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Cancer Risks Related to Electricity Production

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The International Agency for Research on Cancer has previously evaluated the cancer risks associated with fossil fuel-based industrial processes such as coal gasification and coke production, substances and mixtures such as coal tars, coal tar pitch and mineral oils, and a number of substances emitted from fossil-fuelled plants such as benzo[a]pyrene and other polycyclic aromatic hydrocarbons, arsenic, beryllium, cadmium, chromium, nickel, lead and formaldehyde. Based on these evaluations and other evidence from the literature, the carcinogenic risks to the general population and occupational groups from the fossil fuel cycle, the nuclear fuel cycle and renewable cycles are reviewed. Cancer risks from waste disposal, accidents and misuses, and electricity distribution are also considered. No cycle appears to be totally free from cancer risk, but the quantification of the effects of such exposures (in particular of those involving potential exposure to large amounts of carcinogens, such as coal, oil and nuclear) requires the application of methods which are subject to considerable margins of error. Uncertainties due to inadequate data and unconfirmed assumptions are discussed. Cancer risks related to the operation of renewable energy sources are negligible, although there may be some risks from construction of such installations. The elements of knowledge at our disposal do not encourage any attempt toward a quantitative comparative risk assessment. However, even in the absence of an accurate quantification of risk, qualitative indication of carcinogenic hazards should lead to preventive measures.
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INTRODUCTION

ELECTRICITY PRODUCTION entails a wide range of health risks for both the workers and the general public [1]. Some of them are well known (e.g. accidental deaths among coal miners), but many other effects are still matters of debate (e.g. increased cancer risk in populations living close to nuclear power plants). Acute health effects, such as accidents, account for the majority of deaths related to electricity production, according to some researchers [2–5]. However, the burden of chronic diseases is more difficult to estimate, and low estimates may reflect lack of knowledge as well as lack of risk. In particular, cancer is more difficult to quantify than other chronic health effects, because of its long latency, the lack of specificity of most aetiological associations so far elucidated, in particular for the more common cancers (i.e. one exposure may cause several types of cancers and the same cancer can be due to several exposures), and the relative rareness of most cancer types.

A further health consequence of electricity production is psychological, mainly stress, which may lead to increased morbidity. The perception of risk from various forms of electricity production may not reflect the actual risks. In particular, nuclear power, because it is relatively new compared to coal-burning, and because radiation cannot be sensed, is *a priori* more stressful for the neighbouring populations than heavily polluting—but

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more familiar—oil-burning or coal-burning plants. In addition, the long half-life of radionuclides and the sometimes exaggerated awareness that the adverse health effects of radiation may manifest themselves not only on the individuals exposed but also on their descendants, certainly increases the fear of nuclear power. Recently, a study from UK suggested an increase in childhood leukaemia following paternal exposure to radiation in the nuclear industry [6].

This contribution will review the evidence on cancer risks to humans of the electricity production cycles, and will discuss the problems in attempting a quantitative comparative risk assessment. Acute and non-neoplastic chronic health effects will not be discussed in detail, although many of the exposures that are reviewed are known or suspected to be related to non-neoplastic conditions.

Energy from water power and photovoltaic cycles is specific to electricity production, and nuclear-derived energy other than electric has been used for military purposes; fossil fuels such as coal, petroleum and natural gas on the other hand, have for centuries been used as sources of energy for various purposes. Therefore, there is a substantial overlap in exposures to fuel components and emissions between the different uses of fossil fuels. This problem is particularly relevant for populations living in industrialised urban areas, who are exposed to combustion products from vehicular traffic, industrial plants, including power plants, as well as residential and commercial uses of fossil fuels.

Furthermore, any modification in electricity production and use should be considered in the wider context of total energy use: e.g. an increase in electrical power used for domestic heating may result in a similar decrease in the use of other types of energy. This can make it hard to estimate the overall changes in the health effects among the exposed populations.

Methodological problems in evaluating cancer risk from environmental exposures

Data for identifying and quantifying the cancer risk due to exposures related to energy production and distribution and the fuel process come mainly from two types of study: epidemiological (most often cohort) studies of individuals exposed to specific substances or mixtures of substances of interest in particular industries, which are usually occupational studies (because of the greater ease in identifying the populations at risk and measuring the exposures that, as a rule, are much higher than for the general population); and ecological–geographical correlation studies based on comparing mortality or morbidity rates between groups of populations with presumed different levels of environmental exposures.

Occupational epidemiological studies carried out within a given industry are the best suited to determining occupational cancer risks in that industry. For industries involved in energy production or distribution, and in waste storage, however, this approach has not been consistently applied: the published epidemiological studies consider only cancer risk among coal and uranium miners and workers in coal-burning factories, the nuclear fuel cycle and petroleum refining. Except for ionising radiation, exposure data are rarely of sufficient quality to permit extrapolation from occupational exposures to other settings.

Two approaches can be used, and sometimes combined to estimate risk for the general population from environmental exposures resulting from energy production. Geographical correlation studies are interesting because they are studies of the population of interest; however, in order for them to reveal an increased risk based on population statistics, this risk must be quite large [7]. Moreover the precision with which it will be estimated, and its validity for extrapolation to other populations will be poor because of the absence of quantitative individual exposure data and of other information of interest. Finally, the choice of an adequate "non-exposed" reference area is nearly always problematic.

The other approach relies on extrapolation from more precise risk estimates obtained in occupational studies, to populations environmentally exposed; this generally involves obtaining detailed dose measurements at the level of small population subgroups or of individuals [8, 9], for which risk must be estimated; extrapolating risk coefficients to low exposure levels; and taking into account the effects of age, sex, individual susceptibility and other potential exposures which may all modify the risk.

The application of molecular biology methods to epidemiological studies has been recently proposed to better estimate the biologically active dose, study early events in the carcinogenic process and identify genetically susceptible subpopulations [10, 11]. Among the methods already frequently used is the detection of adducts formed between a carcinogen and DNA or other macromolecules [12, 13]. It is likely that a future wider application of molecular biology methods to epidemiological studies will help in the identification and the quantitative assessment of carcinogenic risks from environmental exposures [14].

Additional sources of data for estimating risks are long-term animal tests and short-term tests [15]. For some agents of concern, such as polycyclic aromatic hydrocarbons (PAHs), the only available quantitative data are experimental [16]. The problems involved in assessing risks become even greater because of the need to extrapolate from data on a limited number of species and strains with single, well characterised exposures, to genetically polymorphic human populations who are exposed simultaneously to many different agents, and from cellular and subcellular models to entire organisms.

IARC monograph programme

A number of chemicals which occur in electricity production have been evaluated in the IARC Monographs [11], which publish critical reviews of data on carcinogenicity for chemicals and complex mixtures to which humans are known to be exposed, and on specific cultural or occupational exposures; evaluate these data in terms of human risk with the help of international working groups of experts in chemical carcinogenesis and related fields; and, in some cases, indicate where additional research efforts are needed. The evaluation process results in categorisation of the carcinogenicity of each chemical, group of chemicals or complex mixture or exposure circumstance into one of five qualitative categories (see the preamble of any monograph volume from volume 43 onwards for details).

EXPOSURES OCCURRING IN ELECTRICITY PRODUCTION

Polycyclic aromatic hydrocarbons (PAHs), their mixtures and related compounds

PAHs are formed by the incomplete combustion of almost any fuel (fossil, biomass) and are transported through the atmosphere in the vapour phase and absorbed on particulate matter. PAHs are distributed widely in the human environment, albeit at low concentrations. In industrial environments, the levels of these compounds may be much higher. Epidemiological studies provided evidence to their carcinogenicity, but as PAHs

Table 1. Air pollutants emitted by electricity production plants

Pollutant	Evaluation
Inorganic substances	
Arsenic and arsenic compounds*	1
Asbestos	1
Chromium [VI] compounds	1
Nickel compounds	1
Radon and its decay products	1
Beryllium and beryllium compounds	2A
Cadmium and cadmium compounds	2A
Silica, crystalline	2A
Antimony trioxide	2B
Inorganic lead compounds	2B
Nickel, metallic	2B
Polycyclic aromatic hydrocarbons	
Benz[a]anthracene	2A
Benzo[a]pyrene	2A
Dibenz[a,h]anthracene	2A
Benzo[b]fluoranthene	2B
Benzo[j]fluoranthene	2B
Benzo[k]fluoranthene	2B
Dibenzo[a,e]pyrene	2B
Dibenzo[a,h]pyrene	2B
Dibenzo[a,i]pyrene	2B
Dibenzo[a,l]pyrene	2B
Indeno[1,2,3-cd]pyrene	2B
Polycyclic nitroaromatic hydrocarbons	
1,6-dinitropyrene	2B
1,8-dinitropyrene	2B
6-nitrochrysene	2B
2-nitrofluorene	2B
1-nitropyrene	2B
4-nitropyrene	2B
Others	
Benzene	1
Soots	1
Ethylene dibromide	2A
Formaldehyde	2A

Pollutants are considered to be carcinogenic (group 1), probably carcinogenic (group 2A) or possibly carcinogenic (group 2B) to humans in IARC Monographs volumes 1–53 [11]. For definitions of the groups, see [10].

*This evaluation applies to the group of chemicals as a whole and not necessarily to all individual chemicals within the group.

almost always occur as mixtures, the carcinogenicity to humans cannot be referred to individual compounds. Information from experiments in animals has therefore been essential in establishing their carcinogenicity.

Benzo[a]pyrene (B[a]P) [16, 18] is used as the indicator compound for the presence of PAHs in environmental material, since it has been found consistently in mixtures of such compounds. B[a]P and two others, benz[a]anthracene [16, 19] and dibenz[a,h]anthracene [16, 20], are considered to be probably carcinogenic to humans on the basis of experimental data obtained from both whole animals and *in vitro* test systems. Experimental data indicate that other such compounds are possibly carcinogenic to humans (Table 1).

A number of studies of occupationally exposed groups have addressed exposures to PAHs in detail: Jongeneelen *et al.* used urinary 1-hydroxypyrene as a marker of exposure to PAHs and found markedly increased levels in coke-oven workers [21]. The presence of B[a]P adducts in lymphocyte DNA from coke-oven workers has been confirmed by several methods [22]. In a study in Poland, comparing DNA adducts formed by aromatic compounds including PAHs, a large difference was detected between coke workers and countryside controls: however, adduct levels in local controls, exposed to environmental pollution, were higher than those in countryside controls [23]. Smoking clearly contributes to the human burden of PAH exposure and results in specific DNA damage [24, 25]; this highlights the need for careful control of smoking exposures in evaluation of environmental risks.

More data are available on the carcinogenicity of mixtures containing PAHs, mainly from studies in occupational settings. Thus carcinogenicity to humans has now been established for coal-tar pitches, encountered in a number of industrial situations and in roofers [16]; for coal-tars [16, 26] used medicinally and also present in fossil fuel power plants and other industries; for untreated and mildly-treated mineral oils [16, 27], used in the past in textile production and metal machining and by printing pressmen; for shale oils [16, 28]; and for soots [16, 29]. All of these mixtures produce cancers of the skin, but many also increase the incidences of cancers at other sites, including the urinary and respiratory systems.

PAHs and related compounds, mainly nitroarenes, are component of engine exhausts, and are produced during transportation of fossil fuels (mainly coal and oil) by road, rail or water for electricity production. Several nitroarenes can cause cancer in experimental animals (Table 1), and some of them are potent in various short-term tests for genotoxicity [30]. Exhausts from internal combustion engines have been evaluated for carcinogenic risk by IARC. Diesel engine exhausts have been considered to be probably carcinogenic to humans, and gasoline engine exhausts possibly carcinogenic [30]. No epidemiological study to date has addressed specifically cancer risk among transport workers in the electricity industry.

The evidence with regard to the carcinogenicity of other mixtures containing PAHs, such as creosotes, bitumens and carbon blacks is less clear-cut, mainly because of a paucity of epidemiological data [16]. However, experiments in animals with extracts of both bitumens and carbon blacks have clearly shown a carcinogenic effect, indicating their possible carcinogenicity to humans.

There do not appear to have been any studies on PAH exposure in electricity production workers or in populations living near electricity production plants.

Metals

Arsenic. Electricity generation does not generally give rise to increased arsenic exposure. Environmental contamination with arsenic compounds may, however, occur as a result of electricity production with certain coal types that contain high concentrations of this element. Evidence of human exposure from such sources has been presented from Czechoslovakia [31] and systemic (non-cancer) effects have been reported. In addition to exposure resulting from direct emission of particulate matter with the gases, dissolution can occur if fly ash is deposited so that percolating water can contaminate drinking-water sources. Inorganic arsenic is classified as a human carcinogen, inducing skin and lung tumours in humans [16, 32].

Cadmium. Electricity production in coal-fuelled or oil-fuelled power plants may be performed without significant increases in human exposure to cadmium if coal with low cadmium

concentration is used and optimal technological conditions are employed for combustion and flue-gas cleaning and precautions taken to avoid environmental contamination from deposited fly ash. However, to date most coal-fired power plants have not fulfilled these requirements and conventional handling may result in major increases in human exposures, to some extent by inhalation, but mainly by oral ingestion. The carcinogenicity of cadmium has been evaluated by the IARC [16, 33]. It is classified as a probable human carcinogen, suspected of causing lung cancer in humans.

Chromium and nickel. Although some emission of chromium and nickel does occur from plants burning heavy fuel oil or coal, as with other metals, human exposure to chromium compounds will not be much affected by coal-fired or oil-fired power plants using optimal technology and environmental pollution abatement procedures. Levels of chromium and nickel emissions are dependent on the efficiency of electrostatic precipitators and other devices for control of particulate emissions. Since such precipitators are less frequently found on oil-fired plants and since nickel content of heavy fuel oils is sometimes quite high, nickel emissions may be related more to oil than to coal-fired power plants. The chemical species of chromium emitted from power plants has usually not been characterised but it may be assumed that both hexavalent and trivalent chromium can occur. Chromium [VI] is classified as carcinogenic to humans. An increased occurrence of lung cancer and sinonasal cancer in workers employed in chromate production, chromate pigment production and chromium plating industries constitutes evidence in support of this conclusion. Metallic chromium and chromium [III] compounds are not classifiable as to their carcinogenicity to humans [34]. Nickel compounds are carcinogenic to humans. Metallic nickel is possibly carcinogenic to humans. An increased occurrence of nasal and lung cancer has been observed in workers exposed to nickel compounds in the refining of nickel [35].

Beryllium and lead. Beryllium and lead may occur in some types of coal and in oil shale used for electricity production. Since occupational exposure to beryllium may lead to increased lung cancer risk, beryllium and its compounds are classified as probable human carcinogens [16, 36]. Inorganic lead compounds also produce tumours in experimental animals. Small and inconsistent increases in risk for cancer were observed in workers exposed to lead. These compounds are classified as possible human carcinogens [16, 37].

Sulphur oxides

Sulphur oxides (SO_x) consisting of SO_2, acid aerosols resulting from the oxidation of SO_2 in the atmosphere and SO_2 plus particulate matter, primarily derive from the combustion of fossil fuels [38]. The increasing use of large power stations that disperse pollutants at higher altitudes has led to significant local reductions in concentrations of SO_x in many previously highly polluted urban areas, but has resulted in a more widespread distribution.

SO_x emissions from power plants depend on the sulphur contents of the fuels used. The rate of SO_2 oxidation depends on various atmospheric factors. In particular, sulphur and metals capable of promoting the oxidation of SO_2 to H_2SO_4 in the presence of water and hydrogen peroxide are concentrated in the ultrafine fraction of coal ash [39].

Occupational exposure to sulphuric acid has been associated with increased risk of laryngeal and other respiratory cancers in a number of epidemiological studies [40].

Nitrogen oxides (NO_x)

Combustion of fossil fuels in stationary sources is one of the major sources of emission of NO_x. The oxides emitted in the flue gas of power plants consist of about 95% of nitric oxide (NO) and 5% of NO_2. In the atmosphere, NO is converted to NO_2 via oxidation by ozone. Further reactions convert NO_2 into HNO_3 and various nitrates. In addition, nitrous oxide (N_2O) may account for a substantial proportion of the total NO_x emitted by combustion systems fired with coal or heavy oil. It is converted to nitric oxide in the stratosphere by ultraviolet light. Lipid peroxidation in lungs of experimental animals is increased after exposure to NO_2 or ozone at ambient levels resulting in cell damage. Exposure to both NO_2 and ozone showed a synergistic effect in lipid peroxidation [41].

Other chemicals

Oxidants. Photochemical oxidants are produced in the atmosphere through a series of reactions involving hydrocarbons and NO_x. Among them is hydrogen peroxide, a strong oxidant that has been suggested to be involved in the formation of sulphuric and nitric acids from the respective precursors SO_2 and NO_x. There is limited evidence of the carcinogenicity of hydrogen peroxide to experimental animals, but no data are available on its carcinogenicity to humans [16, 42].

Peroxyacyl nitrates (PANs) are an important group of photochemical oxidants produced by reactions of aldehydes in the presence of NO_2. The two most abundant PANs are peroxyacyl nitrate and peroxyglutaryl nitrate, which are formed by reaction of acetaldehyde and glutaraldehyde, respectively. PANs are known to impair human lung function [43].

Carbon monoxide. Carbon monoxide is the most common air pollutant, and it is emitted during incomplete combustion of organic chemicals. An epidemiological study on US motor vehicle examiners, who were exposed to high levels of carbon monoxide as well as to other automobile exhaust products, found an excess of cancer mortality among individuals with 30 or more years since first exposure. No single type of cancer appeared to be responsible for the increase [44].

Hydrocarbons. A number of hydrocarbons found in the atmosphere can be generated by electricity production cycles. However, other sources of emission, both natural and industrial, are usually more important.

Methane, propane and butane are the major components of natural gas. They leak from natural gas pipelines, and may also be released from coal mines. No data are available on their carcinogenicity to humans. Benzene is a natural constituent of crude oil; it is carcinogenic to humans [16, 45].

Formaldehyde. Formaldehyde is produced during combustion processes in power plants. However, other sources of emission, such as direct production and use, and automobile exhaust, are considered to account for most human exposure to formaldehyde. Formaldehyde is probably carcinogenic to humans [16, 46].

Hydrochloric acid. Coal and other chloride-containing fossil fuels are important sources of airborne hydrochloric acid. There have been reports of increased risk of larynx and lung cancer

among workers exposed to acid mists, including hydrochloric acid [40].

Silica. Crystalline silica is present in oil shale. Large amounts of silicates are found in peat fly ash. Exposure to silica may also occur in the production of photovoltaic cells. Crystalline silica is probably carcinogenic to humans [47].

Asbestos. Asbestos has been used in the past for insulation purposes in areas of electricity-generating power stations with elevated temperatures. Exposures to asbestos may occur in particular during the dismantling and stripping of old lagging material [48]. Asbestos is carcinogenic to humans [16, 49].

Polychlorinated biphenyls (PCBs). Mixtures of mainly hexa- and heptachlorobiphenyls, with some polychlorobenzenes, are used as dielectrics in transformers and pressure gauges. Workers may be exposed during maintenance processes and refilling operations. Exposure to the public has occurred following explosion of transformers. Soot produced during fires and accidents in PCB-filled electrical equipment are highly contaminated with polychlorinated dibenzofurans [50]. PCBs are probably carcinogenic to humans [16, 51].

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs)

Incineration of urban waste is a source of human exposure to PCDDs and PCDFs. However, other industries, such as metal smelters, and pulp and paper mills, as well as motor vehicles, are considered to be more important sources of PCDD and PDCF exposure than power plants [52].

2,3,7,8-Tetrachloro dibenzo-p-dioxin is a potent animal carcinogen and was classified as a possible human carcinogen by IARC [16]. A mixture of two hexachloro dibenzo-p-dioxins was shown to be carcinogenic in animals [53].

Ionising radiation

Radiation is said to be ionising when it has the potential to accelerate electrons in matter. Ionising radiation covers both electromagnetic (such as X-rays and γ-rays) and particulate (such as γ-particles and neutrons) radiations.

In terms of biological effects, distinctions are made between different types of radiation—as a function of the amount of energy transferred in matter over short distances—and between external irradiations and internal contaminations with radionuclides which, as they decay, may irradiate tissue.

Exposure to ionising radiation may result from the nuclear industry and from radioactive materials present in fossil fuels. Much information has been accumulated, both from epidemiological studies and from animal experiments on its carcinogenicity [54, 55].

Most of the human data, however, for low linear energy transfer radiation (such as X-rays and γ-rays), comes from epidemiological studies of population groups with acute or fractionated (over a short period of time) exposures resulting in high doses, such as survivors of the atomic bombings and patients receiving radiation treatment [55]. For higher linear energy transfer radiation, such as α-particles, resulting from the radioactive decay of radon and its progeny, most of the human data is based on more chronic exposures (over an occupational lifetime), but also resulting in high doses, as in the case of hard-rock miners [56]. Much of the concern today is about the effects of protracted exposures resulting in much lower doses at work in the nuclear electricity industry or in the environment.

ELECTRICITY PRODUCTION CYCLES

Coal

Coal is the fossil fuel most widely used in electricity production (Fig. 1). Most of the environmental and health problems associated with coal use come from its impurities, which are higher in lignite or brown coal than in black coal (anthracite) [8].

Clay minerals and carbonates represent a large percentage of inorganic compounds in coal, but they are considered not to represent a carcinogenic risk. Sulphides (mainly iron disulphides, pyrites and marcasite) and sulphates are of great importance, since they determine the sulphur content of emissions. Finally, many metals that may present a carcinogenic risk are found as trace elements in coal: Zn, Cd, As, Pb, Co, Ni and radionuclides [58].

The increase of risk for cancer in coal miners is referred to only two target sites: stomach and lung. Increased risk for stomach cancer among coal miners was observed in early mortality studies examining data from vital statistics, both in the UK and in the USA [59, 60]. Elevated mortality from stomach cancer was also found in five more accurate cohort studies of coal miners in the UK, the USA and Australia [61–65]. In one of these studies, a statistically significant dose-response relationship with increasing dust exposure was observed [65]. No increase was seen among Canadian miners with silicosis [66]. An additional cohort study from the USA did not report an increased stomach cancer mortality [67]. Overall results from cohort studies indicate a 50% increased mortality from stomach cancer among miners. Three case-control studies on stomach cancer carried out among mining populations all identified increased risks, but they were not statistically significant [68–70].

Lung cancer mortality was elevated in the largest US cohort study of miners [62] and also among Canadian silicotic miners awarded compensation before 1956 [66]. In all other studies no increase was observed [61, 63–65, 67]. No dose-response relationship was found in one study examining lung cancer risk in association with coal-dust exposure [65]. The overall SMR for lung as estimated from cohort studies is very close to that expected from national reference mortality rates. Two case-control studies, one conducted in the USA and one in the Netherlands, did not identify an increased risk [71, 72].

Acid coal mine drainage, primarily from coal waste heaps and underground mines, may affect water supplies, increasing the concentration of trace elements such as metals. Coal-cleaning wastes can produce highly acid leachates that dissolve trace elements, such as cadmium, from the wastes [58]. Air emissions from diesel equipment and air pollutants from burning mines

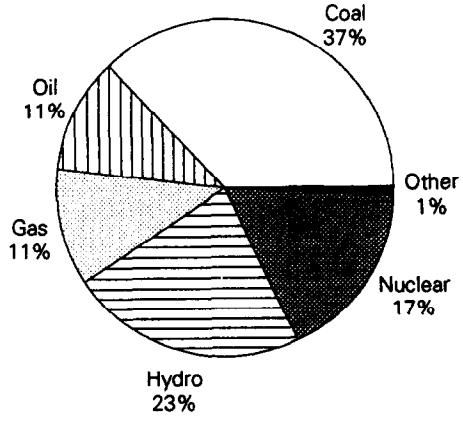

Fig. 1. Proportion of world electricity by energy source, 1985 [57].

Table 2. Estimates of air emissions by fuel type for fossil-fuelled central station electricity generation [T/GW(e).year] [3]

Fuel type	Particulates	SO_2	NO_x	Hydrocarbons
Coal	1160	23 200	23 200	44.9
Wood	4460	3920	9090	18 200
Oil	1160	31 000	11 600	273
Natural gas	1160	21.5	2500	35.6

and waste piles, as well as air emission from coal transportation vehicle engines, may lead to additional environmental exposure to carcinogens.

Among products released in the atmosphere during coal combustion are SO_2, NO_x, CO, hydrocarbons, PAHs, metals and radionuclides. In the US, coal burning causes over 50% of total SO_2 emissions, and about 30% of total NO_x emission. The electricity industry covers about 85% of all US coal consumption [73]. Pollutant emissions depend on the composition of the coal, the operating conditions of the plant and local atmospheric conditions [74]. Therefore, it is difficult to estimate a range of emissions for coal-fired power plants that would be valid in different geographical and temporal settings: examples of estimates based on measurements in the early 1980s in the USA are presented in Table 2. However, in comparison with other fossil fuels used for electricity generation, it is clear that coal will cause higher emissions of particulates. Coal fly ash has shown mutagenic activity in several studies [75, 76].

Concentration of metals in emissions from coal-fired power plants varies according to the composition of the coal. Elements of greatest concern are arsenic, cadmium and lead. Chromium and nickel occur at lower concentrations. The concentrations of trace metals increase as the particle size of fly ash decreases [39].

Coal contains trace amounts of radionuclides from decay series headed by uranium-238, uranium-235 and thorium-232, which are emitted during coal combustion. Coal typically contains about 1–2 parts per million (ppm) uranium and 2–4 ppm thorium [58]. Even if the total amount of radionuclides is orders of magnitude lower than in the nuclear cycle, the maximum dose in individuals in the vicinity of a coal-burning facility may be comparable to that from a nuclear facility; these emissions are not usually monitored around coal-burning plants, which hampers the estimation of the health effects of radioactivity from coal use. In addition to radionuclides in particle emissions, small amounts of radon gas are released during coal combustion and from ash piles.

Peat

Peat is used as an energy source mainly in Nordic countries. Use of peat in energy production yields less sulphur oxides than the use of coal or oil. The production of polynuclear aromatic compounds during peat burning depends on the circumstances: under incomplete burning conditions, the amount of these compounds can be substantial (up to 39 μg/MJ) [77]. Peat ash has shown mutagenic activity [78].

Wood and other biomass

The term "biomass" includes photosynthetically derived materials, which have always been used by humans as a source of food and materials, and now play an important role as an energy source, particularly in developing countries.

Biomass energy can be produced from almost any organic material, including terrestrial and aquatic plants, and agricultural residues. Certain industrial and municipal wastes can also be considered as biomass.

Power from biomass can be generated by direct combustion or by converting biomass first into an intermediate fuel such as synthetic natural gas, methanol, ethanol or vegetable oils, and thereafter combusting them. In developed countries, power is primarily produced by a combined heat and power plant operated by industry using forest litter, wood chips and black liquor. Bagasse from the sugarcane industry can also be burned in on-site plants to provide much of the electricity for sugar refineries. In developing countries, biomass power production is generally produced in very small, widely dispersed units for local use [79].

Because of the range of biomass sources and means of generating electricity, it is not possible to generalise on their impact.

Burning of wood produces only minimal emissions of sulphur dioxide, but can produce high emissions of PAHs and comparable levels of other combustion products such as NO_x and hydrocarbons (Table 2) [80]. Commercial-scale electric power stations use combustion systems which have PAH emissions of the order of 80–700 μg/MJ [77].

Oil

Fuel oils used for electricity generation are mainly of grades nos 4 to 6, also known as "residual oils", derived from distillation residues from refinery processing. They contain 0.2–3.0% of sulphur, and contain mainly straight or branched paraffins and ring structures. Residual oils also contain high concentrations of PAHs [81]. To a lesser extent, distillate fuel oils are also used for electricity production. Potential occupational exposure to fuel oils during electricity production occurs mainly during filling and discharge, maintenance activities of tanks, pipelines, pumps, terminals and depots. Environmental exposure may occur in case of accidental release. The data concerning exposure to fuel oils in humans were considered by the IARC to provide inadequate evidence for their carcinogenicity, mainly because the epidemiological studies were not able to discriminate among exposure to various petroleum products. Residual fuel oils are considered to be possibly carcinogenic to humans [81].

The operation of oil refineries results in the emission of large amounts of airborne SO_2, NO_x, particulates and hydrocarbons, as well as liquid effluents containing, among other compounds, oil and trace metals. Transportation of oil by rail, road or water poses carcinogenic risks comparable to those discussed for coal. Data on air emissions from burning oil to generate electricity (Table 2) do not differ greatly from those of burning coal, except that levels of particulate emissions are considerably lower and there is no ash to dispose of [82].

Ten separate, company-specific cohorts have been studied in the USA [83–90], two in Canada [91, 92], one in the UK [93] and one in Italy [94]. A detailed review of these studies is available [95]. Significant excess mortality from skin cancer was reported among three refinery cohorts. Skin cancer mortality was elevated in three additional cohorts, but the increase was not significant. A case-control study showed a significantly elevated risk for malignant melanoma among men employed in the coal and petroleum products industry, with a cluster of cases in petroleum refineries [96].

Mortality from leukaemia was significantly elevated in two refinery cohorts, and non-significant excess mortality from leukaemia was reported among three additional cohorts. Elevated mortality from unspecified lymphatic leukaemia, unspecified myeloid leukaemia and acute monocytic leukaemia, but not

other cell types, was reported in a subset of workers in the British cohort whose exposures included benzene. A significantly elevated incidence of lymphocytic leukaemia was reported in a large cohort study which included many of the refineries in the USA. Excess mortality from "cancer of other lymphatic tissues", which was not significant, was reported in five refinery cohorts. One report indicated significant excess mortality from leukaemia and "cancer of other lymphatic tissues" combined.

Mortality from malignant neoplasms of the brain was elevated in seven of the refinery cohorts, but this was significant in only one of the studies and only for workers with short duration of employment. A case-control study of astrocytic brain tumours did not find an excess in risk with duration among men employed in petroleum refining during their lifetime [97]. Another case-control study showed a significantly elevated risk for malignant neoplasms of the brain among men employed in petroleum refining [96].

There were reports of an increased risk of stomach, kidney, bladder, pancreas, prostate and lung cancer: these results, however, were not consistent among the studies [95].

One cohort study examined mortality of 11 098 men working at a US oil-producing or pipeline location. Overall mortality was very low and statistically significant deficits were also seen for all major causes of death. The only significant increase was for thyroid cancer among pumper-gaugers [98].

Three small studies on the mortality experience of workers in five Italian coal-fired and oil-fired power plants have been published [99–101]. There was a moderate but consistent excess of lung cancer mortality in all the cohorts analysed. In a study from Milan, there was also a significant excess of total cancer mortality, and non-significant excesses of stomach and bladder cancer mortality, based on few deaths. The authors reported past exposure to PAHs, asbestos, hydrazine, PCBs and metals [99]. In a study from Rome, the lung cancer risk was higher among maintenance workers. No difference in smoking habits was found between power plant workers and the surrounding population, while substantial exposure to asbestos was likely to have occurred [100].

Shale oil

Crude shale oils differ principally from crude petroleum in that they contain higher concentrations of organic nitrogen compounds and arsenic. Numerous PAHs are present. Oil shale contains significant levels of crystalline silica. As, Cd, Pb, Hg and Ni are also frequently found. Shale oil ash has shown mutagenic activity [102]. Shale oils are considered to be carcinogenic to humans on the basis of an excess of skin cancer among workers [16, 28].

Natural gas

Natural gas accounts for about 11% of global electricity consumption (Fig. 1). Exposure to hydrocarbons may occur during extraction of natural gas and from pipeline leaks (see above). Burning of natural gas usually causes lower levels of emissions of SO_2, NO_x and hydrocarbons than the use of coal or oil. Particulate emissions, however, are comparable to those of other fossil fuels (Table 2).

Urban waste

Municipal wastes can be used for electricity generation both directly as fuel at incineration plants and indirectly through the buring of off-gases from landfills. An environmental advantage of waste incinerations is that in burning the organic matter, no greenhouse gases (such as carbon dioxide and methane) are yielded than if the waste were allowed to decay.

Incineration of plastic waste emits hydrogen chlorides, PAHs and other hydrocarbons, especially chlorinated hydrocarbons such as dioxin and furans. Furthermore, heavy metals such as cadmium, lead and mercury should also be taken into consideration [103]. Highly toxic effluents can be avoided by advanced abatement technologies such as two stage wet scrubbers and a particle collection system. Flue ash can be avoided completely by recycling it back into the combustion process and binding it to bottom ash.

Nuclear

Nuclear power provides about 17% of worldwide electricity (Fig. 1). In selected industrialised countries, this proportion is much higher. in France, Belgium, South Korea and Hungary, more than 50% of total electricity generation is produced by nuclear power plants [104].

Direct epidemiological data are available for some parts of the fuel cycle: uranium mining, refining, enrichment and reprocessing, and to a much smaller extent for commercial reactor workers [55, 56]. Additional information on the effects of chronic low-level exposures to ionising radiation is provided by studies of workers in defence establishments and in nuclear shipyards [55].

Uranium miners were at higher risk of lung cancer in studies from the Colorado region in USA [105, 106], Ontario, Canada [107], and Czechoslovakia [108]. In all these studies, there was a linear relationship between lung cancer risk and estimated airborne radiation, due to radon exposure. A multiplicative effect on lung cancer risk was suggested between cumulative radiation exposure and cumulative smoking among Colorado miners [109].

In most studies of nuclear workers, all cause and all cancer mortality rates are substantially lower in the workers than in the reference populations. Possible explanations include the healthy worker effect, and other possible differences between nuclear workers and the general population such as in their socioeconomic status.

Most of the analyses carried out within cohorts, by level of exposure, provide little evidence of an exposure-related increase in overall cancer mortality; the width of the confidence intervals for estimates of radiation-induced risks is such, however, that the estimates are consistent with a range of possibilities, from an absence of a carcinogenic effect to risks several times greater than those on which current radiation protection standards are based (Table 3).

In most studies, analyses of many specific types of cancer have been conducted. These studies have generally been consistent in not showing significant increases in risk for exposed workers for most cancer types examined, although a few associations have been found. Most notably, statistically significant associations between cumulative dose from external radiation exposure and mortality from multiple myeloma have been reported in two studies [115, 116]. Although multiple myeloma has previously been associated with exposure to ionising radiation in the atomic bomb survivors study, the observed excess risk, based on a small number of cases, is not consistent with the estimates obtained from these studies. An association between radiation exposure and mortality from cancer of the prostate has also been found in two studies [114, 117], while non-significant increases in SMR for prostate cancer among nuclear workers as a group were also reported in two additional studies [118, 119]. Statistically

Table 3. Estimates of annual excess mortality risk for all cancers from selected studies [111]

Study [ref.]	Excess deaths (10^6 person-years/10 mSv)	90% confidence interval
Scientific committees		
BEIR V [55]		
Males	9.6*	(6.8–15.5)
Females	10.1*	(7.9–14.5)
UNSCEAR [54]	4–11	
ICRP 26 [110]	4.0	
BEIR III [112]		
Linear	6.0	
Linear quadratic	2.5	
Atomic bomb survivors		
DS–86 [113]	10.1	(8.0–12.4)
Nuclear workers studies		
UKAEA 10-year lag [114]	17.4	(−21.7, 62.3)†
Sellafield 15-year lag [6]	17	(−30, 70)†
Hanford 10-year lag [115]	−29	(<0, 26)

*Based on a single exposure, and an average lifespan of 80 years.
†95% confidence interval.

significant associations between exposure and cancer of the bladder, as well as with leukaemia, were found in one study [120], and for lung cancer in another among those exposed to radionuclides [121]. Increased SMRs for lung cancer were also found in three additional cohorts [120, 121].

Exposure to the general population from power plants, mines and fuel processing may occur through leaks into the local environment (e.g. tritium in drinking water) leading to contamination of water and air, and accidents which may have much wider-scale consequences. Environmental contamination may also occur from accumulated wastes stored in radioactive waste disposal sites.

Since 1983, a number of papers reporting increases in incidence or mortality from childhood leukaemia in the vicinity of UK nuclear facilities have been published: two reprocessing plants (Sellafield [6], Dounreay [122]), on weapons facility [124]. Increases in cancer risk have also been reported, however, near potential sites of nuclear installations [125] and near other "new towns" in Britain [126]. No significant increase in cancer risk has been found in the vicinity of nuclear facilities (including power plants, fuel plants and weapons factories) in the US [127], France [128, 129] or Canada [130].

Recently, the results of two case-control studies investigating the reported increases around Sellafield and Dounreay were published [6, 131]. The study near Sellafield was based on 52 cases of leukaemia and 22 cases of non-Hodgkin lymphoma in children under 15 years; most of the reported increase appears to be attributable to an increased risk in children whose fathers received cumulative occupational radiation doses of 100 mSv or more [6]. The study in Dounreay was based only on 14 cases of leukaemia and non-Hodgkin lymphoma; the increase could not be attributed to parental radiation exposure, but an increased risk was found for using the beaches within 25 km of Dounreay [131].

Levels of occupational and environmental exposures to ionising radiation from the nuclear industry have been regulated since the late 1940s in many industrialised countries. The International Commission on Radiological Protection's recommendations for dose limits are of 100 mSv in 5 years and 50 mSv in any single year for workers, and of 1 mSv per year, averaged over any 5 consecutive years for the general public, respectively [132]. In practice, average annual occupational effective dose equivalents in facilities in Europe, America and Japan are well below 10 mSv, although they tend to be higher in mines and reprocessing plants. For the average individual in the world, an annual dose equivalent from nuclear power production is estimated to be of the order of 0.0002 mSv [54].

Geothermal (hydrothermal), wind, hydroelectric and tidal energy

Environmental effects are small as compared to energy production by more conventional sources.

Airborne emissions from geothermal plants contain carbon dioxide, mercury, armonia and radon (3700–78000 Bq/KW.h) [79]. Hydrothermal fluids (coming from the interior of the globe) contain gases such as CO_2, SO_2 and H_2S, methane, benzene and radon [80]. The most common method of disposal of spent fluids and condensates, that may affect water quality downstream, is reinjection in the reservoir.

Hydroelectric power plants are responsible for a large share of total electricity generation (Fig. 1). No specific cancer risk can be identified in these cycles. However, occupational carcinogenic exposures could occur in related industries, such as metal and construction industry, as well as from earth-moving and transportation activities. The construction of a dam and a reservoir may create environments for parasitic waterborne diseases, such as schistosomiasis, that have been linked to development of cancer in humans [133]. Emissions of particulates, SO_2 and NO_x per unit of energy from geothermal, wind and hydroelectric cycles derive from activities related to materials acquisition, since emissions of these agents during routine operations are negligible, and appear to be one or more orders of magnitude lower than emissions from the coal cycle [80].

Solar

Workers in the device fabrication stage of the photovoltaic energy cycle may be exposed to dusts, fumes and aerosols with large fractions of respirable particles averaging less than 1 μm in diameter. The most commonly used semiconductor material is silicon. Cadmium telluride, gallium arsenide, and copper indium diselamide are also used [79, 134].

Production of silicon may cause exposure to crystalline silica, a probable human carcinogen. However, introduction of closed-top furnaces has reduced occupational and public exposure to this risk. Manufacture of cadmium telluride cells entails occupational and public exposure to cadmium from smelting, refining and fabrication facilities. Exposure to arsenic for workers and the public may result from copper and lead smelting as well as during photovoltaic cell production. Leakage of arsenic-containing fumes from the high-pressure crystallisation furnace in which crystals of gallium arsenide are grown is of special concern. In addition to acquisition and refining of semiconductor materials, workers employed in the fabrication of the cells may also be exposed to strong acids, solvents and welding fumes. Environmental exposure to cadmium or arsenic may occur due to leaks from operating cells and waste disposal after decommissioning of the devices [80].

GLOBAL ENVIRONMENTAL ISSUES

Carcinogenic risk from air pollution and the contribution of electricity production plants

Electricity production plants contribute mainly to the "reducing form" of air pollution, also known as "London smog", the principal components of which are soots, sulphur dioxide and sulphuric acid resulting from the incomplete combustion of coal and oil.

Estimates of airborne emissions of selected air pollutants from different electricity cycles are presented in Table 2. Great caution should be applied when making comparisons between cycles, since the estimates are strongly dependent on assumptions about the composition of the fuels, the technology applied in the plant and other factors that vary with time and space.

A survey made by the US Environmental Protection Agency (EPA) in 1985 estimated that utility combustion was responsible for 69.6% of all US SO_2 emissions and of 32.5% of all NO_x emissions; this proportion was much lower for particulate (1.3%) and volatile organic compounds (2.6%). Coal-fired plants accounted for 95% of total utility SO_2 and 87% of total utility NO_x emissions [135].

Attempts to quantify the human cancer burden due to air pollution have followed two different approaches. On one side, there are examples of quantifications based on low-dose extrapolation of the risk from human or animal carcinogens among air pollutants, such as formaldehyde and metals. A recent authoritative example is the estimate made by the US Environmental Protection Agency (EPA) of the number of cancers in the USA that may be attributable to 90 air pollutants emitted from 60 source categories, among which are coal and oil combustion from utility, industrial, commercial and residential sources. Their overall estimate is 1700–2700 excess cases of cancer per year or an equivalent of roughly 7–11 annual cases of cancer per million population. The largest single source of excess cancer incidence is motor vehicles, which account for 58% of total estimated incidence, while large industrial facilities ("point sources") account for another 20% [136].

In particular, the EPA assessed the risks from coal and oil combustion as sources of air pollution that indicated a burden of 11 cases per year, 1 of deriving from electricity generation [137]. A similar estimate from the UK provided the figure of 1 death per year per 1000 MW of coal-fired and oil-fired plant, due to emission of PAHs and other carcinogens [138].

These estimates must be considered with caution, even for comparative purposes. The information used by the EPA depends primarily on the use of emission, dispersion and exposure models. In only few instances are data available from personal exposure monitoring. These EPA models do not usually incorporate information about how much of the inhaled pollutant or its toxic metabolites is actually delivered to target cells, a kind of pharmacokinetic information rarely available for humans. Furthermore, risk estimation is often based on studies with a small number of cases or poor information on exposure. The use of the linear multistage model for low-dose extrapolation may not reflect accurately the carcinogenic effects at low doses. Interspecies extrapolation adds further uncertainties to the final estimate. Only nine pollutants were included in the EPA analysis on coal and oil combustion, accounting for less than 10% of total organic emissions. The list of pollutants considered by EPA does not in fact include the more abundant air pollutants, such as ozone, SO_2, lead, carbon monoxide, NO_x and particulates, nor does it include hundreds of other chemicals that have not been adequately studied as to their toxicity or human exposure. Finally the emissions depend on a large number of variables and the EPA study used "average" or "typical" emission factors [139].

An alternative approach uses the results of epidemiological studies that analysed the risk of cancer, mainly from the lung, associated with exposure to air pollution.

The major obstacle to a clear evaluation of the role of air pollution in inducing lung cancer is the importance of other sources of exposure to the same chemicals, such as active and passive smoking, occupational exposures and indoor air pollutants including radon gas and combustion products originating from cooking and heating.

Most epidemiological investigations on air pollution and lung cancer are studies on the correlation between mortality from lung cancer with time or across geographical areas with known gradients of air pollution. Attempts have been also made to assess trends in lung cancer mortality in relation to reductions in air pollution and some investigators have attempted to determine how air pollution interacts with smoking.

These studies have provided some evidence of an increased risk of lung cancer among individuals who lived in more polluted areas, compared with individuals living in less polluted areas. A positive interaction between air pollution and smoking or occupational exposure was indicated by a number of studies [140]. The relative risks were in the range of 1.1–1.5. It should be noted that a relative risk of 1.1, i.e. a 10% increase in the incidence of lung cancer over the background, would mean that about 9% of all lung cancers among exposed individuals are attributable to air pollution. The proportion of cancers in the overall population that is attributable to air pollution depends on the proportion of people exposed to it. A recent study from Cracow estimated that 4.3% of lung cancer among men and 10.5% among women are attributable to air pollution [141]. These proportions, if applied to US lung cancer incidence, would represent 4386 lung cancer cases per year in men and 5775 cases in women [142]. It is clear that, even assuming that electricity production contributes only 10% to overall air pollution, this approach provides estimates of extra cancer cases which are orders of magnitude higher than those obtained by analysing specific pollutants, as previously described.

In conclusion, in most studies, residence in urban or polluted areas has been found to be associated with an increase in cancer risk, mainly lung cancer. It therefore seems probable that, in heavily polluted areas, air pollution increases mortality from lung cancer, but the contribution of different sources of pollution, and specifically that of electricity production plants, cannot be accurately assessed. Estimates based on the effects of selected pollutants for which both information on air level and models on cancer risk at low doses are available, on the other hand, are likely to underestimate the magnitude of the contribution of air pollution to human cancer.

Ozone depletion

There has been an awareness that the concentration of ozone in the earth's stratosphere has been decreasing for nearly 2 decades [143].

The concern was mainly about depletion in high latitudes of southern hemisphere, but a recent study reported a substantial decrease over the Arctic as well [144]. Ozone has an important role in the earth's ecosystem, in that it is responsible for absorbing UV radiation from the sun—practically all of that in the UV-C range (240–290 nm), and a large proportion of the UV-B (290–320 nm). It is this range of wavelength that can

cause DNA damage, and hence is important to the viability of terrestrial life forms.

The concentration of ozone in the stratosphere is maintained by a complex series of photochemical reactions [145]. In summary, ozone destruction results from the presence of free radicals, which catalyse the conversion of ozone to oxygen (Cl, Br, NO, H, OH). The principal source of chlorine is the chlorofluorocarbons and that of bromine is various brominated compounds; NO and OH are produced in the stratosphere by reaction of the gases N_2O and H_2O with atomic oxygen. N_2O is emitted naturally from soils.

Some of the so-called "greenhouse gases", important in causing an overall warming of the lower atmosphere, can also be involved in modifying ozone levels.

Future models of atmospheric ozone concentrations are complex, and require many assumptions about the trends in emissions of all of these gases; in general, however, there is a consensus that atmospheric ozone will continue to decline for at least the next 40 years, perhaps by some 6–7% on average [146]. As a consequence of the resulting increased exposure to UV radiation, an increase in skin cancers (non-melanoma and melanoma) can be expected. A 1% reduction in ozone levels will increase the biologically effective UV flux by 2%, and it has been estimated that this would increase the incidence of non-melanoma skin cancer by 1–3%, and mortality from malignant melanoma by 0.8–1.5% [147].

Cancer risk from waste disposal

Few data are available on cancer risk from waste disposal in the electricity cycle. Most industrial processes generate some toxic waste; the carcinogenic risks to the workers and the general population living near toxic waste dumps are still badly characterised and are not specific to electricity production.

The carcinogenic risk of most concern is from radioactive waste disposal. At present, the dominant contribution is from low-level and intermediate-level radioactive waste, mainly mine and mill tailings, which accounts for about three quarters of an effective dose of approximately 200 person-Sv per GWe per year [54]. Most of this dose will be received within about 10^4 years after waste disposal. This is based on the assumption that intermediate-level radioactive waste is stored in "reasonably" covered engineered disposal facilities, built below the water table, preferably in clay soils, thus minimising the contamination of local waters [54].

At present, neither spent fuel nor vitrified high-level radioactive waste has been permanently disposed of; current assessments of risk from radioactive waste disposal do not therefore take that into account [3, 54]. The disposal of high-level radioactive waste and the management of waste sites to prevent future contamination resulting from population settlements, acts of war, geological events, for hundreds of years poses great logistic problems. It is therefore not possible to predict what the health risk from the disposal of today's high-level radioactive waste is going to be for future generations.

CANCER RISK FROM ACCIDENTS AND MISUSE

Accidents are not the only source of non-routine exposure to carcinogenic agents, as has been shown with the burning of oil wells during the war in the Persian Gulf. Acts of war, targeted either on oil or nuclear facilities can lead to extreme levels of environmental contamination with still unclear consequences for humans and the natural envrionment; the carcinogenic risks entailed by these acts are difficult to assess, as is the likelihood of future events of this sort.

In terms of cancer, the accidents in coal mines and dams entail little, if any risk. Refinery, platform or tanker fires may result in high emissions of combustion products. Accidental release of oil or natural gas may also result in heavy environmental exposure to hydrocarbons. Accidents in the nuclear industry can lead to abnormal levels of exposure to ionising radiation locally and possibly on a global scale.

Accidents in the nuclear industry can be divided into two types: those that concern only a limited number of persons (mainly workers in a plant or during transport of radioactive materials) and those that involve large groups of the general population (for example Chernobyl). Accidents limited to a few exposed persons are much more frequent than accidents yielding global environmental contamination, and may go unreported. Most of the known accidents have resulted in relatively high doses to small numbers of individuals [54, 148]. At least ten major accidents occurred between 1945 and 1989, mostly before 1965, since when better knowledge of relevant parameters has allowed the reduction of risk of such accidents; most of these accidents occurred in research reactors. UNSCEAR has also reported a large number of incidents in connection with the transport of radioactive material or with the loss or stealing of such materials [54, 149].

Accidents at nuclear power plants involving large numbers of individuals are fortunately rare, but do occur. There have been four known major documented accidents in nuclear power plants: Kyshtym in September 1957 in the South-East Urals; Windscale in the UK in October of the same year; Three Mile Island in the USA in March 1978 and Chernobyl in the Ukraine in April 1986. Detailed reviews of these accidents are available [54, 148, 149].

The accident in Kyshtym originated from a chemical explosion in concrete tanks containing high-activity radioactive wastes and resulted in the release of approximately 74×10^{15} Bq, mostly short-lived radionuclides (less than 1 year), and 5.6% strontium. An area of 15 000 km^2 was contaminated at levels greater than 3.7×10^9 Bq/km^2, and 1000 km^2 above 74×10^9 Bq/km^2 [148]. About 11 000 persons were evacuated in the 18 months following the accident. The highest doses have been estimated to be of 170 mSv (external) and 520 mSv (internal) [150].

The accident at Windscale originated from a fire; doses to the most exposed individuals were of the order of 1 mSv. The collective effective dose equivalent has been estimated to be of the order of 1300 person-Sv, of which almost half was due to iodine isotopes.

The accident in Three Mile Island originated from a series of human errors; the safeguards and interventions were, however, such that doses to the most exposed individuals were only around 1 mSv. The collective whole-body dose has been estimated to be about 16–35 person-Sv within a 50-mile radius, and of the same order outside this area. The principal radionuclide was xenon-133 [54]. No increase in cancer incidence was found among 5493 persons living in the area who were followed during 1979–1985, nor has any trend in cancer incidence been detected in relation to increasing levels of exposure as estimated using a dispersion model [151].

The accident at Chernobyl occurred while a test was being carried out during a normal scheduled shutdown of one of the reactors, and was due to unsatisfactory written test procedures and design characteristics of the reactor as well as to serious violations of basic operating rules. About 2×10^{18} Bq of

radioactive substances were injected into the atmosphere [152]. The radioactive materials released were carried away in the form of gases and dust particles in air currents, resulting in widely distributed fallout, particularly of caesium and iodine isotopes, in a wide region of the Soviet Union, as well as in other countries, mainly the rest of Europe, and more generally in the northern hemisphere [54].

A very large effort has been undertaken since 1986 to collect exposure and dose data at Chernobyl on the basis of direct measurements (thyroid and whole-body counts for internal contamination and film dosimeters for external exposures) and to model radionuclide deposition and transfer in order to estimate both individual doses and collective dose commitments within the contaminated regions of the Soviet Union and in other countries [54]. Dose and exposure estimates are therefore subject to change as more information becomes available. Approximately 1.5 million persons lived in the areas of Byelorussia, Russia and the Ukraine with the highest iodine-131 contamination, resulting in a collective dose to the thyroid of approximately 22×10^4 person-Sv and to the whole body of 14.5×10^4 person-Sv. An area of 10 000 km^2 in these three republics, with a population of the order of 270 000, was contaminated with radio-caesium at levels in excess of 0.56×10^6 Bq/m^2, resulting in a collective dose equivalent of 5.4×10^4 person-Sv [152]. The estimated collective effective dose equivalent commitment from the accident has been estimated to be of the order of 600 000 person-Sv worldwide [54] or in Europe [148, 153]; the average dose to individuals in the northern hemisphere outside of the USSR has been estimated to be less than 2 mSv [148].

Many reports have been published on the radiological contamination from the Chernobyl accident and on its health consequences. Predictions range from nearly zero to millions of additional cancers worldwide. Using the more accepted risk coefficients for radiation carcinogenesis derived by national and international risk assessment agencies, the variability is somewhat reduced: Ilyin and collaborators [152] predict approximately 1200 extra deaths from cancer among the 74 million inhabitants of the central regions of the USSR, an excess of 0.011% above the spontaneous level, using the UNSCEAR [54] radiation risk estimates. Based on Bengtsson's evaluations which use the Swedish radiation protection authority risk estimates, 8000 extra deaths from cancer would be predicted in the European part of the Soviet Union, and 4000 in the rest of Europe [153]. The US Department of Energy [154] predicts 6500 extra cancer deaths in the Soviet Union (range: 2000–17 000), 10 400 in Europe excluding the USSR and 17 400 worldwide. Using a risk estimate for cancer mortality of 0.1/Gy, the CEC predicts 20 000 additional cancer deaths among the 320 million inhabitants of the European Community [155].

Predictions of cancer risks resulting from the Chernobyl accident depend strongly on the assumptions being made regarding dose distributions and radiation risk estimates; the Chernobyl accident resulted in both external irradiation with low linear energy transfer radiation and internal contamination with a number of radionuclides. There are still uncertainties about the effects of protracted low-dose exposures to ionising radiation, as well as about the relative effects of different types of radiation. Any risk prediction is therefore subject to criticism; only a careful study of very large populations with well known dose levels could help in assessing the validity of the various risk predictions that have been published.

It is impossible at present to estimate reliably the health consequences of future nuclear accidents, since too few data are available on the probability of occurrence of a major accident and the prediction of its effects remains subject to the many uncertainties outlined above.

ELECTRICITY DISTRIBUTION

The methods of transmission and distribution of electricity are similar, regardless of the source from which it is generated. Electricity is transmitted over long distances along high-voltage (220 kV or more) power-lines, in some areas as direct current, but usually as alternating current at 50 Hz (Europe) or 60 Hz (North America). These lines are usually strung overhead on pylons, but engineering constraints or environmental considerations sometimes require underground transmission, though this is considerably more expensive. Voltage step-down transformers are used to obtain the lower voltages (up to 100 kV) used for local distribution of electricity closer to the point of use, and reduction to typical residential voltages of 110 or 220–240 volts is achieved with small transformers very close to the point of use.

Possible adverse health effects of occupational exposure to power frequency electric and magnetic fields (EMF) were reported in a number of early papers [156]. Animal experiments carried out since then have been less conclusive, although no veritable long-term carcinogenicity assay and no initiation-promotion experiment has been carried out. A number of such studies are now in progress and should give results within the next 2–3 years.

Recent experimental research on extremely low frequency (ELF) fields has yielded informative results on the mechanisms by which ELF fields may induce cancer. In particular, ELF fields appear to interact with phorbol esters, the best known promoting agents in experimental carcinogenesis, at membrane receptor sites [157], and to be able to suppress T-lymphocyte cytotoxicity in murine cell lines [158].

Considerable epidemiological data on occupational and residential exposures to ELF fields have been published in recent years. Human exposure to EMF in the ELF range (0–300 Hz) has been suspected of increasing the risk of leukaemia and other malignancies, especially in children in relation to residential exposure [159]. There is still no consensus, however, as to whether cancer risk is increased by exposure to EMF arising from electricity distribution systems. There are several reasons for this: firstly, it is difficult to obtain satisfactory measures of exposure, particularly for retrospective studies, and a number of the earlier studies are defective in this respect [160]. Secondly, observed associations of cancer risk with exposure have usually not been strong (relative risks less than 2.5). Thirdly, there is no accepted explanation for how EMF in this frequency range could produce the biological effects which have been observed, let alone how they might enhance cancer risk, although a number of plausible hypothetical mechanisms are being examined experimentally.

Five published studies have looked at evidence relating childhood leukaemia to residential exposure. Two are essentially negative [161, 162]. Two studies carried out in Denver, USA, provide the strongest evidence of a risk [163, 164]. The later study included extensive direct measurement to ELF fields and control of confounding. Three of the studies also provide evidence on central nervous system tumours in children: all three are significantly positive, with relative risks greater than two-fold [163, 165]. The same three studies also show smaller increased risks for all other tumours combined.

Interpretation of these results is complicated not only by the

aforementioned difficulties of measuring exposure, but by its ubiquity. In practice, everyone living in modern society has some exposure to man-made ELF fields, and it is difficult to draw a clear distinction between exposures to fields from electricity distribution and exposures due to use of electrical appliances, although in homes close to transmission or distribution wires the dominant component of total exposure is derived from these wires.

A number of large epidemiological studies of childhood cancer now in progress are likely to provide new insight into the risk, if any, associated with domestic exposure to ELF fields. They will yield not only precise risk estimates derived from extensive direct measurement of exposure, but estimates of the population attributable risk, i.e. the proportion of childhood cancers which could reasonably be attributed to ELF field exposure.

As for occupational exposure, the results are quite consistent across studies, indicating a relative risk for leukaemia of the order of 1.5–2.0 for "highly exposed" vs. "less exposed". Increases in brain cancer risks have also been reported [159].

CONCLUSIONS

Most electricity-generating processes entail carcinogenic risks to humans at different steps of the cycle. Some of the risks are well characterised and in a few cases serious attempts to quantify the cancer risk have been made. In general, risks to workers seem better characterised than those to the public, and a few occupational groups related to electricity production have been quite extensively studied. In most instances, however, the agents of carcinogenic concern that result from electricity production are not specific to that industry, exposure levels are imprecisely known, and exposures are spread over large areas, where the same or similar agents from other sources are also present. In such situations, the quantification of the cancer burden due to electricity production can be estimated only on the basis of a long list of assumptions, resulting in estimates likely to depend more on the uncertainties than on the data available.

Two examples taken from situations where some data are available will illustrate this point. Air emissions from fossil-fuelled power plants represents one of the exposures of major concern for carcinogenicity of electricity production. As has been pointed out, however, there is a substantial overlap of known or suspected carcinogenic agents, such as PAHs and other hydrocarbons, between the emissions of coal-fuelled or oil-fuelled power plants and other industrial settings, motor vehicle emissions, other sources of air pollution such as residential heating, and personal factors such as smoking or indoor exposure to combustion fumes.

No epidemiological study has yet specifically reported on cancer risks of air pollution from fossil-fuelled electricity-generating plants with control for other sources of PAHs and other chemicals. Little direct assessment of exposures in the general environment has been carried out, and the populations at risk of these exposures are rarely well characterised. Model-based estimates of exposure to carcinogens from power plants depend heavily on assumptions about fuel composition, technology, plant location and atmospheric and population parameters. With a few exceptions such as benzene, the evidence of carcinogenicity to humans for most agents present in power plant emissions is based on the extrapolation of data from experimental animals exposed to single substances and/or the limited data on heavily exposed human populations, such as occupational groups. While there is convincing evidence that air pollution globally considered is associated with an increased risk for lung cancer, the quantification of the carcinogenic risk to humans specifically related to fossil fuel-generating plants could hardly go beyond the stage of an educated guess, even in the case where a reliable model-based estimate of exposure to identified agents would exist.

Ionising radiation is the carcinogenic agent for which the most quantitative information is available. Most studies have relied on detailed dosimetry at an individual level. Notwithstanding the level of quantitative information available on radiation-induced cancer, the magnitude of the carcinogenic effect of low doses and low dose rates is still controversial because of lack of sufficient direct epidemiological data on these exposures: the published studies of workers and other populations of interest still do not have the power to test whether current risk estimates for low-dose chronic exposures—the exposure of concern for workers and the general population from routine operation of nuclear facilities—are adequate. It is notable that as the length of follow-up in studies of populations exposed to ionising radiation has increased, the radiation risk estimates on which regulatory bodies have based their protection standards have tended to increase rather than decrease.

A number of reports have been published in the recent literature [6, 166, 167], raising the question of the adequacy of current risk estimates for low-level radiation exposures. The results of these studies suggest that low-level chronic exposures may actually entail a greater risk than that which has been estimated until now. Although the interpretation of these results is not straightforward, and more studies are needed to confirm or dismiss them, it raises, once again, the question of the soundness of scientific bases for risk extrapolation.

The uncertainties in the assumptions needed to quantify cancer risk from different energy sources are best exemplified in the case of airborne emissions of chemicals and radiation from the different cycles. Similar considerations, however, apply to the other steps of each power cycle, such as mining or extraction of raw materials, transportation, waste disposal and accidents.

The estimates of health effects, and specifically carcinogenic risk, of airborne emissions from electricity generation rely on assumptions about a number of factors, including exposures which depend both qualitatively and quantitatively on the compositions of the fuels, the technology of the power plant, the maintenance of key components, such as boilers and pollutant precipitators, the atmospheric conditions (wind, temperature, humidity), the interaction with other airborne pollutants and other carcinogenic exposures, the structure of the population living in the area concerned by the emissions, and factors such as density, the proportions of subgroups with different susceptibility and the relationship between exposures and diseases. Quantitative estimates of the effects of airborne pollutants or radiation usually present simplified assumptions, such as "standard" fuel composition and plant-operating conditions, and no chemical or biological interaction with other factors [5]. No serious attempt has been made to evaluate how much estimates would vary if some of these assumptions were modified.

Any attempt to quantify cancer risk to human populations has to cope with factors which are even more difficult to quantify or predict than those previously discussed. In the case of electricity production, one must weigh fairly short-term exposures and short-term risks (within the space of an individual's lifespan) against longer-term exposures and risks and probabilistic considerations. In the case of coal burning, for example, the exposures resulting from the generation of 1 GW/y

of electricity can be characterised in space and time, and if the risk assessment models are adequate, the resulting excess number of cancer cases can be estimated. The worldwide ecological modifications resulting from fossil fuel combustion and their influence on the future life of humans and other living species ought also to be taken into account, but the predictions are at present based only on the available knowledge of the processes. In the case of nuclear fuels, however, much of the risk is probabilistic: during routine operations, the nuclear cycle does not pollute very much because of the very strict controls built into it. Most of the risk therefore comes from accidents, from acts of war which cannot be predicted, and from the accumulation of wastes which will need to be managed very carefully for many centuries. Both the carcinogenic risk and the exposure resulting from the present production of electricity from nuclear sources must therefore be considered in the very long term, and the risk must be thought of as a risk of cancer in directly exposed individuals and as a genetic cancer risk in future generations. It is unclear how these issues can be put into the frame of a global comparison of effects.

Another aspect of the problem that is also likely to affect any risk assessment, is the time and space variation of risk from different energy sources. Data on which to base comparisons are specific to existing facilities in countries where studies have been carried out. It is likely that technological improvements will continue to occur in the future, but it is unclear, for instance, what proportion of the hazardous emissions currently produced by fossil-fuelled power plants can be reduced by ameliorating the existing plants. Furthermore, the application of sophisticated and highly hazardous technology, such as the nuclear industry, might create special problems in countries where adequate competence is not available.

Very sparse data are available upon which to base a full quantification of the cancer risk to humans from the electricity production industry. Different approaches have provided conflicting data and it would be misleading to compare estimates on widely studied cycles, such as nuclear, with estimates on cycles whose effects are far from clear, such as fossil fuels. More data are needed on exposures and on humans and risk prediction models must be validated. As mentioned, even predictions on the effects of the Chernobyl accident vary by orders of magnitude.

When quantification of risk is considered not to be feasible, however, qualitative indication of the existence of a carcinogenic hazard should be considered sufficient to implement preventive measures and operate toward a reduction in the opportunities of exposure. However, no cycle seems to be free from cancer risk to humans: even "cleaner" technologies, such as hydroelectric or wind power entail some carcinogenic risks for workers engaged in the acquisition of material. Reduction of electricity consumption seems to be the best way to diminish the cancer burden that might arise from this industry. Reduction of airborne carcinogenic emissions, such as metals, PAHs and radionuclides, will clearly be beneficial, although it will be very difficult to quantify the benefit. Prevention of release of contaminants from nuclear plants, both under normal operating conditions and during accidents will also prevent cancer in exposed populations. It is unrealistic, however, on the sole basis of the current knowledge, to recommend the global adoption of any one energy cycle on the basis of a quantified assessment of the effect of this choice on the carcinogenic risk to exposed human populations.

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