Commission of the European Communities

radiation protection

Feasibility of studies on health effects in western Europe due to the reactor accident at Chernobyl and Recommendations for research

Post-Chernobyl action report

Report

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radiation protection

Feasibility of studies on health effects in western Europe due to the reactor accident at Chernobyl

Report of a Task Group for the CEC

J. Breckow, A.M. Kellerer, E.G. Knox, S. Richardson

Recommendations for research of an international panel of independent experts

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Preface

In December 1988 the Head of the Radiation Protection Programme established an International Panel of Independent Experts in order to draft recommendations on the feasibility of carrying out studies on the health effects from environmental exposure to ionizing radiation as a consequence of the Chernobyl nuclear reactor accident that took place in April 1986. This panel consisted of the following individuals:

Sir Richard Doll	Imperial Cancer Research Fund
(Chairman)	Cancer Epidemiology and Clinical Trials Unit
	Oxford University, UK
Dr. J.D. Boice	Chief of Radiation Epidemiology Branch
	National Cancer Institute
	Bethesda, MD, USA
Dr. J. Estève	Unit of Biostatistics Res. and Information
	International Agency for Research on Cancer
	Lyon, France
Prof. G. Silini	Secretary of UNSCEAR (retired)
	Lovere
	Bergamo, Italy
Dr. J.W. Thiessen	Vice Chairman
	Radiation Effects Research Foundation
	Hiroshima-Nagasaki, Japan

The Panel met in Brussels on 27 and 28 February 1989. A Task Group, also established by the Head of the Radiation Protection Programme prior to that meeting, had prepared a draft report entitled "Feasibility of Studies on Health Effects in Western Europe due to the Reactor Accident at Chernobyl". This Task Group consisted of Prof. A.M. Kellerer and Dr. J. Breckow, University of Würzburg; Prof. E.G. Knox, University of Birmingham; and Dr. S. Richardson, INSERM, Villejuif. At the February meeting the Panel met the Task Group to discuss this draft report, amend it where necessary and draft conclusions and recommendations with respect to the feasibility of health effect studies. The following section of this report contains the recommendations of the Panel, to which is attached the final version of the Task Group report. Together they represent the consensus of both the Panel and the Task Group.

The reactor accident at Chernobyl in 1986 caused widespread concern throughout the population of the European Community because of the potential radiological consequences. The accident also drew attention to some gaps in our knowledge of the behaviour and health hazards of radioactive contamination in the environment and highlighted the need to improve the preparedness to deal with a large-scale nuclear accident. As a consequence, the Commission of the European Communities proposed a revision of the Radiation Protection Programme 1985-1989 (COM(87)332 Final) with the specific aim of initiating additional radiological research to cover the lacunas in knowledge revealed by the Chernobyl accident. The revision of the Radiation Protection Porgramme was approved by the Council on 21 December 1987 with a budget of 10 Mio.ECU (O.J. No. L 16/44 of 21.1.88). The revision outlined 10 specific lines of research:

1. evaluation of the reliability and meaningfulness of long distance atmospheric transfer models;
2. evaluation of data on the transfer of radionuclides in the food chain;
3. feasibility of epidemiological studies on health effects in the population;
4. radiological aspects of nuclear accident scenarios;
5. underlying data for derived emergency levels;
6. improvement of practical countermeasures with respect to the agricultural and aquatic environment;
7. improvement of practical countermeasures with respect to the urban environment;
8. improvement of practical countermeasures with respect to preventive medication;
9. monitoring and surveillance in accidental situations;
10. treatment methodologies of exposed persons.

This report responds to the Post-Chernobyl research action number 3 in considering what health effects might be caused by the radioactive contamination of Member States by the accident at Chernobyl and whether the increased incidences of any anticipated health effects would be amenable to further study. The feasibility study was undertaken by scientists from three institutes with an established reputation in the fields of radiation effects and epidemiology, namely,
Prof. A.M. Kellerer and Dr. J. Breckow from the Institut für Medizinische Strahlenkunde of the University of Würzburg, F.R.G., Prof. E.G. Knox from the Department of Social Medicine of the

University of Birmingham, U.K. and Dr. S. Richardson of the Institut National de la Santé et de la Recherche Médicale in Paris, France. The study was based on a review of the radiation exposures accumulated by the public in different Member States as a result of the radioactive contamination from Chernobyl, a consideration of the possible health effects which might arise from the radiation, an estimation of the incidences using current risk coefficients for different sections of the public, and a calculation of the probability that an epidemiological study might reveal the radiation induced health effects in the background of normal human disease.

The feasibility study has been reviewed by an international panel of distinguished scientists, namely Sir Richard Doll (Chairman) from the Imperial Cancer Research Fund Cancer Epidemiology and Clinical Trials Unit of the University of Oxford, U.K., Dr. J.W. Thiessen (Secretary), Vice Chairman of the Radiation Effects Research Foundation in Hiroshima and Nagasaki, Japan, Dr. J.D. Boice from the Radiation Epidemiology Branch of the National Cancer Institute in Bethesda, U.S.A., Dr. J. Estève from the Unit of Biostatistics Research and Information of the International Agency for Research on Cancer in Lyon, France and Prof. G. Silini (former Secretary of UNSCEAR) from Lovere, Bergamo, Italy. The recommendations of this panel of experts, which provide a concise summary of the feasibility study are presented first in this report.

The feasibility study has been purposely written for the educated layman and should appeal to all those interested in the potential health effects from the reactor accident at Chernobyl, as well as those scientists and medical experts who are professionally involved in research and regulation in radiological protection.

RECOMMENDATIONS
OF AN INTERNATIONAL PANEL OF INDEPENDENT EXPERTS

on

THE FEASIBILITY OF STUDIES ON HEALTH EFFECTS IN WESTERN EUROPE DUE TO THE REACTOR ACCIDENT AT CHERNOBYL

Introduction

The Panel has considered whether studies of health effects potentially related to environmental releases from the Chernobyl reactor accident would be useful. In doing so, it has evaluated the exposure patterns and the dose levels within the European Community, the different health effects that might be induced by such doses, and the likelihood that epidemiological studies could produce scientifically useful information. In the following, the Panel indicates the conclusions of these evaluations and recommends a course of action that it considers to be prudent and scientifically reasonable.

Exposures in the European Community

Doses to populations of member states due to the Chernobyl accident vary from 0.05 mGy to 0.9 mGy in the first year after the accident, with a total exposure in all following years not exceeding that in the first year. Averaged over the population, such exposure levels are less than the normal background radiation levels that result in annual doses of the order of 1 mGy. As a consequence, epidemiologic (or, for that matter, any) health effect studies comparing populations of entire member states could not be expected to be productive and the Panel recommends that no consideration be given to them.

Within member states, and especially in those with the highest depositions of radioactive substances from the accident, smaller areas exist where the doses to the population considerably exceed the average for the country as a whole. If any study is to have a chance of detecting any effect it will only be one in which comparisons are made between relatively small areas within member states that have experienced the biggest differences in exposure.

Possible Health Studies

The Task Group report discusses the health effects in humans that have been shown to be related to previous radiation exposure. These health effects are:

- short-term effects in the exposed, including developmental effects induced in the foetus or embryo, occurring within a few weeks;

- long-term effects, ie cancer and possibly heritable genetic effects that become demonstrable years after exposure, or in future generations.

Most short-term effects require substantially elevated doses of radiation, ie of the order of grays, and are therefore not expected in the siutation discussed here. Some developmental effects, expressed as congenital malformations, could conceivably be induced by doses less than 1 gray, but the low probability of such effects being induced, given the post-Chernobyl exposure situation, precludes their being detected in any of the exposed populations in the European Community.

Cancer is well known as a late effect of radiation exposure and some estimates of the cancer relationship with dose are available. Given the exposure situation described earlier, it is extremely unlikely that any study on the post-Chernobyl cancer rates in adults within the Community will produce useful information and the Panel considers such a study to have no merit. The study of heritable genetic effects in the offspring of those exposed would also be a totally unproductive effort.

Cancers in children differ from cancers in adults in four important respects relevant to the Panel's task. First, they are likely, in the majority of instances, to be related to events that occur during the period of gestation. Secondly, they are normally so rare and the foetus is so susceptible to radiation damage that associations have been reported following exposure of the foetus to excess doses of ionizing radiation of the order of 10 to 50 mGy. Thirdly, the occurrence of the disease can be related to the area in which the mother resided during pregnancy and to the time of birth. Fourthly, the disease occurs relatively soon after birth. For these reasons, the Panel believes that studies of childhood cancer might offer some opportunity for detecting an effect of the Chernobyl accident.

Despite the above considerations, it is the opinion of the Panel on current estimates of radiation risks that even a study of childhood cancer following in utero exposure would be unlikely to demonstrate any attributable increase in risk. The estimated foetal doses resulting from the Chernobyl accident are too low for such an increase to be reliably detected. Moreover, the complexity introduced by the need to allow for the higher doses experienced from natural background radiation or medical x-rays would conceivably mask any possible effect or produce spurious findings. Nevertheless, there is clear public concern about the health consequences of increased radiation exposure from the Chernobyl accident and it might be possible to detect an effect if our estimates of risk were too low by a factor of about 5. The Panel therefore recommends, as a check on our ability to predict risks from doses of the order received, to contribute to our understanding of the occurrence of childhood leukemia, and to allay public anxiety, that a small epidemiologic survey of childhood cancer be conducted within areas where selected cancer registration was in existence at the time of the Chernobyl accident.

Study Design and Methodologic Considerations

A study of the kind that the Panel recommends requires the ongoing registration of childhood cancers, ie those occurring in early life. As stated before, such registration must have been well established before the Chernobyl accident in order to have confidence in the completeness of registration that is necessary for the study of cancer trends with time to be interpretable. One possible approach would compare childhood cancer rates in areas of high and low exposures, or, if adequate dosimetric information is available, as a function of dose, but again, only if the reliability of the registration data has been established before the accident occurred.

In order to improve the statistical power of these studies, joint analysis of data sets from different registration areas needs to be considered. This, however, will require a high degree of uniformity among the registration procedures, a condition that may need special efforts to accomplish, and one that may be hard to meet for some of the existing registries within the European Community. An additional problem is created by the fact that the areas receiving the highest exposures are not included in registration areas.

For all of these reasons, it would not be advisable to initiate ad hoc studies. The Panel recommends, however, that the Commission give serious consideration to the promotion of comparable studies already going on elsewhere. Excellent registries are operated in countries outside the Community, eg in Nordic countries and in countries of Central Europe, some of which have areas where exposures have been high. The International Agency for Research of Cancer (IARC) is organising a collaborative effort for the monitoring of childhood leukaemia in which particular attention is given to comparability of registry data in different areas. Although much of the information to be obtained is not derived from population groups within the Community, the data obtained should be considered extremely useful, particularly if the study could be extended to include all childhood cancer. Finally, close collaboration with scientists carrying out studies in the USSR, ie on people exposed in areas much closer to the accident site and therefore much more likely to result in scientifically useful information, ought to be seriously considered.

Limitations

The Panel is aware that the above suggestions are not easily implemented and that in all likelihood results from the study envisaged might be open to many and varied interpretations. In order to minimise this possibility, the Panel recommends that if any study is to be undertaken, it be planned in detail, with precise definition of the boundaries of high and low dose areas, exposure and control cohorts and statistical methodology before any data are examined. This will diminish misinterpretation of results which occurred purely by chance.

The Panel also recognises that such a study may require a long-term commitment and that its duration should be kept to the minimum required for obtaining reliable results. As most childhood cancers following in-utero exposure may be expected to occur within a period of 8-10 years after exposure, the Panel recommends that a first evaluation of the childhood cancer cohort-specific incidence be made after five years, at which point a decision can be made whether to extend the observations by another period of five years.

Finally, the Panel notes that some of the difficulties of setting up a well-designed investigation arise from the inadequacies of existing cancer registration mechanisms in areas of high exposure within the Community and in comparable adjacent areas. These inadequacies spring partly from inadequate investment - for which remedies could be found - but also from less tractable problems surrounding the release of clinical diagnoses for public health purposes, going beyond the confines of the clinical consultation. The Panel recommends that member states review their arrangements for releasing such information to qualified scientists, for defining its custodianship and for reconciling confidentiality with the effective utilisation of data that may be useful for the detection of environmental hazards.

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FEASIBILITY OF STUDIES ON HEALTH EFFECTS

IN WESTERN EUROPE

DUE TO THE REACTOR ACCIDENT AT CHERNOBYL

Report of a Task Group for the CEC

1. Introduction

The successful control of infectious diseases and generally improved living conditions have, in the technically developed countries, led to substantially increased life expectancy. Parallel to such improvements there has been a growing awareness of health risks due to a variety of environmental, and diet or life style related factors. Some of the major factors are associated with large risks: tobacco smoking is the leading cause of cancer mortality in men, solar UV-light produces the major part of skin cancers, aflatoxins are an important cause of cancer in countries were refrigeration of food is uncommon, exposure to asbestos has been recognized as a grave occupational risk. The list of examples could be extended, and would include factors that have been identified several centuries ago.

The attention to less prominent risk factors is of more recent origin, and it reflects the generally increased apprehension about global environmental, technological, and industrial changes. The need to consider even minor risk factors has been substantiated by epidemiological investigations which suggest that the larger part of all cancers is due to a multiplicity of causes which are largely unrecognized but should be avoidable in principle. This has led to lasting debates about the possible role of chemical carcinogens and about the need to keep the exposure to such carcinogens at safe levels. Such 'safe' levels can exist only if there are dose thresholds for the action of carcinogens, but the existence of thresholds is an unresolved issue. This uncertainty has focussed added attention on the study of potential risks through epidemiological investigations. It has also made it necessary to consider the acceptability of risks that are caused by small doses of carcinogens in the absence of thresholds.

In the recent past there has been especial interest in the mutagenic and carcinogenic action of ionizing radiations. That ionizing radiation is an effective mutagen and carcinogen had become apparent even around the turn of the century when the first skin cancers and the first leukaemias in radiologists were seen. Nevertheless the knowledge was fragmentary and the resulting disregard for the possible hazards led to tragic experiences caused by the use and

misuse of x-rays and of radionuclides in industry and medicine (see discussion of dial painters, thorotrast patients and radium-224 patients in Annex A). During this period there has been - in spite of warning voices - a general expectation of positive effects of small doses of ionizing radiations.

The situation changed dramatically when the atomic bombs where dropped on Hiroshima and Nagasaki, and when, a few years later, a marked increase of leukaemias was seen among the survivors of the bombings. It was then realized that leukaemia and cancer in general can arise from the mutation or transformation of individual somatic cells and that, accordingly, there may be no threshold of dose for the induction of cancer.

It is a characteristic feature of ionizing radiations that individual charged particles can transfer substantial amounts of energy to a cell. The dose is merely a statistical average of energy received by the exposed tissue, i.e. by a multiplicity of cells. When the dose is reduced, fewer cells are traversed by a charged particle, but those cells that are affected receive energies which are independent of dose and dependent merely on the properties of the charged particle. This is an important difference to chemical carcinogens for which the problem of a possible threshold is more complex. With ionizing radiations no dose threshold can be assumed for the mutation of individual cells and, therefore, for hereditary effects. For radiation carcinogenesis the absence of a threshold is a reasonable assumption, although the situation may be complicated due to radiation induced changes of the tissue or of the immune response. Such changes - if they were to occur even at small doses - could alter the chance of a transformed cell to cause uncontrolled growth, and this could lead to complicated dose dependences.

The continued epidemiological investigation of the fate of the survivors of Hiroshima and Nagasaki has provided a wide range of data on radiation induced cancer. At the same time numerous other studies have confirmed and supplemented the resulting insights. Ionizing radiation is, by now, the most widely studied and - in spite of remaining uncertainties - the best understood carcinogenic agent.

These developments were paralleled by a marked reversion of the public perception of the risks of ionizing radiations. The earlier lack of attention was replaced by acute awareness. The concern began to grow already during the years of large scale atmospheric nuclear weapons testing (1950-1965), when the fall-out of the tests began to add appreciably to the general radiation exposure in the northern hemisphere. The impact was far greater after the reactor catastrophe in Chernobyl, when a small part of the Soviet Union received massive radioactive contamination, and when even in some parts of the Western European countries, radioactive contaminations occurred which reached or exceeded the total fall-out during the nuclear arms testing. Outside the immediate area of the reactor accident the resulting doses were too small to cause 'acute' radiation effects, i.e. effects that occur and are observable soon after irradiation. However, the doses were large enough, in some regions even of Western Europe, to cause an appreciable increase above the average natural radiation exposure in the year after the accident (see Annex B). In the first year after the accident the estimated increase over the natural exposure was about 20% in the countries of the European Community (see Annex B). In the five subsequent years the increase will amount to an estimated 3%.

The unexpectedness of the event and the initial uncertainty about the level of the contamination and the resulting doses led to extraordinary public anxiety and to marked changes of life style and dietary habits. Administrations and experts succeeded only partly in informing the public and in avoiding the impression of widely contradictory measurements and interpretations. Even now, after many of the seeming contradictions have been resolved and doses due to external and internal exposures are known with some accuracy, there are lasting concerns and continued requests to perform investigations that might contribute to an assessment of possible health effects of the radioactive contamination after the reactor accident.

The need and the feasibility of an epidemiological follow-up investigation of the evacuees from the immediate surrounding of Chernobyl are apparent. There is international agreement that every effort should be made to perform such studies and to guarantee their continuation over

several decades. The group of evacuees comprises about 35000 people who received doses around 0.4 Gy, which is 100 to 1000 times larger than the exposures resulting from Chernobyl in even the most highly contaminated regions of Western Europe (for details see Annex B).

It is a much more difficult question whether epidemiological investigations should be performed in the far larger populations of Europe which were exposed to much lower increments of their usual exposures. In spite of the difficulty, the question needs to be asked, and it is the purpose of the present document to contribute at least partial answers.

The great public concern and the need for a broad consensus in any resulting decision make it necessary to treat the problem in a way which is intelligible even to the nonspecialist. In the subsequent introductory chapters some essential facts about ionizing radiations and their biological effects are given. Although the treatment is brief, it may facilitate general understanding and the access to sources of more detailed information.

2. Exposure to Radiation

2.1 Exposures from Internal and External Sources

There are various types of ionizing radiations. They differ in their ability to penetrate exposed objects, and they differ in the amount of energy that individual charged particles impart to a cell. However, the principal underlying mechanisms are the same. The most commonly encountered radiations are x-rays or gamma rays; they consist of photons (i.e. electromagnetic quanta) with energies that are large compared to the binding energies of electrons which determine the structure of molecules or biomolecules. The photons can penetrate the body and can release in the body energetic electrons which have comparatively short ranges of a few mm, but which can in turn - while they traverse the cells of the body - liberate thousands of further, less energetic electrons. Electrons can also be emitted directly by a radionuclide which is then called a ß-emitter, and the resulting molecular mechanisms are the same whether the electron is released by a photon or by a radionuclide. Any release of an electron from a molecule is called ionization. Not all the molecular changes of the DNA resulting from ionizations are correctly repaired by the cell, and the remaining damage can impair or alter the cellular functions. It is important to realize that the photons, by themselves, have no biological effects, but that they act merely by producing a 'secondary' radiation within the body, i.e. the high energy recoil electrons.

The failure to understand the role of the recoil electrons has led to lasting confusion after the reactor accident, when radiation exposure due to incorporated radioactive caesium was held to be of entirely different quality than exposure from external caesium. In fact, there is no difference in principle between the action of external and internal exposure. Incorporated caesium is distributed nearly uniformly throughout the body and it produces through the emitted ß- and gamma-rays a general exposure of all tissues and organs. The same occurs, due to the fairly high penetrating power of the caesium gamma rays, when the exposure is from the outside. After the decay of a caesium atom, either outside or within the body, the released photon traverses a substantial distance and then sets into motion an energetic electron; this

process is entirely the same, regardless of whether the photon had originated within the body or outside. Incorporated caesium produces a continuous exposure, until it is removed from the body, with a biological half life (mean excretion time) of about 80 days. A continuous exposure results equally from caesium in the environment. This external exposure decreases much more slowly in time because part of the caesium can remain for years in the superficial layers of the soil and can, therefore, contribute to the terrestrial gamma ray intensity until it decays with its physical half life of 31 years. Caesium is, therefore, to be regarded as a general radiation hazard to all tissues of the body, whether its effect is exerted externally or internally, and any increase in specific cancers would be related to the varying radiation sensitivities of the different organs and tissues, rather than to the location of the caesium itself. The equivalence of external and internal exposures implies the equivalence of equal doses from external sources and from sources through the food chain (for quantities and units see Annex E). It is merely a matter of appropriate dose calculation procedures to evaluate each contribution correctly, including, for example, the contribution of ß-rays (~50%) for internal caesium exposure and excluding this component for external exposure because of the short range of the electrons.

2.2 Organ Specific Exposures

There are, of course, specific effects of incorporated radionuclides, when these concentrate in individual organs. The most critical organ seeking radionuclides after a release from a reactor are iodine, and strontium. Strontium is a 'bone seeker' of long half life, and it was a major contributor to possible health detriments in the fall-out from nuclear weapons testing. The radioactive contamination in Europe after the reactor catastrophe in Chernobyl, on the other hand, contained comparatively little strontium and it added only a minor increment to the contamination which has remained from nuclear weapons testing. Strontium was, therefore, not a nuclide of major concern after the reactor accident.

Radio-iodine, however, has been of special concern because high activities were released and have reached Western Europe. The iodine is effectively accumulated in the thyroid and can cause substantial radiation doses in this organ (see Annex B). Due to the short physical half life of the critical nuclide, I-131, (about 8 days) the period of concern was limited to several weeks. But in the initial phase immediately after the reactor accident, I-131 has been the critical radionuclide of predominant concern; with unreduced consumption of contaminated vegetables and milk, thyroid doses of up to several tens of mGy may have been reached even in the Western European countries. The doses due to caesium were much lower, and were unlikely to exceed about 1 mGy in the first year even in critical groups; however the caesium led to an almost uniform exposure of the whole body.

Summarizing these considerations one can say that the radionuclides of main concern after the Chernobyl accident are iodine which can affect the thyroid, and caesium which can affect all organs of the body. An assessment of the magnitude of doses from these radionuclides for different levels of contamination is given in Annex B. In such considerations one must distinguish between organ doses and whole body doses. As a rough measure of comparison one may note that a whole body dose is assumed to carry a cancer mortality risk that is about 30 times larger than a thyroid dose of the same magnitude (see Annex E).

2.3 Natural Radiation Exposure

Exposures from the radioactive contamination after the accident can usefully be compared with the magnitude of doses from natural exposures. The natural radiation exposure results from terrestrial gamma radiation, cosmic radiation, and from irradiation due to radioactivity always present in the human body (mainly about 60 Bq/kg of K-40). The inhalation of radon and its radioactive decay products accounts for an additional important contribution.

The total annual dose from these three contributions - not including the contribution from radon - is about 1 mGy, but it may vary substantially according to the different levels of

terrestrial gamma radiation. In certain parts of the world, and also in parts of the member states of the European Community, the natural radiation exposure is more than doubled due to higher terrestrial gamma radiation. There are also substantially larger doses in regions of higher altitude because of the enhanced cosmic-ray intensity. The inhalation of radon and its decay products in houses adds to the quoted dose; it results in an α-ray dose to the lung, commonly in excess of 0.5 mGy, and in not infrequent instances in excess of 5 mGy per year. Using the applicable weighting factors for the lung (w = 0.12) and for α-radiation (Q = 20) (see Annex E) one concludes that the contribution of radon in houses equals on the average approximately the contribution of the remainder of natural radiation exposure. The total whole body dose during a lifetime due to the natural sources amounts to about 100 mGy.

The contribution to the exposure of the population due to medical diagnostic procedures adds - in the European countries - another dose averaged over the population and the organs of the body of about 0.5 mGy per year.

3. Radiation Effects

3.1 Acute (Early) Effects

Absence of Acute Effects:

Large doses of radiation cause massive cell killing and radiation syndromes which are termed acute effects. The severity of these effects is dependent on the dose of radiation, and after a whole body dose of several gray the exposed person may die. Moderate doses of radiation, or larger doses to localized tissues or individual organs, as they are employed in therapy, can produce various dose related effects, such as inflammation, scarring, or tissue atrophy.

Acute radiation effects with dose related severity are analogous to effects from other physical agents such as burning or chemical poisoning. For instance, prenatal exposure with sufficiently large doses can cause extensive cell killing in the developing embryo or foetus, and this can lead to abortions or can damage the developing brain. For acute effects, there is a threshold dose below which the effect is seldom or never detected. These effects are not of concern with regard to the radioactive contamination produced in countries of the European Community after the Chernobyl accident. The resulting doses were far below those required for acute effects.

3.1.1 Malformations

The majority of congenital malformations are not demonstrably due to simple specific genetic disorders, although some of them show familial concentrations and some (e.g. cleft palate) offer evidence that abnormal genes may play a part. Most of the malformations are probably caused by environmental factors, or are due to complex random combinations or re-assortments of genes which in other cirumstances would individually be regarded as normal. One must nevertheless consider the possibility that radiation damage to the genetic material of the early embryo, possibly before implantation, could cause an irreparable re-direction of

development. The major concern, however, must be malformations, due to massive cell killing; but these are acute effects with a threshold dose and, therefore, most unlikely to occur at small doses.

3.1.2 Mental Retardation

There is one special effect that is related to cell killing but can occur at relatively low doses. This effect is mental retardation caused by irradiation of a foetus especially between the 8th and the 15th week after conception. Among the children who were exposed in utero by the atomic bombs in Hiroshima and Nagasaki there was an increased frequency of mental retardations, and it was recognized that most of the mentally retarded were exposed during the critical period (8th to the 15th week post conception) which is associated with a wave of proliferation and of migration of neural precursor cells. The statistical analysis of the data suggested that there need not be a threshold in the dose dependence, and a risk factor of 0.4 per Gy (Tab.1) has been estimated. Determinations of the IQ-values of the prenatally exposed children were cited in support of this conclusion. However, it was also pointed out that a threshold cannot be excluded. Regardless of this unresolved question it must be noted that the high sensitivity had not been expected earlier, and that it requires special caution in the avoidance of exposures of the foetus during the critical period.

3.2 Late Effects

Of main concern, with regard to low doses, are the so-called late or long-term effects which result from the mutagenic action of ionizing radiation and which can become manifest years or decades after the irradiation. The main effects of this type are hereditary damages and cancers. The probability for the effect depends on dose but the severity does not. The response to radiation is thus of an all-or-none character; those who escape are entirely unaffected. One cannot exclude that some of these effects have a dose threshold below which

they are not induced. But there is no actual evidence for such thresholds, and it appears certain that there can be no threshold for radiation induced mutations of cells which are responsible for hereditary damage and, at least partly, responsible for radiation carcinogenesis. For radiation protection it is, therefore, generally assumed that there is no threshold and that any dose, however small, will carry a correspondingly small risk of late effects. One must note in this context that there is an emerging consensus, even among toxicologists, that no thresholds of dose or concentration exist for the genotoxic actions of chemical mutagens either.

The cancers and genetic effects induced by radiation exposure are indistinguishable from cancers and genetic effects occurring without radiation exposure. They cannot be attributed to irradiation in an individual case. The effects of the radiation are manifest only in increased incidence rates in exposed groups. Characteristically, the increased incidence rates occur after a prolonged interval of time, from several years to several decades after exposure.

For the purposes of the present discussion and for small doses we will utilize the assumption of linear dose dependences in the sense of a conservative estimate. An implication is, that a given dose causes the same number of disease events (e.g. cancers), no matter whether it is evenly or unevenly distributed across the population. The exposure of a population can then be characterised by the collective dose which is the sum of doses received by individuals of the population. Accordingly, the collective dose (stated in units of personGy) is the product of the mean individual dose and the number of individuals concerned.

In the case of the Chernobyl accident, and except for people exposed in the immediate vicinity of the disaster, late effects are the only ones which one might possibly attempt to detect. That is, we must look for changes in the incidence rate of disorders already known to be radiation related. We should not expect that radiation syndromes or other acute effects (e.g. cataracts) have occurred. Nor should we expect to detect any diseases which are specifically related to radiation, and which could serve as a unique marker of its presence. Furthermore, although we should keep an open mind on the matter, we should not expect to detect previously unknown radiation effects.

3.2.1 Cancer

The major risk of small doses of ionizing radiation is carcinogenesis. It is well known from a variety of studies, and chiefly from the fate of the survivors of the atomic bombings, that radiation can induce most cancers and, especially, leukaemias. The increased frequencies have, in the majority of these studies, been found for persons exposed to relatively high doses of the order of some 100 mGy or more. Any extrapolation to low doses must, therefore, be based on hypotheses. For the puposes of the present study linear extrapolations (see solid line in Fig.1) will be utilized. But a variety of radiobiological investigations suggest that actual dose dependences are linear-quadratic (broken line in Fig.1) with correspondingly decreased risks at low doses. Other forms of the dose-effect relation can also not be excluded. However, there is no apparent reason to believe that there should be a threshold at low doses below which malignancies cannot be induced by ionizing radiations. The important role of somatic mutations for the induction of cancer is, in fact, an argument for the prudent - if possibly conservative - assumption of linearity down to low doses. Radiation induced changes in the tissue response to a transformed cell or in the response of the immune system could occur, but they may be unlikely at very low doses.

The important role of cellular repair processes might suggest that there needs to be a dose threshold, but this assumption would be fallacious because it presupposes fully efficient repair in unirradiated cells or in cells exposed to low doses. In fact it is known that cellular repair processes, while highly effective, are never free from a certain probability of misrepair. Mutations and possibly a resultant loss of growth control are the result of such misrepair. If the probability of misrepair is substantially unaltered at small doses, there is no reason to expect dose thresholds; one will, instead, obtain linear dependences on dose at low doses.

There is, on the other hand, great interest in the study of any possible effect that even small doses might have on the repair systems, and one may, therefore, note certain preliminary evidence that small doses may activate repair systems which, in turn, enable the cell to respond to subsequent (radiation induced) damages more effectively. Such effects could alter

considerably the dose-effect relations, and they would, therefore, be highly relevant to the estimation of risks from low doses. Current research in radiation biophysics and epidemiology deals with these phenomena, but it has not, as yet, led to definite conclusions.

Radiation induced tumours cannot be recognized directly because they do not differ from spontaneous tumours. The statistical proof, on the other hand, of increased rates in a population is difficult because the tumours occur years or decades after an exposure. One has to observe, at least in principle, the total life span of all members of an irradiated group and register all cancer cases. In most investigations this is not feasible, and certain basic assumptions about the temporal course of the tumour incidences must, therefore, be utilized, even if the observation span covers several decades. The required extrapolations are based on lifetime risk projection models which differ for different types of tumours.

For a variety of cancers, mortality rates - and in some instances also incidence rates - have been estimated as a function of dose, of sex, of age at exposure, and of time after exposure. Some of the essential points will be considered.

3.2.1.1 Solid Cancers

From the available data one may conclude that radiation enhances the rates of solid tumours (all cancers except leukaemia) in such a way that they run proportional to the spontaneous age specific rates (see Figs. 2 and 3); bone sarcomas do not seem to follow this pattern. The enhancement factors, i.e. the rates in the exposed divided by the rates in the non-exposed (relative risks), are different for different tumours and they depend on dose, on sex, and on age of exposure (in general they are higher for younger ages at exposure). According to the relative risk model they do not depend on time after exposure (after a latency period of 5 to 10 years). Lifetime risk projections are often obtained on the basis of this model.

The relative risk model has been used in the most recent risk estimates for solid tumours after the revision of the atomic bomb dosimetry (Annex A, Fig.A.1). Averaged over all ages a dose of about 2 Gy is estimated to double the cancer rates; such a dose would, therefore, increase the lifetime cancer-death risk from its present value of about 0.2 to a value of 0.4.

A dose of 2 Gy to double the cancer rates corresponds - if one uses the linear extrapolation to low doses - to a mortality risk factor for solid cancers of about 0.1/Gy (1000 cases per 10000 persons per Gy). This is substantially larger than estimates which have earlier been employed by BEIR, UNSCEAR and ICRP (partly due to different premisses), and which have contained an assumed reduction factor for low dose and low dose rate. For the subsequent estimations in this document a purely linear extrapolation to small doses is employed, and a risk factor of 0.1/Gy for all solid tumours combined is used as an average for all ages at exposure (Tab.1 and Annex B, Tab.B.3). Even if the estimate is conservative, it provides useful general guidance.

3.2.1.2 Leukaemias

For leukaemias the situation is different (Fig.2). Among the survivors from Hiroshima and Nagasaki, and also among patients treated with x-rays for ankylosing spondylitis, leukaemias have occurred in a characteristic wave of increased frequencies. These waves reach, after a short latency of about 2 to 5 years, a maximum 5 to 10 years after exposure. The increased rates disappear largely after about 20 to 25 years (Fig.3). Such temporal dependences that do not parallel the spontaneous age specific rates are referred to by the term absolute risk model. Fig.3 indicates the characteristic differences between the absolute risk model for leukaemia (and bone sarcoma) and of the relative risk model for solid cancers.

From the absolute risk model it follows that most radiation induced leukaemias among the atomic bomb survivors have already occurred. This is in sharp contrast to the situation with solid tumours where - according to relative risk model - a large fraction of the radiation

induced solid tumours is still to occur, especially among those exposed at younger ages. Today, four decades after the beginning of the epidemiological studies in Hiroshima and Nagasaki, the projection of future excess cancer cases is still a central issue for solid tumours. The projection of future cases is of minor importance for leukaemias which follow the specific temporal course characteristic for the absolute risk model (Fig.3 and Annex A).

According to the new risk assessment, the doubling dose for leukaemia - again averaged over all ages at exposure - is approximately 0.7Gy (spontaneous life time rate: about 0.007) which is less than the doubling dose for solid tumours. However, the relative risk model has little meaning for leukaemias; it is more appropriate to state the estimated absolute risk factor of about 0.01/Gy.

3.2.2 Cancer in Children

Cancer in children has been shown to be correlated with radiation exposure, including medical diagnostic exposures and therapeutic irradiations. There is also evidence of a high sensitivity of children to radiation exposure with respect to cancers which occur many years later, i.e. during adulthood. In a few studies increased cancer frequencies have been found even at comparatively low doses (see Annex A). One example of the sensitivity of children is the finding of increased thyroid cancer rates in a follow-up study of children whose heads were irradiated with x-rays for the treatment of tinea capitis (ring worm of the scalp). In this study the estimated average dose to the thyroid was less than 100 mGy; the thyroid cancers occurred many years after the exposures and they paralleled the rise of age-specific cancer rates.

From the follow-up of the atomic bomb survivors one cannot yet infer reliable absolute risk estimates for those who were exposed as adolescents or children; there are still no precise data for the late occurring solid cancers. But it has become clear that the relative risks, i.e. the radiation induced rates of cancer in relation to the spontaneous rates, are substantially larger for those exposed at young ages.

For childhood tumours, and especially for the leukaemias which occur early after exposures during childhood the relative excess risks, i.e. the rates of radiation induced to spontaneous cases are particularly high. Among the children exposed to the atomic bomb radiation about 12 excess leukaemias were observed. This corresponds, under the assumption of linearity in dose, to a risk factor of about 0.01/Gy. This is roughly the same absolute excess rate as that estimated for adults, but it is a much higher relative excess over the low spontaneous rate in childhood. A dose, due to the reactor accident, of 1mGy will nevertheless not lead to observable increases; it may enhance the spontaneous risk of about $3 \cdot 10^{-4}$ to develop leukaemia between age 0 and 10 by roughly 3%; such an increase is certain to remain undetected in any epidemiological survey.

The situation might be different for childhood cancers due to prenatal exposures. But for these cancers there are widely diverging risk estimates. Among 920 children exposed in utero in Hiroshima and Nagasaki with doses in excess of 10 mGy no leukaemia cases were registered. The average dose in these prenatally exposed children was, according to the new dosimetry, only 0.25 Gy. A risk estimate of 0.01/Gy would, therefore, correspond to about 2 expected cases. Even a threefold higher risk estimate of 0.03/Gy cannot be excluded, because it would correspond to only 6 expected cases, and this could have remained unobserved due to statistical fluctuations and due to a lack of observations in the immediate years after the bombings.

Childhood cancers were studied in some recent re-evaluations of data from the 'Oxford Survey of Childhood Cancer' (OSCC) which indicated substantially increased cancer rates in children prenatally exposed (for diagnostic purposes) with x-ray doses estimated to be about 5 to 10mGy. The results can be seen as evidence that prenatal sensitivity for radiation induced cancer exceeds postnatal sensitivity. Estimates of the probability of childhood leukaemia of about 0.07/Gy and of a probability of all fatal childhood cancers of 0.2/Gy were derived for prenatal exposures (Annex A, (A16, A17)). The estimated childhood leukaemia risk appears, as stated, twice as large as maximum values compatible with the observations on the children exposed in utero in Hiroshima and Nagasaki. The overall childhood cancer estimate of 0.2/Gy

is even more debatable; it has not been observed among the children exposed as foetuses in Hiroshima and Nagasaki, and it should have been observed if the value were genuine. Thus, in order to assess the feasibility of epidemiological studies in the European countries after the Chernobyl accident, the risk estimate of 0.1/Gy for prenatally induced childhood cancer (leukaemias and solid tumours between age 0 to 10 years) will be utilized (Tab.1). It can be compared to a 'spontaneous' probability of fatal childhood cancer of about 10^{-3} (one case in 1000 children younger than 10 years), and it corresponds, with these assumed numbers, to the very low dose of about 10mGy to double the spontaneous incidence.

The short and narrow latent intervals and the high ratios of induced to spontaneous rates make childhood malignancies the evident target of potential research efforts (Sect.4.2).

3.2.3 Genetic Effects

Genetic effects, i.e. hereditary effects, are disorders produced through damage of DNA in germ cells and through the resulting mutations. While dominant mutations may appear fairly quickly, recessive mutations may take several generations to manifest their ultimate effects. Almost all specific dominant and recessive disorders are rare.

The only disorders of sufficient frequency to be investigated within a short period after exposure are those recognizable as karyotype disorders, i.e. as wrong chromosome numbers due to faulty germ cell division, or due to abnormal chromosome structures (chromosome fractures, and irregular rejoining). Down's syndrome (trisomy-21) is the classic example of the former case but is unlikely to be a radiation-related disorder. Chromosome rearrangements are dose-related and are readily identifiable.

For hereditary effects no threshold can be assumed below which the induction probability is zero. This applies generally for genotoxic effects of mutagens, but it is especially evident for ionizing radiations, since even individual charged particles can transfer considerable amounts of

energy to the cell and can, therefore, individually cause substantial damage. At sufficiently low doses the number of cells affected is proportional to dose, and each of the affected cells is traversed by only one charged particle.

On the other hand, one has not found in man direct evidence of hereditary damage due to irradiation. Even extensive efforts have failed, up to now, to demonstrate significant increases among the children of those exposed to the radiation from the atomic bombs. However, in view of the size of the population examined and the doses to the parents, recognizable increases were unlikely. While there is no doubt that radiation induces hereditary damage, quantitative estimates must, at present, be based on animal data. From such data one infers that the doubling dose for hereditary damage lies between 0.5 and 2 Gy. This corresponds to a risk factor for genetic disorders of about 0.01/Gy (Tab. 1). However, there is considerable uncertainty how multifactorial diseases may be enhanced due to radiation exposure.

3.3 Indirect Effects

It is not always recognized that some measures to reduce doses after the Chernobyl accident may have been harmful by themselves. For example, iodine salts were taken in order to saturate the thyroid gland and to prevent the further uptake of radioiodine, and this carried some risk. It is well known that an abruptly increased administration of supplementary iodine may provoke the incidence of thyrotoxicosis (hyperfunction of the thyroid) in populations suffering previous iodine deficiency.

Anxieties after the reactor catastrophe have induced various changes in lifestyle and dietary habits in the general population that can have ill effects. Nutritional changes or increased tobacco and alcohol usage are obvious examples. Other deleterious effects were pregnancy terminations, or, possibly, untoward effects on pregnancies by anxiety or changed living patterns. Further consequences included suicides and para-suicides, the consumption of anxiolytic drugs, and enhanced frequency of depressions.

4. Research Targets

In identifying attainable research targets the following points need to be considered. First one must recognize that it is neither possible nor desirable to attempt a direct measurement of all the health consequences of the Chernobyl discharges. Even if such measurement were possible, the inquiry would last so long that it could not provide practical guidance on radiation protection or on energy production policies. Assessment of these kinds can only be based upon existing knowledge and existing models, modified through current research, and not upon direct determinations.

The research strategy must therefore be directed towards the measurement of selected health effects: those which will be detected and measured unambigously and with reasonable accuracy within a reasonably short period of time and those which enhance the theoretical basis of decision making.

The value of the results we seek is in part to support statements of reassurance concerning the effect of the Chernobyl accident itself, and it is partly for this reason that we shall recommend some inquiries although we expect their results to be negative. However, the main value of the results is scientific, helping to set upper limits to the still uncertain current estimates of the relationships between environmental radioactive contaminations and the disease risks which follow. These estimates provide an essential basis for public health planning, including the formulation of energy policies, the definition of permissible radioactive discharges, and the setting of hazard-warning levels. They provide equally important guidance for emergency action in the case of future accidents.

In identifying appropriate research targets, we have also examined resource costs. As it now appears, this issue hinges chiefly upon the quality, location and scope of existing health monitoring facilities and skills. Requirements for supplementary investment are therefore quite limited, and the added expenditure can even have positive spin-offs for other problem areas. However, questions will be raised regarding the adequacy of existing facilities and methods,

and whether they should be enhanced to meet requirements that may arise after any future accidents.

Firstly, although possible health effects of Chernobyl may take some years to emerge, their elucidation demands immediate planning and immediate implementation of a research programme. It is necessary to define in advance the geographic zones, the age groups of the population, and the health criteria through which one can achieve comparisons between different groups. One needs to take immediate steps to avoid substituting what should have been 'prior' declarations by choices made after one has seen the results. If this is not done there is the risk of attributing spurious interpretations to the fluctuations that are bound to arise and that are purely random in nature.

4.1 Cancer in Adults

Cancer induction is seen as the major hazard of small doses of radiation. In the follow-up study of an irradiated population most radiation induced cancers would occur in adults. However, the increased cancer incidences are not readily detected; even with the atomic bomb survivors who were subjected to high doses it took decades of different studies to detect any enhanced rates of solid tumours and to quantify them in continued observation. The follow-up of 76 000 persons led to the conclusion that about 340 excess cancer deaths were caused, in 1985, among a total number of about 6 000 cancer deaths. This represents the excess mortality in 1985 and the number is likely to increase. It is evident that it must be difficult or impossible to detect enhanced rates among populations subjected to exposures that were several orders of magnitude smaller. The difficulty is largely due to the high rate of spontaneous tumours or tumours induced by a multitude of known or unknown factors. Any radiation effect is difficult to recognize against this background rate. In addition, the latent intervals between exposure and the occurrence of solid tumours in adults are relatively long (in the order of 10 to 30 years or more) and variable; cancers resulting from a transient wave of initiations are thus widely spread out in their appearance times.

The collective dose resulting from Chernobyl is estimated as about 60000 personGy during the first year after the accident for the entire population of the European Community (320 millions). This corresponds to an overall average dose of only about 0.2 mGy (Annex B). With an assumed 3 times larger dose accumulation over 50 years a collective dose of nearly 200 000 personGy will be reached. The risk estimate for cancer mortality of 0.1/Gy could be used to infer a hypothetical number of 20 000 added cancer deaths within the population of the European community due to the Chernobyl release. While formally correct and while correctly indicating that the reactor catastrophe had grave environmental consequences, the computation of a possible number of deaths can, nevertheless, be grossly misleading. The reason is that such numbers suggest - at least to those who are unfamiliar with population statistics - a tangible threat to the individual and a substantial increase of prevailing cancer rates. Both suggestions would be quite incorrect. This is readily apparent if one notes that the additional dose due to Chernobyl is only a minor fraction of the natural radiation exposure and that it is also small compared to the geographic variations of the natural radiation background which are normally disregarded, and are certainly not seen as threats. Any quoted number, such as the tentative figure of 20 000 additional cancer deaths, would have to be measured against the expected number of about 80 million cancer deaths to occur among those presently alive in the European community. Prevailing cancer mortalities would, therefore, be enhanced by much less than one part in a thousand and - objectionable as such an increase may be - it is far below the threshold of statistical detectability and equally far below the changes brought about by other risk factors that are voluntarily or unvoluntarily accepted.

A global epidemiologic investigation of possible radiation effects after the Chernobyl accident comprising all countries, and all cancers in the entire populations must clearly be rejected. The question then arises, whether there are specific age groups, specific cancers and specific geographical regions that may still offer possible research targets. This is considered in subsequent sections.

4.2 Childhood Cancer

A critical age group with high radiation sensitivity are those who were infants or were in utero during the period of enhanced radiation exposure, i.e. the first year after the accident. In epidemiological studies the enhanced prenatal sensitivity can be taken into account by reference to the dates of birth of affected children, and not to the date of diagnosis of a tumour. The consideration of birth cohorts would bring out more clearly any transient peak in childhood tumour rates, if it occurred in those prenatally exposed.

According to the statistical analysis of childhood cancers and their association with prenatal diagnostic x-ray exposures, a probability of 0.1 per Gy to develop a solid tumour or a leukaemia before age 10 may - as has been pointed out (Sect.3.2.2) - be a reasonable, if conservative, estimate.

Within the European Community the most highly contaminated regions with the highest doses were Northern Italy, Southern Germany, and Greece. Doses between 1 mGy and 3 mGy for critical groups (e.g. infants and children) were estimated in these regions for the first year after the accident (Annex B).

For all prenatally exposed during this period one estimates a collective dose in the European Community of up to 1 000 personGy due to the reactor accident; this includes a contribution of Italy and Germany each of 300 to 500 personGy. For particular subgroups, such as 60 000 infants born in the southeast of Bavaria in 1987, an increase of childhood cancer of 9 cases within the next 10 years would be assumed, beyond 60 cases expected (background rate). This number, which is based on the tentative risk factor of 0.1/Gy, would correspond to an excess relative risk of about 15% and this would be at the lower limit of eventual detectability (Annex B, Tab.B.3 and Annex D). However, one must note that the considerations in Annex D are based on criteria for a stationary and uniform population. In reality one deals also with secular and geographic variations of cancer rates which would obviate valid conclusions even if the theoretical conditions for statistical discrimination were met.

The comparison between small subpopulations is a central problem in epidemiological designs; one must seek the most suitable balance between a reduction of group size towards higher exposures and an extension of group size towards larger overall numbers of the prenatally exposed. The power of statistical evaluations, i.e. the probability for proving enhancements, may be similar whether one examines a small subgroup with higher exposure or a larger group with lower exposure.

4.3 Cancer of the Thyroid

The thyroid was a critical organ in the initial phase of the radioactive contamination after the reactor accident. Although parents have largely avoided the use of fresh milk in the weeks after the accident, there appears to have been an average thyroid dose of 3 to 10 mGy for children, and a thyroid dose about 5 to 10 times smaller for adults (Tab.B.2).

For adults it is certain that any increase of thyroid cancers will remain undetectable. A Swedish study of a large number of patients subjected to thyroid doses from I-131 that are several thousand times higher than those due to the Chernobyl accident has failed to indicate significantly enhanced rates of thyroid cancers after a mean period of observation of about 25 years.

For children, and for those exposed prenatally, the question deserves a more detailed analysis. In the study of children exposed to external x-ray exposure for ringworm of the scalp a marked increase of thyroid cancers has been seen, although the estimated dose to the thyroid was only about 100 mGy. For children with a thyroid dose of 3 to 10 mGy after the reactor accident, increased thyroid cancer rates might, therefore, not be very far from the limits of detectability. However, animal studies as well as the Swedish epidemiological studies on I-131 patients suggest strongly that the low dose rate exposure from I-131 is substantially less effective per unit dose in inducing thyroid cancer than acute external x or gamma irradiation.

It would, therefore, seem that any increased rates in thyroid cancer, even in those exposed prenatally or as children, will remain undetectable (Annex A, (A13, A14, A26)).

A further argument against epidemiological studies of thyroid cancer after the Chernobyl accident is the difficulty in diagnosis and the resulting incompleteness and uncertainty of registry data. Finally, one must consider that there has been widespread and largely uncontrolled administration of stable iodine in varying doses; this could change substantially the rates of non-malignant alterations in the thyroid. There is, therefore, no possible justification for thyroid studies in connection with the reactor accident and the resulting radioactive contamination in the European countries.

4.4 Genetic Effects

As stated in Section 3.2.3, the dose to double hereditary defects (genetic effects) may be about 1 Gy. An added dose in the order of 1mGy due to the radioactive contamination from Chernobyl could then increase the rate of genetically transmitted disorders by about 0.1%. Such an increase need not be unimportant, but it would be far below the threshold of statistical detectability, and it would remain invisible, even if the risk factor were far higher.

The period after the reactor accident was replete with poorly supported but highly publicised reports of hereditary damages and of prenatal malformations. All these reports remained unsubstantiated, and some particularly sensational assertions were almost immediately traced to faulty data collection or erroneous interpretations. An example was the assumed relation of an observed Down's syndrome cluster in Berlin to a radioactive contamination which, in this city, had never reached radiation levels constantly present in other parts of the country. An even more glaring misrepresentation was a subsequent faulty analysis of data from prenatal analyses performed in the months after the accident within the Federal Republic of Germany. Later coordinated studies with better methodology demonstrated no irregularities.

The continuing study of the children of the atomic bomb survivors who have been subject to much higher radiation doses has, up to this point, demonstrated no increase of hereditary damage; it shows the futility of initiating a study that relates to doses of one or a few mGy. In subsequent sections it will, nevertheless, be pointed out that there are valid general reasons to monitor registries of hereditary defects for known and unknown factors. Even without a decision for a specific radiation study there can thus be surveillance of the frequency of hereditary defects.

4.5 Malformations

There is no evidence that prenatal radiation exposures after the Chernobyl accident in the countries of the EC have caused malformations. Malformations are acute effects with substantial dose thresholds; they can be caused by considerable injury to the developing foetus, and by radiation doses largely in excess of 0.1 Gy. With the possible exception of mental retardation, all malformations would require far higher doses than those caused after the reactor accident in the European countries and any increased probabilities are projected to be far below the level of statistical detectability.

4.6 Mental Retardation

There is evident need to study possible damage to the central nervous system of children who were exposed in utero among the population around Chernobyl that was evacuated after receiving doses of several hundred mGy. However, even in the most highly contaminated countries of the European Community, it is very unlikely that a foetus could have reached an excess dose of 0.2 mGy during the critical period. But even if that dose had been reached, and if the linear estimate of 0.4/Gy were valid, the number of mental retardations due to the exposure would be less than one case in 10 000 newborn exposed in the critical foetal phase which is substantially smaller than the prevalent occurrence of severe mental retardation which

is in the order of 0.002 (two cases in 1 000 newborn). It is, therefore, highly unlikely that any increased frequency could be recognized statistically.

5. Registration Facilities

The feasibility of epidemiological investigations depends crucially upon the existence and the reliable continuity of adequate registration systems for health effects that could be caused by ionizing radiation. The availability and quality of registration and certification systems in different countries and in the different parts of these countries must, therefore, be carefully examined.

5.1 Data Collection and Registries

Studies of health effects following the Chernobyl accident could, in principle, be attempted in several ways:

- by examination of routinely collected data to monitor incidence trends and geographical variations or radiation induced diseases;

- by designed epidemiological studies based on existing and ongoing data collection;

- by specifically designed studies and data bases, as in the follow-up of the population evacuated from the region around Chernobyl.

To study time trends of disease rates and to detect possible changes after the Chernobyl accident, it is necessary to determine reliable baseline rates and to estimate accurately any existing trends before the accident.

For comparisons of rates between areas with different contaminations, it is important to analyse the collected data with comparable methods. The two types of analysis, the studies of temporal or of geographic variations, can be combined in order to contrast time trends in exposed and non-exposed areas. For example, one might compare temporal trends, suitably

standardised for demographic changes, in southern and northern Germany, or in northern and southern Italy. Most other countries of the EC incurred largely uniform contaminations so that they invite no consideration of internal comparisons.

A general criterion for the feasibility of studies is the presence or absence of data registries in areas where the fall-out was highest. This applies equally to designed studies.

An attempt will be made in subsequent sections to list potential sources of data on malformations, hereditary and carcinogenic effects. Such sources need to be utilized for any survey of incidence (or prevalence) rates and their possible changes after the Chernobyl accident. A more detailed review of existing registries on birth defects and on cancers is given in Annex C. It is accompanied by information on ongoing monitoring studies.

5.1.1 Malformations and Genetic Effects

Collecting information on birth defects and chromosomal damage (Annex C) has long been recognised as a task requiring specialised registries and trained pediatricians and nurses. It is well known that the data collected on birth certificates are inadequate. National, sub- or supranational registries are, therefore, required, and such registries have been set up in most countries of the European Community.

Most of the registries were established before 1980 and can, consequently, provide pre-Chernobyl baseline prevalences of particular congenital anomalies calculated for, at least, 5 years. The fraction of total births monitored by the registries in each particular area is high (over 90%), and the possibility of a bias through increased reporting after Chernobyl is correspondingly small. A possible bias must nevertheless be borne in mind. The registries which indicate 100% coverage carry out periodic checks of their completeness for specific anomalies.

Two different types of damage can be studied, developmental abnormalities (such as congenital malformations) and genetic disorders. Their time patterns are different: developmental abnormalities would appear within a limited period corresponding to birth cohorts which were prenatally exposed during the sensitive embryo-foetal period (between 3 and 16 weeks after conception, i.e. during organogenesis); genetic abnormalities, unlike developmental ones, are often transmissible to subsequent generations.

For the detection of developmental effects, anomalies of the central nervous system (Down's Syndrome, Microcephaly, severe mental defects etc.) are of particular interest (cf. Sect.3.1). The ability of monitoring systems for birth defects to detect new teratogenic risks is limited by sample sizes and the magnitude of the relative risk (see Annex D for a discussion of statistical power). The populations which were most exposed due to the Chernobyl release are only very incompletely monitored, and the very small frequency of the expected effects make it virtually certain that any teratogenic risk would remain undetectable (Sect.3.1 and 4.5).

A joint ad hoc evaluation of the frequency of central nervous system malformation in 18 regions in Europe has been carried out by a working group of the EUROCAT Central Registry of Birth Defects for the period since the reactor accident. It shows no increases in the frequency of malformations. The six classes of analysed anomalies were: neural tube defects, arhinencephaly, microcephaly and brain reduction, hydrocephaly, anophthalmos and microophthalmos, and congenital cataract (C5).

Mutations can be detected at the chromosomal or at the gene level. If radiation tends to create chromosomal deletions, translocations and inversions, rather than non-disjunctions which have been recorded but are rare (see Annex C), it would be necessary to distinguish between de novo and inherited mutations, the latter being in the majority.

At the gene level, sentinel phenotypes are indicators of germinal dominant gene mutations. Sentinel phenotypes include sentinel abnormalities which are recorded in congenital anomaly registries, with an expected prevalence rate of 2.6 per 10 000 births. A study of sentinel

anomalies would require specific standardisation throughout the registries and - with few exceptions - it would be difficult to assess their prevalence retroactively. Although the expected effect would be cumulative, and although it might be interesting to monitor trends over the next decade for possible increases in areas with highest exposure, one would be very unlikely to detect effects. To be detectable they would require doses which are orders of magnitude larger than those produced by the radioactive contamination in the European countries (Sect.4.4). Such doses occurred only in the near vicinity of the Chernobyl reactor.

5.1.2 Cancers

Tumour rates can be monitored either in terms of incidence or of mortality data. We shall describe here in detail data bases for measuring cancer incidence, although they exist only in some specialized registries, while age and site specific mortality data are available nationally in all countries of the EC.

Analyses of mortality data would be appropriate with regard to those cancer sites for which mortality is nearly equal to incidence (e.g. lung or stomach). For childhood cancers this is not the case and the situation has become even more complicated because there have been important advances in therapy, so that mortality rates have not paralleled incidence rates in recent years. Hence, incidence rates for childhood cancer may have stayed substantially unchanged, whereas mortality data can show reductions by factors up to 3. Delayed cancer deaths may complicate the data further.

For thyroid cancer which is rarely fatal there is an even more striking lack of connection between incidence and mortality data, and the problem is further complicated by the poorly defined diagnosis of thyroid cancers.

Incidence data recorded in the registries are subjected to a variety of checks for duplication, coding errors, and for completeness. The percentage of histologically confirmed cases is high. Hence assessment errors are minor.

Information on confounding variables, such as new health screening programmes or reorganisations of medical services, is provided by the registries. Age, sex, place of residence and detailed diagnoses are also routinely recorded for each case.

For the comparison of incidence rates in different areas numerous etiological factors - genetic and environmental - need to be taken into account. There are also some specific characteristics of the registration procedure which can influence the comparability of rates. These may differ, depending on the extent of medical facilities, and this has consequences both for the accuracy of diagnosis and the completeness of the records. For all registries in the European Community the notification of cases is voluntary and not compulsory by law. In addition, variations may occur due to the systems of codification and the checking procedures for misclassification.

With regard to adult cancers, any effect of the Chernobyl accident on time trends would not be expected for at least some years; the reason is that, even for leukaemias, the latencies are at least 2 years and usually considerably longer. Other main etiological factors of adult cancers, and especially leukaemias, include exposure to chemicals (solvents, pesticides and insecticides, antineoplastic drugs), and the time variations of such factors would need to be assessed. Hence it would be difficult to distinguish any effects due to the reactor accident from other fluctuations.

In view of the initial high contamination with I-131, thyroid cancer is a concern. However there are difficulties in the registration of thyroid lesions, and latency periods are long. A study of thyroid effects would, therefore, require specially designed surveys and could not be achieved through incidence registries. The Swedish study of patients who had been given substantial activities of I-131 for diagnostic and therapeutic purposes illustrates the difficulties. In spite of far higher doses and an average observation time of 25 years no enhanced rates have been found.

5.1.3 Childhood Cancers

The analysis of time trends needs to be restricted to the most sensitive sites and the most sensitive age groups for radiation induced cancers. These are primarily childhood leukaemias and, more generally, all childhood cancers (Sect.4.2).

A study of childhood cancer based on the registries listed in Annex C, Tab.C.4, would include areas with medium to high caesium contamination (Southern Germany, Northern Italy, Eastern France, Scotland and Wales) and areas of low contamination (Denmark, The Netherlands, Southern England and Spain). Shifts in the time trends in exposed areas could thus be compared to any shifts in less exposed areas.

All the population registries listed in Annex C, Tab.C.4, with two exceptions, were established at least five years before Chernobyl and one can thus assume that near completeness of registration has been attained. Clearly the longer the registry has been operating, the less artefacts will occur in the incidence data and their time dependences. Hence a good baseline estimation for areas ranging from high (Southern Germany and Northern Italy) to low exposure can be obtained. For registries covering only a small population, random fluctuations of a rare disease can be important and can mask any true changes of the mean frequencies.

Presuming a roughly similar time course of all childhood cancers one could pool them, to obtain better statistical resolution. Among the more highly contaminated regions in the EC - such as Northern Italy, Greece, or Southern Germany - there appears to exist only for Germany a childhood cancer registry suitable for the purposes which have been outlined here. This registry has a comprehensive data base, i.e. it contains nearly all cases (about 1 000 per year) (Annex C, Tab. C.4).

With an observation period of about 10 years and with the estimated excess relative risks (see Tab.B.3) due to the Chernobyl accident a study based on the total population of children in the Federal Republic of Germany (6 million) or a study of a regional subpopulation (60 000

to 600 000) could conceivably come close to establishing an effect with statistical significance (Annex D). Therefore, childhood cancer appears to be the only post-Chernobyl effect which might under the tentative assumptions be detectable.

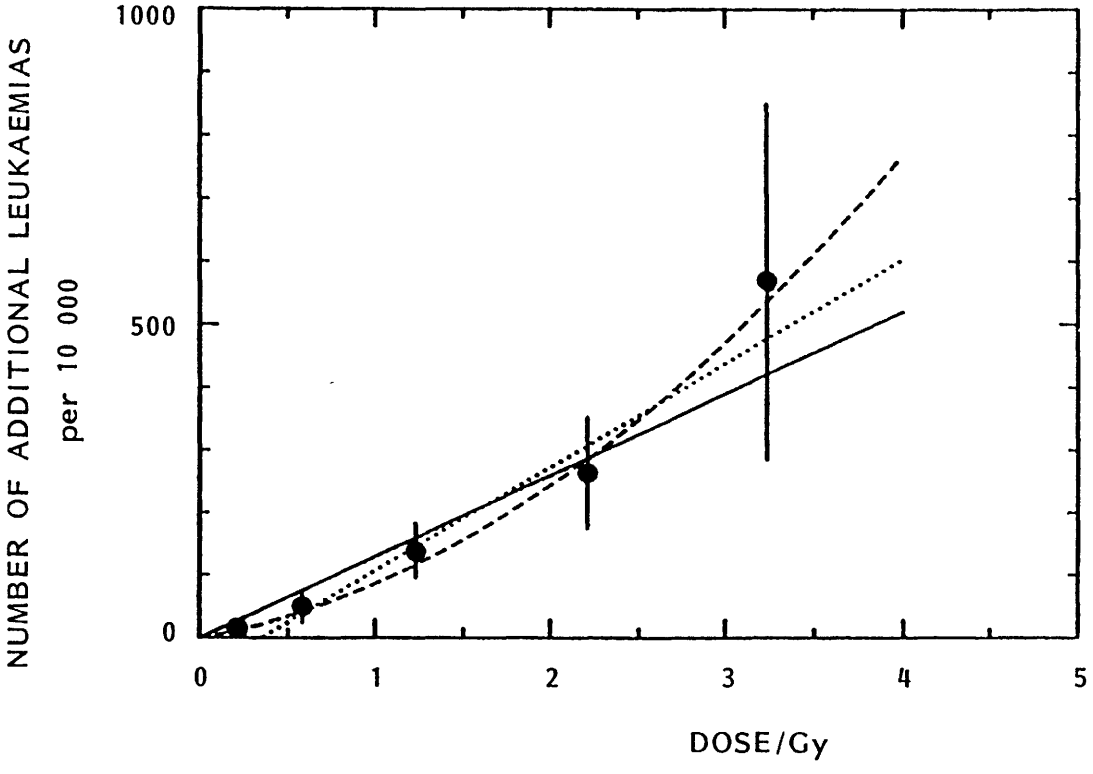

Fig. 1

Diagram of possible dependences of the increased leukaemia mortality on dose. Data are from the atomic bomb survivors of Hiroshima and Nagasaki (A20, cf. Fig. A.2.) for doses between 0.1 Gy and 3 Gy. The coefficient of variation for the plotted points ranges from about 25% to 50%. The linear extrapolation (solid line) is used for a conservative risk estimate at low doses. Radiobiological studies suggest that the actual dependences for sparsely ionizing radiations are linear-quadratic (broken line). There is no evidence for a dose dependence with threshold (dotted line).

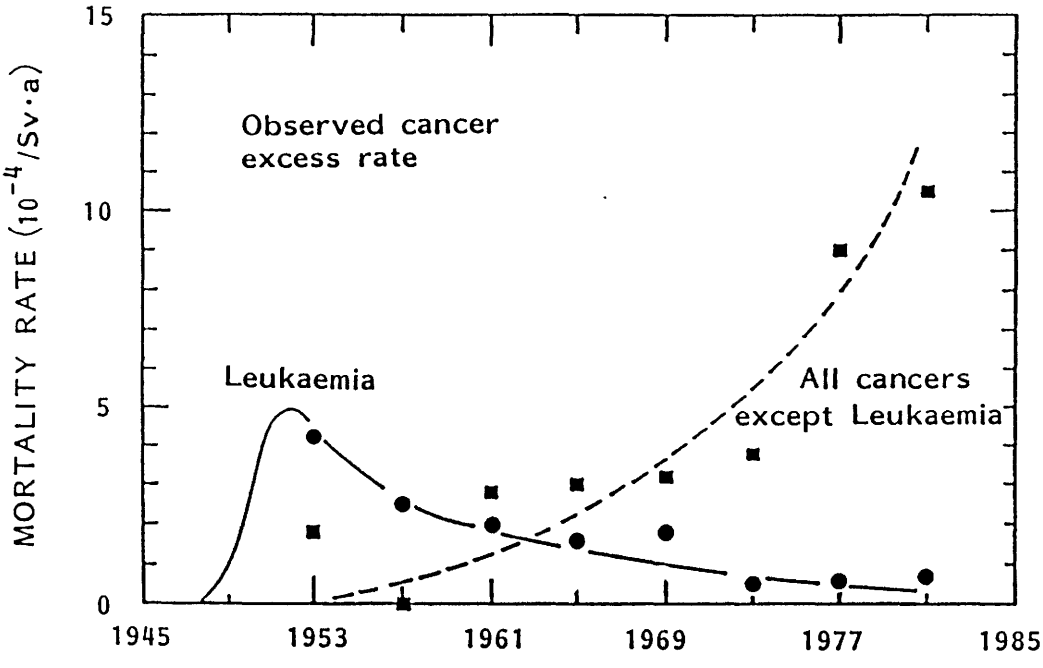

Fig. 2

Excess cancer mortality of the atomic bomb survivors. Each data point represents an observation period of 4 years. The interpolated curves are given merely for easier visualization of the diagram. The data contain a temporal shift of the age distribution of the collective and can, therefore, not provide risk estimates directly (diagram adapted from (A25)).

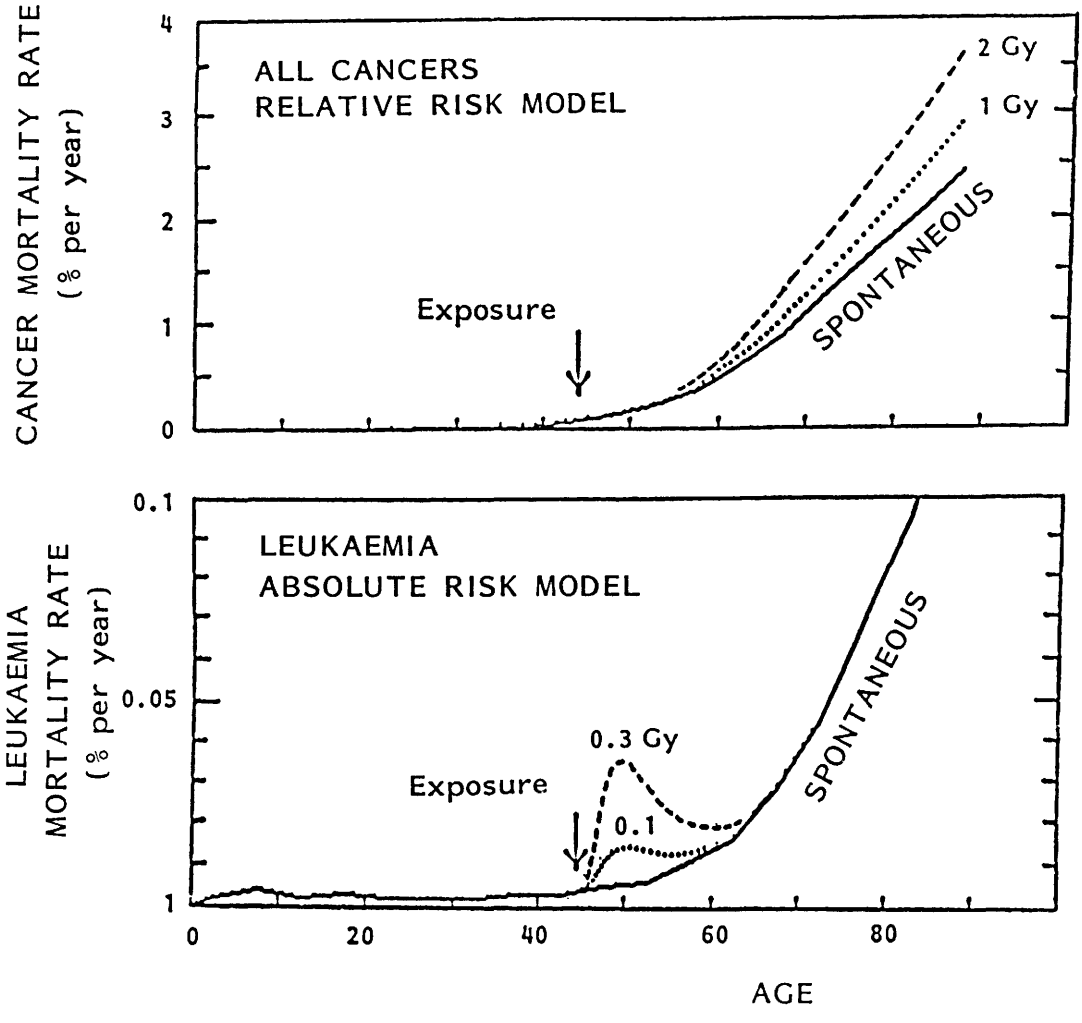

Fig. 3

Schematic diagram of cancer mortality according to the relative risk model and the absolute risk model. The solid curves give the average rates for the male population of the USA. The dotted and the dashed lines indicate the excess rates due to radiation exposure and correspond approximately to the new risk estimates.

Effect	Age at Exposure	Reference	Risk Factor 1/Gy	For Estimations in this Document 1/Gy
Mental Retardation	prenatal (8-15) weeks p.c.)	UNSCEAR[A 9]	0.4	0.4
Hereditary Damage		ICRP26 [A23]	0.004	
		UNSCEAR[A 9]	0.01	0.01
Cancer	working age	ICRP26 [A23]	0.0125	
	age averaged	BEIRIII[A24]	0.01 - 0.02	0.1
	age averaged	RERF [A20]	0.1	0.1
	<20a	RERF [A20]	0.15	0.15
Leukemia	working age	ICRP26 [A23]	0.002	
	age averaged	BEIRIII[A24]	0.002 - 0.004	
	age averaged	RERF [A20]	0.01	0.01
Childhood Cancer				
Leukaemia	prenatal	RERF [A20]	<0.03	
		OSCC [A16]	0.07	
All cancers	prenatal	OSCC[A16,A17]	0.2	0.1

Tab. 1 - Estimates of risk per Gray for various endpoints according to different data sources.

A N N E X E S

Annex A:

Sources of Knowledge on Risk Factors for Radiation Induced Late Effects

General remarks:

Immediately after the discovery of x-rays in 1895, there was a rapid development of medical applications, but soon there were also observations of harmful effects, such as x-ray induced skin damage. Only a few years later, the first skin cancers were observed, and not much later the first leukaemias occurred in radiologists.

In the subsequent decades, tragic experiences resulted from the uncontrolled use of radionuclides, and one of the most serious examples is the fate of the women who painted luminous dials with radium-226 and who incorporated large amounts of this long-lived alpha-emitter when they tipped the paint brushes with their lips. A large number of bone cancers and other cancers occurred among these women.

There was a similar lack of radiation protection for other radium workers, and there was failure to understand radiation risks when an alpha-emitting contrast medium, thorotrast, was widely used for angiography (the representation of blood vessels in x-ray images). There were also instances of medical misuses, such as the injection of high activities of the short-lived radium-224 for the intended treatment of ankylosing spondylitis (a degenerative disease of skeletal joints) and childhood bone tuberculosis. And there was, to name a further example, inadequate radiation protection in uranium mines or in other mines with high radon levels.

The resulting tragic experiences affected numerous individuals, but they have also become a source of knowledge on the risks for radiation carcinogenesis. Further important information has resulted from a variety of diagnostic or therapy-related applications of x-rays or radionuclides in medicine.

The most important data on the late effects of ionizing radiations have been obtained during the last four decades from the observation of the fate of the survivors of the atomic bombings in Hiroshima and Nagasaki. Beyond the enormous extent of destruction and the hundreds of thousands of deaths, immediately and in the days and weeks after the bombings, the surviving population remained subject to increased cancer rates that were seen and are still being seen in those exposed to higher doses.

The subsequent paragraphs contain more detailed information on specific sources of knowledge on risk factors for radiation induced late effects.

Synopsis of data sources:

Radiobiology and epidemiology are the two main sources of knowledge concerning radiation-induced late effects and, specifically, radiation-induced cancer. Radiobiological experiments at all levels from molecular systems to exposed animals are a basic but somewhat indirect source. The studies have led to important general conclusions and they have provided guidelines for comparisons and for extrapolations of more direct data. Actual risk estimates must, however, be based on epidemiological studies.

Epidemiological studies are the major source of information on risk factors. The information they provide is also indirect, because it is based on extrapolations from high doses to the low doses which are encountered in radiation protection.

The main human populations which have been exposed and were later subject to follow-up studies are listed below:

Occupational exposure:

- Dial painters who ingested large amounts of radium-226 containing material

- Uranium miners who were exposed to radon daughters, i.e. the α-emitting decay products of radon, in underground mines
- Workers in nuclear industries who were occupationally exposed to low dose-rate radiation

Patients who were treated by a variety of therapeutic medical procedures:

X-ray therapy of the
- spine and pelvis in adults with ankylosing spondylitis
- chest for enlarged thymus in infants
- scalp in children with ringworm (tinea capitis)
- neck and head for various lymphatic abnormalities
- skin for acne and hemangiomas
- breast in women with postpartum mastitis

Radium-224 therapy
- for tuberculosis and ankylosing spondylitis

Radioiodine therapy
- for ablation of the thyroid gland

Patients who were exposed due to diagnostic procedures:
- fluoroscopy for pneumothorax in women with tuberculosis
- thorotrast injections
- prenatal irradiation
- radioiodine examination of the thyroid

A-bomb survivors of Hiroshima and Nagasaki who represent the largest and most important exposed human collective.

The most relevant groups of these and related studies will be briefly considered here.

Dial painters

Between 1915 and 1930 hundreds of young women, especially in the USA, were employed to paint watch and instrument dials with fluorescent material containing radium-226. Pointing the brushes with their lips they incorporated very large amounts of the long-lived, α-emitting radium-226. Later, they developed grave radiation effects; many of these women died of bone sarcomas, or cancers of the mastoids and nasal sinuses. The doses were very high and various acute radiation damage has been described in the dial painters. A follow-up study published in 1986 (1) covers a period of more than 50 years. It includes about 2 000 US dial painters with known radium body contents. Out of these, about 500 had died up to that date, among them there were 60 deaths from bone sarcoma, sinus or mastoid carcinoma.

The dial painters represent the radiation-exposed group with the longest observation period; however, the data are of limited value for risk estimation. This is so because the exposure was due to densely ionizing radiation and because high activities were distributed inhomogeneously within the body. It was, therefore, difficult to obtain reliable dose or organ-dose estimates, and, more importantly, there was no adequate follow-up study, while most of the exposed were still alive. The probability that bone tumours or other radiation-induced malignancies were reported may have depended on the cumulative dose because the extent of damage determined the degree of medical supervision. The dose-response relationships inferred from these data are, therefore, somewhat uncertain.

Occupational exposure of nuclear industry workers

The lasting debates on cancer risks associated with exposures to low doses and low dose rates has prompted a number of studies of cancer incidence and mortality in workers in the nuclear industry. Together, these studies cover nearly 120 000 employees with a mean follow-up of about 20 years, mainly in the UK and the USA (2, 3, 4, 5). Generally the mortality rates from cancer and other causes of death in these study populations were below national average rates. No excess deaths from malignant diseases were revealed in comparison to the general

population. The low mortality rates are, at least partly, explained by the selection of healthy employees, by above average health care, and by other differences from a normal population in medical care and supervision, social conditions, social services and related factors.

For leukaemias the situation is more complex. Most studies do not reveal an increase of leukaemia rates, but some of the studies would appear by themselves to indicate increases (2, 4). None, however, attain statistical significance.

The total number of observed employees is high (and comparable to the number of A-bomb survivors). However, the number of deaths is small (5 to 15% in different studies); this reflects the age structure of these young working populations. The dose levels are also lower than those of the A-bomb survivor population. Both factors account for the fact that none of the studies of workers has yet provided actual numerical risk estimates, and it is, therefore, apparent that the studies of workers need to be continued.

A variety of partly contradictory studies on cancer risk due to occupational exposure have generated much controversy with respect to dose-effect relations, statistical procedures, risk estimates, etc.

It is beyond the scope of this document to review in detail the existing publications on this topic or to give a resumé of their quality or validity. Some, but not all, of the discrepancies between the investigations appear to vanish as more information accumulates. However, even in their entirety, the worker data cannot provide actual risk estimates. They can merely determine ranges of possible values, and these ranges are equally consistent with no effects and with risks substantially larger than the estimates considered in this document. The epidemiological studies on nuclear industry workers are, therefore, not directly relevant as sources of risk estimates for the present study. However, they are clearly relevant with regard to their methodological aspects. Any epidemiological investigation at low doses would need to utilize the methods that have been developed in these studies.

X-ray therapy for ankylosing spondylitis

In Britain, during the period from 1935 to 1955 a commonly utilized treatment of patients with ankylosing spondylitis was x-irradiation (in several courses) to the spine and pelvis. The patients reported substantial pain mitigation or even improvements of their condition. Subsequently, however, it appeared that the treatment led to an increased risk of cancers, especially of leukaemia. A subsequent extensive epidemiologic investigation, published in 1957 (6), included more than 13 000 patients with doses to the spinal marrow of about 5Gy per irradiation course. At the end of the total follow-up in 1970 (7, 8), more than 14 000 patients were included; of these, about 6000 with an average follow-up of 16 years and only a single course of treatment were selected for further evaluations.

The principal malignancies for which quantitative risk estimates could be obtained were leukaemias. The overall observed/expected case ratio was about 5. After a latency of about 2 years, the rate reached a maximum at 3 to 5 years after irradiation; subsequently, about 20 years after the treatment, it declined toward the control levels. A similar time course was seen in the A-bomb survivors (see below).

The marrow doses ranged from 0.5 to 7 Gy with a mean of about 2.5 Gy and a risk factor of about 0.01/Gy was estimated. The risk estimates for solid cancers are in general agreement, although somewhat smaller, than those derived from the data on the atomic bomb survivors (7, 8, 9).

X-ray exposures of children with tinea capitis

Between 1948 and 1960 children were irradiated in Israel with x-rays for ringworm of the scalp (tinea capitis). The treatments were applied to the head, specifically the scalp. The final cohort study (published in 1984) (10) included more than 10 000 persons with an average follow-up of about 20 years. An estimated mean dose to the thyroid of 100 mGy led to a five-fold increase in malignant thyroid tumours. The absolute risk factor for thyroid cancer

incidence due to irradiation in childhood was estimated to be about 0.03/Gy. Since only one in ten cases is lethal, this corresponds to a mortality risk factor of 0.003/Gy. Children of age between 0 and 5 years appear to be about 1.5 times more sensitive than older ones (9, 10).

Radium-224 therapy

In the years after World War II high activities of the short lived α-emitting radioisotope radium-224 were injected for the intended treatment of ankylosing spondylitis and of bone tuberculosis in children and adults in a German clinic. This treatment has given rise to bone sarcomas, and to various noncancerous lesions. In 1952 a follow-up study of the German radium-224 patients was begun. The study population contained 899 children and adults who received intravenous injections of radium-224, resulting in skeletal doses from 0.05 to 50 Gy (11, 12). Bone sarcomas were reported in 53 patients (compared to only 0.2 cases expected on the basis of population statistics). All of these bone cancers must, therefore, be regarded as radiation induced. A detailed statistical analysis revealed a linear-quadratic dose dependence which did not appear to vary with age at exposure or sex. In contrast to most other solid tumours, the excess risk for bone sarcomas does not run parallel to the age-specific spontaneous rates. Instead, the absolute risk model is applicable, and the course of the bone-sarcoma rate can be approximated by a temporal wave similar to that observed with leukaemias. After a latency of 2 to 4 years, there is a maximum osteosarcoma rate: at about 10 years the rates begin to decline, and, after 20 to 30 years, the increment has virtually disappeared.

The derived lifetime risk factor for bone sarcoma is of the order of 0.01/Gy of α-rays (12); this result cannot, however, be linked to the possible effect of gamma rays.

Iodine-131 in nuclear medicine

For diagnosis of thyroid diseases large numbers of patients were given iodine-131 during the period of 1951 and 1969. A Swedish follow-up study (the most recent results were published in

1988 and 1989 (13, 14)), covers over 35000 persons who were followed for an average period of 20 years after administration of the radionuclide. The population-average dose was 0.5 Gy. The study has not revealed significantly increased thyroid cancer rates among the patients who received diagnostic applications of I-131. However, there has been no fully comparable control group in this study, and it is, furthermore, possible that the diagnostic procedure had some screening effect on thyroid cancers which would otherwise have been found later.

Thorotrast injections

The x-ray contrast medium Thorotrast, an α-emitting thorium-232 dioxide preparation, was introduced for angiography in 1929 and - in spite of published warnings - it was used until about 1950. Its application has caused, apart from a variety of noncancerous effects, various tumours, predominantly liver tumours. The major investigation is the German Thorotrast study; it started in 1967 and it has now reached a total follow-up of about 45 years (15). From more than 5000 patients initially recorded about 2 000 were selected for further investigations. 18% of these developed liver cancer, compared to only 0.14% expected. The mean liver dose rate due to Thorotrast injection was estimated to about 0.02 Gy of α-rays per year for the typical injected amount of the order of 40 ml.

Similar but somewhat less extensive studies have been performed in Denmark, Portugal and Japan, however, their results are less fully documented.

Prenatal exposures from x-ray examinations during pregnancy

Extended case-control investigations of childhood cancer deaths, conducted in Britain between 1953 and 1979 revealed a marked correlation of childhood cancer and prenatal x-ray exposure (16, 17). Analyses based on data from the 'Oxford Survey of Childhood Cancer' include nearly 15 000 geographically-matched and birth-date-matched case-control pairs. They represent the largest studies of prenatal x-ray exposure and childhood cancer. In these studies it was concluded that about 7 to 8% of all childhood cancers were caused by x-ray examinations. For

children with cancer who had been diagnostically exposed in utero, an attributability of 50% was inferred, and a risk factor for fatal childhood cancer of about 0.2/Gy has been stated.

A similar result comes from a case-control study in 32000 twins born in Connecticut from 1930 to 1969; among these 31 cases of cancer were identified (18). Twins who developed leukaemia or other childhood cancer were twice as likely to have been exposed to x-rays in utero than twins who were free of disease. The derived relative risks of 1.6 and 3.2 for leukaemia and other cancers, respectively, at average x-ray doses of 10 mGy correspond to risk factors of 0.03/Gy for leukaemia and 0.2/Gy for other cancers. As noted earlier, such high risk estimates do not fit the data from Hiroshima and Nagasaki.

Follow-up of the atomic-bomb survivors

Observations of the fate of the survivors from Hiroshima and Nagasaki have lasted four decades and they will need to be continued for several more decades. Essential findings have been published in a series of recent documents from the Radiation Effects Research Foundation (RERF) (e.g., 19, 20, 21, 22).

Risk estimates by the United Nations Scientific Committee on the Effects of Atomic Radiations (UNSCEAR) and the International Commission for Radiological Protection (ICRP) were based on data up to the year 1975 and on a dosimetry for the atomic bombs survivors that was subsequently recognized to be faulty. The old dosimetry implied that a substantial, if not the major, part of the radiation induced cancers in Hiroshima was due to densely ionizing neutron radiation, while neutrons played no major role in Nagasaki. This appeared to be consistent with an apparently linear dose dependence for leukaemias, for all cancers, and also for chromosome aberrations in Hiroshima, and with threshold-like dose-response relationships in Nagasaki. It was thought that at low doses the densely ionizing radiation remains effective, while the effectiveness of the sparsely ionizing gamma radiation is considerably reduced. In line with this assumption, risk estimates were obtained which included, for gamma rays, the assumption of a substantial reduction factor for the extrapolation from high to low doses.

The inferred risk factor for cancer mortality averaged over all ages in the exposed population was 0.01 to 0.02 per Gy (Tab.1, main text). For leukaemia the risk estimate was 0.002 to 0.004 per Gy (23, 24).

The revision of the dosimetry was necessitated by the conclusion that the dosimetric computations for neutrons were erroneous. A re-analysis, performed under broad international cooperation, has shown that the contribution of the neutrons to the total dose was minor, even in Hiroshima. This would seem to lead to increased risk estimates, since a substantial part of the effects in Hiroshima was formerly ascribed to neutrons but would now be assigned to the gamma rays. The actual analysis showed that the effects of the change in dosimetry are less marked than one would expect (20, 21, 22), because the revision of the dosimetry has led to higher estimated gamma-ray doses, particularly in the range of low doses. A number of further, largely compensating changes had the overall effect that one obtains virtually the same excess-risk estimates for solid cancers at doses of one or several Gy, regardless of whether one employs the old or the new dosimetry system (Tab.A.1). For leukaemias the situation is different, and there is an approximately 50% increase of the risk estimates due to the changed dosimetry; the technical reason is related to the fact that the bone marrow, as an organ at fairly shallow depth, is differently affected by changes in gamma-ray penetration through the body than other, deep-lying organs.

In spite of the lack, or the small magnitude, of changes due to the revised dosimetry, there is now a substantial increase in the risk estimate for cancer mortality which may altogether amount to a factor up to 10 (Tab.A.1). There are a number of reasons. One of the most important aspects is that there is now less evidence of a decreased slope of the dose-effect relation at low doses. Since the seemingly linear relationship in Hiroshima cannot be ascribed to neutrons, there is less justification for employing a dose-reduction factor for gamma rays as it was introduced by UNSCEAR and employed by ICRP (23), to account for an assumed smaller effect at lower doses and lower dose rates. In the most recent assessment of UNSCEAR (9) possible dose reduction factors between 2 and 10 were suggested, but these tentative values were based on general conclusions of radiobiology, not on direct

epidemiological evidence. The recent studies from RERF (20, 21, 22) have, therefore, quoted risk estimates without this factor.

A further reason for increased risk estimates is the continuation of the observations since 1975 which have in almost all age cohorts shown a persistence of the increased relative risks for solid tumours. This has increased the absolute number of cancer deaths approximately by a factor of 2 at a specified dose, and a further increase of about a factor 2 has resulted from the hypothetical lifetime projection of risk, based on the relative risk model for those who are still alive (Tab.A.1).

Generally it was concluded that the data for solid tumours follow the relative risk model. In this model irradiation causes, as stated, an increase of tumour rates that begins five to ten years after exposure. The increases are taken to persist and to remain proportional to the age-specific rates. The proportionality factors depend on dose and also on age at exposure. They tend to be higher for those exposed at younger ages.

Contrary to observations on the UK ankylosing spondylitis patients (7, 8), and also to observations on the uranium miners, there has been no indication of decreasing relative risks several decades after the exposure for the survivors of the atomic bombings. Accordingly, Preston and Pierce (20) have, in a recent analysis after the dosimetry revision, derived risk estimates which are based on extrapolations throughout life.

Fig.A.1 compares the ICRP risk estimate (lower broken line) from data up to 1975 with the most recent risk estimate (upper broken line) that is based on the new dosimetry and on observations until 1985. The latter relationship is primarily related to the left ordinate which gives relative risk factors and it corresponds to the initial slope of the explicit dose dependence that is given as a solid line. The data represent all nonleukaemia tumours and all age cohorts combined. Assuming a total lifetime cancer mortality of 20%, one deduces the absolute lifetime risk estimates that are provided on the right ordinate. The direct comparison of the earlier estimates with the more recent analysis suggests an increase in risk estimates by

a factor of up to 10. This difference is the product of the previously mentioned individual factors, for which rough values are listed in Tab.A.1.

For leukaemias there has been a somewhat smaller change in risk factors, because there have been few excess cases since 1975; the waves of radiation induced cases had largely disappeared even before 1975. The continued observation has, therefore, not contributed to increased risk estimates. Since there is no need for further projection of risks, the estimates are more certain, except for the question of whether a dose reduction factor applies. However, as stated, there has been an increase by about a factor of 1.5 due to the dosimetry revision. Together with an abandonment of the dose-reduction factor, this leads to an increase of the risk estimates by a factor of about 5. Fig.A.2 compares the earlier ICRP-estimates for leukaemia with the new results.

Figs.A.1 and A.2 are accompanied by tables of scaling factors for three age groups and the two sexes. Applying these scaling factors, one obtains in Tab.A.2 the values of new risk estimates without assumed reduction factors for low doses and low dose rates.

In summary, there is only a minor increase of the risk estimates due to the changed dosimetry. The major change is an indirect consequence of the revised dosimetry system; the abandonment of an assumed reduction factor for low doses reflects the disappearance of direct evidence for a nonlinear dose dependence; for both cities the data are now consistent with proportionality to dose. A further change is due to the extension of the observations to include not only tumours that have occurred since 1975 but also those that are projected to occur in the future, i.e. more than four decades after the exposure in the ageing cohorts. The mounting evidence of enhanced sensitivity of those exposed at younger ages is also of importance, but it will be several more decades until definitive data are available about the frequency at higher ages of solid tumours in those who were exposed as children.

Hereditary disorders in the children of the exposed atomic bomb survivors have been intensively studied and will continue to be studied, but no statistically significant increases

have, as yet, been identified. Surveys of other groups of radiation-exposed persons, too, led to the conclusion that the association between radiation and hereditary damage is too weak to be statistically ascertained. The statistical uncertainty of the data is, however, sufficiently large that there is no inconsistency with risk factors deduced from radiation studies with mice. From such experiments one estimates that hereditary disorders are doubled by doses of 0.5 to 2Gy (9). With a spontaneous rate for naturally occurring hereditary effects of a few per cent, a genetic risk factor of about 0.01/Gy would be estimated. Substantially higher risk factors would be inconsistent with the absence of significant increases of hereditary defects in Hiroshima and Nagasaki.

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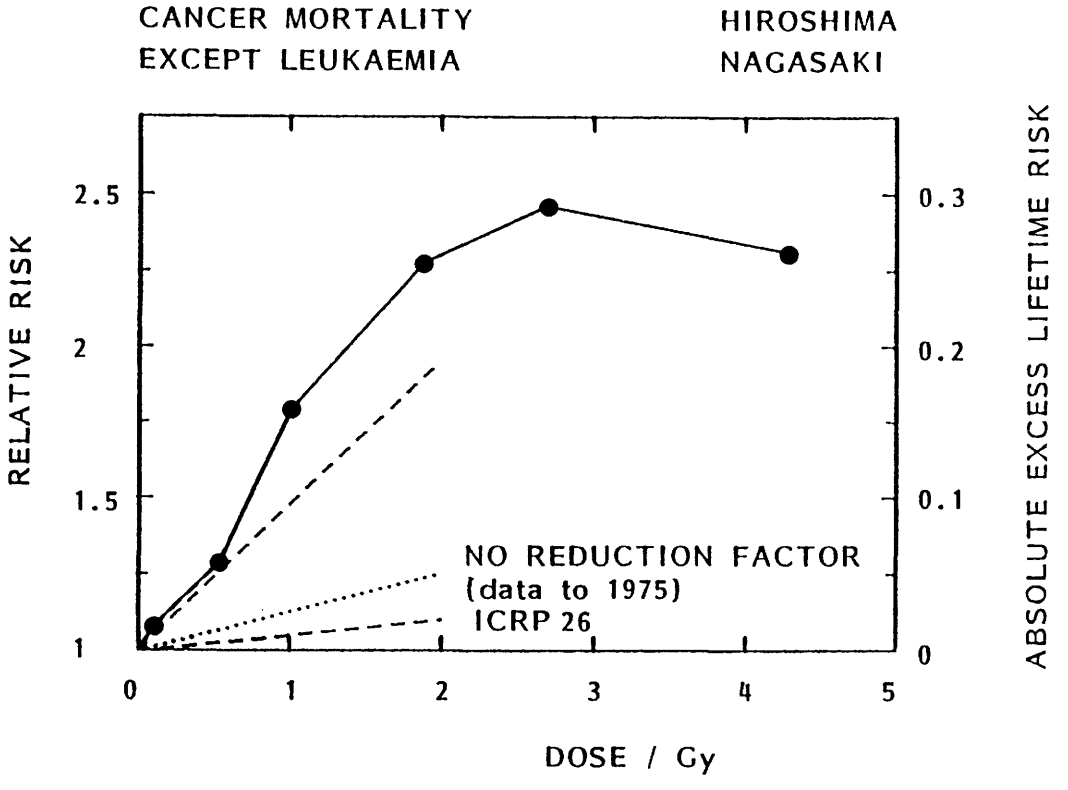

Fig.A.1 Dose dependence of the relative and the absolute risk of cancer mortality for a population of all ages according to the results of RERF (20, 21). The upper dashed line represents the excess lifetime risk. For comparison the risk estimations of ICRP (23) with and without a reduction factor are indicated. The results of the new analyses refer to the study population of all atomic bomb survivors.

The table below contains the adjustment factors for different age groups. The ICRP estimates were based on data up to 1975 and relate to an adult working-age population.

Age at Radiation Exposure	< 20	20-35	> 35
Females	2.16	1.29	0.71
Males	0.97	0.58	0.32

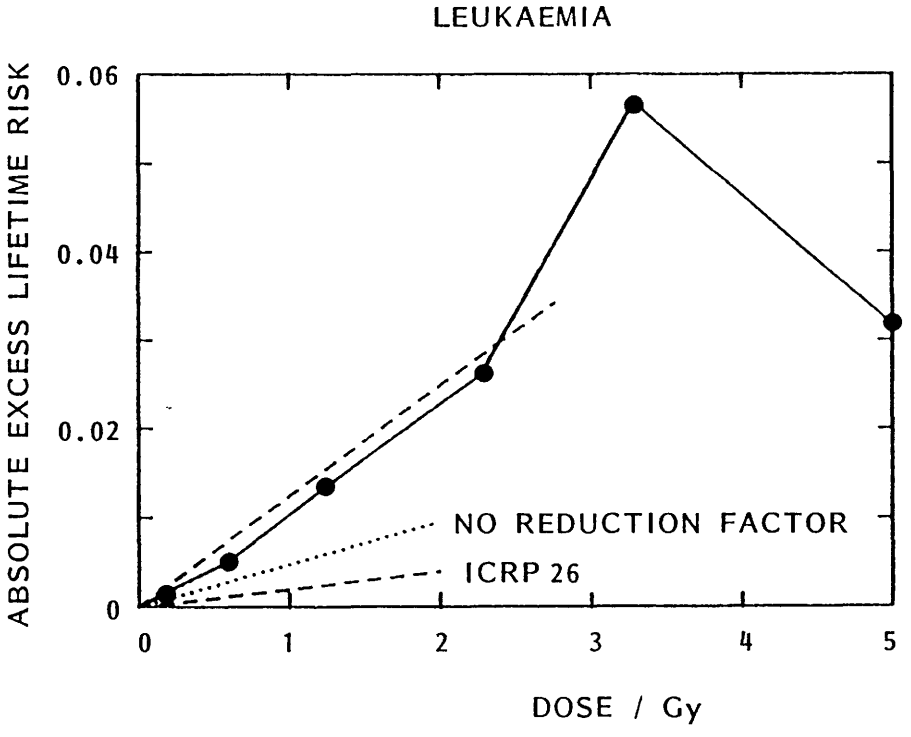

Fig.A.2 Dose dependence of the absolute risk of leukaemia mortality according to the results of RERF (20, 21) under the assumption of a mean time at risk of 32 years. The dashed line represent the mean lifetime risk. For comparison the risk estimates of ICRP (23) with and without a reduction factor are indicated. The results of the new analyses refer to the collective of all atomic bomb survivors. The table contains the adjustment factors for different age groups. The ICRP estimates were based on data up to 1975 and relate to an adult working-age population.

Age at Radiation Exposure	< 20	20-35	> 35
Females	0.57	0.63	0.88
Males	1.01	1.46	1.45

Factors between the old and the new risk estimates.

	Leukaemia	Solid tumors
- Change in dosimetry	1.5	1.0
- Inclusion of younger cohorts	1.2	2.0
- Extrapolation throughout life (depending on age of cohort)	1.0	2.0
- Omission of a reduction factor for small doses and small dose rates	2.5	2.5
Resulting total factor:	4 ... 5	10.0

Tab.A.1 Factors of increase from the previous risk estimates of ICRP26 (23) for a working-age population to the risk estimates for an age-averaged population considered in the present study.

	Additional Risk per Gray					
	Solid Cancers				Leukaemia	
	relative risk		absolute excess-life-time risk		absolute risk	
NEW DATA Age at Exposure	F	M	F	M	F	M
< 20	1.08	0.49	0.19	0.11	0.006	0.01
20 to 35	0.65	0.29	0.12	0.07	0.006	0.014
> 35	0.36	0.16	0.06	0.03	0.009	0.015
Average over Age and Sex	0.5		0.1		0.1	
BEIR III (24)	0.1		0.02		0.004	
ICRP26 (23)	0.05		0.01		0.002	

F: Females

M: Males

Tab.A.2 Estimated risk factors for radiation-induced cancer mortality. The estimates of BEIR refer to higher doses; the estimates of ICRP refer to the age distribution of an adult population. The new data are based on recent results from RERF (20, 21).

Annex B:

Exposures to Radiation from Chernobyl

An unexpected feature of the release of radioactive material from the Chernobyl reactor was not only its magnitude but also the subsequent widespread distribution throughout almost all parts of the northern hemisphere, and mainly across Europe. The patterns of deposition varied according to the meteorological conditions and wind regimes during the period of release. The radioactive cloud contained numerous different fission products and actinides and, traces of all of them were detected in most countries, but only comparatively few nuclides were of radiological concern. The three most relevant radioisotopes were I-131 and the two caesium isotopes, Cs 134 and Cs 137.

After the accident had become known, most countries started or extended monitoring programmes for radioactive contamination. The data have been utilized by a number of national and international committees, to carry out assessments of the radiological impact of the contamination. The data reported in this document are based mainly on reports by the Organisation for Economic Co-operation and Development (OECD) (1), by a task group of the British National Radiological Protection Board (NRPB) for the Commission of the European Community (CEC) (2), by the German Bundesgesundheitsamt (BGA) for the World Health Organisation (WHO) (3), and by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (5). They summarize rough estimates of contamination levels and average doses in member states of the European Community.

Fig.B.1 gives the measured cumulative I-131 and Cs-137 depositions in Europe (3). The levels of contamination vary widely and cover ranges from hardly detectable to several hundreds of kBq/m^2.

The main pathways of exposure have been direct gamma-irradiation from the radioactive cloud, inhalation of activity in the cloud, gamma irradiation from activity deposited on the

ground, and irradiation from ingestion of contaminated food. The conversion from the specific activity in the atmosphere in Bq/m^3 (inhalation), the deposition on the ground in Bq/m^2 (external exposure), or the concentration in food in Bq/kg (ingestion) to organ doses or to whole-body doses is performed through dose conversion factors, and these are specific for the nuclides, for the geometries of distribution, for the biological half lives and for a number of other parameters. The determination of the conversion factors is not a topic of this report; it is dealt with elsewhere (e.g. (3)).

Ingestion (resulting in internal exposure) and external gamma irradiation from deposited material (resulting in external exposure) were the dominant pathways. The relative contribution of these two pathways to the total exposure varied considerably between the different countries. The overall average of the dose in the first year among the population of the member states of the European Community was about 0.2 mGy which resulted in a collective dose of about 60 000 personGy (EC population 320 million).

The reports from OECD and NRPB include certain peak doses which have been estimated in the various countries. The corresponding subpopulations in the higher contaminated regions (termed critical groups) would be the likely target collectives for conceivable epidemiological investigations.

Fig.B.2 shows the distribution of average doses in the first year after the accident in some countries of the European Community. As indicated in the insets of the figure, values for the entire populations are given in terms of the external, internal, and total dose for children, and for critical groups (these groups being partly identical). The most highly contaminated regions with maximal doses are Northern Italy, Austria, Southern Germany, and Greece. In these countries average doses between 0.5 mGy and 1 mGy have been estimated for adults, and 1 mGy to 3 mGy for critical groups.

Tab.B.1 gives the dose estimates from OECD, NRPB, BGA, and UNSCEAR in more detail. The doses are given in terms of whole-body doses that are predominantly due to external and

internal caesium exposure. The values in Fig.B.2 are rough averages of the estimates in Tab.B.1.

When different radionuclides are incorporated they are distributed between the various organs according to their chemical properties. Thus, there can be widely different radiation exposures to different organs by different nuclides; but, in principle, the doses to each organ can be computed or at least estimated in terms of average metabolic parameters.

Organ doses, with the exception of those for the thyroid, need not be considered separately in this document. The bone marrow dose (relevant for leukaemia risk estimation) can for the present purpose be set equal to the whole-body dose by caesium.

Because of the initial predominance of iodine-131 in the contamination and its concentration in the thyroid gland after intake, thyroid doses have been evaluated separately.

The thyroid doses were mainly due to ingestion (internal exposure) and have been accumulated within a few weeks after the accident. Comparing these doses to the thyroid with the whole body exposures due to caesium one concludes that they may contribute about 10% of the total cancer risk due to the radioactive contamination.

The total collective thyroid dose to the EC population is estimated to be about 200 000 personGy (2/3 being contributed by Italy and Germany). The average adult and infant thyroid doses in the first year are given in Tab.B.2. In adults the highest average doses were received in Greece, Italy and the Federal Republic of Germany (several mGy). Infants can have received doses up to several 10 mGy. The contribution of the most exposed subpopulations (about 1% of the EC populations) in North Italy and South Germany to the total collective thyroid dose may be about 10% (i.e. 20 000 personGy).

The estimates from OECD, NRPB, BGA, and UNSCEAR show some deviations but agree qualitatively in the most relevant aspects. OECD gives the highest individual whole-body dose

of 2.9 mGy for a critical group in North Italy. This group, however, remains hypothetical and, accordingly, neither the size nor the collective dose of this group is stated. For the total population of Italy the collective dose is estimated to about 25 000 personGy and for infants (first year of life) about 350 personGy.

Collective doses similar to those in Italy occurred in the Federal Republic of Germany (total: about 20 000 personGy; infants: about 300 to 400 personGy), whereas for most other countries the collective doses are substantially less, namely of the order of 100 personGy and less. Critical groups may be infants or foetuses exposed in utero; they have enhanced sensitivity and have, partly, also incurred increased doses. Childhood cancer is considered as the most sensitive indicator, and one could, therefore, make use of childhood tumour registries, and particularly the well established one in Germany (see Sect.5.1.3 and Annex C).

An adequate choice of regions with high contamination and high average doses could yield values of up to 1.5 mGy for infants in the southeastern parts of Bavaria. Tab.B.3 summarizes the most relevant data for subpopulations and subregions of the Federal Republic of Germany; the values are based on data from Tab.B.1 and on information given by the German Radiation Protection Board (SSK) (4). The essential conclusion is, although very tentative, that for a subpopulation of 60000 infants born in 1987 (roughly those prenatally exposed) in the southeast of Bavaria there might be an increase of 9 cases of childhood cancer in the next 10 years (compared to 60 cases expected, background rate). This would correspond to an excess relative risk of about 15% which could be at the lower limit of detectability (Sect.4.2). If most of the excess cases appeared within the next 5years (say about 5 or 6 cases) a reduced observation time could be sufficient. The background risk for childhood cancer between age 0 and 5 years is about $5 \cdot 10^{-4}$, corresponding to 30 expected cases. This would result in an excess relative risk of about 15 to 20%.

References (Annex B)

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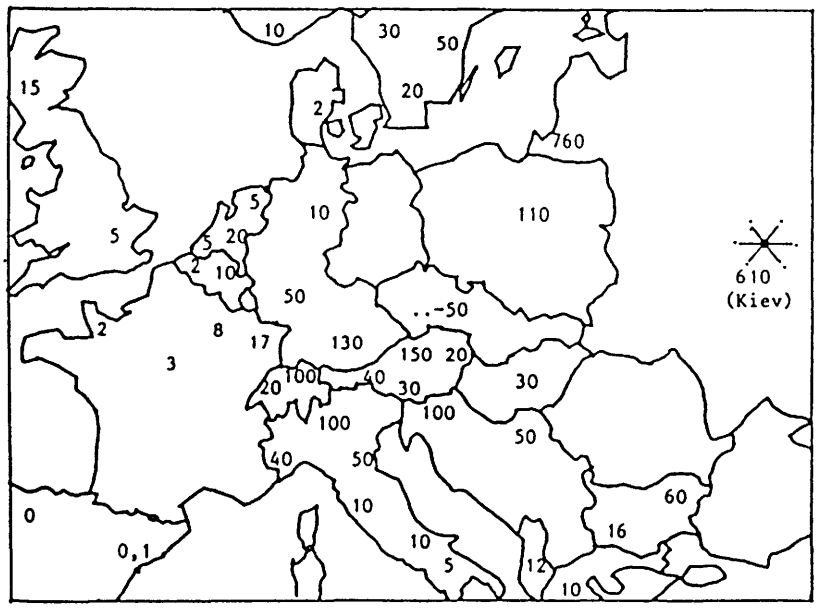
a)



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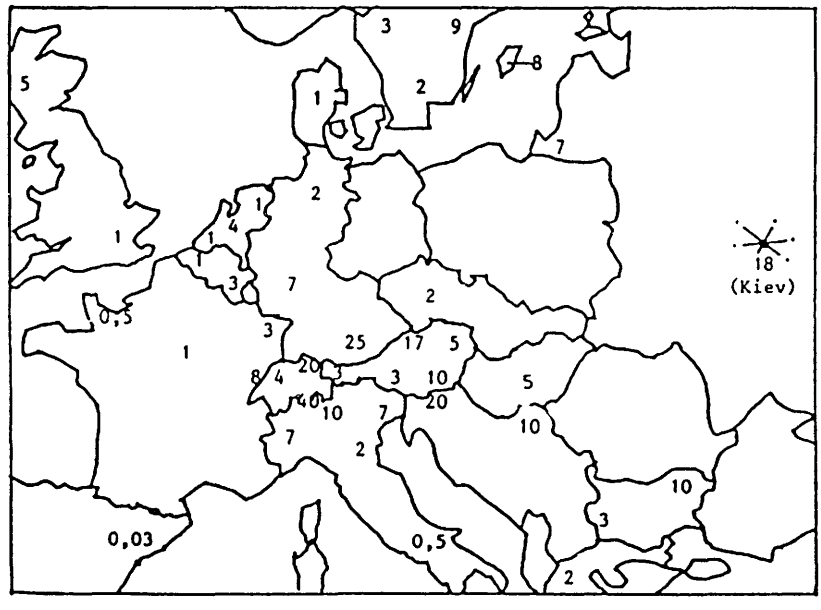

Fig.B.1 Measured cumulated I-131 (a) and Cs-137 (b) activitiy depositions per unit area (the unit is kBq/m^2) (3).

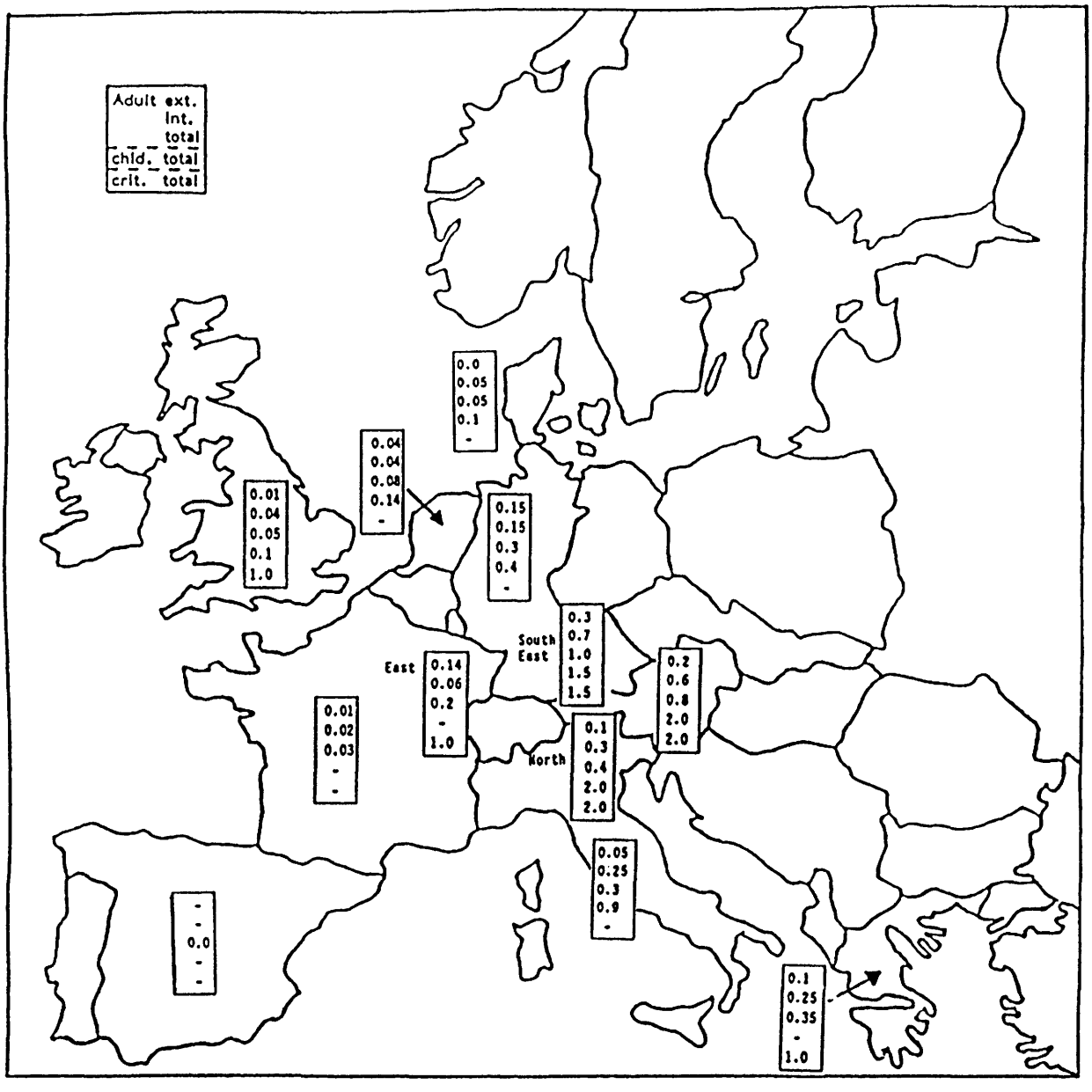

Fig.B.2 Average doses (in mGy) in the first year due to the accident. External, internal and total doses are given for adults. In addition, the averages of the total doses are given for children and for socalled critical groups.

	average individual dose/mGy				contribution from external radiation /%			critical group individual dose/mGy			children individual dose/mGy			infants collective dose/persGy		
	OECD	NRPB	BGA	UNSCEAR	OECD	NRPB	BGA	OECD	NRPB	BGA	OECD	NRPB	BGA	OECD	NRPB	BGA
Belgium	0.04	0.04		0.04	46			0.23	0.4		0.1			20	40	
Denmark	0.02	0.05	0.07	0.03	7		3	0.07	0.4		0.05		0.1	5	20	5
France	0.02	0.04	0.03	0.06	30		30				0.05			60		
" East		0.11	0.25					0.2	0.9							
FR Germany	0.27	0.14		0.13	47			1.1			0.6			350		
" North		0.04	0.4				20		0.4				0.1		200	50
" South		0.34	1.0				25		1.7				1.5		170	160
Greece	0.33	0.27		0.6	7			0.4	1.2		0.63	1.2		45		
Ireland	0.1	0.09		0.12	14			0.33	0.47		0.03			50		
Italy	0.44	0.15	0.2	0.3	17			2.9			0.70	0.9	0.9	270		500
" North		0.18	0.5				30		1.0				1.8			
Luxembourg	0.1	0.05		0.1	29			0.11	0.35		0.13			0.5	2	
Netherlands	0.06	0.06		0.06	53		50	0.14	0.34		0.12		0.2	40		30
Portugal	0.01	0.0			0			0.03	0.0		0.02			2	0.5	
Spain		0.0						0.06								
U.K.	0.03	0.03	0.04	0.03	14		20	1.1	0.8		0.1	0.8	0.1	110		50
Austria	0.6			0.09	19		20	1.1			0.6		2.2	40		170
Sweden	0.2				47			2.3			0.2			30		
Total EC	0.16	0.15			30									960		

Tab.B.1 Whole-body doses in the first year after the Chernobyl accident.

	Entire population thyroid dose / mGy	Infants thyroid dose / mGy
Belgium	0.2	2 - 10
Denmark	0.1	
France	0.2	1 - 10
FR Germany	0.5 - 1.0	5 - 10
South	1.0 - 3.0	10 - 30
Greece	1.0 - 2.0	10 - 20
Ireland	0.3	10
Italy	1.0 - 2.0	4.0
North	1.0 - 2.0	5.0
Luxembourg	0.3	2 - 10
Netherlands	0.4	1 - 5
Portugal	0.0	0.1
Spain	0.0	0.1
United Kingdom	0.2	1 - 10

Tab.B.2 Estimated thyroid doses for the entire population and for infants in the first year after the Chernobyl accident.

	Population	Av. ind. dose mGy	Collective dose persGy	Risk factor 1/Gy	Spontaneous cancer risk	Additional cancer risk	Excess Relative Risk ERR
Total population	$60\text{-}10^6$	0.3	18,000	0.1	0.2	$3\text{-}10^{-5}$	0.00015
Children (0 to 10 years)	$6\text{-}10^6$	0.4	2,400	0.15	0.2	$6\text{-}10^{-5}$	0.0003
Infants	600,000	0.6	360	0.1	0.001	$6\text{-}10^{-5}$	0.06
Infants (Bavaria)	110,000	1.2	130	0.1	0.001	$1.2\text{-}10^{-4}$	0.12
Infants (South-east Bavaria)	60,000	1.5	90	0.1	0.001	$1.5\text{-}10^{-4}$	0.15

Table B.3 Estimates for cancer risk in the Federal Republic of Germany due to exposures in the first year after the accident. The average doses are taken from Tab. B.1 and Fig. B.2; the risk factors are taken from Tab.1 (main text). The risks are projected through life for the entire population and for the subgroup of children. For the subgroup of infants the reference time is taken to 10 years. All infants (up to age 1) are considered who were born in 1987. The spontaneous childhood cancer rate, r_o is assumed to be 10^{-4}/a.

Annex C:

Registries of Congenital Malformations and Tumours

Congenital malformations

Registries of congenital malformations and of chromosomal disorders have been set up in most EC countries. Two coordinating bodies for these registries have been operating: the International Clearing House for Birth Defects Monitoring Systems (Int.Cl.) and Eurocat. The Int.Cl. was founded in 1974 and currently it coordinates data from 24 monitoring systems all over the world. Within the EC, a Concerted Action on Birth Defects was initiated in 1979 with the creation of Eurocat. During the first 5 years, registries were created and the standardization of data collection was tested.

These two coordinating systems function differently: Eurocat centralises all the data from the registries and carries out the analyses; the Int.Cl., in view of its world coverage, relies on registry leaders to provide the necessary information and to perform validation checks.

Tab.C.1 lists EC registries on birth defects and chromosomal anomalies together with their affiliation and some general characteristics (1, 2). Registries operate in eight of the 12 EC countries, monitoring in total about 1.5 million births. The time lag of surveillance varies between registries from 1 week up to 1 year. National centralized registries exist in only two countries, Denmark and Great Britain. Note that there are currently no operative registries in Greece or Southern Germany, and that the registry of Emilia Romagna covers only a minor part of Northern Italy. On the other hand, these 3 regions have had the highest level of caesium deposits.

Tab.C.2 lists the congenital anomalies recorded by Eurocat together with their prevalence (3); those which appear also in the classification by Int.Cl. are underlined. The anomalies which

are detailed per class are those for which defining criteria are most rigorous and least subject to misdiagnosis.

During the past decade, prenatal diagnosis of chromosome aberrations by amniocentesis has been practised to a different extent in European countries. An analysis of the reports from amniocentesis (or chorium biopsy) centres can provide information on incidences, specific to maternal age of chromosome aberrations.

A combined analysis of 52 965 prenatal diagnoses (performed because of age criteria) in 59 amniocentesis centers (51 of which were in EC countries) has been carried out on data collected up to and including 1987 (4). This analysis provides a base line for chromosomal anomalies before Chernobyl. To our knowledge, no further analysis of such data has been planned. The geographic spread of amniocentesis centres in Europe would make such analyses potentially interesting in the context of the Chernobyl accident. In France, there is national centralization of prenatal diagnostic data, but there is no similar centralization in the other countries of the EC.

Most of the registries were started before 1980 and they can, consequently, provide estimates of pre-Chernobyl baseline prevalences of particular congenital anomalies, calculated over at least 5 years. The fraction of total births monitored by the registries in each particular area is high (over 90%), and hence the possibility of a bias through increased reporting after Chernobyl is small. But possible bias must, nevertheless, be borne in mind. The registries which indicate 100% coverage carry out periodic checks of their completeness on particular anomalies.

It will be important to select those anomalies for which the defining criteria are clearest. Indeed many anomalies show a decreasing trend when considered overall, but they exhibit fairly stable trends when specific subsets of anomalies are considered (3). This is the consequence of decreasing levels of reporting for minor anomalies. Particular subsets are outlined in Tab.C.2. In general, there are considerable geographical variations of rates (an

extreme case being that of spina bifida). These variations are certain to outweigh any geographical variation induced by radiation. The rate of chromosomal anomalies, however, is fairly stable; it is lowest in Odense ($10.8/10^4$ births) and highest in Galway ($33.9 / 10^4$ births). The maternal age structure needs to be taken into account as a highly important factor in any analysis of data.

Tumours

Tab. C.3 gives an overall synopsis of existing registration procedures in the EC; it is based on publications of the International Agency for Research on Cancer (IARC) of the World Health Organisation (WHO) ('Cancer Incidence in Five Continents' (Vol. V, in preparation) and a specialised volume on Children's Incidence (in press)).

The listed registries are population based, i.e. they aim to retrieve all cancer cases of persons residing in a specified area. To achieve this, they rely both on voluntary information provided by hospitals and doctors and on an active search through visits and telephone contact. They have permanent staff.

Children's cancer being of primary importance, we list in Tab.C.4 some of the characteristics of the registries which could be used for a study of trends in childhood cancers.

To summarise these tables, one can state that national coverage of childhood cancers is achieved by 4 countries (Denmark, Fed.Rep of Germany, Great Britain and Spain), in the Netherlands only leukaemias are covered. Three countries have only regional registries. The percentage of histologically confirmed cases is around 90% for most registries and nearly all carry out checks on duplicates, coding errors and completeness. The latter is estimated to be about 90%. Hence, there are fairly similar standards of registration.

Among the regions with the highest degree of contamination after the reactor accident and with the highest individual doses there exists only in the Fed.Rep of Germany a sufficiently

comprehensive registry with more than 1 000 cases (age 0 to 14) collected per year (national birth rate: 600 000 per year; childhood cancer rate: 13/100 000 per year). In Italy the registration of childhood cancer is poor, in Greece there is no registry at all.

Studies undertaken in the EC

A study of teratogenic defects in the period after the Chernobyl accident was conducted by Eurocat, and it is to be published shortly (5). The results of this study do not show any systematic increase in the frequency of malformations in nine countries of Western Europe.

A preliminary study of chromosomal anomaly rates before and after Chernobyl has also been conducted by Eurocat. It, too, has not indicated an increase. This preliminary study concentrated mainly on Down's syndrome. A re-evaluation over a longer period is currently planned.

At the 1987 annual meeting of Int.Cl. it was agreed to follow the consequences of Chernobyl in two ways: by an analysis of certain selected malformations and by an analysis of the birth prevalence of the routinely reported malformations. The results of these analyses should be made public in the near future.

A working group comprising most European tumour registries was constituted at the initiative of the I.A.R.C (International Agency of Research on Cancer). Registries have agreed to coordinate the monitoring of leukaemia trends for the next 10 years, and to provide incidence data from 1980 to April 1986 in order to compute baseline incidences. The data will be broken down by sex, age, and region for a total population of about 1-2 million. Exposure data for subregions considered in this study will be provided by OECD and others.

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Table C.1 EEC registries on birth defects and chromosomal abnormalities*

Country	Registries	Affiliation	No. of births monitored (1) (average/year)	Starting date of registry (2)	% of total births monitored (3)
Belgium	West Flanders	Eurocat	7 500	1956	96%
	Hainaut	Eurocat	8 000	1979	90%
Denmark	Odense	Eurocat	4 500	1979	95%
	National	Int. Cl.	54 000	1978	100%
France	Paris	both	40 000	1976	≏100%
	Strasbourg	both	13 500	1979	> 90%
	Rhône-Alpes Auvergne	Int. Cl.	85 000	1973	≏100%
	Marseilles	Eurocat	23 000	1982	100%
Germany	West Berlin	Eurocat	18 000	1980	97%
Great Britain (4)	England & Wales	Int. Cl.	660 000	1972	100%
	Glasgow	Eurocat	13 000	1972	≏100%
	Liverpool	Eurocat	20 000	1960	96%
	Northern Ireland (Belfast)	both	27 900	1971	≏100%
Greece (5)					
Ireland	Dublin	Eurocat	25 000	1979	≏100%
	Galway	Eurocat	NA	1981	≏100%
Italy (6)	Firenze	Eurocat	8 900	1979	98%
	Umbria (7)	Eurocat	7 000	1980	91%
	Emilia Romagna	both	21 800	1978	90%
	Multicentric Reg. (147 hospitals)(8)	Int. Cl.	131 400	1978	NA
Luxembourg		Eurocat	2 200	1980	≃ 50%
Netherlands	Groningen	Eurocat	7 700	1981	≏100%
Spain	32 hospitals nationwide	Int. Cl.	67 600	1978	NA

NA: not available
(1) For Eurocat registries, the number of births monitored was evaluated in 1983.
For Int. Cl. the number of births monitored was evaluated in 1985.
(2) For registries set up before the beginning of Eurocat in 1979, the early data does not often conform to Eurocat methods
For Int. Cl. registries, when the starting date was not available, the earliest year on which a base line is calculated is indicated.
(3) This percentage is evaluated by each registry by comparing the number of births taking place in the monitored maternity units and the number of birth certificates in the given area. A registry which claims almost complete coverage is indicated as ≏100%. National registries are indicated as 100%
(4) A Eurocat registry in South Glamorgan was started in 1985.
(5) A registry of congenital anomalies in Evia was in operation from 1980 to 1983 and could provide a baseline estimation of pre-Chernobyl rates for this area.
(6) A Eurocat registry in North-East Italy was started in 1985.
(7) The registry in Umbria is part of the Multicentric Registration.
(8) This co-ordination started with 28 hospitals in 1978.

* This table was compiled from two sources:
- Registration of congenital anomalies in Eurocat centres 1979-1983 edited by P. De Wals, J.A.C. Weatherall and M.F. Lechat (1985)
- International Clearing House for Birth Defects Monitoring Systems, annual report 1985.

Table C.2 Prevalence of congenital abnormalities in 17 Eurocat registries (per 10 000 births) (1980-1983)

Anomalies of the nervous system	31.4	dysraphic	21.3	anencephaly	0.2
				spinabifida	10.9
				encephalocele	1.8
				iniencephaly	0.4
		hydrocephalus	5.1		
		microcephalus	3.3		
Congenital anomalies of the eye	5.8	anophthalmos	0.4		
		microphtalmos	1.2		
		cataract	1.1		
Congenital anomalies of the ear	8.1	anotia-microtia	0.5		
		absence or stricture of the auditory canal	0.8		
Cardiovascular anomalies (including congenital heart disease) (including a rate of 21.0 of diagnosis not specified)	52.1 50.1	hypoplastic left heart	1.8		
		univentricular heart	1.3		
		common truncus	0.9		
		non corrected transposition of the great arteries	2.9		
Facial cleft anomalies	15.1	cleft palate			6.3
		cleft lip			3.1
		both			5.6
Digestive system anomalies	19.0	tracheo-oesophageal fistula			
		oesophageal atresia and stenosis			3.0
		atresia and stenosis of rectum and anal canal			3.6
		atresia and stenosis of small intestine			1.9
Anomalies of external genital organs	17.6	hypospadias			10.5
		indeterminate sex			1
Anomalies of internal urogenital system	7.4	renal agenesis			3.4
		cystic kidney			2.0
Anomalies of limb	59.1	polydactyly			7.9
		syndactyly			6.2
		limb reduction			5.9
		congenital deformities of feet			24.9
Other anomalies of musculoskeletal and corrective tissues	32.2	anomalies of diaphragm			2.7
		anomalies of abdominal wall			6.1
		including: omphalocele			2.2
		gastroschisis			0.5
Chromosomal anomalies	18.6	Down syndrome			13.2
		trisomy 18			2
		trisomy 13			0.8
		deletion			0.4
		balanced autosomal translocations			0.01
		gonadal dysgenesis (Turner's syndrome)			0.9
		Klinefelter's syndrome			0.2
		other autosomal anomalies and trisomies			1.0
		other sex chromosome anomalies			0.3

Anomalies underlined reported by the International Clearing House in its 1985 report.
* adapted from: Surveillance of congenital anomalies years 1980-1983 Eurocat report 1, edited by P. De Wals and M.F. Lechat (1986).

Table C.3 Population-based registries in EEC countries in operation before 1980

Country	General registries (all ages)	National/Regional	Child registries	National/Regional
Belgium	No (1)	-	No	-
Denmark	Yes	N (2)	-	-
France	Yes	R (3)	[Yes] (4)	R
Germany	Yes	R (5)	Yes	N (6)
Great Britain	Yes	N (7)	Yes	N/R (8)
Greece	No (9)	-	No	-
Ireland	Yes	R (10)	[Yes] (11)	R
Italy	Yes	R (12)	Yes	R (13)
Luxembourg	No	-	No	-
Netherlands	Yes	R (14)	Yes (15)	N
Portugal	No (16)	-	No	-
Spain	Yes	R (17)	[Yes] (18)	N

Entries in [ ] do not strictly meet the chosen requirements

(1) There is a national registration of cancer cases notified by health insurance companies since 1969 and reorganised in 1983.
(2) The Danish cancer registry started in 1942 and covers both adult and childhood tumours.
(3) Calvados, Doubs, Isère and Bas-Rhin established between 1974 and 1978.
(4) The 2 childhood tumour registries started after 1980: Lorraine (1983), Provence-Côte d'Azur-Corse (1984).
(5) Saarland established in 1966.
(6) Co-operative Registry started in 1980.
(7) Cancer registration in England, Wales and Scotland is carried out by population-based regional cancer registries with national coverage since 1962.
(8) There is a national registry of Childhood Tumours for Scotland, England and Wales as well as a registry in Manchester and in the West Midlands.
(9) The Greek Ministry of Social Services collects cases of cancer reported by hospitals and x-ray laboratories.
(10) Southern Ireland Tumour Registry has been operating since 1977.
(11) A childhood cancer registry started in Dublin in 1983.
(12) Lombardy (Varese), Parma, Ragusa (only since 1981).
(13) Province of Torino.
(14) Eindhoven registry started in 1955.
(15) Leukemia only.
(16) Only hospital based registries.
(17) Navarra, Zaragosa, Catalonia (Tarragona) since 1980.
(18) The national registry covers over thirty collaborating centres but is not population based.

Table C.4 Characteristics of registries which could be used for a study of trends in childhood cancers in the EEC

Registry	Annual av. pop. m + f (0-14) (thousands)	First data (year)	(1) % Histol.	(2) Dupl. check	(3) Exhaus. check	(4) % Exh.	(5) Coding errors check	(6) Possibility of more inf.
Danish Cancer Registry	1076	1943	93.5%	Yes	Yes	96%	Yes	Yes
Childhood Cancer Registry of Lorraine	535	1983	93%	Yes	Yes	90%	No	Yes
Childhood Cancer Registry of PAAC	809	1984	97.5%	Yes	Yes	95%	Yes	Yes
Cancer Registry of Bas-Rhin	200	1975	94%	NA	NA	NA	NA	NA
Cooperative Registry of Childhood Malignancies in FRG	9962	1980	70%	No	Yes	90%	Yes	Yes
Cancer Registry of Saarland	237	1967	85.1%	Yes	Yes	90%	Yes	Yes
National Registry of Childhood Tumours in Scotland	1257	1959	NA	Yes	Yes	90%	Yes	Yes
National Registry of Childhood Tumours England & Wales	11116	1962	90%	Yes	Yes	90%	Yes	Yes
Manchester Children's Tumour Registry	943	1954	96%	No	Yes	98%	Yes	Yes
West Midlands Children's Tumour Registry	1025	1957	99.8%	Yes	Yes	95%	Yes	Yes
Childhood Cancer Registry of Dublin	1044	1983	99.8%	Yes	Yes	95%	Yes	Yes
Childhood Cancer Registry of the Province of Torino	500	1967	88%	Yes	Yes	95%	Yes	Yes
South East Registry Eindhoven	192	1955	98%	Yes	Yes	95%	Yes	Yes
Dutch Childhood Leukemia Group Study	3336	1973	100%	NA	Yes	97%	NA	Yes
National Childhood Cancer Registry of Spain	not population based	1980	NA	Yes	Yes	NA	Yes	Yes
Cancer Registry of Zaragosa	186	1960	75.7%	Yes	Yes	90%	Yes	Yes

(1) Percentage of recorded cancer cases confirmed by microscopic examination.
(2) Specific procedure for checking duplicate recordings.
(3) Specific procedure for checking exhaustivity of recordings.
(4) Approximative estimation given by the registry of completeness of recordings.
(5) Checking of diagnosis codification errors.
(6) Possibility of getting back to the initial hospital file of the cancer case.

This table was compiled from two sources. IARC forthcoming publication in Childhood Cancer Incidence on Five Continents and an IARC report on Cancer Registration in the EEC (1987). Apart from specialised childhood registries, only general registries which have been in operation for over 10 years are included.

Annex D

Statistical Power of Monitoring Studies of Incidence or Mortality Trends

The power of any epidemiological study depends on its design. The most critical steps are the choice of the parameters to be assessed, the control of confounding factors, the definition of sample sizes and the use of appropriate statistical techniques. We shall discuss each of these aspects and relate them to monitoring potential health effects of the Chernobyl accident.

Choice of the parameters

The choice must be determined by two principal considerations: minimizing of measurement errors and maximizing the sensitivity of the study. Hence, it will be important to have a detailed map of the radioactive deposits and an assessment both of the collective doses and of the doses to sensitive subgroups, such as children or pregnant women. Any study would need to concentrate on these subgroups and to assess with the best possible methods the most sensitive effects. A study on childhood cancer, especially leukaemia, through cancer registries appears to be the most promising approach to identify any health effect due to the Chernobyl accident.

In these studies comparisons should be made between groups with high and low exposures. A pooling of all the data from European registries would lead to a dilution of the potential effect and to a decrease of power.

Confounding factors

Confounding factors need to be taken into account, both in the design of any epidemiological study and in the statistical analysis of the resulting data. In the analysis of temporal and regional variations in aggregated data, such as incidence rates, confounding factors can be particularly important, and certain parameters can exhibit strong correlation without a direct

causal interrelation (1). It is usually difficult, to determine the geographic variations in such confounding factors. Hence, it will not be feasible to assess possible post-Chernobyl health effects by a mere comparison of rates in areas with different exposure; the expected effects are far smaller than the differences due to unknown genetic or environmental factors.

On the other hand, the influence of confounding factors is largely cancelled when one compares incidence rates before and after an event in the same geographic region. It is then sufficent to control confounding factors that vary on the time scale of the investigation. Furthermore, it may be reasonable to assume overall similarity of time trends between regions with smaller and higher exposures, and this would tend to remove the influence of these confounding factors when temporal changes of incidence between two regions are compared. Such comparisons are, therefore, appropriate for inclusion in the study design.

Sample size and the power of a comparison

Because the minimum detectable risk depends on the sample size of any epidemiological study, this linkage will be examined here, and some notions that are frequenctly considered in this context will be introduced.

Relative risk and excess relative risk

Definition of basic quantities:

Assume that a disease occurs with rate, $r_0(t)$, in a nonexposed reference (control) population and with rate, $r_e(D,t)$, in the exposed population. The latter is a function of dose, D, and time, t. The cumulative rates, R(t), (i.e. $R_0(t)$ or $R_e(D,t)$), are the integrals of r(t) over the observation period 0 to t:

$$R(t) = \int_0^t r(t')\, dt' \qquad (1)$$

For a simple example assume that there are x_i cases in a population of constant size N in the i-th year of observation. The cumulative rate, R(k), up to the k-th year is then obtained as the sum of the x_i ($i \leq k$) divided by N.

The ratio of the rates, $r_e(t)/r_0(t)$, is frequently termed the relative risk, RR(t). In the general case the relative risk depends on the time, t, after the exposure or after the beginning of the exposure:

$$RR(t) = r_e(t)/r_0(t) \tag{2}$$

The excess relative risk, ERR(t), is a measure of the enhanced rates in the exposed group:

$$ERR(t) = \frac{r_e(t)-r_0(t)}{r_0(t)} = \frac{r_e(t)}{r_0(t)} - 1 = RR(t) - 1 \tag{3}$$

The relative risk model (see Sect.3.2.1.1) postulates a rate, $r_e(t)$, that is proportional to the spontaneous age dependent rate, $r_0(t)$. This implies that - after an initial latent period - the relative risk, RR, is constant in time. It will, however, depend on dose D, sex S, and age A at exposure:

$$r_e(t) = RR(D,S,A) \cdot r_0(t) \tag{4}$$

Thus, in the relative risk model, the relative risk, RR, and the excess relative risk, ERR, do not depend on time during a follow-up study.

The subsequent considerations are more general, and include the case of time-dependent factors. In particular they include also the absolute risk model (see Sect.3.2.1.1) that is relevant to leukaemias and childhood tumours.

Power of statistical tests

The attainable power of a one-sided test of the alternative ERR=0 (no excess risk in the exposed group in comparison to the unexposed control group) versus ERR>0 (excess relative risk) can be expressed in terms of the expected number, n_o, of cases in the control group and the magnitude of ERR in the exposed group which is, for the purpose of subsequent examples, assumed to be equal in size to the non-exposed population.

Using the normal approximation to the Poisson distribution (a good approximation for $n_o \geq 10$, otherwise see (2)) one finds the power, which is defined as the probability of obtaining a test result that is significant at a chosen level, α:

$$\text{power } (n_0, \text{ERR}) = 1 - \Phi(z_\alpha - z_\beta) = \Phi(z_\beta - z_\alpha) \qquad (5)$$

with:

$$z_\beta = \sqrt{\frac{n_0}{\text{ERR}+2}} \cdot \text{ERR}$$

$\Phi(z)$ is the standard-normal distribution:

$$\Phi(z) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z} e^{-x^2/2} \, dx$$

To illustrate the use of Eq(5) one can derive, with the assumed data from Tab.B.3, estimates of the probability of detecting an increase in childhood cancer, when a specified population is observed for a period, t, before the exposure and, subsequently, for the same period after the exposure.

The following symbols are employed:

n_o : number of expected cases in the nonexposed group:

N_o : number of individuals in each group

t : duration of observation

r_o : background rate of cases

ERR : excess relative risk

α : significance level of the test

z_α : normal upper one-tailed deviate to the level (z)=1-α

$\varphi(z_\beta - z_\alpha)$: probability that the test result will be significant at the level α

With the assumptions in Tab.B.3 one obtains for infants in southern Bavaria:

ERR=0.15; t=10a; N_o=60 000; r=10^{-4}/a; n_o= 60

From Eq(5) one has: z_β=0.8

and with $z_{5\%}$ = 0.13 one has: $\varphi(0.67)$=0.25.

For the one-tailed 5%-significance level there is a probability 0.25 of detecting the assumed increase.

For general guidance one can use - instead of Eq(5) or its more complicated analogue for unequal group sizes - a simple comparison of the expected number of excess cases with the Poisson variance of the difference of observed cases in the two groups under the null-hypothesis. Thus, one has in the example of the infants in southern Bavaria the variance n_o+n_o=120 of the difference under the null-hypothesis, i.e. a standard deviation of $\sqrt{120}$=11. Under the assumed excess relative risk, ERR=0.15, the expected number of excess cases is 9. Since this lies somewhat below the standard deviation (<11) one expects a poor to moderate chance of finding a 'significant' difference. If the number of expected excess cases were between one standard deviation and twice the standard deviation, one would expect a

moderate chance of seeing the increase. If it were to exceed twice the standard deviation, one would expect a good chance.

These simplified considerations are readily extended to the case of different population sizes. Let N_o and N_e be the population sizes and $r=N_e/N_o$ the ratio of the sizes. The observed number of cases, n_e, in the exposed group minus the product of r and the observed number, n_o, in the control group can then be termed the number, n_{ex}, of excess cases. Under the nullhypothesis, n_{ex} has - as is readily derived - the standard deviation $\sigma = \sqrt{n_e(1+r)}$. The expected number of excess cases, $n_e \cdot ERR$, for the assumed ERR must exceed 2σ for a 'good chance' of a significant observation. Using as index the ratio, $n_e \cdot ERR/(2\sigma)$, one obtains, therefore, the useful necessary condition for a study:

$$\frac{ERR}{2}\sqrt{\frac{n_e}{1+r}} > 1 \qquad (6)$$

where ERR is the excess relative risk, n_e is the expected number of cases in the exposed group under the null-hypothesis (ERR=0), and r is the ratio of sizes of the exposed and control populations.

For the above example one obtains a ratio of 0.41. Thus one concludes that the success probability is poor. This is in line with the more quantitative power calculations from Eq(5).

It must be noted that the condition of a Poisson distribution of cases need not always apply. If interdependent cases occur, Poissonian tests will be invalid; the clustered occurrence of cases of Down's syndrome, due to possibly unrecognized viral infections, are an example. This complication must be kept in mind with regard to the above considerations.

There are additional reasons for failure of the computations of statistical power to correspond to the reality of an epidemiological study. As stated, they disregard confounding factors that

may be different in two populations or may change in time in a population. The presence of confounding factors is usually the major limitation of an epidemiological study, and the formal consideration of the statistics of case numbers must not distract from this far more difficult aspect.

There may be various approaches reducing the influence of confounding factors, but these approaches may often be mutually exclusive. As stated at the beginning of this section, one can, by comparing contemporaneous populations, largely reduce the influence of confounding factors that are due to secular changes; however one incurs, thereby, the usually unavoidable disadvantage that the two populations may differ in other important aspects.

If, on the other hand, secular trends are of minor concern or if they can be corrected in terms of known population rates, it may be more advantageous to compare observations in the same population before and after the event of interest. This is reflected in the choice of the above examples for power computations.

Statistical methods

Time series methods are designed to model the trend, seasonal variations, and the residual fluctuations of an observed set of chronologically ordered data points (3). The trend is estimated either by linear or quadratic variations or by a moving average. The residual fluctuations are assumed to be stationary and modelled by an ARMA (autoregressive moving average) process.

Furthermore, one can incorporate the effect of an 'intervention' into this modelling procedure, and this makes it possible to test at specified times for shifts (sudden or gradual) of the mean (4). The drawback of these methods is that they require a time series of sufficient length with the constraint that the random errors be normally distributed. For instance, the methods might be applicable to the study of childhood cancer or leukaemia incidence over 20 years (10 years

before and 10 years after the reactor accident) for data broken down by trimesters and for a population with at least 5 expected cases per trimester. For shorter series, it might merely be possible to estimate a trend before the event and to test whether the observed rates after the event lie within the confidence interval of the predicted trend (5).

Other methods, such as the cumulative sum technique (CUSUM) or sequential analysis, have been proposed in the context of monitoring of congenital malformation frequencies (6). These methods are designed specifically to detect quickly any increase in the number of cases, rather than to study time trends. A test for detecting a 'change point' in a Poisson process was recently applied to study the rates of hypospadias in Liverpool (7).

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Annex E

Quantities and Units

The multiplicity of radiation quantities and units has led to confusion and to misinterpretations after the reactor accident and the ensuing radioactive contaminations in the countries of the EC.

The use of more than one, or even a few quantities and units is, however, unavoidable because of the different properties of radionuclides, such as iodine or caesium, and because of the complexity of different pathways of radiation exposure (see Annex B).

The complexity is largely reduced, when exposures are specified in terms of the radiation energy actually absorbed by the human body. The concept of absorbed dose (or simply dose) permits this specification. It equals energy per unit mass, and it is measured in joule/kg for which the special name gray (symbol: Gy) is used (1). The older unit of absorbed dose is the rad which equals 10mGy. Apart from two diagrams giving the deposited activities per surface area in different countries, the absorbed dose is the only reference quantity utilized in this report; for brevity the simpler term dose is used for absorbed dose.

A dose can refer to the exposure of one organ only, and it is then of less consequence than the same dose applied to the whole body. Organ doses and whole-body doses must, therefore, be distinguished. The assessment of the doses from fall-out due to nuclear-weapons tests was complex because of the multitude of different radionuclides that had to be taken into account. After the reactor accident it was, at least in the countries of the EC, sufficient to consider the thyroid dose due to iodine and the whole-body dose due to caesium. The dose values quoted in this report refer, therefore, either to the thyroid or to the entire body (including the bone marrow which is relevant for leukaemia-risk estimates).

The concept of effective dose

In radiation protection one uses somewhat more general concepts, which are not employed in this report, but are nevertheless of sufficient interest to be considered briefly. To establish a scale of comparison between the exposure of a single organ and the exposure of the whole body, the organ dose equivalents are multiplied by weighting factors which were specified by the International Commission on Radiological Protection (ICRP) (2). These weighting factors represent the relative contribution of the specified organ to the total risk from a whole body exposure (Tab.E.1). The sum of all weighting factors equals unity, and the sum of all weighted organ dose equivalents is called the effective dose. For example, a thyroid dose of 5mGy is multiplied by the weighting factor 0.03 to result - when no other organ is exposed - in an effective dose of 0.15 mGy. This reflects the judgement that a dose to the whole body carries a mortality risk about 30 times higher than the same dose applied only to the thyroid.

To give the general magnitude of doses resulting from the contamination in the countries of the European Community after the reactor accident, one can state that the average increment of absorbed dose in the first year after the accident in all these countries may have been 0.05 mGy due to the external whole-body exposure, 0.15 mGy due to the whole-body exposure from incorporated caesium, and 0.6 mGy thyroid dose due to the incorporated iodine. To obtain the effective dose, one assigns to the whole-body doses the weighting factor w=1 and to the thyroid dose the weighting factor w=0.03. This results in the contribution of 0.05 mGy, 0.15mGy, and 0.02 mGy, and, therefore, in an effective dose of 0.22 mGy.

A further generalization of the concept of dose is used in radiation protection to account for the different biological effectiveness of sparsely ionizing radiations, such as gamma rays, or x-rays, and of density ionizing radiation, such as α-rays or neutron. The different effectiveness is represented by a qualtity factor Q, which has been set equal to unity for sparsely ionizing radiation and equal, for example, to 20 for α-rays. Multiplying the absorbed dose by the qualtity factor one obtains the dose equivalent, which is measured in sievert (symbol: Sv).

The radioactive contamination in Western Europe after the reactor accident contained - in contrast to the nuclear weapons fall-out - no substantial amount of α-emitters, such as plutonium. The problem of the greater effectiveness of the densely ionizing α-particles was, therefore, not relevant to this report. For the same reason there was no need to use the concept of dose equivalent. Instead it was sufficient to use the absorbed dose and to express even the effective dose in Gy, although this does not agree with the normal convention to treat effective dose as a dose equivalent.

References (Annex E)

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European Communities — Commission

EUR 12551 — Feasibility of studies on health effects in western Europe due to the reactor accident at Chernobyl and Recommendations for research – Post-Chernobyl action report

J. Breckow, A.M. Kellerer, E.G. Knox, S. Richardson, R. Doll, J.D. Boice, J. Estève, G. Silini, J.W. Thiessen

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The report considers whether studies of health effects related to the radioactive contamination of western Europe caused by the releases from the Chernobyl reactor accident would be useful. The report evaluates the exposure patterns and the dose levels within the European Community, the different health effects that might be induced by such doses, and the likelihood that epidemiological studies could produce scientifically useful information. The report concludes that at the exposure levels experienced in the European Community the study of post-Chernobyl cancer rates in adults and the study of heritable genetic effects in the offspring of those exposed would be unproductive. It also concludes that even a study of childhood cancer following *in utero* exposure would be unlikely to demonstrate any attributable increase in risk. However, the report recommends that a small epidemiologic survey of childhood cancer be conducted within areas where selected cancer registration was in existence at the time of the Chernobyl accident to check the ability to predict risks from doses of the order received, to contribute to the understanding of the occurrence of childhood leukemia and to allay public anxiety.

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