

# Atomic Radiation Exposure in Australian Service Personnel in Hiroshima, Japan

# A Report by

# the Australian Radiation Protection and Nuclear Safety Agency

to the

Department of Veterans' Affairs

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#### HIROSHIMA

#### **EXECUTIVE SUMMARY**

This report is divided into several sections. The first section gives the general background information relevant to the problem, and includes a brief summary of the physical processes associated with a typical nuclear explosion and the resulting sources of contamination. An outline of some earlier dose/risk assessments for the Hiroshima area is then given. An outline of dosimetric principles, natural background radiation levels and current recommendations for dose limitation is given in Appendix 1.

Some specific exposure scenarios are discussed in Appendix 2, including external radiation from induced radioactivity (Appendix 2.1), external radiation from fall-out (Appendix 2.2), the potential radiological hazards associated with a souvenired item containing induced radioactivity (Appendix 2.3), a general discussion of potential internal exposures (Appendix 2.4), and a detailed discussion of the potential radiological hazards associated with strontium-90 (Appendix 2.5). A summary of the total committed equivalent dose to bone marrow and total committed effective dose for exposure periods of 6 months, 1 year and 2 years is given in Appendix 2.6. In most cases the conditions under which significant exposures could have occurred are shown to have a very low probability of occurrence. Where the probability of occurrence is potentially significant, an indication of the measurements needed to establish whether the exposure may have occurred is given.

Two Japanese and two American surveys of residual contamination at Hiroshima were carried out between the time of the bomb detonation and the arrival of Australian personnel at Hiroshima (Table 3). The results of these surveys were consistent with each other, in terms of both the location of the residual contamination and the resulting radiation. The survey results were also consistent with calculated estimates of the total amount of fission products produced by the bomb detonation, and with studies of Hiroshima soil samples, roof tiles and bricks that were irradiated with high neutron fluxes to produce neutron activation products.

The dose calculations presented in this report were based on an upper limit estimate of the residual contamination at Hiroshima during the period of occupation by Australian personnel. This upper limit was determined from the results of all the surveys mentioned in the previous paragraph.

The estimated maximum cumulative dose from external exposure received by a person located at the Hiroshima hypocentre from February  $1^{st}$ , 1946, assuming continuous occupation (24 hours per day, 7 days per week) for two years, was approximately 495  $\mu$ Sv. For exposure for 12 hours per day, 3 days per week for two years from February  $1^{st}$ , 1946, the estimated maximum cumulative dose from external exposure was approximately 110  $\mu$ Sv.

The effective dose rate, from external exposure, to a person located in the Koi-Takasu area on February 1<sup>st</sup>, 1946 was approximately 0.38 μSv h<sup>-1</sup>.

The cumulative dose, due to external exposure, received by a person located in the Koi-Takasu district from February 1<sup>st</sup>, 1946 to February 1<sup>st</sup> 1948, for 24 hours per day, 7 days per week, would have been no higher than 6.7 mSv.

By February 1<sup>st</sup>, 1946, the external dose rate from a 1 kg vitrified object, taken from the vicinity of the Hiroshima hypocentre, would have been very much less than that from 1 kg of normal (uncontaminated) soil.

The effective dose resulting from internal exposures at Hiroshima, due to the consumption of a diet consisting entirely of contaminated seafood, fruits, grains, vegetables and drinking water, is unlikely to have exceeded 0.67 mSv.

If all the drinking water consumed by the occupation troops over two years was drawn from a dam with a catchment area uniformly contaminated to the level measured in the Koi-Takasu area, the resulting effective dose is unlikely to have exceeded 0.35 mSv. (Appendix 2.4.3)

If an individual spent 12 months working in the fall-out area (Koi-Takasu), engaged in rubble clearing work or other tasks that may have produced similar levels of dust, for 8 hours per day, every day of the year, it may have been possible to incur a committed effective dose of 10 mSv via inhalation, or approximately 0.03 mSv per day. Calculations indicate that if this did occur the strontium level in a daily urine sample may be detectable today. The total amount of excreted strontium (in urine) could be increased by accumulating urine samples. The ratio of the strontium activity in bone to the strontium activity in urine would also be a crude indicator of the time of intake (for ingestion of strontium). This would involve measurements of strontium in bone, which would be possible via biopsy techniques. A comparison of the results of such measurements (urine and/or bone) with the results of measurements taken on a control group (see earlier) would also be a useful indicator.

Ingestion of  $^{90}$ Sr at a rate which would have led to a committed effective dose of 10 mSv to an individual would result in a rate of excretion of activity in a daily urine sample which should be measurable today, but the daily excretion rate would be similar in magnitude to the excretion rate resulting from the average intake of  $^{90}$ Sr from atmospheric weapons testing fall-out. The  $^{90}$ Sr  $^{90}$ Y activity in the skeleton from the Hiroshima intake would however be approximately 5-10 times higher than the corresponding average activity in the skeleton from the intake of  $^{90}$ Sr from atmospheric weapons testing fall-out.

Calculations of the amounts of strontium that would have to have been taken into the body by inhalation or ingestion at Hiroshima indicate that these intakes would only have occurred under very unusual conditions. The dose per unit intake for entry to the body via injection (open wound) is higher than that for intake by inhalation or ingestion, but the conclusion would be the same.

#### INTRODUCTION

# **General Background**

# Nuclear explosion

A nuclear explosion of the type at Hiroshima is produced by the fission of uranium. This fission process is the result of a chain reaction initiated and sustained by neutrons colliding with uranium nuclei. The uranium is a mixture of <sup>238</sup>U and <sup>235</sup>U. The natural concentration of <sup>235</sup>U has to be enriched to enhance neutron production. The fission process produces other radionuclides (fission products) which are radioactive.

The initial explosion is accompanied by a blast wave, which is followed by the formation of a fireball. The initial blast is accompanied by a very high neutron flux. The neutrons that penetrate the soil can interact with nuclides in the soil to produce "activation products". Some of these activation products are radioactive. Since the neutron flux falls off as the square of the distance from the detonation point, the concentration of activation products also decreases rapidly as the distance (on the ground) from the hypocentre increases.

The fireball is intensely hot and rises rapidly. It also produces extremely large updraughts of hot air which tend to entrain colder air from the surrounding atmosphere. The entrainment process also draws in loose material on the ground surface near the detonation point or hypocentre. This material is drawn up into the fireball and transported rapidly upwards. Radioactive fission products can become attached to this material and can return to the ground surface as fall-out. In most cases the upward transport is sufficient to inject a considerable fraction of this radioactive debris into the stratosphere. This material can remain in the stratosphere for long periods (up to several months or even years) before returning to the ground surface. This explains the world-wide distribution of fall-out from post-1945 atmospheric nuclear weapons testing programs.

Dosimetry, Natural Background, Dose Limitation These topics are discussed in Appendix 1.

# Hiroshima

A report (McRaney and McGahan, 1980) by the United States Defence Nuclear Agency (DNA) stated that an atomic bomb was detonated over Hiroshima, Japan, on 6 August 1945 at an altitude of 1670ft (510m). The same report stated that the blast resulted in "massive destruction but relatively small areas of significant residual radioactive contamination". The bomb was a gun-assembly type that employed <sup>235</sup>U as the fissionable material and had a yield equivalent to approximately 13 kT (McRaney and McGahan, 1980), or 15 kT (Roesch, 1987; Shigematsu et al, 1985) of TNT. Approximately half an hour after the detonation, eyewitnesses reported that "black rain" fell in the Koi-Takasu area west of the city.

The purpose of the Defence Nuclear Agency's report (McRaney and McGahan, 1980) was to present estimates of radiation doses received by U.S. occupation forces in Hiroshima. The doses were estimated from contributions from residual radioactive contamination produced by the nuclear detonation, including those due to the inhalation and ingestion pathways. The estimates were expressed as reasonable upper limits derived from a "worst-case" scenario.

A comprehensive, joint USA-Japan review of the Hiroshima dosimetry was published in 1987 (Roesch et al, 1987). This review included estimates of bomb yield, a summary of measurements of external gamma dose rates and soil contamination levels and a revision of radiation dosimetry. The International Commission on Radiological Protection revised its recommended risk per unit exposure factors on the basis of the Roesch report. The work presented in this document also makes extensive use of the Roesch report.

It was noted (Roesch et al, 1987, p18): that no bomb of the type used at Hiroshima was ever tested. Therefore any assessment of the effects of the residual contamination from the Hiroshima explosion have to be based on calculations and local measurements.

#### Contamination

The Manhattan Engineer District (of the US Defence Nuclear Agency) conducted radiological surveys in Hiroshima from 3-7 October 1945 (Tybout, 1946; McRaney and McGahan, 1980). The group reported that the radiation levels in Hiroshima were very low and that these would not present a hazard to the occupation forces (McRaney and McGahan, 1980). The Naval Medical Research Institute (NMRI) conducted a later survey from 1-2 November 1945 (Pace and Smith,1959; McRaney and McGahan, 1980). DNA found it difficult to arrive at consistent conclusions based on a comparison of the two surveys. However, they did recognise two important points:

- 1. Both of the surveys identified two distinct areas of contamination one centred around ground zero and the other some distance downwind.
- 2. Both of the surveys indicated (very) low residual radiation levels (in general < 1mR hr<sup>-1</sup>).

As the weapon was detonated above 1600ft, fall-out in the vicinity of ground zero was thought to be extremely unlikely (McRaney and McGahan, 1980). The contamination in the ground zero area resulted primarily from activation of the soil and building materials by neutrons released at detonation rather than from fall-out. Comparisons with Nevada Test Site data showed that the roughly circular contamination patterns around ground zero at Hiroshima were typical of neutron-induced fields produced by a high altitude detonation (McRaney and McGahan, 1980).

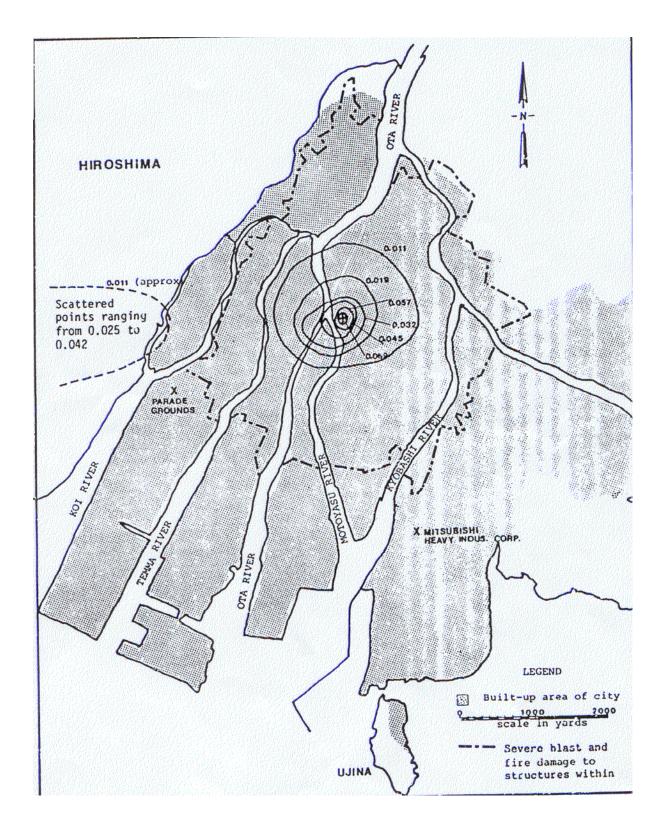
The contamination downwind resulted from the deposition of airborne fission products (and possibly radionuclides produced by neutron activation of atmospheric nuclides) on the ground (fall-out). DNA found from their survey that the decay of fission products was consistent with the "t<sup>-1.2</sup> rule" established for fall-out up to 4000 hours after detonation (McRaney and McGahan, 1980). Beyond 4000 hours the decay was found to depend (approximately) on the time since detonation as t<sup>-2.2</sup> (McRaney and McGahan, 1980).

McRaney and McGahan (1980) reported that the section of the city with a radiation level greater than 0.1mR/hr (at the time of the surveys) comprised less than one percent of the entire built-up area. They also reported that measurable contamination was recorded in only fifty percent of the built-up area.

# Occupation by Australian Personnel

McRaney and McGahan (1980) stated that on 6 March 1946, the 2nd Battalion of the U.S. 34<sup>th</sup> Regiment, including G Company, was relieved by the 67<sup>th</sup> Australian Infantry Battalion

of the 34<sup>th</sup> Australian Infantry Brigade of the British Commonwealth Occupation Forces. Therefore the Australians arrived 211 days after the bomb detonation. The U.S. 34<sup>th</sup> Regiment had been scattered over a very large area with only the one unit, G Company, in the immediate vicinity of Hiroshima. G Company had been quartered on the island of Ujina, approximately 4.5km south of ground zero. Figure 1 shows a map of Hiroshima. The two contaminated areas are shown clearly. Koi-Takasu is the area on the west side of the Koi River. The location of Ujina Island is also shown. The shaded portion of the map corresponds to the built up areas of the city. The contours shown on the map (in mR hr<sup>-1</sup>) are taken from the NMRI survey of 1-2 November, 1945.



**Figure 1:** A map of Hiroshima (McRaney and McGahan, 1980). The contours show external dose rate in mR hr<sup>-1</sup>.

McRaney and McGahan (1980) noted that all known locations for headquarters, command posts, and billets were outside the radiologically contaminated areas. Exposure could have occurred during any periodic trips into these areas, whether they were official or unofficial.

Exposures could have occurred as a result of external radiation from radionuclides on the ground surface (fall-out) or radionuclides in the soil (neutron activation products). Handling of contaminated equipment or suspension of contaminated dust during demolition, clearing and reconstruction of built up areas could also have led to external exposures from radionuclides in air or on the skin, or internal exposures resulting from the inhalation of contaminated dust. Another potential internal exposure pathway would be due to the consumption of contaminated water or food. It would seem reasonable to assume that local water and food supplies were not readily available (from the contaminated areas) due to the disruption caused by the bomb, and that, therefore, the bulk of the water and food consumed by the occupation force personnel came from outside the contaminated areas. This does not exclude the possibility of consumption of local seafood (fish, shellfish, etc.) that may have been contaminated as a result of surface run-off. However, such foods would have comprised only a small fraction of the total dietary consumption, and this, combined with the relatively short time of occupation, would imply a relatively low dose via this pathway.

# Initial activity

This was deduced by measurements of the long-lived fission and neutron activation products at different times after the bomb detonation. The results of these measurements were corrected for radioactive decay to determine their activities at some selected "initial" time. Since the black rain (assumed to be the main source of fall-out) fell approximately half an hour after the detonation over Hiroshima, the initial time was chosen to be one hour. The activities of the short-lived radionuclides at the initial time have to be inferred from measurements of test detonations of nuclear devices, but are irrelevant for this study because the only radionuclides that would contribute to the dose after 212 days are those that could be measured at that time.

Table 1 below gives the half-lives, fraction remaining after 212 days, integrated activity over a period of one year (starting at approximately the time when the Australians arrived in Hiroshima) and dose rate coefficients for ground surface and soil contamination for a number of radionuclides that are considered to be important in this type of assessment. The dose rate coefficients are taken from Eckerman and Ryman (1993)

Table 1:

Nuclide	Half-life	Units	Remaining	Integral of	Dose rate	Dose rate
			fraction after	fractional	coefficient for	coefficient for
			212 days	activity from	surface	soil (bulk)
				212 days for	contamination	contamination
				approximatel		
				y 1 year		
					Sv (Bq s m <sup>-2</sup> ) <sup>-1</sup>	Sv (Bq s m <sup>-3</sup> ) <sup>-1</sup>
Al-28	2	min	0.000	0.000	1.62×10 <sup>-15</sup>	9.32×10 <sup>-17</sup>
Mn-56	2.6	hr	0.000	0.000	1.58×10 <sup>-15</sup>	5.92×10 <sup>-17</sup>
Si-31	2.6	hr	0.000	0.000	3.01×10 <sup>-18</sup>	7.58×10 <sup>-20</sup>
K-42	12.4	hr	0.000	0.000	2.66×10 <sup>-16</sup>	1.01×10 <sup>-17</sup>
Na-24	15	hr	0.000	0.000	3.61×10 <sup>-15</sup>	1.52×10 <sup>-16</sup>
P-32	14.32	day	3.50×10 <sup>-5</sup>	7.40×10 <sup>-4</sup>	2.91×10 <sup>-18</sup>	6.31×10 <sup>-20</sup>
Fe-59	44.5	day	3.68×10 <sup>-2</sup>	2.37	1.12×10 <sup>-15</sup>	4.09×10 <sup>-17</sup>
Sc-46	83.8	day	1.73×10 <sup>-1</sup>	2.00×10 <sup>1</sup>	1.93×10 <sup>-15</sup>	6.79×10 <sup>-17</sup>
Ca-45	163	day	4.06×10 <sup>-1</sup>	$7.55 \times 10^{1}$	4.61×10 <sup>-20</sup>	3.35×10 <sup>-22</sup>
Cs-134	2.1	yr	8.26×10 <sup>-1</sup>	$2.57 \times 10^{2}$	1.52×10 <sup>-15</sup>	5.07×10 <sup>-17</sup>
Co-60	5.3	yr	9.27×10 <sup>-1</sup>	$3.18 \times 10^{2}$	2.35×10 <sup>-15</sup>	8.68×10 <sup>-17</sup>
Eu-152	13.33	yr	9.70×10 <sup>-1</sup>	$3.46 \times 10^2$	1.10×10 <sup>-15</sup>	3.75×10 <sup>-17</sup>
Sr-90	29.12	yr	9.86×10 <sup>-1</sup>	$3.57 \times 10^2$	2.84×10 <sup>-19</sup>	3.77×10 <sup>-21</sup>
Cs-137	30	yr	9.87×10 <sup>-1</sup>	$3.57 \times 10^2$	2.85×10 <sup>-19</sup>	4.02×10 <sup>-21</sup>

The two graphs below show the decay curves for the nuclides listed in the tables. The first graph (Figure 2) shows the decay for the first 10 days.

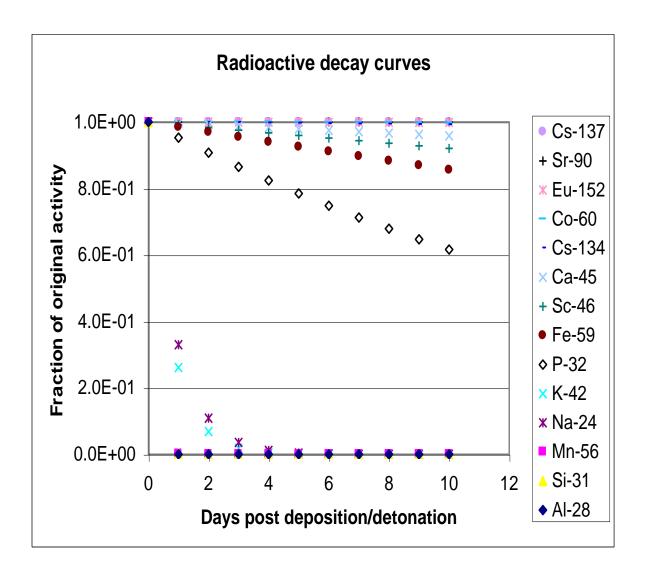


Figure 2:

Figure 2 clearly shows the rapid decay of the very short-lived radionuclides <sup>28</sup>Al, <sup>56</sup>Mn, <sup>42</sup>K, <sup>31</sup>Si, and <sup>24</sup>Na. The order of the keys in the legend is the same as the order (top to bottom) of the curves in the graph.

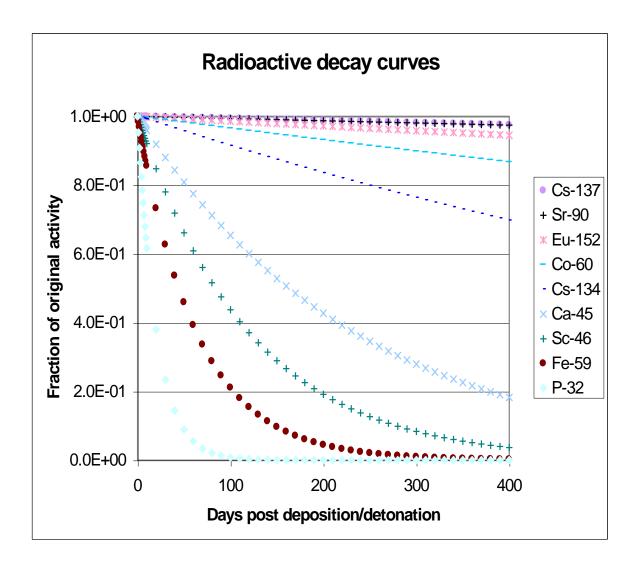


Figure 3:

Figure 3 shows the decay curves for the longer-lived radionuclides as a function of time out to 400 days post detonation/deposition. The order of the keys in the legend is the same as the order (top to bottom) of the curves in the graph.

### Determination of dose

McRaney and McGahan, (1980) calculated the ingestion and inhalation doses from the fall-out area for Nagasaki only, as they considered the comparative estimates for Hiroshima would be considerably smaller. They calculated inhalation doses indirectly from estimates of the amount of soil contamination at the earliest date of occupation by U.S. personnel. To calculate the airborne concentration a resuspension factor was applied, which is the ratio of the activity of material in the air to that on the ground surface. A high resuspension factor of  $1 \times 10^{-4} \text{m}^{-1}$  was chosen for the area around ground zero due to some dust-producing activities by the U.S. occupation forces. This assumed that the top centimetre of soil or building material was available for resuspension. A value of  $1 \times 10^{-5} \text{m}^{-1}$  was selected for the fall-out area. These resuspension factors were considered conservative given the amount of rain that had fallen in the area after the explosion and before occupation. 900 millimetres of rain fell

in Hiroshima during the three months following the bomb detonation. In addition, two typhoons hit Hiroshima on  $17^{th}$  September, 1945 and  $9^{th}$  October, 1945. These events would have caused considerable run-off of surface contamination. Despite studies reporting a rapid decay of resuspension factors with time (McRaney and McGahan, 1980) the same factor was used for the entire duration of occupation. The inhalation dose factors assumed a resuspended particle diameter size distribution with an activity median aerodynamic diameter (AMAD) of  $1\mu m$ . The use of this AMAD would also contribute to a conservative result.

McRaney and McGahan's, (DNA)'s estimate of dose due to ingestion could also be considered conservative given that no attempt was made to separate the fission products into water soluble/insoluble fractions and eliminate the insoluble portion from the dose calculations (the insoluble fraction would be excreted in faeces and therefore not contribute significantly to the dose).

McRaney and McGahan (1980) could not obtain details regarding any specific activities and time spent within contaminated areas, so they calculated dose estimates from reasonable upper limits that had been derived from a "worst case" scenario. This scenario assumed that a serviceman spent eight hours per day within the area of highest radiation intensity. The NMRI survey data was used for calculations as the measurements were made at one metre above the ground and would be more representative of whole body exposure compared with the earlier survey.

HICMCRE (Shigematsu et al, 1995) suggests that activation products could have been:

- 32P due to the reaction of neutrons with the sulphur used to bond insulators to utility poles,
- <sup>60</sup>Co due to the reaction of neutrons with the cobalt used in steel for ferro-concrete bars in concrete buildings,
- and <sup>152</sup>Eu due to the reaction of neutrons with europium, a rare earth metal element present in minute quantities in rock.

Laboratory tests on the soil and building materials of Hiroshima indicated that six weeks after neutron activation the only radioisotopes of significance that remained in the ground zero area were  $^{46}$ Sc ( $t_{1/2} = 84$  days) and  $^{60}$ Co ( $t_{1/2} = 5.2$  years) (McRaney and McGahan, 1980). The nuclides  $^{24}$ Na,  $^{56}$ Mn and  $^{134}$ Cs were identified as potential activation products. However they were not considered in the calculation due to their short half-life ( $^{24}$ Na and  $^{56}$ Mn) or small relative quantity ( $^{134}$ Cs). Pure beta emitters such as  $^{45}$ Ca and  $^{32}$ P were considered present in insufficient quantities to affect the internal dose to the level of significance reported.

Fission products contained in the so-called "black rain" were <sup>137</sup>Cs and <sup>90</sup>Sr (Shigematsu et al, 1995).

# Resultant dose

Wilson (1956) indicated that due to uncertainties in the yield of the Hiroshima bomb, and the uncertainties in test measurements, the best estimate of dose to an individual was an approximate upper limit.

For U.S. occupation forces assigned to Hiroshima the upper limit for the external dose in the ground zero area was found to be 0.03 rem (0.3 mSv) (McRaney and McGahan, 1980). The dose from internal emitters (inhalation and ingestion) was "considerably less", and the dose to the whole body due to inhalation was calculated to be 0.003 rem (0.03 mSv).

The following dose estimates from the DNA report (McRaney and McGahan, 1980) apply to an individual that stayed in an area of maximum contamination for eight hours per day for a considerable period (ie. 2-10 months).

Table 2a:

	Ground Zero Area	Fall-out Area
External Dose	0.030rem	0.019rem (41 <sup>st</sup> Div.)
		0.014rem (24 <sup>th</sup> Div.)
Whole Body Dose due to Inhalation	0.003rem	< 0.068rem*
Whole Body Dose due to Ingestion	< 0.03rem**	< 0.02rem*

In units of Sieverts this table becomes

Table 2b:

	Ground Zero Area	Fall-out Area
External Dose	0.3 mSv	0.19 mSv (41 <sup>st</sup> Div.)
		0.14 mSv (24 <sup>th</sup> Div.)
Whole Body Dose due to Inhalation	0.03 mSv	< 0.68 mSv*
Whole Body Dose due to Ingestion	< 0.3 mSv**	< 0.2 mSv*

- \* The Nagasaki fall-out figure for the 2<sup>nd</sup> Div. Artillery group
- \*\* The Nagasaki ground zero figure

The (DNA) report went on to state that "there is no basis for assuming that any individual in the occupation units received these upper limit doses".

Shigematsu et al (1995) determined the gamma-ray tissue kerma rate at the Hiroshima hypocentre as a function of time. From this they estimated that the absorbed dose rate at the hypocentre, 212 days after detonation, was approximately  $0.6 \mu \text{Gy hr}^{-1}$ .

The cumulative dose on and after the sixth day (post explosion) due to induced radioactivity, at the Hiroshima hypocentre, was estimated to be 81 mGy (Shigematsu et al, 1995). The dose rate due to naturally occurring radioactivity in Hiroshima was estimated to be approximately 8  $\mu$ R hr<sup>-1</sup>. The world-wide average natural background dose rate from external radiation is approximately 7-10  $\mu$ R hr<sup>-1</sup>, which gives an annual dose of approximately 0.3-0.4 mSv.

Shigematsu et al estimated the cumulative dose between 1945 and 1985 due to internal exposure in the Hiroshima fall-out area (Koi-Takasu) as  $< 1 \times 10^{-5}$  Gy for men and  $< 8 \times 10^{-6}$  Gy for women. They reported the estimated radiation dose for the Koi-Takasu fall-out area, due to external gamma-radiation exposure, to be 0.01-0.03 Gy.

These estimates are consistent with one another and strongly suggest that any members of the Australian occupation forces who may have been exposed would have received doses considerably less than 10 mSv.

Roesch et al (1987, p21) commented that the absorbed doses from fall-out at Hiroshima were in the range 0.6 to 2 rad (6 to 20 mSv) for continuous occupancy starting one hour after detonation. This dose estimate was made by integrating the calculated dose rate from t=1 hour after detonation to infinity. Since the members of the occupation forces were in Hiroshima for up to 12 months, starting 212 days after detonation, the expected dose from external exposure can be estimated using the calculated dose rate one hour after detonation, allowing for the decay of the radionuclides known to be present to calculate the dose rate at time t and then integrating the dose rate from t=212 days to t=577 days.

# **Exposure Pathways**

External exposure from surface contamination

The dose estimates in the table above indicate that this contribution would be small (compared with 10 mSv). Direct measurements of external exposure rate due to  $^{137}$ Cs and Plutonium in soil at Hiroshima gave exposure rates ranging from  $11\mu$ R/h to 45  $\mu$ R/h (Roesch et al, 1987, Chapter 6). These results were corrected back to the time of detonation and then used to estimate the cumulative dose to exposed individuals. The following table (Table 3) is taken from Roesch et al (1987).

Table 3:

Investigator	Days after	Exposure rate at time of measurement			Exposure rate at 1	Cumulative
	bomb				hour after	exposure
					detonation	
		Gross		Net		
				(R/h)	(R/h)	(R)
Miyasaki and	H + 188	33 μR/h	maximum	25.5	0.6	3
Masuda (1953)		·				
		30	average	22.5	0.5	2.3
Fujiwara and	H + 49	6×bkgnd	maximum	40	0.19	1
Takeyama (1953)						
	H + 920	2.4×bkgnd		11	1.8	9
Tybout (1946)	H + 60	45 μR/h		37	0.23	1.2
(Arakawa,						
1962)						
Pace and	H + 87	19-20 μR/h		11-34	0.11-0.33	0.6-1.6
Smith (1959)						

Table 3 could be used to estimate the maximum external exposure to an individual spending 24 hours a day in the contaminated areas. The measurements are fairly consistent. However the largest value for the exposure rate corrected back to 1 hour after the bomb detonation was made 920 days after the detonation, and is likely to be the least reliable, due to the effects of surface run-off, etc.

If the Australians took over the American living quarters on Ujina Island, then it is unlikely that any individual would have spent more than 12 hours per day in the contaminated area(s).

Internal exposure from inhalation of resuspended material This pathway is discussed in some detail in Appendix 2.5.

# Surface Run-off

This would cause reduction and redistribution of surface contamination. There were two typhoons over Hiroshima after the bomb detonation and before any Australian occupation troops arrived in the area. The heavy rain that accompanied these events would have caused a significant reduction in surface contamination in the area where fall-out occurred.

# Contaminated drinking water

The source of the drinking water has to be known for the dose contribution from this pathway to be estimated. This possibility is discussed in Appendix 2.5.

# Contaminated food

It would be reasonable to assume that the bulk of the food consumed by the occupation forces personnel was not grown or processed in contaminated areas. The fall-out at Hiroshima occurred close to a residential area; however the consumption of fruits, grains or vegetables produced in the area is likely to have been only a very small fraction of the total dietary consumption by Australian personnel. This possibility is also discussed in Appendix 2.5.

# Contamination on hands (poor hygiene)

The contribution to the total dose from this pathway would usually be small. The possibility of ingesting a significant quantity of radioactivity via this pathway is discussed in Appendix 2.5.

# External exposure from neutron-activation products in soil

Roesch (1987, pp223ff) stated that the potential maximum exposures to external radiation from induced radioactivity at the hypocentre were estimated to be approximately 80 R at Hiroshima. The exposure decreased with both time and distance. These exposures were estimated from measurements of exposure rates, soil samples, neutron activation analyses, etc, and were calculated by integrating the exposure (dose) rate from one hour after bomb detonation to infinity. Roesch (1987) estimated that the cumulative exposure would have decreased to 30% of its original value after 1 day and to only a few percent of its original value after 1 week. This is clearly a consequence of the very short decay times of many of the radionuclides. The decrease with distance is also estimated (Roesch, 1987) to have been 90% at a distance of 500 m from the hypocentre, and 99.9% at 1000 m.

### **SPECIFIC SCENARIOS**

A set of specific scenarios is discussed in Appendix 2. These scenarios are

# 1. External Radiation From Induced Radioactivity (Activation Products)

The external dose rate at the Hiroshima hypocentre on February 1<sup>st</sup>, 1946 was estimated using an analytical formula derived from conservative assumptions. The concentrations of activation products in soil were established from the results of tests carried out on Hiroshima soil samples. The estimated dose rate was compared with the measured dose rate and a correction applied to the formula. The corrected formula was then used to estimate the total external dose to an individual located at the Hiroshima hypocentre from February 1<sup>st</sup>, 1946 for 12 hours per day, 3 days per week for 2 years by integrating the estimated dose rate from February 1<sup>st</sup>, 1946 for two years.

# 2. External Radiation From Fall-out (Fission Products)

The assumptions used for the first scenario were used to derive an analytical formula for the external dose rate resulting from fall-out of <sup>137</sup>Cs and <sup>90</sup>Sr in the Koi-Takasu area. The predicted dose rate was compared with the results of measurements to "calibrate" the formula. The formula was used to estimate the effective dose rate, from external exposure, to a person located in the Koi-Takasu area on February 1<sup>st</sup>, 1946, and the cumulative dose received by a person located in the Koi-Takasu district from February 1<sup>st</sup>, 1946 to February 1<sup>st</sup> 1948, for 24 hours per day.

#### 3. Souvenired Radioactive Item

The radioactivity (due to activation products) in a fist-sized, vitrified, object (10 cm diameter, 1.6 g cm<sup>-3</sup> density) taken from the vicinity of the Hiroshima hypocentre and kept as a souvenir was estimated and compared with the radioactivity in a similar object formed from background soil.

# 4. Internal Exposures

Internal exposures could have occurred via intakes of radionuclides resulting from the inhalation of contaminated dust, the ingestion of contaminated food, the ingestion of contaminated drinking water, and the ingestion of material left on the skin as a result of handling contaminated objects.

The possible maximum doses that could have occurred via these pathways were estimated using a computer model that was designed to estimate the doses resulting from radioactive waste placed on the ground surface.

### 5. Strontium

Possible intakes of strontium could occur via ingestion of contaminated material, inhalation of contaminated material, entry of contaminated material into the body via open wounds, or entry via absorption through the skin. In its most common chemical form, for a scenario such as this, Strontium is not known to be absorbed through the skin, so the last possibility can be disregarded. The other three possibilities were each studied in detail using models to estimate the retention of <sup>90</sup>Sr in the skeleton and the excretion of <sup>90</sup>Sr in urine and faeces 50 years after the intake.

The intake (via ingestion) required to deliver a committed effective dose of 10 mSv was estimated from the results of the model calculations.

For inhalation intakes the external dose rate from fall-out products was estimated using reasonable assumptions about the amount of deposited material and mixing in the soil surface layer. The estimated external dose rate was compared with the results of measurements and the predicted soil concentrations of the fall-out products were adjusted to make the estimated external dose rate agree with the measured external dose rate. These soil concentrations were then combined with an assumed dust resuspension rate to estimate the dose resulting from inhalation of contaminated dust. The amount of material that would have to be inhaled to give a committed effective dose of 10 mSv was estimated, together with the corresponding activity of <sup>90</sup>Sr in the skeleton, urine and faeces 50 years later.

The possibility that the ingestion of a particle of strontium could lead to a committed effective dose of 10 mSy was also examined.

The ingestion rate over 1 year needed to give a committed effective dose of 10 mSv was also estimated.

The amount of soil that would have to enter the body via an open wound to deliver a committed effective dose of 10 mSv was also estimated.

The activities in the skeleton and in urine, as a result of consumption of <sup>90</sup>Sr that is present in the environment as a result of nuclear weapons testing, were estimated.

The results of these estimates were combined in Appendix 2.6 to produce upper limit estimates of the total committed equivalent dose to bone marrow and total effective dose for different exposure periods.

### SUMMARY OF SCENARIO RESULTS

# 1. External Radiation From Induced Radioactivity (Activation Products)

The estimated maximum cumulative dose from external exposure received by a person located at the Hiroshima hypocentre from February  $1^{st}$ , 1946, assuming continuous occupation (24 hours per day, 7 days per week) for two years, was approximately 495  $\mu$ Sv. For exposure for 12 hours per day, 3 days per week for two years from February  $1^{st}$ , 1946, the estimated maximum cumulative dose from external exposure was approximately 110  $\mu$ Sv.

# 2. External Radiation From Fall-out (Fission Products)

The effective dose rate, from external exposure, to a person located in the Koi-Takasu area on February 1<sup>st</sup>, 1946 was approximately 0.38 µSv h<sup>-1</sup>.

The cumulative dose received by a person located in the Koi-Takasu district from February 1<sup>st</sup>, 1946 to February 1<sup>st</sup> 1948, for 24 hours per day, 7 days per week, due to external exposure, would have been no higher than 6.7 mSv.

# 3. Souvenired Radioactive Item

By February 1<sup>st</sup>, 1946, the external dose rate from a 1 kg vitrified object, taken from the vicinity of the Hiroshima hypocentre, would have been very much less than that from 1 kg of soil.

# 4. Internal Exposures

The effective dose resulting from internal exposures at Hiroshima, due to the consumption of a diet consisting entirely of contaminated seafood, fruits, grains, vegetables and drinking water, is unlikely to have exceeded 0.67 mSv.

# 5. Strontium

The preceding discussion indicates that if an individual spent 12 months working in the fallout area (Koi-Takasu), engaged in rubble clearing work or other tasks that may have produced similar levels of dust, for 8 hours per day, every day of the year, it may have been possible to incur a committed effective dose of 10 mSv via inhalation. Calculations indicate that if this did occur the strontium level in a daily urine sample may be detectable. The total amount of excreted strontium (in urine) could be increased by accumulating urine samples. The ratio of the strontium activity in bone to the strontium activity in urine would also be a crude indicator of the time of intake (for ingestion of strontium). This would involve measurements of strontium in bone, which would be possible via biopsy techniques. A comparison of the results of such measurements (urine and/or bone) with the results of measurements taken on a control group (see earlier) would also be a useful indicator.

Ingestion of  $^{90}$ Sr at a rate which would have led to a committed effective dose of 10 mSv to an individual would result in a rate of excretion of activity in a daily urine sample which should be measurable today, but the daily excretion rate would be similar in magnitude to the excretion rate resulting from the average intake of  $^{90}$ Sr from atmospheric weapons testing fall-out. The  $^{90}$ Sr  $^{90}$ Y activity in the skeleton from the Hiroshima intake would however be approximately 5-10 times higher than the corresponding average activity in the skeleton from the intake of  $^{90}$ Sr from atmospheric weapons testing fall-out.

Calculations of the amounts of strontium that would have to have been taken into the body by inhalation or ingestion at Hiroshima indicate that these intakes would only have occurred under very unusual conditions. The dose per unit intake for entry to the body via injection (open wound) is higher than that for intake by inhalation or ingestion, but the conclusion would be the same.

# 6. Total committed equivalent dose to bone marrow and total committed effective dose

In Appendix 2.6 it is shown that for a person spending their entire time at Hiroshima in the Koi-Takasu area, consuming only contaminated food and water (from local sources), and spending 8 hours per day in rubble clearing activities, exposed to dust at the (Australian) nuisance dust limit of 10 milligrams per cubic metre of air, the total committed equivalent dose to bone marrow would 3.15 mSv per year of exposure and the total committed effective dose (whole body) would be 10.6 mSv per year of exposure.

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#### **GLOSSARY**

RERF Radiation Effects Research Foundation (U.S./Japan)

DNA United States Defence Nuclear Agency.

AMAD Activity median Aerodynamic Diameter – the diameter of a particle to

which radionuclides are attached and which has the same aerodynamic

characteristics as a spherical particle of unit density.

HICMCRE Hiroshima International Council for Medical Care of the Radiation-

Exposed (Japan).

exposure the energy to which an individual is exposed, or its equivalent in terms of

the number of ion pairs produced in air per unit energy.

Roentgen (R) the unit of exposure.

absorbed dose the energy absorbed in a unit mass of tissue.

equivalent dose the energy absorbed per unit mass of tissue, corrected for the type of

radiation.

effective dose the whole-body dose, found by summing the equivalent doses (multiplied

by the tissue sensitivity) over all tissues.

rad the non-SI unit of absorbed dose.

Gray (Gy) the SI unit of absorbed dose.

rem the non-SI unit of effective dose and committed effective dose.

Sievert (Sv) the SI unit of effective dose and committed effective dose (1 Sievert = 1

Joule of absorbed energy per kilogram of tissue, corrected for the type of radiation and the type of tissue). The conversion factor from rem to Sv is

100 rem = 1 Sv.

Becquerel (Bq) the SI unit of disintegration rate for a radionuclide. 1 Bq = 1 disintegration

per second.

Curie (Ci) the historical (non-SI) unit of disintegration rate.  $1 \text{ Ci} = 3.7 \times 10^{10} \text{ Bq}$ .

hypocentre the point on the ground immediately beneath the point at which the bomb

was detonated.

detonation point the physical location of the point at which the bomb was located when

detonation occurred.

#### APPENDIX 1: DOSIMETRY AND DOSE LIMITATION

### **Dosimetry**

Radioactive decay or other nuclear processes such as fission can result in the production of ionising radiation (or particles) of several types.

<u>Gamma radiation</u> is high energy electromagnetic radiation (zero mass) that can penetrate easily through tissue. Energy is deposited in the tissue through which the radiation passes. For the gamma radiation energies emitted as a result of radioactive decay the maximum penetration in tissue is approximately 1-2 metres and the maximum penetration in soil is approximately 1 metre.

<u>Beta particles (beta radiation)</u> are electrons (negatively charged) or positrons (positively charged). They can penetrate up to several centimetres in tissue. Electrons passing through tissue lose energy predominantly because of electrical interactions with the electrons in the atoms composing the tissue.

<u>Alpha particles (alpha radiation)</u> are doubly ionised helium nuclei with a total mass approximately 8000 times the mass of an electron. They can only penetrate a few microns in tissue, due to their large mass and their electrical charge.

<u>Neutrons</u> are electrically neutral particles with a mass approximately 2000 times the mass of an electron. Neutrons can easily penetrate large distances through tissue because they do not carry any electric charge and therefore do not interact with the electrons and nuclei in the matter through which they pass, except by direct collision.

There are two ways in which humans can be exposed to ionising radiation released as a result of radioactive decay or other nuclear reactions (e.g. fission). External exposures occur when the radiation source is outside the body. Internal exposures occur when the radiation source is inside the body. The hazard associated with radioactivity is due to the fact that the energy released, as either alpha, beta or gamma radiation, as a result of radioactive decay is high enough to cause ionisation in matter in which some or all of this energy may be absorbed. In some cases this can lead to the induction of cancer. The tissues most at risk appear to be those in which the rate of cell division is highest.

For both types of exposure the risk of harm is estimated in terms of the absorbed dose in the different tissues and organs of the body. Absorbed dose in a tissue or organ is defined as the ratio of the energy absorbed in that tissue or organ to the mass of the tissue or organ.

For external exposures the effects of alpha and beta radiation are small because of their low penetrating power; the major problem is gamma radiation and neutrons, because of their relatively high penetrating power. External alpha radiation cannot penetrate through the skin, and external beta radiation is mostly absorbed in the skin and underlying tissue. For external exposures, the dose is estimated by a combination of direct measurement of the energy incident on the body, laboratory measurements of the rate of absorption of different types of radiation in different types of tissue, and computer modelling of the passage of radiation through tissue. Allowance has to be made for the effects of shielding of one tissue or organ by other tissues.

However, this low penetrating power means that alpha radiation and beta radiation pose the main radiological hazard for internal exposures. Internal exposures usually result from the inhalation, ingestion, injection, entry through wounds or absorption through the skin, of material containing radionuclides that can subsequently move around within the body. These radionuclides may decay within the body or may be exhaled or excreted in urine, faeces or sweat. The chemical form of the material containing the radionuclides, particularly its solubility in blood and other body fluids, has a major influence on the metabolism and movement of the radionuclides within the body. Particular radionuclides can selectively move to particular organs or tissues due to chemical effects. For example, iodine tends to concentrate in the thyroid, and strontium tends to concentrate in the skeleton. When these radionuclides undergo radioactive decay they irradiate the tissue around the point of decay and can also irradiate other tissues. Alpha particles are particularly important in internal exposure, because their low penetrating power and high energy mean that a large quantity of energy is deposited in a relatively small volume of tissue. Beta particles can penetrate further in tissue so they deposit energy in tissue at a lower rate. Gamma radiation can penetrate far enough in tissue to irradiate organs other than that in which the radioactive decay occurred, and can pass out of the body altogether. Neutrons are not released by radioactive decay, so they do not contribute to internal exposures. For internal exposures the dose estimation has to allow for the fact that different types of radiation can have different effects on the same tissue, that the same radiation can have different effects in different tissues and that the radioactive material can move around in the body. In addition, because some radionuclides decay slowly over many years, the energy deposited as a result of a single intake of radioactive material may be deposited over many years and in several different tissues. The estimation of the dose from this single intake also has to take this delayed deposition of energy into account.

To allow for the difference between external and internal exposures, the latter are quantified in terms of the committed effective dose, which allows for the different radiation effects and tissue sensitivities. The unit of committed effective dose is the Sievert (Sv).

The dose to a single tissue is quantified in terms of the equivalent dose, given by  $H_T = \sum_R w_R D_{T,R}$ 

where T denotes the tissue, R denotes the type of radiation,  $w_R$  denotes the radiation weighting factor (which allows for the effects of different types of radiation on the same tissue), and  $D_{T,R}$  is the *total* absorbed dose in tissue T from radiation of type R resulting from the exposure.

The committed effective dose is estimated by summing over all tissues and allowing for the relative sensitivity of each tissue. The committed effective dose is given by

$$E = \sum_{T} w_{T} \sum_{R} w_{R} D_{T,R}$$

where w<sub>T</sub> is the tissue weighting factor for tissue T.

The values of the tissue weighting factors are based on observations of the number of tumours that occur in different tissues for a given exposure. This formulation allows a direct comparison between the effects of external exposures and internal exposures.

# Natural Background Radiation

Radionuclides such as <sup>238</sup>U, <sup>235</sup>U and <sup>232</sup>Th and their radioactive decay products are present in significant quantities in the earth's crust, together with other radionuclides such as <sup>40</sup>K. Humans are continually exposed to radiation from the radioactive decay of these naturally occurring radionuclides and also from cosmic rays. The annual radiation dose, including the committed component resulting from delayed decay within the body, from these natural sources of ionising radiation in Australia is approximately 1.5 to 2 milli-Sievert (mSv). This background dose is made up of (approximately)

- 0.5 1.0 mSv resulting from inhalation of radon (222Rn) and its radioactive progeny,
- 0.3 mSv from gamma radiation from soil,
- 0.3 mSv from gamma radiation from cosmic rays,
- 0.3 mSv from the decay of radionuclides within the body, particularly <sup>40</sup>K.

The cosmic ray contribution increases with increasing altitude.

In some parts of the world the annual dose received from natural background radiation can be as high as 25 mSv, due to the presence of high concentrations of radionuclides in soil. No adverse health effects have been associated with these variations in natural background doses.

#### Dose Limitation

To minimise the risk of harm from exposures to radiation sources other than natural background the International Commission for Radiological Protection has recommended a dose limitation system based on three main principles:

- Justification: no practice should be implemented unless a net positive benefit can be demonstrated;
- Optimisation: ALARA exposures should be kept "as low as reasonably achievable", taking social and economic factors into account;
- Dose Limits: effective dose should be kept below prescribed maximum values. The recommended dose limits are 1 mSv per year (averaged over 5 years, with no more than 5 mSv in any one year) for a member of the public (involuntary exposure), and 20 mSv per year (averaged over 5 years with no more than 50 mSv per year in any one year) for a radiation worker (voluntary exposure). In addition, special limits for the skin and the lens of the eye are also recommended, because of the relatively low sensitivity of these tissues to the effects of ionising radiation.

These recommendations were developed from epidemiological studies of atomic bomb survivors and uranium miners, and were based on the guiding principle that the risk of death from exposure to radiation (for radiation workers) should be comparable with the risk of death for workers in other industries. The currently accepted risk of death from exposure to radiation (other than natural background) is 5% per Sievert, where the entire population is uniformly exposed.

#### APPENDIX 2: SPECIFIC SCENARIOS CONSIDERED IN THIS REPORT

# 2.1. EXTERNAL RADIATION FROM INDUCED RADIOACTIVITY (ACTIVATION PRODUCTS)

# 2.1.1 External dose rate at the hypocentre on February 1st, 1946

# Assumptions:

- there was no fall-out at the hypocentre (confirmed by measurements);
- the assessment only needs to consider the long-lived radionuclides <sup>46</sup>Sc, <sup>60</sup>Co and <sup>134</sup>Cs;
- the initial exposure rate for  $^{46}$ Sc +  $^{60}$ Co +  $^{134}$ Cs was 130  $\mu$ R/h (Roesch et al, 1987; vol. 1, p222.) the half-lives for these radionuclides are 83.8 days, 5.3 years, and 2.1 years respectively;
- the total effective dose rate can be calculated from the general formula

dose rate = activity concentration  $\times$  dose rate per unit activity concentration.

- the soil was uniformly contaminated to infinite depth;
- $1 \text{ R h}^{-1} = 0.0096 \text{ Sy h}^{-1}$  for gamma radiation (Martin et al, 1996);

For this particular case this general formula becomes

$$D_{I}(t) = q_{I}(A_{1}(0)K_{1}.\exp(-\lambda_{1}t) + A_{2}(0)K_{2}.\exp(-\lambda_{2}t) + A_{3}(0)K_{3}.\exp(-\lambda_{3}t))$$

where subscript 1 refers to  $^{46}$ Sc, subscript 2 refers to  $^{60}$ Co, subscript 3 refers to  $^{134}$ Cs,  $A_j(0)$  is the activity of radionuclide j at time t=0,  $K_j$  is the dose coefficient for radionuclide j (Sv (Bq.s.m<sup>-3</sup>)<sup>-1</sup>),  $\lambda_j$  is the radioactive decay coefficient for radionuclide j (decay coefficient = ln(2)/half-life), t is time in hours (t=0 corresponds to 1 hour after detonation), and  $q_I$  is a factor which allows for conversion between different units. This assumes that the effects of leaching and surface run-off can be neglected.

The 
$$K_j$$
 values are given by  $K_1 = 6.79 \times 10^{-17} \ Sv(Bq\ s\ m^{-3})^{-1}, \ K_2 = 8.68 \times 10^{-17} \ Sv(Bq\ s\ m^{-3})^{-1},$  and  $K_3 = 5.07 \times 10^{-17} \ Sv(Bq\ s\ m^{-3})^{-1}$  (Eckerman and Ryman, 1993).

Samples of uncontaminated soil from Hiroshima were tested by bombarding them with neutrons in a reactor and measuring the activation product yield. Using the highest reported concentrations for radionuclides found in samples of Hiroshima soil from these measurements (1×10<sup>-4</sup> pCi gm<sup>-1</sup> for <sup>46</sup>Sc, 6×10<sup>-6</sup> pCi gm<sup>-1</sup> for <sup>60</sup>Co, and 8×10<sup>-4</sup> pCi gm<sup>-1</sup> for <sup>134</sup>Cs (Roesch et al, 1987, vol. 1, p218)), and correcting for the difference between the reactor neutron flux and the bomb neutron flux, the formula for the dose becomes

$$D_I(t) = 0.0096 \times 3.6 \times 10^3 q_I(6.79 \times 10^{-21}.\exp(-3.446 \times 10^{-4}t) + 5.21 \times 10^{-22}.\exp(-1.492 \times 10^{-5}t) + 4.056 \times 10^{-20}.\exp(-3.765 \times 10^{-5}t)$$

At t = 0 this becomes

$$D_I(t) = 1.654 \times 10^{-18} . q_I = 1.248 \ \mu Sv h^{-1}$$

This means that  $q_I = 7.54 \times 10^{17}$ . Therefore the dose rate formula becomes

$$D_I(t) = 0.177 \cdot \exp(-3.446 \times 10^{-4} t) + 0.0136 \cdot \exp(-1.492 \times 10^{-5} t) + 1.057 \cdot \exp(-3.765 \times 10^{-5} t) \mu Sv h^{-1}$$

where t is time in hours from 1 hour after detonation.

The external dose rate from induced radioactivity at the hypocentre on February 1<sup>st</sup>, 1946 was approximately 0.95 micro-Sievert per hour (Roesch et al, 1987, vol. 1, pp221-224).

The slant range from the detonation point (510 metres above the hypocentre) to a point on the ground 1 km from the hypocentre was approximately 1120 metres. With the reasonable assumption that the neutron flux that caused the induced radioactivity in the soil spread out spherically from the detonation point, the estimated upper limit for the dose rate at a point 1 km from the hypocentre on February 1<sup>st</sup>, 1946 would have been approximately 0.2 micro-Sievert per hour (µSv hr<sup>-1</sup>).

The slant range from the detonation point (510 metres above the hypocentre) to a point on the ground 3 km from the hypocentre was approximately 3040 metres. With the same assumption as in the previous paragraph, the estimated upper limit for the dose rate at a point 3 km from the hypocentre on February 1<sup>st</sup>, 1946 would have been approximately 0.03 micro-Sievert per hour.

These are all upper limit estimates. The calculation used here is conservative in that it takes no account of downward leaching of the induced radionuclides by rainwater percolating downwards into the soil. In addition, the soil would not have been contaminated uniformly to infinite depth, because the neutron flux would be attenuated as it passed downwards through the soil. Additional attenuation of the neutron flux would have occurred because of absorption of neutrons in the air between the detonation point and the ground surface. The effect of leaching is important, because the gamma radiation produced by the radioactive decay of the activation products in the soil is attenuated in the overlaying soil. This effectively means that the gamma radiation that contributes to the exposure of an individual standing on the ground comes from approximately the top metre of soil.

Excluding the effects of leaching (and surface run-off), the induced radiation level would have declined with an effective half-life somewhere between 83.8 days ( $^{46}$ Sc) and 5.3 years ( $^{60}$ Co). Using the above formula, the dose rates on February 1<sup>st</sup>, 1946 and February 1<sup>st</sup>, 1950 were 0.952  $\mu$ Sv h<sup>-1</sup> and 0.247  $\mu$ Sv h<sup>-1</sup>. This rate of decrease corresponds to an effective half-life, due to radioactive decay only, of approximately 2 years.

The cumulative dose received by a person located at the hypocentre from February 1<sup>st</sup>, 1946 for 12 hours per day, 3 days per week for 2 years was estimated by integrating the estimated dose rate from February 1<sup>st</sup>, 1946 for two years. For continuous occupation (24 hours per

day, 7 days per week) the estimated cumulative dose from external exposure was 493.4  $\mu$ Sv. For exposure for 12 hours per day, 3 days per week the cumulative dose would have been approximately 493.4×(12/24)×(3/7)  $\mu$ Sv, i.e. approximately 110  $\mu$ Sv.

# **2.1.2 Summary:**

The estimated maximum cumulative dose from external exposure received by a person located at the Hiroshima hypocentre from February  $1^{st}$ , 1946, assuming continuous occupation (24 hours per day, 7 days per week) for two years, was approximately 495  $\mu$ Sv. For exposure for 12 hours per day, 3 days per week for two years from February  $1^{st}$ , 1946, the estimated maximum cumulative dose from external exposure was approximately 110  $\mu$ Sv.

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# 2.2. EXTERNAL RADIATION FROM FALL-OUT (FISSION PRODUCTS)

The long-lived radionuclides that would have to be considered for estimation of external doses from fall-out are <sup>137</sup>Cs and <sup>90</sup>Sr. Making the same assumptions as in the previous section, the total effective dose rate at time t would be given by

$$D_F(t) = q_F(A_1(0)K_1.\exp(-\lambda_1 t) + A_2(0)K_2.\exp(-\lambda_2 t))$$

where subscript 1 refers to  $^{137}$ Cs, subscript 2 refers to  $^{90}$ Sr,  $A_j(0)$  is the deposited activity of radionuclide j at time t=0,  $K_j$  is the dose rate coefficient for radionuclide j (Sv (Bq.s.m<sup>-2</sup>)<sup>-1</sup>), t is time in hours (t=0 corresponds to 1 hour after detonation), and  $q_F$  is a factor which allows for conversion between different units.

Eckerman and Ryman (1993) give values for  $K_1$  and  $K_2$  of  $2.85\times10^{-19}$  Sv(Bq s m<sup>-2</sup>)<sup>-1</sup>, and  $2.84\times10^{-19}$  Sv(Bq s m<sup>-2</sup>)<sup>-1</sup> respectively. The dose contribution from  $^{90}$ Y also has to be included, because the  $^{90}$ Y will be in secular equilibrium with the  $^{90}$ Sr after approximately 50 days post detonation. The dose rate coefficient for  $^{90}$ Y is (Eckerman and Ryman, 1993)  $5.32\times10^{-18}$  Sv(Bq s m<sup>-2</sup>)<sup>-1</sup>. Therefore

$$D_F(t) = q_F(A_1(0) \times 2.85 \times 10^{-19}.\exp(-\lambda_1 t) + A_2(0) \times (2.84 \times 10^{-19} + 5.32 \times 10^{-18}).\exp(-\lambda_2 t))$$

The half-lives for <sup>137</sup>Cs and <sup>90</sup>Sr are 30 years and 29.12 years respectively. Therefore

$$D_F(t) = q_F(A_1(0) \times 2.85 \times 10^{-19}. \exp(-2.636 \times 10^{-6}t) + A_2(0) \times 5.60 \times 10^{-18}. \exp(-2.715 \times 10^{-6}t)$$

For this exercise, since the half-lives are so similar, it is reasonable to approximate this expression by

$$D_F(t) = q_F(A_1(0) \times 2.85 \times 10^{-19} + A_2(0) \times 5.60 \times 10^{-18}) \cdot \exp(-2.636 \times 10^{-6}t)$$

Miyazaki and Masuda (1953) reported the results of measurements of external exposure rates in the Koi-Takasu district during the period January 27, 1946 to February 6, 1946 (approximately 25.5  $\mu$ R h<sup>-1</sup>). The results of these and other measurements summarised by Roesch et al (1987) indicate that an upper limit for  $D_F(0)$  was approximately 40  $\mu$ R h<sup>-1</sup>, or 0.384  $\mu$ Sv h<sup>-1</sup>. Therefore

$$D_F(t) = 0.384 \times \exp(-2.636 \times 10^{-6} t) \mu Sv h^{-1}$$

The effective dose rate, from external exposure, to a person located in the Koi-Takasu area on February 1<sup>st</sup>, 1946 was approximately 0.38 µSv h<sup>-1</sup>.

The fall-out would have decreased with a half-life of approximately 30 years due to radioactive decay. However, other processes such as leaching and surface run-off due to rainfall would have substantially reduced this half-life, and therefore reduced the cumulative dose. It is not possible to provide an accurate estimate of what the effective half-life would have been.

Based on these considerations, the cumulative dose received by a person located in the Koi-Takasu district from February 1<sup>st</sup>, 1946 to February 1<sup>st</sup> 1948, for 24 hours per day, due to external exposure, would have been no higher than 6.7 mSv. Roesch et al (1987) also noted that the estimated life-time absorbed dose (based on measurements of body burden of <sup>137</sup>Cs) for an individual living in the Koi-Takasu area was 0.6 to 2 rad (6 to 20 mSv). This estimate took no account of the effects of surface run-off and leaching.

Another estimate of the cumulative external dose with some allowance for surface run-off and other weathering processes can be made using the t<sup>-1,2</sup> law for the reduction in fall-out levels following a nuclear explosion (Glasstone and Dolan, 1977; Roesch et al, 1987; Eisenbud, 1987). This law approximates the observed decrease in the dose rate due to residual radioactivity (fall-out) by the equation

$$R(t) = R(1) \times t^{-1.2},$$

where t is time (hours) after detonation and R(1) is the dose rate 1 hour after detonation. At Hiroshima the measured dose rate at about February  $1^{st}$ , 1946 in the Koi-Takasu area was approximately 0.4 Sv/hr (Miyasaki and Ikeda, 1953; Miyasaki and Matsuda, 1953). Using this value gives R(1) = 9.1 mSv/hr. Therefore the cumulative dose, due to external exposure, received by a person located 24 hours a day, 7 days a week, from February  $1^{st}$ , 1946 to February  $1^{st}$  1948, in the Koi-Takasu area would be approximately 2.5 mSv. This is consistent with the earlier estimate.

# **Summary:**

The effective dose rate, from external exposure, to a person located in the Koi-Takasu area on February 1<sup>st</sup>, 1946 was approximately 0.38 µSv h<sup>-1</sup>.

The cumulative dose, due to external exposure, received by a person located in the Koi-Takasu district from February 1<sup>st</sup>, 1946 to February 1<sup>st</sup> 1948, for 24 hours per day, 7 days per week, would have been no higher than 6.7 mSv.

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### 2.3. SOUVENIRED RADIOACTIVE ITEM

The factors that would affect the external dose received from a fist-sized, vitrified, object taken from the vicinity of the Hiroshima hypocentre and kept as a souvenir are

- the total activity of the object;
- the proximity of the object to the person being irradiated (the gamma radiation flux would decrease approximately in proportion to the square of the distance from the object;
- the time spent in close proximity to the object.

Unless the object was very close to a specific part of the body for a considerable time, the exposure would have been approximately uniform to most parts of the body. The dose to a specific organ would be extremely difficult to estimate.

The dose rate coefficients used in Scenarios 1 and 2 were derived (Eckerman and Ryman, 1993) for an individual standing on a contaminated surface (fall-out only) or on the surface of contaminated soil (induced radioactivity) contaminated uniformly to infinite distance. The gamma radiation in soil comes (essentially) from the top 1 metre of soil due to the fact that gamma radiation is attenuated as it passes through the soil. If the souvenired object was assumed to be a sphere of approximately 10 cm in diameter and its density was assumed to be 1.6 g cm<sup>-3</sup> (similar to soil), it would have a mass of approximately 850 grams. The radionuclide content of the object, considering only <sup>46</sup>Sc, <sup>60</sup>Co, and <sup>134</sup>Cs in the same concentrations as in Section 1, and correcting for the fact that the neutron fluence used in the soil experiments was measured at  $6\times10^{12}$  cm<sup>-2</sup> (Arakawa, 1962), and the neutron fluence at Hiroshima (prompt + delayed) was estimated to be  $3.2 \times 10^{13}$  cm<sup>-2</sup> (Roesch et al, 1987, vol. 1, p 219), would be approximately 160  $\mu$ Bq of  $^{46}$ Sc, 1100  $\mu$ Bq of  $^{60}$ Co, and 1350  $\mu$ Bq of  $^{134}$ Cs, at 1 hour after detonation. By February 1<sup>st</sup>, 1946 these activities would have decreased to approximately 36.5, 1030 and 1150  $\mu$ Bq respectively. A sample of "normal" soil of the same mass contains approximately 35 Bq of  $^{238}$ U in secular equilibrium with its radioactive progeny (a total activity of approximately 470 Bq) and 45 Bq of <sup>232</sup>Th in secular equilibrium with its radioactive progeny (a total activity of approximately 450 Bq). A comparison of the activity of the activation product concentrations with the concentration of natural radioactivity in the hypothetical souvenired object suggests that the souvenired object would constitute a negligible radiological hazard.

# **Summary:**

By February 1<sup>st</sup>, 1946, the external dose rate from a 1 kg vitrified object, taken from the vicinity of the Hiroshima hypocentre, would have been very much less than that from 1 kg of natural (uncontaminated) soil.

### **REFERENCES**

Eckerman, K.F., Ryman, J.C. *External exposure to radionuclides in air, water, and soil.* Federal Guidance Report No. 12. U.S. Environmental Protection Agency, Washington, DC 20460; 1993.

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#### 2.4. INTERNAL EXPOSURES

Internal exposures could have occurred via intakes of radionuclides resulting from

- inhalation of contaminated dust:
- ingestion of contaminated food;
- ingestion of contaminated drinking water;
- ingestion of material left on the skin as a result of handling contaminated objects.

### 2.4.1 Inhalation of contaminated dust.

A "worst case" estimate for this scenario is discussed in detail in the section on strontium.

# 2.4.2 Ingestion of contaminated food.

There are three main pathways via which contaminated food could be ingested:

- consumption of food grown on contaminated land (grains, fruit, vegetables);
- consumption of animal products (meat, milk, cheese) from animals that have grazed on contaminated land, been fed with fodder grown on contaminated land or which have consumed contaminated drinking water;
- consumption of seafood taken from contaminated seawater. Seawater can become contaminated by surface run-off, directly into the ocean or into rivers that transport the contaminated water to the ocean, or as a result of the transport of radionuclides in groundwater that enters the ocean. The radionuclides have to be transported downwards (by leaching) from the contaminated zone into an aquifer which then transports the radionuclides into the ocean.

It is unlikely that any of these pathways would have contributed significantly to the dose received by any of the occupation force personnel. The greater part of the food consumed by these people would have come from outside the contaminated area.

In the absence of data for the levels of radionuclides in the seawater along the coast near Hiroshima, it is not possible to give a quantitative estimate of the dose resulting from the consumption of contaminated seafood. However, it is reasonable to assume that the behaviour of the radionuclides deposited on the ground at Hiroshima would be similar to the case of radioactive waste deposited on the ground surface. This (waste) problem has been studied, using models that have been validated against measurements. The results indicate that the dominant pathway is exposure to (direct) external radiation from the waste. Estimates made using the computer code RESRAD (Yu et al, 1993), with a scenario similar to the deposition of fall-out, and including all relevant exposure pathways, indicate that over 90% of the dose (per Bq of deposited 137Cs or 90Sr) was due to external exposure.

The assumed diet used in the model calculations is given in Table A2.4.1 below. The calculations were made assuming that:

- (1) all grains fruit and vegetables consumed by the occupation personnel were grown on contaminated soil;
- (2) all animal products (meat, milk, cheese) consumed by the occupation personnel were from animals that had grazed on contaminated land, been fed with fodder grown on contaminated land or had consumed contaminated drinking water;
- (3) all seafood consumed by the occupation personnel was taken from an area contaminated by surface run-off from areas contaminated by fall-out from the bomb explosion.

Assumptions (1) and (2) are equivalent to assuming that the occupation personnel lived in the Koi-Takasu area and that all their food (other than seafood) was grown in this area or taken from animals which grazed and drank in this area. Alternatively the assumptions (with the exception of the calculations of doses due to contamination of drinking water and seafood) are equivalent to assuming that the entire Hiroshima area was contaminated by radioactive fall-out to the level measured in the Koi-Takasu area.

Food type	Assumed consumption		Contamination Fraction
	Amount	Units	
fruits, vegetables, grains	160	kg per year	1
leafy vegetables	14	kg per year	1
milk	92	litres per year	1
meat and poultry	50	kg per year	1
fish	10	kg per year	1
other seafood	5	kg per year	1
drinking water	510	litres per year	1

**Table A2.4.1:** the assumed diet for the purposes of estimating the doses to occupation personnel from consumption of contaminated food.

These values are the default values used in the RESRAD model. The soil to plant concentration factors used in the model for Caesium-137 and Strontium-90 are shown in Table A2.4.2.

Radionuclide	Plant/Soil	Units
	Concentration	
	Ratio	
Cs-137 + progeny	0.04	Bq/kg in edible portion of plant per Bq/kg in soil
Sr-90 + progeny	0.30	Bq/kg in edible portion of plant per Bq/kg in soil

**Table A2.4.2:** the assumed soil to plant concentration factors used in the model calculations.

The meat/livestock intake ratios (beef cattle) and milk/livestock intake ratios (beef cattle) for Caesium-137 and Strontium-90 used in the model are shown in Table A2.4.3.

Radionuclide	Product/Intake	Units
	Concentration	
	Ratio	
Cs-137 + progeny	0.030	Bq/kg in meat per Bq/day in fodder and water
	0.008	Bq/litre in milk per Bq/day in fodder and water
Sr-90 + progeny	0.008	Bq/kg in meat per Bq/day in fodder and water
	0.002	Bq/litre in milk per Bq/day in fodder and water

**Table A2.4.3:** the assumed product to intake concentration factors for meat and milk used in the model calculations.

Table A2.4.4 shows the bio-accumulation factors for fish and other seafood.

Radionuclide	Seafood	Bio-accumulation Factor	Units
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Cs-137 + progeny	fish	2000	L/kg
	crustacea	100	L/kg
	mollusks	100	L/kg
Sr-90 + progeny	fish	60	L/kg
	crustacea	100	L/kg
	mollusks	100	L/kg

**Table A2.4.4:** the assumed bio-accumulation factors for meat and milk used in the model calculations.

These factors are used as the default in RESRAD. The consumption of seafood used in the model calculations was set to 10kg per year for fish and 5 kg per year for other seafoods. Even if the dose from consumption of seafood was as high as 10% of the dose from external exposure (estimated and measured as an upper limit of 6.7 mSv over the two year period in question) this would mean an upper limit for the dose from consumption of seafood of approximately 0.67 mSv.

# 2.4.3 Ingestion of contaminated drinking water.

The calculations made with the RESRAD code included ingestion of drinking water drawn from a well close to the edge of the contaminated area. The dose from this pathway was included in the 10% from non-external exposures, so it is highly unlikely that ingestion of contaminated drinking water would have lead to a significant dose.

Another possible pathway by which contaminated drinking water could have been consumed is surface run-off carrying radionuclides on the ground surface into a dam from which drinking water was drawn. In the absence of evidence of significant surface contamination (from fall-out) other than in the Koi-Takasu area it is not possible to make an estimate of how much this pathway would have contributed to the total dose. However an upper limit to the contribution from this pathway can be estimated as follows. In the period from August 6<sup>th</sup>, 1945, to February 1<sup>st</sup>, 1946, approximately 900 mm of rain fell in the Hiroshima area. Assume that (on February 1<sup>st</sup>, 1946)

- the whole (land) dam catchment area was uniformly covered with water to a depth of 900 mm;
- the entire catchment area was uniformly contaminated with fall-out to the level measured in the Koi-Takasu area.

#### Also assume that

- the water stayed on the ground surface long enough for the radionuclides on the surface (to a depth of 1 mm) to dissolve in the water;
- 80% of the water soaked into the ground; 20% was removed by surface run-off (RESRAD);
- any suspended matter in the water (particles, etc) was removed prior to drinking.

Then, for a surface concentration of 1 Bq per gm each of  $^{90}$ Sr and  $^{137}$ Cs, the concentrations of these two radionuclides in water would have been 33 Bq per litre for  $^{90}$ Sr and 1 Bq per litre

for  $^{137}$ Cs (RESRAD- Yu et al, 1993). Measurements reported by Shizuma et al (1996) on soil samples collected 3 days after the detonation of the atomic bomb over Hiroshima confirmed that the maximum  $^{137}$ Cs concentration in soil was approximately 0.030 Bq g $^{-1}$  in the Koi-Takasu area. In a nuclear explosion of the type at Hiroshima, the total amounts of  $^{90}$ Sr and  $^{137}$ Cs released by the bomb detonation should be approximately equal. Since the radioactive half-lives of both radionuclides is approximately 30 years, it can be assumed that no significant radioactive decay had occurred by February 1 $^{st}$ , 1946. Therefore the consumption of drinking water contaminated to this level at a rate of 510 litres per year would have resulted in an intake of approximately 1000 Bq of  $^{90}$ Sr and 30 Bq of  $^{137}$ Cs over two years. The predicted committed effective (50 year) dose, including the contribution from  $^{90}$ Y, is  $2.8 \times 10^{-8}$  Sv (IAEA, 1996) for an intake of 1 Bq of  $^{90}$ Sr. Therefore the ingestion of 1000 Bq of  $^{90}$ Sr via this pathway would give a committed effective dose of 28  $\mu$ Sv. The predicted committed effective (50 year) dose is  $1.3 \times 10^{-8}$  Sv (IAEA, 1996) for an intake of 1 Bq of  $^{137}$ Cs. Therefore the ingestion of 33 Bq of  $^{137}$ Cs via this pathway would give a committed effective dose of less than 0.5  $\mu$ Sv.

# **2.4.4** Ingestion of material left on the skin as a result of handling contaminated objects. An example of this type of intake is discussed in Scenario 5 on strontium.

### Strontium-90:

Srontium-90 is a fission product, so it would not be present in the induced radiation zone in any significant quantity, unless fall-out also occurred in that area. The only area in Hiroshima where fall-out levels were significant was the Koi-Takasu area, situated approximately 3 km west of the hypocentre.

### Caesium-137:

Caesium-137 is also a fission product, so it would not be present in the induced radiation zone in any quantity, unless fall-out also occurred in that area. Measurements reported by Shizuma et al (1996) on soil samples collected 3 days after the detonation of the atomic bomb over Hiroshima confirmed that the maximum  $^{137}\text{Cs}$  concentration in soil was approximately 30 mBq g $^{-1}$  in the Koi-Takasu area, and that  $^{137}\text{Cs}$  concentrations near the hypocentre were much lower (averaging approximately 1 mBq g $^{-1}$ ). The corresponding deposition densities were approximately 5 Bq m $^{-2}$  and 0.15 Bq m $^{-2}$  respectively. The estimated deposition density of  $^{137}\text{Cs}$  in the 20-30 degree latitude region of the southern hemisphere as a result of atmospheric nuclear weapons testing from 1945 on is approximately 400-500 Bq m $^{-2}$  (UNSCEAR, 2000). The estimated annual dose from exposure to weapons testing fall-out is approximately 1-10  $\mu\text{Sv}$  between 1950 and 1990 (UNSCEAR, 2000). This means that the doses incurred as a result of exposure to  $^{137}\text{Cs}$  at Hiroshima would have been negligible compared with that from global fall-out.

# **2.4.5 Summary:**

The effective dose resulting from internal exposures at Hiroshima, due to the consumption of a diet consisting entirely of contaminated seafood, fruits, grains, vegetables and drinking water, is unlikely to have exceeded 0.67 mSv.

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Shizuma, K., Iatani, K., Hasai, H., Hoshi, M., Oka, T., Okano, M. <sup>137</sup>Cs concentration in soil samples from an early survey of Hiroshima atomic bomb and cumulative dose estimation from the fall-out. Health Physics. **71(3)**:340-346; 1996.

United Nations Scientific Committee on the Effects of Atomic Radiation. *Sources and Effects of Ionising Radiation. Volume 1: Sources.* United Nations: New York, 2000.

Yu, C., Zielen, A.J., Cheng, J.J., Yuan, Y.C., Jones, L.G., LePoire, D.J., Wang, Y.Y., Loureiro, C.O., Gnanapragasam, E., Faillace, E., Wallo, A. (III), Williams, W.A., Peterson, H. *Manual for Implementing Residual Radioactive Material Guidelines using RESRAD*, *Version 5.0.* USDOE Report No. ANL/EAD/LD-2; Argonne National Laboratory; September 1993.

#### 2.5. STRONTIUM

Possible intakes of strontium could occur via ingestion of contaminated material, inhalation of contaminated material, entry of contaminated material into the body via open wounds, or entry via absorption through the skin. In its most common chemical form, for a scenario such as this, Strontium is not known to be absorbed through the skin, so the last possibility can be disregarded. The other three possibilities will each be discussed in turn.

# 2.5.1 Ingestion

To establish the context, a calculation of the effects of an intake of 1 Bq of <sup>90</sup>Sr via ingestion was carried out. This includes the ingestion of material left on the skin as a result of handling contaminated objects (see Appendix 2.4.4)

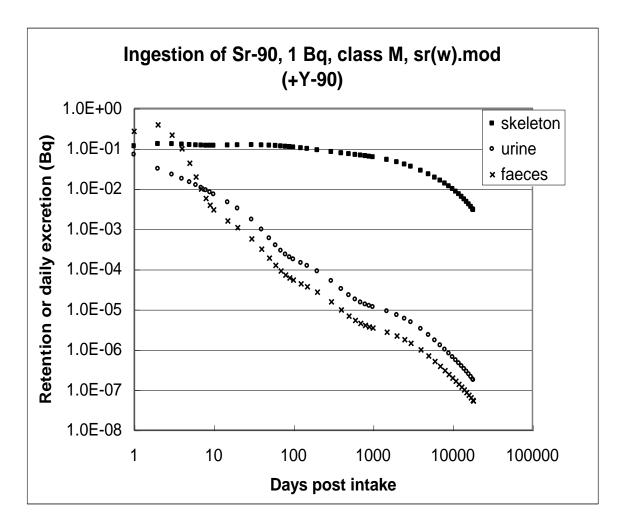


Figure 4:

Figure 4 shows the estimated excretion and skeletal retention (as a function of time to 50 years post intake) of <sup>90</sup>Sr following ingestion of 1 Bq of class M (moderately soluble) <sup>90</sup>Sr.

The calculations were done with a model developed at ARPANSA (O'Brien, 2001). The predicted urine excretion rate agrees with that published (ICRP, 1997) by the International Commission on Radiological Protection (ICRP Publication 78, p69, Fig. A.4.14). The

skeletal retention agrees to within 10% with that predicted by the LUDEP (Jarvis et al, 1995) model developed at the National Radiological Protection Board (U.K.) to implement the new ICRP respiratory tract model (ICRP, 1994).

The predicted amount remaining in the skeleton after 50 years is approximately  $1.46 \times 10^{-3}$  Bq. The predicted daily excretion of  $^{90}$ Sr in urine after 50 years is approximately  $8.65 \times 10^{-8}$  Bq.

This does not include the beta activity due to  $^{90}$ Y. Since the half-life of  $^{90}$ Sr is 29.1 years and the half-life of  $^{90}$ Y is 64.10 hours the  $^{90}$ Y should be in equilibrium with the  $^{90}$ Sr. This should double the beta activity in the skeleton and in urine.

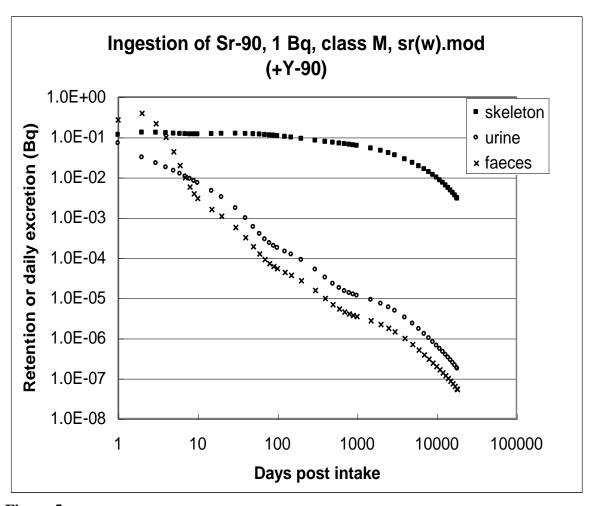


Figure 5:

Figure 5 shows the result of a calculation made exactly as in the case of the first estimate, but allowing for the ingrowth of <sup>90</sup>Y. The effect of this is to double the activity in the skeleton, urine and faeces, as expected.

The predicted committed effective (50 year) dose, including the contribution from  $^{90}$ Y, is  $2.8\times10^{-8}$  Sv (IAEA, 1996) for an intake of 1 Bq of  $^{90}$ Sr. For a committed effective dose of 10 mSv the corresponding intake would be  $3.57\times10^{5}$  Bq (approximately 10 micro Curies), and the activity ( $^{90}$ Sr +  $^{90}$ Y) in the skeleton after 50 years for an intake of this magnitude would be approximately 1000 Bq. The activity in a daily urine sample after 50 years would be approximately 30 mBq per day. The daily volume of urine excreted by reference man (ICRP,

1975) is 1400 mL. This would imply a concentration of ( $^{90}$ Sr +  $^{90}$ Y) in a daily urine sample of approximately 22 mBq per Litre. This should be detectable using spectrometric liquid scintillation counting methods (Boecker et al, 1991; Passo and Cook, 1994). The 29 year half-life of strontium would allow the total activity of strontium to be increased by accumulating urine samples over a period of several weeks and measuring the total activity in the accumulated sample.

The figure above shows that the ratio of the activity in the skeleton to the activity in a daily urine sample increases with time until approximately 20 years post intake and then tends to approach a constant value. Physically this is consistent with the idea that after this time nearly all the activity in the body is concentrated in the skeleton; a small fraction of this activity will recycle to the blood and some of the activity in the blood will be excreted in urine. If the skeletal activity and urine activity can both be measured, this would give a useful indication of the possible time of intake (to within approximately 20 years).

### 2.5.2 Inhalation

#### Assume

- 1. the bomb yield at Hiroshima was approximately 10<sup>15</sup> Bq of <sup>90</sup>Sr (UNSCEAR, 2000);
- 2. all this activity was deposited (fall-out) in an area approximately 3 km by 1 km in extent, located approximately 3 km to the west of the city;
- 3. After 10 months, this activity was uniformly mixed in the soil to a depth of 1 cm;
- 4. An individual worked in this area for 8 hours per day for 12 months;
- 5. The individual was engaged in moderate exercise involving rubble clearing.

The surface activity immediately after the fall-out was deposited was approximately  $3\times10^7$  Bq m<sup>-2</sup>. The concentration of <sup>90</sup>Sr in soil after 10 months would be  $3\times10^3$  Bq cm<sup>-3</sup>. For a soil density of 1.6 g cm<sup>-3</sup> this implies an activity concentration of 2000 Bq g<sup>-1</sup> in the surface soil. In Australia the nuisance dust limit in the workplace is 10 mg m<sup>-3</sup> (ANHMRC, 1978). In the absence of measured dust levels in areas where rubble was being cleared, this value can be used as a reference value for this calculation. Assume that the exercise regime for moderate exercise is 50% heavy exercise (breathing rate 3 m<sup>3</sup> hr<sup>-1</sup>) and 50% light exercise (breathing rate 1.5 m<sup>3</sup> hr<sup>-1</sup>), giving an average breathing rate of 2.25 m<sup>3</sup> hr<sup>-1</sup>, which corresponds to an intake of 45 Bq hr<sup>-1</sup>, or 360 Bq day<sup>-1</sup> (using assumption 4). The particle size for the workplace is normally assumed to be 5 microns Activity Median Aerodynamic Diameter (Jarvis et al, 1995). This corresponds to a physical diameter of 3.5 microns.

Using LUDEP for a continuous intake of 360 Bq d<sup>-1</sup> for 1 year, and including the contribution from the <sup>90</sup>Y produced as the result of the decay of <sup>90</sup>Sr within the body, gives a committed effective dose (50 years) of 6.9 mSv and a total activity in the skeleton after 50 years of approximately 120 Bq. For this case the estimated committed equivalent dose (50 years) to the bone marrow is approximately 18 mSv.

#### Bone measurements

<sup>90</sup>Sr and <sup>90</sup>Y are both beta emitters and weak gamma emitters and are therefore very difficult to detect by whole body (external counting). A measurement of activity in bone would have to be done by biopsy.

#### Urine measurements

The estimated daily excretion rate in urine for this case is approximately 4 mBq per day. These values include the activity due to <sup>90</sup>Y. For the urine the <sup>90</sup>Y would decay sufficiently fast that the measurement would only detect the activity (approximately 2 mBq per day) due to the <sup>90</sup>Sr. The daily volume of urine excreted by reference man (ICRP, 1975) is 1400 mL. This would imply a concentration of <sup>90</sup>Sr in a daily urine sample of approximately 1.4 mBq per Litre. For the intake corresponding to a committed effective dose of 10 mSv this would imply (by simple scaling) a concentration of <sup>90</sup>Sr in a daily urine sample of approximately 6 mBq per Litre. This is close to the limit of detection (Boecker et al, 1991; Passo and Cook, 1994) for measurement by liquid scintillation counting. The 29 year half-life of strontium would allow the total activity of strontium to be increased by accumulating urine samples over a period of several weeks and measuring the total activity in the accumulated sample.

This should be an over estimate of the doses received via inhalation because

- 1. not all the fall-out would have been deposited close to the point of detonation;
- 2. the calculation does not allow for dilution of the deposited material by surface run-off or leaching;
- 3. it is unlikely that any of the Australian personnel would have spent 12 months clearing rubble in the fall-out area.

For a soil concentration of 2000 Bq  $g^{-1}$  each of  $^{90}$ Sr,  $^{90}$ Y and  $^{137}$ Cs uniformly mixed to a depth of 1 cm, which corresponds to a concentration of 3.2 GBq  $m^{-3}$  for each nuclide, and using the dose rate coefficients given by Eckerman and Ryman (1993), the estimated external dose rate would be 1.85  $\mu$ Sv  $hr^{-1}$ .

The measured external dose rate due to fall-out at February  $1^{st}$ , 1946 was approximately 0.384  $\mu Sv$  hr<sup>-1</sup>, so the assumed case gives an external dose rate that is approximately 5 times higher than the measured external dose rate shortly before the Australians arrived in the area. This implies that the estimated dose for the inhalation pathway would be too high by a factor of 5. This would in turn imply that detection (in urine) of strontium inhaled in this way would be difficult, as the excretion rate would be close to the limit of detection.

# 2.5.3 Ingestion of a strontium "particle"

Another possible mode of exposure that has been suggested is the ingestion of a very small (invisible) particle of <sup>90</sup>Sr. Assuming the same concentration in the surface soil as before (2000 Bq g<sup>-1</sup>), a soil density of 1.6 g cm<sup>-3</sup> and that approximately 370,000 Bq of <sup>90</sup>Sr needs to be ingested to give a committed effective dose of 10 mSv, the mass of soil that would need to be ingested to deliver this dose would be approximately 180 g, and the diameter of a spherical particle containing this much activity would be approximately 6 cm (about the size of a tennis ball).

# 2.5.4 Ingestion of <sup>90</sup>Sr at a constant rate

Running LUDEP for a chronic input of 1 Bq  $d^{-1}$  via ingestion for 1 year gives a committed effective dose of approximately 10  $\mu$ Sv. Therefore, to give a dose of 10 mSv via this mode of intake, the intake would have had to be approximately 1000 Bq  $d^{-1}$  for 1 year.

Using this intake (1000 Bq d<sup>-1</sup> for 1 year) in the computer model described by O'Brien (2000) and estimating the activity in the skeleton and urine 50 years after the start of the intake gives

approximately 1000 Bq in the skeleton and approximately 70-80 mBq d<sup>-1</sup> in urine. The results of this calculation are shown in Figure 6.

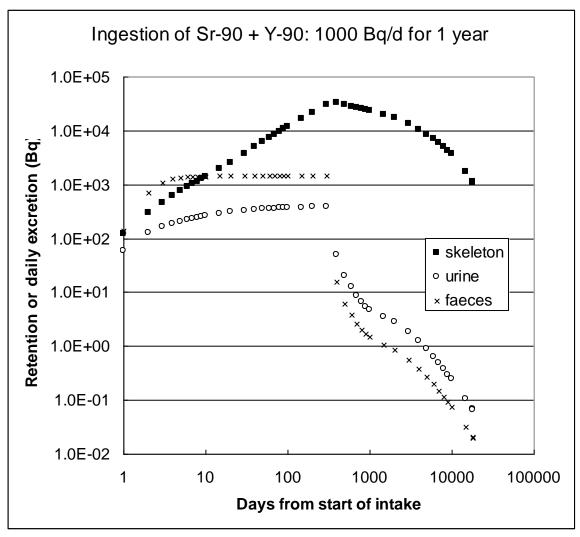


Figure 6:

These results support the conclusions (see Appendix 2.5.1) that the ingestion of <sup>90</sup>Sr at the rate necessary to result in a committed effective dose of 10 mSv would result in an amount of activity in the skeleton that should be detectable after 50 years.

# 2.5.5 Injection of strontium via an open wound

The section on the ingestion of a "particle" of strontium gives a clear indication of the activity, and the corresponding mass of soil, that has to be introduced via ingestion to lead to a committed effective dose (50 years) of 10 mSv. The dose per unit intake for injection is approximately a factor of 3 higher than that for ingestion. This implies that the amount of soil (contaminated with <sup>90</sup>Sr to the level discussed in scenario 2.5.3) that would have to enter the body via an open wound to deliver a committed effective dose of 10 mSv would be approximately 60 grams. The likelihood of this occurring as the result of a single injury would be very small.

# 2.5.6 Effects of Atmospheric Weapons Testing

UNSCEAR (2000) summarise the available data on the contribution to the total annual dose from the ingestion and inhalation of  $^{90}$ Sr from atmospheric weapons testing. The results show that the dose resulting from the ingestion of  $^{90}$ Sr in the environment as a result of atmospheric nuclear weapons testing averaged approximately 2-3  $\mu$ Sv per year from 1950 onwards.

This corresponds to an intake of approximately 100 Bq per year or approximately 0.27 Bq per day (IAEA, 1996).

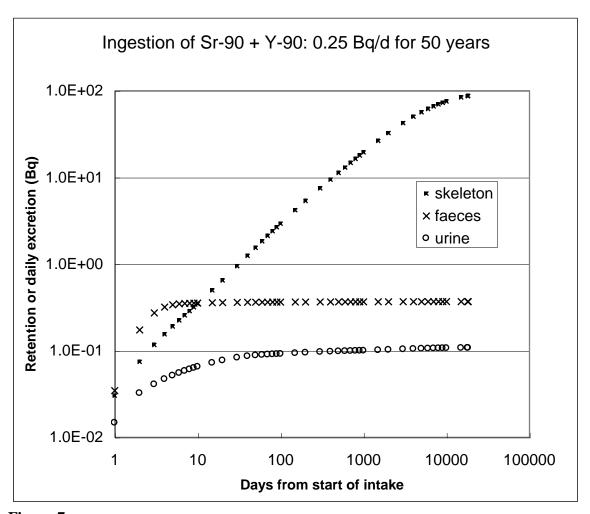


Figure 7:

Figure 7 shows the estimated activity in the skeleton and urine for an intake of  $0.25 \text{ Bq d}^{-1}$  of  $^{90}\text{Sr} + ^{90}\text{Y}$  for 50 years. The estimated activity in the skeleton is approximately 90 Bq, and the estimated activity in a daily urine sample is approximately 100 mBq d<sup>-1</sup>, including the contribution from  $^{90}\text{Y}$ .

These estimates indicate that the contribution to the  $^{90}$ Sr +  $^{90}$ Y activity in a daily urine sample, as a result of consumption of  $^{90}$ Sr present in the environment from nuclear weapons testing, could be the same as that in the urine due to an intake at Hiroshima that would have delivered a committed effective dose of 10 mSv. However the activity in the skeleton as a result of consumption of  $^{90}$ Sr from weapons testing fall-out could be as high as 10% of that

which would result from the intake (via ingestion) of the quantity of  $^{90}$ Sr (+  $^{90}$ Y) at Hiroshima (over 1 year) necessary to deliver a committed effective dose of 10 mSv.

#### 2.5.7 Uncertainties in estimated doses

The uncertainties in the transfer rates between different tissues in the body are considerable (at least  $\pm 25\%$  in most cases and probably as high as  $\pm 50\%$ ). This can lead to large uncertainties in the doses estimated over long periods. One way of countering these uncertainties would be to compare bone samples taken from those people who may have been exposed at Hiroshima with samples taken from people who were not at Hiroshima and who are approximately the same age as the potentially exposed people.

# **2.5.8 Summary**

The preceding discussion indicates that if an individual spent 12 months working in the fall-out area (Koi-Takasu), engaged in rubble clearing work or other tasks that may have produced similar levels of dust, for 8 hours per day, every day of the year, it may have been possible to incur a committed effective dose of 6.9 mSv via inhalation, or 0.03 mSv per day. Calculations indicate that if this did occur the strontium level in a daily urine sample may be detectable. The total amount of excreted strontium (in urine) could be increased by accumulating urine samples. The ratio of the strontium activity in bone to the strontium activity in urine would also be a crude indicator of the time of intake (for ingestion of strontium. This would involve measurements of strontium in bone, which would be possible via biopsy techniques. A comparison of the results of such measurements (urine and/or bone) with the results of measurements taken on a control group (see earlier) would also be a useful indicator.

Ingestion of  $^{90}$ Sr at a rate which would have led to a committed effective dose of 10 mSv to an individual would result in a rate of excretion of activity in a daily urine sample which should be measurable today, but the daily excretion rate would be similar in magnitude to the excretion rate resulting from the average intake of  $^{90}$ Sr from atmospheric weapons testing fall-out. The  $^{90}$ Sr +  $^{90}$ Y activity in the skeleton from the Hiroshima intake would however be approximately 5-10 times higher than the corresponding activity in the skeleton from the intake of  $^{90}$ Sr from atmospheric weapons testing fall-out.

Calculations of the amounts of strontium that would have to have been taken into the body by inhalation or ingestion at Hiroshima indicate that these intakes would only have occurred under very unusual conditions. The dose per unit intake for entry to the body via injection (open wound) is higher than that for intake by inhalation or ingestion, but the conclusion would be the same.

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# 2.6 TOTAL DOSE RATES, COMMITTED EFFECTIVE DOSES AND COMMITTED EQUIVALENT DOSES TO BONE MARROW

In general the total dose resulting from exposures via a number of different pathways can be calculated by summing the doses incurred via each pathway and allowing for differences in the effects of different types of radiation on the same tissue and the different responses of different tissues to the same type of radiation. This is reflected in the equation presented earlier, which can be written as

$$E_{50} = \sum_{T} w_T \sum_{R} w_R D_{T,R}(t,t+50),$$

where E is the committed effective dose,  $w_T$  is the tissue weighting factor,  $w_R$  is the radiation weighting factor and  $D_{T,R}$  is the total absorbed dose in tissue T from radiation of type R resulting from the exposure. This formula allows specifically for the dose contribution from radionuclides that are taken into the body and subsequently decay inside the body, possibly over some considerable period of time after the original intake.

For acute intakes E is calculated by including the contributions from all radioactive decays inside the body out to 50 years following the exposure.

This equation strictly applies only to acute intakes, i.e. instantaneous, once-only intakes. For the Hiroshima situation where the exposures occurred over a period of time the contribution to the committed effective dose resulting from an exposure that occurs during the time interval  $(t,t+\Delta t)$  is given by

$$\Delta E_{50} = \sum_{N} \sum_{T} w_{T} \sum_{R} w_{R} D_{T,R}(t, \Delta t, t + 50),$$

to take the ongoing nature of the exposure into account. For adults the tissue and radiation weighting factors can be assumed to remain constant with time.

For external exposures (where the dose is delivered at the time of the exposure) due to surface contamination  $D_{T,R}$  is given by

$$D_{(\rho S)TR}(t, \Delta t, t + 50) = K_{\rho S}.F_{S}(t),$$

where  $F_S(t)$  is the surface concentration of radionuclides (Bq per cubic metre) at time t, and  $K_{eS}$  is a dose conversion factor (in the appropriate units) which depends on the nuclide

For external exposures due to bulk contamination  $D_{T,R}$  is given by

$$D_{(eB)T.R}(t, \Delta t, t + 50) = K_{eB}.F_{B}(t)$$
,

where  $F_B(t)$  is the bulk or volume concentration of radionuclides (Bq per cubic metre) at time t, and  $K_{eB}$  is a dose conversion factor (in the appropriate units) which depends on the nuclide

For internal exposures the dose is delivered continuously after the intake occurs. For ingestion

$$D_{(ing)T,R}(t,\Delta t,t+50) = K_{ing}(t,t+50).\Delta I_{ing}$$

where  $\Delta I_{ing}$  is the quantity of radionuclides ingested during the time interval (t,t+ $\Delta t$ ). If  $C_{food}$  is the concentration of radionuclides in food, then, assuming that food is consumed at a constant rate,  $\Delta I_{ing} = C_{food}.Q.\Delta t$ , where Q is the quantity of food consumed. Therefore

$$D_{(ing)T,R}(t, \Delta t, t + 50) = K_{ing}(t, t + 50).C_{food}.Q.\Delta t$$

For internal exposures via inhalation,

$$D_{(inh)TR}(t, \Delta t, t + 50) = K_{inh}(t, t + 50).C_{air}.B.\Delta t$$

where C<sub>air</sub> is the concentration of radionuclides in air, and B is the breathing rate.

All these formulae apply to intakes involving a single radionuclide. Where the intake consists of a mixture of radionuclides, the effect of each radionuclide has to be summed. Where ingrowth of radioactive decay products can occur after intake, this has to be allowed for in the dose coefficient.

For the Hiroshima case the significant exposure pathways are

- (1) external due to soil contamination resulting from neutron activation (Appendix 2.1),
- (2) external due to surface contamination by fall-out (Appendix 2.2),
- (3) internal due to inhalation of dust (Appendix 2.5.2),
- (4) internal due to ingestion of contaminated food (Appendices 2.4.2 and 2.5.1),
- (5) internal due to ingestion of contaminated water (Appendix 2.4.3).

For the purpose of calculating the dose to an individual who spent the his/her entire time at Hiroshima in the Koi-Takasu area, pathway (1) can be disregarded.

# **Summary of Results**

Using the assumptions in the previous Appendices, the committed effective doses for the major pathways (assuming that the major contributor to the dose is <sup>90</sup>Sr) are summarised in the following Table for occupancy periods of 1 month, 3 months and 6 months. For clarity the assumptions are repeated here.

External Exposure: the exposed individual was in the Koi-Takasu area for 24 hours per day,

7 days per week for the entire duration of the stay in Hiroshima.

Food: the exposed individual consumed only food grown in soil contaminated

to the level measured in the Koi-Takasu area

Drinking Water: the exposed individual consumed only water contaminated by contact

with soil that was contaminated to the level measured in the Koi-Takasu

area.

Dust:

exposed individual was engaged in rubble clearing in the Koi-Takasu area for 8 hours per day, 7 days per week for the entire duration of the stay in Hiroshima; the dust loading in air was assumed to be 10 mg per cubic metre.

Pathway	Intake Rate	Dose Rate		Dose (mSv)	
	Bq d <sup>-1</sup>	mSv d <sup>-1</sup>	6 months	1 year	2 years
External		9.22×10 <sup>-3</sup>	1.68	3.36	6.72
Food ( <sup>90</sup> Sr+ <sup>90</sup> Y)	185		0.168	0.336	0.672
Water ( <sup>90</sup> Sr+ <sup>90</sup> Y)	1.37		0.00091	0.00182	0.00364
Dust ( <sup>90</sup> Sr+ <sup>90</sup> Y)	360		3.45	6.90	13.8
Total			5.3	10.6	21.2

**Table A2.6.1:** Committed effective doses for different occupancy periods resulting from external exposure, ingestion of contaminated food and water and inhalation of contaminated dust (rubble clearing).

The doses resulting from the ingestion of contaminated food were shown by the model calculations to be approximately 10% of the external doses for continuous occupancy.

The dust inhalation calculations were based on the assumption that the concentration of <sup>90</sup>Sr in the surface soil was 2000 Bq g<sup>-1</sup> (Appendix 2.5.2). As pointed out in that Appendix, this would give rise to an external dose rate of approximately 1.85 micro-Sievert per hour. This value is approximately 5 times higher than the measured external dose rate. However, because of the lack of reported measurements of dust levels, the intake rate for <sup>90</sup>Sr via dust inhalation cannot be reduced below the assumed value with any degree of confidence. It should be noted, however, that the nuisance dust limit corresponds to a level at which respirators would be required in the modern workplace, and would constitute an extremely uncomfortable working environment..

The following Table summarises the committed equivalent doses to bone marrow corresponding to the committed effective dose given in Table A2.6.1.

Pathway	Committed Equivalent Dose to Bone Marrow(mSv)			
	6 months	1 year	2 years	
External	1.17	2.35	4.70	
Food ( <sup>90</sup> Sr+ <sup>90</sup> Y)	0.163	0.325	0.65	
Water (90Sr+90Y)	0.000875	0.00175	0.0035	
Dust (90Sr+90Y)	0.236	0.472	0.943	
Total	1.57	3.15	6.30	

**Table A2.6.2:** Committed equivalent doses to bone marrow for different occupancy periods resulting from external exposure, ingestion of contaminated food and water and inhalation of contaminated dust (rubble clearing).

The values shown in Table A2.6.2 were derived from those in Table A2.6.1 by noting that

- The committed equivalent dose to the bone marrow from external exposure to radiation from <sup>90</sup>Sr is approximately 70% of the committed effective (whole-body) dose (Eckerman and Ryman, 1993);
- The committed effective dose to the bone marrow for ingestion of <sup>90</sup>Sr is approximately 11.6% of the total committed effective dose (Jarvis et al, 1995);
- The committed effective dose to the bone marrow for inhalation of <sup>90</sup>Sr is approximately 0.82% of the total committed effective dose (Jarvis et al, 1995);
- The weighting factor (relative sensitivity) of the bone marrow is 0.12 (ICRP, 1995).

For example, if the committed effective dose from inhalation of contaminated dust for 1 year of exposure is 6.9 mSv, the committed effective dose to the bone marrow will be  $0.0082 \times 10.6 = 0.0566$  mSv. Since the tissue weighting factor for bone marrow is 0.12 the committed equivalent dose to the bone marrow will be 0.0566/0.12 = 0.472 mSv.

These estimates are likely to be upper limits.

The results of this Appendix are summarised in Table A2.6.3 below.

Period of Exposure	Total committed equivalent	Total committed effective
	dose to bone marrow (mSv)	dose (mSv)
6 months	1.57	5.3
1 year	3.15	10.6
2 years	6.30	21.2

**Table A2.6.3:** Total committed equivalent dose to bone marrow and total committed effective dose for different periods of exposure at Hiroshima.

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