IN-DEPTH REVIEW

Occupational exposure, epidemiology and compensation

Richard Wakeford

Occupational exposure

The data summarized in this section are taken mainly from the 2000 Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and generally refer to occupational exposures assessed to have been experienced during the early- and mid-1990s [1]. Occupational exposures in the United Kingdom from 1998 to 2003 have been reviewed recently by the Radiation Protection Division of the Health Protection Agency [2].

Soon after its discovery by Roentgen in 1895, ionizing radiation was exploited in medicine as a diagnostic and therapeutic tool, leading to the inadvertent exposure of medical staff. In the early years of the 20th century, exposures to medical personnel tended to be large, sometimes leading to deterministic effects such as epilation and skin burns. The first substantial epidemiological evidence for the carcinogenic effects of radiation was obtained from observations on radiologists. The use of radiation in medicine grew throughout the last century and currently includes a variety of diagnostic techniques (e.g. fluoroscopy and cardiac catheterization), dental radiography, radionuclides used for organ imaging in nuclear medicine, radiotherapy and other uses such as biomedical research. Initially, it was X-rays that were employed, but techniques became more sophisticated such that other radiations [e.g. densely ionizing alpha particles (\(^ {4}\)He\(^{2+}\)) and neutrons] were introduced. Medical workers now constitute the largest group occupationally exposed to man-made sources of radiation: throughout the world, ~2.5 million medical workers are monitored for exposure to radiation (the majority of these being employed in the countries of the Organization for Economic Cooperation and Development), leading to an average annual collective effective dose of ~1000 person.Sv. In contrast to the high-level exposures experienced by early medical workers, the reported annual occupational doses in excess of 15 mSv are now rare.

The industrial use of radiation is multifarious and includes radiography, irradiation (e.g. the sterilization of medical products), luminizing, radionuclide production and distribution, well logging and accelerator operation. Some 0.75 million workers are monitored for exposure, leading to an average annual collective effective dose of ~400 person.Sv. Although individual doses are generally small, a few workers (such as those involved in site radiography) receive annual doses in excess of 15 mSv, and occasionally serious over-exposures are reported. The luminizing of dials with radium-based paint in the first half of the last century led to accidental ingestion of the paint and, particularly in the United States, high intakes of radium.

Occupational exposure also occurs in educational establishments (~0.33 million monitored workers worldwide) and in veterinary medicine (~50 000 radiation workers), leading to average annual collective effective doses of ~30 person.Sv and 10 person.Sv, respectively.

The discovery of neutron-induced nuclear fission in the 1930s led to the development of the nuclear weapons and power industries in the second half of the 20th century and consequent occupational exposure to natural and man-made sources of radiation. Workers in these industries are involved in mining and processing of uranium, nuclear fuel production, reactor operation, irradiated fuel reprocessing and product and waste management. Additional occupational exposures occur in research and development activities and in production and testing of nuclear weapons. The exposure of workers at the Mayak facility in the Southern Urals to external sources of gamma and neutron radiation and to alpha-emitting plutonium before 1959 were very high, occasionally sufficiently so to produce deterministic effects. The total number of monitored workers in the commercial nuclear fuel cycle is ~0.8 million and the average annual collective effective dose is ~1500 person.Sv. Accurate figures for the defence industry are difficult to come by, but the annual collective effective dose worldwide is likely to be in the range of 100–300 person.Sv.

By far, the largest source of occupational exposure is naturally occurring radiation. The excess risk of lung
cancer in underground hard-rock miners exposed to radon and its radioactive-decay products (principally alpha emitters) has been the subject of epidemiological study. About 0.75 million workers worldwide are involved in underground mining for materials other than uranium and coal (e.g. gold, iron and tin miners), and the annual collective effective dose is just <2000 person·Sv. Coal miners are individually exposed to radon progeny to a lesser extent than hard-rock miners, but the large number of coal miners (∼4 million internationally) leads to an annual collective effective dose of ∼2500 person·Sv. Finally, aircrew are exposed to radon and its radioactive decay products (principally alpha emissions) on board of aircraft. An estimate including a component dose of external gamma radiation from the surrounding rocks. The inhalation of radon-decay products also provides a source of exposure for workers above ground, radon entering a wide variety of workplaces such as offices and shops. Estimates of exposures worldwide are crude, but perhaps 1.25 million workers may be affected with an associated annual collective effective dose of 6000 person·Sv. In summary, ≈6.5 million persons worldwide are occupationally exposed to naturally occurring radionuclides, the overall number of workers so exposed being ∼0.25 million with an annual collective effective dose of 300 person·Sv. In summary, the annual collective effective dose of 6000 person·Sv. In summary, the annual collective effective dose of 11 700 person·Sv.

**Occupational epidemiology**

Occupational exposure to ionizing radiation provided the first epidemiological evidence that radiation increases the risk of cancer when in 1944—a year before the atomic bombs were exploded over Hiroshima and Nagasaki—March 3 reported ∼10-fold excess of leukaemia deaths among US radiologists when compared with the number expected from the mortality rate of other medical practitioners [4,5]. In fact, 13 years earlier, Aubertin [6] had reported an observation of an excess of myeloid leukaemia among French radiologists, although the role of radiation did not appear to have been recognized [4]. Carpenter [7] has summarized the findings of studies of medical personnel published before 1990, and a review of epidemiological studies of cancer among medical radiation workers by Yoshinaga et al. [8] has recently been published. Studies now include radiation-exposed medical staff from the United States, the United Kingdom, Japan, Canada, Denmark and China. The results indicate an excess risk of leukaemia among medical workers who may be inferred to have had occupational exposure to radiation at relatively high levels. For solid tumours, the evidence is more equivocal, although there are indications of an occupationally related raised risk of breast cancer and possibly also of cancers of the skin.

During the first half of the 20th century, and particularly in the United States before 1930, workers (mainly young women) inadvertently ingested substantial quantities of radium-based luminous paint that they were applying to dials [9]. The radium (mainly alpha-emitting 226Ra) was deposited on bone surfaces leading to high localized doses and a large excess of bone cancers among ∼4800 US luminizers with high intakes (average skeletal dose, ∼170 Sv); the excess of bone cancer deaths approached 100 times the number expected from national rates [10]. The highly exposed dial painters also experienced a large excess of cancers of the paranasal sinuses and mastoid air cells, due to the decay of 226Ra deposited in bone to 222Rn, a radioisotope of the noble gas radon, which migrated to the frontal sinuses and mastoid cells leading to their irradiation by alpha particles from radon-decay products [10]. There is also evidence for an excess risk of breast cancer among the luminizers, which may be due to external gamma exposure from the paint pots situated in front of the women [11].

A large number of studies of lung cancer among underground hard-rock miners exposed to radon and its radioactive progeny have been carried out, providing unequivocal evidence for an excess radon-related lung cancer risk [12–14]. There is little evidence from these studies, however, for a radon-related risk of cancers other than lung cancer [15]. Early occupational exposures tended to be high, leading to large alpha particle doses to the bronchial epithelium; but many miners, particularly in recent years, experienced levels of radon-decay products that are comparable to those found in some homes built in high-radon areas [16]. Analyses of data pooled from a number of underground hard-rock miner studies have provided detailed radon-induced lung cancer risk models, notably that derived by the Committee on Health Risks of Exposure to Radon (Committee on the Biological Effects of Ionizing Radiations VI, BEIR VI) based upon 2700 lung cancer deaths among 68 000 miners [12]. The excess risk of lung cancer is approximately directly proportional to the cumulative lung dose from radon and its decay products, and an excess relative risk (ERR, the proportional increase in risk compared to the background level; e.g. an ERR of 1.0 indicates an excess risk that is 100% of the background risk, i.e. the overall risk is double the background risk) coefficient of 0.12 [95% CI: 0.02, 0.25] Sv−1 of dose to the lung may be derived from the pooled miner data [16]. The risk of lung cancer has been found to decrease with increasing attained age and time since exposure but does not vary significantly with age at exposure. The miner data also provide evidence that the risk per unit cumulative dose increases with the length of time over which the dose is received (i.e. an inverse dose-rate effect), although it is
unclear whether this might be an effect confined to high cumulative exposures. The risk of lung cancer from occupational exposure to radon-decay products in underground mines would appear to combine with the risk of lung cancer from tobacco smoke in a manner that is greater than additive but less than multiplicative: a sub-multiplicative interaction between the two exposures exists [12].

Aircrew receive annual occupational doses that are, in general, a few millisieverts above background dose levels (i.e. their occupational doses are comparable to their background doses), so it is unlikely that any effect of these additional exposures could be detected [17]. However, a number of large international epidemiological studies have been conducted, although the consideration of lifestyle factors is important in the interpretation of the results of these studies. Detailed flight histories and measurements of cosmic radiation levels lead to reasonably accurate individual dose estimates. There is little evidence to suggest that aircrew are at a greater risk of radiation-induced cancer than the low level of increase predicted from the low received doses [18–20].

Workers in the nuclear weapons and power industries represent valuable subjects for epidemiological research because of the high-quality dose and personnel records that are generally maintained, and the relatively high occupational doses that were experienced by workers in the earlier years of these industries. Further, many workers received internal doses when radionuclides were taken into the body, usually through inhalation, allowing a study of the risks from internal emitters. However, the level of exposures generally experienced in these industries implies that unless gross underestimation of the risk has occurred, large studies using data pooled from a number of workforces are necessary to provide a realistic expectation of finding any excess radiation-related risk. One interpretational problem encountered in nuclear worker and other occupational studies is the ‘healthy worker effect’, where those employed are usually found to be healthier than the general population due to beneficial differences in a range of background factors, such as smoking, diet and exercise. As a consequence of this effect, most emphasis in occupational studies is placed upon the variation of risk with dose within a group of workers rather than on comparisons with external rates for national or regional populations.

A number of large studies of nuclear industry workers have been undertaken, notably the study based upon the National Registry for Radiation Workers in the United Kingdom [21] and that of workers in the United Kingdom, the United States and Canada co-ordinated by the International Agency for Research on Cancer (IARC) [22]. The IARC three-country study found a positive trend of leukaemia mortality with the cumulative recorded dose of radiation from external sources, which was of marginal statistical significance: the ERR coefficient for leukaemia [excluding chronic lymphatic leukaemia (CLL)], which is considered to have a low sensitivity to induction by radiation] was 2.18 (90% CI: 0.13, 5.7) Sv⁻¹. For the group of all other cancers (i.e. all solid tumours) an increasing trend of mortality with cumulative dose was not discerned: the ERR coefficient was −0.07 (90% CI: −0.39, 0.30) Sv⁻¹. After accounting for the statistical uncertainties associated with these risk estimates, the ERR coefficients are compatible with the equivalent estimates derived from the statistically more powerful study of the Japanese atomic bomb survivors, providing no evidence that the assumptions underlying radiological protection for those exposed protractedly to low doses of radiation lead to a material underestimation of the carcinogenic risk.

Recently, the results of the IARC study extended to include just >400 000 radiation workers employed in the nuclear industries of 15 countries have been published [23]. The workers have predominantly external gamma-ray exposures (average cumulative dose ~20 mSv, giving a collective dose of almost 8000 person.Sv), and the study includes 7000 cancer deaths. In this study, the ERR coefficient for leukaemia excluding CLL was 1.93 (95% CI: <0, 8.47), while that for cancers excluding leukaemia was 0.97 (95% CI: 0.14, 1.97) Sv⁻¹. The latter risk estimate must be treated with some caution because of the influence of lung cancer in producing this statistically significant positive trend with cumulative external dose and other evidence that smoking may be a residual confounding factor. Further, the Canadian data are particularly influential within the data set, even though they contribute just 200 (4%) deaths from cancers other than leukaemia—without the Canadian data, the ERR coefficient for other cancers is 40% less at 0.58 (95% CI: −0.22, 1.55) Sv⁻¹. These issues require investigation before the risk estimates can be properly interpreted.

Some radionuclides distribute heterogeneously between tissues when taken into the body and remain there for a characteristic time that varies with the chemical nature of the material. Plutonium, if inhaled, will reside in the lung for some typical period depending upon the solubility of the chemical form (e.g. as insoluble PuO₂, it remains there for many years), before passing into the bloodstream and being deposited preferentially in the liver and bone surfaces, where it remains for decades before being excreted in urine. Plutonium, in general, emits a short-range alpha particle on decay, so that only those cells in the immediate vicinity of deposited plutonium will be irradiated. It is desirable in epidemiological studies of workers exposed to plutonium to calculate organ- or tissue-specific doses from bioassay data, such as urinalysis results; but it should be noted that for such studies it is the dose already received by tissues that is of relevance, rather than the committed dose (i.e. the dose that will be received over future years) which is used for the purposes of radiological protection.
The calculation of internal doses from bioassay data is complex and comprehensive organ-specific doses have been calculated for comparatively few workforces. One workforce for which the annual organ-specific doses received from internally deposited plutonium have been calculated for the whole cohort is that of Sellafield in north-west England, where moderate exposures to this alpha emitter have been experienced [24]. Omar et al. [25] studied the Sellafield plutonium workers using these organ-specific doses and found no evidence that these workers had a greater risk of cancer than other radiation workers.

Studies of those who participated in nuclear weapons tests have been conducted to address concerns that their health may have suffered. Doses received were, in general, low and no overall raised level of cancer has been found; but there is some evidence of a raised risk of leukaemia among the participants, although the reason for this is unclear [26]. Studies have also been performed on ~0.5 million workers who participated in recovery operations following the Chernobyl reactor accident in 1986. Many workers received doses in the region of 100–200 mSv. To date, no clear evidence of an excess risk of leukaemia, thyroid cancer or other cancer has been detected in these workers, although the length of follow-up is currently limited with respect to an excess risk of solid tumours and some suggestive evidence of an excess risk of leukaemia has been published [27]. The absence of a clear excess risk of thyroid cancer in adult recovery workers is in contrast to the marked excess of thyroid cancer among those heavily exposed as children to short-lived radioiodine released into the environment during the Chernobyl accident [27].

Of potential importance to radiation epidemiology, possibly second only to the Japanese survivors of the atomic bombings of Hiroshima and Nagasaki, are the workers who were employed at the Mayak nuclear complex in Russia. Workers at Mayak were exposed to surprisingly high levels of external radiation and plutonium during the early years of weapons production in the late-1940s and 1950s. Substantial efforts have been made to derive risks associated with protracted external irradiation and with exposure to plutonium from the Mayak worker cohort. For external exposure (average individual cumulative dose 0.8 Sv, with the highest doses exceeding 10 Sv), the risk of both leukaemia and all solid tumours increased significantly with dose after adjusting for the effect of exposure to plutonium; external radiation risk coefficients obtained from the Mayak workers (which take account of their coincident exposure to plutonium) are broadly in line with those expected from the Japanese bomb survivors [28]. Of particular interest is the risk of leukaemia mortality following external irradiation, which shows a marked excess varying with time since exposure, the risk being substantially greatest in the period 3–5 years after a dose was received [28]. This ‘wave’ of excess leukaemia risk following external irradiation is the typical temporal pattern of radiation-induced acute leukaemia risk found in studies of exposed groups [1]. Those Mayak workers assessed to have been exposed to plutonium show clear and large excesses of cancers of the lung, liver and bone—tissues where plutonium will have accumulated—and, to a lesser extent, other solid tumours [28–31]. However, a significant plutonium-related excess of leukaemia is absent [28], possibly because alpha particles emitted by the plutonium deposited on bone surfaces do not reach the cells in the red bone marrow that are sensitive to the induction of leukaemia. At present, organ-specific plutonium doses have been comprehensively calculated for the lung only (mean cumulative lung dose 4.8 Sv, with >50 workers assessed to have a lung dose in excess of 100 Sv), so risk coefficients for plutonium are restricted to lung cancer, which exhibits a pronounced linear dose–response [29]. The lung cancer ERR coefficients are compatible with those obtained from the Japanese atomic bomb survivors [29]: for an attained age of 60 years,

- males: Mayak, 0.23 (95% CI: 0.16, 0.33) Sv⁻¹,
  
  Japan, 0.40 (95% CI: 0.032, 0.86) Sv⁻¹;

- females: Mayak, 0.93 (95% CI: 0.46, 1.9) Sv⁻¹,
  
  Japan, 1.40 (95% CI: 0.76, 2.2) Sv⁻¹,

indicating that, at least for the plutonium lung doses calculated for the Mayak workers, the assumption of a radiation weighting factor for alpha particles of 20 is not seriously in error. The excesses of liver and bone cancers among the Mayak plutonium workers [30,31] are greatest for workers either providing the highest values of excretion in urine or assessed from work histories to have experienced the highest exposures; but the absence of organ-specific doses precludes the estimation of risk coefficients for these cancers. Hopefully, much more information will be derived from a careful study of the Mayak workers, although, unfortunately, risk estimates are likely to continue to be limited by the lack of bioassay data upon which organ-specific doses are based.

Further information on the findings of epidemiological studies of occupational exposure to ionizing radiation may be found in the UNSCEAR 2000 Report [1], the BEIR VII Report [32] and recent review papers [33,34].

**Compensation**

Authoritative bodies have concluded from the currently available scientific evidence that the risk of cancer following low-dose or low dose-rate exposure to ionizing radiation is approximately directly proportional to the cumulative dose received and that there is no threshold dose below which an excess risk does not exist [1, 32], this inference providing the basis for radiological protection [35]. Consequently, an individual exposed to
radiation in the course of his or her work is at some increased risk of cancer as a result of this exposure, although in most instances this increase will be small in relation to the background risk from other causes. At present, a cancer induced by occupational exposure to ionizing radiation cannot be distinguished from a cancer caused either by exposure to other sources of radiation (e.g. natural background) or by other factors such as smoking or diet. Therefore, the assessment of whether a specific cancer in a particular individual has been caused by a specified exposure to radiation, e.g. that incurred occupationally, must be based upon a probabilistic model of radiation-induced risk. The likelihood that the cancer resulted from radiation exposure depends upon the type of cancer, the dose to the relevant organ and a number of potentially modifying factors (such as the sex of the individual, age at exposure and time since exposure), and the possible degree of involvement of radiation must be set against the potential role of relevant background factors (such as whether the individual smoked). Frameworks for assessing the likelihood that occupational exposure to radiation was the cause, or a material contributory cause, of a cancer have been established on the basis of standard radiation-induced cancer risk models, such that the probability that the cancer was caused by the dose of radiation received in the course of work may be balanced against alternative causes. This is generally achieved through the calculation of the ‘assigned share’ (AS, also known as the ‘probability of causation’) for an individual case, where

\[ \text{AS} = \frac{\text{ERR}}{\text{ERR} + 1} \]

and ERR is calculated from standard radiation risk models, given the dose received occupationally and other relevant factors. It must be appreciated that the AS relates to a cancer that has already occurred and should not be confused with the prospective probability of a cancer arising in future as the result of a specified dose of radiation. The AS provides a measure of the weight to be attached to the dose of radiation received occupationally being the cause of the particular cancer in the context of the broad background of possible causes given the circumstances of the particular case. It provides a means of identifying those cases of cancer that are most likely to be attributable to radiation exposure experienced in the course of employment and, therefore, might be deemed most worthy of consequent compensation.

In the United Kingdom, a compensation scheme for radiation-induced cancers was introduced in 1982 by British Nuclear Fuels plc (BNFL) and the relevant trades unions to deal with claims that radiation exposure while employed by BNFL (or its predecessors) had led to cancer in particular workers [36]. This privately run Compensation Scheme for Radiation-Linked Diseases (http://www.csrld.org.uk), based upon the AS concept, now covers almost all the major employers in the UK nuclear industry. A notable feature and strength of the scheme is that it is jointly run and supported by employers and trades unions, and decisions are based upon a consensus. The underlying radiation risk models currently used in this scheme are, in general, those presented in the BEIR V Report [37]. Of some importance to the operation of the scheme are the criteria used to establish whether compensation should be awarded. A criterion often utilized by a court of law is that of the balance of probabilities, which, broadly speaking, implies that if the AS is \( \geq 50\% \), full compensation will be paid, but otherwise no payment will be made. However, uncertainty from a variety of sources is inevitable in the assessment of the AS (e.g. to varying extents within the risk models themselves) and some appropriate account needs to be taken of this uncertainty in compensation schemes. In the UK scheme one process by which uncertainty is accommodated is through proportionate recovery, whereby a fraction of full payment starts to be made at an AS value of 20\%, the fraction increasing with AS, until full payment is made at 50\%. Further allowance for uncertainty is made via the modification of the ERR values generated by direct application of the BEIR V risk models by certain, mutually agreed, generosity factors of benefit to the claimant, and additional generosity may be applied where there is some doubt applying to the dose received at work (e.g. because the dose from high-energy neutrons was not measured accurately by early personal dosimeters) or the dose received by the target tissue relevant to the site of the cancer under consideration relative to that entered in dose records.

In the United States, a government-sponsored compensation scheme based on the AS concept and covering radiation workers involved in nuclear weapons production was introduced in 2000 and includes all cancers with the exception of CLL. The technical basis of the US scheme is the revised National Institutes of Health ‘radioepidemiological tables’, developed by the National Cancer Institute (NCI) and the Centers for Disease Control (CDC) and published in 2003. [Although originally a hard-copy publication, these tables are now an interactive computer program (the Interactive RadioEpidemiological Program, IREP)—http://www.niosh-irep.com/irep_niosh.] The radiation risk models developed by NCI-CDC are primarily based upon the cancer incidence data for the Japanese atomic bomb survivors and provide for modification by certain factors (such as time since exposure), and IREP contains a statistically sophisticated treatment of the various sources of uncertainty in the computation of an AS value [38]. Dose reconstruction to allow the relevant tissue dose and its associated uncertainty to be calculated is also incorporated within IREP. The principal output of IREP is a probability distribution for the AS value for a particular case of cancer, which is generated from an appropriate combination of the uncertainties arising from the values used by the
package. Compensation is paid if the upper 99% confidence limit for the AS is ≥50%, a criterion specified in the pertinent US law setting up the compensation programme to allow for uncertainty in the calculation of the AS in a manner that will benefit the claimant to a degree that is judged to be appropriate.

Conflicts of interest
Formerly an employee of British Nuclear Fuels plc.

References


