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MEMORANDUM FOR: R. Cunningham
D. Cool
F. Congel
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J. Buchanan
C. Feldman
R. Alexander
HEB Staff

FROM: William A. Mills, Chief
Health Effects Branch
Division of Radiation Programs
and Earth Sciences, RES

SUBJECT: DE MINIMIS

The enclosed document is the text of the John Till's panel presentation at the recent HPS midyear symposium. It raises and discusses the important point: Are we ready for de minimis levels of radiation dose or radioactive contamination?

If you have any comments or views on John's opinion, I would appreciate knowing them. Especially, since the Science Panel of the Interagency Committee on Radiation Research and Policy Coordination (CIRRPC/SP) may consider this issue in the near future within its general attention to radiation protection standards.

Mills

William A. Mills, Chief
Health Effects Branch
Division of Radiation Programs
and Earth Sciences, RES

Enclosure:
As stated

G/63

"DE MINIMIS DOSE IN RADIOLOGICAL ASSESSMENT--
ARE WE READY?"

A Summary of Points Made During the Panel Discussion on
"DE MINIMIS ENVIRONMENTAL RADIATION LEVELS: CONCEPTS AND CONSEQUENCES"

Health Physics Society Midyear Meeting

Colorado Springs, Colorado

January 10, 1985

John E. Till, Ph.D.

Radiological Assessments Corp.

Rt. 2 Box 122

Neeses, SC 29107

"*DE MINIMIS* DOSE IN RADIOLOGICAL ASSESSMENT--- ARE WE READY?"

INTRODUCTION

The momentum is rapidly building within scientific and industrial sectors to formally define a dose level that represents a negligible risk to society, a *de minimis* dose. Although the precise terminology for this level of negligible risk is relatively new, the perplexing problem of how to most effectively reduce health detriments or, if you prefer, improve our quality of life, has existed for some time in our profession of health physics and in other scientific disciplines.

The question is not really whether or not a level of *de minimis* dose is defensible but rather are we ready for *de minimis* dose--I suggest that we are not. In the brief statement that follows and through my participation on this panel I will outline my position on this important issue.

DE MINIMIS DOSE--WHERE DID IT BEGIN?

Philosophically, the concept of *de minimis* dose first began with the premise that a threshold dose existed below which there was no harm from radiation. The idea of weighing the importance of risks was presented in the *Health Physics Journal* as early as 1965 (Sowby 1965) in a paper titled "Radiation and Other Risks." Although the Latin term *de minimis* is derived from the legal profession, it was used in the context of radioactive material contamination by the late 1970's (Roger et al. 1976) and associated with risk by Cyril Comar (1979). From a survey of the open literature, it appears that Eisenbud (1980) was the first to combine the two words *de minimis* and dose at the 1980 NCRP annual meeting in his paper "The Concept of *de minimis* Dose." Since 1980 there has been a flurry of articles and letters proposing various levels of *de minimis* dose, interpreting its meaning, and justifying its need. From all of this information about the only thing that can be concluded with certainty is that we are finally learning to spell the term *de minimis* correctly. It is unfortunate that the term is being used loosely, often to have the same meaning as ALARA, threshold dose, and "below regulatory concern." Each of these has a distinctly different meaning; they are not synonyms. Little progress has been made regarding the definition of a level, the justification of the need for *de minimis*, or public acceptance of the concept. The necessity of public acceptance is a common conclusion in many articles and, as emphasized by Mills (1984), is likely more important than reaching any consensus of the level itself.

PROPOSED LEVELS OF *DE MINIMIS* DOSE

Proposed levels of *de minimis* dose range from 0.1 mrem/year to 500 mrem/year, representing a *de minimis* risk ranging between 1.8×10^{-8} and 9×10^{-5} (assuming a risk factor of 1.8×10^{-4} health effects/rem) (Cohen et al. 1984). As yet no level has been accepted within the United States; however, the National Radiological Protection Board in the United Kingdom has adopted a formal position on *de minimis* (Clarke and Fleishman 1984). They suggest 5 mrem to the individual representing a risk of 10^{-7} and 100 man-rem to a population. If the dose meets both of these criteria, then it is considered to be *de minimis*.

To date, neither the ICRP nor the NCRP has proposed a *de minimis* dose for environmental dose assessments. Currently the ICRP endorses the concept of objective health detriment (ICRP 1983). Societal consequences of one man-rem are the same whether they arise from a population of one thousand people exposed to 10^{-3} rem or one million people exposed to 10^{-6} rem. The NCRP is considering formulation of a *de minimis* concept within its Committee on Basic Radiation Protection Criteria (Scientific Committee 1).

Regardless of the effort by industry and regulatory authorities to implement a *de minimis* dose, the concept must have the full support of these scientific bodies if it is to gain public acceptance.

ARE THE ADVANTAGES REAL?

Recently it was pointed out in testimony before the ACRS that a *de minimis* dose could result in a net saving of up to $500,000 per year per nuclear power plant. For 80 plants this is $40 million annually. This saving would be primarily from relaxed restrictions on the disposal of low-level radioactive wastes (Davis 1984). Additionally, it is argued that with a *de minimis* concept, effluent treatment at nuclear facilities could be reduced, thus resulting in more savings (Clarke and Fleishman 1984). Presumably, the same kind of savings applies to all licensees that must deal with nuclear wastes and therefore the economic benefit overall would be significantly higher. Although we may be eager to think that *de minimis* dose would conserve resources, we have made only a cursory examination of the net economic benefits. Exactly how much effluent treatment would we remove? What about the cost of implementing the concept and selling it to the public? Have we considered the costs that will incur trying to prove that doses to the population are truly below the *de minimis* level. These topics deserve more attention in the future before we can say *de minimis* dose is justifiable on an economic basis.

It is also claimed that society will benefit in more than purely economic ways. Davis (1984) noted that a *de minimis* concept would increase public assurance by deregulating strict controls on very low levels of exposure. On the other hand, have we considered how much more aware the public will be of exposures that are above *de minimis* but still of very low risk? To what degree will the public insist that exposures be kept below *de minimis*? Will the public still accept ALARA or will it demand *de minimis*?

LEGAL ASPECTS OF *DE MINIMIS* DOSE

The U.S. Nuclear Regulatory Commission (NRC) regards the concept of *de minimis* dose as largely a legal one and the choice of a numerical level a judgement based on social and technical considerations (Mills 1984). The NRC already has the authority (by the Atomic Energy Act) to exempt from its licensing requirements certain types, quantities, and use of radioactive materials if it finds the exemption will not present an unreasonable risk to the common defense and security or to the health and safety of the general public. The problem the NRC has with this authority is that no level of trifling risk has been identified by the scientific community. Is it therefore the responsibility of the NRC to establish its own guidelines for *de minimis* dose?

The courts have recognized the existence of insignificant risks. In the so-called benzene case (AFL-CIO vs. American Petroleum Institute 1980) the U.S. Supreme Court offered quantitative bounds as follows: "If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk could clearly not be considered significant. On the other hand, if the odds were one in a thousand that regular inhalation of fumes from gasoline which are 2% benzene would be fatal, a reasonable person might well consider the risk significant and take appropriate steps to reduce or eliminate it." The Chief Justice concluded, "Inherent in this statutory scheme is authority to refrain from regulation of insignificant or de minimis risks." Can we argue that the same legal rationale applies to de minimis radiation exposure as well?

It is possible that the outcome of recent key litigation involving persons exposed to radiation would have been different if a formally adopted de minimis dose existed. Contradicting this point of view, however, is the question of how many additional cases would be opened by persons who claim to be exposed to levels greater than de minimis. Specifying a level of de minimis dose may aggrandize these possibilities.

ALARA AND DE MINIMIS--ARE THEY COMPATIBLE?

Many of you followed the exchange of letters that took place in the Health Physics Journal between Rossi (1980, 1982), Lindell and Beninson (1981), and Dunster (1982) which examined the implications and interpretations of de minimis dose and the ALARA principle. Rossi pointed out that by itself, ALARA is inadequate and raises the questions to what level should one aim when performing cost benefit analyses and why should we stop at ALARA if there is a linear response to dose. Dr. Rossi further added that "It is the responsibility of those responsible for radiation protection to see to it that personal exposures are below maximum permissible levels and reduced as near as practicable to de minimis levels."

Lindell and Beninson (1981) responded that ALARA is a recommendation to optimize radiation protection and that de minimis is not ALARA. Rossi (1982) replied that due to the great variability existing in the risk factors and the necessity to put a value on human life and suffering, the ICRP policy of optimization cannot be performed in a meaningful manner and the adoption of a purely de minimis dose would be more prudent.

The concluding letter in the series by Dunster (1982) stated that there is a real and significant difference of objectives evident in this exchange. He suggested that there is the need for both de minimis and ALARA. It is obvious as well that implementation of de minimis dose could provoke even more misunderstanding among the scientific community. If de minimis dose is adopted, then extreme care must be exercised in how it is used. For those wishing to eliminate all exposure to the public from radioactive emissions, then de minimis becomes the standard they are seeking in radiological assessment and ALARA becomes obsolete.

The possibilities for misusing the concept of de minimis dose are great. Only through careful justification of the level selected, a sound understanding and acceptance of de minimis dose by the public, and thorou documentation of the concept will these misuses be avoided.

WHAT ARE THE PROBLEMS?

Assuming the de minimis concept is accepted by the public, at what level should it be set? As already indicated a range spanning nearly three orders of magnitude currently exists. Whatever level is proposed, several important questions must be resolved.

First, how do we assure that the selection of a level of de minimis dose is an international decision, achieved through the ICRP with specific recommendations from individual nations? The implications of small doses on world populations from global cycling radionuclides and the disposal of radioactive wastes in the oceans make the assessment of radionuclides released to the environment an international as well as a national responsibility.

Second, how do we avoid overlooking the buildup of long lived radionuclides that may yield doses to the public that are less than de minimis today but could lead to irreversible levels of exposure that are above de minimis in the future if their release is ignored? Examples include ^{3}H, ^{14}C, and ^{129}I.

Third, are we prepared for the impact that a de minimis dose will have on our research programs investigating the effects of exposures to chronic, low-level radiation and the transport and fate of radionuclides in the environment? It is possible that these programs will suffer greatly by the acceptance of a de minimis concept. Additionally, have we considered how a de minimis dose could suppress the formulation of new ideas on threshold dose such as those discussed recently in The Health Physics Society's Newsletter (Belford 1984) being performed at Brookhaven and the relatively new emphasis on radiation hormesis?

Fourth, have we considered the importance of establishing a de minimis dose that incorporates consideration of risks from all aspects of life including non-radioactive environmental contaminants? Such a broad-based definition of de minimis would certainly be more defensible and may ultimately stand up for a longer period of time.

Fifth, would the public support a de minimis dose even if it were totally endorsed by health physicists? This problem of public support was noted by Dunster (1982) when he emphasized that society is not always logical and ethical in how it spends its resources, as evidenced by its resistance to certain additives in foods and its irrational views on small doses of radiation to large populations such as those resulting from the Three Mile Island accident.

CONCLUSIONS

The question is not really whether we need the concept of de minimis but are we ready for it. The answer is clearly no. We are merely on the fringes of this important issue and still have a long way to go before the need and implications of de minimis are fully understood. Further, we have given little thought to how such an important concept can be explained to and ratified by the public.

The rapidity with which the momentum is building to define a de minimis dose, without international cooperation and, even more importantly, without complete cooperation between our own regulatory agencies is a cause for concern.

The lack of financial support by U.S. government agencies and the nuclear industry to thoroughly evaluate the de minimis dose concept is also disturbing. It would appear that creating such an important new philosophy in radiation protection, especially one resulting in purported significant financial savings to society, should receive more than token assistance.

We must also consider alternatives to de minimis. Certainly one appealing approach would be the use of screening techniques such as those being prepared by the NCRP (Till 1984) in which very simple, calculational methods are used to evaluate radionuclides released to the environment. If the result of the assessment is below a cutoff point, then no further action is taken; if the analysis gives a value of dose above the cutoff point, then the model user must apply more sophisticated methods and perhaps get outside assistance.

Our Society must proceed with caution. The concept of de minimis dose must be approached carefully and, if a[illegible]ied at all, applied with the wisdom that 50 years of health physics knowledge has given us.

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Lord Rothschild

RISK

The Richard Dimbleby Lecture, November 1978

'The true logic of this world is in the calculus of probabilities'
James Clerk Maxwell

British Broadcasting Corporation

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Lord Rothschild's Richard Dimbleby Lecture, 'Risk', was broadcast on BBC 1 on Thursday 23 November 1978

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In a tribute to Richard Dimbleby Lord Mountbatten said:[1]* 'To an amateur appearing on television – which can be a very frightening process – he was kind, helpful, and considerate'.

I wish Richard Dimbleby were here to help me tonight.

I have called this lecture 'Risk'. Let me first make a general comment. There is no such thing as a risk-free society. Even a *virtuous* life has its risks, as illustrated by the Chinese proverb: 'The couple who go to bed early to save candles end up with twins'. But there is no point in getting into a panic about the risks of life until you have *compared* the risks which worry you with those that don't – but perhaps should. Comparisons, far from being odious, are the best antidote to panic. What we need, therefore, is a list or index of risks and some guidance as to when to flap and when not.

We are much more conscious of risks today than people were a hundred years ago. This is not only because we are better educated, and because the accelerating increase in scientific and technological knowledge brings with it new and sometimes imperfectly understood risks; but also because the media – particularly radio and television – bring to your notice infinitely more information than was conceivable in the days before Marconi invented radio-telegraphy. He started the world on its long, unfinished journey towards communication at distances and with detail beyond the range of human imagination eighty-three years ago. It is not the scale of disasters like millions of gallons of oil plastering the beaches of Brittany, or 582 people being killed when two Jumbo jets collided at Tenerife last year, which make the difference. After all, the Black Death killed some twenty-five million people in six years and the Great Plague of 1665 wiped out twenty per cent of the population of London.

What does make the difference is the speed and ubiquity with which information about such events is now disseminated. We learn about them within minutes or even seconds of their having taken place and are subjected to seemingly endless comments about them – fair, unfair, exact, inexact, scarifying and reassuring. That is why we are so conscious of risks and why we must be careful not to be led astray by apparently authoritative statements about the risks we run or don't run, as the case may be. I sometimes think that when talking or reading about risk, the most

*Superior numbers refer to the References Section at the end.

dangerous word in the English language is professor. Even real authorities are from time to time the victims, consciously or subconsciously, of their emotions and blind spots; and for every supposed authority there are a dozen charlatans and axe-grinders.

I want to go back to the past, for a few moments. Living has always been dangerous. In the 1770s, as Christopher Hibbert recounts:[2]

> The Prime Minister (Lord North) was robbed; so were the Prince of Wales and his brother, the Duke of York; a former solicitor to the Treasury was fired upon in his coach; the Lord Mayor was held up near Turnham Green and robbed 'in sight of all his retinue'; two daughters of Admiral Holborn were robbed in St. James's Square, and the Neapolitan Ambassador in Grosvenor Square.

Well, we still go on mugging; but in other ways life has improved and risks have decreased. We no longer remove a person's limb with a saw, without an anaesthetic. No longer are pneumonia and tuberculosis the deadly scourges they used to be, for rich and poor alike. No longer do we apply pigeon dung to the feet of those suffering from Bright's disease, as they did to Charles II.[3] No longer are 12 out of every 100 babies born dead, as they were in 1908.[4] The figure is now a half of 1% instead of 12.*[5] Such improvements will continue, provided our scientists can keep up with the ability bacteria have to develop protective clothing against our latest drugs. 'One drug ahead' should be the applied bacteriologist's motto.

But, new risks, real, sometimes imaginary and sometimes paradoxical, have developed with the accelerating growth of science and technology. And as I said, we are more conscious than our forebears of these new risks, quantified or pseudo-quantified; and thanks in particular to the media, we are more able to be vocal about them. The growth of knowledge is rarely achieved without the creation or emergence of new problems, and the growth of knowledge about risk brings with it a new class of problem. A good example is that at one time marvellous but now abhorred insect killer, DDT. It is harmful and persistent which means that DDT does not, like some other insect killers, decompose readily into harmless constituents.

Rachel Carson, the founder of ecomania, was the first to cash in on these properties of DDT. She wrote a book, *Silent Spring*,[6]

*Actually in 1976.

about DDT, other insecticides and weed killers. It was an emotive, rather plausible but somewhat one-sided indictment. Partly as a result, Ceylon banned DDT in the early 1960s. But then that country got a raging and virulent epidemic of malaria, a disease transmitted by a mosquito which, at that time, could have been controlled or virtually eliminated by DDT. Many therefore died unnecessarily because we had been clever enough to develop ways of measuring as little as 1 part in 10 million of DDT. Such are the risks of a no-risk society. Are we getting too clever by half? Of course not; but we must not allow the results of our cleverness to make us panic, and we must remember that one man's poison may be another man's life.

I mentioned DDT and the possibility of being too clever by half because there is a danger of stifling the analysis of risk, and its treatment, by prematurely asking over-complicated questions and raising over-elaborate difficulties. We have to guard against three dangers: first, not seeing the wood because of the complexity of the trees. I shall be almost entirely concerned with the wood. That is to say I shall not discuss many of the risks which may worry you: for example of smoking, or vaccination against whooping cough, sometimes because the risks in question cannot yet be formulated in a sufficiently clear and simple way – too many ifs and buts. The second danger is the belief that what is all right for America is equally all right in the rest of the world. Gas-guzzling – cars that do ten miles to the gallon or less – should have taught us this lesson. The third danger is to disregard the cost to society of reducing a risk to what it is fashionable to call an acceptable level.

This Dimbleby Lecture is not a disquisition on probability,* either from a philosophical or a mathematical point of view. So I will only say one thing about the concepts underlying risk: when I assert that the risk of a particular event occurring is 1 in 100, in spite of your knowing intuitively what, roughly, that assertion means, it needs some qualifications. The most important of these, in this context, is that the numbers I shall cite are not

*Lowrance' states that 'risk is a measure of the probability and severity of adverse effects' (p. 94). Risk has yet another meaning in Statistics, to do with Loss Functions.

absolute, in the sense that the number 3 is. 3 is neither $2\frac{3}{4}$ nor $3\frac{1}{4}$. It is 3. All the numbers which follow, however, like a 1 in 100 risk, should have known tolerances* attached to them. In other words, 1 in 100 is not exactly 1 in 100 in the world of risks, but, for example, *probably* somewhere between 1 in 95 and 1 in 105. Such tolerances are important. If I say you are 6 feet tall, with a tolerance of plus or minus 2 feet, no one can tell without looking whether you are a dwarf or a giant. One of the most famous mathematicians in history, C. F. Gauss, said: 'Lack of mathematical education is nowhere revealed so clearly as by meaningless precision in numerical studies.'[1]

I said earlier that risk must be described in terms of numbers like 1 in 100. If one wants to *compare* different risks, which I believe to be essential, the numbers must be presented in the same way. Unfortunately, quite a lot of people are frightened of, or dislike numbers. If, as I learned to my chagrin, one says: 'Put a large number of 5p pieces into that one-armed bandit, and 9 times out of 10 you will get at least three-quarters of all your money back', some people, including one eminent member of the Royal Commission on Gambling, become uneasy. 'I found that part of the chapter rather stiff,' he said; 'but then I am not numerate'. Of course that was not true, even though he may have thought it was. The truth is that, many years before, he had been put off appreciating the simplicity and economy of numbers by some idiotic schoolteacher who had pestered and inhibited him with problems of the sort so well known when he was young, to do with leaky bath plugs or the cost of a pound and a quarter of kippers.

I shall assume from now on that I have hypnotised you into forgetting about those problems we had to solve at school and that you are ready for me to answer your question: 'You go on about risks, the numbers attached to them, and comparing one risk with another. What in practice are you talking about? Give an example.' Very well. I shall start with a good one, to do with the number of people who had fatal accidents in certain industries in 1974. You will see that, in Table 1, I have put the industries in order of danger, from which it is clear that it is

*i.e. confidence limits

Table 1

Accidental deaths per million people per year in certain UK industries, 1974[a]

Quarrying	320
Underground coal mining	190
Chemical industries*	172
Farming	109
Food, drink and tobacco	46
Clothing and footwear	2

Confidence limits known

*includes Flixborough

much more dangerous to work in a quarry than in a clothing factory. I said this was a good example. It is for several reasons. First, each number of fatal accidents is related to the same number of people, 1 million, working in that industry. Secondly, each fatal accident is referred to the same period of time, one year, and the same year. The figures are not jumbled up together in an incomprehensible way, as in Table 2. Although these figures will probably elicit comment, such as: 'What! All those suicides?' or 'How on earth did all those people get killed at home?', there is a serious flaw in it, absent from the previous one in which the accidental deaths were given per year (1974), but also *per million people in the same industry*.

Table 2

Non-natural deaths in England and Wales, 1974[b]

Traffic	6372
At home	5747*
At work	692
Suicide	3899

Confidence limits known

*Over 70 % of these are people over 65. It is not a risk shared by the general population aged 5-65.

No conclusions can be drawn from the statement that there were 6372 traffic deaths in 1974, though it may cause some exclamation, unless we know whether it refers only to drivers, to passengers as well, per million drivers, per million miles of driving, per million hours of driving and so on. We can, however, be sure that the 3899 suicides were *not* out of the whole population of England and Wales in 1974; because children under the age of nine do not commit suicide here[11] (though they do in Japan). In fact, the table provides some information about which there is not much to say and even less to learn; and it tells us little about the comparative risks of working, driving, being at home or deliberately killing oneself.

This is not true about the first table, to do with occupational risks. We know, for example, that in 1974 the risk of having a fatal accident if you were a farm worker was 109 in a million or, dividing both these numbers by 109, 1 in 9200. Using a pocket calculator and rounding off the answers, we can recast the information as in Table 3. You might conclude that none of these activities was all that dangerous, a 1 in 3000 risk of being killed during a year seeming pretty remote. But suppose you spend all your working life in a quarry and that no one bothers, as may be the case, to make conditions safer. Then the risk of a quarry worker being killed during that working life becomes only about 1 in 80, rather a different matter.

Table 3

Risk of being killed in certain UK industries, 1974

Quarrying	1 in 3100
Underground coal mining	1 in 5300
Chemical industries*	1 in 5800
Farming	1 in 9200
Food, drink and tobacco	1 in 21,700
Clothing and footwear	1 in 500,000

Confidence limits known

*includes Flixborough

Reverting to incomplete information and the difficulty of making inferences from it, this is a quotation[11] from a recent article by Lord Ashby, the eminent, thoughtful, level-headed and persuasive scientist – I shall use him on more than one occasion as a jumping off point, jumping on him as well from time to time. He said:

> Much more significant and puzzling is the apparently irrational attitude which people have towards environmental hazards . . . Some 7000 people are killed and some 350,000 injured each year on the roads of Britain. Yet this perpetual carnage – nearly 1000 killed or injured every day – generates no public outrage.

Let me be the devil's advocate for once and for a moment. I shall omit the injuries and concentrate on those 7000 deaths. Let us accept without question that they occur 'each year'. Is that 'perpetual carnage'? Should it evoke 'public outrage'? If it does not – *and* it does not – we had better start by putting that figure into perspective, that is by *comparing* it with some other sources of carnage. It is done in Table 4, which some of you may find familiar. Please do not think that this table is intended to persuade you to minimise or ignore the gravity of road fatalities which in this table are expressed as a numerical risk, per year, instead of only deaths in some year or other. But you will find that politicians will be *rather chary* of imposing a maximum speed limit of 50 miles an hour on all roads where the limit is not already 30 or 40, though if they did, both energy and lives would be saved. Why, then, don't they do it? It would not *really* be difficult to enforce. I shall put the answer politely. Their judgment, a concept to which I shall come back later, tells them that people would not like it. And then all the other goodies they

Table 4

Risk of being killed, per year, 1974

Quarrying	1 in 3100
Underground coal mining	1 in 5300
Farming	1 in 9200
Road accident	1 in 7500

Confidence limits known

have in mind for you, less unemployment, less inflation, less taxation, an increasing standard of living, fair shares for all, more pay for the police and the miners – you name it – might be unrealisable: because, you *might* say: 'Maybe we need a change of Government. I want to go faster than 50 miles an hour on all those marvellous motorways I paid for'.

You will realise that I am about to move on to the subject of risk acceptability, which many people divide into two classes: death risks that are incurred voluntarily, like taking contraceptive pills (1 in 50,000 women dead per year),[13] or those that are involuntary, like dying from influenza (1 in 18,000 per year). In the first class, acceptability is a matter for the individual if we ignore his or her friends and family, and the cost of the Health Service. In the second class, involuntary risk, acceptability is of greater public interest, and more complicated. Acceptability by whom and for whom?

Once again I revert to Lord Ashby who had this to say about the acceptability of risk:[14]

> As a very rough generalisation, it can be said that risks of 1 in a million are of no great concern to the average person (though risks of nuclear power plant disasters are in the region of 1 in a thousand million*) . . . When the risks rise to 1 in 10,000 the public are willing to incur expenditure to reduce the risk (for example crash barriers on roads, railings at busy intersections) unless the risk is voluntary, for example cigarette smoking. At 1 in 1000 a risk becomes unacceptable to [the] public and there is strong pressure to have it reduced.

I find this 'very rough generalisation' hard to stomach, and the figures, apart from giving no indication of the duration of the risk, which is essential if they are to have a meaning, confirm my indigestion. What on earth is meant by saying that 'the public are willing to incur expenditure to reduce the risk'? Does the public have any opportunity of saying it is unwilling? Is the public ever asked if it is willing to pay for crash barriers and railings? I doubt it. Of course the public asks for precautions from time to time; and sometimes gets the brush-off as in Cambridge when the Ministry *refused* to reduce a speed limit from 40 to 30 miles per hour. Not enough children killed on the stretch of road in question.

*Quoted from the Rasmussen Report.[15]

To give another example of the problems of public acceptability, far more row is made about the possibility of a major accident at a nuclear power station than about death from influenza. One can compare the death risk from these two sources in the following way. Assume, generously, that some time hence we have 100 nuclear power stations in this country and, improbably, that no safety improvements are made; and, equally improbably, that the risk of death from influenza remains constant at 1 in 18,000 per year. Let us make Lord Ashby's figure of 1 in a thousand million for one nuclear power station disaster ten times more probable: that is, 1 in a hundred million. Then the risk, each year, of dying from influenza is 55 times as great as that of dying as a result of a nuclear power station disaster when there are a 100 of them.

You can look at the danger, or lack of danger, of living near a nuclear reactor in another way, which is shown in Table 5, which refers to the United States. It shows the risk of being killed accidentally in various ways within 25 miles of a nuclear reactor, per year.

Table 5

Annual death risk within 25 miles of a nuclear reactor, USA[1]

Car accident	1 in 4000
Accidental falls	1 in 10,000
Fires	1 in 27,000
Reactor accident	1 in 750,000*

Confidence limits known

*Risk multiplied by 10

We have been discussing the reactions of the 'average person'. Who is he? Certainly not Ralph Nader or the Friends of the Earth. Nor the Westinghouse Corporation which makes nuclear reactors, or others who are emotionally committed to the nuclear age; though all of them can generate a lot of heat in the media, with varying amounts of light. These and many others are pressure groups and one of the objects of this lecture is to remind you that pressure groups will work on you or are doing so already, directly or indirectly, through the media which are not always sufficiently critical or objective about them. Pressure groups should neither be ignored nor rejected; nor should they be unthinkingly accepted. What I hope you will do, if you are sufficiently interested – and I think you should be – is to ask yourself or 'them' some very simple questions.

First Is the risk stated in straightforward language that I can understand, such as 1 in 1000? If not, why not?

Second Is the risk stated per year, per month, per day or per some period of time? If not, I shall ignore the information.*

I would also like you to ask another, more sophisticated question. It is: Are the tolerances on the figures, or the uncertainties associated with them, given or said to be known? If they are not, the information must be of questionable value unless it comes from an acknowledged authority who knows so much that he can make an informed and reasonably accurate guess. That may be the best one can do at a particular moment in time; but I don't like it, even though I shall have to make do with informed guesses later, when discussing nuclear power station risks.

Please keep these questions in mind when, in the next year or two, genetic engineering gets its public bashing and is simultaneously hailed as the saviour of mankind. Insist on both sides telling you, in our language, what the risks are in training bacteria to make cheap Insulin (for diabetics), or cheap Interferon (for treatment of some forms of cancer) – or something cheap and very nasty, by accident or design.

One must not suffocate the rational treatment of risk by overcomplicating the issues too soon, that is before the basic principles of risk assessment are known and accepted. Lord Ashby is, I believe, conscious of this problem; but his solution makes me uneasy. He does not deny the need for hard information – scientific, economic, technological and statistical; but he goes on to say that for what he calls 'political decisions' to be made, another,

*Some risks are properly given per challenge or per event, e.g. trying to cross the Atlantic by balloon which, according to Sir Edward Pochin (personal communication, 8.8.1978), is associated with a death risk of 6/17 (which must now be 6/18).

soft ingredient is necessary: hunch, which he equates, wrongly in my view, with political judgment. Although intuition, which is the same as hunch, has been described as a quick way of coming to the wrong conclusion, I do not believe this description is always fair. There is a famous story,[11] perhaps apocryphal, about Sir Isaac Newton telling the great astronomer Halley about one of his discoveries. 'Yes', said Halley, 'but how do you know that? Have you proved it?', to which Newton replied: 'Why, I've known it for years. If you'll give me a few days, I'll certainly find you a proof of it' – and in due course he did.

So hunch is respectable, provided one remembers that you and I are almost certainly not blessed with the Newtonian variety – few of us are capable of hearing the music of the spheres. Political judgment is a very different matter in democracies. It is not a way, hidden from ordinary mortals, of revealing or illuminating some important truth or need for action. I do not denigrate politicians or their decisions by saying that, with very few exceptions, they are motivated by considerations of personal power, winning elections and the national good, in that order. Those considerations, in that order, are their business, their life blood. To give an example to which I shall come back later: small may not always be so beautiful as some politicians want it to be. But so many people are now convinced that small is beautiful – those fifty-acre farms and charming windmills – that a politician's rational or logical thoughts may have to be adjusted to accommodate the beauty of the small: in other words he has to exercise 'political judgment'.

You will realise that I am not too happy about leaving the treatment of risk to the judgment of politicians. I prefer to believe that if some course of action is made self-evident by *hard* information, and if that course of action is not followed, you and I will say to the politician who denies it what Queen Elizabeth the First said to her man Cecil: 'Get out!'.[12] Of course, insistence on hard information is a counsel of perfection which it will be very difficult to realise in practice. But that does not mean we should allow others to reject the possibility of being rational. We have to try to be rational if only to minimise our reliance on the very occasional Isaac Newton and the not so very occasional exercise of that dubious commodity, political judgment, which is not the art of the possible but that of political self-preservation.

'At what level of risk should I start to get worried?' I hear you say. I am going to make a suggestion which I hope will not be thought subversive. Apart from those who have suffered personal tragedies, I do not believe there is a single one of you, here or at home, who has ever lost a minute's sleep worrying about car accidents. The risk of being killed in a car accident in Great Britain was about 1 in 7500 per year in 1974.[10]* Why don't we start there and say: 'If the risk is less than 1 in 7500 I shan't worry: more, and I shall.' Let's be more precise, by means of examples. You can drink a bottle of wine a day because that's half as dangerous as driving a car.' But you should worry if your boy rides a motorbike because it is 75 times more dangerous than driving a car.[11] We need that index of risks, don't we?

But what do you, a member of the public, do if you are worried? Lobby your MP or local councillor? Join a pressure group? More likely nothing, because most of us are an easy-going lot. It takes a hell of a risk to make us say 'Get out!'

However, things will get easier in due course. Microprocessors will make possible cheap and simple tele-referenda. That is to say, the questions will pop up on your television screens and there will be three new buttons, 'yes', 'no' and 'don't know', for you to push. Then you will be able to watch the results after *Match of the Day* every Saturday evening.

In this lecture I have already given some examples of poorly-formulated risk information and commented adversely on them. Let me give you an example (Figure 1) of an utterance by what

Figure 1

We Almost
Lost Detroit

By John G. Fuller
272 pp. New York
Reader's Digest Press, $8·95

I will charitably call an emotionally over-committed man, and at the same time the follow-up, by an emotionally over-commit-

*1 in 7532 to be accurate, averaged over the total population of Great Britain.

ted lady in the press, in this case *The New York Times Book Review*, on 30 November 1975. There had been an accident in the small Fermi nuclear reactor near Detroit. No one was injured, nor were there any serious consequences outside the reactor. The reviewer, Mary Ellen Gale, said:

> When things went awry at the Enrico Fermi reactor near Detroit, four million people went about their business in happy ignorance, while the technicians gingerly tinkered with the renegade's invisible interior. They knew what the public did not – a mistake could trigger a nuclear explosion.

In fact, a nuclear explosion is no more feasible in a nuclear reactor than it is from chewing pickled cucumbers or gum.[illegible] That does not, of course, mean that nuclear reactors are as harmless as chewing-gum. On the contrary: they must be treated with the utmost care: and they are, I believe, in this country.

I want now to discuss a subject called risk accountancy,[illegible] with particular reference to nuclear power generation. Dr Herbert Inhaber, of the Canadian Atomic Energy Control Board, has recently studied the application of risk accounting to solar energy devices and to those power stations which derive their energy from coal, oil, natural gas, wind or uranium. (There are those who say that Dr Inhaber has not only studied this subject but that he has also stuck his neck out uncomfortably far from his own point of view.) Risk accounting means that if you want to attach risk numbers to an energy machine such as a power station, you must add up all the risks associated with the production of a particular amount of energy, *from the word go*. 'Go' starts with the raw materials out of which the power station is made – how one gets hold of the iron, copper and sand; how one turns them into steel, copper tubing and glass components; how one transports these components; how one converts or constructs them into power stations. Finally, how one gets the coal, oil, uranium and natural gas to the power station. Of course, one doesn't have to 'get' the sun or the wind. They come to us. That's why, apart from their inexhaustibility, they are so attractive to some people, leaving the 'small is beautiful' concept aside.

The results of risk accounting are surprising and perplexing – perplexing because they run counter to our intuition. 'Surely the risk of being hit by the blade of a windmill is negligible', I hear you thinking. Possibly: but have you remembered the risk in getting and fabricating the materials with which the windmill produces that energy, apart from making and erecting the stand-by plant for when the wind happens to have stopped blowing?

When one adds up the total death risk from producing a particular quantity of energy, a surprising picture emerges, Table 6: the risk of energy production from uranium or natural gas is clearly smaller than from coal, oil, wind or the sun. Goodbye huge windmills? Goodbye solar energy? Windmills, yes, I suspect, except for small ones; some solar energy devices, probably not.

Table 6

Estimated deaths for a specified energy output (10 GWy)

Coal	50–1600
Oil	200–1400
Wind	230–700
Solar, space heating	90–100
Uranium	2½–15
Natural Gas	1–4

I must mention, despairingly, that each year the peoples of the world flare or burn as waste 7 trillion* cubic feet of natural gas,† the safest of the lot. No one knows, apparently, what else to do – how to collect it and put it to good use. Those 7 trillion cubic feet are about equivalent to this country's whole energy consumption in 1977. Of course, some waste is inevitable; but if our non-renewable sources of energy are running out so quickly, as we are told at least once a day, would you not think a little more effort might be made to put some of those wasted trillions to good use? As a matter of fact one can make excellent edible protein out of North Sea natural gas and I think I was the first or, perhaps, the second person to taste it, not without some trepidation, I must confess. But my fear was irrational and there-

*1 trillion = 10^{12}.
†Estimate by Shell, 1978.

fore to be deprecated. There was no risk. The protein had already been analysed and there was nothing harmful in it.

Why do I say I was the *first* or, perhaps, the *second* person to taste protein from North Sea natural gas? Dr Norris, who had prepared it, said to me, 'Try it, it's perfectly okay'. For a split second I asked myself, 'Has he really tried it, or am I to be the guineapig?' If Professor Norris, as he now is, happens to be listening to this lecture, he can retrospectively put my mind at rest.

Let's spend a moment on the controversial one, uranium power generation. The table showed that it is one of the safest sources of energy (including one or two left out) and much safer than energy based on coal, oil or wind. Can it be true? The answer is yes. But what *are* the risks associated with a nuclear-powered electricity generating station and the allied operations? There are some eight possible risks, Table 7. With one exception I don't think the odds against these events have been given before.

I believe that most of you are more worried about nuclear power problems than other public and involuntary risks. Things have progressed since the Flowers' Royal Commission reported on this subject in 1976; but there are still problems to be solved. I have, I hope, cleared up quite a few of them. But one headache I have emphasised before in public, the disposal of radioactive waste from nuclear power stations, still persists. If, as I expect, the environmental lobby will continue to sabotage efforts even to examine the possibility of finding suitable places for radioactive waste disposal in this country, far below the surface of the earth, then I say to our Atomic Energy Authority: 'For God's sake sail away and find an uninhabited island, free from volcanoes and with the right geological characteristics. Get cracking *there* and don't tell the econuts where you have gone; or else they may be waiting to welcome you'. There are more than a hundred thousand islands to choose from.

In this lecture I have used the derogatory words econiania and econuts. That does *not* mean that I am against the preservation of the environment. How could I be, given that in 1915 my father prepared, for the then Ministry of Agriculture, the 'Provisional Schedule of Areas in the United Kingdom considered worthy of preservation'. He also *created* the Society for the Promotion of Nature Reserves. In his Will he ordered the destruc-

Table 7

Nuclear Power Station Risks

'Atomic explosion'	0
Death from escape of radioactive substances, 25 miles radius	Less than 1 in 1,000,000 per year
One-man sabotage	1 in 100,000 to 1 in 1,000,000 per year (IG)
Baader Meinhof suicide attack	1 in 100 per year (IG)†
Theft of uranium or plutonium	No figures available. Precautions very good.
Reprocessing used fuel	Less than 1 in 100,000 per year (IG)
Radioactive waste disposal	Not quantifiable. Soluble but not solved.
From the air (bombs or rockets)	By accident: less than 1 in 10,000,000 per year. By intent: 1 in 100,000 to 1 in 1,000,000 per year (IG)
Fast breeder reactor	Potentially important but not ready for general use. Risk intended to be no greater than with thermal reactors.

IG = Informed guess

*At 10[illegible] the 95% confidence interval is estimated to be 10[illegible] – 10[illegible].

†The probability of such an attack, as opposed to that of an adverse result (0·01) is, obviously, secret.

tion of all his maps showing where the rare butterflies we knew so well, like the Large Copper and the Swallowtail, could be found. Thus I was brought up to believe that our natural environment was of paramount importance. But even with this background, I cannot avoid the conclusion that the current exaggerated, if not maniacal, attitude of some people towards our environment is very damaging to our future well-being. Zero growth, the econuts' panacea, won't bail out anybody.

We must also spend a moment or two on coal-fired power stations, which may be even more popular in the next century than now, and need much more risk attention than they have so far had. According to the American Medical Association in June of this year,[21] the number of premature deaths which can be ascribed to the creation and operation of a coal-fired power station of the usual size is 400 times as many as in the case of a nuclear power station, a figure much more in favour of nuclear power even than Inhaber's. The National Coal Board and the Royal Commission on Environmental Pollution should debunk these views if they are wrong – and as soon as possible, at least so far as the United Kingdom is concerned. If they can't, the taxpayer will get an astronomical bill, if the politicians decide to ditch nuclear power.

When I was head of the Government's Think Tank I once asked a Cabinet Minister whether he thought we were of any use to the Cabinet. He replied, 'You make us think'. If this lecture makes you think about risk for a minute or two – quite a long time, incidentally, to think hard and continuously – it will have achieved one of its purposes. Another is to persuade you to say, 'Perhaps there's something in what he said, even if it was only the beginning and the subject gets more complicated later. We really ought to be given a chance to compare the different risks around us before being put into a panic by some apparently authoritative utterance. It's not as difficult as all that'. So why not produce an index of risks so that you can decide above what level – road fatalities, perhaps – you should get into a panic: and below what level – death from influenza – you should relax. That's quite a big spread, from 1 in 7500 to 1 in 18,000 per year for the region of personal choice.

Let me end with a quotation from Spenser's *Faerie Queene*: 'Be bold, be bold and everywhere be bold: but be not too bold' – particularly when making, or walking near, a windmill.

Acknowledgements

I am grateful to those who have helped me in the preparation of this lecture and in particular to Professor F. R. Farmer (United Kingdom Atomic Energy Authority), Sir Edward Pochin (National Radiological Protection Board), Kenneth Rose and Professor E. Rothschild (Massachusetts Institute of Technology).

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SOURCES, EFFECTS AND RISKS OF IONIZING RADIATION

UNITED NATIONS

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NOTE

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INTRODUCTION

1. This is the tenth in a series of substantive reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[a] to the General Assembly[b]. The preparation of this Report and its scientific annexes took place from the thirty-first to the thirty-seventh sessions of the Committee. The material of this report was developed at annual sessions of the Committee, based on working papers prepared by the Secretariat that were modified and amended from one session to the next according to the Committee's requests. During the period of preparation of this Report, which contains seven scientific annexes, another Report containing three scientific annexes was completed at the thirty-fifth session of the Committee. These two reports, referred to as the 1986 and 1988 Reports, constitute the latest comprehensive assessment by the Committee of the sources, effects and risks of ionizing radiation.

2. The following members of the Committee served as Chairmen, Vice-Chairmen and Rapporteurs, respectively, at the following sessions: thirty-first session, Z. Jaworowski (Poland), D. Beninson (Argentina) and T. Kumatori (Japan); thirty-second and thirty-third sessions: D. Beninson (Argentina), T. Kumatori (Japan) and A. Hidayatalla (Sudan); thirty-fourth and thirty-fifth sessions: T. Kumatori (Japan), A. Kaul (Federal Republic of Germany) and A. Hidayatalla (Sudan); thirty-sixth and thirty-seventh sessions: B. Lindell (Sweden), K.H. Lokan (Australia) and J. Maisin (Belgium). The names of those experts who attended the thirty-first to the thirty-seventh sessions of the Committee in an official capacity as representatives or members of national delegations are listed in Appendix I.

3. In approving this Report, and assuming therefore full responsibility for its content, the Committee wishes to acknowledge the help and advice given by a small group of consultants who assisted in the preparation of the text and scientific annexes, upon appointment by the Secretary-General. Their names are given in Appendix II. They were responsible for the preliminary reviews and evaluation of the technical information received by the Committee or available in the open scientific literature, on which rest the final deliberations of the Committee. Additional assistance and financial support for the preparation of some of the scientific annexes were offered to the Committee by various international and national organizations. The Committee would like to express its gratitude to these organizations, which are listed in the relevant annexes.

4. The sessions of the Committee held during the period under review were attended by representatives of the United Nations Environment Programme (UNEP), the World Health Organization (WHO), the Food and Agriculture Organization of the United Nations (FAO), the International Atomic Energy Agency (IAEA), the International Commission on Radiological Protection (ICRP) and the International Commission on Radiation Units and Measurements (ICRU). The Committee wishes to acknowledge their contributions to the discussions.

5. Reports received by the Committee from Member States of the United Nations and members of the specialized agencies and the International Atomic Energy Agency, as well as from these agencies themselves, during the period from 19 April 1986 to 17 June 1988 are listed in Appendix III. Reports received before 19 April 1986 were listed in previous Reports of the Committee to the General Assembly. This information received officially by the Committee was supplemented by, and interpreted with the help of, many other data available in the current scientific literature or, in a few cases, from unpublished communications by individual scientists.

6. In the following Report the Committee summarizes the main conclusions of the specialized studies undertaken, also in the light of previously released substantive documents. The material is presented at the most general level possible, in view of the difficult concepts

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[a] The United Nations Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session in 1955. Its terms of reference are set out in resolution 913 (X). It was originally composed of the following Member States: Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, Union of Soviet Socialist Republics, United Kingdom of Great Britain and Northern Ireland and United States of America. The membership of the Committee was subsequently enlarged by the General Assembly in its resolution 3154C (XXVIII) to include Germany, Federal Republic of, Indonesia, Peru, Poland and Sudan. By resolution A/RES/41/62B the General Assembly increased the membership of the Committee to a maximum of 21 and invited the People's Republic of China to become a member.

[b] Previous substantive reports of the United Nations Scientific Committee on the Effects of Atomic Radiation to the General Assembly are to be found in 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); ibid., Twenty-first Session, Supplement No. 14 (A/6314); ibid., Twenty-fourth Session, Supplement No. 13 (A/7613); ibid., Twenty-seventh Session, Supplement No. 25 (A/8725); ibid., Thirty-second Session, Supplement No. 40 (A/32/40); ibid., Thirty-seventh Session, Supplement No. 45 (A/37/45); ibid., Forty-first Session, Supplement No. 16 (A/41/16). These documents are referred to as the 1958, 1962, 1964, 1966, 1969, 1972, 1977, 1982 and 1986 Reports, respectively. The 1972 Report with scientific annexes was published as Ionizing Radiation: Levels and Effects, Volume I: Levels, Volume II: Effects (United Nations Publication, Sales No. E.72.IX.17 and 18). The 1977 Report with scientific annexes was published as Sources and Effects of Ionizing Radiation (United Nations Publication, Sales No. E.77.IX.1). The 1982 Report with scientific annexes was published as Ionizing Radiation: Sources and Biological Effects (United Nations Publication, Sales No. E.82.IX.8). The 1986 Report with scientific annexes was published as Genetic and Somatic Effects of Ionizing Radiation (United Nations Publication, Sales No. E.86.IX.9).

and notation that characterize this field. After a chapter summarizing the developments and trends that have become apparent throughout the years, the highlights and conclusions to be drawn from the most recent studies in the fields of radiation physics and biology are presented. This main text is followed by the supporting scientific annexes, which are written in a format and a language that are essentially aimed at specialists.

7. Following established practice, only the main text of the Report is submitted to the General Assembly, while the full Report, including the scientific annexes, will be issued as a United Nations sales publication[c]. This practice is intended to achieve wider dissemination of the findings for the benefit of the international scientific community. The Committee wishes to draw the attention of the General Assembly to the fact that the main text of the Report is presented separately from its scientific annexes simply for the sake of convenience. It should be understood that the scientific data contained in the annexes are of great importance because they form the basis for the conclusions of the report.

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[c] United Nations Publication, Sales No. E.88.IX.7.

I. HISTORICAL REVIEW

A. GENERAL CONSIDERATIONS

8. Throughout the thirty-three years of its existence, the Committee has assertively attempted to provide the best possible estimates of: (a) doses received by the world's population in the past, and expected to be received in the future, from various natural and man-made sources of radiation, and (b) risks of induction of various types of harm by radiation, both in the short term and the long term, by individuals directly receiving such doses or by their descendants over many generations.

9. With the passing of time and the increase in number and complexity of the Reports issued by the Committee, it is becoming increasingly difficult, even for the specialists, to trace back to earlier publications the development of the main ideas underlying the Committee's assessments and how these assessments have changed with time and as a result of increasing scientific knowledge. It would seem useful, therefore, to make available in compact, summary form the main conclusions reached in the fields mentioned above. This summary is intended to serve a number of purposes. First, it will inform the General Assembly about the Committee's work and its findings. Second, for the Committee's membership which has been changing gradually over the years, it will form a record of how the Committee's thinking has evolved. Lastly, it will be placed at the disposal of the international scientific community, for whom UNSCEAR Reports and scientific annexes have become a basic reference.

10. What follows in this chapter is therefore a summary of the Committee's assessments in the fields of dose estimation (which pertains closely to the subjects of physics) and risk assessment (which involves physical as well as radiobiological and medical considerations). It aims at giving an account of both the general principles underlying the estimates and the conclusions reached, in a language that is as plain as the complexity of the subjects allows but without much of the discussions supporting the choices made at any particular time. For this, as well as for other technical and methodological details, reference is made to the Reports to the General Assembly issued from 1958 to 1986. A complete list of these publications issued by the Committee appears in footnote *b* to paragraph 1 of this Report. Current assessments are examined in more detail in the following chapter II.

B. CONCEPTS, QUANTITIES AND UNITS

11. Radiation is a transport of energy through space. In traversing material, radiated energy is absorbed. In the case of ionizing radiation, which is the type of radiation that concerns the Committee, the absorption process consists in the removal of electrons from the atoms, producing ions. Ionizing radiation may be produced in man-made devices, such as x-ray tubes, or it may come from the disintegration of radioactive nuclides, the phenomenon that is called radioactivity. While nuclides such as these occur naturally, they may also be produced artificially, as in nuclear reactors. The two basic quantities in the assessment of radiation levels and effects are the activity of a radioactive material and the radiation dose. The Committee uses the system of radiation quantities and units adopted in 1980 by the International Commission on Radiation Units and Measurements (ICRU).

1. Activity

12. The *activity* of a radioactive material is the number of nuclear disintegrations per unit time. The unit that the Committee used for this quantity up to and including its 1977 Report was the *curie* (Ci), which is 37 billion ($3.7\,10^{10}$) disintegrations per second, a number which was originally introduced because it is the approximate activity of 1 gram of radium-226.

13. The present unit of activity has been given the special name *becquerel* (Bq). One becquerel is one disintegration per second.

14. The word radioactivity denotes the phenomenon of radioactive disintegration. It is not a synonym for "activity", nor should it be used to mean "radioactive material".

2. Radiation dose

15. The term radiation dose can mean several things (e.g., absorbed dose, dose equivalent or effective dose equivalent). The *absorbed dose* of radiation is the energy imparted per unit mass of the irradiated material. Up to and including the 1977 Report, the Committee used the *rad* as the unit of absorbed dose (1 rad = 0.01 joule/kg). The present unit of absorbed dose is joule/kg, for which the special name *gray* (Gy) is used. Thus, 1 rad = 0.01 joule/kg = 0.01 Gy.

16. Different types of radiation have different Relative Biological Effectiveness (RBE). The RBE of one type of radiation in relation to a reference type of radiation (usually x or gamma) is the inverse ratio of the absorbed doses of the two radiations needed to cause the same degree of the biological effect for which the RBE is given.

17. When the first UNSCEAR Reports were prepared, the International Commission on Radiological Protec-

tion (ICRP) had recommended certain values of RBE for the purposes of radiation protection. The absorbed doses of various radiations were multiplied by these values to arrive at doses weighted for the purposes of radiation protection (e.g., for comparison with dose limits). The unit of this weighted absorbed dose was called rem.

18. The use of the term RBE in two contexts, radiation protection (where it only meant the standard values recommended by ICRP) and in radiobiology (where it meant the most likely value in a given exposure situation for a specified biological effect), caused some problems. ICRP and ICRU therefore decided to establish a new quantity, the *dose equivalent*. This would be the product of the absorbed dose and a so-called quality factor (first denoted QF and later Q), and its unit would be the rem. The quality factor was given by ICRP as a function of the capacity of each radiation to produce ionization, expressed as the linear energy transfer (LET). For practical applications, ICRP suggested that it would suffice to use approximations of average values, i.e., one unique value of QF (Q) for each type of radiation. It suggested values of Q = 1 for x rays, gamma rays and beta particles, Q = 10 for fast neutrons (changed to Q = 20 in 1985), Q = 10 for alpha particles (changed to Q = 20 in 1977), and Q = 20 for heavy particles. The Committee has also used these factors continued to use Q = 10 for fast neutrons.

19. In the UNSCEAR Reports, when doses are expressed in rem, the ICRP values of "RBE (protection)", QF or Q have been used in most cases; however, when authors express doses in rem, they may have used the primary, LET-related definition of the (Q).

20. When the Committee began in 1982 to apply the new international unit system and the absorbed dose was given in Gy instead of rad, the new unit for dose equivalent was named the *sievert* (Sv).

21. In addition to absorbed dose and dose equivalent, there is a third quantity that may be meant when an author speaks of radiation dose, namely, the *exposure*. Exposure is the total electrical charge of ions of one sign produced in air by electrons liberated by x or gamma rays per unit mass of irradiated air. Since the exposure is a measure of the ionization that x- or gamma-radiation would produce in air, it is therefore only applicable for those types of radiation. The unit of exposure is *coulomb/kg*, but the old unit, the *roentgen* (R) is still in use. One roentgen is equal to 2.58 10^{-4} coulomb/kg. The word "exposure" is also used in this Report in its common meaning of being exposed to something, e.g., a radiation source.

22. In this latter meaning, the exposure to radon decay products can be expressed in two different ways: as the amount of inhaled decay products, taking into account their potential to emit radiation energy, or as the product of the time during which the decay products were inhaled and their concentration in the inhaled air. The potential alpha energy of the inhaled decay products may simply be expressed in *joule* (J). The potential alpha energy concentration in air is expressed in J/m³ or in the older unit, the *working level* (WL), where 1 WL = 2.08 10^{-5} J/m³. For radon in equilibrium with its decay product, this corresponds to a concentration of 3700 Bq/m³. Exposure to the decay products is customarily expressed in terms of the *working level month* (WLM) or, as is now also common, Bq h/m³.

23. In the 1958 Report of the Committee, the word "dose" was used loosely, and the quantity meant had to be inferred from the units used (roentgen, rad or rem). In the UNSCEAR 1962 Report, doses were sometimes expressed in rad, sometimes in rem. However, in the next five Reports (up to and including the 1977 Report), the approach was more stringent. The absorbed dose was used consistently and the dose equivalent was deliberately avoided. The main reason for this was that one use of the physical and biological information was to provide a basis for estimates of RBE and therefore also to evaluate the appropriateness of the recommended values for Q. To present doses as dose equivalents would have been to beg the issue. Sometimes, however, exposures had to be expressed in roentgen because this was how the original data had been presented.

24. With the UNSCEAR 1982 Report, the practice changed. The Committee had gradually become more concerned with risk estimates and was not satisfied with merely reporting levels of absorbed dose. One reason for this was the growing evidence that radon daughter products caused lung cancer and that these daughter products were present in high concentrations in dwellings. Previously, dose contributions from types of radiation with RBEs other than unity had not been considered important and the presentation of absorbed doses was thought to be sufficient. Now, the situation was different. While it was recognized that the dose equivalent was a quantity designed for radiation protection and that the Q values recommended by ICRP might differ from the true values of RBE, the dose equivalent was still believed to give a better indication of risk than the absorbed dose.

3. Development of dosimetric concepts

25. Paragraphs 25-41 review historical development of other concepts and quantities used by the Committee. When the UNSCEAR 1958 Report was issued, two biological effects were prominent: leukaemia and hereditary harm. For that reason, priority was given to calculating dose in the red bone marrow and gonads. In the case of dose in the gonads, it was obvious that the dose would be relevant to risk assessment only if it were calculated for individuals young enough to expect children. In the case of dose in the bone marrow, the question arose whether the mean dose or the peak dose would be relevant; the ensuing discussion led to the concept of mean marrow dose.

(a) *The genetically significant dose*

26. It was realized early that for most populations the medical uses of x rays were the main source of man-made exposure. However, dose distribution within a patient is very uneven, so the dose assessment is not easy. In addition, the age distribution in exposed patient groups differs from that in the general population. To solve these problems, the Committee derived the concept of *genetically significant dose* (GSD), defining it as "the dose which, if received by every member of the population, would be expected to produce the same total genetic injury to the population as do the actual doses received by the various individuals". On the basis of this definition, the Committee developed a formula and an assessment procedure for estimating the genetically significant dose from various types of x-ray examinations. This is described in detail in the 1958, 1962 and 1972 Reports.

(b) *The mean marrow dose*

27. Assuming that the mean dose in the active (red) bone marrow would be the quantity relevant to assessing the leukaemia risk and using information on the distribution of active marrow in the skeleton, this quantity was assessed for various types of x-ray examinations. While it was recognized that this would not be the relevant quantity if the dose-response relationship was non-linear or showed a dose threshold, it was equally clear that if the relationship was linear and showed no threshold, yet another quantity, the *per caput mean marrow dose* in a population would be of interest, and this quantity was assessed in the UNSCEAR 1958 Report.

(c) *The dose commitment*

28. Nuclear test explosions in the atmosphere introduced time elements that made this source of radiation different from, for example, medical exposures, in the sense that the period of practice and the period of exposure were different. After each nuclear explosion, some long-lived radionuclides were released that will persist in the biosphere for many years, causing radiation exposures. To have presented the annual doses caused by the tests that had been carried out up to the time the UNSCEAR 1958 Report was drafted would not have given the full picture: namely, it would not have shown that the contamination was expected to last for a long time, thus committing mankind to exposures in future years. The situation was described by diagrams in the UNSCEAR 1958 Report. These diagrams showed the doses to be expected under various assumptions about the period of future testing.

29. In the UNSCEAR 1962 Report, the Committee introduced the concept of *dose commitment*. The dose commitment from one year of practice is the sum of the per caput annual doses inevitably caused by the resulting environmental contamination over future years. It can be shown that the dose commitment from one year of a practice is equal to the highest annual per caput dose in the future, if the practice continues indefinitely at constant rate. This relationship made it possible to assess the future consequences of continuing various practices.

30. In the UNSCEAR 1964 Report, the dose commitment was defined as "the integral over infinite time of the per caput 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." This definition was repeated in subsequent Reports and a stricter mathematical presentation was given in 1969 and 1977. It should be mentioned that when the integration of the average dose rates is carried out not to infinity but only to some specified time, one is dealing with truncated dose commitments.

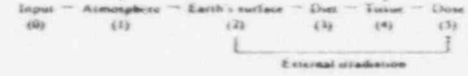
(d) *Collective doses and collective dose commitments*

31. The use of the dose commitment concept did not carry any implication of assumptions with regard to the dose-response relation at the low doses of radiation that were assessed for the environmental contamination; it was merely a mathematical device for adding inevitable dose contributions.

32. Another concept is the *collective dose*. Assuming a proportionality between dose increments and resulting increments in the risk of harm, the expected number of harmfully affected individuals would be proportional to the collective dose, since the latter is defined as the product of the number of exposed individuals and their average radiation dose. Before 1977, the Committee hesitated to assess collective doses, because doing so would have implied an unproven dose-response relation. In its 1977 Report, however, the Committee assessed collective absorbed doses from various sources and practices. Where a practice was expected to cause exposures over future years, the *collective dose commitment* was assessed. This is simply the total collective dose expected from a given practice over all future time.

(e) *Transfer coefficients*

33. Dose commitments from practices causing environmental contamination are proportional to the amount of the relevant radionuclides that have been released into the environment. Thus, the assessment involves the study of a chain of events starting from the primary injection of radioactive material into, for example, the atmosphere and ending with the eventual irradiation of body tissues. This chain of events can be represented schematically:



34. Beginning with the UNSCEAR 1969 Report, the Committee has assessed *transfer coefficients*, i.e., the quotients of the time-integrated quantity (e.g., activity concentration) in each step and the corresponding quantity in a previous step. For example, the transfer coefficient P_{34} is the time-integrated activity concentration in a given tissue divided by the time-integrated concentration of the same nuclide in the diet. The product of all transfer coefficients directly relates the amount of radioactive material injected into the

atmosphere to the resulting dose. The mathematical formulation and assessment procedure were described in detail in the UNSCEAR 1969 Report.

(f) Organs of interest

35. As has already been mentioned, in the UNSCEAR 1958 Report the Committee calculated doses for only two organs: the gonads and the active bone marrow. They were the only organs for which some risk estimates had been made at that time. In the UNSCEAR 1969 Report, the Committee added dose assessments for one more tissue, namely the cells lining bone surfaces. Up to 1972, the dose assessments had thus been made for three organs (gonads, active bone marrow and bone surface cells), although the Committee had in fact made risk estimates for other organs, such as the thyroid (1964 and 1972) and breast and lung (1972). One reason for limiting the number of organs was that the dose assessments would become more complicated the more organs the Committee included and comparisons between various sources would become very difficult.

36. Nevertheless, in the UNSCEAR 1977 Report the Committee added still one more organ, the lung, because it had become increasingly evident that the alpha-emitting daughter products of radon in dwellings were biologically significant and that radon escaping from uranium mill tailings was generating very high long-term commitments.

(g) The effective dose equivalent

37. In 1977, ICRP published a revision (ICRP Publication 26) of its general recommendations, in which it suggested that a weighted sum of the radiation dose equivalents in the most radiosensitive organs and tissues should be the basis for radiation protection assessments. This weighted sum was named the *effective dose equivalent*. It was to have the same unit as the dose equivalent, i.e., the sievert. The effective dose equivalent is determined using only the organ weighting factors recommended by ICRP on the basis of risk assessments. Other types of sums of weighted organ doses, with different weighting factors, must not be called effective dose equivalents.

38. The effective dose equivalent was originally intended to reflect the relative organ risks for an average member of a working population. It gave the same weight to a severe hereditary defect in the exposed individual's first two generations of offspring as to the occurrence of a lethal cancer in that individual. It gave zero weight to curable cancer. The concept was appropriate considering the intended use of the quantity. The same quantity has since found widespread use in the assessment of collective doses to members of the public. Here, where its failure to account for the difference between the age distribution of workers and that of the public at large and its non-inclusion of curable cancer and hereditary harm in generations beyond the second are known deficiencies, the use of the effective dose equivalent may be questionable. Various corrections to compensate for these limitations have been suggested, but for the purposes of radiation protection, and considering all other uncertainties, the extensions of the use of the effective dose equivalent have mostly been accepted.

39. In looking for ways of presenting radiation doses from various sources and practices, UNSCEAR faced problems similar to those faced by ICRP. Particularly in the cases of medical exposure and exposure from radon daughter products in the lung, different organs receive quite different doses, and the idea of a weighted whole-body dose was attractive. The Committee is well aware of the fact that the effective dose equivalent has not been designed for its particular purposes, but it has not been able to find an alternative way of expressing radiation exposures by a single number.

40. In the definition of the effective dose equivalent there is an addition of cancer risk and risk of hereditary harm. The risk coefficients for cancer and hereditary harm, as applied to the effective dose equivalent, are clearly identifiable only if all organs receive one and the same dose. In cases where they do not, the effective dose equivalent gives a basis for estimating the total risk but gives no indication of the relative proportions of the cancer risk and the genetic risk (see section II.C).

41. The effective dose equivalent was used in the UNSCEAR 1982 Report, and comparisons were made on the basis of the collective effective dose equivalent commitment. To simplify the presentation of doses and dose comparisons, the Committee has had to resort to more and more complicated terms, and there is, unfortunately, no easy way out of this dilemma.

C. DOSE ASSESSMENTS

1. Natural sources of radiation

42. In preparing its first Report (1958), the Committee concluded that the three main contributors to radiation doses from natural radiation in soft tissues of the human body were cosmic rays, terrestrial gamma-radiation and potassium-40 within the body itself. When the joint dose contribution of these three sources was assessed in the UNSCEAR Reports of 1958-1977, it varied from 93 to 98 per cent of the total absorbed dose from all natural sources, which was estimated to be about 100 mrad per year. The contribution of the three sources were as follows: about 30 mrad from cosmic rays, 30-50 mrad from terrestrial gamma radiation and 20 mrad from potassium-40 in the body.

43. In all UNSCEAR Reports up to and including that of 1972, doses were assessed for three tissues: gonads, osteocytes and active bone marrow. The per caput doses in these tissues were used for dose comparisons in the main text of the Reports. The assessed values varied only a little from one Report to another, with the exception of an overestimate of the dose from the neutron component of cosmic rays in 1962.

44. In the UNSCEAR 1977 Report, the lung dose from radon daughter products inhaled indoors was given in the summary tables, but it did not look so conspicuous since it was presented as an absorbed dose. In 1982, however, the effective dose equivalent was calculated for the first time, and the significance of this contribution became obvious, since it amounted to about one half of the total, as a world-wide average. The assessed value of the annual effective dose equivalent from natural radiation sources was raised accordingly, to about 2 mSv, i.e., to about twice the value implied in previous UNSCEAR Reports, where the lung dose had not been taken into account.

2. Nuclear explosions

45. Most nuclear explosions in the atmosphere occurred before 1963. Their total yields in equivalent amounts of TNT were estimated in the UNSCEAR 1964 Report as follows:

1945-1951	~1 megaton
1952-1954	60 megaton
1955-1956	28 megaton
1957-1958	85 megaton
1959-1960	0 megaton
1961-1962	337 megaton

These numbers have subsequently been somewhat revised in the light of more recent information (see paragraph 143 and Table 5).

46. The atmospheric tests after 1962 were small in comparison with the earlier explosions, and they ceased completely after 1980. The many underground explosions carried out in later years have had few environmental consequences. This temporal picture gives an indication of the environmental situation that prevailed when the Committee prepared its various Reports.

47. Large explosions in the atmosphere carry most of the radioactive material into the stratosphere, where it remains for some time, the mean retention times being estimated from less than a year to about five years, depending on the altitude and latitude. Fallout can therefore occur years after an explosion has injected material into the atmosphere. Smaller explosions carry the radioactive material only into the troposphere, and fallout occurs within days or weeks.

48. When it prepared the UNSCEAR 1958 Report, the Committee did not yet have sufficient information on the global inventory of long-lived radioactive materials to be able to formulate the assessment models used in later Reports. However, the Committee correlated measured fallout rates and deposits with observed radioactive contamination levels in vegetation and food. As explained in section I.B, the quantities that were first assessed were the genetically significant dose and the per caput mean marrow dose, because for these the Committee could make risk estimates.

49. In the first four UNSCEAR Reports (1958-1966), the Committee described in detail the meteorological processes that depict the stratospheric inventory of radioactive debris. For man, the highest exposure was found to be due to long-lived radioactive material that causes radiation exposures over many years. The dominant radionuclides were strontium-90 (half-life: 28 years), caesium-137 (30 years) and carbon-14 (5,700 years). Some gamma-emitting radionuclides from tropospheric fallout, e.g., zirconium-95 and ruthenium-106, could also contribute significantly through exposure from the ground deposition.

50. Because it was interested in the radiation dose in active bone marrow and in osteocytes, the Committee initially made its most thorough dose calculations for strontium-90. Eventually, however, caesium-137 turned out to cause higher doses because of its double exposure modes: by external gamma-radiation from ground deposition and by internal exposure after intake with food. The exposures from caesium-137 could be verified using direct measurements of the body content, but this was more difficult for strontium-90.

51. With the UNSCEAR 1962 Report, the Committee applied the concept of dose commitment. This made it possible to assess the impact of tests carried out in a particular year or of all the tests up to the time of a Report. In such assessments, however, the contribution from carbon-14 turned out to be high, because of its long half-life. Models for estimating the dose commitment from carbon-14 were developed in the UNSCEAR 1962 and 1964 Reports.

52. In 1964, attention was drawn to the high individual doses caused by enhanced concentrations of caesium-137 in some food chains, in particular the lichen-reindeer chain. This was further discussed in the UNSCEAR 1966 Report, where it was reported that levels of caesium-137 in reindeer meat had in some cases reached 100 nCi/kg (3700 Bq/kg) and in fresh-water fish, 10 nCi/kg.

53. In the UNSCEAR 1969 Report, the mathematical formalism of all calculations was reviewed and the concepts of transfer chains and transfer coefficients were introduced. By the time the UNSCEAR 1972 Report was prepared, the fallout rate had decreased substantially, most of the testing having ceased in 1962. Better estimates could therefore be made of some transfer coefficients, which resulted in somewhat lower dose estimates.

54. In 1977, for the first time, collective dose commitments to most soft tissues of the body from the nuclear test explosions before 1976 were estimated and found to be between 400 and 800 million man rad without the full carbon-14 contribution and about twice as great with the full carbon-14 commitment. For comparison, in the UNSCEAR 1977 Report the annual collective dose to the world population from natural sources of radiation was estimated to be about 300 million man rad.

55. In the UNSCEAR 1962 Report, essentially the same basic information was reviewed. The dose assessment models were then described in a special

Annex, which also listed conversion coefficients, symbols and units. This time the effective dose equivalent was calculated. According to the 1982 assessment, the collective dose contributions from the major radionuclides were as follows:

Radionuclide	Collective effective dose equivalent commitment (10^6 man Sv)	
	External	Internal
Strontium-90	—	0.5
Zirconium-95	0.6	—
Ruthenium-106	0.2	0.1
Caesium-137	1.5	0.7
Others, except carbon-14	0.2	0.7
Subtotals	2.5	2.0
TOTAL	4.5	

56. One of the main problems in estimating future collective doses is that assumptions have to be made about the size of the population. In deriving estimates in the UNSCEAR 1982 Report, the Committee assumed a world population of 4 10^9 persons when calculating collective doses from radionuclides with half-lives of 10-30 years. The dose commitment from these and from shorter-lived radionuclides was estimated to be about 1 mSv. In calculating the collective dose from carbon-14, the Committee used a world population of 4 10^9 in its 1977 assessment, but a projected population of 10 10^9 in its 1982 assessment. The latter assumption made the estimated collective effective dose equivalent commitment from carbon-14 as high as 26 million man Sv.

3. Nuclear power production

57. In 1970, the world-wide total installed capacity for generating electric energy in nuclear reactors was about 20 GW. Over the next ten years, nuclear electric generation increased by more than 10 GW installed capacity per year, to reach 144 GW in 1981. This rapid introduction of nuclear power on a large scale warranted assessments by the Committee starting with its 1972 Report. Facing a situation similar to that which it had faced with the nuclear explosions, the Committee realized its assessment of future doses would depend on the assumptions it made about the continuation and extension of the practice of nuclear energy generation. It is interesting to note that, at that time, the projections for expansion which the Committee quoted were an order of magnitude higher than turned out to be the case.

58. Thus, in addition to assessing of dose commitments and collective dose commitments per year of practice at the current rate, the Committee therefore also estimated these quantities per unit of electric energy produced, i.e., per MW year. The main contributions to the collective dose commitment were believed to come from global contamination by tritium and krypton-85 released during the reprocessing of spent fuel and from local exposures near the power stations. The total was assessed at about 0.4 man rad/MW year. This value, however, was not used in the summary tables or in the main text of the report. Instead, there was an estimate of the annual per caput dose to the world population if nuclear power production would be maintained at the level expected for year 2000 (an installed capacity of 4,300 GW electric power). This annual dose was estimated to be about 0.2 per cent of the dose from natural sources of radiation.

59. In the UNSCEAR 1977 Report, there was a more systematic approach to assessing the collective dose commitments per unit of electric energy produced for each step of the nuclear fuel cycle (mining, milling, fuel fabrication, reactor operation and fuel reprocessing), including occupational exposures. The estimates made in the UNSCEAR 1977 Report were substantially higher than those made in the UNSCEAR 1972 Report, because more data became available and a fuller treatment was possible. Occupational exposure was estimated to contribute nearly 4 man rad/MW year and exposure of the public between 1.5 and 3.8 man rad/MW year to various tissues. The highest single contrib[illegible] was again found to come from global distrib[illegible] to reprocessing. In the Committee's opini[illegible] values may be somewhat pessimistic, because the prior experience of reprocessing and research and development, two contributors that were together assessed to cause between 4 and 6 man rad/MW year, may not be able to indicate future experience. The Committee faced a special problem in dealing with the exposures from radon released from uranium mill tailings. This source would cause lung doses that would not be high for any one individual, but the long time period over which radon might emanate from the tailings (determined by the physical half-life of thorium-230) could make the collective dose commitment quite high.

60. The problem posed by radon was recognized more clearly in the UNSCEAR 1982 Report, where the effective dose equivalent was calculated. The various steps in the fuel cycle were together estimated to cause 5.7 man Sv/GW year (0.57 man rem/MW year), excluding global distribution. About 2 man Sv/GW year were estimated to be caused by global distribution from tritium and krypton-85. Occupational exposure was estimated to contribute somewhat less than 30 man Sv/GW year. The total estimate was therefore about 35 man Sv/GW year (3.5 man rem/MW year), somewhat lower than the 1977 estimate.

61. In addition, however, the Committee expected a contribution from the very long-lived radionuclides carbon-14 (half-life 5,700 years) and iodine-129 (1.6 10^7 years); from radon emanation primarily controlled by thorium-230 (8 10^4 years); and from long-lived actinides leaking from high-level waste repositories. With the exception of carbon-14, these nuclides were not expected to cause any significant cumulative collective dose over any 1000-year period (carbon-14, however, would give 10 man Sv/GW year during the first 100 years). But, over 1 million years, assuming a world population of 10^{10} persons, the collective dose from the long-lived radionuclides was estimated at about 3,400 man Sv/GW year:

Radon from mill tailings	2,800
Uranium from mill tailings	460
Carbon-14	110
High-level waste	30
Iodine-129	28

The corresponding doses to any one individual over a lifetime would be negligible, e.g., compared to the doses from natural background radiation, the large numbers being due merely to the long time periods. It is not a scientific question to what extent exposures over such time periods are relevant in decision-making.

62. Using the concept of incomplete (truncated) dose commitment and assuming future annual nuclear energy generation of 10,000 GW years, the Committee finally projected the annual per caput effective dose equivalent to be 25 microsievert i.e., about 1 per cent of the annual dose from natural background radiation.

4. Medical exposures

63. In 1957, when it was preparing the UNSCEAR 1958 Report, the Committee issued an important statement: "It appears most important ... that medical irradiations of any form should be restricted to those which are of value and importance, either in investigation or treatment, so that irradiation of the population may be minimized without any impairment of the efficient medical use of radiation." The statement also solicited further information on medical exposures, which were recognized to constitute a substantial proportion of the total radiation received by mankind.

64. In the UNSCEAR 1958 Report, the Committee gave priority to the assessment of genetically significant dose. It was realized that the highest genetically significant doses were caused by diagnostic x-ray exposures, which, at that time, were frequently carried out with fluoroscopy rather than with radiography. Diagnostic procedures were classified into 23 types, and the exposure data for these were presented for a few countries, permitting comparisons of doses between the various procedures. In addition, crude estimates were made of the per caput mean marrow dose from these procedures. More than 80 per cent of the genetically significant dose was found to be contributed by only six or seven procedures, which together made up only about 10 per cent of all procedures. The data indicated that it might be possible to reduce the doses considerably, simply by careful attention to techniques. The total genetically significant dose from x-ray procedures ranged from 17 to 150 mrem per year in the various national estimates.

65. In the UNSCEAR 1966 Report, the Committee continued its review of the national data that had been submitted. Detailed data were available from 12 countries. The results were similar to those in the UNSCEAR 1958 Report. The values of the genetically significant doses now assessed ranged from 7 to 58 mrem per year. Ways of reducing patient doses were discussed, and the most effective protective measures were listed, such as the use of the smallest possible radiation field and the reduction of fluoroscopy time. This, in effect, was a protection recommendation, released before ICRP had issued any special recommendations on the protection of patients.

66. Medical exposures were next reviewed in the UNSCEAR 1972 Report. The emphasis was still on the genetically significant dose, and the values now assessed ranged from 5 to 75 mrad per year, although the number of x-ray examinations was reported to have increased by between 2 and 6 per cent per year. The Committee felt that, finally, enough information was available from industrialized countries to provide a basis for attempting to eliminate unnecessary exposures. It noted that a large proportion of the world population did not have easy access to modern x-ray facilities and the health benefits they would provide.

67. In the UNSCEAR 1977 Report, the Committee discussed the problems of comparing doses from exposures to sources as diverse as natural radiation, nuclear explosions, nuclear power production and medical exposures. With regard to the latter, the organ doses caused by diagnostic radiology range from a few millirad to a few tens of rad and are usually delivered at high dose rates. The dose distribution is uneven, both within the body and in the population. Moreover, the emphasis that had so far been put on the genetically significant dose might have hidden the possibility of substantial exposures of other organs, so the Committee extended its assessments to include organs other than the gonads and the active bone marrow.

68. In its attempts to find bases for dose comparisons, the Committee looked for, but failed to find, a satisfactory way of combining doses to various organs into some weighted whole-body dose that would be of relevance in cancer risk assessments. As a compromise, in the UNSCEAR 1982 Report, the Committee decided to assess the effective dose equivalent, which, in spite of its shortcomings, best suited its purposes.

69. The 1982 assessment confirmed that medical exposures constitute the largest man-made contribution to radiation doses received by the population and that in some industrialized countries, this contribution approaches the dose received from natural sources. However, the Committee reminded the reader that medical exposures differ from other man-made exposures in that the practice directly benefits those who are exposed. The yearly number of diagnostic x-ray examinations was now found to vary between 300 and 900 examinations per year and per thousand inhabitants in industrialized countries, excluding mass surveys and dental examinations. X-ray examinations contribute the major portion of the collective effective dose equivalent from medical procedures; radiation therapy and nuclear medicine contribute only a minor portion.

70. The Committee expressed disappointment that very little information was available for the two thirds of the world's population that live in countries where radiological examinations are an order of magnitude less frequent than in the more developed countries.

For developed countries, the Committee estimated the annual collective effective dose equivalent from medical procedures at about 1000 man Sv per million of population, i.e., about 50 per cent of the exposure from natural sources.

5. Occupational exposures

71. The Committee discussed occupational exposures in the UNSCEAR 1958, 1972, 1977 and 1982 Reports and pointed out repeatedly that the data that had been submitted were, for a number of reasons, difficult to analyse. The doses reported are those measured by personal dosimeters, and the quantity measured depends on both the type of dosimeter and on its calibration. These recorded doses depend on the location of the dosimeter on the body, and it must be assumed that they approximate a uniform whole-body dose. The number of persons occupationally exposed is not the same as the number of persons monitored, the difference depending on national requirements for radiation monitoring. The objective of most monitoring programmes is not to provide data for purposes such as those of the Committee, but to check that authorized dose limits are not exceeded. So-called investigation levels are usually applied, below which doses are ignored or recorded as zero. Little information is therefore available for the low-dose region.

72. The treatment of the subject in the UNSCEAR 1958 Report was brief. The number of workers in the medical field in countries that had submitted data was estimated to be between 0.2 and 0.7 per thousand of the total population. The treatment of occupational exposures in the UNSCEAR 1962 Report was brief as well. The number of dental workers was found to be about twice the number of medical workers, while the number of persons occupationally exposed in industries or in research was substantially lower. The contribution of occupational exposures to the annual genetically significant dose was estimated at 0.2-0.5 mrem.

73. At the time of the UNSCEAR 1972 Report, there was still very little published data on occupational exposures. The number of workers in the medical field could now be narrowed down to 0.3-0.5 per thousand in the countries for which data were available, and the total number of persons reported as occupationally exposed was 1-2 per thousand of the total population. The mean recorded dose for most workers exposed to radiation was found to be between 0.2 and 0.6 rad per year, but mean doses as high as 2.7 rad were reported from some industrial radiography workers. The annual dose to crews of supersonic aircraft was assessed to be about 1 rem. Occupational exposures in the nuclear power industry were expressed per unit electric energy produced and were calculated to be 2.3 man rad/MW year (1.6 man rad from fuel reprocessing and 0.7 from reactor operation).

74. In the 1977 Report, an Annex was devoted to occupational exposures. For the first time, the Committee systematically reviewed the purposes and methods of assessment. It was found that the distribution of doses within the exposed occupational groups was mostly log-normal, and on this basis a reference dose distribution was defined. To avoid the problems of determining the actual number of workers exposed and therefore, also, average doses, the Committee emphasized collective doses, the values of which would be largely independent of the administrative requirements on the degree of monitoring. The Committee also calculated the fraction of the collective dose accounted for by annual individual doses exceeding 1.5 rad. The submitted data were analysed on this basis. For most occupations, the mean dose was was 0.1-1 rad per year. A detailed mathematical description of the log-normal distribution and of the reference distribution was given. The collective dose from each step of the nuclear fuel cycle was calculated, with the doses from all steps adding up to about 4 man rad/MW year (see section I.C.3). The collective absorbed dose in the lungs of uranium miners was estimated to be 0.1 man rad/MW year, and examples of high radon levels in non-uranium mines were reported.

75. In its 1982 Report, the Committee continued the analysis on the basis of more data. It noted with satisfaction that its 1977 proposal for methods of analysis had been adopted by several organizations and that the arrangement of submitted data had been influenced by the proposal, thus facilitating the analysis. However, the Committee now found that its suggestion of a reference radiation dose distribution had sometimes been misinterpreted, so it limited its presentation to the average dose, the collective dose and the fraction of the collective dose exceeding 15 mSv (corresponding to the previous 1.5 rad).

76. For countries with a high standard of medical care, medical workers were found to receive a collective dose equivalent of about 1 man Sv per million of population. The number of workers in the nuclear industry had increased substantially since 1977. Occupational exposures in each step of the nuclear fuel cycle were assessed more fully, indicating that the total collective effective dose equivalent might be near 30 man Sv/GW year (3 man rem/MW year). However, half of this came from fuel reprocessing and nuclear research, and it was uncertain whether such high contributions should be expected also in the future. In reactor operation, the highest exposures were to maintenance workers and radiation protection staff during special maintenance operations.

6. Miscellaneous exposures

77. In addition to the main radiation sources discussed thus far, a few other sources were identified by the Committee as far back as in the UNSCEAR 1958 Report. Then, as now, they were referred to as miscellaneous sources. Mentioned in the UNSCEAR 1958 Report were watches with radio-luminescent paint, television sets that could produce soft x rays and shoe-fitting equipment that used x-ray fluoroscopy. None of these sources was expected to cause a genetically significant dose exceeding 1 mrem per year, although the shoe-fitting machines could cause high

local doses. The UNSCEAR 1962 Report mentioned enhanced cosmic radiation to passengers in aircraft but considered the dose insignificant. The total genetically significant dose from all miscellaneous sources was not expected to exceed 2 mrem per year, the largest contributor to which was radioactive watches.

78. In the UNSCEAR 1972 Report, a full Annex dealt with the miscellaneous sources. Incidents, transportation accidents and loss of radioactive material were mentioned as additional sources of public exposure. A number of radioactive consumer goods were also described, such as radioluminescent timepieces and other self-luminous devices, ceramic glazes containing uranium, and thoriated electrodes in welding rods. Radioactive substances in patients released from hospitals, pace-makers with nuclear batteries, and demonstration materials in schools were also mentioned. Television sets were again discussed, particularly the colour ones, whose cathode-ray tubes operate on higher voltages. Finally, it was recognized that enhanced levels of natural radiation could cause problems, as, for example, ~~do~~ radioactive building materials. In later Reports this would become an important topic, no longer treated as a miscellaneous source.

79. In the UNSCEAR 1977 Report, the miscellaneous sources were discussed in an Annex dealing with technologically enhanced levels of radiation. One of the many consumer products added to the list was ionization-chamber smoke detectors. However, the discussion centred on enhanced exposures to natural radiation. Enhanced exposures to cosmic rays in aircraft, including supersonic transports, and in spacecraft, were discussed in detail. Another subject was public exposure due to natural radionuclides emitted from coal-fired power plants. A third subject was exposures due to the industrial use of phosphate products containing uranium-238 and radium; in this case, the exposure pathways were via phosphate fertilizers and by the use of waste gypsum as a building material. Normal exposures from radioactive building materials, whether direct (by gamma-radiation) or indirect (by radon daughter products), were dealt with in the discussion on natural sources.

80. In the UNSCEAR 1982 Report, again, miscellaneous sources were considered together with technologically modified exposures to natural radiation. Essentially the same consumer products were discussed as in the previous reports. It was noted that the radium in wrist-watches had now almost entirely been replaced by tritium, thereby eliminating the external exposure and limiting the annual effective dose equivalent to the wearer from leakage tritium to less than 1 microsievert. The average effective dose equivalent to air passengers passing x-ray fluoroscopic scanners was estimated to be much lower still, about 7 nanosievert per scan. Exposures from coal-fired power plants were reassessed and the collective effective dose equivalent commitment was estimated to average 2 man Sv/GW year (this is 50 per cent of the local and regional collective dose from the same energy production in nuclear power stations, see Table 6). The 1977 production of phosphate rock was estimated to have resulted in a collective effective dose equivalent commitment of 300,000 man Sv, predominantly from the use of gypsum in dwellings; the total contribution from other uses was thought to be only 6,000 man Sv.

7. Accidents and incidents

81. The Committee discussed radiation accidents in the UNSCEAR 1962, 1972, 1977 and 1982 Reports. In 1962, it reviewed the eight major accidents known to it at the time; these had caused at least four deaths. Seven of the accidents were criticality accidents (five in the United States, one in the USSR and one in Yugoslavia). The eighth accident involved pulsed x rays from an unshielded electronic tube at a radar station. The course of the accidents and the clinical symptoms of the exposed persons were discussed in some detail.

82. In the 1972 Report, accidents were treated only briefly. The Committee noted that about 100 incidents in connection with the transport of radioactive material had been reported throughout the world from 1954 to 1968. There had been fourteen accidents involving aircraft carrying nuclear weapons or components of nuclear weapons. Two nuclear submarines had disappeared, and a plutonium-238 isotopic generator had burned up in the upper atmosphere. A number of incidents had also been reported wherein radioactive material had been lost or stolen. An analysis of 115 radium incidents occurring from 1966 to 1969 showed that 55 per cent of the incidents were losses. In another study of 299 incidents involving the loss or theft of radium, 66 per cent of the sources were recovered. The same Report also briefly discussed occupational accidents, showing that they had been particularly frequent in x-ray analytical work and in industrial radiography.

83. In the UNSCEAR 1977 Report, the Committee for the first time discussed accidents at nuclear power plants. In its review of the collective dose commitments from the various steps in the nuclear fuel cycle, the Committee approached the difficult problem of dose commitments from accidents that had not yet occurred. Any nuclear power programme is also a commitment to a certain accident probability, so in that sense, the Committee said, there is also an accident dose commitment.

84. In 1982, the Committee observed that there had so far been only two reactor accidents known to have caused measurable irradiation of the public: one at a military plant at Windscale, United Kingdom, in 1957, and one at a nuclear power station at Three Mile Island, Pennsylvania, United States, in 1979. The collective whole-body dose from the latter accident had been estimated between 16 and 35 man Sv within 50 miles, most of it due to xenon-133, and about of equal magnitude outside 50 miles. The collective effective dose equivalent from the Windscale accident had been estimated at about 1,300 man Sv, of which almost half was due to iodine isotopes and thyroid irradiation. The Committee decided that the probabilistic approaches, which predict the risk from reactor programmes by extrapolating into the future, should not be used as a basis for estimating future components of collective dose commitment.

85. In another part of the UNSCEAR 1982 Report, the Committee reviewed information on occupational accidents. It tabulated those accidents on which it had received data or which had been reported in the open literature. The Committee noted that the serious accidents had occurred early in the development of nuclear technology and that not one serious accident had been reported in reactor operation since the mid-1960s. Radiation accidents in other industries had caused one death since 1960; this death occurred in 1975 in an irradiation facility with cobalt-60. As had been noted in the earlier Reports, industrial radiography seemed to have a special potential for accidents. Some severe injuries had occurred when persons picked up lost radiography sources without being aware of the danger.

D. RISK ASSESSMENTS

1. Hereditary harm

86. The methods used so far to quantify genetic risk can be broadly grouped under two headings: the doubling dose (or relative mutation risk) method and the direct (or absolute mutation risk) method. The doubling dose method aims at expressing the risk in relation to the natural prevalence of genetic diseases in the general population; the direct method aims at expressing absolute risk in terms of expected increases in the prevalence of genetic diseases. Owing to the paucity of direct human data on radiation-induced genetic damage leading to disease states, the rates of induction for the pertinent kinds of genetic damage (mutation and chromosomal aberrations) are based on experimental data in animals. These rates are converted, using a number of assumptions and reduction factors, into the expected number of additional cases of genetic disease in man.

87. To apply the doubling dose method, one needs: (a) an estimate of the doubling dose, i.e., the radiation dose that will produce as many mutations as those occurring spontaneously in a given generation; (b) information on the prevalence of naturally occurring genetic diseases in the population and the extent to which these are maintained by mutation; and (c) an estimate of the dose received by the population. Over the years the doubling dose estimates have been based on experimental data obtained in the mouse; the prevalence figures for naturally occurring genetic diseases are those collected in several epidemiological studies. With the doubling dose method, the risk is the product of the prevalence of naturally occurring genetic diseases, the mutation component, the reciprocal of the doubling dose and the dose sustained by the population.

88. Over the past three decades, there have been shifts in emphasis in the use of these methods and there have also been a number of refinements, as extensively discussed in Annex E. The principles that guided UNSCEAR, as well as other scientific bodies, in its early assessments of radiation-induced hereditary risk in the 1950s were those that had emerged from the extensive investigations in Drosophila, the preliminary results in mammals, particularly the mouse, and the sparse human data. Two of these principles were the following: (a) mutations, induced or spontaneous, are generally harmful, and (b) mutations induced by radiation increase linearly with dose without a threshold.

89. In the light of new data from studies on male mice showing that a chronic gamma dose was only about one third as effective as the same dose given at a high dose rate (and even more reduced in female mice), the UNSCEAR 1962 Report suggested that the previously used doubling dose of 30 roentgen would probably be too low by a factor of 3 to 4. With confirmation and extension of these results and other data showing that the interval between irradiation and conception had a dramatic effect on mutation frequency in female mice (all mutations were found in the progeny conceived during the first seven weeks after irradiation), the Committee in 1966 abandoned the doubling dose approach in favour of other methods, two of which will be mentioned here. In one, the estimated rate of induction of dominant visible mutations in mice (range: 10^{-8} to 10^{-7} per locus and rad) was multiplied by the assumed number of loci determining dominant disorders in man (50-500) to obtain the total risk ($5\ 10^{-8}$ to $5\ 10^{-5}$). In the other, the estimated rate of induction of recessive visible mutations in mice (10^{-7} per locus and rad) was multiplied by the estimated total number of gene loci in man (20,000) to obtain an estimate of total risk from the induction of these point mutations ($2\ 10^{-3}$). The risk to first generation offspring was then computed as a fraction (2-5 per cent) of the above figure.

90. In the UNSCEAR 1972 Report the interest of the Committee in the doubling dose method was revived but was given a low profile. The doubling dose was taken to be 100 rad, and the number of extra cases of severe hereditary diseases per million live births and rad of low-LET radiation was estimated to be about 300 for the irradiation of parental males; of these, six to 15 cases occurred in the first generation and the rest occurred in subsequent generations.

91. By 1977 new data on the natural prevalence of genetic and partially genetic diseases had been obtained. Furthermore, data that had been obtained in the mid-1960s on the induction of dominant mutations having their primary effect in the mouse skeleton had been extended in the mid-1970s, demonstrating transmission. By 1982, new data on the induction of another kind of dominant mutation, namely, those that cause cataracts in the eye of the mouse, became available. All these data allowed the Committee to arrive at direct estimates of genetic risks. It is worth noting that from 1977 onwards, both the doubling dose method and the direct method have been used.

92. In 1977, using a doubling dose of 100 rad, the Committee estimated that, if a population is continuously exposed to low-LET radiation at the rate of 1 rad per generation, there will be a total of about 185 cases of Mendelian, chromosomal and other diseases per million live births at equilibrium, of which about one third would appear in the first generation. The first-generation increase was estimated to be about one third of that at equilibrium.

93. These estimates, as well as those arrived at in the 1982 and 1986 Reports, are summarized in Table 1; for convenience, they are expressed on a per Sv basis. It can be seen that (a) for dominantly inherited diseases, the estimates have remained essentially unchanged, (b) the estimates for chromosomal diseases have become lower, this being a consequence of having excluded diseases attributable to numerical anomalies (such as Down's syndrome), for which there is still no good evidence of induction by radiation; and (c) while in 1977 and 1982 the Committee had provided estimates of risk for congenital anomalies and other multifactorial diseases using certain assumptions, in 1986, concerned about persistent uncertainties over the assumptions used, it no longer did so.

9–. The risk estimates made using direct methods from 1977 up to 1986, are given in Table 2; they include risks from (a) the induction of genetic changes having dominant effects in the first-generation progeny (i.e., dominant mutations, as well as recessive mutations, deletions and balanced reciprocal translocations with dominant effects) and (b) unbalanced products of balanced reciprocal translocations, which may lead to congenitally malformed children.

95. The first of these estimates (item (a) in the paragraph 94) is based on dominant skeletal and cataract mutations in mice and the second (item (b) in that paragraph) on primate cytogenetic data. The estimates based on experience in mice do not include induced genetic changes so severe as to cause death before they can be detected. It can be seen that the changes in risk estimates from 1977 to 1986 are relatively small. Furthermore, a comparison of these estimates with those arrived at using the doubling dose method (Table 1) for the first generation reveals that they are of the same order of magnitude, in spite of the different assumptions and reduction factors.

Table 1

Estimates of the risk of severe genetic disease per million live births in a population exposed to a genetically significant dose equivalent of 1 Sv per generation of low-dose-rate, low-dose irradiation, according to the doubling dose method
(based on UNSCEAR 1977, 1982 and 1986 Reports)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per million live births	Effect of 1 Sv per generation: First generation	Effect of 1 Sv per generation: Equilibrium
1977			
Autosomal dominant and X-linked	10000	2000	10000
Autosomal recessive	1100	Relatively slight	Very slow increase
Chromosomal (due to numerical and structural anomalies)	4000	3800	4000
Congenital anomalies and other multifactorial diseases	43000 / 47000	[illegible]	4500
1982			
Autosomal dominant and X-linked	10000	1500	10000
Autosomal recessive	2500	Relatively slight	Very slow increase
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3000	Probably very small	
Congenital anomalies and other multifactorial diseases	43000 / 47000	450	4500
1986			
Autosomal dominant and X-linked	10000	1500	10000
Autosomal recessive	2500	5	1500
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3400	Probably very small	
Congenital anomalies and other multifactorial diseases	60000 / 600000	Not estimated for reasons given in paragraph 106	

Note: The derivation of the above figures is given in Annex E; see also paragraph 93.

Table 2

Estimates of the risk of genetic disease in the first generation (for a genetically significant dose equivalent of 1 Sv) per million live births following low-dose-rate, low-dose exposure of the parental generation according to the direct method (Based on UNSCEAR 1977, 1982, and 1986 Reports)

Risk associated with	Expected frequency of genetically abnormal children in the first generation per million live births after irradiation of: Males	Females
1977		
Induced mutations having dominant effects	2000	None given
Unbalanced products of induced chromosomal rearrangements	750-1000	None given
1982		
Induced mutations having dominant effects	1000-2000	0-900
Unbalanced products of induced chromosomal rearrangements	30-1000	0-300
1986		
Induced mutations having dominant effects	1000-2000	0-900
Unbalanced products of induced chromosomal rearrangements	100-1500	0-300

Note: The derivation of the above figures is given in Annex C; see also paragraphs 86-95.

2. Cancer

96. As far back as in the UNSCEAR 1958 Report, the Committee emphasized that any attempt to evaluate the biological effects of radiation sources to which the world population is exposed can produce only tentative estimates, subject to wide margins of uncertainty. Despite these reservations, the Report included assessments of the annual numbers of leukaemia and bone cancer cases that could result from natural radiation and fallout. Data relating the incidence of leukaemia to radiation exposure came mostly from the atomic bomb survivors and patients suffering from ankylosing spondylitis.

97. At that time, the Committee estimated the total probability of leukaemia induction over 15 years to be 12 per million population per rem. It noted, however, that in Hiroshima the probability per unit dose decreased markedly with decreasing dose and that the incidence of leukaemia in that city did not appear to be linearly related to dose. The Committee also made what it called a crude estimate of the leukaemia risk to patients suffering from ankylosing spondylitis who had been treated with x-rays. Over 15 years, the risk of induction was estimated to be about 20 per million and rem. Over 35 years, which is the average remaining lifetime of the population and might be the period of risk under conditions of prolonged exposure at lower dose rates, the lifetime risk was assessed to be 52 per million and rem.

98. In discussing the assumed hypothesis of non-threshold linearity between dose and incidence of cancer, the Committee stated in the UNSCEAR 1962 Report that somatic effects were less likely to occur at low dose rates than at the high dose rates employed in many experiments. The only justifications for applying to low doses the relationships observed at higher doses were expediency and the consistency of the assumptions regarding mechanisms in both dose ranges. Nevertheless, the Committee could not say whether, in doing so, it was under- or over-stating the risk. For these reasons, it decided not to estimate absolute risks, but rather to present comparative risk estimates for the gonads (genetic effects), the bone marrow and the cells lining bone surfaces, based on the doses and dose commitments to these tissues from natural radiation sources, medical, occupational and miscellaneous exposure, as well as from nuclear testing.

99. Three basic questions needed to be addressed in the estimation of risk at low dose: the type of effect, the critical tissue for each type, and the function of dose, dose rate and dose distribution to be taken as the relevant parameter for each of the effects. For the somatic effects, the critical tissues were taken to be the active bone marrow and the connective tissue lining endosteal surfaces or trabeculae.

100. Although for genetic effects the experimental data justified an assumption of non-threshold linearity at low doses and dose rates, no such assumption could be made for late somatic effects, because tumour induction at high doses is a very complex function of dose and other exposure factors. Nevertheless, it would be expected that, at low dose levels, the mechanisms by which late effects are produced would be much simpler and any effects that could arise would result from specific changes induced in individual cells. For certain effects having a non-linear relationship at high dose levels, it was thought probable that the dose-effect curve near the origin would be linear. Thus, protraction of exposure and non-uniformity of dose distribution could be ignored. The Committee also discussed the importance of taking into account the way an effect manifests itself over time.

101. Referring to the problems of obtaining estimates of absolute risk, the Committee noted, in 1964, that it had earlier confined itself to estimating comparative risks except for leukaemia. After having reviewed the available information, the Committee saw no possibility of changing this procedure in the UNSCEAR 1964 Report. It immediately went on to state, however, that data published since 1962 had led it to believe that it would be possible, for a few tissues and mainly in the high-dose range, to make estimates of absolute risk that would be valid for the observed range of doses and the given conditions of irradiation. It was considered unlikely that the risk per unit dose at very low doses would be greater than that at higher doses; in fact, at low doses the risk was likely to be much less.

102. By 1964, tentative dose estimates had become available for some of the survivors from Hiroshima and Nagasaki, and the Committee believed that they were almost certainly not in error by a factor of more than 2 or 3. The new dose estimates made it possible to conclude that the annual incidence of radiation-induced leukaemia was approximately proportional to dose in the range from about 100 to 900 rad, with a proportionality factor between 1 and 2 cases per million and rad. The Committee warned that because the Japanese survivors might have been selected by the lethal effects of the irradiation itself, this estimate of risk could only be applied with caution to the general population. The estimate obtained from the atomic bomb survivors was consistent with that determined from subjects who had been irradiated therapeutically for ankylosing spondylitis, at doses between 300 and 1,500 rad. However, as the latter group was also highly selected, the estimate would apply strictly to spondylitic patients only.

103. New information suggested that for children irradiated in utero, the risk of leukaemia per unit dose could be several times higher than for adults. The doses received had been only a few rad, suggesting that under certain conditions, low doses could induce malignancy. As with the ankylosing spondylitis patients, there was the possibility that the irradiated children might not have been representative of all children.

104. A risk estimate for thyroid cancer was obtained from surveys on the induction of cancer as a result of irradiation of the thyroid region during childhood. In the range 100-300 rad, the Committee estimated the annual risk to be about one per million and rad, over approximately 16 years after irradiation. Once again, the Committee pointed out that the subjects might have been a highly selected group.

105. Irradiation was known to cause other malignancies, including tumours of the bone, liver, skin and lung; however, the information was not considered to be reliable enough for deriving risk estimates. The Committee was not optimistic about being able to obtain such estimates for all, or even many, types of human tissue. Indeed, it concluded that leukaemia might well be the predominant type of malignancy produced, and that the overall risk of all malignancies was unlikely to exceed by any large factor that of leukaemia.

106. In 1972, the Committee decided to review again the subject of radiation carcinogenesis in man. The review pointed out that, in order to assess the extent of radiation effects in man, it was essential to obtain empirical information from epidemiological studies. In evaluating such studies it would be necessary to bear in mind a number of inherent difficulties, such as those having to do with the size of the population studied, the dosimetry, the latent period, the relation to natural incidence of cancer, mortality versus morbidity statistics, the confounding effects of illness and the infrequency of true, uniform whole-body irradiation. The Committee discussed all of these points in detail and also considered the question of absolute and relative risks for the first time. It emphasized that the number of people exposed to substantial doses was so small that the relationship between dose and incidence of malignancies in man could be studied only for the most radiosensitive tissues.

107. Evidence on the induction of leukaemia indicated that its incidence increased with dose in the range 50-500 rad and that above this range the frequency tended to decrease, possibly owing to the cell-killing effect of high doses. Radiation-induced leukaemias tended to occur most frequently within a few years of exposure; after 25 years the frequency tended to return to normal, by which time some 15-40 cases per million and rad had been observed.

108. Lung cancers appeared to have been induced at Hiroshima by external gamma exposure at doses of some 30-100 rad. The data indicated a risk coefficient of from 10 per million and rad (at 250 rad) to 40 per million and rad (at 30 rad) during the first 25 years after exposure; this risk estimate was supported to some extent by data from patients treated for ankylosing spondylitis. The Committee noted that an estimate of risk could also be derived from data on uranium miners, but that not much reliance could be placed on such an estimate.

109. The Committee assessed the risk of induction for breast cancer among women exposed in Hiroshima as being between 6 and 20 cases per million and rad during the first 20 years after exposure and over a dose of 60-400 rad. These estimates refer to the 1965 dosimetry. For the induction of thyroid cancers an average risk coefficient was obtained of about 40 per million and rad over a dose of 60-400 rad. For all other malignancies, without clearly identifying their specific types, the Committee tentatively put forward a risk estimate for induction of 40 per million and rad

over the first 25 years after exposure to 250 rad. For a number of reasons, the Committee considered that these risk coefficients were likely to overestimate the risk of environmental exposures, that is, low-dose exposures from both natural and man-made sources.

110. The UNSCEAR 1977 Report also contained a major review of radiation carcinogenesis in man. After dealing extensively with the validity of the data on which risk estimates might be based, the Committee presented its estimates of risk coefficients for leukaemia and tumours in a number of organs. It noted that the risk of a malignancy developing at doses of about 100 rad might vary with the LET of the radiation, sometimes with the age and sex of the subject, and probably with the dose rate and the number of fractions with which the dose is delivered. In that Report the Committee for the first time referred to the induced mortality from leukaemia and other cancers. Previously it had always presented its risk estimates in terms of the incidence of cancer, not in terms of fatality.

111. The thyroid and the breast seemed to have the highest rates of induction, with risk coefficients of around 100 per million and rad. The low mortality rate for radiation-induced thyroid cancers and the moderately low rate for breast cancers were thought to bring the risk of fatality to about one tenth and one half of the incidence values, respectively. Lung cancer also had a high induction rate for males over 35, as judged from the experience of uranium miners. The Committee thought that for lung cancer a mean fatality risk coefficient for all ages of 25-50 per million and rad was probable.

112. The induction of leukaemia, specifically the acute and chronic granulocytic (but not chronic lymphatic) forms, appeared to decrease from about 50 per million and rad at moderately high doses to about 20 per million and rad at lower dose levels. The Committee was rather confident that this estimate would include all the cases likely to appear because, with radiation-induced leukaemia, the average interval between exposure and death appeared to be only about 10 years. With other cancers, which have latent periods of 25 years or greater, it was more difficult to estimate the total number of cases likely to be induced.

113. Risk coefficients were also presented for the stomach, liver and large intestine; brain and salivary glands, all of which had values in the region of 10-15 per million and rad; bone, oesophagus, small intestine, bladder, pancreas, rectum and lymphatic tissue, which had values of 2-5 per million and rad; and skin, for which both the risk of induction and the fatality rate were thought to be low.

114. The Committee also considered the question of estimating the total risk for all fatal malignancies from the observation that this might be four to six times that for leukaemia alone. At doses of a few rad, at which the lower leukaemia risk coefficient of about 20 per million and rad might apply, the total of all fatal induced malignancies, including leukaemia, could be about 100 per million and rad while it was assumed to be about 250 per million and rad at high doses. The risk coefficient for non-fatal malignancies was assumed to be about equal to that for the fatal malignancies. The Committee once again pointed out that the estimate for low doses was derived from mortalities induced at doses greater than 100 rad. The value appropriate to the dose levels involved in occupational exposure, and even more so in environmental exposures, might be substantially less.

115. It was likely that malignancies might be induced by exposure of the foetus in utero at average doses of 0.2-20 rad from diagnostic x rays. The induction rate was difficult to determine with any confidence but was estimated to be around 200 per million and rad.

116. In view of the limited amount of new epidemiological evidence available since the UNSCEAR 1977 Report, and because the dosimetric estimates for the survivors of the atomic bombing of Hiroshima and Nagasaki were in the process of being revised, the Committee decided not to review human carcinogenesis in the UNSCEAR 1982 Report. However, it said that it did not expect that the revisions would change the previous risk estimates by a factor of more than 2. The Committee's risk estimates up to 1977 for cancer are summarized in Table 3 where they are expressed per sievert in order to facilitate comparisons with later estimates.

3. Non-stochastic effects

(a) Irradiation of the adult

117. The Committee considered from time to time the somatic effects of radiation on laboratory animals and human subjects. These effects were first discussed in the UNSCEAR 1958 Report, which attempted to summarize 60 years of knowledge, at a time when information about radiation lesions and their pathogenesis was still rather scanty. Although the Committee had few details on which to base that discussion, the general picture that emerged seemed to be consistent, particularly for the effects induced by high doses. The Committee was aware at that time of the main physical factors affecting the induction of these effects, such as dose, dose rate, fractionation and radiation quality, and it also gave an account of the main biological factors, such as species, age, sex, and partial-body irradiation.

118. The main radiobiological concepts, such as that of cell sensitivity and tissue response, as they manifest themselves in the rate of cell division and differentiation, are to be found in the 1958 Report, although the concept of cell lethality could not be quantified because there were no techniques for single-cell culture. The term recovery was also used in a loose sense, without identifying the many underlying mechanisms. The classification of effects between morphological and functional gave rise to some problems, but the Committee identified, even at that early stage, the difficulties in settling the existence of thresholds, particularly with low doses and late effects.

Table 3

Summary of the Committee's estimates of fatal cancer risk coefficients
(per cent per Sv)

Tissue	1958 Report	1964 Report	1972 Report	1977 Report
Bone marrow	0.2-0.5	0.01-0.02 a/	0.15-0.40	0.20-0.50
Breast	-	-	0.06-0.20	0.50
Lung	-	-	0.10-0.40	0.25-0.50
Thyroid	-	0.16	0.40	0.10
Stomach	-	-		0.10-0.15
Liver	-	-		0.10-0.15
Brain	-	-		0.10-0.15
Salivary glands	-	-		(0.10-0.15)b/
Large intestine	-	-		0.10-0.15
Small intestine	-	-		(0.02-0.05)
Bone	-	-	0.40	(0.02-0.05)
Oesophagus	-	-		(0.02-0.05)
Bladder	-	-		(0.02-0.05)
Pancreas	-	-		(0.02-0.05)
Rectum	-	-		(0.02-0.05)
Mucosa of cranial sinuses	-	-		(0.02-0.05)
Lymphatic tissue	-	-		(0.02-0.05)
Skin	-	-		Low
Estimated total	-	-	-	1.0-2.5

a/ Per year.
b/ Numbers within parentheses refer to total incidence, the fatality risk not having been estimated.

119. Many of the same criteria were used in 1962 in classifying the somatic effects into early and late effects, with the result that effects very different in nature from tumours and leukaemia, such as lens opacification, induction of sterility or non-specific life shortening, ended up being classified together with them just because they also appeared late. The UNSCEAR 1962 Report contained no important departures from the generalizations described above, particularly with respect to the form of the dose-effect relationships, the uncertainties as to the precise form of these relationships at doses below those tested directly, and the pronounced dependence of the effects on the irradiation dose rate.

120. Twenty years elapsed between that Report and the next one, released in 1982, when an extensive Annex discussed the non-stochastic effects of radiation on normal tissues. The new treatment reflected the impressive advances in the understanding of somatic effects that had taken place during the interim. The very title of the Annex implied that there had been a re-classification of the effects into the stochastic and the non-stochastic. To the first class belong those effects for which only the probability of induction is a (linear) function of dose; to the second belong those effects for which severity (as well as probability, for a given severity) is a (sigmoid) function of dose. The Report discussed mainly the effects of irradiation of single tissues and organs; it reviewed a large body of human data interpreted in the light of experience gained in experimental animals.

121. The Committee considered the nature of these effects, their pathogenesis as it results from the interplay of cell killing and tissue kinetics, and the quantitative relationships between them and the time of appearance and degree of the non-stochastic clinical damage. The most general conclusions drawn by the Committee pertained to the existence of a dose threshold for the induction of these effects and the variability of this threshold according to the type of effect. The Annex also contained a detailed analysis of how the dose threshold for each specific type of effect would be expected to vary as a function of the important radiobiological variables such as radiation quality, dose, dose rate, dose fractionation and protraction.

(b) Pre-natal irradiation

122. The earliest mention that the tissues of the embryo and foetus could be particularly sensitive to the action of radiation and that the exposure of pregnant mothers might cause teratological effects to be induced in the product of conception dates from the first UNSCEAR Report (1958). Also, the fact that there are critical periods in development, during which some structures may be particularly vulnerable to the specific action of internal or external irradiation, was already recognized at that time. Finally, it also discussed the shape of dose effect relationships for effects in utero, without specifying the nature of the effects or their induction mechanisms, although implying that the relationships would be of the threshold type.

123. The UNSCEAR 1962 Report reiterated the notion of the special sensitivity of embryonic and foetal structures, pointing out that minor injuries during development could be amplified by the growth

of the relevant structures to produce major anomalies. From data on the pre-implanted mouse it was inferred that doses of 0.25 Gy to the embryo could be lethal to 40 per cent of the animals. The Committee also concluded, on the basis of the fairly large set of experimental results then available, that irradiation during major organogenesis would cause developmental malformations and that there was a good correspondence between the malformed structures of animals and man for corresponding stages in development. In man, malformations were found more frequently in the central nervous system, the eye and the skeleton.

124. In the context of a special discussion of the effects of radiation on the nervous system, contained in the UNSCEAR 1969 Report, the Committee paid special attention to the damage caused in the brain structures of the developing mammal. It confirmed that pre-natal irradiation during the time when the relevant structures are undergoing differentiation could produce severe developmental anomalies. Depending on the time of the irradiation, specific anomalies (microcephaly, encephalocele, hydrocephalus) could be produced in man, probably following threshold-type kinetics as a function of dose. Disorganization of the cortical architecture was described in animals, accompanied by functional impairment in the form of loss of visual, olfactory and distance discrimination. Other learning processes were impaired in animals after doses of 1 Gy or more had been administered during the second or third week of pregnancy in rats; effects of doses below 0.5 Gy were regarded as uncertain. Although changes in conditioned reflexes had been described in animals irradiated near-term with doses as low as 0.01 Gy, the relevance of these effects to risk estimation in man was also doubtful. In man, the Committee recognized small head size and the induction of mental retardation as true effects, but it could not detect any correlation between such morphological and functional abnormalities and structural changes in the central nervous system. The Committee even ventured to derive a risk coefficient for mental retardation in the survivors of Hiroshima and Nagasaki: 1 per thousand and rad for doses over 50 rad delivered at high dose rates.

125. Recognizing the importance of keeping the effects of radiation on growth and development under observation because of their relevance to the general population and to female workers, the Committee undertook another review of this subject in Annex J of the UNSCEAR 1977 Report. This review centred on experimental animal data, which was the only information available, and on the mechanisms whereby effects are induced in utero; it also described dose-time relationships obtained from the more quantitative data.

126. The Annex J of the UNSCEAR 1977 Report generalized the so-called "periods of maximum sensitivity" of the various anatomical structures, to coincide with the growth spurt; it also generalized across species the notion that lethal effects were typical for the pre-implantation period, teratogenic effects for the major organogenesis period and growth disturbances for the foetal period. An analysis of the dose-effect relationships showed that these were mostly curvilinear. The Committee confirmed its previous risk assessment for mental retardation and suggested, on the basis of mouse data, that the risk coefficient for the increment of embryonic killing soon after fertilization could be taken at 1 per cent per roentgen.

127. From this review the Committee concluded that although data in man on the induction of malformations by radiation were very scarce, the data on other animal species were so unanimous and uniform in indicating a pronounced sensitivity to such effects that the human species could not be regarded as an exception. While the Committee found it impossible, given the paucity of human data, to derive reliable, quantitative estimates of risk from pre-natal human irradiation at comparable developmental stages, particularly at low doses and dose rates it could on the basis of experimental animal data exclude that the sensitivity of the human species might be a factor of 10 higher than expected.

4. Other types of harm

128. At various times and in different Reports, the Committee gave special attention to types of harm not easily classifiable into one of those treated above. One such harm is the shortening of life-span, which was said in the UNSCEAR 1958 Report to result from a number of acute or late radiation-induced changes, both specific, such as leukaemia in radiologists, or pathologically diffused in all organs or tissues. These latter conditions were thought to accelerate the normal aging processes and so were termed non-specific, life shortening.

129. The Committee carried out a special study of the so-called aging effects of radiation and presented the results in the UNSCEAR 1982 Report. There seemed to be insufficient grounds to define aging in precise, biological terms, which would allow postulating non-specific effects of radiation at low doses and dose rates that might cause an animal to prematurely age. The Committee therefore focused on the life-shortening action of radiation, an effect that can be more objectively defined. At the doses of greatest interest for practical purposes, that is, those well below the LD_{50} range and down to the smallest doses and dose rates, evidence showed overwhelmingly that irradiated animals live, on the average, fewer years than non-irradiated controls.

130. This life-shortening effect has precise relationships with dose and time. A very large body of evidence in experimental animals allowed the Report to conclude that at low to intermediate doses and dose rates, life shortening is essentially due to the induction of malignancies at a rate above the natural rate characteristic of the species investigated. This conclusion applies to experimental animals and, as far as could be judged from limited human experience, also to man.

131. In the UNSCEAR 1969 Report, the Committee presented a special study of the effects of radiation on the nervous system. That review also covered aspects of morphological and functional disturbances produced by irradiation during the pre-natal stages. Irradiation of the nervous system can cause effects in adults only at high doses, in which case there are profound structural and functional alterations. It was recognized, however, that for doses as low as 0.1 Gy or less, reactions of a "physiological nature" could be induced. The most remarkable finding remained the striking difference in sensitivity between the pre- and post-natal stages, the former being much more vulnerable than the latter.

132. The same Report contained a separate Annex on the induction of chromosomal aberrations in human germinal and somatic cell lines. The induction of chromosomal aberrations in somatic cells is an interesting effect by virtue of its potential use as an in vivo dosimeter and its biological significance with respect to the causation of (or correlations with) induction of malignancies. The Annex covered in depth the dose-time relationships for the induction of chromosomal damage and the variability of aberrations as a function of other physical and biological agents. It concluded that, aside from its practical applications in biological dosimetry, chromosomal analysis could be of little use in assessing the risk of neoplastic, immunological or life-shortening effects of radiation. Risk estimates would continue to be based on the observed incidences of the specific clinical conditions as a function of dose, a conclusion that remains true to this day.

133. The UNSCEAR 1972 Report contained a special study on the effects of radiation on the immune response wherein the Committee, mostly on the basis of experimental data, tried to discuss the role the immune system plays in the development of early and late radiation effects, essentially those of the non-stochastic type. The study concluded that the immune system has large, built-in safety factors that allow it to withstand and recover from substantial injury by radiation. The Committee reported that at whole-body doses around 0.1 Gy, damage to the immune system could be observed but that such damage did not cause great concern. Whole-body doses higher by an order of magnitude could increase susceptibility to infection, while doses of 2 or more Gy could significantly increase the risk of mortality from infection. For non-stochastic effects, these conclusions still appear to be valid.

134. Another special study was carried out of the possible interaction between radiation and other agents that are widely distributed in the environment. This study too, was contained in the UNSCEAR 1982 Report. In it, the Committee paid particular attention to exposure conditions that affect large numbers of people, thereby substantially changing average risk coefficients.

135. The Committee found that for effects of wide practical significance (induction of cancer, genetic effects or developmental abnormalities), there was little systematic information to substantiate claims of non-additive interactions between radiation and other agents. The theoretical analyses, which were accompanied by illustrative examples from experimental or epidemiological work, treated this matter in all its complexity. The different natures of the interacting agents, their different mechanisms of action, the different dose levels and the different ways of administering the doses could all give rise to a variety of possible interactions, in the additive, inhibiting or synergistic sense, but only one case of synergism appeared to be well documented, that between tobacco smoke and radon decay products in uranium miners. This synergism prevents the direct extrapolation of findings in the miners to the general population.

II. THE PRESENT SITUATION

136. This chapter describes the Committee's findings and conclusions in its most recent Reports. For the most subjects the latest account is the one contained in the present (1988) Report, but for some subjects that are not reported here, e.g., exposures from nuclear explosions, the latest account is contained in the UNSCEAR 1982 Report.

A. RADIATION LEVELS AND DOSES

1. Natural sources of radiation[a]

137. The assessment of the radiation doses to humans from natural sources is of special importance because natural radiation is by far the largest contributor to the collective dose received by the world population. The natural radiation sources are classified into: (a) external sources of extraterrestrial origin (that is, cosmic radiation) and radiation of terrestrial origin (that is, the radioactive nuclides present in the crust of the earth, in building materials and in air) and (b) internal sources, comprising the naturally occurring radionuclides that are taken into the human body.

138. Some of the contributions to the total exposure from the natural radiation background are quite constant in space and time and practically independent of human practices and activities. This is true, for example, of the doses received from the ingestion of potassium-40, an element that is homeostatically controlled and also of doses from the inhalation and ingestion of cosmogenic radionuclides, which are relatively homogeneously distributed over the surface of the globe. Other contributions depend strongly on human activities and practices and are therefore ... ly variable. The doses from indoor inhalation of ...don and thoron decay products are examples: building design, as well as the choice of building materials and of ventilation systems, influences the indoor levels, so that as techniques and practices evolve, the doses received from radon will also change. Between those extreme types of exposure, there are some intermediate types: external doses from cosmic rays, which are affected by human practices and are quite predictable but uncontrollable (except by moving to an area where the dose is lower); doses from the inhalation and ingestion of long-lived nuclides of the uranium-238 and thorium-232 decay series, which make a small contribution to the total dose from natural sources and are relatively constant in space; and doses from external irradiation by terrestrial sources, which are also significantly altered by human activities and practices, especially through indoor exposure.

[a]This subject is reviewed extensively in Annex A, "Exposures from natural sources of radiation".

139. The Committee has re-assessed the doses received globally from natural radiation sources (Table 4). The mean annual effective dose equivalent is estimated to be 2.4 mSv; it refers to the adult part of the population. Variation around this mean is due mainly to variations in the external exposure to terrestrial sources and in the internal exposure (inhalation) to short-lived decay products of radon isotopes. The external exposures typically vary around the mean by a factor of 1.5 and the internal ones by a factor of 2.5. For both types of exposure, the extreme values vary around the mean by a factor of 100.

Table 4

Annual effective dose equivalent from natural sources

Source of irradiation	Annual effective dose equivalent (mSv) External		Internal	Total
Cosmic rays				
Directly ionizing component	0.30		-	0.30
Neutron component	0.055		-	0.055
Cosmogenic radionuclides	-		0.015	0.015
Primordial radionuclides:				
Potassium-40	0.15		0.18	0.33
Rubidium-87	-		0.006	0.006
Uranium-238 series:	0.1			1.34
Uranium-238 to uranium-234		0.005		
Thorium-230		0.007		
Radium-226		0.007	1.24	
Radon-222 to polonium-214		1.1		
Lead-210 to polonium-210		0.12		
Thorium-232 series:	0.16			0.34
Thorium-232		0.003		
Radium-228 to radium-224		0.013	0.18	
Radon-220 to thallium-208		0.16		
Total	0.8		1.6	2.4

140. There are several changes from the estimates given in the UNSCEAR 1982 Report: (a) for external exposure to cosmic radiation, the new estimate of the annual effective dose equivalent is higher by 50 microsievert, from taking into account the geographical distribution of the world population as a function of altitude as well as the shielding effect of the building materials; (b) for external exposure to terrestrial sources of radiation, the estimate of the annual effective dose equivalent has been raised by 60 microsievert as a result of a better knowledge of the indoor gamma absorbed doses in air; (c) the estimates of the annual effective dose equivalents from internal exposure to primordial radionuclides have been slightly decreased for the uranium-238 and lead-210 series as well as for the decay products of radon-220, whereas those for the short-lived decay products of radon-222 have been increased by about 300 microsievert on the basis of the results of nation-wide indoor surveys. The net effect of these corrections is a 20 per cent increase in the estimate of the annual effective dose equivalent from all natural sources of radiation.

141. Table 4 shows the paramount importance of doses from the inhalation of radon-222 and its short-lived decay products. Industrial activities that release materials with enhanced concentrations of naturally occurring radionuclides do not significantly alter the overall exposure estimates.

2. Nuclear explosions

142. In the UNSCEAR 1982 Report, the Committee assessed the exposures to the world's population from the release to the environment of radioactive materials produced in nuclear explosions carried out in the atmosphere since 1945. Since no atmospheric nuclear tests have taken place since 1980, the assessment remains complete and valid.

143. The number and yield of atmospheric nuclear explosions are summarized in Table 5, which shows that the most test programmes took place during 1957-1958 and 1961-1962. Large-yield explosions carry radioactive debris into the stratosphere, from where it is dispersed and deposited around the world (this is known as stratospheric radioactive fallout). Exposures to populations are highest in the temperate regions and in the northern hemisphere, where most of the testing occurred. The dose commitment for the southern temperate zone is about 70 per cent of that for the northern temperate zone. The radiation doses are due mostly to the ingestion of radionuclides that have become incorporated in foods and to external irradiation from ground deposition.

Table 5

Number and yield of atmospheric nuclear explosions

Year	Number	Estimated yield (Mt) Fission	Total
1945-1951	26	0.8	0.8
1952-1954	31	37	60
1955-1956	44	14	31
1957-1958	128	40	81
1959-1960	3	0.1	0.1
1961-1962	128	102	340
1963	0	0.0	0.0
1964-1969	22	10.6	15.5
1970-1974	34	10.0	17.2
1975	0	0.0	0.0
1976-1980	7	2.9	4.8
1981-1987	0	No further tests	

144. The most significant radionuclides contributing to the assessed dose commitments for various parts of the world from all atmospheric tests carried out so far are, in decreasing order of importance: carbon-14, caesium-137, zirconium-95, strontium-90, ruthenium-106, cerium-144 and tritium. Residual irradiation from only four of these, carbon-14, caesium-137, strontium-90 and tritium, remains to be received by the present and future world population. An additional contribution of about 0.1 per cent of the total effective dose equivalent commitment will be received from plutonium-239, plutonium-240, and americium-241 at very low dose rates over thousands of years.

145. The collective effective dose equivalent commitment due to all atmospheric nuclear explosions was estimated in the UNSCEAR 1982 Report to be $3\ 10^7$ man Sv, an estimate that is still valid. This value, which takes into account projected future growth of the population of the world, was found to be equivalent to about four years of exposure to natural sources for the population of the late 1970s, on the basis of an annual per caput exposure to natural sources of 2 mSv and a world population of $4\ 10^9$. Owing to the increase in the world population to about $5\ 10^9$ at the present time and to the revised estimate, 2.4 mSv, for the annual per caput exposure to natural sources, the collective effective dose equivalent commitment due to all atmospheric nuclear explosions is now assessed to be equivalent to about three years of exposure to natural sources for the present population.

3. Nuclear power production[b]

146. The number of nuclear reactors being operated to generate electricity has increased since the UNSCEAR 1982 Report. At the end of 1987, the 417 reactors operating in 26 countries had an installed capacity of 298 GW. This represents a 100 per cent increase in capacity since the Committee last reported in 1982, when installed capacity was 144 GW. Projections to

[b]This subject is reviewed extensively in Annex B, "Exposures from nuclear power production".

the year 2000, although still somewhat speculative, amount to around 300 GW, a further growth of 80 per cent from present capacity.

147. The nuclear fuel cycle includes several steps: mining and milling of uranium ores; enrichment of the isotopic content of uranium-235 for some types of reactors; fabrication of fuel elements; production of energy in the reactors; reprocessing (although this is not always undertaken) of irradiated fuel and recycling of the fissile and fertile nuclides recovered; transportation of nuclear materials between fuel cycle installations; and, finally, the disposal of radioactive wastes. Although most of the radioactive materials associated with nuclear power production are present in the irradiated fuel, small amounts are released to the environment in effluents at each of the steps in the cycle. Most of these releases are only of local and regional concern, because the radionuclides have short half-lives and are limited in their environmental mobility. However, some nuclides, because of their long half-lives or rapid transfer through the environment, may contribute to the irradiation of man on a global scale.

148. For each step in the fuel cycle and its associated release of radioactive materials, the Committee has evaluated the doses to workers within nuclear installations and to members of the public. In its evaluations, four population groups have been considered: those exposed in normal conditions because of their work within the fuel cycle; the population living within about 100 km of the plant; the population within a few thousand kilometres; and, finally, the world population.

149. The concentrations of radionuclides in effluents are generally low, and it is hardly feasible and not practicable to monitor members of the population for uptake of radionuclides. Instead, environmental modelling has been used by the Committee to estimate doses at long distances from the plant. The transfer of radionuclides through environmental media can be predicted from measured values obtained by monitoring foodstuffs and water and from experimental studies.

150. The starting point for environmental modelling at long distances is data on the quantities and composition of radioactive materials emanating from various nuclear installations. This information is usually available to the Committee from those countries having nuclear power programmes and has been collected for the six-year period 1980-1985. Since the size of a particular stage in the nuclear fuel cycle is proportional to the nuclear generating capacity served by the stage, the releases have been normalized per gigawatt year of generated electric energy, enabling comparisons to be made and to facilitate the use of averages over all plants of a similar conceptual design; the results are not representative of a specific site, but they do give an idea of the impact of each type of facility. Averaging over all energy production and for all plants of a particular type accounts also for releases that may arise during maintenance shutdowns, when little or no electricity is generated.

151. To assess the collective doses corresponding to the normalized releases, the Committee had previously specified hypothetical sites with broadly representative characteristics for each stage of the fuel cycle: mining and milling, enrichment and fabrication, reactor operation and reprocessing. The Committee also assumed that the environment receiving the releases from each model facility was a hypothetical environment containing the main features of existing sites, so that the most common pathways to man are included. The Committee has used the same models again because it believes they are still adequate for the purpose and because doing so allows the current impact to be compared with the previously assessed impact of 1974-1979.

152. Uranium mines give rise to effluents, which when operating consist mainly of ventilation air in the case of underground mines and of releases into the pit in the case of surface mines. Further effluents are produced during milling operations to extract the uranium. The stockpiles of ore and other extracted materials are the source of airborne emissions when the mine is operating, and this source persists even after the mine has been closed. The tailings that are discharged from the mills also become long-term sources of airborne emissions. The most important radionuclide in all these airborne releases is radon-222. Using the same general models as in the UNSCEAR 1982 Report doses have been assessed both for the operational period and for the long term (10^4 years). Doses from fuel fabrication and transport have also been assessed, but since these are so much smaller than the doses from other components of the nuclear fuel cycle, they are not considered separately.

153. During operation of nuclear power stations and reprocessing plants, solid wastes are produced and have to be disposed of. For purposes of analysis, these wastes have been characterized in terms of volumes and activity concentrations of important radionuclides per unit energy generated. Two typical disposal facilities of the shallow land burial type were specified and terrestrial dispersion models used to calculate the release rates of radionuclides and the resulting effective dose equivalents.

154. The only operating commercial fuel reprocessing plants are at Sellafield in the United Kingdom and at Cap de la Hague and Marcoule in France. The Committee assessed in the UNSCEAR 1982 Report the impact of reprocessing using a notional plant representative of plants that would be reprocessing oxide fuel in the future. At present the throughput of fuel at the three reprocessing plants represents an energy output equivalent to about 5 per cent of that generated by nuclear power. The Committee has therefore decided to assess the impact of the actual reported discharges from these commercial reprocessing plants and weight the resulting collective doses by the fraction of fuel reprocessed to obtain values of exposure per GW year generated.

155. Calculations of collective dose to the world's population and various subgroups require assumptions to be made about the size of these populations, their dietary and other habits, and agricultural and fishing practices. The broadly representative values of these parameters previously used by the Committee have been retained to evaluate the radiological impact of each stage of the fuel cycle.

156. The estimates of collective effective dose equivalent to local and regional populations and to the global population from widely dispersed radionuclides are given in Table 6. Occupational exposures per GW year are approximately three times those received by the local and regional population.

157. Estimates of dose to the public have been reduced, partly because discharges to the environment from reactors have generally decreased and also the estimate for carbon-14, which accounts for half the public exposure from routine reactor releases, is much lower than the estimate in the UNSCEAR 1982 Report due to new, lower measured values of carbon-14 releases from heavy-water reactors.

158. The annual exposure received by the world's population from the release of radionuclides that become globally dispersed is currently much less than that received by local and regional populations. Only if the current levels of discharge of these radionuclides continued and all fuel from all reactors were reprocessed could the global component of the annual collective effective dose equivalent eventually equal the local and regional components.

159. The collective and per caput doses from nuclear power production may be compared to the doses to the world population from natural sources of radiation. The more immediately delivered component of the normalized collective effective dose equivalent commitment has been estimated to be 4 man Sv per GW a from radionuclides in the effluents of nuclear fuel cycle installations. For the present annual nuclear power production of about 190 GW year, the annual collective dose is assessed to be 760 man Sv. Dividing by the world population of $5\ 10^9$ gives an annual per caput dose estimate of 0.15 microsievert. The doses are around 0.01 per cent of the collective and per caput doses from natural background sources.

4. Medical exposures

160. Good data on the frequency of examinations and absorbed doses from medical examinations come mainly from the well-developed countries, which comprise less than 25 per cent of the world's population. There are fragmentary data on examination rates or number of diagnostic units and little or no data on absorbed doses for approximately another 25 per cent of the population. For 50 per cent of the world's population there are no data at all. For this reason, the Committee has developed a modelling approach based upon the good correlation that exists in most countries between population per physician (about which there is more information) and the medical uses of radiation.

161. Access of populations in the world to radiodiagnosis is very uneven: one x-ray machine is shared by fewer than 2,000 people in some countries and by 100,000-600,000 people in other countries. The frequency of procedures is also very uneven: 15-20 procedures per year are carried out per 1,000 population in some countries and 1,000-2,000 procedures per year in others. At the present time, there are about $5\ 10^9$ people in the world, and some estimates are that more than three quarters of the world's population have no chance of receiving any radiological examination, regardless of what disease they have.

162. While absorbed dose data exist for many standard radiographic and nuclear medicine procedures, information now available suggests that the previous absorbed dose estimates for the world population may be somewhat low. An important reason for this is the widespread use of fluoroscopy in developing countries. There are also large numbers of malfunctioning machines, which produce high doses. Neither of these factors was widely appreciated in the past.

163. The collective effective dose equivalent from diagnostic x-ray procedures is far greater than that from dental or diagnostic nuclear medicine examina-

[This subject is reviewed extensively in Annex C, "Exposures from medical uses of radiation".

Table 5

Collective dose per unit practice of nuclear power generation
(man Sv per GW a)

	Over next 100 years	Over all time
Mill tailings (radon), long term	1.5	150 a/
Globally dispersed nuclides and waste	6	60
Local and regional exposures	4	4
Occupational exposures	12	12
Total	24	230

a/ Over 10,000 years.

tions. The per caput annual effective dose equivalent is likely to be no lower than 0.4 mSv (the Committee's previous estimate) and may be as high as 1.0 mSv. Similarly, the annual genetically significant dose may range from 0.1 to 0.3 mSv. However, considering the age structure of the population, the effective dose equivalent may overestimate the detriment. This would be particularly true in countries where the older portion of the population receives most of the medical irradiation.

164. The world-wide collective effective dose equivalent is estimated to be between 2 and 5 10^6 man Sv. Of this, 90-95 per cent is attributable to diagnostic x-ray procedures. Dental radiography, nuclear medicine and radiation therapy (ignoring target doses) together contribute only 5-10 per cent of the collective dose. In developed countries, the contribution to the collective effective dose equivalent is about 0.001 man Sv per examination.

165. There are many possibilities for reducing dose without jeopardizing the benefits of the radiological practices. In the developed countries, it may be possible to reduce the per caput effective dose equivalent by half. In the less-developed countries, the use of radiography rather than fluoroscopy, appropriate collimation, proper film developing, as well as the calibration and maintenance of equipment, would reduce the dose per examination; however, the feasibility and costs of these measures are not known. The genetically significant dose can be significantly reduced through the use of gonadal shielding, a practical, low cost method. Still, the collective effective dose equivalent may increase as x-ray examinations become more widely available to a number of countries, and such an increase may in fact be appropriate.

166. The frequency and total use of medical irradiation is expected to increase over the next several decades because of the aging of the world's population, the growth of this population, and urbanization in the developing countries. By the year 2000, the collective dose will probably have increased by 50 per cent, and by 2025 it may have more than doubled.

5. Occupational exposures[a]

167. Two categories of workers are exposed to radiation: workers in the nuclear industry and in the medical field, where radiation sources are managed, and workers in occupations where higher background radiation levels are encountered (air crews and non-uranium miners are examples). The Committee gave a full assessment of occupational exposures in the UNSCEAR 1982 Report. Updated estimates of exposures to workers in nuclear fuel cycle activities (average annual doses in the range of 3 to 8 mSv for reactor operation, and a collective dose of 12 man Sv for each GW year of electric energy generated, in total for all work in the whole nuclear fuel cycle, cf. Table 6) and to medical personnel (average annual doses in the range of 0.3 to 3 mSv, and a collective dose of about 1 man Sv per million of population, cf. also paragraph 166; in developed countries an average occupational dose of about 1 microsievert per examination) are included along with exposures of the general public in the respective Annexes dealing with these subjects.

[a]This subject is reviewed in Annex B, "Exposures from nuclear power production" and in Annex C, "Exposures from medical uses of radiation".

168. Exposures of radiation workers are subject to detailed regulatory control in all countries and in the majority of cases the doses are but a small fraction of established limits, partly as a result of the current emphasis on optimizing radiation protection. The collective effective dose equivalent commitment per unit of electricity generated to workers in all nuclear fuel cycle installations is estimated to have changed little from the commitment previously estimated by the Committee, but such stability is only to be expected if reductions in exposures are balanced by the greater numbers of workers employed in the expanding industry.

169. Occupational exposure from medical practices includes the contributions from diagnostic x-ray procedures, dental radiography, nuclear medicine and radiation therapy. The average annual collective effective dose equivalent from occupational exposures in these practices is about 1 man Sv per 10^6 population. In spite of the increase in the medical uses of radiation in most countries, the limited trend data indicate that both individual and collective annual occupational doses are decreasing by 10-20 per cent every decade. For developed countries, the average occupational exposure is about 1 microsievert per examination.

6. Miscellaneous exposures

170. Exposures from miscellaneous sources of radiation are evaluated by the Committee whenever warranted by new information or new developments. The latest assessment, in the UNSCEAR 1982 Report, dealt with various consumer devices that contain radioactive materials and with electronic and electrical equipment that emit x rays. Individual exposures to these various sources were generally very small. The Committee believes that assessment to be still valid and feels that no new evaluation is required.

7. Accidents

171. With the large size of the nuclear industry in some countries and the large number of radiation sources used for industrial and medical purposes, accidents are bound to happen. The accidents that have occurred have generally been criticality and other industrial accidents that exposed one or a few workers, transport accidents, including also accidents involving satellites, aircraft and submarines, losses or thefts of radiation sources, and reactor accidents.

172. Three reactor accidents have caused measurable exposures of the public: Windscale in 1957, Three Mile Island in 1979, and Chernobyl in 1986. The Chernobyl nuclear reactor accident was a significant event and is discussed in detail in two Annexes (Annex D, "Exposures from the Chernobyl accident" and Annex G, "Early effects in man of high doses of radiation").

173. In all, six notable accidents have occurred since 1982, when the Committee last dealt with this subject:

1983: Constituyentes, Argentina. An accidental prompt critical excursion occurred during a configuration change in a critical assembly, resulting in the death of an operator, who was only 3-4 metres away. The dose to the victim was estimated to be 5-20 Gy from gamma rays and 14-17 Gy from neutrons.

1983: Ciudad Juarez, Mexico. An improperly disposed of cobalt-60 source found its way into a scrap metal shipment, contaminating the delivery truck, the roadsides and the processed steel into which the scrap was incorporated. Some 300-500 individuals were exposed, ten to doses of 1-3 Gy. There were no deaths.

1984: Mohammedia, Morocco. A source of iridium-192 used to make radiographs of welds at a construction site became detached from the take-up line to its shielded container. The source dropped to the ground and was noticed by a passer-by, who took it home. Eight persons, an entire family, died from the radiation overexposure with doses of 8-25 Gy.

1986: Texas, United States. An accident at a linear accelerator caused two deaths from overexposure.

1986: Chernobyl, USSR. The accident at the nuclear power station resulted in two immediate deaths of reactor operating personnel from the explosion. About 145 firemen and emergency workers suffered acute radiation sickness; 28 of them died during the three months following the accident. There were 30 deaths in all; one worker died from mechanical injury and one from burns. Local residents, none of whom received high exposures, were evacuated. The widespread dispersion of the released materials caused low exposures, primarily to populations of the western part of the USSR and other European countries.

1987: Goiania, Brazil. A caesium-137 source was dismantled in a residential area causing some 240 people to become contaminated. Fifty-four of them were hospitalized and four died.

8. The Chernobyl accident[b]

174. The accident at the Chernobyl nuclear reactor in the USSR, which occurred on 26 April 1986, caused extensive contamination in the local area and resulted in radioactive material becoming widely dispersed and deposited in European countries and throughout the northern hemisphere. The extent to which such a wide region could be affected by an event of this type was unanticipated. Intensive monitoring was undertaken to evaluate the radiation levels.

[b]This subject is reviewed extensively in Annex D, "Exposures from the Chernobyl accident".

175. It was apparent soon after the arrest of releases from the reactor that the radiological impact of the accident, from the point of view of individual risk, would be insignificant outside a limited region within the USSR, either because contamination levels were generally low or because remedial actions to ban the consumption of particularly contaminated foodstuffs prevented high exposures.

176. The accident at the Chernobyl reactor occurred in the course of a low-power engineering test, during which safety systems had been switched off. The uncontrollable instabilities that developed caused explosions and fire, which damaged the reactor and allowed radioactive gases and particles to be released into the environment. The fire was extinguished and the reactor core sealed off by the tenth day after the accident.

177. The death toll within three months from the accident was 30 members of the reactor's operating staff and the fire-fighting crew. Two died immediately, 28 died from radiation injury. Radiation doses to the local population were well below the doses that could cause immediate effects. Local residents were evacuated from a 30 km exclusion zone surrounding the reactor. Agricultural activities were halted and a large-scale decontamination effort has been undertaken.

178. The initial release of radioactive materials from the accident spread with winds, in a northerly direction. Subsequent releases dispersed towards the west and south-west and in other directions as well. Deposition on to the ground was governed primarily by rainfall, which occurred sporadically at the time in Europe. The deposition pattern and the associated transfer of radionuclides to foods and irradiation of individuals was very inhomogeneous, necessitating a regional approach for dose calculations.

179. Measurements since the accident have shown that the radionuclides contributing most significantly to doses are iodine-131, caesium-134 and caesium-137 mainly by external irradiation from deposited material and by ingestion of contaminated foods. The Committee's dose assessment takes most account for these important radionuclides and pathways.

180. Detailed information was available to the Committee to calculate first-year radiation doses in the USSR and all European countries. To extend these results and to estimate the projected doses from deposited materials, wider regions were evaluated. Since there is insignificant interhemispheric mixing of material released into the troposphere, southern hemisphere countries could only have been affected through imported food; this possibility is accounted for in the assessment by considering total food production as well as local consumption in northern hemisphere countries.

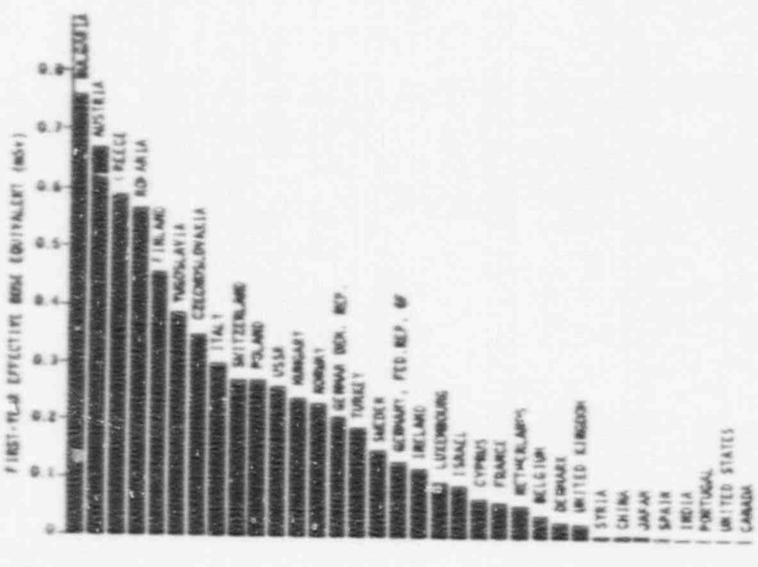


Figure I. Country average first-year committed effective dose equivalent from the Chernobyl accident.

181. The input values for the calculation made full use of measurements during the first year following the accident. Thereafter, projections are required to estimate the further contributions to dose, primarily from ^{137}Cs. The projections are based on experience acquired from past studies of radioactive fallout from the atmospheric testing of nuclear weapons.

182. The results of calculations of the first-year committed effective dose equivalents in 34 countries are illustrated in Figure I. The highest values are for Bulgaria, Austria, Greece and Romania, followed by other countries of northern, eastern and southeastern Europe. Countries further to the west in Europe and also countries of Asia, North Africa, North and Central America were less affected, which is in accord with the deposition pattern.

183. The dose commitments from the accident are delivered over several years, mostly due to continuing exposures from caesium-137. On average, some 30 per cent of the effective dose equivalent commitments were delivered in the first year following the accident. The dose commitments over all time in wider regions of the world are illustrated in Figure II.

184. The main outcome of the dose assessment is the collective effective dose equivalent commitment. This is estimated to be approximately 600,000 man Sv. Of this amount, 40 per cent will be received in the USSR

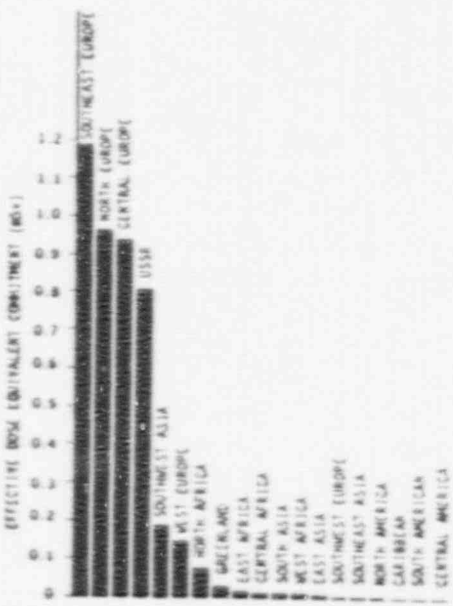


Figure II. Regional average effective dose equivalent commitment from the Chernobyl accident.

and 57 per cent in the rest of Europe. The remaining 3 per cent will be received by other countries of the northern hemisphere.

185. For comparison with Figure I, the one year effective dose equivalent from natural sources is 2.4 mSv. For comparison with Figure II, it should be noted that most of the dose commitment will be received within 30 years of the accident. The 30-year effective dose equivalent from natural sources is about 70 mSv. In using these comparisons, it should be remembered that the doses are averages over large geographical areas within which there will be local variations, in the doses from Chernobyl and those from natural sources.

B. RADIATION EFFECTS

1. Hereditary harm[7]

186. In spite of the considerable progress made during the past few years in understanding the mutation process, there have been no major conceptual changes in the formulation of risk estimates between the UNSCEAR 1986 Report and the present one that would warrant revising the estimates of natural or radiation-induced Mendelian and chromosomal disorders using the doubling dose method. However, an attempt has been made to quantify risks of induction of recessive diseases by this method. New data on the prevalence of congenital anomalies and other disorders of complex aetiology (discussed in 1986) raise a number of questions: Can the doubling dose of 1 Gy be confidently applied to disorders of complex aetiology? What is the magnitude of the mutational component of these disorders? Is it meaningful to provide estimates for these disorders in the continuing absence of experimental or human data bearing on the mechanisms of their maintenance in a population and on their possible response to radiation? Until new data become available, the Committee concluded that it was unable to provide meaningful risk estimates for these disorders. However, even with extreme assumptions (e.g., a 100 per cent mutational component) the risk of severe hereditary harm in the first generation of offspring to the exposed individual does not appear to be higher than the present estimate of the cancer risk. Since this situation remains true in 1988, the risk estimates for hereditary effects that the Committee offers at the present time are those shown in Table 7.

187. Using direct methods, the Committee estimated 10-20 per 10^{-2} Gy per million live born as having genetic diseases caused by induced dominant mutations. The Committee also estimated about 10 extra cases of genetically abnormal children would be expected in the first 10 generations per million live births per 10^{-2} Gy due to recessive mutations. Finally, as to balanced chromosomal rearrangements, the Committee assessed the risk to be between 1 and 15 cases of congenitally malformed children per million live births per 10^{-2} Gy of paternal irradiation (0-5 cases for maternal irradiation). These figures (see Table 2) are also thought to remain valid.

188. Although it did not explicitly say so until 1982, the Committee has always realized that simply presenting the number of serious genetic diseases is to

[7] This subject is reviewed extensively in Annex E, "Genetic hazards".

Table 7

Estimates of risk of severe genetic disease per million live births in a population exposed to a genetically significant dose equivalent of 1 Sv per generation of low-dose rate, low-dose irradiation, according to the doubling dose method
(based on the UNSCEAR 1986 Report and subsequent work)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per million live births	Effect of 1 Sv per generation: First generation	Second generation	Equilibrium
Autosomal dominant and X-linked	10000	1500	1300	10000
Autosomal recessive	2500	5	5	1500
Chromosomal				
Due to structural anomalies	400	240	96	400
Due to numerical anomalies	3400	Probably very small		
Congenital anomalies	60000	Not estimated a/		
Other multifactorial diseases	600000	Not estimated a/		
Early acting dominants / Heritable tumours	Unknown	Not estimated a/		
Totals of estimated risk		1700	1400	12000

a/ See paragraph 188.

ignore the full measure of the harm. In the absence of objective and quantifiable indicators of severity, it is hard to assess the full impact of radiation risks in terms of the individual, familial and social burdens imposed by these diseases. Therefore, starting with the UNSCEAR 1982 Report, the Committee began [redacted]

189. The Committee wishes to stress that there are still no direct data in man on the induction by radiation of hereditary diseases. Until such data become available there is no alternative but to continue to use data obtained in other mammalian species, suitably corrected to accord with what is known of human genetics, to estimate the risk of hereditary diseases in man.

190. All the numerical estimates of genetic risks discussed thus far have been obtained on the basis of genetically significant doses, i.e., on the assumption that the doses are received by individuals before or during the reproductive period. It is obvious that in the exposure of an entire population, the genetically significant doses are markedly less than the total doses received over a lifetime: damage sustained by the germ cells of individuals who are beyond the reproductive period or who are not procreating for any other reason poses no genetic risks. If is assumed that the mean age at reproduction is 30 years and that the average life expectancy at birth is 75 years, the dose received by age 30 is 40 per cent of the total dose.

191. To derive risk coefficients for genetic diseases in a population, one needs, accordingly, to multiply the genetic risk estimates discussed earlier by 0.40. The calculations shown below make use of the most recent risk estimates presented in Table 7 of Annex E "Genetic hazards", and give the risk coefficients per sievert:

(a) Risk coefficient on the basis of gonadal dose in the reproductive segment of the population (from Annex E, Table 7); for quantifiable damage only, over all generations 12,000/10⁶ or 1.2%

(b) Risk coefficient for the whole population, not only the reproductive segment, all generations (0.4 × 1.2%) 0.5%

(c) Risk coefficient for the first two generations, but otherwise as in (a) above 3,100/10⁶ or 0.3%

(d) Risk coefficient for the whole population, for the first two generations (0.4 × 0.3%) 0.1%

2. Radiation carcinogenesis in man[f]

192. The most recent data in the field of radiation-induced cancer in man have been examined with the following in mind: (a) [redacted] advances in under-standing the [redacted] of cancer [redacted] (b) [redacted] in Annex B of the UNSCEAR 1986 Report, "Dose-response relationships for radiation-induced cancer"; (c) extensive additional follow-up data on major epidemiological studies such as those of the survivors of Hiroshima and Nagasaki; and (d) a revised dosimetric system for the survivors of Hiroshima and Nagasaki that allows a better analysis of this important epidemiological series.

193. Several factors influence the probability that an individual exposed to radiation will develop cancer. Some of these, the host factors, pertain to the individual, such as his genetic background, age, sex and state of health; others pertain to the conditions of irradiation, such as the dose delivered, the time period over which the dose was received and the quality of the radiation; still others are factors that may interact with radiation to affect the susceptibility of the host, such as his living habits or his exposure to other toxic agents. Thus, there is no single, simple way to assess the effects, so several approaches have been taken.

194. One approach is to study the effects of different exposure or host conditions on biological models of carcinogenesis. This approach allows analysing one or another aspect of the risk, e.g., its variation with time or with the age of the exposed individuals. Another approach aims at analysing dose-response and risk-projection relationships. A third approach is the direct regression study of epidemiological data, especially through modern multiple regression techniques, which are particularly suited to the complexity of these phenomena.

195. The most informative epidemiological series are those which were carried out in the following groups: (a) people who were chronically exposed to high or intermediate doses of radiation when the dangers of such exposures were as yet unknown; (b) people who were chronically exposed to low doses for occupational, medical or environmental reasons; (c) people who received high doses to some parts of the body over short periods for therapeutic purposes; (d) people who were, and are, exposed to low doses of radiation for medical diagnostic purposes; (e) special cohorts who were irradiated externally as a consequence of the atomic bombings at Hiroshima and Nagasaki or internally as a consequence of fallout from the testing of nuclear weapons; and finally, (f) isolated individuals who received fairly high doses in accidents of various sorts.

196. Two methods have been employed in the epidemiological investigation of the groups listed above: (a) cohort studies, in which exposed individuals are analysed usually prospectively for their cancer experience compared with a suitably chosen non-exposed control group and (b) case control studies in which individuals with cancer are matched with normal individuals of a control population and exposures are determined retrospectively. The first method has distinct advantages but of course can be employed only in special circumstances.

197. Most of the retrospective studies discussed in the UNSCEAR 1977 Report have continued up to the present time, and new results have been reported. In several series, such as that on radiation-induced breast cancer, earlier findings were improved and dose-response patterns were made more precise by combining data from several investigations. In other series, such as that on pelvic irradiation for tumours of the uterine cervix, earlier findings were at least partially called into question. In yet other series, such as those on occupationally exposed groups, the earlier findings have, on closer examination and re-interpretation, been criticized for different types of investigating and reporting bias. Uncertainties in the dosimetry, the unsuitability of control groups and potential or actual difficulties in the ascertainment of tumours were some of the problems encountered.

198. All of the most important prospective studies that were in progress in 1977 are still in progress. Three more sets of mortality data, as well as additional incidence data, are now available from the survivors of Hiroshima and Nagasaki, and these have improved the dose-response estimates for some tumour types and have added other malignancies (colon, ovary, multiple myeloma) to the list of those already known to be radiation-induced. Some information has also been added to the studies of people exposed at the Hanford nuclear facility and to fallout in the Marshall Islands and of patients exposed for medical conditions such as ankylosing spondylitis, mastitis, pneumothorax or thymus-related irradiations. The absolute risks in these cohorts of people continue to increase (save, possibly, in the patients with ankylosing spondylitis and in those who were youngest at the time of the bombings in Hiroshima and Nagasaki). All these studies must obviously continue throughout the life-times of the exposed individuals in order to complete the data on dose- and time-response relationships for cancer induction. Moreover, for the relevant information to be generalized, it is also vital to know to what degree these cohorts are similar to other populations, how, and with what consequences, exposure to non-radiation risks may have changed; and how, for a general population, the risk of a given dose of radiation relates to the background cancer risk. One of the central problems in risk estimation continues to be the shape of the dose-response relationship, an issue extensively treated in the UNSCEAR 1986 Report. Although a number of models may be used to analyse the risk, each of them represents no more than an approximation to the true dose-response relation and has potential limitations or pitfalls.

199. The mortality experience of Hiroshima and Nagasaki survivors has been the single most important source of information on the radiation-related risk of cancer induction. A recent re-evaluation of tissue-absorbed doses in these survivors has made clear that their exposure to neutrons was substantially less than had been thought, and the relevant data, particularly those from Hiroshima, are now believed to be much less informative about the effects of neutrons than had once been presumed. The large body of experimental data and the very limited amount of epidemiological evidence on the relative biological effectiveness (RBE) of neutrons must therefore be carefully re-examined, with a view to arriving at some estimate of risk for this type of radiation.

200. A new international study of patients surviving treatment for carcinoma of the cervix has provided additional data on second cancers at selected sites.

201. Lifetime cancer experience is not yet available for any of the large epidemiological studies. Therefore, to project the overall cancer risk for an exposed population, it is necessary to use models that extrapolate over time data based on only a limited period of the lives of the individuals. Two such projection models have received particular attention: (a) the additive model which postulates that the annual excess risk arises after a period of latency and then remains constant and (b) the multiplicative model in which the time distribution of the excess risk follows the same pattern as the time distribution of natural cancers, i.e., the excess (after latency) is given by a constant factor applied to the age dependent incidence of natural cancers in the population. Data are now available that may provide a deeper insight into the applicability of the two models, and recent findings in Japan suggest that the relative risk projection model is the more likely, at least for some of the most common cancer types. Firmer conclusions should be possible soon.

202. Cancer is generally understood to develop in a number of stages. That is, for malignancies to be expressed a series of events must occur and the rate at which they occur is thought to be reflected in the way cancers appear in the population over the course of time. Analysis of the various epidemiological series in the light of this notion reveals a number of inconsistencies, so that it is not yet feasible to say which stages in carcinogenesis are affected by radiation or whether more than one stage is affected or whether the multistage model is able to explain the actual process. All of these possibilities may apply to some extent. It may even be that events postulated at the cellular or subcellular level cannot be easily related to the clinical data on radiation carcinogenesis.

203. A limited number of genes, known as oncogenes, have been implicated in the malignant transformation of normal cells. The precise ways in which oncogenes can be activated by radiation are not known, but so far data have not revealed any modifications that would suggest radiation plays a special role in inducing cancer or that would help to differentiate, at the genetic level, radiation-induced tumours from tumours induced by other carcinogens.

204. The Committee has carried out a detailed review of the information available on time-specific susceptibility to radiation-induced cancer and has considered separately the evidence pertaining to the exposure of children and adult subjects. Data on children show that the thyroid, the bone, the bone marrow and the breast are definitely responsive to the carcinogenic action of radiation. The bulk of the children successfully treated by radiation for cancer (i.e., those carrying localized primary tumours) who

[f] This subject is reviewed extensively in Annex F, "Radiation carcinogenesis in man".

have developed secondary tumours are those whose primary tumour had a large heritable component of cause. These children are obviously more prone to develop cancer than a normal child. In general, certain sites are susceptible, and the genetic evidence shows that this has to do with gene regions expressed in both the tissue involved in the original primary tumour (e.g., retinoblastoma) and in the tissue of the second tumour (e.g., bone sarcoma). Individuals with the hereditary form of retinoblastoma are also known to develop osteosarcomas away from the irradiated field or in the absence of irradiation. The spontaneous risk of second tumours in retinoblastoma patients is due to the somatic development of homozygosity in those children who inherit a single copy of the relevant mutation, but it is not yet known whether this is also the mechanism by which radiation induces second tumours. There are indications in the case of second tumours following retinoblastoma that a multiplicative projection model may apply, as it does to most adult tumours.

205. A number of general principles concerning the induction of tumours by radiation can be derived. Radiation is detectably carcinogenic if the dose is high enough, but no cancers unique to radiation are induced. Leukaemia (except chronic lymphatic leukaemia) is the most prominently induced cancer but tumours of the breast, thyroid, lung and bone marrow and at a number of other sites are also induced. The frequency of induction per Gy varies with the site. Some tumours such as chronic lymphatic leukaemia, squamous cell carcinoma of the cervix and Hodgkin's disease are not induced by radiation. Induced tumours are expressed some time after exposure, the latency being at least 2-5 years for leukaemia and about 10 years or more for other tumours. Age is the most significant host factor but other factors such as genetics play a role. These features are explained further in Annex F.

206. In general, the results from cancer patients are similar to those from other exposed groups in regard to the post-irradiation pattern of risk. However, in some instances, the risk in cancer patients appears to be different from that in the general population. This could be due to differences in susceptibility to cancer, but it could also be due to differences in exposure to environmental risk factors, e.g., smoking. Second cancers occur in both irradiated and non-irradiated patients, making the estimation of radiogenic risks problematic and suggesting that inferred results may not be generally applicable.

207. The dose-response relationships for various forms of malignancy were discussed extensively in Annex B of the UNSCEAR 1986 Report. The conclusion reached there was that each type of tumour may have a characteristic dose-response pattern and that it is still difficult to assess satisfactorily the pattern for the majority of the tumours. However, a general conclusion could be drawn that for low-LET radiation most dose-response relationships were upward concave, reaching a maximum that would be followed by a decline of the response with further increasing of the dose. This decreasing slope and decline of the curve at high doses seems due to killing of the radiation-initiated cells from which tumours eventually arise.

208. The Committee concluded in 1986 that for some tumours, i.e., carcinomas of the female breast and perhaps of the thyroid a linear relationship at low and intermediate doses of low-LET radiations gave a good fit; for others a linear fit could not be rejected statistically but other models, e.g., linear quadratic and quadratic approximated the data equally well. These observations are still assumed to be basically correct, however, evidence presented recently to the Committee suggests that fractionated doses at very low doses per fraction may be less effective in inducing breast cancer than deduced previously from the linear relationship and apparent lack of dose-fractionation effects. Recent epidemiological studies on patients administered 131-iodine-iodides for diagnostic purposes suggest that low-LET radiation at low dose rates is also significantly less effective than intermediate and high doses delivered at high dose rates. This means probably that the dose-response relationship for induction of cancer of the thyroid gland is also non-linear (upward concave) as was suspected in the UNSCEAR 1986 Report.

209. Many biological differences among human beings are known to modify their susceptibility to radiation-induced cancer, and the Committee examined these differences, known as host factors. Current information generally suggests sex has little or no effect on radiation carcinogenesis, in the sense that the sex ratio for individuals with radiation-induced malignancies (thyroid, breast, lung, leukaemia) is similar to that for non-irradiated individuals with the same malignancies. Data show further that susceptibility to radiogenic tumours decreases with increasing age, the latency periods being related not so much to age at exposure as to the tissue involved. The mean age and the age distribution of cases in adults exposed to single doses are in general similar to those in the population at large. Data on the effect of genetic constitution suggest that there may be a small, but not trivial, fraction of the population which is prone to cancer development and could thus be more susceptible to radiation or other carcinogenic agents. To improve the risk estimates, better means of identifying susceptible individuals should be developed.

210. The concluding section of the Committee's study contains an overall analytical summary of radiogenic cancer effects drawn from the most comprehensive sources available. From only a few epidemiological studies, primarily the survivors of the atomic bombings and patients exposed during treatment of ankylosing spondylitis or cervical cancer (leukaemia only), the carcinogenic risk of radiation can be estimated for many different sites. All three studies comprise large numbers of people exposed to x- or gamma-radiation for short times and followed for long times; however, each set of data has unique characteristics. The Committee considered the results on tissue-specific tumours from these series and compared them with risk estimates produced by various other studies. The Committee's evaluation of risk estimates is discussed in section II.C.2.

3. Early effects in man of high doses of radiation[a]

211. The Committee has reviewed what is known about the effects that occur in man within two to three months from receiving uniformly distributed whole-body doses above approximately 1 Gy of x- or gamma-radiation. The data were collated from three main sources: accidents; the atomic bombings; radiotherapy treatments. Important information on this subject has recently become available as a consequence of the nuclear accident at the Chernobyl power plant, in the course of which about 100 people were exposed to external and internal irradiation amounting to 1 Gy or more. The USSR delegation has prepared especially for UNSCEAR a detailed report entitled "Acute radiation effects in victims of the Chernobyl nuclear power plant accident", which is presented as an Appendix to Annex G.

212. Early prodromal responses during the first 48 hours after irradiation are mediated through the autonomic nervous system and appear as gastro-intestinal and neuromuscular signs. The incidence and latency periods for these effects are dose-dependent. For instance, the dose that induces vomiting in 50 per cent of individuals is approximately 2 Gy, and the mean latency period after this dose is about 3 hours.

213. Doses higher than 50 Gy generally lead to death within two days from cerebrovascular and neurological injuries (the so-called neurological syndrome). Uniform, whole-body doses between 10 and 50 Gy cause the gastrointestinal syndrome, which is generally fatal, with most deaths occurring during the second week after irradiation. In spite of the experience of those who died after the atomic bombings, there is insufficient information to estimate precisely the relationship between the dose and the probability of death due to this syndrome. The time to death of the gastrointestinal syndrome depends on the renewal time of the intestinal lining and is influenced by secondary factors such as infection, haemorrhage, loss of fluid, protein and electrolytes.

214. Uniform, whole-body doses of less than 10 Gy but greater than 1 Gy cause the bone-marrow syndrome, the incidence and severity of which depend on dose. The initial marrow damage after low doses reduces the number of white cells in the blood, the lymphocytes being the most sensitive indicators of injury. Doses of 1-2 Gy reduce the concentration of blood lymphocytes to about 50 per cent of normal within 48 hours of irradiation. Neutrophils show an initial increase over the first few days, then a dose-related fall. Ten days after 2-5 Gy, there is the beginning of a second abortive rise; however, if the marrow does not recover, a final decline is observed. The loss of neutrophils is associated with the onset of fever and is predictive of survival. The time course of platelet loss is broadly similar to that for granulocytes. Platelet levels in the blood below 30,000-50,000 per microlitre are associated with bleeding. People with the bone-marrow syndrome show an increased susceptibility to infection due to injury to the haematopoietic and the immune system.

215. In addition to the systemic effects described, irradiation may also cause damage to many other tissues and organs exposed separately. The resulting clinical symptoms vary as to time for appearance and severity. They may or may not be part of the syndromes described, depending upon the tissues irradiated, the dose level, the modalities of irradiation, and other physical and biological factors.

216. Irradiation of the skin causes lesions that are well known and very dependent on the dose and the area irradiated, in the sense that smaller doses have to take place over larger areas to elicit the same level of damage. Skin lesions include erythema, abnormal hair growth, epilation, desquamation and vascular and dermal injury. The dose in the basal layer of the epidermis determines the amount of cell killing and hence the degree of desquamation.

217. Injury to the mucous membranes in the mouth and throat evokes inflammation and swelling, with ulceration and necrosis after high doses. Mucosal injury is greatest in the cheeks, soft palate, and hypoglossal region. Acute effects on the eye are also well described and very dependent on the structures irradiated and the doses received.

218. When the thorax is irradiated, pneumonitis is the earliest sign of radiation injury in the lung. It appears at 1-3 months for doses greater than 8 Gy. The time of onset of pneumonitis is not significantly dose-dependent between 6 and 12 Gy. At Chernobyl there were some patients with early lung reactions. These changes were probably multifactorial in origin.

219. High acute doses of up to 4 Gy induce temporary sterility in some male individuals, but the dose inducing prolonged sterility in all males is at least 6 Gy. Although some of the differentiating forms of spermatogonia respond early and are very radiosensitive, the sperm count begins to decrease only after 6 weeks. In women, temporary sterility is induced by high doses up to 4 Gy and prolonged sterility by 4-10 Gy. Older women are more susceptible, probably because the number of ovarian follicles decreases with age.

220. It is of interest to know the dose of radiation that causes, on average, 50 per cent of individuals to die within 60 days ($LD_{50/60}$). The LD_{50} is a concept widely used in experimental work but there is doubt as to its applicability in human radiation biology, except for statistical purposes. The epidemiological series available for estimating this dose in man comprise radiotherapy patients, accident cases, and the Japanese exposed to atomic bombings in the Second World War. The $LD_{50/60}$ reflects marrow failure. The most recent studies of the $LD_{50/60}$ from experience in Japan (after revision of the doses) yield values of 3 and 3 Gy. The figure is thought to apply to the very special conditions prevailing after the bombing, for irradiated human beings who have no access, or only minimal access, to medical treatment.

[a] This subject is reviewed extensively in Annex G, "Early effects in man of high doses of radiation".

221. Some groups of radiotherapy patients have been useful for assessment of the $LD_{50/60}$. None of 20 children and adolescents given 3 Gy to the whole body to treat Ewing's sarcoma died of marrow failure. The $LD_{50/60}$ for groups of adults irradiated for disseminated cancers was 2.9 Gy in one series and 3.4 Gy in another. All these data indicate that for cancer patients, although they receive supportive treatment, the $LD_{50/60}$ is probably about 3 Gy, while [redacted]

222. In the accident at Chernobyl, 43 individuals received doses estimated to have been between 2 and 4 Gy, and one of them died. Of 21 people receiving doses between 4.2 Gy and 6.3 Gy, seven died. Of 20 patients receiving doses between 6 and 16 Gy, 19 died. Because of the complications suffered by many of the patients during the accident, such as thermal and skin injury, it is difficult to derive a value for $LD_{50/60}$ from these data.

223. From its review and discussion of the above data, the Committee concludes that it is impossible to assign a unique value to the LD_{50} in man; it may change substantially depending on age, the state of health of the individuals irradiated and on the prophylactic or therapeutic measures adopted before and after irradiation. For the planning of emergency responses, it is important to know which value of the LD_{50} would apply in which situation. The Committee [redacted]

224. Neutrons are more efficient in causing acute injury than x- or gamma-radiation, by a factor of 2-3, using single doses. There is little experience in man of the lethal effects of neutrons, except in a few isolated accidents. The neutron component of the doses to the survivors of the atomic bombings is now considered to be much smaller than had previously been estimated so the data collected from this group of people are therefore of little use in assessing the effects of neutrons.

225. As is well known in the field of radiobiology, dose protraction and fractionation cause less effect than the same total dose given singly. The early effects of high doses in man are no exception to this general rule. Thus, prodromal responses are somewhat alleviated by dose protraction or fractionation. Similarly, low-dose-rate or multi-fractionated irradiation markedly reduces injury to the intestine and the bone marrow in all species including man. Various quantitative formulae have been proposed to estimate the changes in dose or effect brought about by protracted irradiation; however, because the data base for many tissues is sparse, these formulae are only very rough guidelines for prediction. There is, moreover, one exception—the testis—to the general rule on protraction and fractionation; the progression of cells into sensitive phases makes this organ more sensitive to fractionated doses than to single doses.

226. In general, large amounts of internal emitters are required to produce early effects in man. Bone marrow depression is observed after single large intakes of iodine-131 and caesium-137. Gold radiocolloids have produced mild radiation sickness and haematological complications, as have phosphorus-32 and sulphur-35. Severe acute intestinal injury in man from internal emitters has not been reported, and lung injury has been rare. Treatments for internal contamination with radionuclides are based on local removal, reduced retention, enhanced excretion and diminished translocation.

227. A small fraction of the population may be particularly sensitive to early radiation injury by virtue of inherited genetic disorders, such as ataxia telangiectasia. Persons with this disease are more radiosensitive than normal. Many other genetic disorders predispose to increased chromosomal or cellular injury, but quantitative estimates of this increase are not available.

228. It is difficult to form a prognosis in irradiated patients solely from an estimate of the dose. There are many confounding factors, including intercurrent disease, dose protraction and radiation quality. The type and duration of prodromal symptoms, including erythema, may assist in the prognosis. Haematological signs, particularly the lymphocyte count, are good prognostic indicators. The lowest blood counts and their time of occurrence for the various blood cell types are also important, as is the duration of marrow aplasia after high doses. The appearance and persistence of immature cells in the blood is usually a favourable sign of marrow recovery. A valid prognosis must be founded on a wide range of different types of data and constantly updated.

229. The information provided by the USSR and contained in the Appendix to Annex G on the victims of the Chernobyl accident is exhaustive and valuable. While the nature of the lesions observed is not unexpected, the degree of precision achieved in the analysis of their time of onset and their magnitude and duration adds considerably to our understanding of the biological effects of high doses of radiation in man. Further analysis of these findings is definitely warranted, particularly in respect to the following points: the precise assessments of the doses received by the victims, the correlation of the various symptoms and signs with the causal agents (the pattern of exposure was complex and involved internal and external irradiation and additional thermal exposure in a few cases). These new studies will substantially enhance the present knowledge and will eventually allow the data collected at Chernobyl to be consolidated with other findings discussed in Annex G. The Committee is indebted to all those who contributed to the Appendix for their willingness to share this experience and wishes to commend them for the professional skill and the human compassion shown on such a tragic occasion.

4. Effects of pre-natal irradiation

230. In its latest study of the biological effects of pre-natal irradiation contained in the UNSCEAR 1986 Report, the Committee reviewed the most recent information on developmental events, particularly in the brain of mammalian embryos and foetuses; the irradiation of experimental animals before birth; and children exposed to radiation pre-natally by the atomic bombings at Hiroshima and Nagasaki. Its review centred as much as possible on human experience and included effects that had not previously been considered before in this light, such as the carcinogenic effects of irradiation *in utero*.

231. The 1986 data showed that mental retardation is the most likely type of developmental abnormality to appear in the human species. In essence, analysis as a function of time showed that the probability of radiation-related mental retardation is essentially zero with exposure before eight weeks from conception, is maximum with irradiation between eight and 15 weeks, and decreases between 16 and 25 weeks. After 25 weeks and for doses below 1 Gy, no case of severe mental retardation had been reported. On the assumption that the induction of the effect is linear with dose (as the data seemed to indicate), the probability of induction per unit absorbed dose was estimated at 0.4 per Gy at the time of the peak sensitivity and at 0.1 per Gy between 16 and 25 weeks from conception.

232. Using all the data available, the Committee attempted to derive quantitative risk estimates for the radiation effects for which there is positive evidence or, at least, reasonable presumption of induction. In addition to mental retardation, these effects include mortality and the induction of malformations, leukaemia and other malignancies. Under a number of qualifying assumptions, the Committee estimated that a dose to the conceptus of 0.01 Gy delivered over the whole pregnancy would add a probability of adverse health effects in the live born of less than 0.002. The normal risk of a non-irradiated live born carrying the same conditions is about 0.06. Information becoming available suggests that the risk estimates in the last two paragraphs may need substantial revision downward (particularly in the low-dose ranges). The Committee intends to review this in the near future.

C. DERIVATION OF RISK COEFFICIENTS

233. In the situations described in the Annexes, people are exposed to a range of types of radiation, and the resulting doses in their bodies are often non-uniform. In order to add the doses from groups of sources, e.g., natural sources, it is necessary to use a quantity that takes account of these different kinds of radiation and dose distributions in the body. The quantity used by the Committee is the effective dose equivalent. This quantity is obtained by weighting the absorbed dose in a tissue of the body, first by a factor to take account of the effectiveness of the type of radiation and then by a factor to take account of the different biological sensitivities of the tissues. The sum of these weighted absorbed doses is the effective dose equivalent.

234. The values of the two sets of weighting factors are those recommended by the International Commission on Radiological Protection. From time to time, the Committee has considered other systems of weighting, but has so far decided that the effective dose equivalent remains adequate for its purposes. The use of the effective dose equivalent is limited to assessments of long-term effects such as carcinogenesis. For assessing the early effects of high doses, the absorbed dose is an appropriate quantity.

235. When it uses the term "risk" (in a quantitative sense) the Committee means the probability of a harmful event, e.g., a radiation-induced death and often expresses this probability in per cent. The number of projected events in a population is expressed either as cases per thousand or cases per million. The term "risk coefficient" is used in a general way to indicate the risk per unit dose (risk per gray in the case of absorbed dose or risk per sievert in the case of effective dose equivalent). Since the relationship between dose and risk is not always proportional, it is sometimes necessary also to specify the dose or dose range for which the coefficient is valid.

236. In addition to estimating risk, the Committee has also estimated the projected number of years of life lost in an exposed population due to radiation-induced mortality. This quantity and also the projected number of cases or deaths in an exposed population are sometimes called measures of collective detriment.

1. Hereditary harm

237. Genetic risk coefficients may be defined to apply either to the gonad dose equivalent or the effective dose equivalent. It is also necessary to decide whether they should apply to genetically significant doses (i.e., doses to reproductive individuals) or average doses to the population at large. Opting for the latter might seem absurd from the scientific point of view, but sometimes only average doses or total collective doses are known; moreover, risk coefficients for cancer often apply to average doses.

238. In the UNSCEAR 1986 Report and in Annex E of this Report, "Genetic hazards", the Committee has reviewed the present body of knowledge of the hereditary effects of ionizing radiation. These reviews are summarized in section I.D.1. There are several customary ways of presenting the scientific information. One is to make the assessment for an equilibrium situation, wherein a stable population has been exposed over many generations, with each reproductive individual, male or female, receiving a unit gonad dose, and to estimate the fraction of the offspring who would then be expected to be affected by hereditary harm. Another way is to assess the affected number of offspring to a parent generation where the parent generation, males or females or both, have received a given collective dose.

239. In both cases, the information can be translated into a risk coefficient that expresses either the probability of a reproductive individual giving birth to a child affected by hereditary harm or the expected number of affected children, per unit individual or collective gonad dose to reproductive individuals. The risk coefficient may also be extended to include harm in all future generations.

240. Such risk coefficients can be applied directly to estimates of the genetically significant dose, such as those which have been made for various medical diagnostic x-ray procedures. However, they cannot be applied to effective dose equivalents unless there is uniform whole-body exposure. In other cases, the applicable genetic risk coefficient could range from zero (if the gonads are not exposed) to four times the risk coefficient that is applicable to the gonad dose (in the case that only the gonads are exposed), the organ weighting factor for the gonads being 1/4.

241. If the effective dose equivalent is assessed not for reproductive individuals but for average individuals in the population at large, then the relevant risk coefficient is only F/L of the genetic risk coefficient that would apply to reproductive individuals, F being the main reproductive age and L the life expectancy at birth. If F is about 30 years and L about 75 years, the genetic risk coefficient for the average individual becomes 40 per cent of the coefficient for reproductive individuals.

242. Table 8 summarizes the Committee's present estimates of genetic risk coefficients. Extensive information about the nature of the genetic risk is presented in the UNSCEAR 1986 Report.

243. A comparison with previous estimates (see Table 1) shows that present estimates are lower than those made in 1977. The 1977 estimates were used when the ICRP defined the effective dose equivalent. The risk coefficients refer only to the expected number of cases of quantifiable, severe, hereditary disease. What this means in terms of detriment is a question the Committee will continue to study.

2. Cancer

244. Cancer risk coefficients may be expressed either as (a) the site-specific individual probability of future radiation-induced cancer (death) per unit dose or (b) the collective detriment. The latter may be presented either as the expected number of cancer deaths (or cases) in the exposed population or as the number of person years lost because of cancer deaths per unit collective dose.

245. The new assessments in Annex F, "Radiation carcinogenesis in man", relate to the cancer risk at doses of 1 Gy at high dose rate of low-LET radiation. It has to be stressed, however, that statistically significant excess cancer mortality in Hiroshima and Nagasaki has been observed for the first time for some cancers and at several specific sites at doses between [redacted]. Not only have the risks from nine types of cancer been assessed with reasonable confidence, but also the total risk from all other types of cancer has been independently assessed. The risk estimates include a projection into the future of observations on the exposed populations at Hiroshima and Nagasaki. The new estimates have taken into account the revised dosimetry. All of this has had the combined effect of making the risk estimates at these doses and dose rates higher than before.

(a) Site-specific individual risk

246. Table 9 shows the results of the Hiroshima-Nagasaki study with regard to the individual probability of death from site-specific radiation-induced cancer. Two sets of numbers are given: one is derived from projections based on the additive (absolute) risk model, the other from projections based on the multiplicative (relative) risk model.

247. The total cancer mortality risk coefficient for the average individual (averaged also over both sexes) is 4.5 per cent per gray on the additive risk model and 7.1 per cent per gray on the multiplicative risk model. These numbers may be compared with the 1977 estimate for high doses, which was about 2.5 per cent per sievert on the basis of the additive model (see Table 3). Further summary values of risk coefficients for populations of other ages and other circumstances are given in Table 10. These lifetime risks range from 4 per cent to 11 per cent per gray.

Table 8

Revised genetic risk coefficients a/
(per cent per Sv)

	For gonad dose equivalent		For effective dose equivalent	
	Reproductive population	Total population	Reproductive population	Total population
First two generations	0.3	0.1	0-1.2	0-0.5
All generations	1.2	0.5	0-5	0-2

a/ Risks from diseases of complex aetiology were not estimated. See also paragraph 186.

Table 9

Per cent lifetime excess cancer deaths probability following exposure to 1 Gy organ absorbed dose at high dose rate of low-LET radiation
(per cent)
(based on the population of Japan using an average age risk coefficient)

	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.97	0.93
All cancers except leukaemia	6.1	3.6
Bladder	0.39	0.23
Breast a/	0.6	0.43
Colon	0.79	0.29
Lung	1.5	0.59
Multiple myeloma	0.22	0.08
Ovary a/	0.31	0.26
Oesophagus	0.34	0.16
Stomach	1.3	0.86
Remainder	1.1	1.0
Total	7.1	4.5

a/ Value has to be divided by 2 to calculate the total and other organ risks.

$\frac{0.07}{100 \text{ Rad}} = 7 \times 10^{-4}$

Table 10

Estimates of projected lifetime risk, for 1000 persons (500 males and 500 females) exposed to 1 Gy of high dose rate low-LET radiation

(based upon the population of Japan)

	Risk projection model	Excess fatal cases	Years of life lost
Total population	Additive	40-50	950-1200
	Multiplicative	70-110	950-1400
Working population (aged 25-64 years)	Additive	40	880
	Multiplicative	80	970
Adult population (over 25 years)	Additive	50	840
	Multiplicative	60	640

248. The problems in deriving risk coefficients that are also applicable at low doses are the same as before. Such risk coefficients can only be inferred from the observed values at moderate to high doses. In 1977, when the total cancer risk coefficient at high doses was estimated to be about 2.5 per cent per sievert, the Committee pointed out some of the uncertainties; these included the fact that this estimate was an underestimate because no projection had been made into the future, but it was also an overestimate in the sense that the risk per unit dose at low doses was believed to be lower than the estimates for high doses.

249. In this Report, the problems in deriving risk coefficients at low doses and for low dose rates remain. The Committee agreed that there was a need for a reduction factor to modify the risks shown in Table 9 and Table 10 for low doses and low dose rates. The Committee considered that such a factor certainly varies very widely with individual tumour type and with dose rate range. However, an appropriate range to be applied to total risk for low dose and low dose rate should lie between 2 and 10. The Committee intends to study this matter in detail in the near future.

250. The Committee has not presented risk estimates for high-LET radiation in general in this Report (except for the exposure to radon of uranium miners). For low doses of external high-LET radiation it would be necessary to multiply the risks for low-LET radiation by an appropriate quality factor. No dose or dose rate reduction factor is considered necessary for high-LET external radiation at low doses.

(b) Collective detriment

251. The product of risk coefficients appropriate for individual risk and the relevant collective dose will give the expected number of cancer deaths in the exposed population, provided that the collective dose is at least of the order of 100 man Sv. If the collective dose is only a few man Sv, the most likely outcome is zero deaths.

252. The Committee has also assessed the person years lost per unit collective dose because of radiation-induced cancer mortality. The results at high doses and high dose rates of low-LET radiation are summarized in Table 10. The total loss amounts to about 1 person year per man Gy with both projection models.

D. COMPARISON OF EXPOSURES

1. Previous UNSCEAR comparisons

253. The way in which to present radiation exposures from various sources has always been a problem for the Committee. In its 1958 Report, the Committee assessed the per caput mean marrow dose and the genetically significant dose to the world population from various sources and practices. At that time, the Committee even calculated the expected number of cases of leukaemia and hereditary harm from natural background radiation and nuclear explosions.

254. In the UNSCEAR 1962 Report, the Committee assessed the per caput doses from natural irradiation of the gonads, the bone surface layers and red bone marrow. It also calculated the dose commitments to the world population for the three organs. The genetically significant dose was assessed for medical and occupational exposures. However, in that Report the Committee felt that it had less confidence in the risk coefficients used in the UNSCEAR 1958 Report and that it was not able to assess any detriments. It stated, instead, that the estimated doses and dose commitments could be used for comparative risk assessments and gave this comparative risk in relation to natural background radiation, which was assigned the value of unity. This comparison was made for medical exposures and nuclear explosions with reference to leukaemia, bone tumours and hereditary effects. On the same basis, the Committee said, the detriment of various sources could be expressed in terms of exposure to natural background radiation that would give the same per caput dose or dose commitment.

255. In the UNSCEAR 1964, ... and 1972 Reports, the Committee continued to express the risk from nuclear explosions in terms of the equivalent period of exposure to natural background radiation. Until 1972 the Committee had calculated per caput doses or dose commitments for the whole world population. For a population of a given number, this implies an assessment of the collective dose from each source. In the UNSCEAR 1977 Report, the Committee for the first time explicitly presented collective dose assessments for various sources and practices. At the same time, however, it also drew comparisons on the basis of equivalent periods of natural background exposure. In the UNSCEAR 1982 Report, the Committee included more information on the ways in which individual exposures vary, and it assessed collective dose commitments. In the summary and conclusions, the collective dose equivalents were translated into equivalent periods of natural background radiation.

256. From this short review it can be seen that comparison with the natural background dose rate has always played an important role in the Committee's presentation of its assessments. When, in 1958, the Committee estimated the number of affected persons, it drew a comparison with the natural occurrence of cancer and hereditary disease. Since then, per caput and collective doses have been compared with the corresponding doses caused by natural radiation.

2. Purpose of comparisons

257. Comparisons usually have a purpose and may be presented in different ways depending on that purpose. Comparisons with doses or detriments caused by natural sources of radiation may help to clarify the relative radiological importance of man-made radiation sources, but they say little about justifiability or acceptability of these other sources. Information on where doses are low or high in relation to the natural background may help in determining whether there is a potential for meaningful epidemiological studies. Comparing the radiation doses or risks of alternative procedures for achieving one and the same objective, e.g., medical diagnostic information, may disclose what might be preferable from the radiation protection point of view, but it will not reveal other risks or disadvantages. Since the Committee has no use of its own for comparisons, it wishes to present its data in such form that they can be used for a number of different purposes.

3. Comparison of collective doses

258. If risk coefficients are known and if proportionality between dose and response can be assumed, radiation detriments, such as the expected number of cancer deaths, can be calculated from information on collective dose commitments. For relative comparisons, however, it suffices to compare collective doses or per caput doses (which amounts to the same thing) from the various sources, thereby eliminating the uncertainty in the risk coefficients. In such comparisons, the annual collective dose from natural sources of radiation may be taken as the reference; the contribution from other sources may be expressed in terms of the equivalent periods of natural background radiation, as has been the Committee's practice since 1962.

259. When collective doses from different sources are compared, it is important that the comparison be on a relevant basis. This is simple for sources and practices aimed at achieving one and the same objective, such as energy production or medical diagnostic information. In other cases, one must be careful to find a common basis for comparison. For example, it is of doubtful relevance to compare collective doses to arbitrarily selected populations and time periods. However, although comparisons of collective doses from entirely different practices will often not be very meaningful, they may sometimes help in setting priorities for dealing with concerns of radiological consequences.

4. Comparison of individual doses

260. The radiation doses an individual receives from various man-made sources are normally compared with the dose he receives from natural sources of radiation. An extra dose that is small in relation to the background dose will not significantly affect an individual, i.e., it will not change his total exposure situation noticeably. While the individual might still wish to avoid such a small extra dose, he should know that it does not in itself present any substantial risk. This does not mean that the dose is acceptable just because it is small; rather, acceptability would depend on the total harm the source is likely to cause and on society's appraisal of that harm.

261. Comparing per caput doses in the case of an uneven dose distribution within a population may be misleading, since no individual may actually receive the per caput dose but instead will receive either higher or lower doses. In that case, comparing typical doses as well as extreme doses may be more appropriate.

5. Summary of dose comparisons

262. Table 11 summarizes the various estimates of radiation doses. As in previous Reports, the equivalent period of exposure to natural background radiation is given along with the collective dose commitments. In comparing these estimates with those in previous Reports, it should be remembered that the estimate of the annual dose from natural background radiation has increased, from less than 100 mrad (corresponding to about 1 mSv) in the 1977 Report to 2.4 mSv in the present Report. This increase came about for two reasons: (a) instead of giving a number of organ doses, the effective dose equivalent is now given and (b) the large contribution from radon daughter products has been recognized.

263. Table 11 is of necessity a considerable condensation of the available information. It is worth noting that about half of the natural background radiation is contributed by lung irradiation by radon daughters. Occupational exposures are experienced by those who work in the medical field as well as those who work in the nuclear power industry and in industrial radiography. Exposures from nuclear power production are due to radionuclides released from uranium mining and waste disposal activities, as well as from the operation of reactors to produce electric energy. About one third of the current exposures from nuclear power is attributable to radon emissions from mine tailings and another third to carbon-14 discharges from reactor operation, primarily heavy water reactors.

264. Of the collective effective dose equivalent commitment (other than from ^{14}C) from all atmospheric test explosions, 1.5 million man Sv have been contributed by short-lived radionuclides and 3.5 million man Sv

Table 11

Summary of estimates of effective dose equivalent

Source or practice	Present annual individual doses (mSv): Per caput (world population)	Present annual individual doses (mSv): Typical (exposed individuals)	Collective dose commitments: Million man Sv	Collective dose commitments: Equivalent years of background
ANNUAL			Per year of practice	
Natural background	2.4	1 - 5	11	1
Medical exposures (diagnostic)	0.4-1	0.1 -10	2-5	0.2-0.5
Occupational exposure	0.002	0.5 - 5	0.01	0.001
Nuclear power production	0.0002	0.001-0.1	0.001 (0.03) a/	0.0001 (0.004) a/
SINGLE			Per total practice	
All test explosions together	0.01	0.01	5 (26) a/	0.5 (2.4) a/
Nuclear accidents			0.6	

a/ The additional long-term collective dose commitments from radon and carbon-14 for nuclear power production and carbon-14 for test explosions are given in parentheses.

represent contributions to present individual life-time doses primarily from strontium-90 and caesium-137. Because the Chernobyl accident led to doses mainly in Europe, the collective effective dose equivalent commitment rather than the global per caput dose is presented.

6. Direct comparison of detriments

265. In this Report, the Committee has reviewed the existing knowledge on radiation risks and has ventured to indicate the magnitude of the risk factors for low doses as well as for high doses. The Committee has also assessed the collective doses from various sources and practices. It is tempting to combine the estimates and calculate the expected number of cases of cancer and hereditary disease.

266. Many estimates of this type, with different degrees of reliability, depending on the risk coefficients assumed, and with widely different purposes on the part of those who made them, have been reported. The results have been very scattered, depending on the general assumptions. The Committee hesitates, for a number of reasons, to add its own detriment assessments to those already provided for the various sources of radiation.

267. First, the Committee needs to bear in mind the terms of reference under which it operates: its purpose is to evaluate doses, not to make value judgements or engage in setting standards. As is made clear by the discussion in section II.D.4, even those assessments of risk that purport to be scientific involve assumptions and decisions that are not, strictly speaking, scientific. Indeed, the physical quantities used by the Committee reflect such assumptions. For example, the effective dose equivalent, by definition, includes weighting factors that depend on subjective judgements as to what constitutes radiation-induced harm. For each further step in processing the basic information, non-scientific judgements are likely to be needed or implied.

268. Next, the way in which the basic scientific facts are presented influences the impression they give. For example, thousands of cancer deaths from a single accident would undoubtedly be a high number of deaths. However, since such deaths could be expected to occur over a long period of time, the annual incidence will be low. This means a very small increase of the normal incidence of cancer, an increase which is not expected to be noticeable in health statistics. This shows that it is possible, by selecting the form of presentation, to convey different impressions.

269. Lastly, there is the great uncertainty of such estimates. It was stressed in section II.C that the risk coefficients for cancer at low doses can only be inferred from observations at high doses and that the risk coefficients for hereditary effects are not even deduced from observations in man. Even though the Committee believes that its estimates are the best that can be given at the current state of knowledge, it must qualify them by drawing attention to the underlying assumptions and uncertainties. Unfortunately, any estimate of a finite number of cancer deaths is soon taken out of context and the qualifications forgotten.

270. For these reasons, the Committee prefers to follow its previous practice of comparing collective dose commitments from the main radiation sources rather than estimated detriments.

Appendix I

MEMBERS OF NATIONAL DELEGATIONS ATTENDING THE THIRTY-FIRST TO THIRTY-SEVENTH SESSIONS

ARGENTINA

D. Beninson (Representative), D. Cancio, A.J. Gonzalez, E. Palacios

AUSTRALIA

K.H. Lokan (Representative)

BELGIUM

M. Errera (Representative), J. Maisin (Representative), J.B. C. A'au, P. Lohman, F.H. Sobels, A.D. Tates

BRAZIL

E. Penna Franca (Representative), L.R. Caldas (Representative)

CANADA

E.G. Letourneau (Representative), A.M. Marko (Representative), W.R. Bush, G.C. Butler, B.C. Lentle, D.K. Myers

CZECHOSLOVAKIA

M. Klimek (Representative)

CHINA

Wei Lüxin (Representative), Li Deping, Wu Dechang

EGYPT

S. El-Din Hashish (Representative), H. Roushdy (Representative), M. El-Kharadly

FRANCE

H. Jammet (Representative), A. Bouville, R. Coulon, M. Bertin, B. Dutrillaux, J. Lafuma, G. Lemaire, R. Masse, P. Pellerin, M.R. Tubiana, G. Uzan

GERMANY, FEDERAL REPUBLIC OF

A. Kaul (Representative), J. Ehling, W. Jacobi, H. Kriegel, F.E. Stieve, C. Streffer

INDIA

N.K. Notani (Representative), K. Sundaram (Representative)

INDONESIA

S. Wiryosimin (Representative), A. Baiquni (Representative), O. Iskandar (Representative), M. Ridwan (Representative), C.J. Sugiarto

JAPAN

T. Kumatori (Representative), H. Matsudaira (Representative), T. Terasima (Representative), A. Kasai, A. Yamato

MEXICO

E. Araico (Representative), J.R. Ortiz Magaña (Representative)

PERU

L.V. Pinillos Ashton (Representative), M. Zaharia (Representative)

POLAND

Z. Jaworowski (Representative), J. Liniecki (Representative), Z. Szot

SUDAN

A. Hidayatalla (Representative), A.A. Yousif

SWEDEN

B. Lindell (Representative), G. Bengtsson, K. Edvarson, L.-E. Holm, K.G. Lüning, S. Mattsson, J.O. Snihs, J. Valentin, G. Walinder

UNION OF SOVIET SOCIALIST REPUBLICS

L.A. Ilyin (Representative), A. Guskova (Representative), K.M. Bochkodarov, V. Denim, E. Golubkin, D.F. Khokhlova, A.A. Moiseev, Yu.I. Moskalev, V. Pavlinov, O. Pavlovsky, O. Piatak, V.V. Redkin, V.A. Shevchenko

UNITED KINGDOM OF GREAT BRITAIN AND NORTHERN IRELAND

J. Dunster (Representative), R.H. Clarke, S.C. Darby, J. Denekamp, J.H. Edwards, K.E. Halnan, P.S. Harper, A. Searle

UNITED STATES OF AMERICA

F.A. Mettler (Representative), R.D. Moseley (Representative), R.E. Anderson, L.R. Anspaugh, R. Baker, C. Edington, J.H. Harley, R.C. Ricks, H.H. Rossi, W.L. Russell, P.B. Selby, W.K. Sinclair, J.W. Thiessen, E.W. Webster, H.O. Wyckoff

Appendix II

SCIENTIFIC STAFF AND CONSULTANTS WHO HAVE CO-OPERATED WITH THE COMMITTEE IN THE PREPARATION OF THIS REPORT

L. R. Anspaugh
B. G. Bennett
A. Bouville
R. H. Clarke
F. Fagnani
L. Frittelli
A. Hagen
J. Hendry
B. Lindell
F. A. Mettler
M. Morrey
O. Pavlovsky
W. J. Schull
G. Silini
F. D. Sowby
K. Sankaranarayanan
G. A. M. Webb
K. Weiss

Appendix III

REPORTS RECEIVED BY THE COMMITTEE

1. Listed below are reports received by the Committee from Governments between 19 April 1986 and 17 June 1988.

2. Reports received by the Committee before 19 April 1986 were listed in earlier reports of the Committee to the General Assembly.

Document	*Country*	*Title*
A/AC.82/G/L.		
1732	United Kingdom of Great Britain and Northern Ireland	Environmental radioactivity surveillance programme: results for the UK for 1984
1733	Japan	Radioactivity Survey Data in Japan Number 72, March 1985
1734	Japan	Radioactivity Survey Data in Japan Number 73, June 1985
1735	United States of America	Environmental Measurements Laboratory: A compendium of the EML's research projects related to the Chernobyl nuclear accident
1736	United States of America	Environmental Measurements Laboratory: The high altitude sampling program: radioactivity in the stratosphere
1737	Japan	Radioactivity Survey Data in Japan Number 74, Sept. 1985
1738	Japan	Radioactivity Survey Data in Japan Number 75, Dec. 1985
1739	Union of Soviet Socialist Republics	Assessment of population doses from x-ray examination in the USSR (1970-1980)
1740	Union of Soviet Socialist Republics	Genetic effects of radionuclide decay
1741	Union of Soviet Socialist Republics	Acute radiation effects in man
1742	Union of Soviet Socialist Republics	Production and release of carbon-14 in nuclear power stations with RBMK reactors
1743	Union of Soviet Socialist Republics	Body burden of fallout caesium-137 in the inhabitants of Moscow 1960-1983
1744	Union of Soviet Socialist Republics	Radiation doses to the far north inhabitants
1745	Union of Soviet Socialist Republics	Occupational exposure of radiographic workers
1746	Japan	Radioactivity Survey Data in Japan Number 76, March 1986
1747	Japan	Radioactivity Survey Data in Japan Number 77, June 1986
1748	Japan	Radioactivity Survey Data in Japan Number 78, October 1987
1749	Japan	Radioactivity Survey Data in Japan Number 79, October 1987
1750	Union of Soviet Socialist Republics	Proposals for setting possible intake limits for transuranium radionuclides absorbed from the gastro-intestinal tract
1751	Union of Soviet Socialist Republics	The evaluation of non-stochastic effects in man for low doses of internal irradiation

Document	Country	Title
1752	Union of Soviet Socialist Republics	Tritium production in LWGR power plants and its release into the environment
1753	Union of Soviet Socialist Republics	Medical treatment in the case of uranium intoxication
1754	Union of Soviet Socialist Republics	Dynamics of effective dose equivalent from intake of strontium-90 and caesium-137
1755	Union of Soviet Socialist Republics	Specific activities of natural radionuclides in building materials used in the Soviet Union

Scientific Annexes