Journal of Radiation Research, Vol. 59, No. S2, 2018, p. ii1–ii10
doi: 10.1093/jrr/rrx093
Advance Access Publication: 8 February 2018
Supplement – Highlight Articles of the First International Symposium

Typical doses and dose rates in studies pertinent to radiation risk inference at low doses and low dose rates

Werner Rühm[1,*], Tamara Azizova[2], Simon Bouffler[3], Harry M. Cullings[4], Bernd Grosche[5], Mark P. Little[6], Roy S. Shore[7], Linda Walsh[8] and Gayle E. Woloschak[9]

[1]Institute of Radiation Protection, Helmholtz Zentrum München, Ingolstädter Landstr. 1, 85764, Neuherberg, Germany
[2]Southern Urals Biophysics Institute (SUBI), Ozyorskoe Shosse 19, 456780, Ozyorsk, Chelyabinsk Region, Russian Federation
[3]Centre for Radiation, Chemical and Environmental Hazards, Public Health England (PHE), Chilton, Didcot OX11 ORQ, UK
[4]Radiation Effects Research Foundation, 5–2 Hijiyama Park, Minami-ku, Hiroshima 732-0815, Japan
[5]Federal Office for Radiation Protection, Ingolstädter Landstr. 1, 85764 Oberschleißheim, Germany
[6]Radiation Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, 9609 Medical Center Drive, Bethesda, MD 20892-9778, USA
[7]New York University School of Medicine, 650 First Ave., New York, NY 10016, USA
[8]Medical Physics Group, Department of Physics, Science Faculty, University of Zürich, Winterthurerstrasse 190, CH-8057 Zürich, Switzerland
[9]Departments of Radiation Oncology, Radiology, and Cell and Molecular Biology, Robert H. Lurie Comprehensive Cancer Center, Feinberg School of Medicine, Northwestern University, 300 E. Superior St., Tarry 4-760, Chicago, IL 60611, USA
*Corresponding author. Institute of Radiation Protection, Helmholtz Zentrum München, Ingolstädter Landstr. 1, 85764, Neuherberg, Germany. Tel: +49 89 3187 4011; Fax: +49 89 3187 3323; Email: werner.ruehm@helmholtz-muenchen.de
Received 25 July 2017; revised 6 September 2017; editorial decision 24 November 2017

ABSTRACT

In order to quantify radiation risks at exposure scenarios relevant for radiation protection, often extrapolation of data obtained at high doses and high dose rates down to low doses and low dose rates is needed. Task Group TG91 on 'Radiation Risk Inference at Low-dose and Low-dose Rate Exposure for Radiological Protection Purposes' of the International Commission on Radiological Protection is currently reviewing the relevant cellular, animal and human studies that could be used for that purpose. This paper provides an overview of dose rates and doses typically used or present in those studies, and compares them with doses and dose rates typical of those received by the A-bomb survivors in Japan.

Keywords: radiation risk; ionizing radiation; low doses; low dose rates

INTRODUCTION

After more than a hundred years of radiation research, much is known about the effects of high doses of ionizing radiation on the human body. For stochastic effects such as solid cancers and leukemia, data from the Japanese atomic bomb survivors in Hiroshima and Nagasaki have played a key role in quantifying the risks from external radiation exposure. Because the atomic bomb survivors were exposed to high rather than to low dose rates, however, inference of radiation risk at low doses and low dose rates is still challenging. Furthermore, even after more than six decades of follow-up, radiation risks from several tens of milliGray are still difficult to quantify among this cohort. Estimates of radiation risks at low doses and low dose rates are important, for example for populations living in contaminated areas after nuclear accidents or in high natural background radiation areas, or for radiation protection of individuals occupationally exposed to ionizing radiation.

Recently, the International Commission on Radiological Protection (ICRP) established Task Group 91 to research radiation risk inference for radiological protection purposes at low-dose and low-dose rate exposure, based on cellular, animal and epidemiological

studies, and to examine the weight of evidence for and against the continued use of the dose and dose rate effectiveness factor (DDREF) [1]. This factor was introduced by the ICRP in the 1990s for radiation protection purposes, to extrapolate radiation risks at high doses and high dose rates, where an abundance of data are available, down to low doses and low dose rates, where there is much less human data. The numerical value of the DDREF has been subject to much discussion among various international organizations (reviewed by Rühm *et al.* [2]). For example, in its Report 103 [3], ICRP continued to recommend a value of 2 for this factor, emphasizing the considerable uncertainties associated with this value. In contrast, the National Academy of Sciences (NAS, USA), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) and the German Radiation Protection Commission (SSK) suggested somewhat lower values or to abandon the use of this factor [4–6]. Members of TG91 are currently reviewing the recent scientific literature with particular emphasis on the results of molecular and cellular studies, of animal studies, and of epidemiological studies on human cohorts who have been exposed to ionizing radiation [7], and the first results have already been published [8–10].

The present paper summarizes the dose rates and corresponding cumulative doses that are typical of the various exposure scenarios in cellular experiments, animal experiments, and exposures of various human cohorts, and the results are compared with doses and dose rates typical of those received by the Japanese atomic bomb survivors. In this context it is important to note that UNSCEAR considers an ionizing radiation dose of <100 mGy as being low. Additionally, a dose rate of <0.1 mGy/min averaged over 1 h (corresponding to 6 mGy/h) is considered low [11].

DOSE RATES AND CORRESPONDING CUMULATIVE DOSES TYPICALLY USED IN CELLULAR EXPERIMENTS

In cellular and molecular studies relevant to the evaluation of dose- and dose-rate effects, many differing radiation exposure conditions have been used. The majority of studies of dose-rate effect utilize ^{60}Co gamma irradiation, but some utilize ^{137}Cs gamma irradiation. Low-dose studies, by contrast, tend to be conducted with X-ray sources; though both X- and gamma radiation sources have been used, only a few studies compare X- and gamma-radiation sources.

Among the more sensitive endpoints currently in use are the various chromosomal protein foci assays (γ-H2AX, 53BP1, etc.). The low doses used in most studies are in the range of 1 to a few mGy up to a hundred or more mGy, delivered at a range of dose rates [for the lower dose range, generally around 1 mGy/min (eg Rothkamm and Löbrich [12])]. Endpoints such as chromosomal aberrations and mutations have been assessed at dose rates as low as 20 mGy/d with accumulated doses of 8 Gy [13]; more commonly dose rates of 1–10 mGy/h are reported with accumulated doses of 0.5 to several Gray (e.g. Okudaira *et al.* [14]; Manesh *et al.* [15]). Reference [16], a review specifically considering cellular and molecular data suitable for dose rate effect evaluation, provides a more extensive summary and tabulation of the irradiation conditions used in the relevant studies.

DOSE RATES AND CORRESPONDING CUMULATIVE DOSES TYPICALLY USED IN ANIMAL EXPERIMENTS

Several large-scale animal studies have been carried out in the USA, the current EU, Russia, Japan, and other countries from the 1960s until the present, using dogs, mice, rats and a few other species as model systems in order to study the effects of radiation dose and in some cases dose-rate on life-shortening and cancer incidence. To a large extent, these studies are—for financial end ethical reasons—unrepeatable. Archives from some of these experiments are available, for example, at the Janus archive (janus.northwestern.edu/wololab). Few of these animal studies have been included in considerations of radiation risk inference at low doses and low dose rates and DDREF [4]. For the JANUS gamma-exposed mice, the range of the dose rates was 1.295×10^{-5} to 0.378 Gy/min, while for neutron-exposed mice it was 1.5×10^{-5} to 0.113 Gy/min [17]. Cumulative doses for gamma exposure ranged from 0 to 49.0 Gy, and for neutron exposure from 0 to 3.2 Gy [17].

The European Radiobiological Archives (ERAs) were developed to retain primary data from past large-scale radiobiological animal studies [18]. The ERAs include information from almost all European long-term studies carried out from the 1960s to the 1990s. From the many European studies included in the ERAs, those that used rodents, X-ray or gamma radiation, and had overall cancer or specific malignancies as an endpoint, are of most relevance to the DDREF discussion. After those studies were excluded for which the relevant information was not available, 25 were left, 6 of which used rats, and 19 used mice. Typically, for gamma radiation, the dose rates were from 1.35×10^{-3} to 240 Gy/h, while the dose range was 0.02–68.2 Gy, with a dose of 5.89 Gy averaged over all studies. For X-rays, the dose rates varied from 2 to 60 Gy/h, while the dose range was 0.02–15 Gy, with a dose of 2.88 Gy averaged over all studies.

Recently, a large animal experiment on mice was initiated at the Institute for Environmental Studies at Rokkasho, Aomori prefecture, Japan. The cumulative doses chosen for that study are (i) close to the annual dose limit recommended by ICRP for workers (20 mGy) and (ii) the annual doses astronauts might receive in space (400 mGy). The dose rates chosen are 0.05 mGy/day and 1 mGy/day, respectively. Thus, the typical exposure times of the investigated mice are 400 days starting at an age of 8 weeks. In the experiments, a third group of mice are exposed to a dose rate of 20 mGy/day (with a total cumulative dose of 8000 mGy), serving as a positive control. Irradiation is interrupted for 2 h every day, in order to examine the health condition of each mouse, as well as to supply new bedding, food and water [19].

DOSE RATES AND CORRESPONDING CUMULATIVE DOSES OF HUMAN COHORTS

Natural exposures

General population

Ionizing radiation is a natural phenomenon present in the environment due to various natural sources: (i) cosmic radiation from space and from the sun produces secondary particles in the atmosphere such as protons, neutrons, electrons, etc., but also cosmogenic

radionuclides such as ^{14}C; (ii) primordial radionuclides emitting various sorts of ionizing radiation during their decay have existed—due to their long physical half-lives—on Earth since its formation; and some of their decay products—in particular the radioactive isotopes ^{222}Ra and ^{220}Ra of the noble gas radon—may reach buildings through diffusion from the Earth's crust. Some of these primordial radionuclides (for example ^{40}K) reach the human food chain and, consequently, become part of the human body. As a result, the global annual population-weighted mean effective dose from natural sources of ionizing radiation is ~2.4 mSv [20]. Because the level of radiation exposure from natural sources depends on various factors such as the soil and rock composition of the land, latitude, altitude, etc., this value varies locally, between 1 and 10 mSv per year [20]. Thus, populations may be exposed to an annual effective dose of up to 10 mSv per year, corresponding to a dose rate of 1 μSv/h [20].

For a typical life expectancy of 80 years, the above-mentioned mean annual effective dose results in a mean cumulative effective dose of ~2.4 mSv/year × 80 years = 200 mSv. Due to the regional variation in dose rates, however, cumulative effective doses might be between 80 mSv and 800 mSv.

Population in high background radiation areas

Several geographical regions of the world, including Yangjiang, China and Kerala, India are known to contain high background radiation areas (HBRAs). Epidemiological studies have focused on these regions to determine whether there are any detrimental health effects such as cancer linked to the natural high background radiation dose rates.

In Karunagappally, Kerala, India, a cancer incidence registry provided data between 1990 and 2005 for about 70 000 adults. This data was used to compare cancer rates between the low background radiation areas and the HBRA there, where the high background exposure was primarily due to external gamma radiation from monazite sand containing thorium [21, 22]. The cumulative colon dose ranged to over 500 mGy, with a mean cumulative colon dose from terrestrial sources at end of follow-up for the HBRA study group of 161 mGy. Dose rates measured for a randomly selected subset of the cohort ranged to over 10 mGy per year, corresponding to ~1 μGy/h.

A Chinese study on ~31 600 adults was done with data collected from the Yangjiang area of Guangdong province in China, between 1979 and 1998 from the HBRA there, divided into three dose groups (high, medium, low) on the basis of environmental dose rates per year [23]. The estimated mean cumulative colon doses were 84.8 mGy in the HBRA cohort. Average annual dose rates from external radiation from natural sources, including thorium, were estimated to be 2.10 mSv/year (0.24 μSv/h) in the HBRA and 0.77 mSv/year in the control area [24, 25].

Occupational exposures

General considerations

The ICRP most recently recommended [3] an effective dose of 20 mSv per year as a dose limit for workers dealing with ionizing radiation. Assuming an annual working time of 2000 h, and that the occupational exposure is homogeneously distributed over the working year, a worker reaching this annual dose limit might be continuously exposed to a dose rate of up to 10 μSv per working hour. It is important to emphasize, however, that in most cases the annual dose limit of 20 mSv per year will not be reached, due to effective radiation protection measures. For example, in a recently reported pooled international nuclear workers cohort, the mean cumulative colon dose, over all years of work to date, was 20.9 mGy, and the maximum 1331.7 mGy [26]. With a mean length of work of 12 years, a mean dose rate of 1.7 mGy/year follows (corresponding to 0.9 μGy/h if an annual working time of 2000 h is assumed).

If one considers a worker who is occupationally exposed during a whole career (which may last for 40 years) to an effective dose close to the dose limit, a life-time effective dose of 40 × 20 mSv = 800 mSv would accumulate; however, as noted above, this is only rarely, if ever, the case. In Germany, a limit for the life-time cumulative occupational effective dose of 400 mSv has been introduced [27].

It is also important to note that individual workers—depending on their profession—might have accumulated their recorded doses in a much shorter time. Thus, although the assumption of a continuous exposure over the whole working time is more or less realistic for some occupations (e.g. air crew), it may not always hold for others (e.g. nuclear workers). In such cases, dose rates higher than estimated might occur.

Air crew

In Germany, for example, the average annual occupational effective dose to air crew was 1.9 mSv, in 2012 [28]. Maximum allowed flight hours for air crew are typically ~900 h per year, and some individuals accumulate up to 5 mSv per year or more. Assuming a maximum of ~40 working years, cumulative career doses of up to 200 mSv can therefore not be excluded. In such cases, mean effective dose rates of ~5–6 μSv/h can accumulate, which is consistent with typical dose rates at flight altitudes from secondary cosmic radiation of 2–7 μSv/h depending on altitude, latitude, and solar activity [29, 30]. Astronauts are exposed to highly variable radiation fields, depending on whether or not they are exposed in near-earth orbit (NEO), when they would be largely protected by the Earth's Van Allen magnetic fields [31]. However, even the Apollo astronauts, some of whom were exposed in deep space, had an average film badge dose of 4.3 mGy, with a mean dose rate of 0.43 mGy/day [31].

Nuclear workers

In a recent meta-analysis of cohorts exposed to low dose rates of ionizing radiation, Shore and co-workers performed a review of recent epidemiological studies including workers employed in nuclear installations [10]. Typical cumulative effective doses (for workers in nuclear installations excluding the Russian Mayak Production Association (PA) (see below) are in the range of 6 (Australian nuclear workers) to 36 mSv (Rocky Flats plutonium facility). Although mean durations of exposure are not always explicitly given, typical effective dose rates are 1–2 mSv/year.

Mayak workers

Immediately after the Second World War, the Soviet Union intensified their efforts to develop nuclear weapons. Accordingly, in the Southern Urals the Mayak PA was established, including nuclear reactors, radiochemical and plutonium production facilities, and auxiliary units. Workers there were exposed externally, and internally due to incorporation of radioisotopes such as those of plutonium. Exposures, as estimated based on the Mayak Workers Dosimetry System 2008 [32, 33], were particularly high during the first years of operation, resulting in mean personal dose equivalent [$H_p(10)$] —dose equivalent at a depth of 10 mm in the human body at the position where an individual dosimeter is worn, as defined by the International Committee on Radiation Units and Measurements [34]—values of ~300 mGy per year from external exposure during the years 1949–1952 [35] (corresponding to dose rates of ~150 μGy/h if continuous exposure during 2000 working hours per year is assumed). Note, however, that individual workers might have accumulated their doses in much shorter times, particularly during the early phase of Mayak operation, resulting in higher than estimated dose rates, up to several mGy per second [36].

Similarly, annual absorbed internal doses from plutonium incorporation reached more than a 15 mGy liver dose during the years 1953–1958 [37]. If it is assumed that this annual dose is continuously irradiating the organ during the whole year, this corresponds to typical absorbed dose rates of 1.7 μGy/h. Incorporation of radionuclides by Mayak workers decreased over the decades of Mayak operation. However, it should be highlighted that the dose in any one year is not just dependent on the incorporation in that year but also on the incorporation in previous years.

The mean cumulative external gamma $H_p(10)$ dose is 0.51 Gy, with a maximum of 6.8 Gy [35]. The mean cumulative internal liver dose, largely from alpha-particle exposure, is 0.31 Gy, with a maximum of 36 Gy [35]. This compares with average annual effective doses of up to 1000 mGy recorded in the late 1940s, as reported by [38]. During 1948–1953, when doses to Mayak workers were the highest, the unshielded dosimeters had significant variability in photon energy dependence and angular response, and bias by high-energy beta exposures provided overestimates of doses for workers in some settings. Efforts were made to correct for those estimation biases, but such corrections likely had substantial uncertainties [33, 38]. In addition, before 1957 unmeasured intermediate and fast neutrons were thought to have contributed 10–15% of the total dose [36]. These uncertainties temper the cumulative dose and dose-rate estimates.

Chernobyl workers

After the Chernobyl accident, large areas in the near and far field from the reactor site were contaminated by a variety of radionuclides from the reactor inventory. In the aftermath, considerable efforts were made to clean up the reactor site and adjacent areas. Cohorts of workers engaged in these efforts include >53 000 individuals employed for this work from 26 April 1986 until 25 April 1987, >31 000 individuals from 26 April 1987 until 31 January 1988, and ~21 400 individuals from 1 February 1988 until 31 December 1990. Mean accumulated doses correspond to 161 mGy, 81 mGy and 35 mGy, respectively. However, doses to certain small groups of workers in certain specific periods were much higher, in excess of 500 mGy [38]. Based on these doses, it was estimated that daily average doses of those individuals correspond to 7.6 mGy/day (320 μGy/h), 4.7 mGy/day (200 μGy/h) and 2.6 mGy/day (110 μGy/h), respectively, if one assumes continuous exposure over the whole day [39].

Windscale accident workers

The Windscale reactors, also called 'Piles', used uranium metal as fuel, were moderated by graphite, and were air-cooled. Their main purpose was the production of plutonium for the UK atomic weapons program [40]. Pile No. 1, on which the accident mentioned below happened, was operational in October 1950 [41]. Unfortunately, a fire broke out in the reactor core on 10 October 1957, resulting in a partial core meltdown [40]. McGeoghegan and Binks [42] documented the recorded external doses of the 471 workers who were involved in Windscale fire activities. For October 1957, the median dose was 3.52 mSv, and the maximum dose was 43.93 mSv [42].

Populations exposed to man-made contamination

Techa River population exposure in the Southern Urals

In the early phase of the Soviet Union's weapons program, liquid radioactive waste produced in the course of the program at the Mayak PA was released into the nearby Techa River. As a consequence, the population living in downstream villages was exposed to ionizing radiation, either externally or internally through radionuclide incorporation. The releases began in 1949 and peaked in the early 1950s. Based on the currently used Techa River Dosimetry System (TRDS) 2009, mean annual external doses to the red bone marrow (RBM) were highest in 1951, reaching almost 40 mGy, with maximum individual annual RBM doses reaching ~220 mGy. This would correspond—if the dose was continuously spread over the year—to a mean RBM dose rate of 4.3 μGy/h, with maximum individual values up to 25 μGy/h. As for internal RBM doses, mean annual doses were highest in 1951, with a mean value of 125 mGy and a maximum individual value of 2700 mGy, corresponding to dose rates of 14 μGy/h and 340 μGy/h, respectively. Mean cumulative RBM doses were 400 mGy, with individual doses up to 9000 mGy. These doses were used to quantify radiation-induced leukemia risks (e.g. Krestinina *et al.* [43]; M Degteva, priv. comm). A new dosimetry version (TRDS-2017) will provide further refinement of the doses and dose rates.

Population exposure after the Fukushima accident

During the Fukushima accident, considerable amounts of radionuclides—mainly radiocesium and radioiodine—were released into the environment. Among them were ^{137}Cs and ^{131}I, with estimated ranges of activities released into the atmosphere of 6–20 and 100–500 PBq, respectively. Other radionuclides released were less important in terms of their contribution to the human dose [44–46]. Adult members of the public in Japan who were not evacuated after the Fukushima accident, but continued to live in Fukushima prefecture, might have accumulated on average up to 4.3 mSv effective dose and up to 17 mGy thyroid dose, during the first year [44].

Individuals who lived within the 20 km evacuation zone or the deliberate evacuation area, received higher effective doses, but <10 mSv. For example, inhabitants of 'precautionary evacuated settlements' received up to 2.2 mSv effective dose before and during evacuation, while those of deliberately evacuated settlements received up to 8.5 mSv [44]. 'Precautionary evacuated settlements' refers to the evacuations instructed 12–15 March 2011 as an urgent protective measure. Thus, inhabitants of these settlements received their 2.2 mSv effective dose within a mean period of ~2 days. 'Deliberate evacuation' refers to evacuations performed between late March and 21 June 2011. Taking the 102 days between 11 March and 21 June, the average time after the accident for those deliberately evacuated was 51 days.

During the Fukushima accident, a maximum ambient dose rate of 12 mGy/h was automatically measured on 15 March at the main gate of the nuclear power plant. Measurements recorded by other automatic monitoring stations on site were generally lower. However, surveys performed between 20 March and 23 March indicated local spots with much higher dose rates [44]. In contrast, extensive measurements of ambient dose rate made at numerous locations outside the plant indicated much lower dose rates. For example, ambient dose rates reached ~25 μGy/h between 12 March 12 and 17 March, at Fukushima and Minamisoma, and continuously decreased thereafter [44]. These and other results suggest that typical dose rates to the population after the Fukushima accident were low (according to the UNSCEAR definition mentioned above).

Exposures after the A-bomb explosions over Japan

When the atomic bombs were dropped over Japan in August 1945, ~340 000 inhabitants were affected in Hiroshima. About 140 000 of these inhabitants had died by the end of 1945, due to blast wave, fire or radiation exposure. In Nagasaki, out of 270 000 inhabitants who stayed in the city at the time of the explosion, ~70 000 had died by end of 1945, for the same reasons.

As a result of the explosions, the populations of both cities were exposed to gamma and neutron radiation. For those who were close to the hypocenters, total kerma free-in-air (FIA) was up to 35 Gy of neutrons and 120 Gy of gamma-rays in Hiroshima (19 Gy and 328 Gy, respectively, in Nagasaki) [47]. Without heavy shielding and/or proper medical treatment, exposure to such dose levels is lethal (the LD_{50} dose for humans is ~3–4 Gy, which is the dose at which ~50% of exposed individuals will die without proper medical treatment [48]). For Hiroshima and Nagasaki, the FIA kerma reached levels of 4.5 and 8.7 Gy at 1000 m from the hypocenters. Beginning at this distance it was possible to survive 1) in the open, 2) inside wooden houses, and 3) outside but partially shielded by wooden houses (if one did not succumb to thermal injuries to exposed skin or blast injuries) and, accordingly, the cohort of A-bomb survivors which has been investigated for radiation-induced late-effects were mostly exposed at distances larger than ~1000 m from the hypocenters. Thus, cumulative gamma and neutron FIA kerma values for the A-bomb survivors span a dose range of a few milliGray or less [for those who were far away from the hypocenters at the time of bombing (ATB)] to several Gy (for those who were at distances of ~1000 m from the hypocenters ATB [49, 50]. A small number of survivors included in the risk studies, likely closer than 1000 m and supposedly not protected by heavy shielding (~280), have estimates of total shielded kerma of >4 Gy, ranging up to several tens of Gray, but these estimates are truncated to 4 Gy. Other survivors with 'heavy shielding' are not included in the risk estimation, because their doses cannot yet be calculated accurately. Some of the heavily shielded survivors were located even closer to the hypocenter ATB.

The emitted radiation consisted of five major components listed at the time the survivor was exposed: prompt primary gamma radiation, prompt neutron radiation, prompt secondary gamma radiation, delayed gamma radiation, and delayed neutron radiation. Prompt primary gamma and neutron radiation from the fission processes occurring during the explosions were emitted within <1 μs after detonation, while the bombs were structurally still intact, and reached the ground almost immediately (within less than ~1 μs for photons, and less than ~10 μs for neutrons) [47]. As these emissions ceased when the fissioning material had thermally expanded just enough to go subcritical and was no longer able to sustain the chain reaction, the duration of these emissions was less than ~1 μs [51]. Note that prompt gamma radiation also included secondary gamma radiation produced by prompt neutrons interacting with the atmosphere and the soil. This was from inelastic scattering of fast neutrons up until 100 μs, and from thermal neutron capture in air and ground, ending at ~0.2 s. For simplification, the average dose rate is defined over the duration of each component, even though dose rates decrease substantially towards the end of its duration. For Hiroshima at 1000 m from the hypocenter, where prompt primary gamma radiation kerma with no shielding was ~70 mGy, and at 2000 m ~2 mGy, typical dose rates were between 70 mGy/1 μs = 7×10^4 Gy/s and 2 mGy/1 μs = 2×10^3 Gy/s (assuming a spread in time of the gamma pulse at the ground of 1 μs) ([47], chapter 3, Table 11). For prompt neutrons, FIA kerma was ~0.24 Gy at 1000 m and 0.4 mGy at 2000 m, corresponding to dose rates of between 0.24 Gy/10 μs = 2.4×10^4 Gy/s and 0.4 mGy/10 μs = 40 Gy/s (assuming that the spread in time of fast neutrons reaching the ground is also of the order of 10 μs). Finally, for prompt secondary gamma radiation, FIA kerma was ~1.38 Gy at 1000 m and 35 mGy at 2000 m, corresponding to dose rates of between 1.38 Gy/0.2 s = 6.9 Gy/s and 34 mGy/0.2 s = 0.17 Gy/s (assuming that the spread in time of the thermal neutrons being captured in the air and ground ends at 0.2 s) [51]. Since survivors typically had their doses reduced by a factor of ~0.4 by shielding from houses, whether they were inside or outside, the shielded doses and dose rates were correspondingly smaller.

Delayed gamma rays and neutrons originated from fission products in the fireball. The ground-level arrival of delayed radiation is governed by the development of the ground-reflected fireball with time, which in turn is influenced by atmospheric rearrangements, due to changes in air temperature and density (pressure) during the explosions. The delayed radiation dose rate received at ground level was at first almost constant, because the fireball expanded while the fission products decayed. After a few seconds, the overall dose rate decreased rapidly as the fireball rose into the upper atmosphere due to the ground-reflected shockwave and thermal convection.

Table 1. Summary of dose rates and cumulated doses as estimated in the present study

Samples/Animals/ Human cohorts	Mean dose rate (range)[a]	Cumulative dose	Reference	Dose quantity	Remark
Cellular experiments	1000–60 000 μGy/h	1 mGy–8 Gy	[16]	Absorbed dose	Various endpoints including chromosome protein foci assays, chromosomal aberrations, and mutations
Animal experiments					
Mice	780 μGy/h–22.6 Gy/h	0–49 Gy	[17]	Absorbed dose	US Janus database
Rats/mice	1350 μGy/h–240 Gy/h	20 mGy–68.2 Gy	[18]	Absorbed dose	European ERA data base
Mice	2, 42, 830 μGy/h	20, 400, 8000 mGy	[19]	Absorbed dose	Japanese IES experiment
Human cohorts					
General population	0.3 (0.1–1) μSv/h	192 (80 800) mSv	[20]	Effective dose	Calculated from annual effective dose for world population; cumulative life-time doses assume an age of 80 years
HRBA population, India	<1 μGy/h	161 mGy	[22]	Absorbed dose to colon	Mean dose for cohort; dose rate estimate based on measurement of a randomly selected subset of the cohort
HRBA population, China	<0.24 μSv/h	84.4 mGy	[23–25]	Absorbed dose to colon; effective dose rate	Mean colon dose for HRBA cohort; dose rate estimate based on Yuan *et al.* [25], Morishima *et al.* [24]
Air crew	2 (<6) μSv/h	<200 mSv	[28–30]	Effective dose	Dose rate estimate based on mean annual effective dose and assumed 900 flight hours per years; cumulative dose assumes 40 years of work
Astronauts	~18 μGy/h	4.3 mSv	[31]	$H_p(10)$, film badge	Dose rate and cumulative dose for 10 days Apollo mission
Mayak workers	<150 μGy/h	510 (0–6800) mGy	[35]	$H_p(10)$, film badge	Dose rate estimated based on annual dose and assuming 2000 working hours per year
Chernobyl clean-up workers	320 μGy/h	160 mGy	[39]	$H_p(10)$, film badge	Dose rate and cumulative dose for the first year after the accident; dose rate calculated based on individual time of employment and assumed continuous exposure
Windscale workers		3.5 (<43.9) mSv	[42]	Effective dose	

Techa population	External: 4.3 (<25) μGy/h Internal: 14 (<340) μGy/h	400 (0–9000) mGy (external + internal)	[43]; M Degteva, priv. comm.	Red bone marrow dose	Dose rate estimates based on highest annual dose (in 1951) assuming chronic exposure
Fukushima: prefecture, not evacuated		<4.3 mSv (during first year)	[44]	Effective dose	
Fukushima: precautionary evacuated settlements		<2 mSv (before and during evacuation)			
Fukushima: deliberately evacuated settlements		<8.5 mSv			

HBRA = high background radiation area; $H_p(10)$ = personal dose equivalent; LSS = Life Span Study; [a]doses rates are given in units of μGy/h or Gy/h, for ease of comparison; priv. comm. = private communication. Note: UNSCEAR considers a dose rate of <6000 μGy/h, and a dose of <100 mGy as being low [11]. Note also that individual workers—depending on their profession—might have accumulated their recorded doses in a shorter time, i.e. that the assumption of continuous exposure over the whole working time may not always hold. In such cases, dose rates higher than estimated might have occurred.

Typically, and for Hiroshima at a distance from the hypocenter of 1500 m, exposure due to the delayed gamma and neutron radiation was not much longer than 10 s [57]. For a distance of 1000 m and a FIA kerma from delayed gamma rays with no shielding of 2.77 Gy and from delayed neutrons of 17.7 mGy, corresponding dose rates were of the order of 2.77×10^{-1} Gy/s for gamma rays, and 1.77×10^{-3} Gy/s for neutrons (assuming an exposure time of 10 s). At a distance of 2000 m from the hypocenter, the corresponding dose rates were 3.96×10^{-2} Gy/10 s = 3.96×10^{-3} Gy/s and 1.24×10^{-5} Gy/10 s = 1.24×10^{-6} Gy/s. Again, these numbers were typically reduced somewhat by shielding.

To summarize the previous two sections, at a distance of 1000 m from the hypocenter in the open in Hiroshima, delayed gamma radiation dominated FIA kerma (2.77 Gy), followed by prompt secondary gamma radiation (1.38 Gy) and prompt primary gamma radiation (0.071 Gy), followed by prompt neutrons (0.24 Gy) and delayed neutrons (0.0177 Gy) ([47], Chapter 3, Table 11). The corresponding values for Nagasaki are 4.61 Gy, 2.48 Gy, 1.52 Gy, 0.098 Gy and 0.026 Gy, respectively ([47], Chapter 3, Table 13). Therefore, the total gamma FIA kerma by far exceeded that from neutrons. Note, however, that if neutron doses are weighted by a factor 10, to take account of the relative biological effectiveness of neutron compared with gamma radiation (as done in recent analyses of cancer mortality and incidence among atomic bomb survivors [52, 53], the FIA neutron radiation at ~1000 m in Hiroshima becomes nearly half as important as the gamma radiation, but this importance is reduced for dose to the survivor's organs, which are shielded by body tissue, as neutrons are far more strongly absorbed by tissue than gamma-rays, or as distance to the hypocenter increases, as the neutron/gamma ratio in the FIA radiation decreased rapidly with distance from the hypocenter. We note also that the evidence for higher values for the relative biological effectiveness of neutrons in Hiroshima and Nagasaki has been emphasized [54–56].

The estimates given above do not include gamma radiation originating from neutron activation of the ground, which dominates the dose of those 'early entrants' who entered the cities immediately after the bombing (i.e. well after 1 min and therefore after the prompt and delayed irradiation from the bombs and the fission debris in the fireballs). Imanaka and co-workers estimated ground dose rates of 6 and 4 Gy/h (1.7×10^{-3} Gy/s and 1.1×10^{-3} Gy/s) at the hypocenter at 1 min after the explosion in Hiroshima and Nagasaki, respectively [57]. These dose rates had decreased rapidly, by a factor of 1000, 1 day later, and by a factor of 1 million 1 week later (more rapidly than fission product decay). Imanaka *et al.* [58] calculated doses ranging from 2.6 to 24 mGy for four early entrants whose time-and-location data were established by detailed interviews, and who passed close to the hypocenter in Hiroshima on the day of the bombing or the next day. Doses and health effects from this or other residual radiations are not discussed in more detail in the present review, given the fact that these are currently being re-evaluated [59].

SUMMARY AND CONCLUSIONS

Table 1 summarizes the values for cumulative doses and dose rates estimated for various exposure scenarios. For comparison, Table 2

Table 2. Summary of dose rates and cumulative doses as estimated for the Japanese A-bomb survivors

Cohort	Mean dose rate (range)	Cumulative dose	Reference	Dose quantity	Remark
LSS A-bomb survivors, all radiation sources		114 mGy (0–3388 mGy)	Based on Cullings *et al.* [50]	Weighted colon dose	All numbers for Hiroshima; similar estimates hold for Nagasaki
LSS A-bomb survivors: prompt radiation	Prim. γ, 1000 m: 7×10^{4} Gy/s Prim. γ, 2000 m: 2×10^{3} Gy/s Sec. γ, 1000 m: 6.9 Gy/s Sec. γ at 2000 m: 0.17 Gy/s n at 1000 m: 2.4×10^{4} Gy/s n at 2000 m: 40 Gy/s	70 mGy 2 mGy 1.38 Gy 35 mGy 240 mGy 0.4 mGy	[47, 51]	Kerma free-in-air	All numbers for Hiroshima; similar estimates hold for Nagasaki; prompt primary gamma pulse assumed to last 1 μs; Prompt secondary gamma pulse assumed to last 0.2 s; prompt neutron pulse assumed to last 10 μs
LSS A-bomb survivors: delayed radiation	γ at 1000 m: 2.77×10^{-1} Gy/s γ at 2000 m: 3.96×10^{-3} Gy/s n at 1000 m: 1.77×10^{-3} Gy/s n at 2000 m: 1.24×10^{-6} Gy/s	2.77 Gy 39.6 mGy 17.7 mGy 0.0124 mGy	[47, 51]	Kerma free-in-air	All numbers for Hiroshima; similar estimates hold for Nagasaki; exposure due to delayed radiation assumed to last for 10 s.
Hiroshima, early entrants		<24 mGy	[58]	Kerma free-in-air	Estimates for Hiroshima, based on four example cases [58]; doses received within a few hours or days

LSS = Life Span Study; Prim. = primary; Sec. = secondary; dose rates are not given in terms of dose per hour as in Table 1, because exposure times due to the explosions over Hiroshima and Nagasaki were much shorter than 1 h; actual duration of radiation pulse for various sources of radiation is given in the last column.

summarizes cumulative doses and dose rates estimated for the A-bomb survivors in Hiroshima and Nagasaki, Japan.

It is clear from Table 1 that the experimental studies include dose rates and doses that span a wide range of values, and very often exceed the ranges typical for human exposures, as well as the ranges described by UNSCEAR as low dose rates and doses. We note, however, that the animal experiments previously and currently performed in Japan were designed to include values for dose rates and doses that are typical of some radiation protection scenarios. Exposures to very low dose-rates on a daily basis often involve higher accumulated doses; exposure facilities capable of carrying out these low dose-rate exposures are not commonly found, and this limits the number and type of such studies that can be performed.

Therefore, data from experimental studies (on cells or animals) have often been obtained at radiation doses and dose rates that are not typical of exposure scenarios relevant to radiation protection. Thus, extrapolation of radiation-induced effects obtained in these studies to humans for application in radiation protection should be done with care.

In contrast, most of the low-dose-rate epidemiological studies listed in Table 1 have dose rates directly relevant to radiation protection considerations and provide an important complement to the A-bomb survivor studies. The total dose rates received by the majority of exposed A-bomb survivors (at a distance of <2000 m from the hypocenters) due to prompt and delayed gamma and neutron radiation were much larger (in part because of the very short pulse duration) than those received by any other human cohort listed in Table 1. A comparison of cancer risk estimates from these cohorts with those from the A-bomb survivors, as recently described in [10], thus provides valuable information on the magnitudes and ranges of uncertainties in dose-rate effects relevant to human cancer risks after exposure to ionizing radiation. The study accounted for variations among studies in the distributions with respect to sex, ages at exposure and at observation, as well as in differences in types of dosimetry and study outcomes. The results—which particularly reflected a difference in risk estimates between the two most statistically influential studies, the Mayak and INWORKS studies—had a range of uncertainty between no reduction and a 2-fold reduction in excess risk per unit dose for the low-dose-rate studies compared with the A-bomb study excess risk.

ACKNOWLEDGEMENTS

The authors thank S.D. Egbert (Science Applications International Corporation, USA) for his comments on the dosimetry of the Japanese A-bomb survivors. RERF is a private, nonprofit foundation funded equally by the Japanese Ministry of Health, Labour and Welfare and the U.S. Department of Energy through the National Academy of Sciences. We thank the two referees for their detailed and helpful comments.

FUNDING

This work was supported by the Intramural Research Program of the National Institutes of Health, National Cancer Institute, Division of Cancer Epidemiology and Genetics.

REFERENCES

1. ICRP. *Task Group 91. Radiation Risk Inference at Low-dose and Low-dose Rate Exposure for Radiological Protection Purposes*. http://www.icrp.org/icrp_group.asp?id=83 (2 February 2018 date last accessed).
2. Rühm W, Woloschak GE, Shore RE et al. Dose and dose rate effects of ionizing radiation – a discussion in light of radiological protection. *Radiat Environ Biophys* 2015;54:379–401.
3. ICRP. The 2007 Recommendations of the International Commission on Radiological Protection. ICRP Publication 103. *Ann ICRP* 2007;37:1–332.
4. National Academy of Sciences (NAS), Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation NRC. *Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII – Phase 2*. Washington, DC: National Academy Press, 2006.
5. SSK. 2014. Dose- and dose-rate-effectiveness factor (DDREF), Recommendation by the German Commission on Radiological Protection with scientific grounds. http://www.ssk.de/SharedDocs/Beratungsergebnisse_E/2014/DDREF_e.html?nn=2876278 (2 February 2018, date last accessed).
6. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Epidemiological Studies of Radiation and Cancer. *UNSCEAR 2006 Report. Annex A*, pp. 13–322. New York: United Nations, 2008.
7. Rühm W, Azizova TV, Bouffler SD et al. Dose-rate effects in radiation biology and radiation protection. *Ann ICRP* 2016;45:262–79.
8. Haley B, Paunesku T, Grdina D et al. The increase in animal mortality risk following exposure to sparsely ionizing radiation is not linear quadratic with dose. *PLoS One* 2015;10:e0140989.
9. Rühm W, Eidemüller M, Kaiser JC. Biologically based mechanistic models of radiation-related carcinogenesis applied to epidemiological data. *Int J Radiat Biol* 2017;93:1093–117.
10. Shore RE, Walsh L, Azizova T et al. Risk of solid cancer in low dose-rate radiation epidemiological studies and the dose-rate effectiveness factor. *Int J Radiat Biol* 2017;93:1064–78.
11. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Biological mechanisms of radiation actions at low doses. A white paper to guide the Scientific Committee's future programme of work. *Report V.12-57831*. New York, 2012.
12. Rothkamm K, Löbrich M. Evidence for a lack of DNA double strand break repair in human cells exposed to very low x-ray doses. *Proc Natl Acad Sci U S A* 2003;100:5057–62.
13. Tanaka K, Kohda A, Satoh K. Dose-rate effects and dose and dose-rate effectiveness factor on frequencies of chromosome aberrations in splenic lymphocytes from mice continuously exposed to low dose rate gamma radiation. *J Radiol Prot* 2013;33:61–70.
14. Okudaira N, Uehara Y, Fujikawa K et al. Radiation dose-rate effect on mutation induction in spleen and liver *gpt* delta mice. *Radiat Res* 2010;173:138–47.
15. Manesh SS, Deperas-Kaminska M, Fotouhi A et al. Mutations and chromosomal aberrations in hMTH1-transfected and non-transfected TK6 cells after exposure to low dose rates of gamma radiation. *Radiat Environ Biophys* 2014;53:417–25.
16. Brooks AL, Hoel DG, Preston RJ. The role of dose rate in radiation cancer risk: evaluating the effect of dose rate at the molecular, cellular and tissue levels using key events in critical pathways following exposure to low LET radiation. *Int J Radiat Biol* 2016;92:405–26.
17. Grahn D, Wright BJ, Carnes BA et al. Studies of acute and chronic radiation injury at the biological and medical research division, Argonne National Laboratory, 1970–1992: the JANUS program survival and pathology data. Argonne, Illinois: Argonne National Laboratory, University of Chicago, 1995.
18. Birschwilks M, Gruenberger M, Adelmann C et al. The European radiobiological archives: online access to data from radiobiological experiments. *Radiat Res* 2011;175:526–31.
19. Tanaka S, Tanaka IB, Sasagawa S et al. No lengthening of life span of mice continuously exposed to gamma rays at very low dose rates. *Radiat Res* 2003;160:376–9.
20. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Sources and effects of ionizing radiation. *UNSCEAR 2000 Report to the General Assembly, with scientific annexes, Volume I: Sources*. Vol. E.00.IX.3. pp. 1–654. New York: United Nations, 2000.
21. Akiba S. Cancer risk associated with low-dose and low-dose-rate ionizing radiation exposure. *Genes Environ* 2013;35:80–7.
22. Nair R, Rajan R, Akiba S et al. Background radiation and cancer incidence in Kerala, India—Karunagappally cohort study. *Health Phys* 2009;96:55–66.
23. Tao Z, Akiba S, Zha Y et al. Cancer and non-cancer mortality among inhabitants in the high background radiation area of Yangjiang, China (1979–1998). *Health Phys* 2012;102:173–81.
24. Morishima H, Koga T, Tatsumi K et al. Dose measurement, its distribution and individual external dose assessments of inhabitants in the high background radiation areas in China. *J Radiat Res* 2000;41:9–23.
25. Yuan YL, Morishima H, Shen H et al. Recent advances in dosimetry investigation in the high background radiation area in Yangjiang, China. In: *High Levels of Natural Radiation*. Amsterdam: Elsevier; 1997, 223–33.
26. Richardson DB, Cardis E, Daniels RD et al. Risk of cancer from occupational exposure to ionising radiation: retrospective cohort study of workers in France, the United Kingdom, and the United States (INWORKS). *BMJ* 2015;351:h5359.
27. Bundesgesetzblatt (BGBl), German Radiation Protection Ordinance. Zweite Verordnung zur Änderung der Strahlenschutzverordnung. Bundesgesetzblatt Teil I 1989; 23 (in German) .
28. Bundesamt für Strahlenschutz (BfS). Bundesamt für Strahlenschutz/Fachbereich Strahlenschutz und Gesundheit, Autoren: G. Frasch, L. Kammerer, R. Karofsky, E. Mordek, A. Schlosser, J. Spiesl, Die berufliche Strahlenexposition in

Deutschland 2012: Bericht des Strahlenschutzregisters 2014; BfS-SG-22/14 (in German).
29. Mares V, Maczka T, Leuthold G et al. Air crew dosimetry with a new version of EPCARD. *Radiat Prot Dosimetry* 2009;136:262–6.
30. Bottollier-Depois JF, Beck P, Latocha M et al. Comparison of codes assessing radiation exposure of aircraft crew due to galactic cosmic radiation. *EURADOS Report 2012*; 2012-03, ISBN 978-3-943701-02-9.
31. Cucinotta FA, Hamada N, Little MP. No evidence for an increase in circulatory disease mortality in astronauts following space radiation exposures. *Life Sci Space Res* 2016;10:53–6.
32. Khokhryakov VV, Khokhryakov VF, Suslova KG et al. Mayak Worker Dosimetry System 2008 (MWDS-2008): assessment of internal dose from measurement results of plutonium activity in urine. *Health Phys* 2013;104:366–78.
33. Vasilenko EK, Khokhryakov VF, Miller SC et al. Mayak worker dosimetry study: an overview. *Health Phys* 2007;93:190–206.
34. ICRU. Conversion Coefficients for use in Radiological Protection Against External Radiation. *ICRU Report 57*. International Commission on Radiation Units and Measurements, Bethesda, MD, 1998.
35. Hunter N, Kuznetsova IS, Labutina EV et al. Solid cancer incidence other than lung, liver and bone in Mayak Workers: 1948–2004. *Br J Cancer* 2013;109:1989–96.
36. Khokhryakov V, Suslova K, Aladova E et al. Development of an improved dosimetry system for the workers at the Mayak Production Association. *Health Phys* 2000;79:72–6.
37. Azizova TV, Haylock RGE, Moseeva MB et al. Cerebrovascular diseases incidence and mortality in an extended Mayak worker cohort 1948–1982. *Radiat Res* 2014;182:529–44.
38. Bouville A, Kryuchkov V. Increased occupational radiation doses: nuclear fuel cycle. *Health Phys* 2014;106:259–71.
39. Ivanov VK, Chekin SU, Maksoutov MA et al. Radiation Risk of Incidence of Hypertensia among Russian Recovery Operation Workers of the Chernobyl Accident. *Med Radiol Radiat Saf* 2017;62:32–7. [in Russian].
40. Arnold L. *Windscale 1957: Anatomy of a Nuclear Accident*. New York: Palgrave McMillan, 2007.
41. Wakeford R. The Windscale reactor accident—50 years on. *J Radiol Prot* 2007;27:211–5.
42. McGeoghegan D, Binks K. Mortality and cancer registration experience of the Sellafield employees known to have been involved in the 1957 Windscale accident. *J Radiol Prot* 2000;20:261–74.
43. Krestinina LY, Davis FG, Schonfeld S et al. Leukemia incidence in the Techa River Cohort: 1953–2007. *Br J Cancer* 2013;109: 2886–93.
44. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Levels and effects of radiation exposure due to the nuclear accident after the 2011 great East-Japan earthquake and tsunami. *UNSCEAR 2013 Report (Volume I)*, 2014.
45. World Health Organization (WHO). *Preliminary Dose Estimation from the Nuclear Accident after the 2011 Great East Japan Earthquake and Tsunami*. Geneva, 2012.
46. World Health Organization (WHO). *Preliminary Health Risk Assessment from the Nuclear Accident after the 2011 Great East Japan Earthquake and Tsunami*. Geneva, 2013.
47. Young RW, Kerr GD (eds). Reassessment of the atomic bomb radiation dosimetry for Hiroshima and Nagasaki – Dosimetry System 2002. *Report of the Joint US–Japan Working Group*. Radiation Effects Foundation, Hiroshima, Japan, 2005.
48. Mole RH. The LD50 for uniform low LET irradiation of man. *Br J Radiol* 1984;57:355–69.
49. Cullings HM, Fujita S, Funamoto S et al. Dose estimation for atomic bomb survivor studies: its evolution and present status. *Radiat Res* 2006;166:219–54.
50. Cullings HM, Grant EJ, Egbert SD et al. DS02R1: Improvements to atomic bomb survivors' input data and implementation of Dosimetry System 2002 (DS02) and resulting changes in estimated doses. *Health Phys* 2017;112:56–97.
51. Glasstone, S. *The Effects of Nuclear Weapons*, 3rd edn. U.S. Departments of Defense and Energy, 1977.
52. Grant EJ, Brenner A, Sugiyama H et al. Solid cancer incidence among the life span study of atomic bomb survivors: 1958–2009. *Radiat Res* 2017;187:513–37.
53. Ozasa K, Shimizu Y, Suyama A et al. Studies of the mortality of atomic bomb survivors, Report 14, 1950–2003: an overview of cancer and noncancer diseases. *Radiat Res* 2012;177:229–43.
54. Kellerer AM, Rühm W, Walsh L. Indications of the neutron effect contribution in the solid cancer data of the A-bomb survivors. *Health Phys* 2006;90:554–64.
55. Rühm W, Walsh L. Current risk estimates based on the A-bomb survivors data – a discussion in terms of the ICRP recommendations on the neutron weighting factor. *Radiat Prot Dosimetry* 2007;126:423–31.
56. Walsh L. Neutron relative biological effectiveness for solid cancer incidence in the Japanese A-bomb survivors – an analysis considering the degree of independent effects from γ-ray and neutron absorbed doses with hierarchical partitioning. *Radiat Environ Biophys* 2013;52:29–36.
57. Imanaka T, Endo S, Tanaka K et al. Gamma-ray exposure from neutron-induced radionuclides in soil in Hiroshima and Nagasaki based on DS02 calculations. *Radiat Environ Biophys* 2008;47:331–6.
58. Imanaka T, Endo S, Kawano N et al. Radiation exposure and disease questionnaires of early entrants after the Hiroshima bombing. *Radiat Prot Dosimetry* 2012;149:91–6.
59. Kerr GD, Egbert SD, Al-Nabulsi I et al. Workshop report on atomic bomb dosimetry: review of dose related factors for the evaluation of exposures to residual radiation at Hiroshima and Nagasaki. *Health Phys* 2015;109:582–600.