低線量電離放射線の影響に関する諸問題

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Problèmes liés aux effets des faibles doses de radiations ionisantes

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Conclusions

1   RESEARCH PROGRESS FROM 1990 TO 1994

Since the publication of the ICRP recommendations in 1990, fundamental research in molecular genetics and cellular biology has progressed extensively [60].

Fundamental data

a) The linear extrapolation without threshold is based on the assumption that radiocarcinogenesis is a random process in which one event occuring in a single cell is sufficient to induce cancer; from such an hypothesis, reducing the dose should reduce the number of events without modifying either their nature or their severity. Consequently, any dose, even the lowest, should be associated with a risk proportional to the dose. However, work conducted since 1989, particularly in molecular biology, provides evidences against this view.

b) Molecular events, induced by high doses delivered at high dose rates, are quantitatively different from those induced by low doses at low dose rates. Simple extrapolation is therefore not justified. In addition, generally, the influence of dose rate, insufficiently taken in to account up to now, is of prime importance, even at low doses.

c) All cells contain highly sophisticated mechanisms which allow continuous repair of DNA damaged by radiation, chemicals, as well as those resulting from oxidative reactions of normal metabolism. Almost forty enzymes are known to be involved in these DNA repair, while some are « constitutive », others are « inducible » in response to DNA lesions they have to repair. These latter ones, which appear at high dose rates, are a major source of error-prone repair and thus of potential carcinogenic mutations.

At low dose rates, repair is effective because there is sufficient enzymes present to repair a relatively small number of lesions. At high dose rates, however, repair is less effective, because the same amount of enzymes must face rapidly a large number of lesions within a short time interval.

d) Another phenomenon, linked to cell death, appears for doses higher than 1000 mSv. Non-dividing cells repair lesions more efficiently than those undergoing division. At doses above 1 000 mSv, cell death initiates in surviving cells the enter into their division cycle.

e) The mechanisms involved in carcinogenesis are highly complex and include: multiple genomic changes, disturbance of cell-cell interactions and loss of efficiency of the organism's immune response. These phenomena differ according to the nature of the cancer, as for example in leukemia versus solid tumor.

The use of an exclusive linear relationship without threshold is therefore a simplification, open to criticism.

f) In animals experiments, depending of the type of cancer, at different dose levels, practical thresholds are observed, below which the risk of radiocarcinogenesis is negligible if not zero. A dose rate effect is observed on cancer induction, even at low doses. In this regard it is noteworthy that no induction of human cancer has ever been observed for doses lower than 200 mSv.

g) Recent developing work has shown that pretreatment with low doses of radiation can reduce the harmful effects of high doses received later.

Epidemiological data

a) No epidemiological survey (including those on the survivors of Hiroshima and Nagasaki) revealed a carcinogenic effect for doses lower than 200 mSv. This is particularly the case for studies on cancer rate in areas where natural radiation (including radon) is high. Therefore, the potential effect must be very weak. At low dose rates, excess of leukemias is observed only for doses higher than 400 mSv and excess of solid tumors only for doses higher than 1000 mSv.

b) Preferential reference to the results from Hiroshima and Nagasaki is therefore questionable considering the high dose rate at which exposure was delivered.

The effects observed at high doses, in different studies, are generally lower in the adult than those predicted by risk coefficients actually proposed and calculated from Hiroshima and Nagasaki data. This, may be due to a reduction of the carcinogenic effect associated with fractionation of the dose or low dose rate.

c) Concerning the basic principles adopted by ICRP in 1990 to revise the carcinogenic risk coefficient, the following remarks can be made:

1) The use of a non threshold linear relation deserves discussion. Several epidemiological data, especially that concerning α-radiation, suggests the existence of a practical threshold, that is due either to repair to damaged DNA or to a latency period longer than the average lifespan.

Furthermore, the epidemiological data suggest that it is inappropriate to use a single reduction coefficient for the dose and the dose rate because a dose rate effect is observed, even for relatively low doses [61].

2) Concerning the delayed excess of cancer deaths at Hiroshima and Nagasaki, it is noteworthy that it is cancers of the digestive tract in particular. In studies made on patients treated by irradiation for Ankylosing Spondylitis such an

increase is not seen. This difference may result from the fact that the radiation at Hiroshima and Nagasaki was delivered at very high dose rates or because of particular features, especially nutritional, in the Japanese population.

3) The dose reestimates, particularly for neutrons, for Hiroshima and Nagasaki are controversial. The neutron dose at Hiroshima is now considered to have been higher than was estimated in 1986. Thus, the reevaluation of the carcinogenic effect is questionable given the reevaluation of dosimetry.

4) Higher sensitivity of young people to radiation effects is obvious but it is reduced when the age at exposure increases.

5) The use of a multiplicative model with a constant coefficient to predict the excess of malignant tumors during life expectancy is open to criticism. In fact, new data, particularly that concerning young subjects (Hiroshima and Nagasaki survivors and children treated by radiotherapy), show a rapid reduction in the relative excess after a time period greater than 15 years. This reduction is also observed in irradiated adult subjects.

2   RECOMMENDATIONS

In 1989, the Academy concluded its report on the same topic as follows :

« *In conclusion*, it can be considered, *that the present standards appear already well set, that there is no scientific basis to change them and that, in a few years, the data on low dose effects should be completed, ... »*

The Academy had emphasized however, the necessity of not exceeding a total dose of 1000 mSv during occupational lifetime. This dose should be distributed homogeneously over time. The accumulated dose should be monitored at regular intervals of about ten years.

Consequently, to answer the question submitted in 1994, the Academy has performed an extensive analysis on numerous and informative studies, particularly in molecular genetics and epidemiology, published from 1989 through 1994. From this analysis, and considering only the scientific aspect of the question, it can be concluded that:

1) There is no scientific data, which serves as evidence in favour of lowering the dose for the members of the public in France to 1mSv/year.

2) There is no recent, indisputable scientific data which support lowering the current standards for workers in France. Therefore the conclusions of the 1989 report remain unchanged, particularly that concerning the life-time dose of 1000 mSv which implies monitoring every ten years to ensure that the rhythm of acquisition respects the objective.

3) The new contributions from molecular biology lead to the concept that the process of induction of potentially carcinogenic persistent genomic lesions, is significantly different at low or high doses and likewise at low and high dose rates. The differences are mainly due to DNA-lesion repair mechanisms which are not similar in the two situations. Recent epidemiological data are in complete agreement with this conclusion.

4) Medical examinations are the second largest source of exposure of populations, after natural exposure, way before other sources. Thus, to decrease significantly the radiation doses of the population, effort should be directed toward reducing the doses received during radiological examinations, especially among the young.

5) There are good reasons for encouraging current and future research on the mechanisms of biological effects of ionizing radiation, on the development of sensitive methods to detect the effects of radiation on the human genome and on the epidemiology of low doses of radiation.

6) A small minority within the group, while in general agreement with point (1) of the recommendations, although lowering of the maximum doses for the members of the public in the new ICRP proposals are difficult for them to accept, also, consider it undesirable that France distinguishes itself from the positions of other countries.

Concerning the standards for workers, this small minority of members disagreed with the point (2) of the recommendations. Although they approved the proposal of a life-time-dose of 1000 mSv, they considered it better to accept individual follow-up, including moving people to other work places, to ensure that the dose received over five years does not exceed 100 mSv.

For these two reasons, some members of this minority consider it better to accept (as a protective measure) all the maximum doses proposed by the ICRP (for the public and workers) but acknowledge the possibility of revising upwards the standards proposed for the public in line with future developments in radiobiology and radiotherapy.

Finally, again among this small minority, others advocate that only the ICRP proposals concerning workers should be taken into account and therefore support the recommendations of the majority concerning point (1).

Dose limits expressed in millisieverts per year

	Workers	Members of the public[1]*
Current regulations (CIPR 26)	50	5
ICRP 60	100 over 5 years (i.e on the average 20 per year provided not exceeding 50 per year)	1
Proposals from the French Academy of sciences	50**	5

– For workers it is recommended that the cumulated dose should not exceed 1 000 mSv over the occupational lifetime, and that the annual dose should never exceed 50 mSv.

Remarks: In ICRP 26, the estimated detriment is 1.25×10^{-2} per Sievert.

In ICRP 60 the estimated detriment is 5.6×10^{-2} per Sievert for workers and 7.3×10^{-2} for members of the public.

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* In addition to the dose from medical exposure (average 1 mSv/year) and to the dose received from natural exposure (2.5 mSv/year on average but varies in France between 1.5 and 6mSv/year, depending on the region).