] (51)	5,600[b] (30)	4,600[b] (25)	4,600[b] (25)				307
Brookhaven, N.Y.	~ 60 (10)	70 (10)	95 (10)	120 (10)	130 (10)	140 (10)	180 (10)							308
Los Alamos, N. Mex.		72 (86)	92 (80)	95										309
Los Angeles, Cal.			59 (14)	95 (36)	93 (36)	114 (25)	99 (15)	91 (34)	91 (50)	86 (40)	84 (51)	76 (50)	63 (23)	310
Europe and Asia														
Belgium	73 (507)		117 (529)		146 (552)		169 (623)		144 (407)		125 (381)		105 (167)	280
Denmark														
Riso			89 (14)	132 (14)		143 (10)	195 (10)	176 (10)	191 (20)		170 (15)	153 (20)		206, 207, 227
Federal Republic of Germany	47 (42)	54 (42)	83 (40)	120 (40)	141 (40)	163 (40)	152 (40)	148 (40)	131 (40)	119 (40)	104 (40)	101 (40)		311
Finland														
Helsinki	112 (49)	120 (49)	175 (49)	200 (49)	204 (24)	218 (49)				205[h]	200 (78)	160 (27)		173, 284, 312, 313
Lapland (H)	1,430–4,420[a]				2,260–8,360[a]				2,370–10,640[a]				2,000–8,200[a] (121)	173, 312, 314
France (northern)	70 (~ 60)	103 (~ 60)	132 (~ 60)	168 (~ 60)	197 (~ 60)	240 (~ 60)	273 (~ 60)	198 (~ 60)	242 (~ 60)	220 (~ 60)	159 (~ 60)	155 (~ 60)	103 (~ 60)	315, 316
Italy														
Bologna	74 (13)	81 (13)	124 (13)	150 (13)										183
Norway (western) (R)	332[f] (11)			651 (169)										288, 317
Oslo[e]	178 (23)			338 (23)	357 (23)					337 (25)				288, 317
Masfjorden (R)	870 (11)			1,430 (10)				2,000 (10)				1,230 (10)		288, 317
Poland														
Lodz		133 (23)		181 (23)		128 (22)		200 (22)						318

TABLE XV. CAESIUM-137 IN MAN (*continued*)

All values are group averages in pCi Cs^{137}/g K unless otherwise indicated

(Figures in parentheses indicate the number of measurements performed)

Region, country or area of residence	1963 I	1963 II	1963 III	1963 IV	1964 I	1964 II	1964 III	1964 IV	1965 I	1965 II	1965 III	1965 IV	1966 I	References
Sweden														
Stockholm	60 (13)	72 (9)	122 (8)	151 (22)	182 (20)	235 (10)		191 (13)						194
Lapland (H)	1,700-2,900[a] (105)				2,600–5,400 (94)				3,000–6,400 (96)					174
Switzerland														
Geneva		74 (~ 15)	107 (~ 15)	151 (~ 15)	180 (15)	188 (17)	196 (14)	184 (22)						237, 238
USSR (far north) (H)	3,570–23,600[b, d] (60)				7,140–25,700[b, d] (30)				1,400–34,000[b, d]					175, 319
United Kingdom														
Berkshire	54 (10)	64 (12)	90 (11)	115 (13)	138 (13)	150 (13)	162 (15)	170 (17)	166 (18)	160 (21)	149 (19)	124 (21)	104 (24)	320, 321
West Cumberland	107 (14)	138 (14)	198 (14)	249 (14)	255 (14)	256 (14)	272 (14)							322
Far East														
Japan	64[g] (68)	59[g] (68)	66[g] (74)	40 (23)		96 (20)	101 (42)	107 (41)	90 (29)	82 (54)	71 (16)			275, 323
SOUTHERN HEMISPHERE														
Oceania														
Australia														
Adelaide	36 (24)	33 (19)	33 (22)	21 (21)	32 (29)	36 (6)	31 (4)	45 (4)	67 (7)	66 (29)	67 (11)	62 (11)		324

Note:

(H) = High levels of Cs^{137} due to operation of the lichen-reindeer (caribou) food chain mechanism (paragraphs 98–101).

(R) = High levels of Cs^{137} due to very high average rainfall in the area and poor grazing conditions for cattle.

[a] Average values for different groups of local population (Lapps or Eskimos only).

[b] Approximate values calculated only for adults from reported total body burden assuming 140 g of potassium in the body, or from data on caesium-137 body weight ratio assuming approximately 1.9 g K/kg in females and 2.3 g K/kg in males.

[c] Approximate values calculated from excretion of Cs^{137} in urine in groups of population subsisting to a varying degree on reindeer meat in their diet.

[d] Range of concentrations observed in individual reindeer breeders.

[e] Group of schoolboys sixteen to eighteen years old in 1963 and eighteen to twenty years old in 1965.

[f] Masfjorden residents.

[g] Estimated from radio-chemical analysis of muscles.

[h] Calculated from total body burden assuming 140 g K in an average adult male.

TABLE XVI. C^{14} CONTENT OF SOME FOODSTUFFS AND HUMAN TISSUES COLLECTED IN VARIOUS PLACES[209-211]

The values are expressed in per cent increase of C^{14} specific activity above pre-test levels

(Number of samples in parentheses)

Area or locality	*Year:*	*1962*		*1963*				*1964*			
	Quarter:	*III*	*IV*	*I*	*II*	*III*	*IV*	*I*	*II*	*III*	*IV*
Foodstuffs											
Cereals, Norway	64°N									94 (3)	
Milk, Norway	64°N					84 (1)	71 (2)	71 (1)		91 (3)	
Milk, United States	30–50°N				40 (1)						
Meat, poultry and eggs, United States	30–50°N			28 (4)		16 (2)					
Fruits, United States	30–50°N		12 (1)	18 (3)							
Potatoes, United States ..	30–50°N					48 (2)					
Total diet, United States	30–50°N					26 (7)	33 (6)	34 (1)			
Human tissues											
Soft tissues and blood											
Norway	64°N					34 (2)	41 (3)	43 (3)	46 (4)	53 (2)	53 (2)
United States	30–50°N	24 (2)				33 (1)	16 (3)	38 (14)	54 (1)		
Australia, Melbourne ..	38°S								57[a'] 14[a'']		

[a] Data for a subject from Melbourne, Australia, who stayed in the United States for two weeks prior to death (a' — plasma; a'' — erythrocytes).

TABLE XVII. DOSE COMMITMENTS FROM NUCLEAR EXPLOSIONS

Tissue	*Source of radiation*	*Dose commitments (mrad) for period of testing 1954-1965*	*Paragraphs*
Gonads	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Cs^{137}	15	145
	C^{14} [a]	13	147
	TOTAL[b]	76	
Cells lining bone surfaces	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Sr^{90}	156	143
	Cs^{137}	15	145
	C^{14} [a]	20	147
	Sr^{89}	0.3	146
	TOTAL[b]	240	
Bone marrow	External, short-lived	23	137
	Cs^{137}	25	135
	Internal, Sr^{90}	78	143
	Cs^{137}	15	145
	C^{14} [a]	13	147
	Sr^{89}	0.15	146
	TOTAL[b]	150	

[a] As in the 1964 report, only the doses accumulated up to year 2000 are given for C^{14}; at that time, the doses from the other nuclides will have essentially been delivered in full. The *total* dose commitment to the gonads due to C^{14} from tests up to the end of 1965 is about 180 mrads.

[b] Totals have been rounded off to two significant figures.

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Annex C

THE GENETIC RISKS OF IONIZING RADIATION

CONTENTS

I. Introduction

1. The 1958[1] and 1962[2] reports of the Committee surveyed in some detail the subject of the genetic effects of ionizing radiation. The present paper updates those parts of the earlier reviews that require significant revision in the light of recent progress, with particular regard to those results that more or less directly bear on the estimates of risk from radiation for human populations.

2. The present annex is not self-contained and should be read in the context of the earlier reviews made by the Committee, particularly of that contained in its 1962 report. Many problems that were earlier considered in some detail by the Committee will not be reviewed here, since they are not considered to be directly relevant to the question of risk estimates.

II. The prevalence of naturally-occurring hereditary defects and diseases

3. The surveys of naturally-occurring hereditary defects and diseases made by the Committee in its 1958 and 1962 reports are still in large part valid. The present annex will therefore refrain from discussing again the hereditary factors involved in the general morbidity of the population or in the occurrence of congenital malformations, since too little has been recently added to our knowledge both with regard to the extent of their genetic component and to the mechanisms through which that component acts.

4. Likewise, no detailed discussion will be made of those unfavourable traits that are maintained in human populations by mechanisms which, though reasonably well known, are not predominantly mutational. Exposure of a population to radiation is likely only slightly to affect the prevalence of these traits by comparison with those primarily maintained in the population by recurrent mutations. For the purpose of estimating risks, the relevance of a discussion of these traits is therefore, at most, accessory.

5. Finally, the distribution of quantitative traits in human populations also will not be discussed in the present review. Their importance from the point of view of risk estimates cannot be doubted, but our knowledge has not sufficiently progressed to warrant a revision of the rather detailed discussion contained in the 1958 report of the Committee.

Major disabilities due to single gene traits maintained by recurrent mutation

6. In earlier reports, the Committee considered as an individual category those harmful traits whose mechanism of inheritance was understood and whose prevalence in the population was mainly determined by recurrent mutations, excluding those traits that were due to a cytologically detectable chromosome anomaly. This category, apparently due to gene mutations, included autosomal dominant, autosomal recessive and sex-linked traits.

7. In its 1958 report, the Committee accepted a figure of about 1 per cent as the proportion of all live-born infants falling into this category. Subsequent information suggests that this figure is slightly low. Of this 1 per cent, about 70 per cent are believed to be accounted for by approximately fifty dominant traits determined by autosomal genes. The terms "dominant" and "recessive" as used here are conventional in human genetics. Thus the term "dominant" includes traits usually recognized by the heterozygous expression of the mutation, although in the great majority of examples the condition of individuals homozygous for such genes has never been observed.

8. *Dominant autosomal traits.* The relative frequency of the fifty dominant autosomal traits referred to in the previous paragraph is now estimated to be approximately 80 per cent. An analysis of the proportion of sporadic cases at birth (i.e., cases having no affected relatives) among all living cases suffering from these disabilities indicates that about 4 per cent of the cases are due to new mutations arising in parental gametes.[3] The frequency of live-born children carrying new dominant mutations responsible for major disabilities appears therefore to lie around 3×10^{-4} ($0.01 \times 0.80 \times 0.04$).

9. This frequency corresponds to a rate of 1.5×10^{-4} mutations per generation per gamete. The average mutation frequency of the individual loci responsible for those traits is not known, estimates at individual loci being available only for a few of them which are rather more mutable and not likely to be representative of the rest. It is a safe assumption, however, that the fifty traits referred to in paragraph 7 are determined at fifty loci at least, and, for the purpose of the present discussion, this will be assumed to be true. With fifty loci the average mutation rate per locus per generation would be around 3×10^{-6}. This rate is an upper limit to the average mutation rate at all loci at which dominant visible mutations occur, but it seems unlikely that the average mutation rate can be lower than by, at most, one order of magnitude. If this were so, the number of loci involved would be correspondingly higher.

10. Other limitations may result from the possible inclusion of phenocopies and of recessives mimicking dominant phenotypes. Again, however, the cumulative frequency of traits here considered is unlikely to be more than slightly over-estimated by ignoring these complications.

11. Finally, the frequency of sporadic cases among all cases of the traits considered here, though justified by the need to obtain an over-all mutation rate, may be misleading in as much as it may suggest that the traits involved confer a rather small reduction in fitness. It must be realized, however, that the traits on which the estimate rests span the whole range of severity, from epiloia where the mean fitness is very low, to ichthyosis and alopecia areata in which it is almost unimpaired, even though the latter two diseases may be a cause for serious physical or social hardship to some affected individuals.

12. *Sex-linked traits.* The category of traits that can be confidently attributed to mutations at loci lying on the X chromosome includes, according to a recent survey of the literature,[4] some sixty different disabilities. Another study[5] indicates that the number of clearly sex-linked harmful traits that are maintained in the population by recurrent mutation is forty-nine, most of them with very severe effects.

13. A tentative analysis of the distribution of frequencies of these forty-nine traits suggests that the corresponding over-all mutation frequency is approximately 1.3×10^{-4} mutations per gamete per generation. Assuming that each trait is determined at a single locus, the rate per locus per generation is therefore 2.7×10^{-6}, in reasonable agreement with the upper limit of the estimate obtained independently in paragraph 9 for autosomal dominants.

14. It is important to note, however, that only sixteen of the forty-nine sex-linked traits occur at a rate higher than 10^{-6}. While it is likely that our inventory of relatively common traits maintained by high spontaneous mutation rates is almost complete, it is to be expected that we shall continue to recognize an increasingly large number of very rare traits caused by X-linked mutations. It follows, therefore, that, as in the case of autosomal dominants, the average rate per locus here obtained applies only to those loci that have been sampled so far, and that it would be misleading to assume that they are representative of the spontaneous rates per locus over the rest of the genome.

15. *Autosomal recessive traits.* Recessive traits in man are usually much more severe in their effects (homozygous expressions) than those due to dominant genes where, in general, only the heterozygous expressions are observed. Special methods are now available by means of which it is possible to recognize with a high degree of probability individuals heterozygous for recessive genes, at least when the corresponding gene frequencies in the population are sufficiently high. Many recessive gene traits, probably the great majority, are so uncommon that they are very seldom seen or their genesis recognized. Finally, although recessive detrimental traits are less common, the corresponding gene frequencies, on the average, must be very much higher than those of genes whose expression is easily recognizable in heterozygotes.

16. The estimation of the rate of spontaneous mutation over the whole genome in man can be approached in several independent ways. Most of them make use of information obtained from studies of the sex-ratio and are based on the hypothesis that differences in the values of the ratio at birth and during various stages of intrauterine life may result from the elimination of various stages of development of males hemizygous for recessive lethals on the X chromosome. The assumption is obviously very crude, as it ignores the physiological factors involved in the differential survival of foetuses of either sex, and the complications due to non-disjunction, to loss or to structural rearrangements of sex chromosomes which are now well documented in man. The sex-ratio might also be affected to some extent by sex-limited autosomal mutations. Accepting the assumption that the sex-ratio is largely controlled by the occurrence of sex-linked recessive mutations and using data on the sex-ratio of dead foetuses, the rate of mutation over the whole X chromosome has been estimated to be 6.8×10^{-3} per generation for mutations expressed between the twenty-eighth week of pregnancy and birth.[6] The same method applied to different data yields similar estimates.[7, 8]

17. Other data were analysed by a more refined method[9] based on the assumption that lethal recessive mutations on the X chromosome accumulate with the age of the mother. A negative correlation between the sex-ratio of live-born children and maternal age is therefore expected and makes it possible to estimate the rate of accumulation of mutants with time. This study also presents several limitations, because the effect of birth order and paternal age cannot in these data be separated from that of the age of mothers, and because maternal age may be correlated with maternal factors differentially affecting foetuses of either sex. The method also fails to remove the bias due to the possible correlation between maternal age and the occurrence of chromosome losses that might alter the sex-ratio at birth. Besides, very large samples are needed to achieve a sufficient resolving power, because the rate of accumulation of mutations in oöcytes is expected to be low if at least part of the mutations that occur spontaneously are due to copying errors, whose frequency of occurrence is more dependent on the rate of cell multiplication than on time alone.

18. The study yielded an estimate of $(2 \pm 0.75) \times 10^{-3}$ mutations per generation per X chromosome in female gametes. Neglecting the theoretical limitations that were outlined above, the estimate applies to recessive lethals acting between conception and birth.

19. Some of the limitations that weaken the validity of the previous methods are removed by studying the relationship between the sex-ratio and the age of the maternal grandfather at the birth of the mother.[10] The relationship is believed to be determined by the accumulation with age of recessive lethals that arise during the continued multiplication of spermatogonia. X chromosomes carrying mutations will be transmitted to daughters, and, from them, to 50 per cent of the male offspring that will thus be eliminated. The age of the maternal grandfather is therefore expected to be positively correlated with male mortality *in utero* and therefore with the sex-ratio of aborted foetuses and stillborn, and negatively correlated with the sex-ratio of live-born children.

20. Although it is not possible even with this method to allow for the occurrence of mutations with sex-limited expression and for structural rearrangements of the sex-chromosomes, the three-generation approach has the advantage that the correlation between time and sex-ratio is unlikely to be obscured by maternal physiological factors or by the occurrence of chromosome losses.

21. The method has been applied to a sample of live-born children with inconclusive results.[11, 12] When applied to a sample of some 7,000 dead foetuses,[13] the method showed a significant positive correlation of grandpaternal age with the sex-ratio, once the confounding effects of other concomitant variables, such as the age of the parents, age of the maternal grandmother, birth order and so on were eliminated. It was thus possible to estimate a rate per generation in male gametes of $(3.6 \pm 0.9) \times 10^{-3}$ sex-linked recessive lethals acting between twenty weeks after conception and birth. An earlier survey of the same size based on still-born gave an estimate of 2.5×10^{-3}, in good agreement with the previous one.

22. The rate thus obtained is probably an underestimate of the total rate of mutation, since, judging from *Drosophila* data,[14-16] sex-linked lethals, as opposed to autosomal lethals, are strongly selected against in spermatogonia, most studies indicating a reduction of around 50 per cent. If the same severe selection holds in man, the rate of sex-linked recessive lethals per generation in the viable male gametes is only half that in the genome before meiosis.

23. A certain number of sex-linked mutants must also be acting later in life. Their rate of induction must be lower, however, since the excess male mortality attributable to genetic causes is certainly very small. If, on the other hand, the rate of spontaneous mutation on the X chromosome is higher in spermatogonia than in oöcytes, the estimate of the spontaneous mutation rate over the whole genome will over-estimate the over-all rate representative of both sexes, but not by a factor of more than two. Although not all the reservations that may be advanced have been reviewed here, as a first approximation the estimate just given, corrected for germinal selection, may be taken as the total rate of induction of sex-linked recessive lethals. Assuming that the X chromosome comprises about 5 per cent of the human genome and that its mutability per unit length is the same as that of the rest of the genome, the total rate of recessive mutations over the whole genome is approximately 14 per cent per gamete per generation.

24. The total rate of mutation as estimated above, divided by the rate of mutation per locus per generation, gives an estimate of the size of the genome in terms of number of loci at which detectable mutations may arise. The average rate of recessive mutations per locus per generation is not known but is likely to lie between 2×10^{-5} and 0.2×10^{-5}, indicating that the genome may contain anywhere between 7,000 and 70,000 loci. A figure that has frequently been used—10,000 loci—was derived from *Drosophila* data by considering the ratio between the over-all rate of induction of sex-linked lethals by radiation and the average rate per locus.[17] For purposes of computation, the figure of 20,000 loci in man will be used in the rest of this review.

25. The significance of an over-all mutation rate in terms of individual or collective hardship is very difficult to assess. While sex-linked recessive lethal genes are promptly eliminated in the hemizygous state *in*

utero, fully recessive mutations arising on autosomes are eliminated mainly in homozygotes and these, because of the low levels of inbreeding prevailing in most human populations, are rarely observed. As was mentioned in an earlier section, strictly recessive mutations that occur spontaneously in man have usually a very severe effect in the homozygote.

26. Evidence from *Drosophila*,[18-21] however, suggests that newly arising mutations on the average are not completely recessive but find a varying measure of phenotypic expression in heterozygotes as well and, in fact, range from fully dominant to completely recessive. Their phenotype is usually harmful, and the mutations are thus eliminated at a rate between 1 and 7 per cent per generation. No such figures are available for any other species, and the *Drosophila* estimate can only be applied to man with the greatest caution.

27. The manner in which mutants are eliminated through heterozygotes in man is also unknown. A number of mechanisms[22] can be thought of, such as loss of embryos and foetuses *in utero*, premature deaths and reduction of fecundity due to physical or mental defects of all shades of severity, and that ill-defined but significant entity known as the genetic component of morbidity. At present, it is not possible to determine to what extent any of these mechanisms contributes to the elimination; in particular, it is unknown what fraction of the elimination takes place through losses assessable in socially meaningful terms.

28. On the other hand, the very concept of unconditional harmfulness entailed by the assumption that mutant genes are eliminated in the heterozygous state has been questioned[23] because a number of genes, both in man and in experimental animals, are known to be advantageous in the heterozygous state, at least in some environments, even though they are detrimental or lethal in homozygotes. The existence of such genes is a matter of experimental observation, but it seems unlikely that they represent more than a small fraction of the newly arisen mutants.

29. Since the phenotypic expression of any gene depends on the expression of the other components of the genome, it could also be argued that, with such a large number of mutants as arise spontaneously in each generation, their interactions with the rest of the genome should be taken into account before reaching conclusions regarding how and when they are eliminated. This cannot at present be done with the available experimental data. Until evidence to the contrary accumulates, however, it is reasonable to assume that the net effect can be neglected, as positive and negative interactions presumably tend to cancel each other.

30. If the rate of elimination observed in *Drosophila* is accepted as valid for man—and there is no denying the tentativeness of such an assumption—and if, as a first approximation, the rate of elimination of new mutations is assumed to be between 1 and 7 per cent per generation, the total number of detrimental genes carried, on the average, by human gametes can be estimated to be between two and fourteen ($0.14/0.07 = 2$; $0.14/0.01 = 14$).

31. Some indirect support for this estimate comes from the study of the offspring of consanguineous marriages in man.[24] Consanguinity of the parents increases the likelihood of their offspring being homozygous for a fraction of the genome, including lethal and detrimental genes. The amount of detriment thus observed in the offspring is directly related to the number of detrimental genes carried by the parents on the one hand, and on the degree of consanguinity on the other. As discussed in earlier reports of the Committee, the relationship between detriment and degree of consanguinity, when established on an adequate sample of consanguineous marriages, makes it possible to estimate the number of genes, or rather of lethal equivalents, in the population which, in the homozygous state, give rise to the type of detriment that is being studied. The estimates vary from one sample to another. A recent review[25] of the available data sets three as a likely upper limit to the mean number of lethal equivalents per gamete acting after birth.

32. The estimate based on the three-generation approach refers to the whole of the newly arisen mutational detriment. The estimate derived from consanguinity studies, on the other hand, is a measure of the detriment carried by the population, as expressed in homozygotes after birth. Estimates of the corresponding damage expressed before birth have so far been so erratic[25, 26] as to preclude conclusions regarding the importance of its contribution to the total detriment, although there is reason to believe from results of animal experiments that it may be far from negligible. Considering that, as discussed in the previous paragraph, the damage estimated on the basis of consanguinity data and the damage quite independently assessed through sex-linked lethals are not entirely the same, and taking into account the errors involved in either estimate, the two approaches appear to lead to reasonably consistent results. Too much weight should not be attached to their closeness, however, because the estimates in both cases are dependent, in different ways, on a large number of assumptions.

33. It must also be mentioned that the damage estimated through consanguinity studies may only in part be due to balance between recurrent mutation and selective elimination through heterozygotes. Even if, as was mentioned in paragraph 28, mutations conferring selective advantage to heterozygotes were much rarer in the population than the others, they could contribute more to the detriment expressed in the homozygous state.[27]

34. The method of assessment of genetic detriment through consanguinity studies was originally expected to yield quantitative information on the respective role of either mechanism in maintaining the detriment in human populations. So far, however, the results of human surveys have been inconsistent. Besides, the approach through which the role of the alternative mechanism can be assessed has recently been challenged on the ground that some of the assumptions on which it rests are unjustified.[28] The estimate based on the three-generation approach can only be regarded as applying to mutational damage. Comparison with that obtained from consanguinity studies suggests that the latter may include a large mutational component.

35. Even consanguinity studies do not provide information about the manner in which genes are eliminated at the levels of inbreeding commonly prevailing in human populations. The damage revealed by these studies is, by definition, due to detrimental genes acting in the homozygous state. There is no way at present to guess at what period of life this damage is being expressed, although it seems unlikely that it can be expressed earlier than in homozygotes.

36. This section has shown that estimates of the over-all spontaneous mutation frequency in man may be derived mainly from human sources, but the degree of confidence in these is still limited by deficiencies both of theory and of the empirical data. However, there is merit in making as much use of human information as possible, and the methods themselves are potentially capable of improvement. It may be noted that the only figure obtained in this section which is used in estimating risks is the estimate of the total number of mutable gene loci in the human genome. Although this figure is based on human data alone, the uncertainty of the assumptions that are used in obtaining it must be clearly borne in mind.

CONSTITUTIONAL CHROMOSOME ANOMALIES

Introduction

37. Despite the considerable advances that have been made in recent years in the field of human cytogenetics and that require a revision of the points discussed by the Committee in its 1962 report, it is still too early to achieve a balanced inventory of the chromosomal anomalies prevailing in the human species. This is not only due to the fact that it is often impossible to establish a close correlation between a clinical pattern and the chromosome anomaly that is observed, but also to the fact that it is difficult to identify certain pairs of chromosomes[a] and that still relatively few subjects have been studied. Nevertheless, several specific syndromes can now be characterized by the chromosome anomalies associated with them.

38. It is customary to consider separately autosomal and sex-chromosome anomalies and, within each of these, changes in the number of chromosomes and structural rearrangements. Among the autosomal anomalies, four syndromes are especially frequent. These are trisomy 21 (i.e., Down's syndrome associated with trisomy of one of the two pairs of chromosomes of the G group which is conventionally called No. 21); trisomy 18 (i.e., of a No. 18 chromosome in the E group) and trisomy 13 (i.e., of one of the 13-15 or D group chromosomes. Conventionally the extra chromosome is assumed to be a No. 13). The fourth syndrome is due to deletion of part of the short arm of chromosome 5 (*cri du chat* syndrome).[b]

39. In addition to these four common anomalies, the occurrence of other types of autosomal alterations has been described:[30-34]

(*a*) An extra chromosome (e.g. 21) from which a portion is deleted, so that a completely trisomic state is not present.

(*b*) Deletions, particularly involving the short arm or the long arm of chromosome 18.

(*c*) Isochromosomes.

(*d*) Ring chromosomes.

(*e*) Translocations of two general types:

(i) Those in which essentially all of one chromosome is exchanged to another, with no identifiable reciprocal product. This produces a chromosome number of 45 in the individual, without phenotypic change. This type of translocation always involves one acrocentric chromosome (groups D and G).

(ii) Translocations in which parts of two chromosomes are reciprocally translocated. This does not produce phenotypic changes in the balanced state, but results in severe damage in the unbalanced condition.

(*f*) Mosaicism—a mixture of two cell lines in the same individual. These may include various combinations of monosomic, disomic and trisomic cells (e.g. involving chromosome 21); a mixture of diploid and triploid cells; or a mixture involving any of the types of abnormalities described above.

40. Among sex-chromosome anomalies,[30-32,35-37] three types are particularly frequent, the XO (Turner's syndrome[c]), the XXY (Klinefelter's syndrome[d]) and the XXX[e] complements. The other complements that have been described—XXYY, XYY, XXXY, XXXXY, XXXX, XXXXX—are much rarer. Structural anomalies are also known: ring X (X_R), isochromosome X involving the long or the short arm of the X chromosomes, deletion of the short (X_{DS}) or of the long arm (X_{DL}) of the X chromosome.

41. Several different types of mosaics, associating, for instance, an XO with an XX line or an XY with an XXY line, have been described. Sometimes structural anomalies of the X chromosome are involved, such as XO/XX_R, $XO/X_{iso}X$, etc. (table I). Each type of mosaic determines different phenotypes depending on which line is preponderant. In true hermaphroditism, karyotypes vary from one case to another; both XX and mosaics are known, the latter of the types XX/XXY, XX/XY and XO/XY.

Prevalence of constitutional chromosomal anomalies

Autosomal anomalies

42. *Trisomy 21.* Most autosomal anomalies have been too recently known to make it possible to estimate accurately their individual rates of occurrence. Several investigations, however, have dealt with trisomy 21, the earliest among the anomalies to be defined as a syndrome. The frequency of trisomy 21 seems to be of the same order of magnitude in all areas where it has been estimated,[38, 39] approximately 1.5 per 1,000 live

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[a] The international conferences held at Denver and London[29] defined the characteristics of the normal human karyotype and the relevant terminology. At the London conference it was agreed that chromosomes could be classified according to two systems, alphabetical and numerical, as follows:
1-3 (A), 4-5 (B), 6-12 and X (C), 13-15 (D), 16-18 (E), 19-20 (F), 21-22 and Y (G).

[b] The syndromes due to these autosomal anomalies consist of serious mental retardation associated with a constellation of malformations that makes possible the clinical diagnosis of each syndrome. The most frequent malformations are, schematically:

Trisomy 21: Peculiar facial features with palpebral fissures slanted upwards and outwards, epicanthus, short nose with broad base, thick lips, small and malformed ears, small and spherical skull, hands and feet short and broad, muscular hypotonism.

Trisomy 13: Microcephaly, microphthalmia, harelip, ear and heart malformations, polydactyly, characteristic foot deformity, arrhinencephaly.

Trisomy 18: Ear and skull malformations, abnormal position of hands and fingers, cardiac malformations.

Cri du chat syndrome: roundish face, hypoplasia of the lower jaw, hypertelorism and highly characteristic cry, particularly in the first years of life.

[c] Turner's syndrome: gonadal agenesis, absence of secondary sex-characters, small stature and various malformations in phenotypic females with amoenorrhea.

[d] Klinefelter's syndrome: phenotypic males with high stature, gynecomastia, gonadal hypoplasia and sterility.

[e] The XXX syndrome is a chromosomal rather than a clinical entity that has been observed both in seemingly normal and in feeble-minded women without serious genital disorders.

births. Not all cases of trisomy 21 are regular but the frequency of translocated trisomies 21 is difficult to assess because the samples that have been studied in different laboratories (table II) are usually biased, since karyotype analyses are carried out in most cases in children from mothers under thirty years of age when the chances of detecting a translocation are highest. Based on these selected cases, the frequency of D ~ G and G ~ G translocations is 7-12 per cent,[30, 40, 41] but the most reliable estimates cluster around 2 per cent of all trisomies 21.[42, 43, 44] These translocations are usually of the type D ~ G and G ~ G, other types being exceptional. It should be noted that only in one-third of the families where two or more sibs are affected is trisomy 21 accompanied by a detectable translocation.[45] Translocations involving chromosome 21 have recently been estimated to arise at an approximate rate between 2.1×10^{-5} and 2.7×10^{-5} per chromosome 21 per gamete per generation. The expected frequency of translocated chromosome 21 among live-born children would then be approximately 5.4×10^{-5}.[46]

43. Among these translocations, only a minority is inherited from parents carrying the translocations themselves. Only one in four (3/12) is found when data from several authors[41] are pooled. According to a recent survey,[46] 49 per cent of D ~ 21 translocations and 5.6 per cent only of G ~ 21 translocations are inherited from parents. It can be estimated on the basis of current knowledge that *de novo* translocations in trisomy 21 represent from one-half to three-quarters of all these translocations. The frequency of mosaics of which one cellular line at least is trisomic 21 is low, probably less than 1 per cent,[47] and partial trisomy with deletion of part of a chromosome 21 is exceptional.[48]

44. *Trisomies 18 and 13.* Few statistical data on autosomal trisomies 18 and 13 are available. The first estimates for trisomy 18 give frequencies ranging from 0.2 to 1.6 per 1,000 births.[30, 49-51] For still unknown reasons, a clear predominance of females is apparent—forty-five females as against thirteen males according to a recent survey.[52] Out of sixty-five cases of trisomy 18, sixty regular trisomies have been observed, two translocation trisomies and three mosaics,[52] but the relative proportions of the different chromosomal complements are probably not representative of the actual population frequencies, inasmuch as a bias occurs in the selection of published observations. The same is true for trisomy 13[53] where, out of forty-eight cases, two mosaics, three partial trisomies and three translocation trisomies have been observed. The frequency of trisomy 13 at birth appears to be lower than that of trisomy 18.[51]

45. Cri du chat *syndrome.* No data are yet available on the frequency of this anomaly of which some fifty cases are now known and which consists of deletion of part of the short arm of chromosome 5. Seven cases (including two sibs) have been observed in a population of 1,562 feeble-minded subjects.[54] The frequency of the syndrome is probably higher than that of trisomy 13. The association of the anomaly with a translocation has been described in one case.[55]

46. *Translocations.* It is also difficult to assess the frequency of translocations in the general population, since balanced translocations are phenotypically undetectable and are usually identified through anomalies occurring among the descendants of carrier individuals. Thus, only systematic surveys can give an idea of the frequencies involved. A recent investigation on 438 adults[56, 57] yielded a frequency of five per 1,000 for reciprocal translocations and possibly pericentric inversions, which represent major karyotypic anomalies. Minor variations affecting the size of the short arm of group D and G chromosomes or the location of the centromere of a chromosome 16 are probably fairly frequent (2-3 per cent). It is not proved, however, that those anomalies are associated with any detectable malformation.

Sex-chromosome anomalies

47. The frequencies of sex-chromosome anomalies are better known than those involving autosomes since the number of special stainable bodies (Barr bodies or sex-chromatin) in interphase nuclei makes it possible to know how many X chromosomes are present in a cell. In general, as many bodies are observed in the interphase nucleus as there are X chromosomes less one. Normal male individuals have no sex-chromatin and their cells are called sex-chromatin negative. Cells of normal females have one Barr body which is visible only in a certain percentage (from 20 per cent upwards) of cells called sex-chromatin positive. Cells with three X chromosomes have two Barr bodies and are called double-positive.

48. Sex-chromatin tests therefore make it possible to recognize anomalies involving the number of X chromosomes (XXX, XXY, etc.) and certain mosaics in which normal cells are in sufficient proportion to be detected in a relatively small sample of cells. If the abnormal cell population is a small minority, the mosaic situation will be detected by this means only with great difficulty.

49. In samples from certain selected populations the frequency of sex-chromosome anomalies is higher than in the general population. This is the case with feeble-minded subjects,[36, 58] with sterile subjects,[58] with criminals[59, 60] and with females with stature below normal[61] (table III).

50. A number of surveys have dealt with the frequencies of sex-chromosome anomalies among live-born children. By pooling the data from five of these surveys[62-66] involving altogether over 25,000 children, the following frequencies of subjects with abnormal sex-chromatin are observed: 1.5 per 1,000 females, of whom three-fourths, or 1.2 per 1,000, were double-positive (XXX), 0.3 per 1,000 negative, and 1.7 per 1,000 chromatin-positive males. In eighteen of the 10,725 males who underwent a chromosome examination, twelve were XXY, one was XXXY and five were mosaics XX/XXY.[64] In twelve girls among 10,000 that were investigated, nine proved to be XXX, two were XO, and one was a mosaic XO/X_{DL}X (deletion of part of the long arm). Those figures show the high frequency of mosaics among individuals with sex-chromosome anomalies, a frequency which has been estimated to be about 18 per cent of these anomalies.[36] Finally, Y-chromosome polymorphism is known in man[62] as it is in other organisms, but its significance is not clear.

Conclusions

51. Although more information is needed, it is generally agreed that chromosome anomalies are observed in about ten per 1,000 live new-born infants. Autosomal trisomies and sex-chromosome anomalies

account for about 3.5 per 1,000 each, the rest being accounted for by translocations (table IV). It must be borne in mind, however, that only relatively major anomalies of the karyotype are detectable through present techniques. A sizable number of chromosome anomalies escape cytological diagnosis. On the other hand, attention has recently been drawn[67] to the existence in man of complex structural changes, for example insertions, which can produce genetic imbalance in the children of carriers (duplications, deficiencies). These events do not seem always to be accompanied by detectable morphological differences between the chromosomes of children and those of their parents. Their frequency cannot at present be assessed.

Chromosome anomalies in miscarried foetuses

52. A certain number of spontaneous abortions are associated with chromosome anomalies. Sometimes it is the abnormal chromosome constitution of one of the parents that is responsible[68-71]—mosaics or structural rearrangements, particularly translocations. More frequently, the anomaly concerns the foetus alone—monosomies and trisomies, triploidies—with no apparent anomaly detected in the parents.[72-75] The proportion of chromosome anomalies observed in a number of studies of spontaneous abortions is large: thirty cases out of eighty-two.[76-80] The largest homogeneous survey includes 200 cases.[81,82] Chromosome anomalies were obtained forty-four times: 11 XO, 9 triploids, 7 E-trisomics, 6 D-trisomics, 5 G-trisomics, 2 C-trisomics, 2 tetraploids, 1 B-trisomic and 1 A-trisomic.

53. About one-fourth of all spontaneous abortions therefore appear to be associated with chromosome anomalies, particularly anomalies of the number of chromosomes. The high pre-natal lethality of XO individuals probably explains why the proportion of XO new-born children is much lower than that of XXX or XXY new-born children. Assuming that 15 per cent of all pregnancies result in spontaneous abortions,[83] chromosome anomalies would be responsible for the interruption of about 4 per cent of the pregnancies. The over-all burden of chromosome anomalies would therefore be 5 per cent per generation.

Factors affecting the prevalence of chromosome anomalies in human populations

Modes of transmission

54. Many individuals with constitutional chromosome anomalies have no descendants owing to sterility related to a sex-chromosome anomaly or to some debility associated with an autosomal anomaly. The viability of trisomic subjects, particularly with regard to chromosomes 18 and 13, is severely reduced, so that it must be assumed that the frequency of these anomalies is maintained in the population almost exclusively through recurrent chromosome mutation. Nevertheless, XXX females have been reported to have children; thus, eleven women have had thirty-one children, about half of whom were examined and in whom no anomaly was detected.[35] Recently,[84] two children with Klinefelter's syndrome (XXY) were observed, who were born respectively of an XXX and of an XX/XXX mother, and an XX/XXX girl born of a mother also XX/XXX.[85] This confirms the possibility that sex-chromosome anomalies can be transmitted as has been shown in other organisms, including mice. Among the few children of XXY/XY mosaics, no anomaly related to the parental mosaicism has been observed.[86, 87]

55. Diplo-triplo-21 mosaics have also been observed in six families among the parents of children who were regular trisomics 21. The risk of recurrence of trisomy 21 is all the more difficult to assess in these cases as the respective proportions of the different cell lines are not necessarily the same in the germ line cells as in the somatic cells where they are detected.

56. Another problem regards the transmission of translocations to the descendants of the carriers. The most accurately studied cases are those concerning D ~ 21 translocations. According to certain authors, when the mother carries the anomaly, on the average one-third of her children are normal, one-third carry the balanced translocation and one-third are trisomics 21.[39] Other authors believe that the latter proportion is between one-third and one-tenth.[42] The inconsistency may be due to the different statistical methods employed. When the father is the carrier, on the other hand, the frequency of trisomics 21 among the offspring is much lower, certainly less than 5 per cent,[39] the reason for such a difference in transmission with the sex of the carrier being unknown.

57. In the case of G ~ 21 translocations, the sex of the transmitting parent does not seem to play a role. Therefore 21 ~ 22 translocations will give rise to one-third, one-third, one-third proportions, whereas the offspring of 21 ~ 21 translocations will be trisomics 21 since the lethality of haplo-21 zygotes is probably very high.

58. The frequency of transmission of other types of translocations is less known. A familial observation of a D ~ D translocation showed a 1:1 segregation of the anomaly in the course of three generations.[89] It is not unlikely that the size of the chromosomes involved in the translocation may influence the frequencies of abnormal[90] segregation, but the available data are still insufficient. Interchromosomal effects[91] are an additional cause of aneuploidy among the descendants: the presence of an anomaly, e.g. a translocation, may increase the likelihood of an anomaly of a different type in the offspring; several observations of regular trisomies 21 have been reported in children born of parents carrying translocations of the D ~ D type and sex-chromosome anomalies in children of a parent carrying an autosomal translocation have also been observed.

Mechanisms of induction

59. Still relatively little is known on the mechanisms of induction of chromosome anomalies in man. Anomalies involving the number of chromosomes may be due to non-disjunction or to loss of a chromosome during meiosis. But both loss and non-disjunction may also take place in the zygote.

60. When abnormal segregation or losses of chromosomes occur at the blastomeric stage, they give rise to mosaics consisting of two or more cell lines. The existence of monozygotic twins with different karyotypes argues in favour of such a mechanism.[30] If one of the cells resulting from non-disjunction is unviable (e.g. YO), the complementary cell (XXY) only will give rise to an anomalous homogeneous line.

61. Abnormal gametes may arise at meiosis in translocation carriers. Certain structural anomalies may also take place during meiosis (production of X isochromosomes) but not necessarily so. Other mechanisms have been suggested to account for certain aneuploidies: asynchronous duplication of a single chromosome,[92]

polyspermy or double fertilization, of which several examples have been recently described in the case of XY/XX mosaics.[93-95] The frequency of those accidents, however, is still wholly unknown.

62. In certain cases it is possible to identify the stage at which the accident has occurred. Thus, an $X^M X^P Y$ complement[f] is due to a non-disjunction during the first meiotic division in the male, whereas $X^M X^P X^P$ and XYY are believed to result from non-disjunction at the second division.[35] By means of the markers located on the X chromosome (colour blindness, glucose-6-phosphate-dehydrogenase deficiency, Xg blood group), it is occasionally possible to determine the paternal or maternal origin of an X chromosome. Thus, among the XO subjects studied in one series, the maternal origin of the X chromosome has been ascertained twenty times and its paternal origin once,[96] but because of the difficulty of the technique this cannot be taken as a reliable estimate. Likewise, XXY subjects are known to have arisen as a consequence of non-disjunction occurring during gametogenesis in either parent.[30, 35, 97, 98] Finally, an XXYY subject has been reported to originate from two successive non-disjunctions.[99]

Possible aetiologic factors

63. A certain number of factors are known to induce chromosome anomalies, but their actual effect in man can only be indirectly inferred, since few cytogenetic data on human gametogenesis and the early stages of development of the human embryo are available.

64. *Maternal age* is positively correlated with the occurrence of aneuploids. This is clearly proved in the case of trisomy 21,[38, 39] and is observed also with trisomies 13 and 18 though to a lesser degree,[30] and perhaps in the case of Klinefelter's syndrome (XXY)[100] as well, but apparently not with Turner's syndrome (XO).[101]

65. *Certain viruses* may be responsible for chromosome or chromatid breaks, both *in vivo* and *in vitro*, and also for cases of aneuploidy, sometimes preferentially involving certain chromosome pairs.[102] Attention has been drawn to time fluctuations of certain constitutional chromosome anomalies and to their accumulation in certain limited areas.[38, 103, 104] A correlation has been suggested between an increased frequency of X-chromosome anomalies and of trisomy 21 on the one hand, and an epidemic of rubella in the preceding months on the other, as well as between an increased frequency of trisomy 21 and an outbreak of infectious hepatitis.[105, 106]

66. *Ionizing radiations*, particularly x rays, have been suspected of inducing aneuploidy in man. It does not seem, however, that exposure to radiation is actually recalled more frequently among mothers of trisomics 21 than among mothers of normal children,[107, 108] as had earlier been held,[109] although the results of a recent survey[110] reopen the problem once more. The possible role of parental radiation before conception has again been suggested recently with regard to one case of trisomy 13[111] and in several cases of trisomy 18.[112]

67. Very few cases of chromosome anomalies in human somatic cells have so far been observed after pre-natal irradiation. In a recent observation the anomaly consisted of a mosaic of minute chromosomes, with no apparent structural rearrangements, in a child who had been exposed to radiation in the first weeks of intra-uterine life.[113] Additional studies have also been made in recent years on the effects of both x rays and internally-deposited radio-active isotopes (I^{131}, P^{32}) on chromosomes of human somatic cells. The studies were carried out on blood, skin or aponeurotic cells drawn from subjects exposed to radiation accidentally[114-117] or occupationally,[114, 118-123] or exposed for therapeutic or diagnostic reasons.[113, 114, 121, 124-149]

68. These *in vivo* studies have yielded information on the types of anomalies observed and on their persistence in the organism. Both aneuploidies and polyploidies, frequently due to endo-reduplications, and also structural anomalies have been observed: chromatid and chromosome breaks, acentric fragments, minute chromosomes, di- and polycentric chromosomes, and ring chromosomes. Some of these changes persist for years. Observations have been reported in which the irradiation had taken place more than twenty or thirty years prior to the observation.

69. In other studies, human somatic cells were exposed to radiation *in vitro*.[150-160] *In vitro* irradiation makes it possible to select the quality of the radiation, to estimate more accurately the doses and dose rates received by cultured cells, and to evaluate the number of various types of chromosome rearrangements as a function of the dose. The frequency of deletions has thus been shown to be linearly related to the dose of both x rays and neutrons.[153-157] The frequency of two-hit events (dicentric and ring chromosomes) bears a quadratic relationship with the x-ray dose and a linear relationship with the dose of neutrons (table V).[153, 161, 162]

70. *Conclusion.* The respective role of each of these factors cannot at present be assessed, especially as still other factors that are not mutagenic *stricto sensu* may also play a role. The association of several different chromosome anomalies in certain families or in certain subjects suggests the existence of factors predisposing to these anomalies. An interchromosome effect (paragraph 58) may enter into play in certain cases, while others have been attributed by some authors to the effect of a gene similar to those already known to be responsible for non-disjunction in *Drosophila*. The role of a gene responsible for familial mosaicisms has also been suggested.[85, 163]

III. Experimental data on radio-sensitivity of germ cells *in vivo*

Changes in the number of chromosomes

Changes of ploidy

71. Several attempts have been made to induce changes of entire sets of chromosomes in mammals. Where radiation was used as the inducing agent the attempts have not been successful. Experiments not involving radiation showed that triploid embryos can survive. They were observed at nine and one-half and twelve days gestation in mice and rats, respectively, and at five months of age in human foetuses.[30, 164] In experimental animals, embryos of higher ploidy are eliminated before implantation. If the same phenomenon

[f] X^M and X^P indicate X chromosomes of maternal and paternal origin, respectively.

obtained in man, these embryos would be eliminated during the first week after conception.[g]

72. In contrast to what has been observed in mammalian species, viable triploid animals have been described in *Drosophila*, the parasitic wasp *Habrobracon*,[165] the land isopod *Trichoniscus* and the salamander *Triturus viridescens*.[166] Polyploidy has been observed in the silkworm *Bombyx mori* and the butterfly *Solenobia triquetiella*.[167-172]

Loss or addition of individual chromosomes

73. Animals monosomic for one of the autosomes have rarely been observed. The best known example is that of the fruit fly which can survive the loss of one of the fourth chromosomes, representing a few per cent of the total genome. Mammals monosomic for one of the autosomes have never been observed, and several lines of evidence suggest that monosomics are always lethal before birth, although mosaics may survive.[173]

74. Instances of trisomy have been studied extensively in plants for a quarter of a century.[174] They are known for the fourth chromosome of *Drosophila* and have recently been reported in the mouse by Griffen and Bunker[175] who described three different autosomal trisomics by analysing the male offspring of males, the spermatogonia of which had been irradiated with 350 and 700 R of x rays. Two of the trisomics were sterile and the third semi-sterile. In each case, the extra chromosome was a member of the smaller chromosome classes, and none of the males showed external deviations from the normal phenotype. A phenotypically normal but sterile male trisomic for one of the smallest chromosomes has also been found by Cattanach.[176]

75. Anomalies involving sex-chromosomes are much better known than those involving autosomes. Information on the spontaneous occurrence of the sex-chromosome anomalies in mammals has been reviewed extensively by Russell.[173] The spontaneous occurrence of these anomalies in mice and in *Drosophila* was discussed in the 1962 report (annex C, paragraphs 71 and 72).

76. In experimental animals, the most commonly observed anomalies are of the XO and XXY type, irrespective of whether male or female germ cell stages have been irradiated. XO individuals are observed more frequently than XXY individuals, the reverse being true in man (paragraph 53), because, in addition to non-disjunction, XO individuals can arise through loss of the paternal X or Y chromosome or the maternal X chromosome during germ cell formation as well as during or before the first cleavage of the zygote. By contrast, it is likely that the majority of XXY individuals arise from non-disjunction.

77. Experiments with mice have demonstrated that a considerable increase above the spontaneous frequency of XO and XXY individuals can be obtained by irradiating various germ cell stages as well as early zygotes.[177, 178, 179] Table VI summarizes the data.

78. With regard to the induction of sex-chromosome losses (XO individuals) in mice, the sensitivity is highest in the early zygotes, namely, shortly after sperm entry (completion of second meiotic division) and also in the early pronucleus stage. Sensitivity then becomes relatively low in the mid-pronucleus stage (probably post-DNA synthesis). Male and female germ cells at all stages are much less sensitive than are early zygote stages. Spermatids appear to be the most sensitive germ cells in males.

79. In females, the early prophase stages of the primary oöcyte, which are only present in the ovaries of foetuses and new-born children, are about as sensitive as early spermatocytes. From the point of view of genetic risks, it is the dictyate stage of the oöcyte that is most important. Dictyate oöcytes in various developmental phases are present in the ovaries from shortly after birth until about eight hours before each ovulation. In man, cells at that stage therefore absorb more natural and man-made radiation by several orders of magnitude than any other female cell stage.

80. Oöcytes in the early phases of the dictyate stage are easily killed by exposures as low as 8 R.[180] Genetic effects have therefore not yet been studied for this early stage. Preliminary results on later phases of the dictyate stage (table VI) have shown that the sensitivity of these cells with regard to sex-chromosome losses is on the average of the same order as that of the male germ cell stages.

81. Studies on the induction of sex-chromosome loss in the metaphase of the first meiotic division of the oöcyte are in progress. In view of the results of the induction of dominant lethals (paragraph 108), it is to be expected that radiation-induced sex-chromosome loss in this stage will be high compared to other female germ cell stages.

82. In adult *Drosophila* males, several independent attempts have been made to determine the rate of induction of XO animals.[181-186] The absolute frequencies, however, are dependent upon the age of the males at the time of irradiation and the type of X chromosome carried by the irradiated males. In decreasing order of sensitivity, male germ cells rank as follows: spermatocytes > spermatids > spermatozoa > spermatogonia. Below 1,000 R, the results for spermatocytes are consistent with a linear dose-effect relationship and suggest that the induced rate of XO animals per R is about 2.3×10^{-5}. This figure is remarkably close to that obtained for spermatocytes in mice (table VI).

83. Information on the induction of sex-chromosome loss in spermatogonia of *Drosophila* is scanty. The induced frequencies observed are close to those of the controls. Sensitivity would therefore seem to be much lower than for other stages.

84. With regard to the induction of non-disjunction by irradiation, the peak sensitivity for XO and XXY animals is found in the same stage(s) of spermatogenesis.[181, 186, 187]

85. Experiments with oöcytes in different developmental stages have clearly shown that XO and XXY animals can result from the irradiation of *Drosophila* oöcytes in the prophase of the first meiotic division.[188, 189] The most recent study provides good evidence that the frequency of XO animals increases faster than linearly with dose in the exposure range 500-5,000 R. The dose-effect relationship for the production of XXY animals is more complicated and difficult to interpret.

86. The induced frequency of occurrence of X^M (maternal X) loss in *Drosophila* is 0.58×10^{-5} per roentgen (calculated by taking account of the fact that YO is lethal) at an exposure of 500 R.

[g]

	Pre-implantation period in days	*Major organogenesis period in days*	*Foetal period in days*
Mouse	0-5	6-13	14-20
Man	0-8	9-56	57-270

87. The evidence presented in the preceding paragraphs demonstrates that loss of sex-chromosomes (and most probably of other chromosomes as well) and non-disjunction of these chromosomes can be induced by ionizing radiation in experimental animals. There are no reasons to doubt that chromosome changes of this type can also be induced by radiation in the germ cells of man. In fact, there is already some evidence that this is the case.[110] It is perhaps not coincidental that a similar frequency of induction of sex-chromosome loss is observed in the spermatocytes of *Drosophila* and of the mouse. Results for dictyate oöcytes of the mouse are of the same order of magnitude. Information on the induction of sex-chromosome loss and non-disjunction in dictyate stage oöcytes and spermatogonia is still far from complete. In view of the results obtained with *Drosophila,* one can surmise that the rate of induction of sex-chromosome losses in the spermatogonia of mice and man may also be much lower than the corresponding frequencies obtaining at other stages of spermatogenesis. Special emphasis needs to be given to the fact that the early zygote stages in mice have proved to be extremely sensitive to the induction of chromosome loss by irradiation.

Dominant lethals

Introduction

88. For the purpose of this section "dominant lethals" are defined as those genetic changes, irrespective of their exact nature, that cause embryonic death and early post-natal death in heterozygotes. However, the term has more specifically come to be applied to those embryonic deaths that result from point mutations or chromosome breaks in parental germ cells. All induced changes that affect the germ cells themselves or render the gametes incapable of participating in fertilization are excluded from the dominant lethal category.[104]

89. There should be a clear distinction between dominant lethals arising in mature gametes as compared to those in immature germ cells. In post-meiotic male germ cells, the frequency of dominant lethals rises linearly with dose at low x-ray doses. The damage primarily results from chromosome losses due to single chromosome breaks. If the broken ends of chromosomes remain unrestituted and form acentric fragments and dicentric chromosomes during the development of the zygote, they cause death. At higher doses of irradiation, two-break events lead to structural chromosome anomalies which may also result in zygotic death. However, the main contribution to zygotic mortality is probably from single chromosome breaks.

90. In pre-meiotic male germ cells, most, if not all, of the chromosome breaks which yield acentric and dicentric chromosome combinations will be expected to lead to death of the descendant cells and therefore to be selectively eliminated prior to becoming mature germ cells. On the other hand, most dominant lethals induced by treatment of pre-meiotic cells are the consequence of multiple (two or more) chromosome breaks leading to simple translocations, inversions, etc. In their original form, such changes frequently do not kill either the germ cells carrying them or the resultant zygotes. However, as a result of events at meiosis, they may give rise to genetically unbalanced gametes still capable of fertilization but eventually leading to unviable zygotes.

91. In the mouse, two methods are available to estimate the dominant lethality induced by radiation. The first method measures changes in litter-size of irradiated animals. The second method takes into account the components of pre-natal mortality as observed by dissecting pregnant females about twelve to eighteen days after mating, and counting the number of *corpora lutea* and of dead and living implanted embryos, thus making it possible to estimate the proportion of pre-natal deaths that occur before or after implantation. Not all pre-implantation losses can be attributed to zygotic deaths. They can also be due to (*a*) lack of fertilization; (*b*) lack of fertility of the egg. Similarly, not all zygotic deaths result from dominant lethal action. They may also be due to maternal factors, such as failure of the uterus to respond to the appropriate endocrine stimulus, thus precluding implantation of fertilized eggs.

92. Both methods have been in use since 1932. Litter-size, however, has proved to be an extremely variable character, so that more reliance is now placed on estimates based on pre-natal death data. Litter-size can be affected by many factors, such as the number of implanted embryos in the uterus and the lactation stimulus which in turn is also dependent on the number of sibs in a litter. In experiments with highly inbred animals, litter-size data can be misleading, because the death of an embryo may increase the chances of survival of the remaining embryos by reducing intra-uterine competition.

Male germ cells

93. *Spermatogonia.* As early as 1956, it was found that the irradiation of mouse spermatogonia led to a significant decrease in mean litter-size as measured three weeks after birth.[190, 191] These reductions were observed following acute exposures of 600 and 1,000 R. Even after a single exposure of 300 R, a reduction in litter-size of about 3-4 per cent was found.

94. In more recent years, most data have been obtained through the pre-natal death method. There is, however, no common opinion as to how the measurements of pre-natal death should be used to estimate dominant lethal damage. Therefore in this review, comparing results obtained by different authors[192-197] made it necessary to recalculate their data, as the figures given in their tables were such that comparisons could otherwise only be made for post-implantation deaths. Table VII gives the results of the recalculations and shows that the total rate of induction of dominant lethals increases significantly when spermatogonia are irradiated with acute x-ray exposures of 300 R and higher.

95. The data obtained by the various authors are conflicting with respect to the question whether pre-natal death occurs predominantly before or after implantation.

96. There is evidence that the induction of dominant lethals is dose-rate dependent.[194, 198] Results presented in table VII show that irradiation at 600 R + 600 R acute x rays increases the frequency of dominant lethals significantly,[198] whereas a single dose of chronic radiation does not.[194]

97. With regard to the question of the persistence of irradiation-induced dominant lethal damage in spermatogonia, the results obtained by Sheridan[196] are of particular importance. His data showed that the frequency of dominant lethals, as measured by the proportion of dead implants among total implants, remained constant over a period of at least twenty-four weeks. These results indicate that dominant lethals

once induced in primary spermatogonia may be indefinitely transmitted to the more mature germ cells that originate from these spermatogonia.

98. It is not unlikely that the dominant lethality which is observed over the twenty-four-week period can at least partly be accounted for by translocations induced in primary spermatogonia. Translocations will be dealt with in the section on translocations (paragraphs 114-129).

99. *Other germ cell stages.* The most recent information on sensitivity with regard to the induction of dominant lethals in the male germ cells of mice at other stages is presented in table VIII. Older data have recently been reviewed.[164] In interpreting the data in tables VII and VIII, it must be borne in mind that part of the spermatocytes and spermatogonia could have been killed almost instantaneously by the x-ray doses that were employed, thus leading to sperm depletion. As a consequence, an unknown fraction of the non-implanted eggs may be accounted for by lack of fertilization rather than by the induction of dominant lethals.[164] It is therefore more appropriate to base estimates of dominant lethal damage on post-implantation data.

100. The post-implantation data in table VIII show that spermatids are the most sensitive cells. Spermatozoa in the vas deferens and in the epididymis are more sensitive than those in the testis tubules, whereas part of the spermatocytes are probably as sensitive as spermatozoa, others being less sensitive. Finally, spermatogonia appear to be somewhat less sensitive than spermatocytes.

101. The changes in sensitivity in maturing male germ cells of *Drosophila* are very similar to those observed in mice.[199-205] Dominant lethal induction is highest in spermatids and spermatocytes. Spermatozoa are roughly half as sensitive as spermatids, and, finally, spermatogonia are about half as sensitive as spermatozoa. The pattern of response of male germ cells of the silkworm to the induction of dominant lethals, although less well known, follows essentially the same pattern as observed with *Drosophila*.[206] Studies on various species of insects have provided a powerful tool in programmes to control insect pests by means of radiation-induced sterilization.[207]

102. Induction of dominant lethals in post-meiotic cells has not only been studied in the mouse, but also in guinea pigs, rats and rabbits. The data, summarized in table IX, indicate the variability in sensitivity between mature spermatozoa of different species. It was calculated that the LD_{50}'s due to induced dominant lethals in these species are 730, 430, 380 and 305 R, respectively.[208, 209] However, general conclusions on the relative sensitivity of these species cannot be made since it varies with dose.

103. Dominant lethal induction has also been studied in swine.[210-214] Since in these experiments only the effects on spermatogonia have been studied, these data cannot be compared with those of the species mentioned in the preceding paragraphs.

104. The objective of the studies with swine was to measure the effects of paternal irradiation (300 R x rays) on the first generation offspring. Changes in litter-size at birth, mortality between birth and weaning, and increase of weight within the first 152 days of life were some of the criteria which were used to detect the genetic effects. In these experiments, two different breeds of pigs were used—Duroc and Hampshire. Thus far, 2,315 litters have been examined. In the Duroc breed, radiation caused a slightly significant increase (4.2 per cent) in the litter-size at birth and a rise in the sex-ratio. On the other hand, irradiated Hampshire males sired smaller litters than their unexposed brotheers though the difference was not significant. At present the discrepancy between the two breeds cannot be adequately explained.

105. The mortality of new-born pigs was analyssed during three periods (0-1 day; 2-6 days; 7-42 days) between birth and weaning. In each of these periods the mortality rate in Duroc pigs was higher in the irradiated than in the non-irradiated series. The irradiated group of the Hampshire breed showed higher mortality only in the first period. In the Duroc breed, total mortality at forty-two days was significantly higher ($P < 0.05$) in the irradiated than in the non-irradiated series. The total mortality rate in the control and irradiated series of the Hampshire breed was not significantly different.

106. In general, mortality was greater in large litters than in small ones. Furthermore, male pigs had higher mortality rates than females, the relationship between sex and mortality being more pronounced in the Duroc than in the Hampshire breed. All data taken together indicate that the results obtained with the Duroc breed are inconsistent with those obtained with the Hampshire breed and make it premature to generalize on the consequences of irradiation of sires in swine.

Female germ cells

107. Information on dominant lethal induction in the different stages of oögenesis in mice and other species is not as extensive as that for spermatogenesis. The earlier data on various mammalian species have been extensively reviewed[164] and show that stages which possibly correspond to the diakinesis stage or to the metaphase stage of the first meiotic division are much more sensitive than those between the early dictyate and the diakinesis stages. In more recent years, the relative sensitivity of mouse oöcytes at different stages has been studied by using the technique of induced ovulation, by means of which it is possible to irradiate oöcytes at different stages throughout the meiotic divisions.[215]

108. The results of these studies are summarized in table X and show that the total rate of induction of dominant lethals reaches its largest values in oöcytes irradiated during the metaphase of the first meiotic division. Anaphase I and metaphase II are less sensitive than metaphase I oöcytes, but still considerably more sensitive than the dictyate and pro-nucleus stages. The same pattern of sensitivity can be demonstrated for induced post-implantation death. For induced pre-implantation death, as defined in table VII, no clear sensitivity pattern can be observed. In practically all stages, pre-natal death occurs predominantly after implantation; in particular, this is evident in the series in which the females had been exposed to 200 R.

109. The data obtained with oöcytes lend themselves to a comparison with those obtained in male germ cells. The best possible comparison is that between the data of Bateman[195] (table VIII) and those of Edwards and Searle[215] (table X), since both experiments were performed at the same exposure of 200 R. Although it is realized that these experiments were carried out by different authors, and that the total rates of induction of dominant lethals can vary because of differences in experimental technique, it seems justified to conclude that metaphase I oöcytes seem decidedly

more sensitive than spermatids. From a comparison between oöcytes in the dictyate stage and spermatogonia, it may be presumed that oöcytes in the dictyate stage are more sensitive than spermatogonia by a factor of ten to twenty. However, this has only been observed at 200 R. At other doses this factor may be completely different, since sensitivity to cell killing and to the induction of genetic damage can vary widely among cells of the spermatogonial population and among cells in the dictyate stage.

110. In insect species, as in mice, it has repeatedly been observed that oöcytes in metaphase I are much more sensitive to dominant lethal induction than during prophase stages (*Drosophila*,[216-218] *Habrobracon*,[219] *Cochliomyia hominivora*[220]). Similarly, studies with the silkworm (*Bombyx mori*) have shown that the frequency of dominant lethals observed after irradiation of oöcytes immediately before metaphase is twenty times higher than the frequency observed after irradiation of oögonia when only lethals which act in the embryonic stage are taken into account. Most lethals induced in the oöcytes were found to be eliminated during embryonic and larval stages, whereas death caused by lethals induced in oögonia was evenly spread over all developmental stages.[221]

Summary and conclusions

111. With regard to dominant lethal induction, it is clear that the sensitivity pattern for the various stages of gametogenesis is strikingly similar in widely different species. In all species studied, the highest frequencies of dominant lethals are observed in spermatids and the lowest frequencies in spermatogonia. Among female germ cell stages, the highest frequencies are found in metaphase oöcytes in the first meiotic division, whereas the lowest frequencies are encountered in dictyate stage oöcytes of mammals and in oögonia of insects. Induced frequencies of dominant lethals in spermatogonia vary widely among species, even within mammals.

112. Experiments with mice, rats and guinea pigs have shown that death due to dominant lethality occurs mainly at about the time of implantation or shortly thereafter. In rabbits, however, dominant lethals act predominantly before implantation of the embryo. Predictions concerning the time of action of dominant lethals in man are difficult to make on the basis of results obtained with other mammals. This is so because man is monotocous, whereas the other mammals are polytocous.[h] Furthermore, there exist differences in nidation and placentation between man and primates on the one hand, and other mammalian species on the other.

113. Results of studies with mice indicated that dominant lethals induced in primary (predefinitive) spermatogonia of the mouse can be transmitted to the immediate offspring of the carriers of these spermatogonia for over a period of at least twenty weeks and possibly much longer.

Translocations

Male germ cells

114. In mice, the presence of translocations is usually ascertained by analysing the F_1 progeny of treated and control animals for heritable semi-sterility. In practice, semi-sterile animals are recognized as those animals of the F_1 progeny which have considerably less than the normal number of F_2 offspring. In addition to the criterion of semi-sterility, most authors confirm the presence of translocations by examining cytologically pre-meiotic and meiotic germ cells of presumed translocation carriers. Table XI surveys the data presently available on translocation induction in spermatogonia of mice.

115. It is highly probable that translocation induction in spermatogonia is an important cause of dominant lethality in zygotes descended from those cells, since in the mouse each spermatocyte (division product of spermatogonia) heterozygous for a translocation will produce one gamete carrying the translocation, one gamete with the normal chromosome complement and two unbalanced gametes carrying duplications and deficiencies. Unbalanced gametes, upon fertilization, give rise to zygotes which usually die at about the time of implantation. However, some zygotes die between implantation and birth, while a few apparently survive to maturity.[222]

116. Experiment 1 in the table shows that irradiation of spermatogonia with two acute exposures of 600 R each results in a significant increase in the percentage of translocations in both female and male F_1 progeny. The pooled data from experiment 1 show that about 4 per cent of mature offspring of the irradiated males carry induced reciprocal translocations. From this percentage it was calculated that the frequency of translocation heterozygotes among spermatogonia was 14.8 per cent.[223] Since radiation was given in two fractions to prevent substantial killing of spermatogonia, the translocation frequency might have been different if the total had been given in one single exposure instead of two.

117. The experiment mentioned above was not only performed to obtain an estimate of the rate of induction of translocations but also to provide information on the total rate of induction of dominant lethals. By studying data both on translocation induction and on dominant lethal induction, it is possible to estimate that about 67 per cent of the total amount of dominant lethality (total amount of pre-natal death) in the 1,200 R experiment can be attributed to translocations, whereas the remaining dominant lethality is thought to be caused by "primary dominant lethal mutations".[198] The situation is different when the haploid post-meiotic germ cells are irradiated. In that case, the contribution of translocations to dominant lethality is much smaller, because in these cells translocations of the aneucentric[i] type will cause death of the zygotes, whereas eucentric translocations will be tolerated.

118. Experiments 1 and 2 in table XI are the only ones that provide information on the effect of dose rate on translocation induction in spermatogonia of mice. As expected, the dose-rate effect is very pronounced[194, 198] (see also paragraph 124).

119. Experiment 3 provides information on translocation induction in spermatogonia following irradiation with a single acute exposure of 700 R.[224] The frequency of translocations in this experiment is significantly lower than the frequency in experiment 1, even when the difference in dose is taken into account. Differences in the experimental procedure and in the strains used are mentioned as the possible factors which might account for the observed discrepancy.

[h] Monotocous and polytocous mammals produce respectively one and many young at a birth.

[i] Aneucentric translocations involve the centromere so that there results an acentric and a dicentric chromosome.

120. Results obtained from experiment 6 do not provide evidence in favour of or against the validity of the estimates derived from experiments 1 and 3. The percentages of semi-sterility obtained at the three different doses in experiment 6 do not differ significantly; neither do they differ significantly from the percentage of semi-sterility (4.14) observed in experiment 1. The data of experiment 6, however, must be interpreted with some care, because only an unknown percentage of the semi-sterile F_1 individuals has been analysed cytologically. Since it can be deduced from experiment 6 that not all semi-sterile F_1 animals had cytologically detectable translocations, it might well be that the frequencies obtained from this experiment lead to over-estimates of the rate of induction of translocations by a factor which is unknown and that probably varies with dose.

121. Experiments 4, 5 and 7 in table XI were not primarily designed to detect translocations.[106, 107, 225] The results of these experiments seem to be consistent with those of experiment 1, but accurate estimates of the rate of induction of translocations cannot be obtained from them because of the fairly small number of animals involved and because cytological analyses were not performed.

122. The experiments summarized in the previous paragraphs have shown conclusively that translocations can be induced in pre-meiotic male germ cells. The rate of induction being low, as had already been noted in the 1962 report on the basis of much more limited information, translocations have been recovered only at high doses and dose rates. It must be pointed out that the rates of induction thus obtained refer to those translocations alone that are recoverable through semi-sterility. This procedure may lead to an underestimate of the total rate of induction of translocations.

123. Older data on translocation induction in other stages of spermatogenesis have been reviewed by L. B. Russell.[164] These data, together with the more recent data of Griffen,[226] show that post-meiotic cells are much more sensitive than pre-meiotic cells, and that among the post-meiotic stages the spermatid stage is the most sensitive one. Pooling the data reviewed by Russell and those of Griffen indicates that approximately 25 per cent of the offspring are semi-sterile when post-meiotic cells are irradiated with a dose of about 700 R. It thus seems that post-meiotic cells are about six times as sensitive to the induction of translocation as spermatogonia.[223] On the basis of theoretical considerations, Auerbach and Slizynska[227] conclude that post-meiotic cells are twelve times more sensitive than spermatogonia.

124. Besides genetic studies, cytological studies also have been performed on the induction of translocations. Arsenieva and Bochkara[228] discovered that, when primary spermatocytes are irradiated with 50 R or less, both balanced and unbalanced translocations occur twice as frequently in monkeys (*Macaca mulatta*) as in mice. Other cytological studies provided information on the effect of dose rate on translocation induction in spermatogonia of mice which were exposed to 600 R of x or gamma rays.[292] Frequencies of multivalent configurations (indicative of heterozygosity for reciprocal translocations) in spermatocytes at dose rates of 913, 89 and 9.7 R per minute did not differ significantly, all being 12-13 per cent. At lower dose rates, however, frequencies were much lower, being about 5 per cent at 0.86 R per minute, and falling to only 1.5 per cent at 0.02 R per minute. It therefore seems that, with regard to translocation induction in type A spermatogonia at the dose level of 600 R, high dose-rate irradiation is about eight times as effective as low dose-rate irradiation.

125. The application of the cytological technique has also revealed a marked discrepancy between the translocation frequency in spermatogonia as found in the genetic experiment (600 R + 600 R, paragraph 116), and that observed cytologically in spermatocytes.[229, 290-292] The observed frequency of translocations in the genetic experiment is about half that observed cytologically. It is thought that the observed discrepancy does not result from a failure of translocation heterozygotes to show semi-sterility, but rather from a selective process between meiotic metaphase and fertilization.

126. As far as the genetic experiments are concerned, it is remarkable that the sensitivity pattern for translocation induction in the different stages of spermatogenesis in mice is very similar to that observed in *Drosophila*. Information on the latter species is, however, much more detailed.[205, 230-233] *Drosophila* spermatogonia appear to be less sensitive to translocation induction than mice. For the F_1 male progeny, the frequency of induction of autosomal translocations is around 0.5 per cent (3,280 tested gametes).[231] No translocations were found in an experiment by Oster (2,000 R, 813 tested gametes)[230] and only one in the experiment of McCarthy and Nafei (400 R, 901 tested gametes).[232]

Female germ cells

127. Information on translocation induction in oöcytes of the mouse is very scanty. Irradiation of late oöcytes with 400 R acute x rays, resulted in a frequency of induced heritable partial sterility of about 2 per cent.[164] This figure is not far removed from the figure of 4 per cent semi-sterility which has been obtained for spermatogonia irradiated with 600 R + 600 R (paragraph 116).

128. The observations on the induction of translocations in female germ cells of mice confirm the findings in *Drosophila*, where it has also been found that the frequency of radiation-induced reciprocal translocations in female germ cells is very low.[234-236]

129. The preceding paragraphs on the induction of translocations in germ cells of experimental animals have shown that these anomalies are induced in both male and female germ cells. The studies indicate that the frequency of radiation-induced translocations is higher in spermatogonia than in dictyate oöcytes. At present, only results obtained with spermatogonial irradiation can be used to estimate the rate of induction of translocations in man.

Point mutations

Specific locus mutations

130. In its 1962 report, the Committee reviewed extensive data on the induction of recessive mutations at seven specific loci in the mouse. Since then, more data have become available. Both old and new data are listed in table XII. They only refer to specific locus mutations induced in spermatogonia and oöcytes. Ignoring variability between loci, the spontaneous mutation rate at the seven specific loci in male germ cells lies between 0.46×10^{-5} and 1.0×10^{-5} mutations per locus per generation (95 per cent confidence limits).

131. In spermatogonia the rate of induction of specific locus mutations rises linearly with acute exposures

up to 600 R. At higher doses, the yield of mutations falls off. This has been attributed to "selective elimination at high doses of the more mutationally sensitive cells in the spermatogonial population".[237] In spermatogonia, the rate of induction is about 2.2×10^{-7} mutations per locus per roentgen. Owing to the sampling variability of the data and to variations in mutability between loci, the rate of induction may lie anywhere between 0.75×10^{-7} and 5.5×10^{-7} (95 per cent confidence limits). These data were obtained from the mutation rates observed after exposure of spermatogonia to 600 R of acute x rays.

132. Recently, results have been published of a study on mutation rates at a set of six specific loci in the mouse.[238] Only one locus in the new set is common to both sets. So far, only three mutations have been recovered in the new stock, yielding a rate of induction of about 0.5×10^{-7} mutations per locus per roentgen. Because of the small numbers involved, this estimate is affected by a large error. As it stands, the estimate lies below the mean of 2.2×10^{-7} obtained with the seven specific loci stock, but two of those loci had even lower rates.

133. A joint estimate is difficult to obtain, but the data taken together suggest that it may be around 1×10^{-7} mutations per locus per roentgen, with presumable confidence limits ($P = 0.95$) one order of magnitude apart.

134. The spontaneous mutation rate in female germ cells is not well known. With one mutation observed in 98,828 offspring, the confidence limits are very wide. Data on the induction of mutations at seven specific loci are also much scantier in mouse oöcytes than in spermatogonia. The data from high doses of acute x rays lead to an estimate of 4.8×10^{-7} mutations per locus per roentgen with 95 per cent confidence limits 2.5×10^{-7} and 7.7×10^{-7}, suggesting that acute irradiation of oöcytes might yield more mutations than irradiation of spermatogonia. Oöcyte data have not yet been obtained for the new set of six specific loci.

135. The results of experiments on the induction of recessive mutations at specific loci in the mouse and in other species are summarized in table XIII. It is apparent that *Drosophila* stands out as the species least susceptible to mutation induction by radiation, even though the comparison of species can be based only on very small samples of loci.

Over-all rates of mutation

Recessive lethal mutations

136. *Drosophila.* Abrahamson exposed spermatogonia of *Drosophila* males to 8,500 R gamma rays from a caesium source and analysed the induction of second and X-chromosome lethals in these cells.[16] The rate of induction of recessive lethals in the second chromosome turned out to be 12.01 per cent, whereas in the X chromosome it was 2.17 per cent. Since the number of mutations in the very small fourth chromosome is negligible and the third chromosome contains as much genetic material as the second chromosome, it can be estimated that the over-all rate of induction of recessive lethals in the whole genome is equal to 26.19 per cent at an exposure of 8,550 R. From this it follows that the over-all rate of recessive lethals per gamete per roentgen is 3.1×10^{-5}.

137. In Abrahamson's data, the ratio of second chromosome lethals to X-chromosome lethals is 5.5. The second chromosome contains about twice as much genetic material as the X chromosome; therefore it would be expected that the ratio between second and X-chromosome lethals would be roughly 2 instead of 5.5. That the ratio is 5.5 and not about 2 is explained by germinal selection, whereby about half the lethal-carrying spermatogonia are killed by the action of these lethals on the cell's own metabolism. Germinal selection does not occur in post-meiotic cells, because in these cells the ratio of second to X-chromosome lethals is in accordance with what one would expect on the basis of the length of the respective chromosomes.[14-16, 239]

138. In experiments of McSheehy,[240] spermatogonia of larvae or adult males received gamma ray doses in the range 200-800 rads. The dose-effect curve for second chromosome lethals proved to be linear. Taking into account germinal selection and the relative length of the chromosomes, it is possible to derive from McSheehy's data an estimate of the over-all rate of induction of recessive lethals per gamete per rad that is equal to 2.9×10^{-5}. This value is close to that obtained by Abrahamson. The induction of second chromosome lethals in spermatogonia has also been studied by Ytterborn.[241] The over-all frequency of recessive lethals in his experiment is 3.4×10^{-5} per gamete per roentgen. Because of the absence of data for non-irradiated controls, it is not possible to estimate the over-all rate of induction of recessive lethals. The over-all rates obtained by Abrahamson and McSheehy are consistent with the rate of induction of recessive visible mutation at specific loci in the same species.

139. *The mouse.* In 1959, a first attempt was made to calculate the rate of induction of autosomal recessive lethals in mouse spermatogonia by following a method proposed by Haldane.[242] It was found that the efficiency of the method was rather disappointing.[243] The same method has also been used by other authors.[244, 245] The results, however, were inconclusive. In 1961, another method was used to estimate the frequency of recessive lethals per gamete.[193] This attempt was also unsuccessful because, among other things, the presence of inbreeding depression made a proper evaluation of the amount of induced recessive lethals impossible.

140. In an experiment by Lyon *et al.*[198] inbreeding was avoided, and, furthermore, the scale of the experiment as well as the doses were increased. Hybrid F_1 males were given two exposures of 600 R high intensity x rays eight weeks apart. Twelve weeks after the application of the second dose, the males were outcrossed to females from a different stock. F_1 males free of translocations and of other factors likely to interfere with tests for recessive lethality were mated to females derived from a stock different from that used for F_1 females. F_2 females were then backcrossed to F_1 males. The offspring from father-daughter matings was used to analyse the presence of recessive lethals (and visibles) which had originally been induced in the spermatogonia of P_1 males. A control experiment was also carried out in which the males were not irradiated.

141. Evidence on the rate of induction of recessive lethals was obtained from three different sets of data: (*a*) embryonic lethality at fourteen days gestation in first litters of F_2 daughters backcrossed to their sires; (*b*) litter-size at birth and weaning of litters from F_2 daughters allowed to produce three litters; (*c*) fourth litter dissection data of daughters having already produced three litters.

142. The final estimate of the radiation-induced recessive lethality was derived exclusively from the first

and third sets of data. These data showed that embryonic survival in the irradiated series was 96.8 per cent of that in the controls. From this figure, it could be estimated that the rate of induction of recessive lethal mutations was about 29.5×10^{-2} per gamete, or 2.46×10^{-4} per gamete per roentgen. The estimate has a large standard error and only makes it possible to establish an upper 95 per cent confidence limit of 4.3×10^{-4} mutations per gamete per roentgen.

143. A different experimental procedure to estimate the rate of induction of recessive lethals per genome was followed by Lüning.[246] Lüning exposed males of a mouse population to 276 R acute x rays in each of seven consecutive generations and prevented inbreeding by starting every generation with a sufficient number of different pairs. A similar experiment was performed in which the males were not irradiated. The presence of recessive lethals in the third and following generations was analysed by making sib matings in each generation. Any induced recessive lethal was thus expected to become manifest in the offspring of those matings. The analysis was similar to that made by Lyon *et al.*[198] on the progeny of father-daughter matings, information being obtained on embryonic death and on litter-size at birth and at weaning.

144. As in Lyon's experiments, data on embryonic deaths were regarded to be the most reliable. They were obtained from the offspring of the fourth to eighth generation of sib matings. The data were then pooled, making allowance for the different doses of radiation that individual generations had received. To evaluate what percentage of deaths among the offspring of sib matings between control and irradiated individuals could be attributed to dominant mutations, Lüning performed an additional experiment using non-sib crosses in the irradiated and control series. The ratio of embryonic death among the offspring of irradiated sibs and irradiated non-sib crosses, once corrected for the rate of spontaneous recessive lethals, indicated that the embryonic survival in the irradiated population was 98.9 per cent of that in a standard population. Such a survival ratio leads to an estimate of the rate of induction of $0.8\text{-}2.0 \times 10^{-4}$ per roentgen per gamete, a figure remarkably close to that obtained by Lyon *et al.* Here also the error is large, but the upper limit to the rate is 6.4×10^{-4} mutations per gamete per roentgen.

145. Despite their large error, the observed rates of induction of recessive lethals per gamete are, at first sight, apparently lower than might be expected from the estimated average rate of induction at the seven specific loci most extensively studied, if it is assumed that the number of mutable loci in the mouse is the same as information in man and *Drosophila* leads us to expect. However, allowance must be made for only 75 per cent of the mutations induced at the seven loci being lethal. Furthermore, it must be noted that radiation-induced lethality was scored *in utero* in the studies of over-all induction, whereas in the investigation at the seven loci the scoring included lethals acting perinatally and post-natally. Allowing for these two factors alone narrows the gap between the two sets of estimates to the extent that, rather than being inconsistent, they may support each other.

146. If the recent data on five additional loci are taken into account as was done in paragraph 133, where an estimate per locus per roentgen of 1×10^{-7} mutations was suggested, the apparent discrepancy between over-all rates and rates per locus is reduced even further.

Recessive visible mutations

147. The over-all spontaneous rate of mutation to recessive visibles in mice has been estimated to be 7.04×10^{-3} per generation. The confidence limits of this figure are very wide since only one mutation has been observed in 142 tested gametes.[198] The over-all rate of induction of recessive visibles in the same experiment was estimated to be 1.8×10^{-5} mutations per gamete per roentgen. At first sight, such an estimate seems inconsistent with the rates observed at specific loci and with the presumable size of the mouse genome, even when account is taken of the fact that only about 25 per cent of the induced mutations at specific loci are viable. It must be observed, however, that the results of comparisons of the over-all frequency of visible mutations with the frequency of mutations at specific loci are difficult to assess, since ascertainment is practically complete in the case of specific loci and incomplete to an unknown extent in the other.

Dominant visible mutations

148. The over-all spontaneous rate of mutation to dominant visibles in mice appears to be 1.7×10^{-5} per generation with 95 per cent confidence limits of 2×10^{-6} and 6×10^{-5}.[247] The estimate is lower than the spontaneous rate observed in man, but it would be unwise to draw conclusions regarding the mutability of the two species with respect to dominant visibles, as too many traits that would require intensive investigation in the mouse are easily detected in man.

149. The over-all rate of induction of dominant visibles by acute radiation can be estimated on the basis of one experiment to be 4.6×10^{-7} mutations per gamete per roentgen.[198] The over-all rate of induction of recessive visibles as estimated by the same observers (1.8×10^{-5}) appears to be considerably higher, the ratio between the two rates being about 40. Such a ratio is based on few mutations only, but it is highly significant, with approximately 95 per cent confidence limits of 7 and 250.

150. Two extreme hypotheses may be advanced to account for that ratio. According to one hypothesis, the ratio would indicate that the genome is made up of two groups of loci, a smaller one where only dominant mutations arise and a larger one where only recessive mutations arise, and that the average rates of induction per locus are the same in the two groups. Alternatively, one could assume that most or all of the loci can mutate both to dominants and to recessives though at different rates, the rates of induction of dominants being forty times lower than those for recessives. The true explanation probably lies somewhere between these two extreme hypotheses, but both theory and empirical data are inadequate to provide a satisfactory interpretation of the results. The ratio between induced recessive and dominant mutations in *Drosophila* is known to be about 5,[248] a value significantly lower than in the mouse. A larger proportion of the induced mutations appears therefore to be recessive in the mouse than is known in *Drosophila*. The reason for the difference between the two species is not known.

151. In the past few years, Ehling[249-252] has shown that irradiation of pre- and post-meiotic male germ cells of mice can lead to the induction of mutations that give rise to skeletal abnormalities. Since these mutations become manifest in the first generation offspring of the irradiated male parents, it is thought that they represent a type of dominant visible mutation.

152. A rather large number of skeletal malformations were observed, and it was considered quite likely that only a small proportion of the abnormalities were due to freshly induced mutations. To distinguish between the possible causes of abnormality, the malformations were divided according to whether they occurred only once in particular experiments (class 1 abnormality) or more frequently (class 2 abnormality). Most of the class 1 abnormalities were considered to be due to dominant point mutations. In view of what is generally known about mutation frequencies at specific loci, it was thought that under the conditions of these experiments a specific dominant mutation would have been likely to occur at most once in the testing of 3,000 to 4,000 gametes.

153. In experiments in which pre- and post-meiotic germs cells were irradiated with 600 R acute exposure, Ehling observed a significant increase of class 1 abnormalities (scored at four weeks of age). The excess over control of class 1 abnormalities per gamete per roentgen was 3.5×10^{-5} for post-meiotic stages and 2.3×10^{-5} for pre-meiotic stages.

154. Excluding the portion of class 1 abnormalities that may not be mutational in origin, the frequency of dominant mutations affecting the skeleton after single exposure with 600 R is 2.9×10^{-5} mutations per gamete per roentgen for post-meiotic germ cells and 1.1×10^{-5} mutations per gamete per roentgen for pre-meiotic germ cells. Additional data were obtained from two experiments in which spermatogonial cells had been irradiated with a split dose. A total of twenty-three dominant mutations was observed in 1,968 offspring derived from irradiated males, and one mutation was observed in 1,739 offspring derived from unirradiated control males.

155. The ratio between dominant skeletal mutations induced in spermatogonial and post-spermatogonial stages is similar to that found in the specific locus experiments. This observation indirectly supports the notion that a sizable proportion of the skeletal abnormalities may result from point mutations. If they had been predominantly chromosomal aberrations, one would have expected a larger difference between the frequencies in spermatogonial and post-spermatogonial stages. The question whether these dominant mutations are transmissible to the second and later generations after irradiation is still open but under investigation.

Dose-rate effect on specific locus and recessive lethal mutations

Mice

156. *Spermatogonia.* Table XII shows that most of the information on dose-rate effects in the spermatogonia of mice was reviewed by the Committee in its 1962 report. The final results of experiments which had not been fully completed when the 1962 report was adopted do not significantly alter the results available at that time and largely confirm the following conclusions arrived at by the Committee:

(*a*) When spermatogonia are given exposures of 300 to 600 R gamma radiation at a rate of 0.009 R per minute (90 R per week), the frequency of induced specific locus mutations is lower by a factor of about three than when the same doses of x rays are delivered at a rate of 90 R per minute.

(*b*) At 0.8 R per minute (gamma rays) most of the dose-rate effect has already occurred, and the yield does not differ significantly from that observed at 0.009 R per minute (gamma rays).

(*c*) At the rate of 9 R per minute (x rays) the response is intermediate between the responses at 90 R per minute (x rays) and 0.8 R per minute (gamma rays).

(*d*) A reduction of the rate from 0.009 R per minute (gamma rays) to 0.001 R per minute (gamma rays) does not result in a further lowering of the mutation frequency.

157. *Oöcytes.* Data on dose-rate effects in oöcytes are fewer than those obtained in spermatogonia because the early follicle stages of the oöcyte development are easily destroyed at those doses that are necessary to obtain adequate mutation frequencies. The data collected thus far lead to the following conclusions:

(*a*) The dose-rate effect in oöcytes is in the same direction as in spermatogonia but considerably larger. At comparable exposures, the mutation frequency for 90 R per minute x irradiation in oöcytes is substantially higher than for spermatogonia irradiated at the same rate. At a rate of 0.009 R per minute gamma irradiation the opposite is true, the mutation frequency for oöcytes being lower than for spermatogonia.

(*b*) The mutation frequency at 0.8 R per minute (gamma rays) is intermediate between the frequencies at 0.009 (gamma rays) and 90 R per minute (x rays). This finding differs from the results for spermatogonia where mutation frequencies at rates of 0.8 R per minute and 0.009 R per minute are not significantly different.

158. In the course of studies on dose-rate effects in oöcytes,[253] it was shown that the genetic response of old females to irradiation (six to nine months old) was different from that of young females (two to four months old), the mutation frequency after exposure of 400 R (0.8 R per minute) in second litters of old females being considerably higher than the frequency in second litters of young females. No such difference has been found for first litters. No satisfactory explanation for this age effect in females (which has also been observed at a rate of 0.009 R per minute) has yet been given.

159. Since the publication of the 1962 report, the interpretation of the dose-rate effect has not changed. It is believed that the dose-rate response is a direct intracellular effect on the mutation process, although it may be influenced to some extent, and particularly at high doses, by secondary processes such as cell selection or change in cell stage during irradiation. Accepting the evidence that the dose effect operates at the intracellular level, it can be explained in terms of repair of pre-mutational damage. Russell suggests two alternative ways in which the dose rate might affect repair of pre-mutational damage. Firstly, there is more damage to the repair system at high than at low dose-rate irradiation. Secondly, the repair system is saturated by high dose irradiation, because its capacity for repair is limited and it has only a limited time in which it can act. Whatever the correct interpretation of the dose-rate effect may be, it seems evident that there is a threshold dose rate below which the repair system is not affected. This threshold is somewhat higher in spermatogonia than in oöcytes.

Drosophila

160. *Spermatogonia.* In a series of experiments with *Drosophila*, Purdom *et al.*[254-256] investigated the dose-rate effect in spermatogonia. The data indicate the presence of an effect at very low dose rates in a single

experiment, but not in others, and the investigators do not interpret their data as providing conclusive evidence for such an effect at the dose levels studied.

161. Although different dose rates were used in experiments of Oftedal,[257-259] there is no clear evidence of a dose-rate effect for mutation induction. There is, however, strong evidence of a total dose effect at high doses similar to that reported by Russell[237] with x rays and neutrons and by Batchelor *et al.*[200] with neutrons. In experiments with *Drosophila* (table XIV) Oftedal reported a linear increase in mutation frequency at low doses, while the frequency of mutations decreased at higher doses. These results suggest that spermatogonial mutation rates in *Drosophila* may be severely biased by the irradiation technique used. Oftedal, in fact, suggests that acute doses of radiation will kill more of the sensitive cells (that is, the more mutable component) of a heterogeneous spermatogonial population, thus allowing sampling of a less sensitive cell type. The data of Abrahamson *et al.*[261] and McSheehy,[240] measuring recessive lethal induction at doses higher than those used by Oftedal, showed a linear increase in mutation frequency as a function of dose. These results indicate that the less sensitive spermatogonial cells were sampled.

162. *Oögonia.* In adult *Drosophila* females, the oögonial stage is the only one that is maintained for a time long enough to permit the application of chronic radiation doses that extend over several weeks. Results of a pilot study of dose-rate effects in oögonial cells[262] were mentioned in the 1962 report, where it was indicated that 4,000 R Co^{60} gamma rays given at the rate of 7,333 R per minute were significantly more mutagenic by a factor of 2.5 than the same dose applied at the rate of 0.2 R per minute.

163. In subsequent large scale experiments,[263] summarized in table XV, it was found that the results obtained were different depending on (1) the experimental conditions under which the acute or chronic radiation was given and (2) whether the radiation doses were measured physically or biologically. On the basis of more recent results[264, 265] which have not yet been published, Muller favours the view that in *Drosophila* there is no significant difference in the mutagenic effectiveness of gamma rays over the approximate sixty-fold range of rates (0.016 to 1 R per minute) used. It was observed, however, that at high dose rates there was a reduction of effectiveness of about 50 per cent as compared to that at low dose rates.

164. *Oöcytes.* The effect of changes in dose rate on the induction of sex-linked lethals in stage seven oöcytes of *Drosophila* is now being studied by Himoe.[402] Preliminary results show that the yield of mutations is the same whether the oöcytes are irradiated at 330 R/min or 9.4 R/min.

Silkworm (Bombyx mori)

165. Two types of dose-rate effects have been discovered in the silkworm, one being the reverse of the other.[200, 266] In one type (type 1) which occurs in the primordial germ cells (spermatogonia and oögonia) in the gonads of newly hatched larvae, the yield after chronic irradiation (0.1 R per minute) is lower than that after acute irradiation (100 to 300 R per minute). In the other type (type 2), where the effect is found in larvae about eight days old in which the gonial cells are in later developmental stages, the mutagenic effectiveness is higher for chronic irradiation than for acute irradiation. After extensive cytological studies and dose-fractionation investigations (paragraph 176) and neutron studies (paragraph 200), the authors proposed the following hypothesis for the interpretation of the complicated features of the dose-rate effects in the silkworm. At least two mechanisms are involved:

(*a*) A certain part of the radiation-induced premutational damage is subject to repair, and the extent to which this damage will be repaired is primarily dose-rate dependent. This results in the production of higher mutation frequencies following acute irradiation.

(*b*) Because the gonial cell population is changing with time from exclusively primordial germ cells to a mixture of primordial and primary spermatogonial cells, and because the metabolic cycle of many cells in the population is blocked by radiation at a sensitive stage, chronic irradiation can give rise to higher mutation frequencies than acute irradiation. Presumably, the degree and extent of the occurrence of either phenomenon depend largely upon the metabolic activity of the irradiated cells.

The chalcid wasp (Dahlbominus)

166. In contrast to what was mentioned in the 1962 report, more recent information indicates that a small dose-rate effect is detected in oögonia of *Dahlbominus.*[267] Significantly more eye colour mutations are induced with 1,000 R acute irradiation (1,000 R per minute of 2 MVp or 300 kV x rays) than with chronic irradiation (0.08 to 0.17 R per minute gamma rays). The mutational yield following acute irradiation is about one and one-half times that obtained after chronic irradiation. Although these results could be explained as a dose-rate effect, other explanations are possible, for example, differences in radiation quality (paragraph 199).

Conclusions

167. Dose-rate effects have been and are being studied in widely different organisms and in various stages of germ cell development. The above survey is not comprehensive, because it has dealt only with the induced genetic damage that most probably finds its origin in one-hit events (specific locus and recessive lethal mutations) and with the germ cell stages that are most important in the consideration of genetic radiation hazards.

168. Among the several species studied, the mouse is the most closely related to man. In this species, we now have exhaustive and indisputable evidence of a dose-rate effect in spermatogonia and oöcytes. The dose-rate effect is more pronounced in oöcytes than in spermatogonia (see table XII).

169. Dose-rate effects have been reported also in *Bombyx mori, Drosophila* and *Dahlbominus.* These effects are not only less pronounced than those obtained in the mouse but also conflicting in nature, and more work is necessary to clarify the situation.

Effects of low doses

170. As early as 1958, on the basis of the dose-rate effect observed in mouse spermatogonia and oöcytes, it was suggested that repair of pre-mutational damage might be affected not only by the dose rate at which the radiation was given but also by the dose itself, it being argued that acute high doses might hamper the repair of pre-mutational damage to a larger extent than would acute low doses. Preliminary results[268, 269] now indicate that in oöcytes the mutation yield after

50 R acute x rays (table XII) is significantly lower than would be expected from the results of irradiation at 400 R, if the dose-effect curve on which the frequency at 400 R lay was a straight line through the origin. This finding is supported by the results of new fractionation experiments in which the total exposure is partitioned into small acute exposures of 50 R separated by intervals of time presumably long enough for the repair process to recover (paragraph 175). These experiments now in progress yield mutation frequencies below those obtained with single, unfractionated exposures.

171. These results from small doses and small dose fractions indicate that the saturation of the repair system or the damage to that system is dose-dependent. It appears that the repair of pre-mutational damage that takes place at low dose rates can also occur to an appreciable extent with acute exposures as high as 50 R.

172. Additional evidence on the effects of small doses on repair mechanisms is provided by the experiments with primary cultures of human and monkey (*Macaca mulatta*) cells in tissue culture.[270-272] Although these data concern chromosome damage rather than point mutations, and somatic cells rather than germ cells, they may be pertinent. Dubinin found that chemicals like cysteamine were able to protect the cells against chromosome damage (most probably due to two or more hit events) resulting from exposures to 25 and 50 R x rays or 50 and 100 R gamma rays. The same chemicals had no protective effect at 12.5 R x rays or at 25 R gamma rays. More evidence that the effectiveness of the repair system is dose-dependent has recently been obtained for the early spermatids of *Drosophila* (paragraph 219).

173. There is now evidence to support what had already been anticipated as soon as the dose-rate effect was discovered, namely that, when acute irradiation is delivered in very small doses, the induced mutation frequency may be as low as that occurring with low dose-rate irradiation. The finding that a substantial repair effect of this kind is occurring in the mouse with acute exposures as high as 50 R indicates that the principle may apply to most of the range of doses usually encountered in human genetic hazards.

The effect of dose fractionation on the induction of point mutations

174. Several lines of evidence indicate that fractionation of the radiation dose sometimes results in a rate of mutation induction that is different from that observed after a single dose. In experiments with spermatogonia of mice, it was observed that the most striking effect of fractionation was found when a total dose of 1,000 R x rays (90 R per minute) was given in two 500 R fractions separated by twenty-four hours (table XVI).[273, 274] Under these circumstances, the mutation frequency in the fractionated series proved to be approximately five times as large as that obtained from the unfractionated series. Some of the other fractionation procedures listed in table XVI also resulted in some increase of the mutation frequency.

175. This effect is presently explained partly in terms of cell-stage synchronization and partly by cell selection. On the other hand, an effect of dose fractionation working in the opposite direction was expected under certain conditions. Thus, since small doses of radiation are now known to produce fewer mutations than would be predicted on the basis of results at large doses (paragraph 170), it was anticipated that a reduced mutation frequency might be obtained when large doses were fractionated into small doses separated by intervals of time sufficiently long for the repair process to recover. There is already some evidence that this is occurring in mice.[269]

176. Fractionation effects similar to those observed in the mouse were reported for spermatogonia and oögonia of the silkworm.[266, 275-277] When a total exposure of 1,000 R was delivered in two or three fractions separated by twelve- or twenty-four-hour intervals, a striking increase in the induced frequency of specific locus mutations was observed in comparison with the unfractionated exposure. The mutation frequencies in the fractionated series were about two times or four to eight times as high as in the unfractionated series, depending on whether the spermatogonia and oögonia were sampled immediately after the hatching of the larvae or seven to nine days later. As in the fractionation experiments with mice, other fractionation procedures increased the mutation frequencies to a lesser extent. The results of the most recent experiments indicate that the depression of repair is due to post-radiation blocking of the cell cycle at the G_2 and/or G_1 phases, in which the amount of repair of the pre-mutational damage is assumed to be small.

177. In *Drosophila*, the effect of dose fractionation has been studied for pre- and post-meiotic male germ cells. Purdom's results of a study on the effect of dose fractionation in spermatogonia were inconclusive.[256]

178. Alexander and Bergendahl[278] reported a fractionation effect for sex-linked recessive lethals in spermatids when irradiation was carried out in the absence of oxygen. Glembodsky *et al.*[279] investigated the effect of acute and fractionated doses of radiation on the induction of sex-linked recessive lethals in spermatids, and no differences were observed. Tates,[280] however, has reported a significant decrease in the frequency of sex-linked recessive lethal mutations in germ cell stages that probably correspond to young spermatids and late spermatocytes when the dose is delivered in five equal fractions, each separated by two-hour intervals, instead of being acute unfractionated.

179. A number of investigators[281-283] have reported that the sex-linked recessive lethal mutation frequency induced in mature sperm of *Drosophila* is the same whether the dose is given in a single exposure or in fractions separated by intervals of days or weeks.

Conclusions

180. The increased mutation frequency observed as a result of dose fractionation occurred only in experiments in which the dose in each fraction was quite high. Therefore, these results would probably apply only to very rare conditions in the exposure of man to ionizing radiation. However, the preliminary evidence on the reduced mutation frequency obtained when a dose is delivered in small fractions may well have many applications in estimating the genetic effects of radiation in man. Quantitative information is needed before these estimates can be made.

The effect of the interval between irradiation and conception

181. In the male mouse, extensive data on spermatogonia have shown no evidence of any significant change in mutation frequency with time after irradiation.

This holds true even to the end of the animal's reproductive life.[237]

182. In contrast to these findings in the male, recently published results from irradiation of female mice with fission neutrons clearly show that the interval between irradiation and conception has a very pronounced effect on the mutation frequency observed in the offspring.[284] In the first seven weeks after irradiation, a period in which two litters are usually conceived, the mutation frequency is high. With a dose of approximately 63 rads, a total of 59 specific locus mutations was observed in 89,301 offspring conceived in that period. After that, no mutations were found in a total of 120,483 offspring. Of all the other biological factors affecting mutation frequency that have been studied, none has produced such a striking effect. There is preliminary evidence (table XII) that the same effect occurs with x rays.

183. The low mutation frequency in the later period comes from oöcytes that were irradiated in immature follicle stages. It is not yet known whether the marked difference in mutation frequencies in the two time intervals is due to a low mutational sensitivity of oöcytes in early follicle stages, to an efficient repair mechanism in these stages, or to cell selection.

184. The extreme lowness of the mutation frequency in the later period is worth emphasizing. In the neutron experiment, the dose was high enough to be highly mutagenic in the early interval after irradiation. Yet in the later period the observed value of the mutation frequency was zero, and even the upper 99 per cent confidence limit of this zero figure was below the spontaneous mutation rate in male mice. (A reliable value for the spontaneous rate in females is not available, although it appears to be lower than that in males).

Conclusions

185. The mutation frequency in female mice is markedly dependent on the interval between irradiation and conception. The frequency is high in the first few weeks after irradiation but then plunges rapidly to an extremely low value. Caution should be used in applying the results to the human female, because the oöcyte stages involved in the two species may not be comparable in their responses. However, there is certainly a possibility of a similar effect and, therefore, an indication that the genetic hazard from the exposure of women may, on the average, be much less than that calculated on the basis of female mouse mutation rates observed after irradiation.

The relative biological effectiveness (RBE) of radiations of different qualities

186. In 1963, the RBE Committee set up by the International Commission on Radiological Protection and the International Commission on Radiation Units and Measurements[285] reviewed the information available at that time on the relative biological effectiveness of different types of radiation with regard to the induction of genetic damage. In general terms, the conclusions of the RBE Committee were that neutrons of various energies were about 2.3 to 7.3 times more effective than x or gamma rays in inducing dominant lethal mutations in spermatozoa of *Drosophila* and the mouse. The actual values depended on the doses and dose rates at which they were determined. RBE values for the induction of sex-linked recessive lethal mutations were of the order of 1.1 to 1.6. The following paragraphs update these conclusions, but the main emphasis is on the recent results obtained with mice, since they have a more direct bearing on the problem of estimation of human risks.

The mouse

Pre-meiotic male germ cells

187. Studies of Russell[237] showed that the rate of induction of specific locus mutations increases with dose when spermatogonia are irradiated with high dose-rate (79 rads per minute) fission neutrons. At doses higher than approximately 100 rads, the mutation frequency is lower than the actual value at 100 rads (table XII). The mutagenic effect of high dose-rate neutrons was compared with that of low dose-rate (0.17 and 0.79 rads per minute) fission neutrons. It was found that there was no dose-rate effect at about 60 rads. However, above 100 rads, high dose-rate irradiation was mutagenically less effective than low dose-rate irradiation. Such a reverse type of dose-rate effect can be explained by assuming that there is a difference in the degree of cell selection under high and low dose-rate irradiation.

188. Comparison of the results of 60 rad neutron irradiation with x irradiation at 90 R per minute indicated an RBE of 5.8 for specific locus mutations. Since the mutagenic effect of 300 R high dose-rate x rays is about 3.3 times larger than the effect of 300 R low dose-rate gamma rays (0.009 R per minute), it follows that an RBE of 19.1 is obtained when the mutagenic effectiveness of neutron irradiation is compared with that of low dose-rate gamma irradiation. Russell arrived at a closely similar RBE value (18.1) on the basis of results of another experiment in which he compared the mutagenic effects of 100 rads of low dose-rate neutron irradiation (0.14 rads per minute), with that of 600 R low dose-rate gamma irradiation (0.13 R per minute).

189. Searle and Phillips reached essentially the same conclusions as Russell with respect to a reversed dose-rate effect at doses above 100 rads (table XII).[260, 280, 288]

190. An analysis of the dose-mutation relationship for very low dose-rate neutron irradiation revealed that it was linear between 0 and 307 rads. The relationship applies to both specific locus mutations and dominant visible mutations. Comparison of the mutagenic effectiveness of 307 rads very low dose-rate neutron radiation with that of 608 rads very low dose-rate gamma radiation led to an estimated RBE of about twenty-three for specific locus mutations and to a figure of twenty for dominant visible mutations.[288]

191. There is little information on the induction in spermatogonia of mutations resulting in dominant lethality. Comparison of litter-size at birth after 307 rads of chronic neutrons compared to 608 rads of chronic gamma rays showed a significantly smaller litter-size in the former.[288]

192. Recent cytological studies have shown that about 21 per cent of the spermatocytes derived from irradiated A type spermatogonia showed multivalent configurations (indicative of translocation heterozygosity) when the spermatogonia were irradiated with 307 rads of low dose-rate neutrons. A percentage of 3.5 was obtained when similar cells were irradiated with 207 rads of higher dose-rate neutrons.[289, 290] If the data obtained for low dose-rate neutron irradiation are compared with those for low dose-rate gamma

irradiation (paragraph 124), and if it is, furthermore, assumed that the dose-mutation relationship for low dose-rate neutron and gamma induction of translocations is linear, then fast neutrons may be as much as forty times as effective as gamma rays from this point of view.

Post-meiotic male germ cells

193. Recently Searle *et al.*[286] obtained some information on the induction of dominant lethal mutations in spermatozoa which had been exposed to 0.7 MeV neutrons at 0.01 rads per minute. The biological effectiveness of these neutrons was calculated by comparing them with x rays on the basis of induced dominant lethality as estimated through live embryos/*corpora lutea* ratios in the control and in the irradiated series. At about 50 per cent induced dominant lethality (100 rads for neutrons, 600 R for x rays), neutrons turned out to be 5.8 times more effective than x rays. Searle's results confirm the results of Russell's studies[293] on the relative effectiveness of neutrons from a nuclear detonation and from a cyclotron. Both Searle and Russell observed that neutron irradiation induced more mutations in spermatids than in spermatozoa. This had been found in earlier studies with x irradiation.

194. The RBE values obtained by Searle and Russell are fairly close to those published by Pomerantseva.[294, 295] The post-meiotic germ cells in her experiments were irradiated with fast neutron (1 MeV) doses from 17 to 228 rads (4.3 to 11.6 rads per minute). Using pre- and post-implantation death as criteria for dominant lethal induction, Pomerantseva estimated that fast neutrons were five to six times more effective than Co^{60} gamma irradiation and four times more so than x rays.

195. Pomerantseva also studied the RBE of 660 MeV protons and observed that this type of radiation was about half as effective as x rays and 0.65 times as effective as Co^{60} gamma irradiation. As a result of studies with rats, Plotnikova *et al.*[296] found that the RBE of 500 MeV protons for dominant lethals in spermatozoa was 0.6 to 0.7 when compared to 180 KeV x rays.

Female germ cells

196. Comparison of the effects of high and low dose-rate neutron irradiation of oöcytes with a dose of approximately 60 rads showed that high dose-rate irradiation is significantly more mutagenic than low dose-rate irradiation.[284] However, the dose-rate effect for neutrons is smaller than that for gamma rays. At high dose rates, the published data on mutation frequency in oöcytes indicate an RBE similar to that obtained for spermatogonia.[284]

Drosophila

197. Abeleva and Lapkin[297, 298] irradiated spermatozoa and spermatids of *Drosophila* with doses of 600, 1,200 and 2,400 rads fast neutrons (55 rads per minute), on the one hand, and 1,200 and 2,400 rads acute x rays on the other. The relative biological effectiveness of neutrons, compared to x rays, as measured by the induction of dominant lethals, was 2.4 to 2.8 for spermatozoa and 1.3 to 1.5 for spermatids. If one takes into account the fact that RBE values vary with dose, dose rate and energy spectrum of the neutrons, the RBE values obtained by Abeleva and Lapkin are in good agreement with those mentioned in the review of the RBE Committee. The same applies to the results of Abeleva and Lapkin's studies on the induction by neutrons of sex-linked recessive lethals in spermatozoa and spermatids. For this category of genetic effects, the RBE for neutrons compared to gamma rays is 1 to 1.5.

198. The finding that the RBE value for spermatids is lower than for spermatozoa is presumably due to the fact that spermatids have a higher sensitivity than spermatozoa to the induction of genetic damage by x rays in the presence of oxygen.[299] Dauch *et al.*[300] observed that, for spermatozoa sampled on the second day after irradiation, the RBE for fast neutrons, as measured by the induction of both recessive lethals and translocations, was considerably higher than in sperm sampled during the first day after radiation exposure. This difference can be ascribed to a difference in oxygen tension between the two stages, because Sobels[301] was able to show that the higher sensitivity to x irradiation of fully mature spermatozoa compared to that of almost mature sperm cells is a consequence of a higher degree of oxygenation in fully mature spermatozoa.

199. With 660 MeV protons, Rappoport *et al.*[302] obtained a linear yield of sex-linked recessive lethals in spermatozoa at doses between 500 and 12,000 rads and a rate of induction of 2×10^{-5} mutations per rad. The data suggest that the RBE of 660 MeV protons compared with gamma rays is approximately one. The early studies of Edington[303] and Edington and Randolph[304] on the induction of sex-linked recessive lethals in spermatozoa had shown that the RBE of x rays compared to gamma rays was approximately 1.1 or 1.4 depending on the dose level at which it was estimated. The fact that high dose-rate x rays are mutagenically more effective than high dose-rate gamma rays has more recently been confirmed by Seeley *et al.*[305] for sex-linked recessive lethals in spermatozoa and by Purdom and McSheehy[255] for IInd chromosome lethals in spermatozoa and spermatogonia.

Silkworm

200. The relative biological effectiveness of 14 MeV neutrons, fission neutrons and gamma rays was determined for specific locus mutations induced in early (type 1 dose-rate effect) and late gonia (type 2 dose-rate effect).[306-309] Since mutation frequencies were found to increase faster than linearly with dose regardless of the type of radiation applied, it is impossible to describe the difference in mutagenicity of these types of radiation by one single RBE value over the whole dose range. Therefore the authors estimated the RBE values at a fixed mutation frequency. The RBE values of 14 MeV neutrons in comparison to gamma rays are the following: early spermatogonia (0.8 pe-locus; 1.0 re-locus); late spermatogonia (3.2; 2.1); early oögonia (1.2; 1.2) and late oögonia (1.7; 2.8). For 1.5 MeV fission neutrons, these values are: early spermatogonia (1.7; 1.9); late spermatogonia (4.2; 3.5); early oögonia (2.1; 2.4) and late oögonia (3.8; 3.0). These RBE values make it possible to rank the three types of radiation according to their relative efficiency of mutation induction as follows: gamma rays, 14 MeV neutrons and fission neutrons.

Dahlbominus *and* Mormoniella

201. In experiments on the induction of specific locus mutations in early and late oöcytes of *Dahlbominus* by 750 rads of 14 MeV neutrons and 750 rads

of gamma rays, Baldwin[310] observed that high dose-rate neutrons were mutagenically more effective than high dose-rate gamma rays, but not significantly so. The relative mutagenic effect of neutrons and gamma rays in early and late oöcytes was the same.

202. The induction of specific locus mutations in oöcytes of *Mormoniella* has been studied by Kayhart[311] who compared the relative mutagenic effectiveness of thermal neutrons, fast neutrons and x rays. The RBE for thermal neutrons could not be estimated, but the RBE for fast neutrons was seventeen to twenty-one in the low dose range (45 to 70 rads) and two to four at higher dose levels (240 to 1,400 rads).

Conclusions

203. All evidence confirms that neutron irradiation is mutagenically more effective than x or gamma irradiation. There are indications that the relative mutagenic effectiveness of neutrons increases with their linear energy transfer. Results of studies with such widely different species as the mouse and the silkworm indicate that RBE values for neutrons are almost always in the range of one to six. An RBE value for the mouse of about twenty was obtained when the mutagenic effects of low dose-rate neutrons or of low doses (60 rads) of high dose-rate neutrons were compared with low dose-rate gamma radiation. It has been established that RBE values can change with doses and dose rates, and it is also known that the RBE values may be different for germ cell stages and types of genetic damage.

Repair of pre-mutational damage

204. Since dose-rate effects and some fractionation effects are usually interpreted on the basis of repair of mutational damage, it seems appropriate to review some of the recent advances in this field.

Paramecium

205. Kimball's extensive studies[312-314] with *Paramecium* showed (*a*) that various post-irradiation treatments can reduce the amount of x-ray induced mutation but only when applied before the first chromosome duplication that follows irradiation, thus demonstrating the existence of reparable pre-mutational damage; (*b*) that the mutation yield is inversely related to the length of the interval between irradiation and chromosome duplication. By sufficiently prolonging the time interval between irradiation and DNA synthesis, up to 60 per cent of the maximum mutation yield can be eliminated. Irradiation damage produced during post-duplication (G_2 phase) and early prophase is subject to most effective repair, and probably almost all of the initial lesions produced during these phases are potentially reparable; (*c*) that the reduction in mutation yield, and therefore the amount of repair, depends on growth conditions and on the presence of metabolic inhibitors, suggesting that an enzymatic process is responsible for the observed reduction.

206. The hypothesis that enzymes are involved in repair of pre-mutational damage in *Paramecium* is made probable by the observation that the mutation yield is increased when the time between irradiation and DNA synthesis is decreased. If there is little time between irradiation and DNA synthesis, there is less opportunity for repair to occur, and, as a consequence, most of the pre-mutational damage will be irreversibly fixed at the time of chromosome duplication.

207. Kimball's investigations into the nature of of the initial radiation damage led him to conclude that thethere were at least two different kinds of pre-mutatiotional damage, one producing permanent alterations (i (i.e., mutations) through misrepair, the other through m misreplication. The results obtained with *Parameciecium* suggest that misrepair is less efficient than misrepliplication,[313] since the longer the time between irradiatiation and replication, the lower the yield of mutations. ThThus, potential mutagenic lesions tend to disappear withthout producing mutations prior to replication but tend to to be converted to mutation at replication. Studies of tf the pre-mutational lesions that cause mutations by m misreplication revealed that these lesions usually affeciected both conserved strands of the DNA and therefore re all progeny of such cells.[313, 315]

208. Repair processes have also been observed fl for mutations induced by 2,537Å ultra-violet radiation, n, by alpha particles,[316, 317] by nitrogen mustard, and d by tri-ethylene melamine (TEM).[318] At least with TETEM mutations, however, the pre-mutational damage tl that is repaired differs from that due to x rays, since mmost TEM-induced mutations, unlike those induced byby x rays, affect only a half or a quarter of the progeny ny of the cell in which a mutation has been induced. Frfrom observations on the repair of x-ray and TEM-indudduced mutations, it appears that *Paramecium aurelia a* is capable of repairing more than one kind of pre-mututational damage. The two mutagens, furthermore, diffliffer in that the x-ray damage incurred in the G_2 phase se is almost completely repaired,[319] whereas a comparativeively high yield of mutations is still obtained after TETEM treatment of G_2 cells.[318]

Bacteria

209. Studies of repair in bacteria deal mainly wiwith damage affecting the survival or the growth of tl the treated cells and, therefore, would not seem to b be strictly relevant to the problem of repair of prpre-mutational damage. The reason for mentioning sonsome of these studies in this report is that they have throwown considerable light on the molecular and enzymatnatic mechanisms of repair of ultra-violet or chemicalcally induced damage in the DNA. Since no comparabable information is available with regard to ionizing radiadiation, these results may help us to understand tl the phenomena resulting from x irradiation.

210. Part of the damage affecting the survival d of ultra-violet-irradiated bacteria (or bacteriophages) coconsists in the formation of thymine dimers and can b be repaired through the intervention of two groups o of enzymes. The photo-reactivating enzymes have beeeen shown[320, 321] to split the ultra-violet-induced thymimine dimers in the DNA into the original separate thymimine residues in the presence of light with wave-lengtlgths ranging from about 320 to 450 mμ. Dark-repapair enzymes, on the other hand, excise the ultra-violeblet-induced thymine dimers.[322-325] The excision of dimeners is followed by *restitutio ad integrum* of the originainal DNA molecule through a process of reparative repliçaication that fills the gaps with new material. Unlike the photo-reactivating enzymes, the dark-repair enzymes do not require visible light.

211. Both enzymatic systems are genetically controlled and many radio-sensitive mutants, lacking either the photo-reactivating or the dark-reactivating enzymes, have been isolated. On one occasion, an enzyme capable of initiating dark-repair *in vitro* has been isolated from *Micrococcus lysodeikticus*.[326-328]

212. Investigations into the action of the difunctional alkylating agent mustard gas on the growth and survival of *E. coli* strains, with and without dark-reactivating enzymes, have provided evidence that chemically induced lesions in the DNA can also be repaired.[329] The strain carrying the dark-reactivating enzymes was able to excise (i.e. repair) the inter-strand cross-links between guanine moieties that are induced by mustard gas. The strain which did not have the dark-reactivating enzymes could not remove these inter-strand cross-links in the DNA.

213. Less is known with regard to repair of pre-mutational damage. Witkin *et al.*[330] studied mutation induction in three different strains of bacteria. The first strain (H/r) was used to study the induction of mutations to streptomycin resistance. The second strain (H/r30) was an arginine requiring substrain of the first and was used to study mutations to prototrophy. Neither of these strains carried the photo-reactivating enzyme, whereas the third strain (B/r) did. The mutations studied in the B/r strain were mutations to streptomycin resistance. Mutation induction by ultra-violet in all strains was studied in the absence or presence of post-treatment with photo-reactivating light (wavelength 320 to 450 mμ).

214. It was found that the H/r strain was not capable of repairing potential mutations to streptomycin resistance in the presence of photo-reactivating light. The H/r30 strain, on the other hand, was able to repair potential mutations to prototrophy in the presence of photo-reactivating light. The B/r strain which has the photo-reactivating enzyme was capable of repairing potential mutations to streptomycin resistance in the presence of photo-reactivating light. From these results, it was concluded that ultra-violet light induces two kinds of pre-mutational damage. Repair of the first kind of damage (i.e., potential mutations to streptomycin resistance) can only take place in the presence of the photo-reactivating enzyme and photo-reactivating light. The second type of damage (i.e., potential mutation to phototrophy) requires only photo-reactivating light for its repair.

215. The mechanism of repair of pre-mutational damage in bacterial strains which have or lack the photo-reactivating enzyme is not yet well understood. This applies even more to our knowledge concerning the process of repair of ultra-violet-induced mutations in bacterial strains having or lacking the dark-reactivating enzymes.

216. The examples given above have shown striking parallelism between the error-correcting mechanisms for such different lesions in the genetic material as those induced by ultra-violet and mustard gas. There is no such detailed evidence for the mechanism of the repair processes in bacteria exposed to ionizing radiation. However, it has been demonstrated that bacterial strains show a difference in sensitivity to ionizing radiation depending on whether they are equipped with repair enzymes or not. These results seem to suggest that damage from ionizing radiation may be repaired in a way similar to that observed with ultra-violet or chemically treated bacteria.

Metazoan germ cells

217. In the sections on dose-rate and dose-fractionation effects, it was shown that results of dose rate and dose fractionation on mutation induction could be explained by assuming that the mutation process in metazoan germ cells was subject to repair. The possibility of interfering with repair processes in metazoan germ cells by means of pre- or post-irradiation treatments with metabolic inhibitors has been amply demonstrated in the past decade. The details of the evidence are comprehensively analysed in recent reviews.[314, 331] The main conclusions only will therefore be outlined in this report.

218. Evidence for the operation of repair processes in early spermatids and mature spermatozoa of *Drosophila* has been obtained in studies on the effects of different post-treatments by Sobels *et al.*[229, 301, 331-337] It is remarkable that early spermatids and spermatozoa give an opposite response to the same treatments. This suggests that the metabolic pathways involved in the mutational event are essentially different in these two different stages of sperm development.

219. For early spermatids, post-treatment with cyanide or nitrogen following radiation exposure in air leads to an increase of the frequency of recessive sex-linked lethals in a ring X chromosome.[331-335] Since the formation of peroxides could be ruled out in the cyanide experiments[335] it was concluded that inhibition of respiratory enzymes enhanced the mutation frequency by inhibiting a repair process. Further evidence for the oxygen-dependence of the repair system in early spermatids was provided by the observation that, after inhibition of the repair process by anoxia before and during irradiation, the mutation frequency was markedly lowered by post-treatment with O_2, as compared to that observed with N_2.[299] By applying the same treatments (pre-treatment with N_2, followed by post-treatment with either N_2 or O_2) at three dose levels (1,250, 2,500 and 3,750 R), Watson[338] observed that the absolute reduction of the mutation frequencies in the O_2 post-treated series was the same at all three doses. These results suggest that the repair system can only cope with a limited amount of damage, because at the low dose level repair is considerably more effective than at the high dose level where the system apparently becomes saturated. The repair process in early *Drosophila* spermatids therefore shows a remarkable similarity to that postulated by Russell to explain the effect of dose (paragraph 171) and dose rate (paragraph 159) in the mouse.

220. In contrast to the findings in early spermatids, post-treatment with O_2 of mature spermatozoa irradiated under anoxia, increases the frequency of mutations as compared to that observed after post-treatment with N_2.[299, 336] Post-radiation interaction of radicals with O_2 cannot explain this effect.[301] As in spermatids, the post-radiation reduction of the genetic damage in spermatozoa by N_2 is thought to be mediated by an enzymatic repair process which derives its energy from glycolysis. The modification by O_2 versus that by N_2 shows a remarkable similarity to glycolysis, which is also adversely affected by oxygen, but favoured by anoxic conditions. This idea is supported by the finding that pre-treatment with two specific inhibitors of the glycolytic pathway, i.e. sodium fluoride and iodoacetamide, leads to a considerable increase of the radiation-induced mutation frequency in spermatozoa.[301, 339]

221. A contrasting response of early spermatids and spermatozoa has also been observed after inhibition of protein and/or RNA synthesis, since pre-treatments with chloramphenicol, ribonuclease or actinomycin-D all result in an increase of the radiation-induced mutation frequency in sperm, but in a decrease in early spermatids.[301, 331, 337] It is clear therefore that protein

and/or RNA synthesis also play a part in the mutation process in *Drosophila*. It is not yet possible, however, to state more precisely which specific steps are involved from the induction of premutational damage to mutation fixation.

222. Several lines of evidence indicate that the observed post-radiation modifications in early spermatids and mature spermatozoa cannot be explained by:

(*a*) Shifts in the sampling of germ cells with different radio-sensitivities among groups of flies (or pupae) having received different post-treatments,[301, 336, 338] nor by

(*b*) Selective elimination of cells with recessive lethal mutations or with translocations.[301, 339]

223. Experiments of Tazima *et al.*[340] with silkworm showed that it was possible to increase the yield of specific locus mutations in spermatogonia and oögonia by subjecting these cells to post-irradiation treatments with cyanide, chloramphenicol, nitrogen gas and low temperature.

Quantitative traits

224. The general problems associated with radiation-induced changes in quantitative traits were discussed in great detail by the Committee in its 1958 report. In particular, the Committee analysed the implications of induced changes of the variance and the mean of such traits as birth-weight, intelligence, life span and fertility. The discussion was largely based on data collected in *Drosophila* and in plant material. Further experimental results were reviewed more briefly in the 1962 report.

225. Although more data on *Drosophila* have been obtained in recent years,[341-345] in this report attention will only be given to recent results obtained with vertebrates, on which almost no data were available for inclusion in earlier reports. While the information available on the induced changes in quantitative traits in vertebrates is much less complete than in *Drosophila* and plants, the results reviewed here may have a far greater relevance to the situations likely to obtain in man.

226. *Body-weight.* Touchberry and Verley[346] obtained evidence that body-weight at thirty-two days was increased and growth rate speeded up in the offspring of mice whose ancestors had been irradiated during six generations with doses from 10 to 240 R. Newcombe and McGregor[347] studied radiation-induced changes in body-weight in the offspring of rats whose male ancestors had received gonadal exposures of 600 R of x rays over thirteen successive generations. It was found that rats in the irradiated series tended to be heavier than their controls. The relative incidence of "heavy" as compared with "light" animals (defined as those in the upper and lower halves of the weight distribution for irradiated and control groups combined) was significantly higher in the irradiated than in the control group by factors of 3.4 for males and 2.2 for females. It could be demonstrated that the radiation-induced changes in body-weight were primarily associated with induced hereditary changes and not merely a secondary effect of the radiation-induced reductions in litter-size.

227. *Maze-performing ability* was studied in rats which descended from a population in which males (in one experiment both males and females had been irradiated) had been exposed to 400 to 1,000 R acute x rays during twelve consecutive generations. Newcombe and McGregor,[348] who performed these studies, observed a decline in maze-performing ability in the irradiated series. In the same series, they noticed a 70 per cent increase in "dull" animals (animals with error scores that exceeded the mean by more than one standard deviation) and a 30 per cent decrease in "bright" animals (animals with scores lower than the mean by more than one standard deviation). Contrary to expectations, analysis of the variability of error scores revealed a decreased variability in the irradiated series. By analogy with the author's findings with regard to radiation-induced changes in body-weight, it is assumed that radiation-induced reduction in maze-learning ability could also be explained as being due to hereditary changes and not to differences in litter-size between control and irradiated series. At present, it is difficult to evaluate to what extent maze-performing ability in rats can be equated to a clearly defined component of human intelligence.

228. *Life span.* As early as 1957, Russell[349] obtained evidence of a reduction in life span in the offspring of male mice that had been exposed to 30 to 80 rads of detonation neutrons. Since then, several other workers became interested in this subject, because radiation-induced life-shortening represented a genetic hazard for the human population that had been hitherto unforeseen. Spalding[350] irradiated male mice with 30 to 180 rads acute fission neutrons in one experiment and with 60 to 300 rads gamma rays in another. His results did not indicate any reduction in life span in the offspring from irradiated sires or grandsires. On the contrary, the average life span of all control females was shorter than that of the female offspring from irradiated sires or grandsires. The effect was even more pronounced in the male progeny. The results for neutrons and gamma rays were the same.

229. Frölen[351] observed no shortening of the life span in the first generation offspring of male mice exposed to 500 R acute x rays. The results were the same when the irradiated males received a pre-treatment with cysteamine. The life span of the female offspring of irradiated males was not analysed.

230. Studies were also made concerning the life span of descendants of mice populations which had received irradiation during five or more generations. Spalding and Strang[352] failed to demonstrate any reduction of life span attributable to ancestral irradiation in male and female mice from sires exposed for five, ten and fifteen generations, to 200 R acute x rays per generation. Gowen and Stadler[353] exposed mice to gamma irradiation from mating to death. Each following generation was maintained in the irradiation field from conception to death. Extensive analyses of the life span were made when the strains of mice had completed lives through the sixth generation. The average ancestral radiation doses for each generation were, in order of generation, 370, 680, 820, 980, 1,180 and 1,290 R. The results of these experiments indicated that ancestral irradiations over six generations had little effect.

231. All studies listed above, with the exception of that of Russell, have shown that irradiation has little or no effect on life span in offspring from mice irradiated during one or more generations. Russell's experiment was the only one in which fission neutrons were used to irradiate the most sensitive of the post-meiotic male germ cell stages, these conditions being chosen as likely to increase the probability of obtaining an effect.

232. *Reproductive life* and its modification by irradiation have been studied by Spalding *et al.*[352] They observed that the average period of fertility was longer in the offspring of mice that had been irradiated with 200 R per generation during twenty successive generations than in controls.

233. *Skeletal abnormalities.* Searle[354] studied continuous and quasi-continuous skeletal variation in descendants of sublines of an inbred strain of mice which had been kept in a radiation field for about nine generations, receiving 1 R per night and about 80 R per generation. The study has not yet produced a clear picture, and further studies are indicated.

234. The only other vertebrate species in which the effect of irradiation on a quantitative trait has been studied is the chicken. In these experiments,[355] cocks were irradiated to induce genetic variability in stocks which would otherwise no longer respond to selection for high egg numbers. Irradiation of cocks with 1,000 to 1,500 R acute x rays per generation over a period of seven generations, followed by selection for high egg numbers during six generations, led to a negative result; namely, the response to selection was no different, or perhaps slightly less, in the irradiated lines than in controls.

235. In conclusion, studies on radiation-induced changes of quantitative traits in vertebrates have yielded results that must be considered as still fragmentary. The information available does not make it possible to evaluate how the means and variances of these traits are changed by radiation. It must, however, be noted that the only experiment concerning an intellectual function reveals a decrease in the mean without an increase in the variance. Estimates of risk of induced changes of quantitative traits in man must wait until further results are obtained with vertebrates, particularly with mammals.

Miscellaneous genetic effects

Space-flight results

236. In the context of the present report, results of genetic experiments performed in orbiting spacecraft are not of immediate relevance to risk estimates, since the doses received by man and experimental animals have thus far been extremely low and mostly of the order of tens of millirads. It seems, however, appropriate to mention this subject, because some results suggest that genetic damage is induced during space flights even if the detectable amount of ambient radiation in the spacecraft has been small, indicating that other space-flight parameters, such as vibration, weightlessness and acceleration, may induce genetic effects either by themselves or in combination with the ambient radiation.[356-367]

237. Two lines of research have been followed in studying the influence of space-flight parameters. Firstly, several attempts have and are being made to simulate these parameters in ground tests. Thus far, these model experiments on the ground have mainly focussed on the action of vibration alone and in combination with acceleration or with irradiation. The results have been found to be dependent on the type of vibration, the duration of the treatment, the type of genetic damage under study and on whether the vibration was applied before or after the irradiation.[368-371]

238. In the second line of research, the biological material (human blood cells) was treated by irradiation from a man-made radiation source during the orbital phase of the spaceship mission.[372, 373] The frequencies of single and multiple-break aberrations induced in the blood cells were compared with the frequencies in non-irradiated cells aboard the spaceship and with the frequencies in irradiated and non-irradiated cells that remained on the ground. The in-flight control gave the same result as the control on the ground, indicating that the space flight by itself did not induce aberrations. Comparison of in-flight irradiated cells with those irradiated on the ground on the other hand revealed that, while there was no significant difference with regard to yields of multiple-break aberrations, the frequency of single-break aberrations was significantly higher in the samples irradiated in flight. Apparently, synergism exists for the production of human chromosome aberration between radiation and some space-flight parameters.

239. It is still too early to obtain a complete picture of the interaction of irradiation with space-flight factors, and, so far, there is no common opinion as to whether space-flight factors by themselves are able to induce some kinds of genetic damage. The current findings are important, because they lead us to realize that the magnitude of the genetic and somatic risks encountered in space flights is not only determined by dose, dose rate and quality of the radiation received in those circumstances but also by parameters that are peculiar to space-flight situations.

Effects of internally-deposited radio-active isotopes

240. Increased rates of dominant lethals in male mice have been observed by Lüning *et al.* after injection with Sr^{90}. New and extensive data indicate that the increase of dominant lethals is not observed in matings within the first three weeks after injection, but only from the fourth week onwards. This corresponds to effects on spermatocytes and spermatogonia.[374-376]

241. The genetic effects of C^{14} incorporated into *Drosophila* have been studied by Purdom. Preliminary results suggest that the recessive lethals induced by C^{14} are largely due to the emitted beta radiation and probably not, or to a much lesser extent, to transmutation.[377]

242. Tritiated thymidine and deoxycytidine have been studied for their capability to induce sex-linked recessive lethals in *Drosophila* males.[378, 379] The distribution of the lethals along the X chromosome was different for the two types of radio-active chemicals. Two regional differences have been noted, one of high mutability after tritiated thymidine and one of high mutability after tritiated deoxycytidine.

243. Olivieri and Olivieri[380] studied the mutagenic effect of tritiated thymidine and uridine in *Drosophila* males and found that tritiated uridine increased significantly the frequency of sex-linked recessive lethals in spermatocytes. The mutagenic effect of tritiated uridine was even more pronounced when applied in combination with actinomycin D.

IV. Risk estimates

244. Risk estimates express a probable quantitative relationship between doses of radiation and frequencies of certain effects. In this report, risks of genetic effects will be expressed in terms of expected frequencies of genetic changes (point or chromosome mutations) induced per unit dose or function (e.g.

power) of dose. In earlier reviews of genetic effects by the Committee, risks were expressed in terms of doubling doses, these being the doses required to produce a number of mutations equal to those occurring naturally in one generation. Doubling doses can easily be computed when both the natural incidence and the rate of induction of a certain category of mutations are known. When both figures are available, the doubling dose is a compact way of summarizing the information regarding a given effect in given circumstances. The use of the doubling dose, however, is not necessary in arriving at risk estimates, and for that reason the more direct approach is employed in this report.

245. Risk estimates as defined in paragraph 244 have the advantage that they can be obtained in the non-linear case in which a single doubling dose would have little meaning. They are also absolute estimates which give at once the risk in terms of effects, whereas this type of information is less directly obtained from the doubling dose and involves unnecessary assumptions regarding the proportionality between spontaneous and induced rates. Finally, risk estimates as expressed in this report are consistent with the practice followed by the Committee with regard to the risk of induction of malignancies.

246. It may be pointed out that, while estimates of risks of induction of malignancies in man can be derived from the results of irradiated human populations, this is not possible with regard to genetic risks. As will be discussed below, *in vivo* human data are inadequate to provide estimates of genetic risks. These must be based on results of experiments with animals —mainly mice—and with human somatic cells *in vitro*. While there appears to be no alternative at present to the use of such experimental material, its limitations must be clearly borne in mind and will be stressed throughout the following paragraphs. Because of unavoidable inferences from one species to another or from one type of cell to another, the estimates thus obtained are less reliable than the data from which they are derived.

247. An additional difficulty with genetic risks is encountered in expressing them in meaningful terms. The eventual result of the great majority of genetic changes is, sooner or later, the failure of cells carrying those changes to be transmitted to the following generations. Only in a minority of cases—such as certain dominant traits and certain chromosome anomalies that occur frequently in the population and that are easily detected—can we make assumptions as to the manner in which the damage will be expressed. For most genetic changes even conjectures are not permissible regarding the actual manifestation of the damage throughout generations in terms of individual or collective hardship.

248. The estimates reviewed in the following paragraphs were obtained for acute irradiation of spermatogonia by low LET radiation at high single doses. The consequences of irradiation of oöcytes, and those of exposure to radiation of different quality at different doses and dose rates, will be considered separately.

Point mutations

Total risk of induction

249. Much as the total rate of spontaneous mutation can be derived from an analysis of the excess of female over male new-born children, so the total risk of induction could, in principle, be obtained from the shift of the sex-ratio to be expected in the offspring of irradiated mothers as a consequence of the induction of sex-linked recessive lethal mutations. Such a shift has, in fact, been observed and was used by the Committee in its 1958 report to obtain risk estimates. Further observations were summarized and discussed in the 1962 report, in which the Committee discarded estimates of risk of point mutations based on the sex-ratio shift, because, while the shift undoubtedly was largely due to genetic damage and might have reflected a point mutational component, it was not possible to rule out or separate the possible confounding effect of induced chromosome anomalies, the high frequency of which was not known in 1958.

250. Such a reservation is still valid now, despite new data[381, 382] on the offspring of irradiated mothers which confirm earlier observations, and although no increase in the frequency of sex-chromosome anomalies was noted in a survey[383] of the female offspring of irradiated mothers. The size of the survey was too limited, however, to exclude the possibility that induced anomalies of the sex-chromosomes may account for at least part of the effect on the sex-ratio.

251. A further reason why risk estimates based on the sex-ratio shift are not being made in this report lies in the limitation of the data themselves. The largest and dosimetrically best known material is still that collected among the irradiated populations of Hiroshima and Nagasaki.[384] Doses in the parental populations were between 0 and 200 rads, however, and the observed shift was not significant.

252. Some effect on the sex-ratio may also be expected after paternal irradiation, primarily as a consequence of the induction of sex-linked dominant lethals. The expectation has not been conclusively borne out by observations in man, and experimental studies have shown that in mice the results of paternal irradiation cannot be explained on the sole basis of the induction of sex-linked dominant lethals.[384, 385]

253. No other human data are yet available that would make it possible to obtain risk estimates for the induction of point mutations. As in the 1962 report, it will therefore be necessary to base risk estimates in man on rates of induction observed in the mouse. However, it is no more possible now than it was in 1962 to assess how close the rates of induction in man and in the mouse might be. For want of better data, it will be assumed that rates of induction of mutations are the same in man and in mice, but the arbitrary nature of such an assumption needs to be underlined. The possibility that rates of induction may be higher in man than in mice should not be overlooked.

254. The average rate of induction of mutations at twelve specific loci in mouse spermatogonia exposed in the range 300-600 R acute x rays is estimated to be about 1×10^{-7} per locus per roentgen (paragraph 133). The confidence limits, when this figure is used as an estimate of the average rate for all loci in the mouse, are presumed to be about one order of magnitude apart.

255. The size of the human genome in terms of loci at which detectable mutations arise was estimated in paragraph 24 to be between 7,000 and 70,000. As mentioned earlier, the estimate, although very crude, is in agreement with similar but more precise estimates valid for *Drosophila*. It also agrees with a number of other published estimates of the number of mutable loci in man.

256. If the rate of induction of specific locus mutations assumed to apply to man (paragraph 254), is multiplied by the estimated size of the human genome, the resulting estimate of total risk of point mutation in man is 2×10^{-3} mutations per gamete per roentgen. Taking into account the variability of the data on which it is based, it can be assumed that the approximate confidence limits of the estimates are between one and two orders of magnitude apart. While this range reflects the sampling variability of the estimate, the dubiousness of some of the underlying assumptions must also be borne in mind.

257. It will be recalled that direct estimates of the total rates of induction of lethal recessives in mice have been obtained from two independent sets of experiments and are remarkably close (paragraphs 143, 144). Allowing for the fact that these estimates measure only a known part of the damage measured by experiments at specific loci makes it passible to compare direct estimates of the rate of induction over the whole genome with those obtained indirectly. The direct method gives a lower estimate ($\sim 0.5 \times 10^{-3}$) than the indirect method. The upper confidence limit of the direct estimate (1.6×10^{-3}), however, is well within the range of the indirect one. Such an agreement gives strong support to the estimate discussed in paragraph 256, especially as the direct estimate is based on a smaller number of assumptions.

258. The nature of the damage measured by the total rates of induction is as difficult to assess as is that measured by the total rate of spontaneous mutation which was discussed in paragraphs 25-27. The total rate of induction includes all mutations of every degree of dominance and harmfulness. They will all eventually be eliminated from the population.

259. The mechanisms through which the spontaneous mutational damage could be eliminated were mentioned in paragraph 27. These mechanisms also apply to the induced damage, but the relative contribution of any mechanism to the process of elimination cannot in the current state of our knowledge be assessed. It is therefore not possible to express damage, as measured by the total rate of induction, in terms of individual or collective hardship.

260. If observations made in *Drosophila* can be used as a model for the situation obtaining in man, the induced damage will initially be eliminated at the rate of 4-7 per cent per generation. The rate will, after a few generations, taper off into a rate between 1 and 2 per cent that will persist approximately at the same level until all the induced damage has been removed. Genes will persist in the population for periods of time inversely proportional to their rate of elimination and therefore dependent upon the severity of their expression in heterozygotes. If a population was steadily exposed to a constant amount of radiation per generation for a number of generations, the rate of elimination of the damage would tend to become equal to the rate of induction.

Risk of induction of dominant mutations

261. The difficulty of evaluating the total mutational damage in socially meaningful terms justifies attempts to obtain independent estimates of that part of the damage that can be expected to find its expression in an indisputably injurious way. Experimental data that lead to high estimates of dominant skeletal damage in the mouse were discussed in paragraphs 151-155. While it is too early to evaluate from these data the effects at low doses, the Committee wishes to emphasize that this type of observation may in the future offer a clue to the estimation of risks of induction of dominant mutations in man. In the meantime, dominant damage in man can only be estimated for that portion of the genome that is responsible for a selected group of dominant traits (paragraphs 8-11).

262. When the Committee reviewed genetic effects in 1958 and 1962, it gave estimates of the expected frequency that these traits would reach in the population at equilibrium under conditions of steady irradiation. It is, however, more informative and more consistent with the approach adopted for estimating the over-all mutation rate if the rates of induction after a single exposure are obtained, and this approach will be followed here.

263. For that purpose, the rate of induction per locus per roentgen (1×10^{-7}) as observed at specific loci in the mouse will be used. The rate, however, applies to recessive mutations. It will be recalled (paragraphs 149, 150) that limited mouse data show that the over-all rate of induction of dominant visibles is considerably lower than that of recessive visibles. The interpretation of that phenomenon is difficult, but it cannot be excluded that it may in part reflect a lower average rate of induction of dominant mutations. The rate of induction used therefore can only be considered as an upper limit, for it probably over-estimates the rate of induction of dominants, though by not more than two orders of magnitude (paragraphs 149, 150).

264. As discussed in paragraph 9, the part of the human genome under discussion, namely that responsible for some fifty dominant traits most commonly observed and easily detected, consists of at least fifty loci and is unlikely to consist of as many as 500. Multiplying the assumed number of loci by the rate of induction discussed in the previous paragraph gives a total rate of induction ranging from 5×10^{-5} to 5×10^{-8} mutations per gamete per roentgen depending upon the assumptions concerning the number of loci involved and the proportion of dominant mutations induced.

265. Assuming full penetrance, the damage thus estimated will become apparent in the offspring of irradiated subjects and, because of the reduction of fitness that it entails (paragraph 11), will, on the average, persist in the population for some twenty-five generations. The genes responsible for those traits that more drastically impair fitness will be eliminated in the first generation, whereas the mildest ones will persist for a very long time. Under conditions of steady irradiation for several generations, the frequency of the induced traits in the population would build up to a value equal to the rate of induction.

Effects of cell stage and type of irradiation

266. As mentioned in paragraph 248, all mouse data used for numerical estimates have been derived from experiments in which mouse spermatogonia were irradiated with high, unfractionated doses of acute, low LET radiation. However, it needs to be emphasized that the final yield of mutations has proved to be different when the germ cells of mice are irradiated with (*a*) low doses, (*b*) fractionated doses, (*c*) chronic radiation, and (*d*) high LET radiation.

267. As has been discussed in part III of this annex, experimental results in a number of species show that matters may differ considerably when other germ cells are irradiated. On the basis of results ob-

tained at seven specific loci in the mouse, acute x-ray irradiation of oöcytes at high doses yields more mutations per unit dose than acute irradiation of spermatogonia. Although the rate of induction in oöcytes is known with little precision, data suggest that it may be twice as high as in spermatogonia. When individuals of both sexes are irradiated, the total number of mutations induced will therefore be about 50 per cent higher than if oöcytes had the same sensitivity to radiation as spermatogonia.

268. Preliminary results indicate that in oöcytes the yield of specific locus mutations per unit dose after 50 R acute x rays (paragraph 170) is significantly lower than would be expected from results of irradiation at higher doses. It seems therefore that low doses of radiation are relatively less mutagenic than high doses of radiation, at least in oöcytes. Since human populations are more commonly exposed to low than to high radiation doses, it might well be that the estimates of genetic risks which are presently made will eventually prove to be too high.

269. Experiments with spermatogonia and oöcytes have shown that chronic radiation is mutagenically less effective than acute radiation. Under conditions of chronic irradiation of spermatogonia the yield of mutations per unit dose at rates of about 1 R per minute or less is about one-fourth of that at 90 R per minute (paragraph 156). With oöcytes, the reduction of the mutation yield is even more pronounced (paragraph 157). When both sexes are exposed to low dose-rate x or gamma radiation, the over-all yield of mutations can therefore be expected to be between one-eighth and one-fourth of that expected when the same population is exposed to high dose-rate radiation. Preliminary data indicate that a small dose-rate effect obtains with low doses of neutrons in mice oöcytes but not in spermatogonia (paragraphs 187, 196). More detailed information is needed before this dose-rate phenomenon with neutrons can be taken into account.

270. Results of new fractionation experiments (paragraph 175), in which the total radiation exposure is partitioned into small acute exposures of 50 R, indicate that this type of fractionation procedure yields mutation frequencies which are below those obtained with single, unfractionated procedures. Although mutation frequencies in the fractionated and unfractionated series differ by less than one order of magnitude, it is thought that this effect may be of importance for the estimation of human genetic hazards, because the fractionation procedures used are similar to those used in some medical practices.

271. Results of investigations at seven specific loci in spermatogonia show that low doses (up to about 100 rads) of acute or chronic fast fission neutrons are mutagenically more effective than x and gamma rays, suggesting an RBE of five for acute irradiation and of twenty for chronic irradiation (paragraph 188). Since human populations are usually exposed to low doses given at low dose rates, it seems that in spermatogonia the rate of induction of mutations per unit dose of neutrons may be some twenty times higher than the corresponding rate for x or gamma rays.

272. The final yield of mutations is not only affected by factors associated with radiation procedures but also by biological factors. One of the latter factors has recently been discovered and may have an important bearing on the estimation of genetic risks from irradiation of germ cells of females. Experiments with female mice have shown that the interval between irradiation and conception has a very pronounced effect on the mutation frequency observed in the offspring (paragraph 182). The frequency obtained after irradiation of females with low doses of neutrons is high in the first few weeks after irradiation, but, after that period, drops to a very low value, in fact zero in the sample size studied so far. Similar results have been obtained with x rays. There is a possibility of a similar effect in man and, therefore, an indication that the genetic radiation hazard from the exposure of women may, on the average, be less than that calculated on the basis of female mouse mutation rates obtained in the early time interval after irradiation.

Chromosome anomalies

273. The estimation of risks of induction of chromosome mutations can only be made on grounds as tenuous as those on which the estimates of risks of induced point mutations are based. While with regard to the induction of point mutations detailed and reliable quantitative information from *Drosophila* and from the mouse can be used, no comparable amount of data is available concerning the induction of chromosome anomalies. But the induction of point mutation is not borne out by direct observations in man, and the corresponding quantitative relationships between dose and effect are unknown. To estimate risks of induction of point mutations in man, a very major step is therefore necessarily involved in extrapolating from the experimental animals to our own species.

274. With regard to the induction of chromosome anomalies, on the other hand, there is clear evidence that a number of them can be induced by radiation in human cells *in vitro*. Preliminary observations suggest that some can be radiation-induced *in vivo* in germ cells. However, information on rates of induction *in vivo* in man is absent, and that obtained from human peripheral blood cells irradiated *in vitro* must be supplemented with observations in experimental animals.

275. Inferences regarding the induction of chromosome anomalies in our species based on animal material are especially open to criticism, inasmuch as the radiation sensitivity of chromosomes is known to change from one species to another. Thus, there is some evidence that human somatic cells might be more sensitive to the induction of chromosome anomalies by radiation than those of mice. Likewise, extrapolations from *in vitro* studies of human cells can also be quite misleading because of the known dependency of chromosome sensitivity on a number of factors associated with the stage and metabolism of the irradiated cells.

276. It was shown in part II of the present review that constitutional chromosome anomalies are responsible for a large part of the defects of genetic origin carried by human populations. Most of the anomalies are eliminated either pre- or post-natally in the generation immediately following the one in which they have arisen, and are associated with very severe hardship. Some, however, notably translocations, can be transmitted for a number of generations and are also the cause of serious harm to those who carry them in the unbalanced state.

277. Only for some types of chromosome anomalies can tentative risk estimates be obtained. These will be discussed in the following paragraphs. The estimates apply to a minor fraction of the total spontaneous chromosome damage detectable in the population. No estimate of the over-all risk of induction of chromosome

anomalies can be obtained in the current stage of our knowledge.

Changes in chromosome numbers

278. Experimental results indicate that in *Drosophila* the frequency of sex-chromosome loss rises linearly with dose below 1,000 R (paragraph 82). The rate of induced loss per pre-meiotic cell is very close to that obtained from irradiation of mouse spermatocytes at 200 R—between one and four chromosomes per 100,000 cells per roentgen. A comparable figure for non-disjunction cannot be obtained, because in that case the dose-effect relationship as observed in *Drosophila* is more complicated.

279. The possible importance of the induced sex-chromosome loss in man becomes apparent when it is recalled that XO karyotypes have been identified in 5 per cent of a sample of aborted foetuses and may therefore be responsible for a sizable proportion of spontaneous miscarriages. However, the possibility that at least part of the observed losses may have occurred after fertilization cannot be excluded.

280. No estimate of risk can, in the present stage of our knowledge, be obtained for the induction of losses or additions of autosomes. Some still inconclusive evidence, indicating that they may be induced by radiation in man, was mentioned in paragraph 66.

Translocations

281. Translocations in experimental animals are associated with, and frequently recognized through, the incidence of semi-sterility. In man, semi-sterility is a hardly applicable criterion, since the family-size usually falls very short of the natural fecundity of the species. The importance of translocations in human populations lies, therefore, much more in the suffering that they involve for those who receive them in the unbalanced state than in the effect they may have on the fertility of carriers of balanced translocations.

282. The estimation of risks of induction of translocations in man may be approached either from results obtained in mice or from results obtained in human cells *in vitro*. As discussed in part III (paragraph 116), from the incidence of semi-sterility in mice irradiated with 1,200 R of x rays, the number of induced translocations has been estimated to be approximately 14.8×10^{-2} per pre-meiotic cell. The estimate is based on the assumption that translocations are not further transmitted unless they are balanced, that no selection takes place between normal cells and cells carrying a balanced translocation, and that non-disjunction does not bias the observed frequencies of the translocations that are recovered.

283. In this connexion, it must be borne in mind that some of these assumptions may not strictly apply to man, since in our species the association of translocations with trisomies 21 does occur with a frequency of about 5×10^{-5} of all live-born children (paragraph 42), and the viability of cells carrying balanced translocations may, in fact, be different in mice, since the spontaneous frequency of translocations seems to be lower than in man.

284. The use of cell cultures to estimate the frequency of radiation-induced translocations is also far from being free from objections. For example, it is not possible to determine directly the rate of induction of translocations by radiation, because, even if the karyotype of each scored cell were established, present techniques would not make it possible to detect those translocations that involved small quantities of chromosome material or fragments of equal size. Finally, *in vitro* observations are available only on somatic cells, and it does not necessarily follow that, if the anomalies that were observed *in vitro* occurred in pre-meiotic cells *in vivo*, they would be transmitted to a viable gamete, as is indicated by the fact that haplo-21 zygotes appear not to be viable, whereas haplo-21 somatic cells are.[113]

285. Rather than determine the frequency of translocations *in vitro*, most authors have therefore assessed the frequency of breaks, dicentrics and ring chromosomes. Breaks are events whose frequency rises linearly with dose, whereas the frequency of dicentrics and ring chromosomes, like that of translocations, at least when induced by x rays, is proportional to the square of the dose at low doses and to its 1.5th power at high doses. At very low doses, the effect may be proportional to the first power of the dose.

286. The number of dicentrics and of ring chromosomes obtained through irradiation of blood cells at exposures between 50 and 200 R is 0.52×10^{-5} per cell per roentgen squared of which 0.45×10^{-5} are dicentrics.[187] The rate of 0.27×10^{-5} dicentrics per cell per roentgen squared was also obtained,[380] but it is perhaps less relevant because it is based on observations at exposures ranging between limits too far apart (25 to 1,200 R).

287. If it is assumed that translocations on one side and rings and dicentrics on the other are induced at the same rate, and that rates at high doses increase with the 1.5th power rather than with the square of the dose, the expected translocation rate after 1,200 R based on *in vitro* data is approximately 21×10^{-2} translocations per cell (or 18×10^{-2} if only the results on dicentrics are taken into account). This rate is fairly close to that deduced from semi-sterility data in mouse spermatogonia. Under the same reservations as were formulated for that case, the rate of transmission of translocations through the gametes would be four to six times less.

288. The rate of induction of translocations is known to be highly dependent on the rate of delivery of radiation. The estimates of rates of induction discussed in the previous paragraphs refer to acute irradiation. The actual rates under chronic irradiation may be considerably lower, as indicated by the mouse data discussed in paragraphs 118 and 124.

Deletions

289. Estimates of rates of induction of deletions in human germ cells are not available, but an idea of the possible magnitude of the risk of induction of certain clinically significant deletions can be obtained on the basis of the rates of induction of deletions by radiation in human cells *in vitro*. Induced rates *in vitro* are probably reliable, since they are consistent with scantier observations on peripheral cells of subjects irradiated accidentally *in vivo*.[154]

290. It is not known whether one single break is sufficient to bring about a stable "terminal" deletion or whether, in fact, an additional break is required to make it possible for the telomere to attach itself to the centric fragment. The linear rise of the frequency of terminal deletions *in vitro* (paragraph 69) speaks in favour of the one-hit theory.

291. To obtain estimates of the risk of induction of given syndromes due to terminal deletions, it is necessary to know the size of the fragments whose loss is responsible for each syndrome. As mentioned in paragraphs 39 and 40, the following terminal deletions are known to be associated with clinical syndromes severely detrimental but compatible with survival: deletion of part of the short arm of chromosome 5 (*cri du chat* syndrome), of the short arm and of the long arm of 18, and of the short and of the long arm of the X chromosome. It is not known whether any other deletion, be it terminal or interstitial, is compatible with survival nor to what sort of detriment it may be associated.

292. In the *cri du chat* syndrome, the size of the target, i.e., the length of the segment of chromosome 5 where a break must occur to produce the required deletion, amounts to over 50 per cent of the short arm of this chromosome or to about 1 per cent of the length of the diploid chromosome complement. This has been estimated[387] by studying the variations in length of the residual fragment of the short arm of chromosome 5 in the known cases of *cri du chat* syndrome.

293. Observations[157] on blood cells irradiated *in vitro* have shown that x rays induce 1.1×10^{-3} deletions per cell per roentgen. If a single break were enough to bring about the *cri du chat* syndrome, the deletion would be expected to occur with a frequency of $1.1 \times 10^{-3} \times 10^{-2} = 1.1 \times 10^{-5}$ per cell per roentgen. If two breaks were required, the expected frequency would be lower than the square of this (i.e., 1.2×10^{-10} per cell per roentgen squared).

294. Similar estimates can be obtained for the other deletions mentioned previously. Deletions of part of the short and of the long arm of chromosome 18 compatible with survival involve 0.25 and 1 per cent of the length of the diploid chromosome complement, respectively, leading to estimates of 0.3×10^{-5} and 1.1×10^{-5} deletions per cell per roentgen in the case of single events, and of 0.8×10^{-10} and 1.2×10^{-10} deletions if two breaks are required. Likewise, deletions of the short and of the long arm of the X chromosome, involving 3 and 4 per cent of the complement length, respectively, would occur with probabilities of 3.3×10^{-5} and 4.4×10^{-5} deletions per cell per roentgen if one break was required, and 11.0×10^{-10} and 19.0×10^{-10} deletions per cell per roentgen squared otherwise.

295. Nothing is known about the selection that deletions arising in germ cells might undergo. It is conceivable that a fraction of those that radiation may induce would be eliminated sometime before birth or perhaps before fertilization. Neither human nor experimental data are available which would make it possible to assess the extent of the elimination.

V. Conclusions

296. The estimates given in the preceding paragraphs must be examined in the light of the practical value they have in assessing the detriment that will result from exposure of human populations to any source of radiation. For that purpose, risk estimates must, ideally, be comprehensive, therefore taking into account all major genetic effects of social, rather than merely biological, import. If this did not prove possible, it would still be valuable to know the range in which the over-all risk estimate lay or even an upper limit to the estimate.

297. Even taken together, the estimates given earlier do not meet these requirements. The risk of induction of dominant mutations (paragraphs 261-265) applies to those major and easily recognizable traits that are clearly undesirable from the individual and social points of view. These traits are frequently observed in all known populations. That damage would always be a minor fraction of the over-all damage due to point mutations, though a particularly conspicuous one, both because of its immediate manifestation and its persistence for a number of generations, and because of the nature of the detriment to which the risk estimate applied.

298. An approach to the estimation of damage from induced dominant mutations could in the future result from the application to man of the observed frequencies of skeletal defects in first generation irradiated mice (paragraphs 151-155). However, it is not certain that comparable rates of induction apply at low doses.

299. The over-all risk of induction of all point mutations, which are all assumed to have some degree of dominance and to be eliminated predominantly in heterozygotes (paragraphs 253-260), includes the risk of induction of dominant mutations discussed in paragraph 297. One major practical limitation of the over-all risk estimate is due to the fact that the damage that is thus assessed is expressed in terms of loss of mutants through generations. This loss has a clear biological meaning, and has an undesirable character for the individual and for society. However, we do not know how many of the harmful mutations induced by radiation will at some point be eliminated through, say, loss of a zygote before implantation—an event which is not usually detectable in man—rather than through drastic reduction of fertility, miscarriages or serious genetic defects. But the estimate does, at any rate, provide the required upper limit to the damage due to point mutations.

300. This is, however, only part of the induced damage, since it does not include that due to chromosome anomalies. At present, we have no way of estimating the over-all risk of induction of chromosome anomalies. Their high frequency in human populations makes it likely that such a risk may not be negligible. We only have estimates of the induction of sex-chromosome loss (paragraphs 278-280), of translocations (paragraphs 281-288) and of those deletions that are known to be associated with severe clinical syndromes (paragraphs 289-295). The total damage from induced chromosome anomalies is likely to be higher, but our present knowledge is inadequate even to guess its possible magnitude, while such partial estimates as we have discussed are based on assumptions that make conclusions conjectural or, at best, very tentative indeed.

301. In considering the significance of radiation damage to the genetic material, it may be of interest to compare it with the rate of naturally-occurring genetic changes. It was estimated in the report that, on the average, a total of 140 point mutations arose spontaneously in 1,000 gametes in each generation and that under conditions of acute irradiation at high doses one rad induced a total of two mutations per 1,000 gametes. Thus a dose of one rad per generation would add about one-seventieth to the total number of mutations arising spontaneously per generation. To this point mutational damage must be added that due to chromosome anomalies which occur spontaneously in 1 per cent of live-born children. It is not possible at present to estimate

the over-all rate of induction of these anomalies by radiation, but the rate is expected to be very low at low doses.

302. Since neither a comprehensive estimate of the genetic risk, nor an upper limit to that estimate is available, the assessment of genetic damage from main sources of radiation must still be made by means of comparative risks. This is possible only at low doses and dose rates, in so far as linearity of the dose-effect relationship can be accepted as a computational approximation even for those effects that take place as a consequence of more than one event. No such approximation is allowed at high doses and dose rates, and even comparative risks cannot be determined for them.

TABLE I. MAIN TYPES OF SEX-CHROMOSOME MOSAICS OBSERVED IN MAN[30–32, 35–37]

A. *Without structural anomalies of the X chromosome*	
(1) In Klinefelter's and related syndromes ..	XY/XXY
	XX/XXY
	XXXY/XXXXY
	XXXX/XXXXY
	XXY/XXXXY/XXXXXY
	XO/XY/XXY
(2) In Turner's and related syndromes	XO/XX
	XO/XXX
	XO/XX/XXX
	XO/XYY
(3) In the XXX syndrome	XX/XXX
(4) In other syndromes ...	XO/XY
	XY/XXXY
	XX/XXY/XXYYY
	XX/XY
B. *With structural sex-chromosome anomalies*	
	XO/XX_{DL}
	XX/XX_{DL}
	XX/XX_{DS}
	XO/XX_{DS}
	XO/XX_R and $XO/XX_R/XX_RX_R$
	$XO/X_{iso}X$ and $XO/X_{iso}X/X_{iso}X_{iso}X$
	$XO/X_{iso}Y/X_{iso}Y/Y_{DL}$
	$XO/XY_{DL}/XXY_{DL}$
	$XO/XY/XY_{DL}$
	$X_{iso}X/X_{iso}X_{iso}Y$

TABLE II. FREQUENCY OF TRANSLOCATIONS AMONG TRISOMICS 21

	Observed cases	*Per cent frequency*	*References*
Heavily biased samples ..	(13/110)	11.8	30
	(18/227)	7.9	40
	(3/41)	7	41
Less biased samples	(5/101)	5	389
	(25/652)	4	30
Samples with little bias ..	(1/58)	1.7	43
	(4/203)	2	390
	(2/96)	2.1	44
	(6/127)	4.7	388

TABLE III. FREQUENCY OF ANOMALIES IN SEX-CHROMOSOME NUMBER IN SELECTED POPULATIONS

	Observed cases	*Frequencies per 1,000*	*References*
Feeble-minded			
Chromatin-positive males..	(29/3,306)	8.77	2
	(70/7,358)	9.51	2
Double-positive females ..	(12/2,689)	4.46	2
Chromatin-negative females	(1/2,689)	0.37	2
Criminals			
Chromatin-positive males..	(15/760)	20	59
XYY males	(7/197)	35	60
Sterile subjects			
Chromatin-positive males..		30	2
Females with sex-chromosome anomalies		280	2
Childless males with sex-chromosome anomalies..	(8/130)	62	58
Females with small stature			
Chromatin-positive females		73	61

TABLE IV. FREQUENCY OF SOME COMMON CHROMOSOME ANOMALIES

	Observed cases	*Frequency per 1,000*	*References*
A. Individual anomalies among live-born children			
Trisomy 21	1,522/1,022,042	1.5	2, 39
Trisomy 13	2/10,345	0.2 (0.021–0.69)[a]	51
Trisomy 18	3/10,345	0.3 (0.058–0.85)	51
Cri du chat syndrome		> 0.2[b]	
Klinefelter's and related syndromes	31/18,147	1.7 (1.16–2.98)	62–66
XXX syndrome	12/10,000	1.2 (0.62–2.1)	64
Turner's syndrome	5/16,920	0.29 (0.095–0.69)	62–66
B. Over-all frequency of anomalies in spontaneous abortions	44/200	220 (165–284)	82
C. Frequency of structural changes in an unselected adult population		5	56, 57

[a] 95 per cent confidence limits.

[b] Higher than frequency of trisomy 13.

TABLE V. RATES OF INDUCTION OF CHROMOSOME ANOMALIES BY ACUTE IRRADIATION OF BLOOD CELLS

		References
X rays		
Chromatid breaks per cell per roentgen	0.26×10^{-2}	154
Chromosome breaks per cell per roentgen	0.24×10^{-2}	154
	0.39×10^{-2}	155
	0.69×10^{-2}	156
Deletions per cell per roentgen	$0.11 \times 10^{-2} \pm 0.012$	157
Dicentrics per cell per roentgen squared	$0.45 \times 10^{-5} \pm 0.07$	157
	$0.27 \times 10^{-5} \pm 0.014$	386
Dicentrics per cell per rad squared	0.09×10^{-5}	156
14 MeV neutrons		
Deletions per cell per rad	$0.23 \times 10^{-2} \pm 0.022$	161
Ring and dicentrics per cell per rad squared	$0.81 \times 10^{-2} \pm 0.06$	161
25 MeV neutrons		
Deletions per cell per rad	0.26×10^{-2}	162
Fission neutrons		
Deletions per cell per rad	0.45×10^{-2}	162

TABLE VI. FREQUENCY OF SPONTANEOUS AND RADIATION-INDUCED SEX-CHROMOSOME ANOMALIES IN MALE AND FEMALE GERM CELL STAGES IN THE MOUSE

Irradiated germ cell stage	*Exposure (R)*	*Animals classified*	*Spontaneous frequency (per cent) XMO*[a]	*Adjusted*[b] *induced frequency per R per* 10^5			*References*
				Loss[c] *of XM*	*Loss of XP or Y*	*XXY*	
FEMALE GERM CELLS							
Prophase primary oöcyte (irradiated foetuses and newborn, 13½-20½ days post-conception)	150–250 (weighted mean, 220.5)	2,402		1.51	—	0	179
CONTROLS		785	0.0				
Dictyate (irradiated adults; ovulations 1-32 days post-irradiation)	100–400 (weighted mean, 342.0)	331		3.34	—	0	
MALE GERM CELLS							
Spermatocytes (mating 36-42 days post-irradiation)	200	1,508		—	1.6	0	
Spermatocytes (mating 29-35 days post-irradiation)	200	2,370		—	3.6	0.4	177
Spermatocytes (post-pachytene, 22-28 days post-irradiation)	200	1,752		—	0.7	0	
CONTROLS	—	3,059	0.06				
Spermatids (mating 15-21 days post-irradiation)	200	1,656		—	5.7	—	177
CONTROLS	—	1,299	0.13				
Spermatozoa (vas and epididymis)	600	1,112		—	2.0	—	177
CONTROLS	—	1,285	0.14				
PRONUCLEUS STAGES							
♀ and ♂ early pronucleus	100	422		19.0	23.2	0	177
	100	227		17.6	12.5	0	178
♀ and ♂ mid-pronucleus	100	193		0	0	0	178
	200	70		0	2.1	0	177
CONTROLS	—	822	0.97				177
	—	196	0.51				178

[a] XMO was the only spontaneous sex-chromosome abnormality observed in controls. OXP and XXX occur spontaneously with very low frequencies.[173]

[b] Irradiated minus control frequency. In the case of XM loss, frequency is calculated by taking account of the fact that OY is lethal.

[c] Loss of entire chromosome as well as a few cases of deficiency.

TABLE VII. DOMINANT LETHALS IN SPERMATOGONIA OF MICE

Exposure (R)	*Rate of delivery (R/min)*	*Induced pre-implantation losses*[a]	*Induced post-implantation losses*[b]	*Total rate of induction of dominant lethals*[c]	*Reference*
0		—	—	—	196
550	69	—	0.02	0.02	
0		—	—	—	391, 392
300	100	0.06[d]	0	0.04[d]	
0		—	—	—	195
200	100	0.01	0	0.01	
0		—	—		198
600 + 600 8 weeks apart	217	0.01		0.11	
0		—	—		194
1,200	0.017	0.02		0.02	
0		—	—	—	197
275	75	—	0.04	—	
55 × 5[e]	75	—	—	—	

[a] $1 - \frac{\text{implanted embryos/}corpora\ lutea\text{ in irradiated series}}{\text{implanted embryos/}corpora\ lutea\text{ in control series}}$.

[b] $1 - \frac{\text{live embryos/total number of implants in irradiated series}}{\text{live embryos/total number of implants in control series}}$.

[c] $1 - \frac{\text{live embryos/}corpora\ lutea\text{ in irradiated series}}{\text{live embryos/}corpora\ lutea\text{ in control series}}$.

[d] Significantly higher than controls.

[e] 5 R per day for fifty-five consecutive days.

TABLE VIII. INDUCTION OF DOMINANT LETHALS IN GERM CELLS OF MALE MICE

Exposure (R)	*Weeks after irradiation*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*References*
300	Total rate of induction of dominant lethals[a]	0.21	0.15	0.38	0.28	0.28	0.24	0.05	0.04	391, 392
300	Post-implantation death[b]	0.19	0.01	0.27	0.08	0.05	—	—	—	
200	Total rate of induction of dominant lethals[a]	0.23	0.13	0.42	0.30	0.40	0.55	0.44	0.01	195
200	Post-implantation death[b]	0.20	0.12	0.32	0.13	0.21	0.09	0.13	—	

[a] $1 - \frac{\text{live embryos/}corpora\ lutea\text{ in irradiated series}}{\text{live embryos/}corpora\ lutea\text{ in control series}}$. [b] $1 - \frac{\text{live embryos/total number of implants in irradiated series}}{\text{live embryos/total number of implants in control series}}$.

TABLE IX. RATE OF INDUCTION OF DOMINANT LETHALS IN POST-MEIOTIC CELLS OF VARIOUS MAMMALS

Mammal	*Exposure (R)*	*Corpora lutea*	*Per cent induced pre-implantation losses*	*Per cent induced post-implantation losses*	*Total rate of induction of dominant lethals*	*References*
Mouse[a]	0	387	—	—	—	208
	400	248	13	40	48	
	670	178	28	51	65.6	
Mouse[b]	0	1,244	—	—	—	391, 392
	400	1,029	15	21	33	
	500	1,052	16.6	35	45	
	600	1,120	18	41	51	
	700	1,165	23	45	58	
Rat[a]	0	758	—	—	—	208
	400	419	13	49	55	
	670	403	29	61	72	
Guinea pig[c]	0	59	—	—	—	393
	500	95	2	43	44	
Guinea pig[a]	0	69	—	—	—	209
	300	47	—	13	10	
	450	46	—	28	25	
	700	40	—	50	45	
Rabbit[a]	0	203	—	—	—	208
	450	214	65	18	71	
	600	195	63	17	69	
Rabbit[c]	0	32	—	—	—	393
	500	105	49	16	57	

[a] Matings within 3 days after irradiation.
[b] Matings within 4 weeks after irradiation.
[c] Matings immediately after irradiation.

TABLE X. DOMINANT LETHALS IN OÖCYTES OF MICE[215]

Exposure (R)	Oöcyte stage	Corpora lutea	A	B
	Control	668	—	—
100	Dictyate	250	0.20	0.02
100	Late prophase I	217	— 0.07	— 0.09
100	Metaphase I	266	0.43	0.35
100	Anaphase I	184	0.37	0.20
100	Metaphase II	227	0.29	0.22
100	Pronucleus stage before or during DNA synthesis	149	0.11	0.05
	Control	668	—	—
200	Dictyate	279	0.11	0.14
200	Late prophase I	309	0.38	0.36
200	Metaphase I	185	0.74	0.75
200	Anaphase I	193	0.69	0.68
200	Metaphase II	274	0.57	0.55
200	Pronucleus stage before or during DNA synthesis	130	0.15	0.21

A—total rate of induction (see footnote [a] to table VIII).
B—induced post-implantation deaths (see footnote [b] to table VIII).

TABLE XI. TRANSLOCATIONS IN SPERMATOGONIA OF MICE

Experiment	Exposure (R)	Rate of delivery (R/min)	F_1 Males	Per cent semi-sterile	F_1 Females	Per cent semi-sterile	Total F_1	Per cent semi-sterile	Checked cyto-logically	References
1	0		427	0.2	109	0	536	0.2	Yes	
	600 + 600	217	427	3.5 (1.7–5)[a]	104	6.7	531	4.1 (2.4–5.8)		198, 223
2	0		216	0.0					Yes	
	1,200	0.017	214	0.9						194
3	0		?	?					Yes	
	700	?	1,010	0.5 (0.02–1.6)						224
4	0		80	0.0	25	0.0			No	
	550	69	80	0.0	25	0.0				196
5	1,092	Acute	110	2.7 (0.6–8.7)					No	225
6	0						1,037	0.0	Partly	226
	350	Acute					452	2.2 (1.1–4.0)	Partly	
	700	Acute					444	3.2 (1.4–4.8)	Partly	
	1,000	Acute					238	2.5 (0.9–5.4)	Partly	
7	0		112	0.0						197
	275	75	112	0.0						
	55 × 5[b]	75	112	0.9					No	

[a] 95 per cent confidence limits.
[b] 5 R per day for fifty-five consecutive days.

TABLE XII. NATURAL AND INDUCED MUTATION RATES AT SEVEN SPECIFIC LOCI IN ADULT MOUSE SPERMATOGONIA AND OÖCYTES

Source	Total exposure (R)	Rate of delivery (R/min)	Number of offspring tested	Number of mutations observed	Mutations per locus per gamete × 10^5	Reference
			SPERMATOGONIA			
X ray	300	80–90	65,548 (40,408)[e]	40 (25)	8.7 (8.8)	237
X ray	600	80–90	119,326	111	13.3	237
X ray	1,000	80–90	31,815	23	10.3	394
X ray	600	60–70	10,761	11	14.6	395
Co^{60} x ray	600	24	44,352	33	10.6	396
X ray	600	9	40,326 (28,339)[e]	23 (14)	8.1 (7.1)	237
Cs^{137}	600	0.8	28,059 (27,840)[e]	10 (10)	5.1 (5.1)	237
Cs^{137}	300	0.009	58,457	10	2.4	237
Cs^{137}	516	0.009	26,325	5	2.7	237
Cs^{137}	861	0.009	24,281	12	7.1	237
Co^{60}	603	0.007–0.009	10,763	2	2.7	395
Co^{60}	609	0.005	58,795	16	3.9	288
Co^{60} and radium	37.5	0.0011–0.0078	63,322	6	1.4	247
Cs^{137}	86	0.001	59,810 (56,993)[e]	6 (6)	1.4 (1.5)	237
Cs^{137}	300	0.001	49,569[f]	15	4.3	237
Cs^{137}	600	0.001	31,652[f]	13	5.9	237
Fission neutrons[a]	307	0.002–0.003	41,875[f]	67	22.9	288
Fission neutrons	207	55–60	39,028[f]	8	2.9	287, 397
Fission neutrons	104	0.001	39,083[f]	27	9.9	287, 397
Fission neutrons	101	0.13	19,506[f]	20	14.6	237
Fission neutrons	63	0.17	18,194[f]	13	10.2	237
Fission neutrons	59	0.79	17,041[f]	12	10.1	237
Fission neutrons	59	79	16,758[f]	10	8.5	237
CONTROL	—	—	531,500 (544,897)[e]	28 (32)	0.8 (0.8)	237
			OÖCYTES			
X ray	400	90	11,124 (12,853)[b, e]	15 (16)	19.3 (17.8)	237
Cs^{137}	400	0.8	20,827 (36,083)[e]	7 (13)	4.8 (5.2)	237
Co^{60}	600	0.05	10,117	1	1.4	401
Cs^{137}	400	0.009	37,049[f]	2	0.8	237
Cs^{137}	258	0.009	27,174	1	0.5	237
Co^{60}	450	0.004	11,225[f]	0	0	397
X ray	50	81	127,391[c, f]	10	1.1	268, 269
X ray	50	81	54,621[d, f]	0	0	268, 269
Fission neutrons	63	79	43,000[c, f]	37	12.2	284
Fission neutrons	63	79	40,092[d, f]	0	0	284
Fission neutrons	63	0.17	46,301[c, f]	22	6.8	284
Fission neutrons	63	0.17	80,391[d, f]	0	0	284
Fission neutrons	104	0.001	12,058[f]	1	1.2	397
CONTROL	—	—	98,828	1	0.14	396
CONTROL	—	—	13,402	0	0	397

[a] Neutron doses in rads (gamma component is included).
[b] Includes data from an old experiment, which was later excluded.
[c] Oöcytes sampled up to first seven weeks after irradiation.
[d] Oöcytes sampled more than seven weeks after irradiation.
[e] Revised from the 1962 report.[2]
[f] New data.[237]

TABLE XIII. SPECIFIC LOCUS MUTATIONS IN EXPERIMENTAL ANIMALS

Species	Number of loci tested	Exposure (R)	Rate of delivery (R/min)	Spontaneous mutation rates at individual loci per generation × 10^6 induced mutation rates per locus per gamete per R × 10^6			
				Spermatogonia	Oögonia	Oöcytes	References
Mouse	7	0		752		144	2
		400	90			48	
		600	60–90	22			
Mouse	6	0		0			238
		600	88	5			
Drosophila	8	0					398
		900	85	1.5			
Dahlbominus ..	4	0			599		267, 399
		1,000	1,000		9		
		0				552	400
		1,000[b]	100			30	
		1,000	100			45	
		1,000	100			65	
Bombyx	Pe-locus[a]	0		8,900	9,200		266
		1,000	60–100	65	28		
		1,000	60–100	23	12		
		1,000	60–100	28	18		
	Re-locus[a]	0		0	8,800		
		1,000	60–100	32	24		
		1,000	60–100	9	5		
		1,000	60–100	6	12		
Mormoniella ..	5	0				717	311
		1,136	854			14	

[a] Cells irradiated on the seventh, eighth or ninth day after hatching of the larvae.
[b] Progressively older stages of oöcyte development.

TABLE XIV. SEX-LINKED RECESSIVE LETHALS IN SPERMATOGONIA OF *Drosophila* LARVAE[257, 258]

Total exposure (R)	Rate of delivery (R/min)	Number of offspring tested	Mutation frequency per cent
		ACUTE RADIATION	
0	—	25,650	0.30 (0.24–0.37)[a]
56	25	21,538	0.40 (0.33–0.49)
109	25	21,154	0.42 (0.34–0.52)
163	25	20,860	0.43 (0.53–0.36)
0	—	8,405	0.26 (0.18–0.39)
307	25	8,330	0.26 (0.18–0.39)
		CHRONIC RADIATION	
0	—	25,738	0.31 (0.25–0.39)
144	0.30	9,583	0.50 (0.38–0.66)
267	0.55	8,310	0.66 (0.51–0.86)
300	0.60	5,705	0.75 —
400	0.83	5,793	0.40 (0.27–0.60)
542	1.13	7,641	0.38 (0.27–0.55)

[a] 95 per cent confidence limits.

TABLE XV. INDUCTION OF SEX-LINKED RECESSIVE LETHALS IN OÖGONIA OF *Drosophila melanogaster* BY "4,000" R

(*Modified from Muller* et al.[263])

Rate of delivery (R/min)	*Room of irradiation*	*A*	*B*
7,333	Hot	1.6 ± 0.2	1.67 ± 0.23[a]
7,333	Hot	1.7 ± 0.15	2.07 ± 0.16
7,333	Hot	2.0 ± 0.15	2.21 ± 0.17
2; 1	Hot	1.7 ± 0.17	1.44 ± 0.18
1.7; 1	Dilution	1.0 ± 0.09	1.23 ± 0.11
0.2	Hot	0.86 ± 0.11	0.68 ± 0.09
0.2	Hot	1.0 ± 0.15	0.9 ± 0.14
0.1-0.2	Dilution	1.08 ± 0.11	1.28 ± 0.13
0.1-0.2	Dilution	1.4 ± 0.11	1.5 ± 0.11
0.1; 0.05; 0.02	Dilution	1.25 ± 0.15	1.42 ± 0.17
0.05; 0.02	Dilution	1.5 ± 0.5	1.7 ± 0.6

A — Percentage observed minus spontaneous lethals.
B — Percentage after correcting for results of sperm irradiation in females.
[a] Spermatid instead of spermatozoan frequency was used for obtaining correction factor.

TABLE XVI. MUTATION FREQUENCY IN THE MOUSE FROM SINGLE AND FRACTIONATED IRRADIATION

Cell stage	*Total exposure (R)*	*Exposure in each fraction (R)*	*Interval between fractions*	*Number of offspring*	*Mean number of mutations per locus per roentgen[a] × 10^8*	*References*
Spermatogonia	0	—	—	531,500	—	274
	300	—	—	65,548	26.6	274
	600	—	—	119,326	20.9	274
	1,000	—	—	44,649	8.5	274
	600	100 and 500	24 hours	24,811	39.1	274
	1,000	600 and 400	>15 weeks	4,904	28.4	274
	1,000	500	2 hours	14,879	10.8	253
	1,000	500	24 hours	11,164	49.2	274
	1,000	200	24 hours	8,588	25.9	253
	1,000	200	1 week	10,968	18.8	274
Oöcytes	200	—	—	37,297	40.2	274
	400	—	—	12,853	44.5	274
	400	200	24 hours	6,086	52.8	274

[a] Mutation rate in oöcytes is not adjusted for the control value. Spontaneous mutation rate in females is not accurately known.

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Annex D

LIST OF REPORTS RECEIVED BY THE COMMITTEE

1. This annex lists reports received by the Committee from Governments and agencies of the United Nations between 10 July 1964 and 7 June 1966.

2. No documents were issued under symbols A/AC.82/G/L.1060 and A/AC.82/G/L.1061.

3. Reports received by the Committee before 10 July 1964 were listed in annex I of its first report, annex J of its second and annex C of its third.

Document No.	Country and title
A/AC.82/G/L.	
	JAPAN
949	Radioactive contamination of rice in Japan with reference to Sr-90 and Cs-137 content in rice until 1962.
950	Radioactive contamination of milk in Japan (1961-1963).
951	Japanese dietary habits and the fallout problem—II.
952	External doses of radiation from fallout in Tokyo and its vicinity.
953	Recent variation of atmospheric contents of C-14 in Tokyo and transfer problem of atmospheric carbon dioxide.
954	Assay of strontium-90 in human bone in Japan, 1961-1963.
	AUSTRALIA
955	Strontium-90 in the Australian environment during 1962.
	INDIA
956	Measurements on the environmental radioactivity in India from nuclear weapon tests data collected during 1962-63.
	UNITED STATES OF AMERICA
957	Radiological Health Data, volume V, No. 6, June 1964.
958	Fallout program quarterly summary report, July 1, 1964. HASL-146.
959	HASL contributions to the study of fallout in food chains, July 1, 1964. HASL-147.
960	Spectrometric determination of dose rates from natural and fallout gamma-radiation in the United States, 1962-63.
961	Radiological Health Data, volume V, No. 7, July 1964.
	UNITED STATES OF AMERICA
962	Radiological Health Data, volume V, No. 8, August 1964.
963	Terrestrial and freshwater radioecology. A selected bibliography—Supplement 2.
	UNITED KINGDOM
964	Annual report 1963-64. ARCRL-12.
	UNITED STATES OF AMERICA
965	Farming practices and concentrations of fission products in milk.
966	Background material for the development of radiation protection standards. FRC report No. 5.
967	Radiation protection guidance for Federal agencies.
	SWEDEN
968	Correlations between Cs^{137} fallout rates, food levels and body burdens.

Document No.	Country and title
A/AC.82/G/L.	
	FRANCE
969	Contribution à l'étude de la radioactivité du milieu ambiant pendant les années 1960 à 1963, dans la région de Paris.
	UNITED STATES OF AMERICA
970	Radiological Health Data, volume V, No. 9, September 1964.
971	Health and Safety Laboratory fallout program quarterly summary report, October 1, 1964. HASL-149.
972	Ichiban: The dosimetry program for nuclear bomb survivors of Hiroshima and Nagasaki—A status report as of April 1, 1964. CEX-64.3.
	UNITED KINGDOM
973	Assay of strontium-90 in human bone in the United Kingdom; Results for 1963, Part II. MRC report No. 9.
	BELGIUM
974	La retombée radioactive mesurée à Mol, année 1962.
	SWEDEN
975	Chromosome investigations on the embryo progeny of male mice treated with ^{90}Sr.
	UNITED STATES OF AMERICA
976	Radiological Health Data, volume V, No. 10, October 1964.
977	Revised fallout estimates for 1964-1965 and verification of the 1963 predictions. FRC report No. 6.
978	Summary report on fission product radioactivity in the air along the 80th meridian (West), 1957-1962. NRL-6104.
	SWITZERLAND
979	7. Bericht der Eidg. Kommission zur Uberwachung der Radioaktivität zuhanden des Bundesrates für das Jahr 1963.
	UNITED STATES OF AMERICA
980	Radiological Health Data, volume V, No. 11, November 1964.
981	Filter pack technique for classifying radioactive aerosols by particle size. Part 2 —Isotopic fractionation with particle size.
	ITALY
982	Data on environmental radioactivity collected in Italy (January-June 1963). CRU/137/63
	FRANCE
983	Surveillance de la radioactivité, année 1961.
984	Surveillance de la radioactivité, année 1962.
985	Surveillance de la radioactivité, année 1963.

Document No.	Country and title
A/AC.82/G/L.	
	BELGIUM
986	Contrôle radiologique de l'environnement du C.E.N. à Mol.
987	La retombée radioactive mesurée à Mol. Rapport d'avancement, année 1963.
	UNITED STATES OF AMERICA
988	Radiological Health Data, volume V, No. 12, December 1964.
989	Flight data and results of radiochemical analyses of filter samples collected during 1961 and 1962 under Project Star Dust, January 1, 1965. HASL-153.
990	Fallout program quarterly summary report, January 1, 1965. HASL-155.
991	Study of adolescents exposed *in utero* to the atomic bomb, Nagasaki, Japan. 1. General aspects: clinical and laboratory data.
992	Radiological Health Data, volume 6, No. 1, January 1965.
993	The HASL surface air sampling program, summary report for 1963. HASL-156.
994	X-ray equipment survey in Polk County, Florida, September 1961-August 1963.
995	Procedure for determination of stable elements and radionuclides in environmental samples.
	UNITED KINGDOM
996	Radioactive fall-out in air and rain; results to the middle of 1964. AERE-R-4687.
	UNITED STATES OF AMERICA
997	Radiological Health Data, volume 6, No. 2, February 1965.
998	Atmospheric radioactivity and fallout research. TID-12616 (Rev.2).
	UNITED KINGDOM
999	Interim report: Radioactivity in milk, 1964. ARCRL-13.
	UNITED STATES OF AMERICA
1000	Radiological Health Data, volume 6, No. 3, March 1965.
1001	The central nervous system in leukemia.
1002	Fallout program quarterly summary report. HASL-158.
1003	Carbon-14 measurements in the atmosphere—1953 to 1964. HASL-159.
	UNITED KINGDOM
1004	Interim report: Radioactivity in milk, 1964. ARCRL-13.
	SWEDEN
1005	The occurrence of Cs^{137} in Swedish food, especially dairy milk, and in the human body after the nuclear test explosions in 1961 and 1962.

Document No.	Country and title
A/AC.82/G/L.	
	UNITED STATES OF AMERICA
1006	Malignant lymphoma in survivors of the atomic bomb in Hiroshima.
1007	Terrestrial and freshwater radioecology. A selected bibliography—Supplement 3. TID-3910 (Suppl.3).
	SWEDEN
1008	Genetic effects of supra-lethal X-ray treatment of male mice.
	UNITED KINGDOM
1009	Assay of strontium-90 in human bone in the United Kingdom. Results for 1964, Part I. MRC report No. 10.
	UNITED STATES OF AMERICA
1010	Radiological Health Data, volume 6, No. 4, April 1965.
	AUSTRALIA
1011	Strontium-90 in the Australian environment during 1963.
	UNITED STATES OF AMERICA
1012	Background material for the development of radiation protection standards. Protective action guides for strontium-89, strontium-90 and cesium-137. FRC report No. 7.
	UNITED STATES OF AMERICA
1013	Radiological Health Data, volume 6, No. 5, May 1965.
	ITALY
1014	Data on environmental radioactivity collected in Italy (July-December 1963). BIO/04/64.
	UNITED STATES OF AMERICA
1015	Fallout program quarterly summary report, July 1, 1965. HASL-161.
1016	Aging in Hiroshima atomic bomb survivors.
1017	Autopsy study of leukemia in Hiroshima.
1018	Radiological Health Data, volume 6, No. 6, June 1965.
1019	Measurement of the exposure of human populations to environmental radiation.
1020	Distribution of strontium-90 in surface air during 1963.
	INTERNATIONAL ATOMIC ENERGY AGENCY
1021	Panel on the molecular basis of radiosensitivity (IAEA reports PL-115/1-PL-115/12
	UNITED STATES OF AMERICA
1022	Medical uses of radium and radium substitutes.

Document No.	Country and title
A/AC.82/G/L.	
1023	Study of adolescents exposed *in utero* to the atomic bomb, Nagasaki, Japan. II. Growth and development.
	NORWAY
1024	Precipitation as a cause of seasonal and latitudinal variations in radioactive fall-out.
	UNION OF SOVIET SOCIALIST REPUBLICS
1025	Радиоактивные выпадения на территории СССР в 1963 году.
1026	Содержание стронция-90 в костной ткани населения Советского Союза в 1959-1963 годах.
1027	Характеристика радиоактивного загрязнения биологической цепочки мох-олень-человек на крайнем севере СССР в 1961-1964 годах.
	UNITED STATES OF AMERICA
1028	Offsite ecological research of the Division of Biology and Medicine—Terrestrial and Freshwater. TID-13358 (Rev.2).
1029	Radiological Health Data, volume 6, No. 7, July 1965.
1030	The HASL bone program 1961-1964. HASL-163.
1031	Behavior of certain radionuclides released into freshwater environments; annual report 1959-1960.
	CZECHOSLOVAKIA
1032	Relationship of radiosensitivity to the rate of growth.
	ARGENTINA
1033	Estudio de evolución de materiales radioactivos en el medio terrestre. Informe No. 133.
1034	Radioestroncio y estroncio estable en los huesos y dietas de los niños. Informe No. 149.
	UNITED STATES OF AMERICA
1035	Radiological Health Data, volume 6, No. 8, August 1965.
1036	Fallout program quarterly summary report, October 1, 1965. HASL-164.
	FOOD AND AGRICULTURE ORGANIZATION/ INTERNATIONAL ATOMIC ENERGY AGENCY
1037	Survey of radionuclides of natural origin in the soil-vegetation-human food chain.
	SWEDEN
1038	The effect on the length of life in the offspring of X-irradiated male mice.
	UNITED STATES OF AMERICA
1039	Radiological Health Data, volume 6, No. 9, September 1965.
1040	Iodine-131 in children's thyroids from environmental exposure.
	SWITZERLAND
1041	8. Bericht der Eidg. Kommission zur Uberwachung der Radioaktivität zuhanden des Bundesrates für das Jahr 1964.
	UNITED KINGDOM
1042	Annual report 1964-65. ARCRL 14.
1043	Assay of strontium-90 in human bone in the United Kingdom; Results for 1964, Part II. MRC report No. 11.
	JAPAN
1044	Radioactivity survey data in Japan, Number 5, November 1964.
	INTERNATIONAL ATOMIC ENERGY AGENCY
1045	Interim report on naturally occurring radionuclides in human tissues.
1045/ Rev1.	Report on naturally occurring radionuclides in human tissues.
	UNITED STATES OF AMERICA
1046	Natural environmental radioactivity. An annotated bibliography. WASH-1061.
1047	Radiological Health Data, volume 6, No. 10, October 1965.
1048	Project Springfield report. DASA-1517.
	AUSTRALIA
1049	Strontium-90 in the Australian environment, 1961 to 1963.
1050	Meteorological implications of measurements of strontium-90 in Australia.
	INDIA
1051	Measurements of airborne radioactive fallout in India. A.E.E.T.-208.
1052	Fission products data and its application in studying fallout from nuclear weapon tests. A.E.E.T.-209.
1053	Strontium-90 content of food samples in India. Data summary through 1963. A.E.E.T./A.M./40.
	UNITED KINGDOM
1054	Radioactive fallout in air and rain. Results to the middle of 1965. AERE-R 4997.
	UNITED STATES OF AMERICA
1055	Radiological Health Data, volume 6, No. 12, December 1965.
1056	Radiological Health Data, volume 6, No. 11, November 1965.
1057	Radioactive fallout from nuclear weapons tests.

Document No.	Country and title
A/AC.82/G/L.	
1058	Health and Safety Laboratory fallout program quarterly summary report, January 1, 1966. HASL-165.
1059	Carbon-14 measurements in the atmosphere. HASL-166.
	ITALY
1062	Data on environmental radioactivity collected in Italy, Jan.-June 1964. BIO/08/64.
	UNITED STATES OF AMERICA
1063	Late irradiation effects conference. WASH-1059.
	BELGIUM
1064	La retombée radioactive mesurée à Mol—Année 1964. R.2348.
	AUSTRALIA
1065	Concentration of caesium-137 in Australian milk during 1963.
1066	Concentration of caesium-137 in Australian milk during 1964.
	UNITED STATES OF AMERICA
1067	Radiological Health Data and Reports, volume 7, No. 1, January 1966.
1068	Atmospheric radioactivity in Antarctica, 1956-1963. NRL 6341.
	UNITED KINGDOM
1069	Interim report: Radioactivity in milk, 1965. ARCRL-15.
	DENMARK
1070	Sr-90 in human bone—Denmark 1962-1964.
	UNITED STATES OF AMERICA
1071	Flight data and results of radiochemical analyses of filter samples collected during 1963 under Project Stardust. HASL-168.
1072	Flight data and results of radiochemical analyses of filter samples collected during 1964 under Project Stardust. HASL-169.
	FOOD AND AGRICULTURE ORGANIZATION/ INTERNATIONAL ATOMIC ENERGY AGENCY
1073	Dietary levels of strontium-90, caesium-137 and iodine-131 for the years 1964-66.
	JAPAN
1074	Radioactivity survey data in Japan, Number 7, May 1965.
	UNITED STATES OF AMERICA
1075	Fallout program quarterly summary report, April 1, 1966. HASL-171.
1076	Radiological Health Data and Reports, volume 7, No. 2, February 1966.
	SWEDEN
1077	Placental transfer of strontium 85 in mice.

Document No.	Country and title
A/AC.82/G/L.	
	UNITED STATES OF AMERICA
1078	The global strontium 90 budget.
1079	Retained thorium dioxide media in seminal vesiculography.
	UNION OF SOVIET SOCIALIST REPUBLICS
1080	Сравнительный анализ глобальных выпадений продуктов ядерных взрывов на материки и океаны.
1081	К оценке общего количества стронция-90 в мировом океане.
1082	Основные черты глобального распределения стронция-90 на поверхности мирового океана (1960-1961 гг.).
1083	Поступление продуктов испытаний ядерного оружия населению Советского Союза с пищевым рационом и водой в 1963-1964 годах.
1084	Содержание стронция-90 в костной ткани населения СССР (материалы 1964 года).
1085	К вопросу о содержании цезия-137 в костной ткани человека.
1086	К вопросу о распределении стронция-90 в скелете взрослого человека.
1087	Исследование экстрагированных зубов как метод массового контроля за содержанием стронция-90 в организме людей.
1088	Некоторые гигиенические аспекты проблемы радия-226.
1089	Некоторые данные о зависимости между содержанием стронция-90 и цезия-137 в окружающей среде и организме людей.
1090	Поведение живых организмов в полях излучений.
1091	К вопросу о сезонных колебаниях радиочувствительности кроликов.
1092	К вопросу об общих проблемах радиочувствительности организма.
1093	О поступлении стронция-90 в растения.
1094	К вопросу об изучении форм поступления некоторых продуктов деления на земную поверхность.
1095	Особенности распределения стронция-90 в различных типах почв европейской части СССР в 1961 году.
1096	О подвижности и формах нахождения стронция-90, стабильного стронция и кальция в дерново-подзолистой и черноземной почвах.
	UNITED STATES OF AMERICA
1097	Radiological Health Data and Reports, volume 7, No. 3, March 1966.
	UNITED KINGDOM
1098	Assay of strontium-90 in human bone in the United Kingdom; Results for 1965, part 1, with some further results for 1963 and 1964. MRC report No. 12.

Document No.	*Country and title*
A/AC.82/G/L.	
	SWEDEN
1099	Preliminary report on X-ray induction of recessive lethals in mouse spermatogonia.
	UNITED KINGDOM
1100	Traits in man whose frequencies appear to be preponderantly determined by recurring mutations on the X chromosome. A note on birth frequencies and mutation rates.
A/AC.82/G/R.	
	UNITED STATES OF AMERICA
225/Add.10	Health and Safety Laboratory manual of standard procedures. Inserts and addenda, NYO-4700.
225/Add.11	Manual of standard procedures—NYO-4700. Addendum.

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.

ARGENTINA

Dr. D. Beninson (*Representative*)
Dr. A. Placer
Dr. E. Van der Elst

AUSTRALIA

Mr. D. J. Stevens (*Representative*)

BELGIUM

Professor J. A. Cohen (*Representative*)
Professor F. H. Sobels

BRAZIL

Professor L. R. Caldas (*Representative*)
Professor C. Pavan (*Representative*)

CANADA

Dr. G. C. Butler (*Representative*)
Dr. A. H. Booth
Dr. W. E. Grummitt
Dr. H. B. Newcombe

CZECHOSLOVAKIA

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