REVIEW ARTICLE

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Radiation risks in perspective: radiation-induced cancer among cancer risks

Abstract The majority of the public in industrial countries believes that pollution and low doses of radiation are threats to good health. As a matter of fact, when these putative risks are compared to those originating from lifestyle, they appear very small. In particular, the risks associated with low doses of irradiation, even when they are assessed with the most pessimistic models, appear extremely small. Public anxiety is fuelled by the uncertainty regarding the magnitude of this risk and the use of the linear no threshold (LNT) hypothesis, which gives credence to the concept that even the smallest doses are harmful. There are a number of scientific and epidemiological data currently under debate that are not consistent with the LNT hypothesis. For example, no difference in the incidence of cancers or of birth defects has been observed between regions with low or high natural irradiation. This inconsistency between perceptions and data underlines the role of psychological factors studied since 1957 which should be placed in the perspective of the public's present attitude toward risk and technology. Social amplification or attenuation of risk may occur in several ways. Fearful concern about radiation began in 1955, with the beginning of the Cold War, when the possibility of a nuclear holocaust appeared very real. Analysis of the data shows that these fears of technology could have a detrimental effect; they should therefore be investigated and understood.

Key words LNT hypothesis · Radiation carcinogenesis · Non-radiation carcinogenesis · Dose-response relationships · Radiation risk · Risk perception

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Introduction

In the industrialized countries, the majority of the public believes that the modern environment is a threat to good health. They feel that air and water pollution, pesticides and fertilizers which are present in most food, the so-called modern industrialized agriculture, and nuclear energy have impaired their health. Even when ill, some hesitate to take antibiotics because of their possible toxicity. In a survey carried out in the USA in 1995, the following question was asked: Do you feel that the risks to which our health is currently exposed are greater, equal to or smaller than 30 years ago? The answers were 75%, 15% and 10%, respectively [1].

Fortunately, the actual data do not support these fears. On the contrary, they show that in industrialized countries health is continuously improving [2]. Life expectancy is the most reliable index for the health of a population because it integrates and sums up all the physical and mental stresses to which human beings have been submitted since their birth. Life expectancy has dramatically increased in the Western world since the end of World War II. For example, Table 1 shows that in France the life expectancy of the population has increased during the 20th century from 44 years in 1900 to 78 years in 1997. This amazing progress continues. In France, women's life expectancy is the longest in the world, together with that of Japanese women, which shows that medical and environmental factors (despite the development of nuclear energy from which over

Table 1 Health indicators in France [2]

	1900	1980	1997
Life expectancy at birth	Male 44 Female 45	Male 70.2 Female 78.4	Male 74.2 Female 82.1
Life expectancy without major handicap		72.6	75.1
Infant mortality	150‰	15.5‰	5‰

Table 2 Causes of death in USA in 1990 [97]

a: Leading causes of death		b: Estimated causes of death due to lifestyle	
Heart disease	720,058	Tobacco	400,000
Cancer	505,322	Diet/inactivity patterns	300,000
Cerebrovascular disease	144,088	Alcohol	100,000
Unintentional injuries	91,983	Certain infections	90,000
Chronic lung disease	86,679	Toxic agents	60,000
Pneumonia and influenza	79,513	Firearms	35,000
Diabetes	57,664	Sexual behavior	30,000
Suicide	30,906	Motor vehicles	25,000
Chronic liver disease	25,815	Drug use	20,000
HIV infection	25,188	Total	1,060,000
Total	1,757,216		

Table 3 Male mortality in France in 1996 (according to Haut Comité Santé Report, 1998)

Estimated deaths due to various diseases	Deaths	Premature (before 65 years of age)	Estimated deaths due to lifestyle	
Cancers	90,000	28,000	Tobacco	60,000
Cardiovascular diseases	79,500	12,500	Alcohol	35,000
Violent deaths	26,000	16,300	Accidents	25,000
Respiratory diseases	22,000		Total	120,000
Gastrointestinal diseases	14,000			
Total	276,000	80,000	Between 25 and 65 years of age≥55% deaths due to lifestyle	
			Pollution	about 3,000

Table 4 Potential years of life lost (men) in France due to life-style.

	%		Years
Cancer due to tobacco and alcohol	10	Traffic accidents	500,000
Cardiovascular diseases (tobacco)	4	Tobacco	700,000
Cirrhosis and psychosis (alcohol)	5	Passive smoking[a]	45,000
Accidents	12	Air pollution[a]	15,000
Suicides	12		
AIDS	5		

[a] The evaluation of the potential number of years of life lost due to passive smoking and environmental pollution was not carried out by HCSP [2] and are, therefore, imprecise estimations of which the former is based on a report by the French Academy of Medicine on passive smoking (1997 [98]) and the latter is based on a report by the French Academy of Science [10]

80% of electricity is made) are among the best in the world. The excess mortality in men has been well analysed and is mainly due to three causes: accidents, alcohol and tobacco. Furthermore, life expectancy without major disability increases at the same pace of about 2 months per year and is only three years shorter than that of life expectancy. That means, not only do we live longer each year, but we also enjoy life longer. This conclusion is valid for all Western industrialized countries as well as for Japan. Unfortunately, in the countries of Eastern Europe, in particular those of the former Soviet Union, life expectancy over the past three decades has not only not increased but has in fact decreased. The causes of the higher death rates during adulthood appear to be alcohol, tobacco, a diet too rich in animal fats (sausages, pork fat, etc.) and psychological distress rather than a failure of the medical system, since in most Eastern European countries the death rates of infants did not increase significantly [3].

If we now consider all causes of death (Tables 2 and 3) by disease and by causes of diseases, we find that diseases related to human behaviour (tobacco, alcohol, accidents, particularly traffic accidents, suicide, obesity and lack of exercise) account for about half of the deaths and for two-thirds of premature deaths (before 65 years of age). On the other hand, pollution causes at worst about 3000 deaths per year in France, less than l% of spontaneous deaths. If we express these data in loss of years of potential life (Table 4), the difference becomes even more impressive since unfortunately deaths due to behaviour occur at younger ages rather than those due to pollution or natural diseases.

Non-radiation carcinogenesis

The main aim of this paper is to compare cancer risks caused by radiation and other environmental factors with

those originating from lifestyle. Doll and Peto [4] estimated their respective roles about two decades ago. More recently, Hill et al. [5] updated these risk estimates and adapted their data to the situation in France. In 1996, the Harvard report on cancer prevention provided another estimation [6]. In conclusion, the three estimations concur to show that about 75% of human cancers are associated with lifestyle (Table 5). In industrialized countries, about half of the human cancers could easily be prevented by changes in individual behaviour such as the implementation of the recommendations of the European Action Against Cancer: smoking cessation, drinking less than the equivalent of half a litre of wine per day, a diet with plenty of vegetables and fruit and less rich in animal fat and, furthermore, avoiding excessive sunbathing, in particular under the age of 15 years (Table 6). On the other hand, the total percentage of cancer attributable to pollution (air, water and food) appears to be less than

2%. For example, the relative risk (RR) of lung cancer in workers exposed to severe air pollution (in garages or for truck drivers) is equal to 1.33 [7]. Thus, it is the same as for individuals exposed to passive smoking (RR between 1.25 and 1.4) [8, 9]. For the general public, the cancer excess due to air pollution is not detectable [10], and the relative risk is at least ten times smaller than from passive smoking. It has been estimated that in France passive smoking causes about 150 lung cancers per year [9]. The number of cancers related to outdoor air pollution in the general public should therefore be much smaller. The absence of any significant influence of air pollution on the frequency of lung cancers is corroborated by the fact that the lung cancer incidence in non-smokers (Table 7) has been constant during the past four decades despite air pollution and the growing importance of passive smoking. Similarly, it should be recalled that the incidence of stomach cancer in Western countries is currently four times lower than it was four decades ago [5, 6] despite the presence in food of increasing amounts of potentially carcinogenic chemical products (pesticides, fertilizers, herbicides, etc.). It has often been claimed that nitrates could be transformed in the stomach into nitrites and nitrosamines, which are potent carcinogens. However, there is no correlation between the nitrate content in drinking water and stomach cancer incidence.

Table 5 Causes of cancer deaths [6]. The proportion of cancers that could be avoided through changes in lifestyle (tobacco, alcohol, half of those associated with nutrition and sedentary lifestyle, the sun) can be calculated: approximately 50%. The numbers in this table are based on American studies; in France, the numbers are similar, except for tobacco, which is slightly lower (25%), and alcohol, which is significantly higher (about 10%)

Tobacco	30%	Avoidable by changes in lifestyle (mean 50%)
Nutrition	30%	
Sedentary lifestyle	5%	
Alcohol	3%	
Solar and ionizing radiation	2%	
Chronic infections	5%	
Reproductive factors	5%	
Socioeconomic status	3%	
Medicinal drugs	1%	
Food additives (salt)	1%	Total pollution
Pollution	2%	(2%–2.5%)

Table 7 Approximate constancy of US non-smoker lung cancer death rates in the American Cancer Prevention Studies I and II (prospective studies of more than one million men and women), taken from Garfinkel [99] (annual mortality/100,000, age-adjusted for US population 1970)

	Male	Female	Both sexes
1960s	6	4	5
1980s	6	5	5
Mean of both periods	6	4	5

Table 6 'Avoidable' cancer mortalities in France by prevention, either collective (reduction in pollution) or individual (changes in lifestyle), according to [5] and [100]

Acts of prevention[a]	In the present[b] (%)	In the future[b] (%)
Avoid tobacco	22	35
Avoid alcohol	12	10–15
Avoid tobacco & alcohol	27.4	36
Avoid obesity[c]	2	≤2
Avoid & treat sexually transmitted diseases	2	≤1
Avoid excessive exposure to UV radiation	<1	≈1
Limit carcinogenic medical acts: radiology, hormones, cytotoxins	<1	≈1
Eliminate known human carcinogens:	4	<1
a) from industrial environment	2	<1
b) from general environment	<1	<1
Eliminate hepatitis B	<1	<1
Reduce radiation of military & industrial origin	<0.01	0.01

[a] The means listed are changes in environment or lifestyle that are, at least in part, achievable and do not include: pregnancy, prophylactic mastectomy, prostatectomy, hysterectomy, etc.
[b] Mortality in the present is the result of exposures in the past; mortality in the future is the result of exposures in the present.
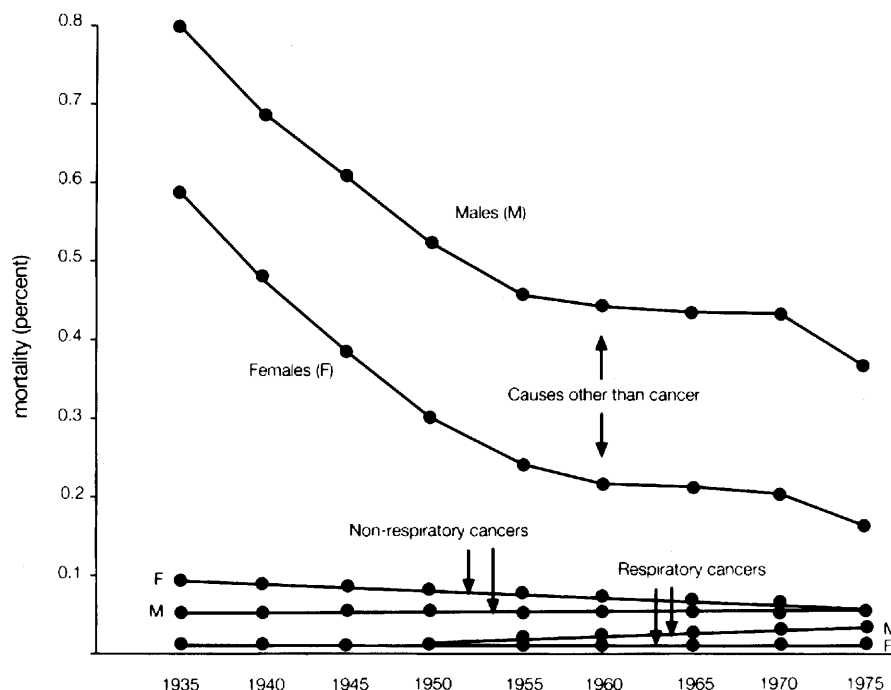[c] Reduction in the consumption of lipids (in particular saturated animal fat) would reduce cardiovascular risks and perhaps also some cancer mortalities.

Fig. 1 Time course of changes of mortality due to cancer and other causes for an age-standardized population in the USA (1935–1975)

More generally, in populations aged 35–70 years in Western Europe and the USA, there has been no significant variation of cancer incidence for an age-standardized population since 1930, although millions of tons of thousands of new chemicals (pesticides, fertilizers, herbicides, fibres and plastics, etc.) which were synthesized during and after the Second World War have been released into our environment. The increase in cancers of the respiratory system which is entirely due to tobacco has been balanced by a decrease in non-respiratory cancers (Fig. 1). The misconception regarding carcinogenic agents present in the environment has been emphasized by Ames and his colleagues in several papers [11, 12, 13]. Plants contain natural pesticides, many of which are carcinogens, as a defence against predators and parasites. Indeed, natural carcinogens are present in nearly all vegetables and fruits, e.g., apples, bananas, and carrots as well as coffee, lettuce, potatoes, tomatoes, cabbages, and many others. Ames and Gold [13] estimate that there are about 10,000 times more pesticides from natural plants than those originating from pollution. Yet, the cancer incidence is inversely correlated with the amount of vegetables eaten each day.

These data show that the carcinogenic effect of low concentrations or low doses of genotoxic agents is very small, if any, even when these agents, such as benzene, are highly carcinogenic at high doses. In fact, there is no evidence of any carcinogenic effect of carcinogens at low concentrations or doses. The study of tobacco is of great interest with regard to the dose-effect relationship. At the world level, tobacco is by far the primary human carcinogen since it causes about 30% of cancers in men [4, 5, 6, 14]. The relationship which has been proposed is $F = kD^2 t^{4...5}$ where the dose, D, is the number of ciga-

rettes smoked per day, t the duration of the smoking time in years, F the probability of lung cancer induction, and k a constant [15]. In France, active smoking causes about 21,000 lung cancers per year (corresponding to a mean number of 15 cigarettes smoked per day), and passive smoking (corresponding to about 1 cigarette per day) causes about 100 lung cancers per year [9]. These figures are compatible with the above quadratic relationship, whereas a linear relationship would predict about 1500 cancers induced each year by passive smoking.

Among smokers, after smoking cessation, the relative incidence of lung cancer decreases and plateaus at an incidence 5–10 times lower than in current smokers [16]. The relative risk (RR) increases with age at quitting and decreases with years since quitting. For men who started smoking at age 17.5 and smoked 26 cigarettes/day, at age 75 the RR for former smokers compared with current smokers is approximately 45% for those quitting in their early 60s, 20% for those quitting in their early 50s, 10% for those quitting in their 30s and 5% for those who have never smoked. Thus, 40 years after smoking cessation, the incidence in men who inhaled huge amounts of carcinogens every day for about 20 years is only two times greater than in non-smokers. This relatively small impact emphasises the predominant role of the promoting effect of tobacco such as chronic irritation, cell proliferation and epigenetic factors. The mutagenic effect is at least five times smaller than the promoting effect [17].

Most carcinogens identified by epidemiology are promoters such as alcohol, chronic irritation, infection or inflammation, parasites, etc. There is not only a quantitative but also a qualitative difference between low doses and high doses of genotoxic substances because the latter
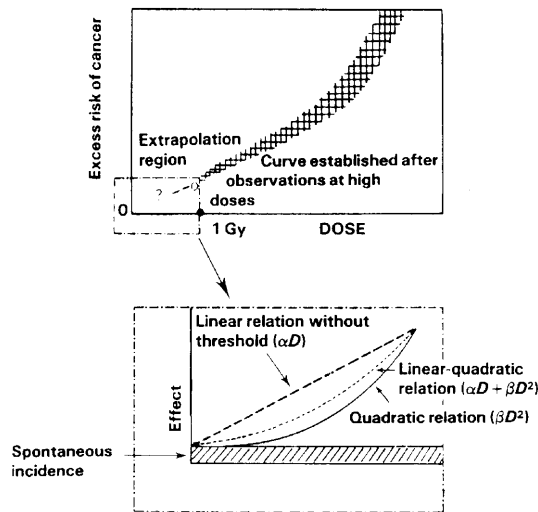
Fig. 2 Extrapolation of the dose-effect relation to low doses. Several studies have established a dose-effect relation with doses greater than 1 Gy, but none has provided reliable data below 0.5 Gy. The effect of these low doses is the relevant factor in radiation protection. For this purpose it is necessary to know the shape of the dose-effect relation in order to extrapolate between the spontaneous incidence and that found after irradiation to 1 Gy. The figure shows that the evaluation of risk depends on the function chosen

causes cell death and thereby a compensatory increase in cell proliferation [18, 19]. Cell division is a critical factor in mutagenesis. Due to the occurrence of errors during DNA synthesis and mitosis, rapidly dividing cells have a greater probability of mutating. After exposure to a physical or chemical agent which causes DNA damage, DNA repair mechanisms are most effective when cells do not divide. DNA repair is impaired or arrested during DNA duplication and cell mitosis [11, 18, 19]. During these stages of the cell cycle, the lesions are fixed and become irreversible. The proportion of irreversible lesions, both in vitro and in vivo, is much smaller when cells remain quiescent. Therefore, any agent causing chronic mitogenesis can be indirectly mutagenic just because it increases the probability of converting DNA damage into mutations [11]. High doses of any toxic agent which provoke the death of a significant proportion of cells enhance the carcinogenic effect of the mutagenic agent, whereas low doses do not kill cells [19]. These data do not exclude the possibility of carcinogenesis in quiescent tissues, but they are not consistent with the hypothesis of proportionality between the dose of a mutagenic agent and its carcinogenic effect.

For risk assessment, the main problem from a medical point of view is the shape of the dose-effect relationship for doses or concentrations below which an effect is detectable in epidemiological studies. The absence of detectable effects does not preclude the existence of an excess in cancer incidence too small to be detectable by epidemiological studies. These risks have been called 'virtual' [20] because their existence is not certain and their estimation is to a large extent subjective (Fig. 2).

Table 8 Variations with age of radiation risk (RR) for thyroid cancer (after exposure to 1 Gy) (thyroid weights: birth 1 g, 6 months 2 g, 4 years 4 g, 10 years 20 g, 18 years 20 g; *NS* not significant)

Patients [21]		Survivors of Hiroshima and Nagasaki [101]	
Age	RR	Age	RR
0–4 years	40	0–9 years	9.5
5–9 years	20	10–19 years	3
10–14 years	10	20–39 years	0.3 NS
20–30 years	1	>40 years	0.2
>30 years	0		

Radiation carcinogenesis

Radiation carcinogenesis does not differ fundamentally from chemical carcinogenesis. For example, the delays between inception of smoking or exposure to an occupational carcinogen and the emergence of cancer are similar to those observed in radiation carcinogenesis: 20–50 years. In radiation carcinogenesis, as well as for other carcinogenic agents, cell division following exposure greatly enhances the incidence of cancer. This is particularly true for thyroid [21] or breast cancer [22]. In girls having undergone radiotherapy for Hodgkin's disease, the risk of breast cancer seems to be highest when irradiation was delivered during breast growth associated with menarche. This is consistent with an increased risk in rapidly proliferating breast cells [102]. In infants or children under 10 years of age, the thyroid is the organ with the greatest susceptibility to radiation carcinogenesis, whereas after 20 years of age the carcinogenic effect is indeed extremely small, even following acute irradiation probably because the cell mitotic rate has become very low (Table 8).

An increase in the incidence of cancer following irradiation has been reported in two situations:

- chronic irradiation: occupational exposure (e.g. uranium miners) or heavy contamination. In these cases, radiation acts like tobacco, as both a mutagenic factor and a promoter. Excess leukemias have been reported for doses of ≥500 mSv, particularly in workers at the Sellafield Plant. There is, however, no evidence of excess leukemia due to lower doses. Even in regions of high natural background irradiation (10–100 mSv/year), no excess in cancer or leukemia incidence has yet been observed.
- acute irradiation such as radiotherapy, or A-bomb survivors in Hiroshima: an excess is found only for doses ≥200 mSv [23]. Among radiotherapy patients, an increase in the incidence of thyroid cancer has been reported in children for doses ≥100 mSv. A comparison between high-dose and low-dose rates (brachytherapy with radioactive implants) has shown that the latter are less leukemogenic [24, 25, 26].

Dose-response relationships

As is the case for chemical carcinogens, the main problem is the shape of the dose-effect relationship for small doses (below 200 mSv) [25, 26, 27, 28]. The magnitude of the risks calculated by extrapolation varies widely with the mathematical relationship used for their assessment. The aim of the scientist should be to reduce the uncertainty by excluding the relationships which are not consistent with scientific knowledge on carcinogenesis and by increasing the accuracy of epidemiological studies, but this is a difficult task because both scientific considerations and the precautionary principle must be taken into account. It is, therefore, not surprising that the choice of the dose effect relationship is controversial and that the choices are different for radiobiologists, who put emphasis on scientific validity, and for some radioprotection specialists, who insist on the precautionary principle. It is in fact impossible to exclude the carcinogenic effect of low doses of radiation or chemical carcinogens even on quiescent tissues [29, 30]. Moreover, as human beings are exposed to a large variety of carcinogens, the possibility of synergistic interactions cannot be excluded. However, the theoretical possibility of a small carcinogenic effect of low doses does not signify that the relationship ought to be linear. There could be a practical threshold, if for very small doses the probability of a carcinogenic effect is very low, or other relationships such as a quadratic one.

LNT hypothesis

In 1965, the International Commission of Radiological Protection (ICRP) [28] advocated a linear no threshold relationship (LNT) and explained this choice as follows: 'As the existence of a threshold dose is unknown, it has been assumed that even the smallest doses involve a proportionately small risk of induction of malignancies. Also, because of the lack of knowledge of the nature of the dose-effect relationship in the induction of malignancies in man – particularly at those dose levels which are

Table 9 Risks attributable to human activities (according to [31])

There is a 'one in a million' risk of death from:

650 km in an aeroplane
100 km in a motor car
Smoking one cigarette
2 h of passive smoking
1–5 min of rock climbing
1–5 weeks of work in a normal factory
1 h of sea fishing
2–5 weeks on the contraceptive pill
Drinking half a bottle of wine
Exposure to 0.1 mSv, i.e.
   (a) exposure to the maximum permissible dose (workers)
      for half a day
   (b) 3 years of life close to a nuclear power station
   (c) mean dose received during 3 months from diagnostic
      radiology

relevant in radiological protection – the Commission sees no practical alternative, for the purposes of radiological protection, to assuming a linear relationship between dose and effect, and that doses act cumulatively. The Commission is aware that the assumptions of no threshold and of complete additivity of all doses may be incorrect, but is satisfied that they are unlikely to lead to the underestimation of risks.' Pochin [31], at that time chairman of ICRP, aimed at reassuring the public by demonstrating the insignificance of the risk, as assessed by pessimistic linear extrapolations, in comparison with other risks of daily life (Table 9). Unfortunately, the opposite was achieved. The LNT hypothesis fuelled anxiety because it supported the concept that any dose, even the smallest, is carcinogenic. The detrimental psychological impact of the LNT hypothesis was brought to light after the Chernobyl accident in 1986, when minute doses of the Chernobyl radiation were utilized to calculate that 53,400 persons would die of Chernobyl-induced cancer over the next 50 years [32, 33]. This frightening death toll was derived simply by multiplying the trifling Chernobyl doses in the USA (0.0046 mSv per person) by the vast number of people living in the Northern Hemisphere and by a cancer risk factor that was based on epidemiological studies of 75,000 atomic bomb survivors in Japan who received more than 200 mSv at a dose rate of 6 Sv/s.

The scientific validity of the LNT has been challenged over the past decades [25, 26, 34, 35, 36, 37, 38, 39]. Abelson, in a critical editorial published in *Science* [40] stated: 'To calculate effects of small doses, a linear extrapolation from large doses to zero is employed. The routine use of this procedure implies that pathways of metabolism of large doses and small doses are identical. It implies that mammals have no defence against effects that injure DNA. It implies that no dose, however small, is safe. Examples of instances in which these assumptions are invalid are becoming numerous. Linear extrapolation of effects from high to lower doses is often not valid. In a third or more of instances in which a maximum tolerated dose elicited extra tumours in rodents, one-half that dose did not. Ames and others have pointed out that huge doses of non-genotoxic substances are accompanied by toxicity, cell death, and cell replacements. This creates conditions favourable for growth of tumours [...] Thus the linear extrapolation is not applicable to the majority of chemicals. Recently, short term experiments have measured the extent of DNA damage caused by different levels of doses of test substances. In one example, 11 chemicals known to cause cancer at high doses were administered at low levels. With 8 of 11 substances, the minimum amounts of damaged DNA were found not in controls but in the animals that received an amount intermediate between zero and a high dose. Instead of damaging the DNA of the rodents' livers, the low doses were apparently beneficial to them. In another study, female rats that had been administered 0.001 µg/kg of dioxin per day developed fewer breast, uterine, pituitary, and liver tumours and fewer overall tumours than did the controls.

When doses of 0.01 µg/kg per day were administered, the incidence of liver tumours exceeded that of the controls, but the number of breast, uterine, pituitary, and total tumours was markedly less than in the controls. In the above instances, safe (diminished cancer) levels of exposure exist for substances known to cause cancer at higher doses. The use of linear extrapolation from huge doses to zero implies that 'one molecule can cause cancer.' This assertion disregards the fact of natural large-scale repair of damaged DNA.'

This question was once again discussed at a symposium in Paris in 1998 [38]. It was pointed out that the LNT relationship assumes that: (1) the probability of a DNA lesion repair is constant whatever the dose and, hence, the number of other lesions provoked in the same cell and in the surrounding cells; (2) the probability for a damaged cell to evolve toward an invasive cancer is not influenced by the possible promotional effect of further irradiation or induced cell proliferation, nor the control exerted by surrounding cells. These assumptions were criticized. A single mutational event may evolve toward a clinical cancer, but in the absence of cell proliferation or promotion, this progression probably occurs very infrequently. The probability of the promotion and progression from a DNA lesion to a clinical cancer is likely to be strongly influenced by the presence of other lesions in the same cell or surrounding cells [17, 27]. Induced genetic instability probably plays a prominent role in radiation carcinogenesis [38]. Only initiated cells which are also immortalized and have a defect in the apoptosis mechanism can gradually accumulate other genomic lesions [17]. Moreover, the LNT hypothesis is not consistent with the impact of dose rate [24, 25, 26], which is observed even for irradiation with alpha-particles [41]. It has also been shown that some data cannot be fitted into a LNT hypothesis, such as the leukemia incidence in Hiroshima and Nagasaki [42] and osteosarcoma and hepatoma induced by contamination with alpha-emitter radionuclides in human beings or experimental animals [17, 43, 44, 45, 46, 47]. These high-dose studies are based on a high accuracy of the data and a follow-up of more than half a century; however, a fit is acceptable within a quadratic relationship. The BEIR IV report of the US National Academy of Sciences in 1988 [47] discussed the existence of a threshold or a quasi-threshold. Recently, the epidemiological data concerning alpha-irradiation have been supported by experimental data showing that the carcinogenic effect of one alpha-particle passing through a cell nucleus is so small that most of the carcinogenic effect must be due to several particles [48, 49]. The variation of natural background irradiation, which in some regions is as low as 1.5 mSv and in other regions as high as 100 mSv [50, 51, 52, 53, 54], does not seem to have any impact and does not suggest a LNT relationship. Indeed, most epidemiological and experimental data [55, 56, 57, 58, 59] support a quadratic relationship, which is also compatible with the excess of solid tumours observed in A-bomb survivors [23], particularly when the presence of a neutron component is taken into consideration [60, 61]. The possibility of an effect of low doses (10–20 mSv) on the fetus irradiated in utero [49, 62] deserves more investigation; it should, however, be recalled that the rapidly dividing immature cells of a fetus are not comparable to solid tissue but rather to bone marrow.

In view of all these data, it may seem surprising that so many specialists in radioprotection still adhere to the LNT hypothesis. The report of the OECD committee in 1998 [28] on radiation protection and public health summarizes the situation as follows: 'We have to note that some data reinforce the use of a LNT model and some clearly demonstrated the existence of a threshold [...]. The latest data from the analysis of Hiroshima and Nagasaki survivors [...] do not contradict the risk coefficient estimates used in ICRP 60 and do not suggest changes in the validity of the 'precautionary principle' which is the basis for the ICRP recommendations [...] however, it provides no improvement of our knowledge on the shape of the dose-effect relationship ... In view of the current status of knowledge and of the precautionary principle, the use of the LNT assumption is still justified when a unified approach must be applied to all sources and practices [...], however, this approach need not be automatically applied by experts to estimate risk in specific circumstances. In all cases, experts should use the best scientific information available concerning a given exposure situation. They may choose not to use the LNT assumptions ...' This cautious conclusion is instructive. Although most data and current models of carcinogenesis support a quadratic relationship or show evidence of a practical threshold, the LNT relationship is not rejected in radioprotection, because it does not underestimate the risk. This is why some radioprotection specialists are not willing to reject LNT formally until there is a clear demonstration of inadequacy in all situations. However, most of them, and this is a major step forward, no longer consider LNT as dogma. They are now willing to discuss other concepts and models in order to avoid, after the delivery of a very small dose to a large population, frightening and unfounded figures for predicted casualties which are estimated by the incautious use of LNT.

Natural and man-made radiation

As pointed out by Jaworowski [32], it was a judgement based on the LNT assumption that led an ad hoc Soviet government commission to evacuate and relocate more than 270,000 people from many areas of the former Soviet Union where the 1986–1995 average radiation doses from the Chernobyl fallout ranged between 6 and 60 mSv. By comparison, the world's average individual lifetime dose due to natural background radiation is about 160 mSv. In the Chernobyl-contaminated regions of the former Soviet Union, the lifetime dose is 210 mSv, while in some regions of the world it is about 1000 mSv. The forced evacuation of so many people from their – presumably – contaminated homes calls for ethical scru-

tiny. A comparison ought to be made between the psychological and medical burden of this measure (anxiety, psychosomatic diseases, depression and suicides) and the harm that may have been prevented. In this regard, a statement has been made by Gonzales, director of the Radioprotection Department at the International Atomic Energy Agency (IAEA) [63], who considered that doses below 10 mSv/year (total of natural and man-made irradiation) do not justify any action. He felt that between 10 and 100 mSv the condition should be analysed, but that action becomes mandatory only for doses higher than 100 mSv/year. This stance is of great interest, particularly because no distinction is introduced between natural and man-made radiation. This proposal may open the way for producing recommendations acceptable both from a scientific and public health point of view. It reconciles a precautionary approach with the assumption that doses lower than 10 mSv/year are without any significance for health and that it is unjustified to frighten people when they receive doses of this magnitude. Utilization of the LNT relationship, even for the smallest doses, is undoubtedly one of the factors which has greatly contributed to the anxiety of the general public and raised a controversy between, on the one hand, oncologists, radiobiologists and physicians and, on the other, engineers who work in radioprotection without any training in biology or medicine.

Life appeared and developed in a bath of ionizing radiation which was more intense a billion years ago than now. Man-made radiation represents only a small percentage of ionizing irradiation (10%–30% in industrialized countries). The main artificial source of irradiation is by far medical radiology. There is no country where the industrial use of radiation, and in particular nuclear energy (including uranium extraction, the release of radioactivity in the atmosphere or water, nuclear waste) results in doses higher than 0.01 mSv/year, whereas e.g. in France, natural irradiation varies from 1.5 to 6 mSv/year [49], throughout the world it varies from 1.5 to 30 mSv/year, and in a few inhabited regions may be as high as 100 mSv/year or more [50]. Thus, the additional irradiation due to nuclear

energy or nuclear waste represents less than 1/100 of natural irradiation, less than for a Parisian receiving additional radiation when spending a weekend at a winter resort (because of a high flow of cosmic rays) or at the seaside in Brittany (granite region). Yet, these minute doses provoke fears whereas, fortunately, no one takes into account supplementary irradiation during vacation. This inconsistency stresses the role of psychological factors.

In 1986, Richard Doll, using a LNT relationship, estimated that in the UK approximately 1265 cancer deaths were attributable to ionising radiation each year, 25 of them to man-made irradiation and 6 of the 25 to occupational exposure and discharges in the environment, which include all aspects of nuclear energy. In France, similar calculations have been made with the ICRP 60 cancer risk coefficient. It was also calculated that natural radiation could cause a maximum of 6000 and perhaps zero excess cancers, while for nuclear energy the corresponding figures were 25 and 0 (Table 10). In France, all the anxiety was focused on nuclear energy. This example shows that LNT has increased public aversion to radiation risks because by enabling the calculation of virtual risk, it has given credence to its existence. In parallel, optimization and cost-benefit analysis of virtual risk have introduced the idea that the protection of health in this field is severely limited by financial considerations. This led to the philosophy of zero risk. Whereas the lay person will accept the cancer caused by natural or medical irradiation (because their benefits are tangible), they may not accept the much smaller risks of nuclear energy when they do not appreciate its advantages.

Radiation risk assessment and psychological factors

The paramount importance of psychological and sociological factors in risk perception is well known. As early as 1957, a working party of WHO experts [64] emphasized the role of the rapid pace of change as contributing to the perception of the world as increasingly unstable and hazardous. It was said 'It would seem that societies have a certain threshold of tolerance for rates of changes, which if exceeded tend to social disintegration. [...] From the standpoint of mental health, irrational emotional states and unsatisfactory human relationships tend to be proportional to social disintegration.' The pace of change in our society over the past four decades may have provoked the crises and pessimism that we observe today. Moreover, in atomic energy the huge amount of energy released from matter is mysterious and raises 'dread of the unknown'. To transform matter into energy appears as a transgression, like the Greek myth of Prometheus who stole fire from the gods. Exaggerated apprehensions of radiation have been extensively studied since the advent of nuclear energy [65, 66, 67, 68]. Numerous examples have been given of discrepancies between the actual risk and the emotions raised. For example, according to Masse and Carde [69], nuclear energy incidents causing a dose excess of about

Table 10 Carcinogenic effects of ionizing radiation in France. Higher risk limits are calculated from ICRP data given a linear no threshold (LNT) hypothesis. The lower limit assumes that annual doses lower than 10 mSv have no carcinogenic effect. The potential reduction is calculated using data discussed in the symposium 'Les avancées de la radiologie médicale et ses contraintes' (in *Concours Médical*, suppl. 35, 1996)

Virtual risks due to ionizing radiation	Virtual cancer deaths
Natural irradiation: dose 2.5 mSv/year	0–6000
Potential reduction (houses with high concentration of radon)	0–600
Medical irradiation: dose 1 mSv / year	0–2500
Potential reduction (better techniques, modern equipment)	0–800
Nuclear energy and waste: dose 0.01 mSv/year	0–25
Potential reduction	≈0–10

1 μSv (corresponding to the excess of cosmic rays received due to living in Paris on the fourth floor of a building rather than on the first floor) is sufficient to arouse great anxiety, whereas dose increments one thousand times higher are ignored when they correspond to variations in natural irradiation or man-made irradiation not related to nuclear energy. In a poll carried out by the European Commission in 1990, artificial radioactivity was cited as one of the main sources of cancer, after tobacco but before alcohol, obesity, diet or sunbathing, whereas in fact it has very little impact, if any.

However, in order to understand fearful concerns on radiation, it should be placed in the perspective of the public's present attitude toward risk and technology [70, 71, 72, 73]. One cannot summarize in a few sentences the huge amount of literature published on this subject. The discrepancy between the risks as assessed by the experts and as perceived by the general public is particularly large for nuclear energy, but is by no means limited to that. It is observed in our society when attention is focused on small or hypothetical risks which are associated with new technologies. This may explain that while life has become safer and healthier, most individuals believe that the situation is getting worse rather than better. A similar phenomenon had been observed after the First World War and was analyzed in one of Freud's most celebrated books: *Das Unbehagen in der Kultur,* translated into English under the title *Civilization and Its Discontents* (1929). Freud pointed out that during prosperous periods the fear of death (Thanatos), which is always present in the subconscious, becomes more ominous because the struggle for life is less intense, which leaves more time for people to reflect on the meaning of life. Moreover, those who feel frustrated or victimized in the society tend to blame it for their unease. Therefore, they criticize the most obvious characteristic of contemporary society: its technology. In parallel, the myth of nature, which had been prevalent during the romantic period, is revisited, and the past is idealized. Since 1976, we as well as others [65, 66, 70, 74] have emphasised the role of the unconscious in the perception of risk. Currently, physical (ionizing radiation, microwaves, etc.), chemical (pesticides, synthetic products, etc.) and biological (genetically modified organisms, etc.) technologies are being stigmatized by being perceived as unnatural and entailing great risks.

Risk is a socially constructed concept. Perception of risk is complex and subjective; it is the result of a long process during which a representation of the technology and of its risks is constructed. This process is influenced by social, psychological and political factors. World views have a strong impact on risk judgement. When there are negative feelings, the risks are amplified, and the technology is judged as having high risks and low benefits. Providing information calling attention to the benefits favourably alters the evaluation of risks. Conversely, information about risks alters perceptions of benefits.

An important point is that education and social status are strongly correlated with risk judgement and attitude. There is a wide heterogeneity in perception with respect to the magnitude of risk and its acceptability. Public attitude varies greatly with social subgroups, and analysis of the data reveals that a wide range of social and cultural factors shapes risk perception. Gender is strongly related to risk judgement. Men tend to judge risks as smaller and less problematic than do women, even among scientists. Perceived risk is also inversely related to income and educational level, trust in institutions, in experts and in science. People who create, manage, control and benefit from technologies see less risk in the world. About one-third of white males perceive the risk to be quite low. This subgroup is characterized by a good educational and income level, and as having trust in institutions, science and experts [75].

Risks of ionizing radiation are greatly overestimated by those who do not believe in progress, who are reluctant to see economic growth or feel powerless in society. This paramount influence of subjective factors challenges the theory of rational behaviour [65, 66, 70, 75, 76]. However, it has been found difficult to integrate the technical and social aspects of risk [76, 77]. Cultural theorists have proposed that individuals select what and how much to fear as the product of a particular cultural bias [78, 79] and in order to support a given world view.

Social amplification or attenuation of risk may occur in several ways [76]. Mass media cover risks selectively [70, 76], emphasizing those that are more dramatic or to which the public is sensitized. When hazards are iteratively mentioned or overestimated, fears are built up. Anti-nuclear campaigning is a self-sustaining reaction. The campaign exploits fear and anxiety. Due to this anxiety any incident or accident occurring in a nuclear plant or any study, even if unreliable, suggesting that small doses of radiation are carcinogenic becomes a headline for the mass media. These rumours create the impression that nuclear energy is unsafe. This in turn enhances fears, and public opinion more readily trusts studies proclaiming that atomic energy is hazardous even if their scientific basis is unreliable. General scepticism, alleged unreliability of the official sources of information, and questioning of potential benefits are predominant factors in these reactions. When information accords with subconscious fears or expectations, it is readily believed, and the risk perception is amplified. To these well-known factors, Jaworowski [32] adds others:

- the psychological warfare during the Cold War that played on the public's fear of nuclear weapons,
- lobbying by fossil fuel industries,
- the interests of radiation researchers striving for recognition and budget,
- the interests of news media that profit by inducing public fear.

The representations of a technology and its risks are slowly established in the unconscious under these various influences, and thereafter they have a predominant influence on risk perception [80].

The role of representation is well illustrated by the comparison between ultraviolet and ionizing irradiation. The incidence of melanoma and the number of resulting deaths

are increasing each year in the northern EU countries. This increase is due to sunbathing and clothing styles. Sun exposure is particularly dangerous for infants and children. In most countries, information campaigns, those targeting mothers in particular, have failed to significantly alter the pattern of sun exposure even for young children (<5 years). Sun is associated with nature, holidays, the pleasant moments of life. A suntan during the 19th century was characteristic of farmers and poor peasants, while ladies protected themselves with parasols and gloves. The suntan became fashionable after the First World War, when it was a feature of the 'happy few' who enjoyed summer holidays on the beach and winter holidays at skiing resorts. In 'nature' camps, young children were exposed bare to the sun. After the Second World War, plane charters made it possible for many Europeans to holiday on the shores of the Mediterranean Sea. The incidence of melanoma started to increase rapidly and, despite the warnings, continues to increase.

Ultraviolet (UV) radiation also provides an illustration of the impact on public opinion of campaigns launched and supported by commercial rivalry. The Anglo-French Concord was the first commercial supersonic plane. Built in the late 1970s, its main aim was to link London and Paris with New York. Supersonic transports were accused of endangering stratospheric ozone with the water vapour discharged by their jet engines. Ozone depletion would then expose animal life to strong UV rays which would induce widespread skin cancer. A huge campaign against supersonic planes was launched in New York and New Jersey but was stopped 18 months later when it became obvious that the Concord was not a commercial success. Authorization to land in New York was granted amidst total public indifference [80].

Interestingly enough, most fears and actions have focused on ozone-depleting chemicals. But while the depletion of stratospheric ozone has slightly increased UV exposure in the Southern Hemisphere, it has had little, if any, significant effect in the Northern Hemisphere. Calculations showing the relatively small impact compared with individual behaviour were ignored. These observations confirm the strong tendency of the public to minimize individual responsibility and to put blame on others (society, the manufacturers of new chemicals). As stated by H. Tazieff [81]: 'The same shrewd if not exactly honest use has repeatedly been made of fears over the possible depletion of stratospheric ozone whenever powerful lobbies have sought to torpedo a project they opposed ... frightening the public is an easy task for anyone able to afford press and media campaigns.' A similar distortion of perception was induced for over two decades regarding lung cancer: underestimation of the effects of tobacco and overestimation of the effect of air pollution. This was greatly enhanced by tobacco manufacturers who spent millions of dollars to promote it.

Role of mass media in risk perception

Let us now consider the image of ionizing radiation and radioactivity. It has markedly evolved during the 20th century, but in a direction opposite to that of sun UV [80]. The first radiation-induced cancers were observed in physicists and physicians manipulating radium or x-rays between 1902 and 1906. Among the pioneers of x-rays and radioactivity, over 400 died from leukemia or skin cancers, a figure comparable to that of the excess of cancers in A-bomb survivors. From 1920 to 1938, the incidence of leukemia was ten times higher in radiologists than among other physicians. Nevertheless, radiation and radioactivity were felt to be a source of vigour. Radioactive water was sold at high prices, and mineral waters were advertised with proud proclamations of their high content of radioactive elements. In France, the most popular beauty cream in the 1930s, ThoRadia, boasted its enrichment in thorium and radium. The same was observed for a toothpaste in Germany.

Radioactivity was equally prestigious in the UK and the USA. Throughout the Western world it was associated with science, the fight against cancer, and Marie Curie (who died from a radio-induced leukemia in 1930). The first users of x-rays or radioactivity were not discouraged by the dangers. Just like mountain climbers, they knew the dangers but wanted to face the challenge.

Contrary to what is often claimed, an analysis of contemporary newspapers shows that the A-bomb explosions were hailed in August 1945 because they ended the war [65]. Radioactivity remained popular for several years because the prospect of abundant and inexpensive nuclear energy raised great hopes. These culminated in 1955 in the UN Conference on 'Atoms for Peace' in Geneva. In the French newspapers from this period, 'good' news (discovery of uranium ore, the inauguration of the first reactor) were on the front pages, while accidents were in small type on the back pages. The attitude toward radiation evolved slowly in the mid-1950s after the series of H-bomb tests by the Americans and the Russians [65]. At that time, the threats of the Cold War and the growing number of H-bombs on both sides of the Iron Curtain increased fears of a nuclear holocaust. But probably the main source of the change in attitudes during that period was the huge misinformation campaign launched by Stalin and his successors, which was most likely one of the biggest ever. The so-called 'Peace Movement' and its Stockholm resolution aimed at blocking the development of nuclear weapons in the Western world by fuelling fears about their biological effects. In the late 1960s, the fears expanded from weapons to peaceful uses and nuclear reactors. Nuclear energy became the symbol of high technology, big enterprise and dread and was chosen by environmentalists as their main target [65]. The Three Mile Island and Chernobyl accidents further increased anxiety because they associated nuclear energy with the uncontrollable, global catastrophe. On June 12, 1992, a prominent French environmentalist, Brice Lalonde, wrote an article in the newspaper *Le Monde* speaking about the beginning of the environmental movement in France in the 1970s: 'We had to have an enemy. We hesitated between the car (*la 'bagnole'*) and nuclear energy. We chose nuclear energy

because it was associated with the EDF (the French state-owned electricity producer) and the army.'

Recently, the role of psychological and cultural factors was illustrated by the controversies related to the incidence of leukemias in the vicinity of nuclear plants [80]. Since 1983 numerous papers have reported an increase in the incidence of leukemias around nuclear plants, especially in the UK: Sellafield, Dounreay, Aldermaston, Burghfield, and Hinkley Point. Each of these papers received important coverage in the mass media and aroused great emotion. However, it should be remembered that a statistical result is called significant when the risk of error is equal or inferior to 5% ($P<0.05$). This means that among 100 surveys, up to 5 will detect an effect which actually does not exist, the excess being due only to expected statistical variation. When surveys are carried out around nuclear plants, it is therefore to be expected that one survey out of 20 will give a spurious positive result ($P=0.05$). Indeed, in most cases, further investigations revealed statistical flaws (too short duration of the period studied, bias in the delineation of the geographical area, etc.) and have not confirmed initial claims. It is mostly around the Sellafield plant that there is a real cluster of leukemias. However, there are also clusters around some non-nuclear plants, and the current explanation is viral infections related to the migration of large numbers of workers [82]. Relation with irradiation has now been excluded by the COMARE studies [83]. Furthermore, surveys carried out around all of the several hundred nuclear plants in the UK (1994), the USA (1991), France (1995), Canada (1993) and Japan (1995) did not observe an increased incidence of cancers or leukemias in their vicinities [84, 85].

The incidence of leukemias in the vicinity of La Hague recently caused a controversy in France. La Hague (near Cherbourg in Normandy) is one of the two radioactive waste reprocessing plants in France. A survey carried out in 1989 did not reveal an increase in leukemias. Another one in 1995 reported a marginally significant increase in the incidence around the plant (1.6 expected and 4 observed, $P=0.06$) during the period 1978–1992. In 1997, a case-control study showing that the leukemia cases were associated with consumption of seafood and swimming on the beaches near the plant was published by the same authors [86]. Despite the statistical flaws in this article immediately pointed out by experts, the article received huge coverage in the media and incited great anxiety. It had economic consequences in the region for which the sale of seafood is an important resource. A scientific committee was appointed. Its interim report in July 1997 pointed out the statistical biases of the study [87]. In October 1997, an extension of the period studied from 1993–1996 revealed that no further cases of leukemia had been diagnosed around the plant [88]. Thus, the small excess for the period 1978–1996 is clearly not significant ($P=0.15$). This new result was barely mentioned in the press. Most mass media are not interested in reassuring news, possibly because it does not increase sales. Currently, despite the lack of any increase in leukemia incidence, the general public

in France remains convinced that La Hague has been the source of a leukemia epidemic.

A more dramatic example of the failure of the information system was given during the weeks that followed the Chernobyl accident in 1986. In this case, despite the small size of the dose received by the public in Central Europe (about 1.5 mSv, while the dose due to natural irradiation in 1 year varies between 1.5 and 6 mSv in the various regions of France and Europe), there were about 100,000 abortions, whereas the threshold dose for teratogenic effects is about 200 mSv.

These examples illustrate the great vulnerability of the public when risk is associated with a technology which is questioned or feared. Risk communication in these situations is difficult [70], but this does not mean that nothing can be done. The main points are to change the representation, to establish or re-establish trust, and to give evidence of the benefits. A risk can be accepted if it is associated with benefits for those who are affected. European studies [70] have shown that the risks are scored higher in Latin countries (France, Spain) than in Nordic or Anglo-Saxon countries, probably because the trust in the government's ability to control risks is lower.

With regard to radioprotection, the decrease in the dose limit (from 5 mSv to 1 mSv/year) has not been reassured and may even have increased anxiety because it revealed previous uncertainty with regard to risk and triggered controversies among scientists. Moreover, doses delivered are often expressed as a percentage of the limit dose, therefore the same dose caused by an incident became more frightening as it was now equal to half of the limit dose rather than to one tenth of it. Optimization and the principle of ALARA (as low as reasonably achievable) were well accepted by workers who saw the benefits, but they had a detrimental effect on public acceptance because it was perceived that radioprotection is controlled by economic considerations. These reactions to ionizing radiation further confirm that reluctance is actually not related to data but to mental models.

In summary, for the public, risk is much more than a rational assessment of danger. It includes the voluntary nature of the risk, catastrophic potential irreversibility, tolerability, the unknown, and uncontrollability. Acceptability is improved by benefits, reliability of institutions and sources of information. It is impaired by a lack of consensus among scientists. As we have seen, health risk perceptions are easily exploited and manipulated for commercial or political purposes. Conflicts and controversies surrounding the risks of a technology are not due to public irrationality or ignorance but instead to a series of complex psychological, social and cultural factors, and to mental models established over decades. Recently, Le Déaut from France [70], Slovic from the United States [75], and Jaworowski from Poland [32] have stressed the formation of powerful special interest groups well funded by a fearful public and sometimes by commercial rivalry. These groups are sophisticated in using their own experts and the media to communicate their concerns and their distrust to the public in order to

Table 11 Law of decreasing returns. Example: water purification (chemical or microbiological pollution). If the cost of the elimination of 90% of pollutants is taken to be equal to 1, the cost of eliminating a further 9% is about one hundred times more and that of eliminating a further 0.9% may be a thousand times higher. An additional life gain through the elimination of 99.9% instead of 99% assumes the existence of a LNT relationship, which is very hypothetical. Note the possible negative consequences of a cost increase above a certain limit: a decrease in water use for hygiene

	Measure of detriments avoided	Cost
Eliminate 90% of pollutant	90	1
Eliminate 99%	9	100
Eliminate 99.9%	≈0.9	10,000

Table 12 Costs for 1 year of life saved (taken from [90]). Real costs could be higher (probable overestimation of number of deaths due to low concentrations of toxic agents). In the USA, it was calculated that 60,000 lives could be saved through a better allocation of resources without any increase in expenditure (statistical deaths)

Prevention or cessation (alcohol, tobacco)	$1000–$4000
Medical care (USA):	
Cardiovascular diseases	$20,000
Cancers	$50,000
Prevention of traffic accidents	$40,000–$80,000
Toxic agents in environment (USA)	($150,000–$1 billion)

Fig. 3 Costs for 1 year of life saved: medicine vs toxin control

Fig. 4 Vicious circle according to [91]: scientific uncertainty [whether it is an agent whose effect is uncertain (BSE) or a virtual risk calculated for low doses of a carcinogenic agent] gives rise to public anxiety and an overestimation of risk by the experts who want to be cautious. This in turn increases public fears, which leads to stricter regulations through pressure from politicians

influence debates and decisions. Trust has a fundamental role to play in risk perception, but it is fragile and easier to destroy than to create [70].

Finally, an ultimate problem must be mentioned: the unbalanced allocation of budget due to the weight of public opinion and lobbying. Abelson [89] raised this problem in an editorial published in *Science* in 1990. He wrote: 'Stringent regulation and frightening publicity have led to public anxiety and chemophobia ... the cost of cleaning up phantom hazards will be in the hundreds of billions of dollars with minimal benefit to human health. In the meantime real hazards are not receiving adequate attention.' This means that the governments of industrialized countries are spending huge amounts of money for a very limited health benefit [90]. The amount of money which is spent to save a life varies enormously and is markedly increased when a zero loss is sought (Table 11). It is much lower in preventive or curative medicine (Table 12, Fig. 3).

Since that time the problem has become even more serious, as pointed out by Breyer [91], who considered the anxiety of the public a vicious circle ([91], Fig. 4). The amount of money that a nation can devote to health protection and prevention is necessarily limited [90]. Choices must be made, and they should rely on the assessment of the cost and benefit of any action [90, 91, 92].

However, public reactions are not irrational, and they should not be overlooked: they should be investigated and understood. The role of the scientific communit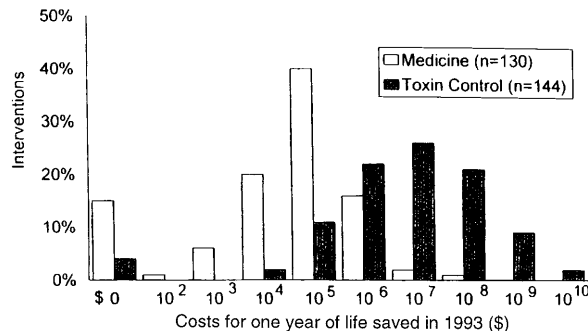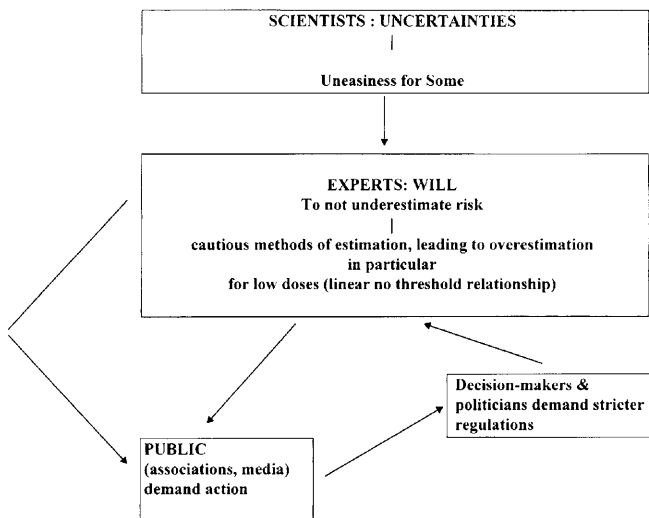y is to state what is known and what is uncertain, to distinguish actual and virtual risks, and to correct biased information. When information is in accordance with subconscious fears or expectations, it is readily believed, and the risk perception is amplified [95, 96].

References

1. Institut Européen de Cindyniques (1996) Risque, santé, environnement: la place de l'outil économique dans les transactions sociales. Cahiers du Groupe Epistémiologie des Cindyniques 3: 5–97
2. Haut Comité Santé Publique (1998) Rapport sur la santé en France. La documentation Francaise, Paris
3. Tubiana M (1998) Histoire de la pensée médicale – les chemins d'esculape. Flammarion Edit, Paris
4. Doll R, Peto R (1981) The causes of cancer – quantitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst 66: 1191–1308
5. Hill C, Doyon F, Sancho-Garnier H (1997) L'épidémiologie des cancers. Flammarion Edit, Paris
6. International Agency for Research on Cancer (1997) Cancer research for cancer control. (Harvard report on cancer prevention) Harvard, p 12

7. Bhatia R, Lopipero P, Smith A (1998) Diesel exhaust exposure and lung cancer. Epidemiology 9: 84–91
8. Hackshaw AK, Law MR, Wald NJ (1997) The accumulated evidence on lung cancer and environmental tobacco smoke. Br Med J 315: 980–988
9. Trédaniel J (1997) Tabagisme passif et risque cancérogène pour les poumons. Bull Acad Med 181: 736–742
10. Académie des Sciences (1999) Pollution atmosphérique due aux transports et santé publique. (Rapport no. 12) Académie des Sciences, Paris
11. Ames BN, Gold LS (1990) Chemical carcinogenesis: too many rodent carcinogens. Proc Natl Acad Sci USA 87: 7772–7776
12. Ames BN, Gold LS, Willett WC (1995) The causes and prevention of cancer. Proc Natl Acad Sci USA 92: 5258–5265
13. Ames BN, Gold LS (1997) Environmental pollution, pesticides and the prevention of cancer: misconceptions. FASEB J 11: 1041–1052
14. Muir CS (1990) Epidemiology, basic science and the prevention of cancer. Implication for the future. Cancer Res 50: 6441–6448
15. Doll R, Peto R (1978) Cigarette smoking and bronchial carcinoma: dose and time relationships among regular smokers and lifelong non-smokers. J Epidemiol Community Health 32: 303–313
16. Halpern MT, Gillespie BW, Warner KE (1993) Patterns of absolute risk of lung cancer mortality in former smokers. J Natl Cancer Inst 85: 457–464
17. Tubiana M (1999) Contribution of human data to the analysis of human carcinogenesis. C R Acad Sci [III] 322: 215–228
18. Cohen SM, Ellen LB (1990) Cell proliferation in carcinogenesis. Science 249: 503–504
19. Tubiana M, Lafuma J, Masse R, Latarjet R (1991) The assessment of the carcinogenic effect of low dose radiation. In: Gerber GB, et al (eds) The future of human radiation research. Br Inst Rad Report 22: 109–119
20. Adams J (1998) A Richter scale for risk? The scientific management of uncertainty versus the management of scientific uncertainty. In: Vitale M (ed) Science and technology awareness in Europe: new insights. European Communities, Brussels, pp 93–111
21. Ron E, Lubin JH, Shore RE, Mabuchi K, Modan B, Pottern LM, Schneider AB, Tucker MA, Boice JD Jr (1995) Thyroid cancer after exposure to external irradiation: a pooled analysis of seven studies. Radiat Res 141: 259–277
22. Hancock SL, Tucker MA, Hoppe RT (1993) Breast cancer after treatment of Hodgkin's disease. J Natl Cancer Inst 85: 25–31
23. Heidenreich WF, Paretzke HG, Jacob P (1997) No evidence for increased tumor rates below 200 mSv in atomic bomb survivors. Radiat Environ Biophys 36: 205–207
24. Boice JD Jr, Blettner M, Kleinerman RA, Stovall M, Moloney WC, Engholm G, Austin DF, Bosch A, Cockfair DL, Krementz ET (1987) Radiation dose and leukemia risk in patients treated for cancer of the cervix. J Natl Cancer Inst 79: 1295–1311
25. Académie des Sciences (1989) Risque des rayonnements ionisants et normes de radioprotection. (Report no. 23, 72) Académie des Sciences, Paris
26. Académie des Sciences (1997) Problèmes liés aux faibles doses des rayonnements ionisants (Problems associated with the effects of low doses of ionizing radiation). (Report no. 834) Académie des Sciences, Paris
27. Tubiana M (1 999) Les effets cancérogènes des faibles doses de radiations. Cancer Radiothér 3: 203–214
28. OECD (1998) Report on developments in radiation health science and technology and their impact on radiation protection. Nuclear Energy Agency Committee on Radiation Protection and Public Health. OECD, Paris
29. Infante PF (1991) Prevention versus chemophobia: a defence of rodent carcinogenicity tests. Lancet 337: 538–540
30. Weinstein IB (1991) Mitogenesis is only one factor in carcinogenesis. Science 251: 387–388
31. Pochin EE (1987) Radiation risks in perspective. Br J Radiol 60: 42–50
32. Jaworowski Z (1999) Radiation risk and ethics. Physics Today 52: 24–29
33. Jaworowski Z (1998) Radiation risks in the 20th century: reality, illusions and ethics. Exec Intell Rev 25: 15–19
34. Bond VP, Wielopolski L, Shani G (1996) Current misinterpretation of the linear no threshold hypothesis. Health Phys 70: 877–882
35. Latarjet R, Tubiana M (1989) The risks of induced carcinogenesis after irradiation at small doses. The uncertainties which remain after the 1988 UNSCEAR report. Int J Radiat Oncol 17: 237–240
36. Rossi HH, Zaider M (1997) Radiogenic lung cancer. The effect of low doses of low LET radiation. Radiat Environ Biophys 36: 85–88
37. Rossi HH (1997) It is time for change. Health Phys Soc Newsl, pp 8–9
38. Gros F (1999) Carcinogenic risks due to ionising radiation. Life Sciences. C R Acad Sci [III] 322: 81–256
39. Tubiana M (1992) The carcinogenic effect of exposure to low doses of carcinogens. Br J Industr Med 49: 601–605
40. Abelson PH (1994) Risk assessment of low level exposure. Science 265: 1507
41. Morlier JP, Morin M, Chameaud J, Masse R, Bottard S, Lafuma J (1992) Importance du rôle du débit de dose sur l'apparition des cancers pulmonaires chez le rat après inhalation de radon. C R Acad Sci [III] 315: 463–466
42. Little MP, Muirhead CR (1996) Evidence for curvilinearity in the cancer incidence dose response in the Japanese atomic bomb survivors. Int J Radiat Biol 70: 83–94
43. Raabe OG (1984) Comparison of the carcinogenicity of radium and boneseeking actinides. Health Phys 46: 1241–1248
44. Raabe OG, Book SA, Parks NJ (1980) Bone cancer from radium. Canine dose-response explains data for mice and humans. Science 208: 61–64
45. Rowland RE, Stehney AF, Lucas HF (1983) Dose-response relationships for radium-induced bone sarcomas. Health Phys 44 [Suppl 1]: 15–31
46. Van Kaick G, Wesch H, Luehrs H, Liebermann D, Kaul A (1991) Neoplastic diseases induced by chronic alpha irradiation. Epidemiological, biophysical and clinical results by the German Thorotrast study group. J Radiat Res 32 [Suppl 2]: 20–33
47. BEIR IV (1988) Health risks of radon and other internally deposited alpha-emitters. Committee on the Biological Effects of Ionizing Radiations (BEIR). National Research Council, National Academy of Sciences, Washington DC
48. Miller RC, Randers-Pehrson G, Geard CR, Hall EJ, Brenner DJ (1999) The oncogenic transforming potential of single alpha particles through mammalian cell nuclei. Proc Natl Acad Sci USA 96: 19–22
49. Thé G de, Aurengo A, Legrain M, Masse R, Tubiana M (1999) Energie nucléaire et santé. Bull Acad Natl Med (Paris) 183: 1233–1246
50. Wei C, Sugahara T, Tao E (eds) (1997) High levels of natural radiation. Radiation dose and health effects. (International congress series 1136) Excerpta Medica, Elsevier, Amsterdam
51. Ye W, Sobue T, Lee VS, Tanooka H, Mifune M, Suyama A, Koga T, Morishima H, Kondo S (1998) Mortality and cancer incidence in Misasa, Japan, a spa area with elevated radon levels. Jpn J Cancer Res 89: 789–796
52. Wei L, Zha Y, Tao Z, He W, Chen D, Yaan Y (1990) Epidemiological investigation of radiological effects in high background radiation areas of Yangjiang, China. J Radiat Res 31: 119–136
53. Kesevan PC (1997) Indian research on high levels of natural radiation: pertinent observations for further studies on high levels of natural radiation. In: Wei L, Sugahara T, Tao Z (eds) High levels of natural radiation 1996. Elsevier, Amsterdam, pp 111–117
54. Nair MK, Amma NS, Gangadharan P, Padmanabhan V, Jayalekshmi P, Jayadevan S (1997) Epidemiological study of

cancer in the high background radiation area in Kerala. In: Wei L, Sugahara T, Tao Z (eds) High levels of natural radiation 1996. Elsevier, Amsterdam, pp 271–276

55. Thomas RG (1994) The US radium luminisers: a case for policy of below regulatory concern. J Radiol Protect 14: 141–153

56. Cohen BL (1995) The test of linear no threshold relationship theory of radiation carcinogenesis for inhaled radon decay products. Health Phys 68: 157–174

57. Sanders I, Lauhala KE, McDonald KE (1993) Life span studies in rats exposed to $^{239}PuO_2$. Survival and lung tumors. Int J Radiat Biol 64: 417–430

58. Tanooka H, Ootsuyama A (1993) Threshold-like dose response of mouse skin cancer induction for repeated β irradiation and its relevance to radiation-induced human skin cancer. Recent Results Cancer Res 128: 231–241

59. Tubiana M (1998) The report of the French Academy of Science: problems associated with the effects of low doses of ionizing radiation. J Radiol Protect 18: 243–248

60. Kellerer AM, Nekolla E (1997) Neutrons versus y-ray risk estimates: inferences from the cancer incidence and mortality data in Hiroshima. Radiat Environ Biophys 36: 73–83

61. Kellerer AM (1999) The effects of neutrons in Hiroshima – implications for the risk estimate. CR Acad Sci [III] 322: 229–237

62. Doll R (1997) Effects of small doses of ionizing radiation on human health. Nucl Energy 36: 435–441

63. Gonzales A (1999) Regulation of low level radiation. C R Acad Sci [III] 322: 241–243

64. WHO (1957) Questions de santé mentale, posées par l'utilisation de l'énergie nucléaire à des fins pacifiques. (Technical report no. 151) World Health Organization, Geneva

65. Tubiana M, Pelicier Y (eds) (1977) Colloque (symposium) 'Les implications psycho-sociologiques du développement de l'énergie nucléaire'. Socité Française de Radioprotection, Paris

66. Tubiana M, Pelicier Y (eds) (1997) Colloque (symposium) 'Atom et Societé'. Centre Antoine Béclère, Paris

67. Dupont RL (1980) Nuclear phobia: phobic thinking about nuclear power. The Media Institute, Washington DC

68. Peters E, Slovic P (1996) The role of affect and world views as orienting disposition in the perception ad acceptance of nuclear power. J Appl Social Psychol 26: 1427

69. Masse R, Carde C (1997) La contrivers des faibles doses. In: Tubiana M, Pelicier Y (eds) Colloque 'Atome et Societé'. Centre Antoine Béclère, Paris, pp 77–82

70. Tubiana M, Vrousos C, Carde C, Pagès JP (1999) Colloque (symposium) 'Risque et Societé'. Nucléon, Gif sur Yvette, France

71. The Royal Society (1992) Risk: analysis, perception and management. Study report. The Royal Society, London

72. Adams J (1995) Risk. UCL Press, London

73. Kunreuth H, Slovic P (eds) (1996) Challenges in risk assessment and risk management. Ann Am Acad Polit Social Sci [special issue]

74. Fritzsche AF (1995) The role of the unconscious in the perception of risks. In: Risk health, safety and environment, pp 215–240

75. Slovic P (1999) Trust-emotion, sex, politics and science. In: Tubiana M, Vrousos C, Carde C, Pagès JP (eds) Colloque 'Risque et Société'. Nucléon, Gif sur Yvette, pp 85–110

76. Kasperson RE (1999) The social attenuation and amplification of risk. In: Tubiana M, Vrousos C, Carde C, Pagès JP (eds) Colloque 'Risque et Société'. Nucléon, Gif sur Yvette, pp 111–122

77. Stem P, Fineberg H (1996) Understanding risk: informing decisions in a democratic society. National Academy of Sciences, Washington DC

78. Douglas M, Widlasky A (1982) Risk and culture – an essay on the selection of technological and environmental dangers. University of California Press, Berkeley

79. Douglas M (1985) Risk acceptability according to social science. Russell Sage Foundation, New York

80. Tubiana M (1998) Health risks data and perceptions. In: Vitale M (ed) Science and technology awareness in Europe: new insights. European Communities, Brussels, pp 113–123

81. Tazieff H (1992) CFCS: an imaginary danger. Projections (science and environment), Paris, special issue on the Heidelberg appeal, 7: 61–67

82. Kinlen LJ (1995) Epidemiological evidence for an infective basis in childhood leukaemia. Br J Cancer 71: 1–5

83. COMARE (1996).The incidence of cancer and leukaemia in young people in the vicinity of the Sellafield site, West Cumbria: further studies and an update of the situation since the publication of the report of the Black Advisory Group in 1984. Fourth report. Committee on Medical Aspects of Radiation in the Environment

84. Hatchouel JM, Laplanche A, Hill C (1 995) Leukaemia mortality around French nuclear sites. Br J Cancer 71: 651–653

85. Jablon S, Hrubec Z, Boice JD (1991) Cancer in population living near nuclear facilities. A survey of mortality nationwide and incidence in two states. JAMA 265: 1403–1408

86. Pobel D, Viel JF (1997) Case-control study of leukaemia among young people near La Hague nuclear reprocessing plant: the environmental hypothesis revisited. Br Med J 314: 101–106

87. Spira A, Oton O (1999) Rayonnement ionisant et santé. La Documentation Française, Paris

88. Guizard AV, Spira A, Troussard X, Collignon A (1997) Incidence of leukemias in people aged 0 to 24 in north Cotentin. Rev Epidemiol Santé Publ 45: 530–535

89. Abelson PA (1990) Testing for carcinogens with rodents. Science 249: 358

90. Frantzen C, Birraux C, Graham J, Francis RT, Gail de Planque E, Chevrillon H (1996) Risques: choix politiques, évaluations économiques et sociales. Ann Mines (Paris) 96: 31–69

91. Breyer S (1993) Breaking the vicious circle: towards effective risk regulation. Harvard University Press, Cambridge, Mass.

92. Arrow KJ, Cropper ML, Eads GC, Hahn RW, Lave LB, Noll RG, Portney PR, Russell M, Schmalensee R, Smith VK (1996) Is there a role for benefit-cost analysis in environmental, health and safety regulations. Science 272: 211–222

93. Health Physics Society (1996) Radiation risk in perspective. Position statement. In: Mossman K, Godman M, Masse F, Mills WA, Schiager K, Vetter RJ (eds) Health Phys Soc Newsl 24: 3

94. Wingspread Conference (1998) Exposures below 10 rem are no danger. Nucl News 50–51

95. Taylor LS (1980) Some non-scientific influences on radiation protection standards and practice. Health Phys 39: 851–874

96. Walinder G (1996) Has radiation protection become a health hazard? Kärnkraftsäkerhet & Utbildning, Nyköping, Sweden

97. Huttunen JK (1997) Health promotion and its wider dimension. In: Les nouvelles stratégies de prévention en santé publique (Rome). Europe Blanche XIX-Institut des Sciences de la Santé, Paris

98. Tubiana M, Tredaniel J, Thomas D, Kaminsky M (1997) Rapport sur le tabagisme passif. Bull Acad Natl Med 181: 727–766

99. Garfinkel L (1981) Time trends in lung cancer mortality among non-smokers and a note on passive smoking. J Natl Cancer Inst 66: 1061–1066

100. Peto R (1981) Why cancer? Times Health Suppl (Lond), pp 12–14

101. Thompson DE, Mabuchi K, Ron E, Soda M, Tokunaga M, Ochikubo S, Sugimoto S, Ikeda T, Terasaki M, Izumi S, Preston DL (1994) Cancer incidence in atomic bomb survivors. Part II. Solid tumors 1958–1987. Radiat Res 137: S17–S67

102. Bhatia S, Robison LL, Oberlin O, Greenberg M, Bunin G, Fossati-Bellani F, Meadows AT (1996) Breast cancer and other second neoplasms after childhood Hodgkin's disease. N Engl J Med 334: 745–751