Health Risks Due to the Use of Nuclear Energy for Electric Power Generation

by M. Tubiana

Many attempts have been made in recent years to compare the health risks from various methods of power production. The purpose of this article is:

1. To review the data which form the basis for an evaluation of the biological effects of ionizing radiations;

2. To evaluate the doses received by members of the public and workers as a consequence of nuclear power production; and

3. To examine the possible biological consequences of these exposures — both external and internal — for members of the public and workers.

Data on which Evaluation of the Effects is based

It is often said that the effects of ionizing radiation on man are mysterious and unpredictable. In fact, of all physical and chemical agents present in our environment, radiation is undoubtedly the best known. Roentgen discovered X-rays in 1895, and Becquerel natural radioactivity in 1896, and it was realized almost immediately that radiation had an effect on man. The first cancer caused by radiation was observed in 1902. The first experiment demonstrating the possibility of causing cancer in animals was carried out in 1910. The mutagenic effect of X-rays has been known since 1925.

Thus, from the beginning of the century we have been aware of the biological effects of ionizing radiation, ranging from the effects of doses of several thousands of rem (6000–7000 rem) used for the treatment of cancer to those induced by the accumulation of low doses. In view of the potential dangers of ionizing radiation, research on the subject went on side by side with the rapid development in the use of ionizing radiation in medicine (radiodiagnostics and radiotherapy). The first victims of ionizing radiation were the very physicists and physicians who were studying the radiation and its effects.

The feelings of guilt and concern which Hiroshima and Nagasaki engendered among the whole scientific community explain why since 1945 the effects of ionizing radiation on

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man have been studied in even greater depth. Since then the sums spent on studying the biological effects of ionizing radiation have run into thousands of millions of dollars.

It is paradoxical that the extent of these studies should have led to misgivings, but the understandable fear is that their scope reflected the seriousness of the hazards. Moreover, as the other methods of power generation (coal and oil) had been developed at a time when less attention was paid to health risks and when some of these, such as mutagenic or carcinogenic risks, were not known at all, there is a tendency to minimize the danger. It is therefore understandable that as radiation risks were the first to be stressed and quantified — by the very promoters of nuclear energy — attention should be focused on them; so that ionizing radiation was until recently the easiest of all toxic agents to detect, thanks to the high precision and extreme sensitivity of the detectors.

Owing to the early interest shown by radiobiologists in radiation protection and the efforts made to quantify the risks and develop dosimetric methods, health experts are unanimous in believing that radiation protection should serve as a model for all work aimed at combating potentially harmful agents. The lead of radiotoxicologists over other health experts is demonstrated by the fact that not until 50 years after the establishment of the International Commission on Radiological Protection (ICRP) was a similar body setup for carcinogenic and mutagenic chemicals.

Apart from experimental research, our present knowledge of the late effects of ionizing radiation on man is derived from the study of populations exposed to known doses of radiation — populations in which the consequences of exposure have been studied scientifically. These groups include several tens of thousands of patients treated with X-rays and radioisotopes for non-malignant diseases; several groups of workers (radium dial painters, radiologists, uranium miners, etc.), and the 285 000 survivors of Hiroshima and Nagasaki who have been studied since 1945 by a team of 500 specialists established jointly by Japan and the United States.

Many conferences and symposia have discussed these studies and several expert committees, national and international, have analysed the whole gamut of information obtained in this field. In particular, the 1977 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), a document of more than 700 pages, and the reports of the United States National Academy of Sciences must be mentioned. Moreover, in 1928 — a long time before the advent of nuclear power — groups of specialists, radiologists, and physicists met to discuss the risks of ionizing radiation together with the precautions to be taken to limit those risks and established the International Commission on Radiological Protection, the first recommendations of which were published the same year. Since then ICRP has regularly published its recommendations, made on the basis of the most recent scientific data. These recommendations have always been accepted by every country, and the role of the ICRP is universally recognized. In 1977 it published its 26th report. A further point to be stressed is that all these expert groups arrived at essentially the same conclusions and that, contrary to what is sometimes believed by the public, there is little disagreement among experts on the nature and extent of the biological effects caused by radiation.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 1. Main Sources of Irradiation in France

Sources: UNSCEAR 1977 and US National Academy of Sciences Report (Washington 1980)

NATURAL IRRADIATION	Average dose (mrem/yr) (Paris area)	Variations in France
Cosmic rays	30	100 at 2500 m altitude
Ground radioactivity	46	200 in granitic areas
Natural radioisotopes incorporated in the body (^{40}K, Ra, Th, etc.)	24	200 in regions with high radioactivity in water
	100	250 to 300 in Brittany, Massif Central, etc.

MAN-MADE IRRADIATION	Average value reported for total population (in mrem/yr)	
Medical X-rays	100[1]	Considerable individual variations depending on age and frequency of X-ray examinations
Dose due, in 1977, to fall-out from nuclear tests in the atmosphere (mainly carried out in 1956–1962)	5	
Air travel	0.1	Considerable individual variations: as much as 250 to 500 for air crews
Nuclear power production in 1976 (external irradiation sources and internal contamination)	0.15	Small variations depending on place of habitation, reaching 4 mrem for persons living in the vicinity of a plant

[1] This value was calculated in the USA. In France, the figure is very probably similar according to the data of the Central Service for Protection against Ionizing Radiations and the Ministry of Health. The average value for the whole world population is 20 mrem/yr.

Table 1 (cont.)

MAN-MADE IRRADIATION	Average value reported for total population (in mrem/yr)	Variations in France
Dose predicted for the year 2000	2	
Luminous dial watches	0.50	Up to 10 for persons who regularly wear certain types of luminous watch
Miscellaneous (phosphate fertilizer, television, coal burning, etc.)	1	
TOTAL irradiation other than medical or military	4	

Doses to the Public and Workers

By virtue of being present on the earth's surface, everyone is exposed to cosmic rays and to radiation emitted by natural radioactive bodies (radium and thorium): the average annual dose is of the order of 100 mrem at sea level. However, this dose varies considerably from one point to another on the earth. In France, for instance, it is of the order of 100 mrem a year in the Paris region but attains values of 200—300 mrem in high-altitude regions (Alps) or regions withgranitic soil. In several regions of the globe, annual doses can reach 500 or 600 mrem.

Apart from this natural exposure, there are other sources of irradiation (see Table 1). The most important source of human origin is the medical use of ionizing radiation, mainly in radiodiagnostics, from which every inhabitant of the earth receives, on average, an annual dose of the order of 20 mrem. However, in some countries like the United States and France the average dose is as much as 50 or even 100 mrem a year.

What does nuclear power contribute in comparison? In 1976, according to the UNSCEAR report, it accounted for 0.15 mrem per annum, which is lower than natural irradiation by a factor of about 600. In other words, the annual dose from nuclear power was, on average, about half a day's dose due to natural radioactivity. In France, the dose attributable to nuclear power in 1976 was the equivalent of the excess dose which a Parisian would receive during a half-day spent in Brittany or in the Massif Central.

For the year 2000 UNSCEAR quotes values between 2 and 4 mrem per annum if 60 per cent of the electricity generated comes from nuclear power. This includes doses due to uranium mining and processing, reactor operation, nuclear waste and pollution of

air, water and foods, with allowance for possible concentrations along the food chain and the resulting fixation of radioactive substances in the human body. Thus, for a Parisian, this would be tantamount to spending a week or two in the Massif Central or Brittany.

Contrary to what has sometimes been said, no significant difference in the frequency of cancers or genetic malformations has been detected between regions exposed to different doses of natural radiation.

The doses which workers at nuclear power plants receive are known accurately in all countries. For example, Table II refers to data taken from the latest report of the United States National Academy of Sciences. The order of magnitude of the doses is close to that of other countries, especially France As will be seen, these doses are, on average, appreciably lower than the ICRP limit (5 rem/yr). It should further be noted that exposure of workers and contamination of members of the Public due to fast breeder reactors will be lower, for a given number of kilowatt-hours produced, than from conventional thermal-neutron reactors.

Biological Effects of Low Doses

The first type of biological effect corresponds to the immediate or delayed effects on irradiated tissue, e g. skin lesions, reduced fertility and sterility, cataracts and impeded growth in subjects irradiated during childhood. When the doses are quite high (above a threshold of a few hundred or thousand rad), these effects are observed in all irradiated subjects and the seriousness increases with dose. Conversely, if the dose is low, no effect is detectable, there is thus a threshold dose below which no effect is observed. This varies with the effect and the volume of irradiated tissue; it is always higher than a few tens of rem per annum.

In the case of two other types of biological effect, namely induction of cancer and genetic mutations, the dose-effect relationship is quite different. What varies as a function of dose is not the biological effect itself, which remains identical, regardless of dose, but the probability of this effect. In other words, the percentage of irradiated subjects in whom the effect is observed increases with dose but the effect does not vary.

For example, a cancer is always a cancer, whether it occurs spontaneously or is caused by radiation, and there is no way of distinguishing one from the other. The only way in which induction of cancers in irradiated subjects can be shown is to demonstrate that they have a statistically higher frequency of cancers than a group of subjects of the same age and sex who were not irradiated.

Genetic effects have the same probabilistic or, as the experts would say, stochastic character, there being nothing to distinguish radiation-induced mutations from natural ones.

In these two cases, therefore, the concept of threshold is replaced by that of risk varying with dose, very low for small doses and rising with dose.

While the first ICRP recommendations were based on the non-stochastic somatic effects of radiation and on the existence of a threshold, from the early fifties onward greater

attention was devoted to the carcinogenic potential of low doses It had become clear by then that random effects without a threshold, i.e. carcinogenesis and mutagenesis, were the risks which had to be considered as a priority following exposure to low doses of radiation. Radiation protection experts therefore placed emphasis on the evaluation of these risks.

This situation is not unique, for in numerous areas of industrial health and medicine the same trend occured. Protection developed in the same way in the case of asbestos, for example. Initially, asbestosis was regarded as the main hazard and the aim of industrial medicine was to bring the exposure to asbestos dust to a level lower than that which caused the disease. Later, when working conditions were better and workers no longer suffered from asbestosis, they lived longer, and it was then found that they had a slightly higher risk of lung and pleura cancers than the population in general. It was therefore necessary to introduce protection measures taking into account the relationship between the asbestos concentration in the air and the probability of the occurrence of cancer.

This evolution of radiation protection, from the original criterion of lesions which appear in all irradiated subjects to a new criterion based on the probability of an extremely serious risk that will ultimately affect only a very small proportion of irradiated subjects, is thus a tendency frequently found in industrial medicine and is a consequence of the improvement of general working conditions.

EVALUATION OF THE RISK OF CANCER

In quantifying the carcinogenic effects of radiation, we are helped by several studies on populations of irradiated subjects, notably the prolonged and meticulous studies carried out on the survivors of the atomic bomb. Of the 285 000 registered survivors at Hiroshima and Nagasaki, 80 000 died a natural death between 1950 and 1978. It is estimated that approximately 400 to 500 of these deaths were due to radiation-induced cancer. Among the 1200 survivors who had received the highest doses (average dose of 330 rad, i.e. very close to the lethal dose when delivered to the whole body, which is between 350 and 450 rad approximately), the increase in the frequency of leukaemia between 1950 and 1974 was 1 per cent. Thus, there was undoubtedly a carcinogenic effect but in absolute terms the number of survivors affected was relatively small and did not appreciably change the overall survival rate of the population exposed to the atomic bomb.

We should note in this connection that the studies on survivors did not reveal any particular disease due to irradiation, apart from opacity of the eye lens and retarded development of height and weight in children exposed when very young. In particular, no increase in morbidity, acceleration in the process of aging or shortening of lifespan, apart from that related directly to the higher frequency of cancers, was observed.

Nothing justifies us to say, as some do at times, that estimates based on the survivors are erroneous because of high mortality during the first few years which left only the more resistant subjects to survive longer.

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The dose received by each survivor was determined by the distance between his position at the time of the explosion and the hypocentre. However, the radiation received by the inhabitants of the two cities differed. The Hiroshima bomb was made of uranium-235 and about a third of the irradiation was due to neutrons and the rest to gamma-rays. Since the relative biological effectiveness (RBE) of neutrons is about 10, 80 per cent of the biological effect was due to neutrons. At Nagasaki, on the other hand, the bomb, made of plutonium-239, was of a different design and released almost entirely gamma-rays. This difference justifies separate analyses of the Hiroshima and Nagasaki data, comparison of which provides direct information on the relative carcinogenic effects of neutrons and photons on man.

In these two cities, the carcinogenic effect of radiation first manifested itself in an increase in the number of leukaemia cases at the end of the forties. After passing through a peak during the period from 1952 to 1970, the rate of leukaemia decreased consistantly even while the frequency of radiation-induced solid tumours, mainly of the thyroid, breasts and lungs, increased. The latent period between the time of exposure and the appearance of cancer varied according to the nature of the tumour and the age of the subject at the time of exposure — for acute leukaemia it was 5—10 years in subjects who were irradiated during childhood and 10—15 years in those irradiated as adults. In the case of solid tumours, the latent period varied between 15 and 30 years and could be even longer for cancers of the digestive tract.

It was further established that the carcinogenic effect varied as a function of age. For many cancers the relative risk was higher in subjects exposed before the age of 20. The rate of acute leukaemia was higher in subjects irradiated at an age less than 10 or above 50. As for breast cancer, the relative risk was greatest in women between the ages of 10 and 19 at the time of irradiation.

Until 1975 leukaemia represented about a third of malignant tumours and accordingly offered the best sample for a quantitative study of carcinogenic effects. At Nagasaki, the frequency of leukaemia did not increase in subjects who had received less than 100 rad. It exceeded that observed in the non-irradiated group only in the group of subjects who had received more than 100 rad. Above this dose, the frequency of leukaemia rose with dose. The excess of leukaemia frequency observed in the Nagasaki survivors corresponds to an induction rate of about 20—30 cases of leukaemia per 10 000 subjects who had received 100 rad. In the Hiroshima survivors the induction rate seems higher; there appears to be no threshold and the frequency is already enhanced in subjects who had received more than a few tens of rad

Let us now consider the second source of information, namely subjects who have received radiation treatment for a non-malignant disease. One of the diseases for which patients have been most frequently irradiated is a specific form of rheumatism — ankylosing spondylarthritis. In Great Britain a study was conducted on about 20 000 subjects who had been treated by radiotherapy for this disease with doses of some hundred rads. No effect was observed in those who had received less than 300 rad, whereas in subjects who had received higher doses the frequency increased with dose. The number of leukaemia cases observed in the patients was about ten times greater than would have

been expected, given the age and sex of the persons irradiated. Apart from this increase in leukaemia, various other cancers showed a rise which was much smaller although statistically significant. There was likewise an excess of laukaemia or cancer cases in different groups of patients treated for other non-malignant diseases, while none was observed in other studies on various diseases (cervical cancers treated by radium, hyperthyroidism treated by radioactive iodine and so on).

A third source of information is the observation of subjects who were subjected to irradiation for occupational reasons. Only one example of these should be cited here, namely radiologists, they undoubtedly form the group of workers who have received the highest doses. A study of those who practised between 1920 and 1939 — a period when the hazards of ionizing radiations were still not very well known and precautions such as those used today were not taken — shows that the frequency of leukaemia among them was 10 times greater than that found in physicians who did not use radiation, such as ear, nose and throat specialists; the frequency among general practitioners who frequently used X-rays was intermediate.

It is interesting to note that among radiologists who practised after 1946, i.e. at a time when the effects of ionizing radiation were better known, no increase in the frequency of leukaemia was observed, undoubtedly because doctors and pracitioners, as generally all workers exposed to ionizing radiation, complied with the ICRP standards. This shows the importance and effectiveness of those recommendations.

On the whole, the numerical data from these different studies agree satisfactorily. In the three cases we observe that the incidences are close to one another if we express the results per 10 000 persons irradiated with 100 rem or per million persons irradiated with 1 rem, which is quantitatively the same thing. Let us note that the latter form of expression is artificial, because no effect was observed in subjects who had received less than 100 rem; nevertheless this is the presentation used most frequently in international reports for reasons of simplicity. Thus, for example, for 10 000 subjects who have received 100 rem we should see about 30 cases of leukaemia, if we judge from the observations on the Hiroshima and Nagasaki survivors. Judging from the observations on ankylosing spondylarthritis patients we should observe only 12, but in this case only 40% of the haematopoietic tissue, i.e. bone marrow, was irradiated. With allowance for the percentage of irradiated bone marrow, we find for whole-body irradiation nearly the same figure as in the Hiroshima and Nagasaki survivors. Lastly, in the case of radiologists, it is much more difficult to evaluate the leukaemia-inducing effect because it is not known what doses they received. In attempting to evaluate their doses, we can take into account the apparatus used, the number of X-ray examinations per day, the duration of practice, and so on. We thus arrive at estimates lying between 400 and 2000 rad. If the upper limit of this range is taken as the basis for calculation, the figure would be 10 cases per million and per rad. If the lower limit is taken as the basis, we would get 50 cases per million and per rad. Well within this range lies the figure of 30 cases per million and per rad observed at Hiroshima and Nagasaki and in patients treated for ankylosing spondylarthritis, for whom the dose evaluation is more accurate. The data as a whole are thus consistent and suggest that chronic irradiation may be nearly as effective in causing leukaemia as irradiation for a shorter time.

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We have so far considered only leukaemia because this is the most frequently observed form of cancer in irradiated subjects. Needless to say, other cancers, too, have been studied with equal attention. For example, at Nagasaki no significant increase was found in the frequency of other cancers at doses below 100 rad, whereas above 100 rad the increase was statistically significant.

The susceptibility of different tissues or organs to radiation-induced cancer is highly variable. For some tissues doses have to be very high — several thousand rad — to cause cancers in 1 per cent of the irradiated subjects, while for others the same effect is obtained with doses ten times smaller. The most radiation-sensitive tissues include, in particular, the thyroid, breasts, lungs and bone marrow. For all these cancers, different studies have provided estimates which appear to agree with one another to within the limits of accuracy of each study. Several studies have compared the frequency of all tumours as against that of leukaemia alone and found the ratios to vary between 3 and 5. Knowing that the risk of leukaemia is about 2 per thousand for 100 rem, the total risk of cancer should be of the order of 1 per cent for 100 rem. This estimate agrees quite satisfactorily with that obtained by adding the risks for each tissue, and this is the evaluation arrived at in the reports of UNSCEAR and ICRP.

However — and we have to come back to this question, for it is basic — these evaluations are based on X- or gamma-ray doses above a hundred rem. A hundred rem is already a very high dose, much higher than may be received by workers occupationally exposed to radiations. Where lower doses are concerned, even if the possibility of an effect cannot be ruled out, there are no data which can be used to estimate the magnitude of the risk, because it is too low to be estimated directly. Although some results suggest that moderate doses of X-rays — a few tens of rads — can cause cancer, they cannot be relied upon for evaluation of the frequency of induction following such doses in view of the large statistical uncertaingy associated with the data relating to these levels of irradiation.

Some studies have claimed to observe an increase in the incidence of leukaemia and/or other malignant tumours in adults who have received low doses occupationally or from radiological examinations. The best known of these is the study by Mancuso, Stewart and Kneale.

These authors analysed the causes of death among 2184 workers exposed to radiation and 1336 non-exposed workers at the Hanford nuclear plant in the United States of America, concluding that those who died from cancer had received higher doses than the other workers. From this they deduced that the carcinogenic risk associated with radiation was greater than had previously been thought. A second analysis based on 4033 deaths, although producing smaller differences, seemed in their opinion to confirm the first study. However, other research workers using the same data on the mortality of workers at Hanford arrived at completely different conclusions. The study by Mancuso and co-workers has also been criticized from the statistical point of view by many authors and committees of experts, who considered in particular that the differences reported for some cancers were probably due to statistical fluctuations caused by the use of too small a population sample. This error was compounded by

splitting the instances of cancer into too large a number of sub-divisions. If variations in the incidence of cancer in a large number of small populations are sought, then it is not surprising that for some of these and for some cancers a statistical difference may be observed which appears significant but in reality is meaningless.

It seems improbable today that this type of study will produce any insights into the effects of low doses of radiation. Further, calculations in the UNSCEAR report show that unless the present estimates of the total cancer induction rate are substantially in error, it would require studies covering some millions of man-years on workers exposed to annual average whole-body doses of more than 1 rad to assess directly the cancer risk associated with such doses. There is thus little hope of obtaining reliable data on the effects of low doses in the near future.

Evaluation of the Low-Level Dose Risk and the Dose-Effect Relationship

To evaluate the risk from doses to which workers or the population are exposed, a very large extrapolation has to be made as the risk being assessed is that from doses approximately 100 or 10 000 times lower than those for which reliable data are available. An extrapolation of this magnitude requires accurate knowledge of the shape of the function which links dose and effect.

The most pessimistic theory — the one which predicts the greatest number of cancers — is the linear relationship with no threshold. This assumes that if a dose of 200 rem delivered to 10 000 people produces 200 cancers, then a dose of 1 rem to 10 000 people will produce one cancer. A relationship of this type is rarely observed in pharmacology. For most medicines or toxic substances (e.g. alcohol), the effect of low doses is proportionally very small or non-existent. The probability therefore is that this relationship overestimates the actual risk. However, there are other theories on the dose-effect relationship which are plausible and various sources of information can be used to determine which of them is the most appropriate.

The first consists in analysing data on human beings. In the case of the Nagasaki survivors exposed to a mean dose of 350 rad, the probability of radio-leucosis is 35 per million per rad. If the dose-effect relationship were linear, the probability per rad should remain the same for people exposed to 100 rad. The effect per rad is in fact almost halved, which means that between 100 and 350 rad the relationship is definitely not linear. This reduction of the effect as a function of dose suggests strongly though not conclusively that the dose-effect relationship in Nagasaki survivors is not linear but rather a power function (of the type $y = bx^n$). For doses less than 100 rad no increase in the incidence of leukaemia has been noted, the incidence observed actually being slightly lower than normal although this difference is not statistically significant. It should be pointed out, however, that although a linear relationship fits the data less well, it cannot be entirely excluded.

On the other hand, in the case of people irradiated at Hiroshima in whom the leukemogenic effect is due to neutrons, the dose-effect relationship seems to be linear — in other words, the leukaemia incidence is proportional to the neutron dose.

This difference between the forms of the dose-effect relationship for X-rays and neutrons is consistent with experimentally observed effects on cells and with theoretical predictions. Without going into details, the effect is proportional to the dose in the case of densely ionizing particles such as neutrons, because there is a very high probability that a single particle will create serious lesions in the nucleus of a cell (rupture of the two chains of the desoxyribonucleic acid (DNA) molecule). In this case, an effect proportional to the dose is expected — that is, a linear relationship ($N = \alpha D$).

However, in the case of weakly ionizing particles such as X- or gamma rays, energy transfers are much smaller and the lesions caused by the passage of each particle are much less serious and much easier to repair, except when two or more particles pass almost simultaneously through the same region of the cell nucleus (the breaking of the two chains of the DNA molecule usually requires the joint action of two particles). The probability that two particles will pass through the same volume is proportional to the square of the dose (βD^2).

For most human cancers, a linear relationship seems statistically unlikely and appears to overestimate the effect of low doses. However, some data on human beings, particularly on thyroid and breast cancers, are equally compatible with a quadratic or a linear relationship, thus making it impossible to exclude either of these types of dose-effect curve.

What information can we expect to gain from experimental research to help us to choose the correct dose-effect relationship?

It is generally accepted that the induction of a tumour is the final outcome of a succession of separate events. The study of cancer induced by chemical products shows that at least two separate stages can be distinguished: initiation and promotion. Initiation is a fast, irreversible process which confers neoplastic characteristics on a normal cell. Promotion is the process whereby a transformed cell gives rise to a tumour capable of growing and invading neighbouring tissues. The promoters are usually agents which stimulate cell proliferation. Initiation is probably associated with a lesion in the genetic material, and some data suggest that for this transformation a linear relationship for low doses of X- or gamma-rays would overestimate the effect. However, "transformation" and "carcinogenesis" cannot be equated, particularly as the "initiated" or "transformed" cell may remain in a "dormant" state without proliferation indefinitely.

The difficulties associated with the transformation from cellular initiation to cancer as such are underlined by the considerable variations in cancer induction depending on the tissues concerned, following total irradiation in which all cells have received the same dose. Thus, the incidence of cancer is relatively high in the thyroid and breast and very low or virtually zero in the prostate and testicle, but the reason for these differences remains unknown. Moreover, very appreciable differences are observed in a given tissue, depending on age; in the case of women, for example, the susceptibility of the breast to radiocarcinogenesis is relatively high between the ages of 15 and 20, but very low before puberty and in adult and elderly women.

The appearance of tumours following irradiation has been the subject of extensive study in animals. For tumours following exposure to X-rays, the effect per unit of absorbed

dose decreases with the dose rate; this is not true for heavy particle irradiation. For most tumours observed in animals (leukaemias, kidney, skin and lung tumours), data on carcinogenesis due to low X-ray doses can be represented by a quadratic polynomial or by a linear dose-effect curve with a threshold or quasi-threshold, whereas cancer induction by heavy particles is better represented by a linear dose-effect relationship. In some cases the incidence of tumours actually decreases at low doses, while in one instance (mammary neoplasia in the Sprague-Dawley rat) a linear no-threshold curve was obtained after exposure to X-rays. These differences in the mathematical form of the dose-effect relationship are not surprising in view of the diversity and complexity of carcinogenic processes.

Radiation protection is concerned with the effect of low doses delivered over long periods. Ideally, therefore, one should not just estimate the effect of low doses on the basis of that observed for high doses: one should also take into account the fact that the dose rate is much lower. All experimental data suggest that when a given radiation dose is spread over a longer period of time its effect is reduced — for example, the mutagenic effect of radiation at low dose rate is three times less than at a high rate. This is also true of most other toxic agents (the effect of drinking a litre of alcohol in an hour is not the same as drinking it over a month). Nonetheless, this factor is deliberately disregarded and, for radiation protection purposes, the risks associated with low rates are considered to be the same as those associated with high rates.

To sum up, although extensive data exist which allow us satisfactorily to assess the risk to man posed by high doses and dose rates, they do not allow definitive conclusions regarding the form of the dose-effect relationship for carcinogenesis in man nor regarding the effect of low X-ray doses. Theoretical analyses and data obtained from simple or animal systems suggest that for X- or gamma-rays a relationship of the linear type overestimates the effect of low doses, and all information concurs to suggest that for doses of the order of a few rads the real risk is probably 4—10 times lower than that predicted by the linear extrapolation. The risk is probably still lower for smaller doses and low dose rates.

However, in any evaluation of a harmful effect it is preferable to overestimate than underestimate the risk. It is probable therefore that a linear no-threshold relationship will continue to be used for some time in risk estimations.

GENETIC RISK EVALUATION

The human environment contains a large number of mutagenic agents which, when the mutations affect the cells of the ovary or testicle, are capable of causing genetic damage. Radiation was the first of these to be discovered and is the best known, but there are other agents — molecules produced during the burning of coal, oil and tobacco are mutagenic, for example. The most recent data suggest that the genetic risk to man from radiation is smaller than had previously been thought. The most important fact in this respect is that the Hiroshima and Nagasaki studies revealed no abnormal genetic effect;

the data obtained on the frequency of congenital defects, morphology and life expectancy have shown no discernible effects in the descendents of Hiroshima and Nagasaki survivors by comparison with the children of parents who had not been irradiated. In studies on animals, more than 100 generations of mice have been exposed to 200 rads per generation without it being possible to detect any adverse effect on the viability or fertility of their descendents. The results of these intensive radiation studies are thus reassuring.

However, this does not mean that radiation has no genetic effect. The absence of deleterious effects in individuals suggests rather that the body's safeguard mechanisms eliminate most embryos with serious genetic defects right at the beginning of pregnancy. This mechanism is not, however, taken into account in genetic risk evaluations, which are based on a large number of studies measuring the frequency, per unit of dose, of various types of genetic mutations induced in mice and insects, in particular the fruit fly, or drosophila. Comparison with recent studies on cultured human cells suggests that at the cellular level the dose-effect relationship takes the same form in all species.

UNSCEAR, ICRP and BEIR recently carried out independent reviews of the huge volume of data currently available on genetic effects, and their conclusions, although not identical, confirm each other.

The UNSCEAR and the BEIR reviews estimate the number of induced defects at a little under 200 in the descendants of a million subjects who received 1 rem. Of these, perhaps a quarter or half are likely to be expressed in the first generation and the remainder in the next 10—15 generations. The ICRP critical review reached similar conclusions for all generations. If we consider the dangers to which an individual is exposed, the genetic harm of greatest personal impact may be thought to be that of the risk of abnormalities in children, grandchildren and great-grandchildren. The average risk would be 30—40 genetic defects per million children following the exposure of their parents to 1 rem, or about $\frac{1}{3}$ of the figure obtained for the somatic risk of cancer. To put this risk in its proper perspective it should be compared to the so-called natural incidence which is about 107 000 per million.

Further, when assessing the harm per man-rem distributed through a working population aged 20—65, or among the population as a whole, it must be remembered that $\frac{2}{3}$ of the man-rem will have no harmful genetic consequences since they will affect individuals who are going to have no more children in any case. Similarly, it should be pointed out that the cancer risk for workers is overestimated because average induction rates are used whereas it is known that the rates are higher for children and adolescents.

RISKS TO THE PUBLIC AND TO WORKERS

The maximum total risk, including both the carcinogenic risk and the genetic risk for descendents, is 1.3×10^{-4} per rem. It is thus easy to assess the maximum risk limit knowing the doses received by the public from the UNSCEAR estimations and by workers from direct measurements (Table 2). It should be noted firstly that this risk is relatively

Table 2. Occupational Irradiation
(In the USA, from the National Academy of Sciences Report, Washington 1980)

	Average dose (mrem/yr)
200 000 occupationally exposed to medical X-rays (doctors and technicians)	300–350
200 000 occupationally exposed to dental X-rays (dentists and technicians)	50–125
100 000 occupationally exposed medical personnel handling radioisotopes (doctors and technicians)	260–540
30 000 occupationally exposed in civilian nuclear power plants and reactor installations	600–800
35 000 occupationally exposed in naval nuclear propulsion plants (submarines etc)	130–330
100 000 occupationally exposed in research and development (civilian research centres, universities etc.)	130–330

low even with very high, near-lethal doses. The excess in the number of cancers is relatively small by comparison with that caused by other human activities; for example, in the group of atomic bomb survivors who received the highest doses, it is equivalent to the excess observed in Californian women treated with oestrogen for menopause problems or, to give another example, the number of cancer deaths caused by radiation in the Hiroshima and Nagasaki survivors is at least twenty times lower than the number of cancer deaths in that population caused by the use of tobacco. It is, furthermore, the very fact that the effect is so small which explains the difficulties involved in carrying out a precise evaluation.

For a population dose of 2 mrem/yr in the year 2000, this would mean that in a population equivalent to that of France (50 million inhabitants) the use of nuclear power for electricity production would cause about 2–10 cancer deaths each year This figure may seem large, but it should be emphasized that, on the one hand, it is calculated on the basis of a deliberately pessimistic assumption and that the actual figure may be lower or even zero, and, on the other hand, that this risk is small compared to the total number of cancer deaths (about 120 000 annually in France, see Table 3) and that cigarettes alone cause 20 000 cancer deaths in France every year. The table gives an indication of the main causes of death in France and this allows us to compare radiation risks with others.

Table 3. Main Causes of Death in France

Causes	Number of deaths per annum (France)	Annual risk rate (per 10^6 persons)
Total death rate	≅600 000	11 500
Traffic accidents	≅15 000	285
Accidents in the home	≅5 000	≅100
Accidents on holiday	≅3 600	180
Industrial accidents and occupational illnesses	≅2 300	160
Fires	≅2100	40
Lightning	≅25	0.5
Electrocution	≅200	≅4
Total cancers	≅120 000	2 300
Leukaemias	≅4 000	77
Tobacco	≅70 000	1 300
Alcohol	≅40 000	750

The risk attached to other elements of everyday life can also be assessed in the same way — take for example the carcinogenic effect of tobacco. Statistical data compiled on several million people show that from three cigarettes a day (about 1000 cigarettes a year) upwards there is a significant increase in the risk of lung cancer; if ten cigarettes are smoked a day, this risk is increased by a factor of about 8 and, for 20 cigarettes a day, by a factor of about 20. If we assume a linear relationship between the number of cigarettes smoked and the effect, the risk from one cigarette smoked a day, or a year, or in a lifetime can be assessed using a rule of three. Similar assessments can be made for the risk of alcohol-induced cirrhosis or cancer of the oesophagus due to one gram of alcohol, the risk of a fatal traffic accident per kilometre travelled and so on.

If we now compare these risks to those from irradiation (Table 4), we see that the risk attached to one cigarette is equivalent to that from the radiation dose received by workers during half a day of irradiation at the maximum permissible dose or from three years of living in the vicinity of a nuclear plant.

If the risk is to be assessed objectively in the case of workers, it must be seen in the light of other occupational risks.

Table 5, taken from Reissland, indicates the order of magnitude of the average reduction in life expectancy which, under the most pessimistic hypotheses, would result from

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Table 4. Comparative Risks

The following incur a one in a million risk of death:

650 km air travel

100 km car travel

$\frac{3}{4}$ cigarette

1.5 minutes mountaineering

20 minutes of life at age 60

Use of oral contraceptive pills for $2\frac{1}{2}$ weeks

$\frac{1}{2}$ bottle of wine

Exposure to 10 mrem: $\frac{1}{2}$ day exposure at maximum permissible dose (occupational) or living three years in the vicinity of a nuclear plant.

Based partly on Pochin.

Table 5. Average Reduction in Lifespan (in days) (New Scientist 13.9.79)

	For one year of working life (person aged 40)	For 35 years of working life
Deep-sea fishing	31.9	923
Coal mining	3.6	103
Oil refinery	2.6	74
Railways	2.2	63
Construction	2.1	62
Industry (average value)	0.5	13.5
Occupational exposure to radiation at 5 rem/yr	1.3	32
Occupational exposure to radiation at 0.5 rem/yr	0.1	3

Based on Reissland.

irradiation at the maximum permissible dose. It is comparable to that observed in common occupations. Moreover, the average dose received by exposed workers is, as experience has shown, on average ten times less than the permissible dose. The nuclear industry is thus to be classed with moderate-risk occupations.

Finally, many papers (which are reviewed elsewhere and which have been presented at several scientific meetings) have attempted to compare the risks to the population and workers of the different methods of energy production. For the purposes of comparison these risks must be related to the production of 1 MWe/yr by each system. It should be pointed out, however, that the nuclear power industry suffers from a serious handicap in these comparisons. Statistical data are available on the health effects of other systems but since no health effects have ever been noted in the nuclear industry, the risk is evaluated on the basis of theoretical considerations which, as has been seen, result in an overestimation of the risk for the sake of caution. Moreover, risk estimation for nuclear power includes the genetic risk for descendants; this is not the case in traditional health methodology nor in the risk estimation applied to the other systems.

It will no doubt be pointed out that while nuclear power risks may be minimal during normal operation, they can soar if there is an accident. The answer to this is, firstly, that the experience already acquired (several thousands of reactor years) shows that accidents are rare and, secondly, that they are statistically predictable (see the Rasmussen report among others). Most reports include in the health risk estimation those risks caused by contamination from accidental releases (Windscale or Three Mile Island type) and even the possibilities of a nuclear disaster. Even when these are taken into account the risk to health presented by the nuclear power industry remains low in comparison with the risks associated with other types of power production; nor does the nuclear industry have a monopoly on accidents — they occur in all power systems. One need only recall breaks in hydroelectric dams, accidents during the transport of oil or methane, fires in refineries, accidents in coal mines and so on.

In sum, thanks to the extraordinary sensitivity and accuracy of measurement methods and to the research carried out since the end of the 19th century, the risks from ionizing radiation are undoubtedly among the best-known. They seem to be, relatively speaking, very low in the vicinity of nuclear plants which have benefited from radiological protection measures developed by radiologists to protect themselves and to protect those who are ill. The health effects of accidents can be estimated and they seem to be low as a result of the elaborate safety measures taken — lower than or comparable to the risks from accidents in traditional industrial or hydroelectric plants. Paradoxically, anxiety has been aroused precisely by the cautious attitude adopted and the extremely sophisticated nature of the precautions taken as well as perhaps by the special connotations which the word "atom" evokes in the minds of the public and even some scientists.

References

CROW, J F., "Can we assess genetic risks? ".

Proceedings of the Sixth Congress of Radiation Research (Ed. Tokada, et al.), Tokyo (1979) 70–8.

FINCH, C.S , "Hiroshima and Nagasaki", A review of 30 years of study
Proceedings of the Sixth Congress of Radiation Research (Ed Tokada, et al.), Tokyo (1979) 50–8

ICRP Report No.26, Recommendations of the International Commission on Radiological Protection, Annals of the ICRP, Vol 1, No 3, Pergamon Press, Oxford (New York City, 1977)

United Nations Scientific Committee on the Effects of Atomic Radiation, 1977 Report to the General Assembly, Sources and Effects of Ionizing Radiations, United Nations publication Sales No.E.77.IX., Vol 1 (New York, 1977) 1–725.

Colloque sur les risques comparés des différents modes de production d'énergie (Paris, janvier 1980).