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Assessment of the possible risks of radiation induced health effects from contamination at the University of Manchester

KA Jones, WB Oatway, RGE Haylock, S Holmes and JR Simmonds

Contract Report

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EXECUTIVE SUMMARY

This study was carried out to estimate the risks from exposure to radioactive contamination in two buildings at the University of Manchester used by Professor Rutherford and his colleagues in early research work with radioactive materials. The study is an input into the inquiry being carried out by Professor David Coggon following concerns from staff about possible health effects from working in those buildings.

Radionuclides from each of the uranium-238, thorium-232 and uranium-235 decay chains were used by Professor Rutherford and his colleagues. The first stage in the assessment was to collect all relevant information on radioactive contamination at the affected locations at the University. This information was then used to derive representative levels of contamination at a generic location which were used to estimate radiation doses and risks of radiation induced health effects. The aim was to calculate doses to hypothetical individuals from exposure to radioactive contamination that could have occurred in the past and from current exposure levels, in each case assuming a working lifetime of 40 years. As measurements of radioactive contamination were not available before 1999, assumptions had to be made to determine levels in earlier years (1950 to 1989 were considered) and a cautious approach was adopted to try to ensure that the risks were not underestimated. Two source terms were used in the study, one representing the more likely amount of radioactivity (referred to as the 'base case') and the other representing an upper bound of the possible levels of contamination. Remediation of the buildings was carried out between 2000 and 2004. Measurements made after the remediation were used to estimate the contamination levels for the assessment of exposure for current and future occupants.

The possible radiation exposures to two groups of staff were considered. The first group represented office-based university staff, who were likely to have spent most time in the rooms where contamination had been found. The second group consisted of maintenance workers who would have spent less time in the relevant areas but who may have had raised exposures compared to the office-based workers in the short term owing to the nature of their work. For each group and time period considered, radiation doses were estimated and used to estimate the risks of radiation induced health effects.

The radiation doses calculated in this study were committed effective doses and the committed equivalent doses to bone surfaces, brain, liver, pancreas, red bone marrow and lungs. Radioactive material taken into the body is retained in organs and tissues until it is excreted while at the same time undergoing radioactive decay. The period over which a radionuclide delivers dose can vary from a few days to many decades depending on its chemical and physical decay characteristics. Committed dose takes account of the time radioactive material remains in the body by estimating the dose which will be delivered over a 50 year period following its intake. The doses estimated for the office-based university worker were higher than those for the maintenance worker because of the greater time spent in the area. The highest estimated committed effective dose, summed over the period from 1950 to 1989, was about 75 mSv for the

office based worker assuming the upper bound source term. The dose estimate for the base case was slightly lower at 72 mSv.

The organs and tissues estimated to receive the highest doses were the lungs and bone surfaces. Estimated doses for the pancreas and the brain were about an order of magnitude lower.

The highest risk estimated was for the hypothetical office-based workers, assuming the upper bound contamination scenario from 1950 to 1989. The highest risk of cancer mortality in this hypothetical scenario would be from lung tumours, with a lifetime risk of exposure-induced death (REID) of 0.6%. In other words, this estimate means that approximately 60 in every 10,000 people exposed to this level of radiation would die from a radiation-induced lung cancer. This compares with a baseline risk of dying from lung cancer in the general population of about 7% (700 deaths per 10,000). The average years of life lost in an individual who died of lung cancer as a consequence of their exposure would be approximately 12.

The estimated risks of pancreatic and brain cancer were much lower with REIDs of 0.004% and 0.003% respectively (less than 1 in every 10,000 deaths), about a hundred times lower than the baseline risk of dying from these cancers.

Extensive remediation of both buildings was carried out between 2000 and 2004. Cautious estimates of current and future doses were obtained by using measurements that included the contribution from natural background. The estimated effective dose over 40 years was 48 mSv for the office-based worker and 7 mSv for the maintenance worker. These doses are lower than those estimated for past exposures and the associated risks are correspondingly low. Any residual contamination by radionuclides is not at a level that indicates a need for further decontamination or routine exposure monitoring in order to protect the health of people working in the buildings. However, before carrying out any future intrusive maintenance work that will significantly disturb floor or wall materials, a radiological risk assessment should be made to determine whether other additional measures are needed to protect those involved in the work.

Uranium-238, thorium-232 and uranium-235 and their decay products occur naturally in the earth's crust and everyone receives doses from exposure to these and other natural sources, including from cosmic radiation. In order to put the doses estimated in this assessment into context the exposure from natural background radiation is given for comparison. The exposure from natural background for the average member of the UK population is estimated as about 90 mSv in 40 years (2.2 mSv per year). However, this value does vary considerably, with the lower end of the range being about 40 mSv and the upper end being about 4000 mSv over the 40 year period.

Measurements made of post-mortem samples and a tooth from a former staff member, indicate levels of exposure which are consistent with those found in the general population. This provides some evidence in support of the assumption that the assessed doses for a hypothetical individual will not have significantly underestimated the actual exposures received by University of Manchester staff.

HPA would like to acknowledge valuable contributions from a number of individuals. This work would not have been possible in the available timescale without the large

amount of research carried out by Neil Todd and John Churcher. Also, discussions have taken place throughout with Professor Coggon and this has also been extremely beneficial. An important aspect in this study was that the concerned staff from the University of Manchester and their families were kept informed and consulted about the work and were given the opportunity to question what was being done. In addition comments received on HPA's provisional report were taken into account in this final report. All of these inputs have improved the study.

CONTENTS

1 INTRODUCTION

Concerns were raised by staff who worked from the 1970s to 1990s at certain locations on the University of Manchester campus about possible health effects from radioactive and chemical contamination. These locations were used by Professor Ernest Rutherford in carrying out work with radioactive material between 1907 and 1919 and residual radioactivity has subsequently been found in these areas. An inquiry was set up under the independent chairmanship of Professor David Coggon to examine the possibility of health effects resulting from the contamination. The Health Protection Agency (HPA) has expertise in providing guidance and advice on the health effects of radiation in both the environment and the workplace. As input into the inquiry, HPA published a report providing background material on the risks from exposure to radiation (Oatway et al, 2009). APPENDIX A gives information taken from that report, including details of the nature of ionising radiation and the radionuclides of interest. Professor Coggon also asked HPA to carry out an assessment of the possible health risks to University staff from exposure to the radioactive contamination and this report details the work.

2 LOCATIONS OF INTEREST

The assessment relates to two buildings at the University where Professor Rutherford and his colleagues carried out their work with radioactive materials, the Physical Laboratory (opened in 1900) and its extension (opened in 1912). Both buildings have undergone many changes of use and name since the early 20th century. Table 1 gives the most significant names and uses of the buildings based on information provided by the University of Manchester and given in a report by Dr Neil Todd (Todd, 2008). Throughout this report these two buildings will be referred to by their current names: the Rutherford Building and the Coupland 1 Psychology Annex. APPENDIX B contains the floor plans and indicates the main areas of interest.

Table 1 Names and uses of the Rutherford Building and its Annex from 1900 onwards

Year	1900	1912	1945	1968	2000
Name	Physical Laboratories		Schuster laboratory	Coupland 1 (part) Manchester Museum (part)	Rutherford building (part) Manchester Museum (part)
Use	Department of Physics			Department of Psychology (part) Manchester Museum (part)	International Student Admissions Manchester Museum (part)
Name		Extension to Physical Laboratories or 1912 Extension	Schuster Extension	Coupland Extension (part of Coupland 2)	Coupland 1 Psychology Annex
Use		Department of Physics		Department of Psychology	

3 APPROACH USED IN THE DOSE AND RISK ASSESSMENT

HPA were asked to perform two assessments of the radiological consequences of the radioactive contamination present in the Rutherford Building and the Coupland 1 Psychology Annex: one for past exposures to ionising radiation resulting from contamination in the Rutherford Building and the Coupland 1 Psychology Annex and the other for current and future exposures.

The assessments were intended to be representative of the highest exposures of typical occupants of the buildings. It was the aim that no person who may have been exposed while working at the University in the period of interest would have a dose or risk greater than that estimated for one of the generic groups, and in most cases individuals would be expected to have a dose, and hence risk, significantly less than that estimated here.

For the assessment of past exposures HPA considered the doses and risks to people working in the building between the years 1950 and 2000. The year 2000 was chosen as the end point of the assessment of past exposures as the refurbishment of the Rutherford Building, which included the removal of radioactive contamination, began in this year. Following this remedial work the exposure levels would have been significantly reduced. The year 1950 was chosen as an early enough start point to include anyone who had worked in the buildings but who had now retired. In discussion with Professor Coggon, it was decided to assume a maximum working time in the buildings of 40 years. Therefore, results in this report are presented assuming that exposure occurred between the years 1950 to 1989; exposures between 1960 and 1999 would have been lower and so were not presented. It was thought that no individual had worked in the buildings over such a long time period and this is therefore an example of the cautious approach adopted in the assessment, considering the exposure of a hypothetical worker at the University rather than specific individuals.

It must be stressed that the assessment is intended to be representative of the highest exposure of typical occupants of the building. Generic parameters have been assumed for inadvertent ingestion rates, inhalation rates and occupancy of the rooms and no variation over time of these parameters has been assumed. It should be noted that these generic parameters are cautious and likely to result in higher doses than would be expected. If variations in the exposure routes had been assessed the doses would not be higher than those reported.

For the assessment of past exposures, the first stage in the assessment was to review the available information on the potential contamination at University of Manchester over the last 100 years or so. This included a review of the available monitoring reports as well as historical records that provided information on the work of Professor Rutherford and his colleagues with radioactive materials.

Unfortunately, monitoring records of radioactive contamination only exist for 1999 onwards. Information provided by the University (Peters, 2008) does not indicate that any substantial building work or remediation was carried out prior to 1999. There is anecdotal information that rooms in the Rutherford Building were replastered and repainted in the early 1960s due to high level of contamination found in them (Private

correspondence to Professor Coggon, 2008). John Churcher, who worked into the Rutherford Building from the early 1970s to 1999, has indicated that there was no substantial building or remediation work during this time although he did comment that walls and ceilings were drilled through in the 1990s to create cable ducts for the Ethernet (Churcher et al, 2008). If remediation work was carried out in the 1950s or 1960s then exposures received before this remediation work may have been higher than those reported.

The assessment is mainly based on measurements made in 1999 and decay corrected back to earlier years to estimate the contamination levels from 1950 to 1989. More detail is provided in the following section and APPENDIX C.

The exposure levels for current and future exposures were derived from monitoring results gathered after completion of remediation work to relevant parts of the buildings.

4 CONTAMINATION LEVELS

The first stage in any assessment is to estimate the amount of radioactivity to which people may have been exposed. For the natural uranium and thorium decay chains, all radionuclides are isotopes of solid elements, except for isotopes of radon which is a gas. The contamination levels for solid materials are either given as surface contamination (Bq cm^{-2}) or activity concentration (Bq g^{-1}). Radon levels are expressed as activity concentrations in air (Bq m^{-3}). APPENDIX A gives more information about the natural decay chains.

4.1 Contamination levels used for assessment of past exposures

Remediation of the buildings was carried out from 2000 to 2004, with monitoring records prior to remediation being available from 1999. Contamination levels in years prior to 1999 were derived using these monitoring records. To take account of the potential uncertainties it was cautiously assumed that the highest measured level of any radionuclide in a decay chain, rounded up to the nearest order of magnitude where necessary, was representative of the level of the other radionuclides in the decay chain. Two different source terms were used in this assessment.

- **Base case**. This source term was derived from information obtained from monitoring reports and included only those radionuclides that were mentioned explicitly or which, owing to their short half-lives, were expected to be present due to the radioactive decay of longer lived radionuclides that were mentioned in the monitoring reports. In addition, thorium-230 and actinium-227 were included as there was sufficient historical information for estimates of total amounts to be made (see APPENDIX C for more details). It should be noted that this source term specifically did not include those radionuclides at the top of the uranium-238 and uranium-235 decay chains as these radionuclides were not

reported (from uranium-238 to uranium-234 and uranium-235 to protactinium-231 respectively).

- **Whole chain case**. Although they are not mentioned in the monitoring reports there is evidence (Todd, 2008) that uranium and thorium compounds were used by Professor Rutherford and his colleagues and could have been part of any contamination caused by the work. Therefore, a further source term was used in which it was cautiously assumed that the entire decay chains of the uranium and thorium series were present.

The source term for the base case is presented in Tables 2 to 4 and for the whole chain case in Table 5. The activities in the Tables represent the activity present in the year 2000. More information on the derivation of the activities is given in APPENDIX C.

Radon-222 monitoring data were only available for one room prior to remediation of the buildings. The highest measured radon-222 concentration was 30 Bq m^{-3}. In order to supplement this lack of data, the radon concentration in air was also derived based on the activity of radium-226. This calculation (described in section C2.8) resulted in a radon-222 concentration of 180 Bq m^{-3}, and this more cautious value was used for both source terms.

No measurements were available for radon-220 (thoron) in any of the surveys. This is not unusual as radon-222 doses are typically about ten times higher than those from radon-220 in the UK (Watson et al, 2005) and so usually just radon-222 is measured. However, radon-220 was considered in this study for completeness, using an activity concentration in air of 50 Bq m^{-3} at 1 m above floor level derived from the measured activity concentration of actinium-228. This calculation is detailed in APPENDIX C, section C2.8.

The activity concentrations and surface contamination levels of the other radionuclides were based on monitoring records. From these monitoring records there is evidence that there is some activity disequilibrium within the decay chains, specifically for radium-226 compared with lead-214, bismuth-214 and lead-210. The activity concentrations of lead-214, bismuth-214 and lead-210 varied from a few percent to 50% of the value for radium-226 (Turner, 2000a, b). This range was probably due to the fact that some of the radon-222 gas, produced by the decay of radium-226, would have escaped from the building. As a result, there would be less lead-214, bismuth-214 and lead-210 and other progeny present than radium-226. Nevertheless, to be cautious it was assumed that all of the radionuclides were in secular equilibrium with radium-226.

Wherever measurements for a particular radionuclide were available for surface contamination (Bq cm^{-2}) and activity concentration (Bq g^{-1}), they were similar or within an order of magnitude (see APPENDIX C for more details). Where both types of measurement were available, the higher of the two values was used and in all cases the same value was assumed for surface contamination (Bq cm^{-2}) and activity concentration (Bq g^{-1}).

Table 2 Source term for the base case assessment for year 2000 – uranium-238 decay chain

Radionuclide	Surface contamination Bq cm^{-2}	Activity concentration of dust Bq g^{-1}	Comment
^{238}U to ^{234}U	-	-	Assumed not to be present
^{230}Th	10	10	Based on estimate from historical inventory (see APPENDIX C for details)
^{226}Ra	200	200	Activity concentration of dust based on (Turner, 2000b) (see APPENDIX C for more details)
^{222}Rn	Air concentration of 180 Bq m^{-3}		Estimated using measured activity concentration of ^{226}Ra (see APPENDIX C for details)
^{218}Po to ^{210}Po	200	200	Assumed to be in equilibrium with radium-226

Table 3 Source term for the base case assessment present for year 2000 – thorium-232 decay chain

Radionuclide	Surface contamination Bq cm^{-2}	Activity concentration of dust Bq g^{-1}	Comment
^{232}Th to ^{228}Ra	1	1	Assumed present due to presence of ^{228}Ac
^{228}Ac	1	1	Activity concentration of dust based on (Turner, 2000b) (see APPENDIX C for more details)
^{228}Th to ^{224}Ra	1	1	Assumed to be in equilibrium with ^{228}Ac
^{220}Rn	Air concentration of 50 Bq m^{-3}		Estimated using measured activity concentration of ^{228}Ac (see APPENDIX C for details)
^{216}Po to ^{208}Tl	1	1	Assumed to be in equilibrium with ^{228}Ac

Table 4 Source term for the base case assessment present for year 2000 – uranium-235 decay chain

Radionuclide	Surface contamination Bq cm^{-2}	Activity concentration of dust Bq g^{-1}	Comment
^{235}U to ^{231}Pa	-	-	Assumed not to be present
^{227}Ac	10	10	Based on estimate from historical inventory (see APPENDIX C for details)
^{227}Th to ^{223}Ra	10	10	Assumed to be in equilibrium with ^{227}Ac
^{219}Rn	-	-	Assumed not to be present[(a)]
^{215}Po to ^{207}Tl	10	10	Assumed to be in equilibrium with ^{227}Ac

(a) Radon-219 has a very short half-life (4 seconds) and was assumed to decay away before it can be inhaled

Table 5 Source term for the whole chain case assessment present for the year 2000

Decay series	Surface contamination Bq cm^{-2}	Activity of dust Bq g^{-1}	Comment
^{238}U	200	200	Based on the maximum measured activity which was ^{226}Ra activity concentration of dust (Turner, 2000b). See APPENDIX C for more details.
^{232}Th	1	1	Based on the maximum measured which was ^{228}Ac activity concentration of dust (Turner, 2000a)
^{235}U	10	10	Based on estimate from historical inventory of ^{227}Ac (see APPENDIX C for details)

It was assumed that contamination was present in a discrete area of an office, represented by a circular patch 1 m in diameter, both as dust on the floorboards and under the floorboards. Comments made in the monitoring reports indicate that in general the contamination tended to be located in small areas often only a few centimetres in dimension, although several such areas could be present within a single room, ie, the contamination was distributed heterogeneously. By assuming that the contamination was a circular, homogeneous patch 1 m in diameter on and under the floorboards, the assessment was likely to overestimate the area of contamination and hence the resulting dose. The dust under the floor was assumed to have a thickness of 0.5 cm whereas the contamination on the floor was assumed to be just on the surface, ie, to have no depth. In addition, this patch was cautiously assumed to be located under an area of high occupancy, for example under a chair at a desk, rather than in areas of the room where people are not likely to spend time, under a cabinet for example. In estimating external doses from this contamination, exposures were averaged over the body assuming that the effective height of the person was 1 m above the contamination.

One monitoring survey also reported 'hotspots' of lead-210 contamination in room C1.10 (Turner, 2000a, b). These hotspots were reported to have a relatively small area but an activity that could be over an order of magnitude higher than that measured in other areas. These hotspots were treated as a separate source of exposure. The hotspots contained much higher activity concentrations of lead-210 than of radionuclides higher up the decay chain. It was, therefore, assumed that lead-210 was the primary contaminant rather than being present as the result of radioactive decay. The assessment of the dose from the hotspots was made assuming an activity concentration of 5000 Bq g^{-1} of lead-210 and its progeny, based on the monitoring data for two discrete patches of contamination (Turner, 2000a, b) rounded up to an order of magnitude (see Table 6). APPENDIX C gives more detailed information about the monitoring data used.

The hotspot was taken to be a circular patch of contamination on the wall, measuring 0.1 m in diameter. The assumed distance between the contamination and the person being exposed was assumed to be 0.3 m, representing someone near the wall whilst sitting at a desk or working on the wall using a drill. Figure 1 provides a schematic of the different areas of contamination and their location with respect to the person assumed to be exposed.

Doses from the exposure to the hotspot contamination were summed with the doses from either the base case or whole chain contamination scenarios to give an estimate an overall dose.

Table 6 Contamination scenario for the hotspot present in the year 2000

Radionuclides	Surface contamination Bq cm^{-2}	Activity of dust Bq g^{-1}	Comment
^{210}Pb, ^{210}Po, ^{210}Bi	5000	5000	Based on the maximum measured ^{210}Pb activity concentration (Turner, 2000a, b)

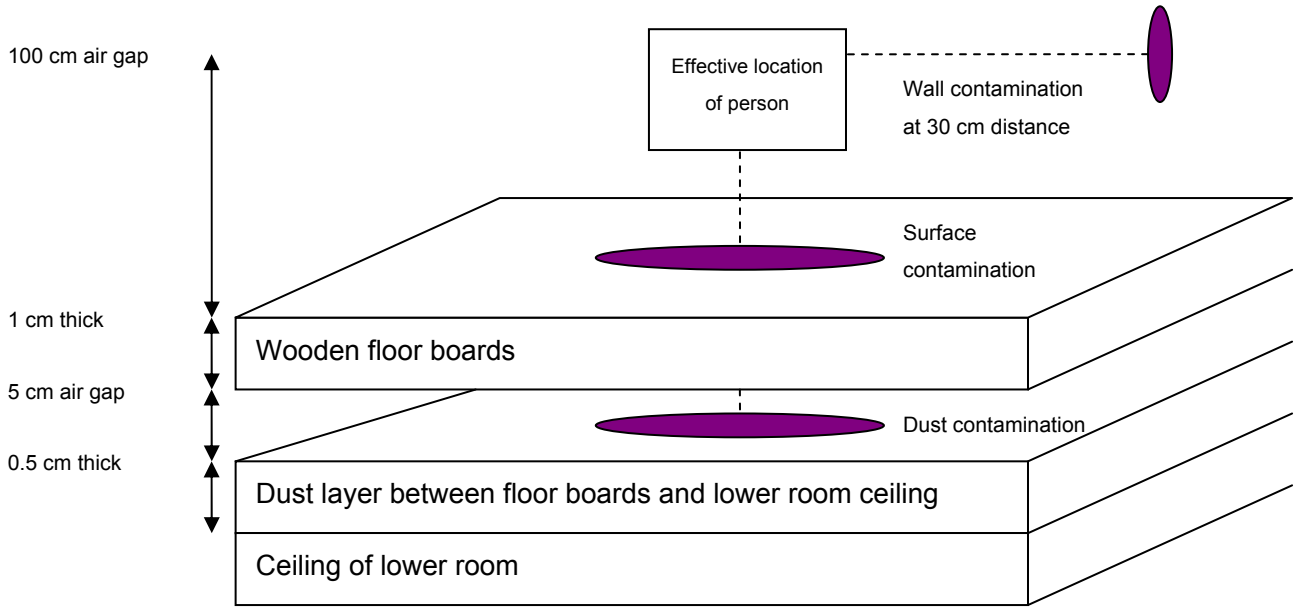

Figure 1 Diagrammatic view of the contamination profile together with the materials of the floor and the location of the person being exposed. Dimensions are not to scale.

4.1.1 Decay corrections

As discussed above, data from measurements made prior to remediation of the buildings (mainly for the years 1999 and 2000) were used to estimate contamination levels in previous years (1950 to 2000). To take account of radioactive decay the radionuclides in each decay series were divided into 3 categories, noting that by the year 2000 any contamination present could have been in place for up to 100 years.

- Long-lived radionuclides (with a half-life of more than 1000 years). This assessment assumed that the activity of any radionuclide with a half-life longer than 1000 years could be considered constant over this period. For reference, the decrease in the activity of a radionuclide with a half-life of 1000 years over a period of 100 years is less than 3%.
- Short-lived radionuclides (with a half-life less than 10 years). Radionuclides with short half-lives could only plausibly be present in 1999 as a result of decay from a longer lived radionuclide. These radionuclides were assumed to have the same activity concentration as their most immediate longer lived parent radionuclide.
- Radionuclides which have a half-life of between 10 and 100 years were assumed to be present in 1999/2000 due to being part of the original source of contamination rather than as a result of decay from a long lived parent. Only two radionuclides required the application of correction for radioactive decay: lead-210 and actinium-227, both of which have half-lives of around 22 years.

A decay correction factor, F, was used to scale the activities assumed for the year 2000 to estimate past activities, as shown below. The year 2000 was used as the year in which most of the monitoring data were gathered.

$F = 1 / [\exp (-\lambda * t)]$

Where

λ = Decay constant of the radionuclide, y^{-1}, taken from ICRP (ICRP, 1983)

t = Number of years before the year 2000, y

Table 7 shows the decay correction factors for selected years within the assessment timeframe. These factors were applied to the activities shown in Tables 2 to 4 and Table 6 in order to account for the reduction of activity over time. For example, levels of lead-210 in 1950 were estimated to be about five times greater than reported in the monitoring surveys carried out around the year 2000.

Table 7 Decay correction factors to be applied to the activities in base case and hotspot source terms

Radionuclide	Year correction applies to:					
	1950	1960	1970	1980	1990	2000
^{210}Pb[(a)]	4.7	3.5	2.5	1.9	1.4	1.0
^{227}Ac[(a)]	4.9	3.6	2.6	1.9	1.4	1.0

(a) No decay correction was applied for the progeny but they were assumed to be in equilibrium with either ^{210}Pb or ^{227}Ac

Figure 2 illustrates the effect of the decay correction. The yellow line shows the variation over time of lead-210 contamination based on the levels assumed for the year 2000 allowing for radioactive decay. The pink line shows the levels of lead-210 as a function of time assuming that it was present only as a result of radioactive decay from radium-226. For this assessment it was cautiously assumed that lead-210 was present in the original contamination and had decayed, rather than being present at the same level as the radium-226 throughout the period.

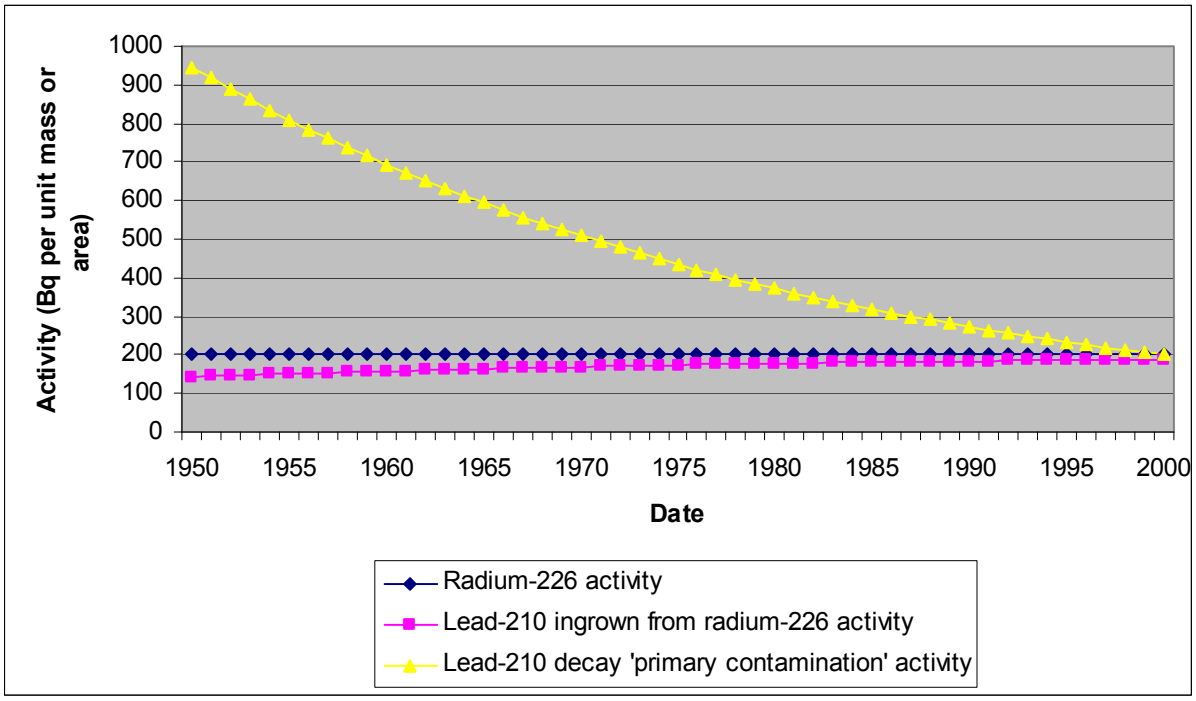

Figure 2 Activities of lead-210 from 1950 to 2000 assuming ingrowth from radium-226 activity or present as a primary contaminant.

4.2 Contamination levels used for assessment of current and future exposures

For the assessment of current and future exposures, dose and risk estimates were based directly on available monitoring data following remediation of the buildings in 2000 to 2004.

The monitoring reports indicated that there may be patches of contamination present at less than 1 Bq cm^{-2} ((Robinson, 2005), 2009; (University of Manchester, 2009). For this assessment a nominal surface activity of 1 Bq cm^{-2} was used to represent the upper end of the possible contamination to be found in the rooms. Using the approach adopted in the assessment of doses from past exposures, this was assumed to equate to an activity concentration of 1 Bq g^{-1}. Monitoring results following remediation of the buildings do not provide information on the relative activity of decay chain members and hence it was cautiously assumed that all members of the uranium and thorium decay chains were present at this activity concentration.

This highest external dose rate measured at 1 m above the floor was 1.7 10^{-4} mSv per hour (see section C 2.9). This dose rate includes the contribution from natural sources, for example naturally occurring radionuclides found in bricks. It is not possible to distinguish between the dose rates from any residual contamination and from natural sources. However, if a residual surface contamination level of 1 Bq cm^{-2} is assumed for the uranium and thorium decay chains and standard models are used to estimate external doses, the resulting dose rate would be 5 10^{-6} mSv per hour. On this basis only

about 5 % of the measured dose rate would result from residual contamination, with the remaining 95% being due to natural sources such as bricks and tiles. For the assessment, doses from external irradiation were cautiously calculated using a rounded dose rate of 2 10^{-4} mSv per hour.

As with any radon-222 measurements made in different rooms in any building the measurements made in the Rutherford Building and Coupland 1 Psychology Annex vary considerably from less than 10 to 410 Bq m^{-3} (University of Manchester, 2009). Measurements will include the contribution from natural sources. The average UK indoor radon-222 concentration is 20 Bq m^{-3} although this can vary up to 17 000 Bq m^{-3} (Watson et al, 2005). The highest radon-222 concentrations in air were measured in a storeroom (G37) in the Coupland 1 Psychology Annex. Since this room is not occupied it was decided that it would not be appropriate to use these measurements for assessing doses. All other radon-222 concentrations were below 180 Bq m^{-3}, the value which had been used for the assessment of past exposures. It was decided to cautiously also use this value for assessments of current and future exposures. For radon-220, it was also decided to cautiously assume that the concentration in air for current and future exposures was the same value as for past exposures, ie, 50 Bq m^{-3}.

No exposure to hotspots of lead-210 contamination was considered in the assessment of current and future doses, since it was assumed that these were removed during the remediation works.

The source term used to assess the current and future doses is summarised in Table 8.

Table 8 Source term for current and future exposures

Dust activity concentration	1 Bq g^{-1}	Estimated from surface contamination (Robinson, 2005 and 2009) and (University of Manchester, 2009)
External dose rate	2 10^{-4} mSv hr^{-1}	From dose rates measured in (Frith, 2001) and (Adams, 2001a).
Radon-222 air concentration	180 Bq m^{-3}	Assumed to be same as value for past exposures.
Radon-220 air concentration	50 Bq m^{-3}	Assumed to be same as value for past exposures.

5 EXPOSED GROUPS AND THEIR EXPOSURE ROUTES

This assessment considered exposure to two groups of people:

- **Office-based staff member**, representing someone who spends all their time within a single office
- **Maintenance workers,** representing those who perform routine maintenance work within the building, which also includes office cleaners.

For both groups the following exposure pathways were considered:

- **External irradiation**
- **Inhalation of radon gas**
- **Inhalation of contaminated dust**
- **Inadvertent ingestion of contaminated dust.**

Each of the pathways is briefly discussed in the following sections. A full description of the methodology, including the equations and the parameters used, are given in APPENDIX D.

5.1 External irradiation

External irradiation occurs when radionuclides, particularly those that emit gamma radiation, are present in an area where someone spends their time. The radiation emitted will pass through the air and into and, possibly through, the body, irradiating a person's organs. The level of exposure will be dependent on the radionuclide and its radioactive emissions (eg, gamma ray energy), the activity present, the location of the radionuclides with respect to where someone spends time, and the length of time spent in the area.

From reviewing accounts of the work performed by Professor Rutherford and his colleagues (Todd, 2008) and data available from monitoring reports, it was judged that most of the contamination found on and under floor boards was due to spills of liquids used in experimental work. There is a possibility that floors have been sanded in the past (Frith, 2000). Dust created by this sanding would have fallen under the floor boards and formed a layer of contaminated wood dust, mixed with other dust. For this assessment, radionuclides were assumed to be located both on the floor boards, represented by a surface layer of activity, and as a layer of dust under the floor boards. For material under the floor boards, account was taken of shielding provided by the overlying wooden floor. A schematic of the position of the contaminated areas with respect to the location of someone using the room is shown in Figure 1 and is discussed in section 4.1.

5.2 Inhalation of radon gas

Radon-222 and radon-220 are radioactive gases that are created from the radioactive decay of isotopes of radium. Once these gases have been created they may be able to escape from the contamination patch and may be inhaled. In addition to direct exposure from radon-222 and radon-220, both of these radionuclides also produce a series of radioactive progeny that irradiate the lungs if inhaled. Exposure from radon-222 and radon-220, and their progeny, was estimated for the entire time that a person occupied a contaminated office. Since radioactive decay following inhalation occurs largely in the lungs, doses to body organs are very small and assessments were therefore made only for dose to the lungs.

5.3 Inhalation of contaminated dust

Inhalation of radionuclides other than radon-222 and radon-220 gas depends on their attachment to dust particles that could get resuspended into the air and subsequently inhaled. For the assessment the activity concentration of dust in the office was assumed to be the same regardless of whether it was in the air or on surfaces. The exposure of a person occupying an office from inhalation of airborne dust was estimated for the entire time they were present.

For an office-based staff member it was assumed that exposure was to ambient dust levels. However, for the maintenance worker some enhancement of the dust levels was expected to occur due to their work. In addition, the maintenance worker was also assumed to be exposed to significantly enhanced dust levels for example as a result of drilling. It was also cautiously assumed that drilling was at the site of a hotspot of contamination (as described in Table 6). Therefore, while carrying out certain tasks the maintenance worker was assumed to inhale more dust per unit time than the office-based staff member. However, overall the maintenance worker was assumed to be exposed for a shorter period of time as they would also have worked in other parts of the building.

The important factors when assessing the inhalation of dust are the inhalation rate and the amount of airborne dust available for intake (called dust loading). The inhalation rate was taken from Smith and Jones (2003) based on data given by the International Commission of Radiological Protection (ICRP, 1975) as representative of an adult spending approximately two thirds of their time doing light exercise (walking) and one third of the time spent sitting. This was considered to provide a cautious estimate for an office-based worker and to be reasonably representative for the maintenance worker.

The dust loading was based on a review of monitoring results carried out over the last few decades in various situations, a summary of which is given in Table 9 (Simmonds et al, 1995). For an office-based staff member a dust loading of 10^{-5} g m^{-3} was thought to be applicable. For the maintenance worker a higher dust loading of 10^{-4} g m^{-3} was assumed to take account of the general work that they carried out. In addition account was taken of significantly raised dust levels experienced by the maintenance worker, during work such as drilling or sanding, leading to a further order of magnitude increase in the dust loading used, ie, 10^{-3} g m^{-3}. For reference, this is an order of magnitude below the Occupational Exposure Limit (OEL) for total inhalable dust of 10^{-2} g m^{-3} specified by the Health & Safety Executive in its Guidance Note EH 40/2005 (HSE, 2005). The OEL is the concentration of an airborne substance, which should not be exceeded, over an 8-hour weighted-average reference period.

Table 9 Typical dust loadings appropriate for a range of activities

Dust Loading (g m^{-3})	Scenario
10^{-2}	Dusty Environment (ploughing, etc). Generally short exposure duration
10^{-3}	Enhanced outdoor ambient dust levels (digging in domestic gardens, site excavation). Short or long durations, occasionally representing an "average" over dusty environment and outdoor ambient levels.
	Urban locations
	Enhanced workplace levels
10^{-4}	Outdoor ambient dust levels, also used in some studies for general ambient exposure levels (ie, indoor and outdoor)
10^{-5}	Indoor ambient dust levels

5.4 Inadvertent ingestion of contaminated dust

Inadvertent ingestion of contaminated dust was considered to occur when someone put their hands, food or other objects in their mouth after they had been placed on surfaces covered with contaminated dust. The amount of dust ingested was considered to vary depending on the action the person was undertaking, with manual work considered to result in a higher dust ingestion rate than office use.

The ingestion rates used were taken from Smith and Jones (2003), a report widely used in radiation protection as a source of data on habits. The ingestion rate used for the office-based staff member and the maintenance worker was derived from studies that considered the ingestion of household dust by infants at home. The value used here of 1 mg per hour is potentially an overestimate as it was based on a daily rate for someone who spends the majority of time at home, in excess of 20 hours a day. In this assessment this daily rate was simply divided by the time spent at work of around 10 hours, that is, the total amount of dust ingested in a day was assumed to be all ingested within the time spent at work. The enhanced rate used for the maintenance worker was assumed to be an order of magnitude greater, 10 mg per hour. No information could be found to refine these rates further.

5.5 Other assessment parameters

The parameters used to define the exposure pathways were assumed to be applicable for past, current and future exposures. This was considered justifiable given the generic nature of the assessment. The parameters used in this assessment were considered to be realistic but cautious.

5.5.1 Exposure times

The exposure times used to assess the dose for each of the exposed groups are given in Table 10. For the office-based staff exposure was assumed to last for an entire working year, taken to be 2000 h y^{-1}. For the maintenance workers it was considered

that only some of the year would be spent in areas where contamination was present due to these workers moving around different buildings. It was assumed that 10% of the working year would be spent in contaminated areas. For most of the time members of this group were considered to be doing tasks that slightly enhanced the dust levels. In addition, a further 1% of the working year was considered to be spent performing actions that raised the dust levels significantly, such as drilling or sanding. These exposure times were taken to be additive; that is, the total time a maintenance worker was exposed to contamination was 220 hours per year, 200 hours at an 'enhanced' level of exposure and 20 hours at work 'creating significant dust levels.

Table 10 summarises the main parameter values used in the assessment.

Table 10 Summary of the parameters assumed in the assessment

	Office-based university staff exposure[(a)]	Maintenance workers undertaking normal work with some 'enhanced' level of dust[(b)]	Maintenance workers undertaking work 'creating significant dust' levels[(c)]
Exposure time	2000 h y^{-1}	200 h y^{-1}	20 h y^{-1}
Ingestion rate of dust	1 mg h^{-1}	1 mg h^{-1}	10 mg h^{-1}
Inhalation rate of dust	1.2 m^3 h^{-1}	1.2 m^3 h^{-1}	1.2 m^3 h^{-1}
Dust loading	10^{-5} g m^{-3}	10^{-4} g m^{-3}	10^{-3} g m^{-3}

(a) Represents those workers who spend the majority of their time at a desk within a single office.

(b) Represents those workers who move between offices and in their work disturb dust within the office. This corresponds to cleaners and maintenance workers performing routine tasks.

(c) Represents the time workers spend performing tasks that involve a significantly raised dust level, for example drilling or sanding.

5.5.2 Fraction of dust that may have been contaminated

As detailed in section 4.1 it was cautiously assumed that the contamination was present as a 1 m diameter patch. If a room measured 3 m by 3 m then the patch would be just under 10% of the floor area. It was therefore assumed that 10% of dust in a room was contaminated.

In addition some of the dust within the air would originate from the hotspot. A cautious approach was used to estimate the potential amount of dust that could have originated from such a hotspot. Taking the hotspot to be represented by a circular patch of diameter 0.1 m, the area of the floor or wall covered by the hotspot would be approximately 0.008 m^2. Again, assuming a total area of 9 m^2, the hotspot would represent about 0.1% of the area. This percentage was used in the assessment.

For a worker drilling into a wall, the fraction of contaminated airborne dust would be related to the fraction of drilled wall material that was contaminated. Assuming only the first 2 mm of the wall material was contaminated but that drilling was to a depth of 20 mm, then only 10% of the dust created by drilling would be contaminated. Insufficient information is available to refine this further but it was considered that representing the contaminated dust as being approximately 10% of the total dust was more realistic than assuming that all of the dust inhaled was contaminated.

5.5.3 Fraction of contamination that may have been fixed to surfaces

If most of the radionuclides were adhering strongly to the floor or walls, then little would be available for inhalation or ingestion. It is very likely that by the 1950s any remaining contamination would have been fixed to surfaces as otherwise it would have been removed by cleaning, movement of people, furniture etc over time. However, as there was little information available on the amount of activity fixed to surfaces, it was cautiously assumed that none of the contamination was fixed and that all activity could be resuspended or transferred onto hands, food and other objects. This is unlikely to reflect the real situation and will lead to an overestimation of the dose.

5.5.4 Dose coefficients for intakes

The dose coefficients used in this assessment were those published by the ICRP in publication 72 (ICRP, 1996) supplemented with organ specific equivalent dose coefficients published on CD ROM (ICRP, 2001). The models used to derive the dose coefficients are referenced in the ICRP publications. These sources are the UK recommended reference for dose coefficients for members of the public. The report giving background material on the nature of radioactive contamination (Oatway et al, 2009) gives further information on these dose coefficients.

6 HOW THE RISKS WERE ASSESSED

Research into the risks of radiation-induced cancer has been going on for many years. The risk of around 12 specific cancer types has been found to be raised by radiation exposure and dose-response models for the estimation of these risks in different populations have been developed by a number of national and international organisations.

Models for solid cancers (those cancers apart from leukaemia and other haematological cancers) have a linear relationship between dose and risk which assumes that the excess risk increases in proportion to the radiation dose. For leukaemia a linear quadratic dose response relationship has been found to be better to allow for the finding that the slope of the dose response relationship is greater at higher doses than at lower doses. These dose response relationships vary between specific cancer types and may also vary with other individual level factors such as sex or age at exposure.

The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) recently published a new set of radiation risk models based mainly on updated data from a study of the survivors of the atomic bombings at Hiroshima and Nagasaki in 1945 (UNSCEAR, 2008). These models were used in this risk analysis. They are relative risk models in which the excess risk due to the radiation exposure is a product of a dose related function multiplied by the underlying baseline risk of the specific cancer in question. This means that for a constant dose the radiation induced cancer risk will vary if the underlying risk also varies.

Research has also shown that the risk from an exposure is not expressed immediately following the exposure but a number of years later. The best estimates for this 'lag' period are currently ten years for solid cancers and two years for leukaemia. Further, once the period of raised risk has started it can continue for many years. The current best estimate for this 'expression' period is 40 years for leukaemia and to the end of the individual's life for solid cancers. Additionally, the raised risk is not constant over the expression period but varies with time since exposure. Thus it can be seen that the relationship between cancer risk and radiation exposure is complex.

To quantify the radiation risk to a specific population or individual, the measures used to quantify risk must be selected with care. For this risk assessment, account must be taken of the complexity of the pattern of raised cancer risk from an extended period of radiation exposure. For example, because of the lag period and the extended expression period, the risk of solid cancer from an exposure is less for a person exposed at age 70 compared to the risk for someone exposed at age 20, as the 70 year old might succumb to some other cause of death before any risk associated with the radiation could be expressed, while the 20 year old has many years of life expectancy left in which the radiation risk could be expressed.

The measure used in this risk assessment to estimate lifetime risk is the 'Risk of Exposure Induced Death' (REID). This quantity is the lifetime risk that an individual will die from a cancer (of the type in question) that has been caused by that person's radiation exposure (Thomas et al, 1992). It is presented in this assessment as a percentage.

This measure is widely used in radiation epidemiology to measure lifetime risk of radiation induced cancer. It is appropriate for this risk assessment as it takes account of both radiation risks over the whole of a person's life and risks from competing causes of death. While other lifetime risk measures such as 'excess lifetime risk' have sometimes been considered in risk assessments, because the doses involved in this risk assessment are small, there will only be very small differences between these measures and REID. Risk measures that are calculated at a specific age following first exposure such as attributable risk are not appropriate as they do not adequately take account of the fact that the risk extends over a long period of time or of risks from competing causes of death

For the calculations in this report, the REID measure can be thought of as a sum of risk values over the years from the age at first exposure to an age of 100 years. In an individual year this risk value is derived as the probability that the subject survives to that age multiplied by the difference in the probability of death (from the cancer in question) between the exposed person and a similar but unexposed person.

In addition to the REID measure, other supplementary risk measures were calculated. Firstly, the baseline risk was estimated for a similar but unexposed individual drawn from the same population. This quantifies, in the same units as the REID (ie, as a percentage), the underlying level of risk of dying from the cancer in question in the absence of additional radiation exposure. Secondly, the baseline life expectancy was included. This is the predicted life expectancy of a person of the same age at first exposure and sex and drawn from the same population as the subject for which the

REID was calculated. Thirdly, the loss of life expectancy if death occurs was calculated. This measures the predicted years of life a person would lose if they die from a radiation induced cancer.

Risks were calculated separately for each type of cancer of interest, based on a hypothetical male (typical of the England and Wales population) who was exposed from age 20 for a period of 40 years. Lifetime risks for women exposed in the same way would be slightly higher for some cancers and slightly lower for others, but overall the risks would be very similar. The population and cancer mortality data used in the calculations were obtained from the Office for National Statistics (2007). The risk values calculated for this person represent an upper limit for the male working population as anyone who started work at a later age would have less time for any risk to be expressed.

Calculations were performed based on exposures in two different calendar periods: one for the assessment of past exposures starting in 1950 and the other for the assessment of current and future exposures nominally assumed to be from 2000. Both the underlying overall mortality rate and the mortality rates for specific types of cancer varied over this 50 year period. This would affect the derived radiation induced cancer risk, even if the doses received over the 40 years of exposure were the same in both exposure scenarios. Underlying specific cancer death rates and the overall age specific mortality rate for England and Wales were obtained from the Office for National Statistics (ONS), 1950 to 1999. For all subsequent years the 1999 rates were used (Office for National Statistics, 2007).

These models calculate risk based on annual doses. For the purposes of this assessment, the estimated committed effective dose to 50 years from each annual intake was assumed to be expressed in the year in which the exposure occurred. While this assumption is not strictly accurate, it does represent a ‘worst case scenario’ for the actual dose distribution and so provides an upper bound on the variation in the radiation risk related to this factor.

7 RESULTS

The following sections give a summary of the results, with the detailed results being given in APPENDIX F. It should be stressed that the results are for a hypothetical individual and are not specific to a particular person. The results are given to a maximum of three significant figures but it should be noted that this is for comparative purposes only and does not indicate this degree of precision.

7.1 Doses from past exposures

Table 11 and Table 12 give the committed effective and organ equivalent doses summed for the years 1950 to 1989, for the whole chain and base cases respectively. All of the doses in both tables include the contribution from both the widespread

contamination and from the hotspot of lead-210. The equivalent dose is multiplied by the appropriate tissue weighting factor and then the doses for all the tissues and organs of the body are summed to give the effective dose (see section A3.3 for more detail). For information, the tissue weighting factors (taken from (ICRP, 1991)) are also given in Table 11. Effective doses were calculated and are presented in this section to give the reader an understanding of the dose to the whole body. However it should be noted that effective doses were not used in the calculation of risks but rather risks to the specific organs, such as the lungs, brain and pancreas were calculated.

Table 11 Committed effective and organ equivalent doses for hypothetical office-based and maintenance workers, assuming the whole chain source term summed for exposures from 1950 to 1989

Organ	Tissue weighting factor (ICRP, 1991)	Office-based university worker, mSv	Maintenance worker, mSv
Bone surface	0.01	270	370
Lungs	0.12	300	85
Liver	0.05	90	120
Red bone marrow	0.12	65	70
Pancreas	See footnote	31	8
Brain	See footnote	31	8
Whole body (effective)	Not applicable	75	37

For the purposes of calculation, the brain and pancreas are two of a number of organs which make up the Remainder tissues and organs which have a combined weighting factor of 0.05 (ICRP, 1991)

Table 12 Committed effective and organ equivalent doses for hypothetical office-based and maintenance workers, assuming the base case source term summed for exposures from 1950 to 1989

Organ	Tissue weighting factor (ICRP, 1991)	Office-based university worker, mSv	Maintenance worker, mSv
Bone surface	0.01	240	360
Lungs	0.12	300	82
Liver	0.05	87	120
Red bone marrow	0.12	61	70
Pancreas	See footnote	30	8
Brain	See footnote	30	8
Whole body (effective)	Not applicable	72	37

For the purposes of calculation, the brain and pancreas are two of a number of organs which make up the Remainder tissues and organs which have a combined weighting factor of 0.05 (ICRP, 1991)

For the office-based worker, the organs/tissues estimated to have received the highest doses were the lungs and bone surface with 300 mSv and 270 mSv, respectively for a 40 year period for the whole chain case. For the maintenance workers, the organs/tissues estimated to have received the highest doses were the bone surface and the liver with 370 mSv and 120 mSv respectively for a 40 year period for the whole chain case. For both groups the estimated doses to the pancreas and brain were an order of magnitude lower than these doses.

Estimates of effective doses were higher for office-based university staff than for the maintenance workers. This was unsurprising given that the office-based staff have the highest assumed occupancy of the buildings. The most significant exposure pathways for office-based university staff were estimated to be external irradiation and inhalation of radon-222 (see Table 13). The important radionuclides for external irradiation were bismuth-214 and lead-214 which are both progeny of radium-226. It should be noted that there was variation in the contributions for individual radionuclides and the importance of exposure pathways over time. For example, the ingestion of contaminated dust contributed significantly more to the total dose in 1950 than in 1989 (about 25% to 10%, respectively).

Table 13 Breakdown of the summed effective dose (1950 to 1989) mSv by pathway assuming whole chain source term

Pathway	Office-based university worker, mSv	Maintenance worker, mSv
Inhalation of radon-222	22	2
Inhalation of radon-220	10	1
Inhalation of contaminated dust	2	9
Inadvertent ingestion of contaminated dust	12	22
External exposure	29	3
Total	75	37

As can be seen from Table 13, the ingestion and inhalation of contaminated dust resulted in higher doses for the maintenance worker than for office-based workers. For the maintenance worker the inhalation of dust was estimated to contribute about 20% to 25% of the total dose over the period 1950 to 1989 while inadvertent ingestion contributed about 50% to 65%.

For the lungs, inhalation of radon-222 and radon-220 contributed about 90% of the dose for office-based workers for both source terms and at all times considered. For maintenance workers, doses to the lungs were about a third lower as they spent less time in the office with consequent reductions in doses from radon-222 and radon-220. However, inhalation of contaminated dust was estimated to be of greater importance, contributing 65% of the lung dose in 1950 and 40% in 1989 for both source terms.

For bone surfaces, for both groups and source terms, ingestion of contaminated dust was estimated to be the most significant pathway contributing about 70% to 80% of the dose. Most of this dose was estimated to be due to lead-210. The percentage contribution of this pathway was not calculated to vary much over time but the dose to the bone surfaces for both groups and contamination scenarios roughly halved from 1950 to 1989.

For doses to the liver, the ingestion of contaminated dust was estimated to be a significant pathway for office-based and maintenance workers. For office-based staff the contribution from contaminated dust was about 60% of the dose in 1950 but by 1989 the

external dose pathways contributed about 50% of the dose for both source terms. For maintenance workers, who were estimated to have higher doses to the liver than office-based workers, the ingestion of contaminated dust was estimated to be the most important exposure route at 80% of the dose for both source terms. Lead-210 and polonium-210 account for most of this dose. The dose to the liver was calculated to drop by over half from 1950 to 1989.

Doses to both the pancreas and brain for office-based workers are mainly due to exposure to external radiation, which was estimated to have contributed about 90% of the dose in 1950 and about 95% in 1989 for both source terms. The radionuclides which contributed significantly to the estimates of external irradiation are lead-214 and bismuth-214, both progeny of radium-226. For the maintenance workers, the relative importance of the exposure pathways varied over time. In 1950 the ingestion of contaminated dust was estimated to be the most important exposure route, contributing over 60% of the dose, but in 1989, nearly 60% of the dose was estimated to come from external irradiation.

7.2 Doses from current and future exposures

Table 14 gives the total committed effective and organ equivalent doses based on current estimates of exposures and assuming that these levels remain the same for the next 40 years. These doses are lower than those estimated from past exposures due to the lower levels of contamination found following remediation of the buildings. However, since a number of cautious assumptions were made in the assessment, mainly the inclusion of natural background in some of the estimated exposures, the actual doses to current and future occupants resulting from residual contamination will be much lower than the values given in this section.

For the office-based workers, the lungs were estimated to receive by far the highest organ dose, 280 mSv, with most of the dose due to the inhalation of radon-222 (about 65%) and the inhalation of radon-220 (about 30%). Doses to all the other organs/tissues, including the brain and pancreas, were estimated to be at least ten times lower than to the lung. For the maintenance workers the bone surface was estimated to receive the highest dose, 49 mSv, followed by the lung at 33 mSv. The doses to the brain and pancreas were estimated to be at least ten times lower than to the bone surface.

Table 14 Committed effective and organ equivalent doses for an office-based worker and a maintenance worker based on estimated current contamination levels for 40 years

Organ	Tissue weighting factor (ICRP, 1991)	Office-based university worker, mSv	Maintenance worker, mSv
Bone surface	0.01	20	49
Lungs	0.12	280	33
Liver	0.05	17	9
Red bone marrow	0.12	16	6
Pancreas	See footnote	16	2
Brain	See footnote	16	2
Whole body (effective)	Not applicable	48	7

For the purposes of calculation the brain and pancreas are two of a number of organs which make up the Remainder tissues and organs which have a combined weighting factor of 0.05 (ICRP, 1991)

It should be noted that the Ionising Radiations Regulations 1999 (IRR99) ((TSO, 2000) specifies annual limits on committed effective dose for workers and members of the public. For workers it is 20 mSv y^{-1} and for members of the public it is 1 mSv y^{-1}. The guidance to these regulations states that 'for the assessment of compliance with the dose limits relating to members of the public, realistic estimates should be made of the average effective dose (and where relevant equivalent dose) to representative members of the appropriate reference group'. In addition it says that 'exposures received as a result of natural background radiation at normal levels are not considered in determining compliance with the dose limits'. The intention of this dose assessment was to represent the highest likely dose to be received, ie, it is not a realistic estimate of doses. The assessment of current and future doses also includes some contribution from natural background radiation. The highest estimated annual effective dose was for office workers in 1950 at around 2 mSv. This compares with the annual dose limit of 1 mSv for members of the public (TSO, 2000) but it should be noted that the dose limits have reduced over time. For example in the previous set of regulations (HMSO, 1985) the annual dose limit was 5 mSv. Although it is of interest to compare the doses estimated with the doses limits given for workers and member of the public the dose limits are not directly applicable for the reasons discussed above.

7.3 Risks from past exposures

Table 15 shows estimated risks for office-based staff from the estimated past exposures.

Table 15 Risks of cancer associated with exposures to ionising radiation based on hypothetical office-based workers employed from 1950 to 1989 assuming whole chain case

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.94	0.005	13.1
Brain	0.51	0.003	16.5
Liver	0.15	0.005	13.0
Lung	7.08	0.597	12.3
Leukaemia	0.61	0.022	18.2

The largest risk of cancer mortality in this hypothetical scenario was estimated to be from lung tumours, with a lifetime risk of exposure-induced death (REID) of about 0.6%. In other words, approximately 60 in every 10,000 people so exposed would die from a radiation-induced lung cancer. This compares with a baseline risk of dying from lung cancer in the general population of about 7% (700 per 10,000 deaths). The average years of life lost for an individual who died of lung cancer as a consequence of his exposure would be approximately 12 years.

Risks from irradiation of other organs are much lower. For pancreatic cancer, the REID was estimated as 0.004% (less than 1 in every 10,000 deaths), corresponding to slightly less than half of one percent of the baseline risk which is 94 per 10,000 deaths.

For brain cancer, the REID was estimated as 0.003% (less than 1 in every 10,000 deaths). This is slightly over half of one percent of the baseline risk of dying from brain cancer which is 51 per 10,000 deaths.

Table 16 summarises the risks of cancer mortality for the maintenance workers assuming the whole chain source term. Risks of leukaemia and liver cancer are slightly higher than for the office-based workers, but those for lung, brain and pancreatic cancer are all lower.

Table 16 Risks of cancer associated with exposures to ionising radiation based on hypothetical maintenance workers employed from 1950 to 1989 assuming whole chain case

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.94	0.001	13.4
Brain	0.51	0.0008	16.8
Liver	0.15	0.006	13.1
Lung	7.08	0.175	12.5
Leukaemia	0.61	0.023	19.2

7.4 Risks from current and future exposures

Table 17 and Table 18 give the estimated risks of radiation-induced health effects from current and future exposures for office-based and maintenance workers respectively. It can be seen that these risks are lower than the risks from past exposures.

Table 17 Risks of cancer associated with exposures to ionising radiation based on hypothetical office-based workers based on estimated current contamination levels for 40 years

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.97	0.002	12.6
Brain	0.51	0.001	16.2
Liver	0.16	0.001	12.9
Lung	6.49	0.535	11.3
Leukaemia	0.64	0.006	16.7

Table 18 Risks of cancer associated with exposures to ionising radiation based on hypothetical maintenance workers based on estimated current contamination levels for 40 years

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.97	0.0002	12.6
Brain	0.51	0.0001	16.2
Liver	0.16	0.0005	12.9
Lung	6.49	0.0624	11.3
Leukaemia	0.64	0.0020	16.7

The largest estimated risk of cancer mortality in this scenario was from lung tumours, with a lifetime risk of exposure-induced death (REID) of 0.5% for office-based workers and less than 0.1% for maintenance workers. The risks of exposure-induced death are much lower for the other types of cancers, typically by a factor of one hundred.

8 DISCUSSION

There are uncertainties in all assessments of dose and risk because every factor used in the assessment will not be known precisely. In this assessment a major uncertainty is the estimation of contamination levels to which people were exposed from the 1950s in the absence of monitoring records prior to 1999. The uncertainties associated with the other factors are likely to be minor in comparison. The following section discusses this uncertainty and the cautious assumptions made in the assessment to try to ensure that the assessed doses and risks were not underestimated.

- It was assumed that the hypothetical office-based worker was sitting directly above a 1 m diameter patch and 0.3 m from a spot of contamination on the wall for 2000 hours per year. Information from the monitoring reports indicates that the majority of the contamination was in small, discrete patches. If it is more realistically assumed that the office-based worker was only exposed to a few small contamination patches then the assessed doses would be reduced significantly.

- It was assumed that exposures occurred every year for 40 years. In practice the longest occupancy is likely to have been less than this.

- The highest reported contamination values have been assumed to apply to all of the decay chain being considered (except for thorium-230 and actinium-227 for the base case, as discussed in section 4.1). For example, in all of the rooms except one, which was probably used for lead-210 experiments, measurements of radium-226 progeny such as lead-214, bismuth-214 and lead-210 were found to have lower values than those for radium-226; probably due to the escape of radon-222 gas. However, when estimating surface contamination levels it was assumed that the progeny were present at radium-226 contamination levels. A separate but cautious assumption was made when estimating radiation doses from the inhalation of radon. An emanation fraction of 50% was assumed which means that 50% of the radon-222 gas was assumed to have escaped from the surface.

- It was assumed that none of the contamination was fixed and was therefore all available for resuspension. Experience from other similar situations indicates that any contamination present from 1950 onwards would be fixed, ie, any non-fixed contamination would have been removed by cleaning, general movement etc. Assuming that only 10% of the 1 m patch was still easily removed the estimated committed effective doses for 1950 to 1989 for the office-based workers would reduce by about 10% and for the maintenance worker by up to 25%.

- Risks were calculated using annual doses beginning in 1950 for a 20 y old male. Exposures occurring at older ages carry smaller risks because there is less time for the expression of the risk. In addition, the annual doses used were committed organ doses which are integrated over a 50 year period. For long-lived radionuclides that are retained in body organs over many years, dose is received over many years and assigning dose to the year of intake will overestimate risks.

While there are good reasons to believe that doses have been overestimated, it is possible that if remediation had been carried out at some earlier time contamination levels in the past could have been higher than indicated by recent monitoring. Information provided by the University (Peters, 2008) did not indicate that any substantial building work or remediation was carried out prior to 1999, although there was some documentation (see section C2.13) which surmised that some sanding of the floors may have occurred and anecdotal information that some of the rooms had been replastered and repainted (Private correspondence to Professor Coggon, 2008).

Many different scenarios could be postulated but given the available information it is the judgement of the authors based on experience from previous assessments that the assessed doses are not likely to be more than a factor of five below the actual doses and certainly would not be orders of magnitude lower. In reality the assessed doses are more likely to overestimate actual doses, as discussed above.

The assessments have been done for adults. In the Coupland 1 Psychology Annex there is an observation room for work with children. Although children may have higher inadvertent ingestion rates, dose per unit intake values and REID values than adults, they will have spent significantly less time in the Building than adults (a few hours as opposed to 2000 hours per year). Therefore, it can be assumed that any dose and associated risk they received will have been much lower than those estimated for adults.

Other work has been considered to see if it provides information on the exposure levels received by staff who worked in the Rutherford Building and Coupland 1 Psychology Annex. HPA was asked by HM Coroner, Manchester, to analyse post-mortem samples from two former University of Manchester staff. Given the nature of the contamination, the samples (bone, liver, lung and kidney) are being analysed for isotopes of uranium and thorium, radium-226, lead-210 and polonium-210. These radionuclides occur naturally in the environment and are present in small quantities in everyone. Results of the samples indicate levels that are consistent with those found in the general population.

In addition, the enamel from a tooth from a former University of Manchester staff member, who worked in the Rutherford Building for many years, was analysed for radiation exposure using electron paramagnetic resonance. The dose measured in this tooth was of the order of that typically measured in teeth from average members of the population (Fattibene, 2009).

Items belonging to a former staff member of the University, which had been in the Rutherford Building for several decades, were monitored for the presence of radioactive contamination. No radioactive contamination, either from radium-226 or other radioactive substances, was detected during the survey (Belford and Shaw, 2009).

These additional factors provide some evidence in support of the assumption that the assessed doses for a hypothetical individual will not have underestimated the actual doses received by University of Manchester staff.

In order to put the results into context, it is important to point out that all of the radionuclides used by Professor Rutherford and his colleagues (uranium-238, thorium-232 and uranium-235 decay chains) are present in the soils and rocks around us. Everyone receives a dose from exposure to this radiation. The following paragraphs discuss the doses that are received from these exposures to provide some context to the doses which have been estimated in this study.

In the UK, the average activity concentration of members of the uranium-238 decay chain in soil is 40 Bq kg^{-1} while that for the thorium-232 decay chain is 25 Bq kg^{-1} (UNSCEAR, 2000). However, there are wide ranges in these activity concentrations in soil across the UK with the range for uranium-238 being between 2 and 330 Bq kg^{-1} and

between 1 and 180 Bq kg^{-1} for thorium-232 (UNSCEAR, 2000). Table 19 gives some examples of the natural levels of the uranium and thorium decay series.

Since building materials are extracted from the earth, they too are mildly radioactive. Concentrations vary but concrete might be expected to contain around 40 Bq kg^{-1} activity concentration of radium-226, clay (red bricks) around 50 Bq kg^{-1}, and natural building stones around 60 Bq kg^{-1} (European Commission, 1999). Studies conducted in the UK in the 1980s suggested that the average gamma-ray dose rate from simply living in a house is around 0.3 mSv y^{-1} and the average gamma-ray dose rate from living in a house in the Greater Manchester area is slightly higher at around 0.4 mSv y^{-1} (Wrixon et al, 1988).

As these naturally occurring radionuclides are present in soil, they are also taken up by plants and ingested by people. The most significant contributor to the dose from the ingestion of these naturally occurring radionuclides in foods is from members of the uranium decay chains, notably lead-210 and polonium-210. The average annual dose to a member of the UK population from the presence of uranium and its decay products in food is approximately 0.07 mSv (Watson et al, 2005).

Tobacco grown for the manufacture of cigarettes (mainly *Nicotiana tabacum*) also takes up naturally occurring radionuclides. One study estimated that smoking around 30 cigarettes daily gives an effective dose of around 0.25 mSv y^{-1} (Papastefanou, 2009).

Table 19 Some examples of natural levels of members of the uranium and thorium decay series

Material	Levels	
Soil (UK average)	40 Bq kg^{-1} uranium-238 decay chain	25 Bq kg^{-1} thorium-232 decay chain
(UK range)	2 – 330 Bq kg^{-1} uranium-238 decay chain	1 – 180 Bq kg^{-1} thorium-232 decay chain
Building materials - Concrete	40 Bq kg^{-1} radium-226 (average)	30 Bq kg^{-1} thorium-232 (average)
	240 Bq kg^{-1} radium-226 (maximum)	190 Bq kg^{-1} thorium-232 (maximum)
Clay (red brick)	50 Bq kg^{-1} radium-226 (average)	50 Bq kg^{-1} thorium-232 (average)
	200 Bq kg^{-1} radium-226 (maximum)	200 Bq kg^{-1} thorium-232 (maximum)
Natural building stones	60 Bq kg^{-1} radium-226 (average)	60 Bq kg^{-1} thorium-232 (average)
	500 Bq kg^{-1} radium-226 (maximum)	310 Bq kg^{-1} thorium-232 (maximum)
Mussels	42 Bq kg^{-1} radium isotopes	
Brazil nuts	30 Bq kg^{-1} radium isotopes	
Radon-222 (UK average outdoor)	4 Bq m^{-3}	
(UK average indoor)	20 Bq m^{-3}	
(UK highest - indoor)	17 000 Bq m^{-3}	

The average annual dose to the UK population from all natural sources is about 2.2 mSv (Watson et al, 2005). A summary of the annual average dose to a member of the UK population is presented in Table 20. Inhalation of radon decay products is the most significant contributor to the average dose and is most variable. Terrestrial gamma radiation results from the radionuclides in the earth which emit penetrating

gamma rays as discussed earlier. Other natural sources of exposure include that from other naturally occurring radionuclides such as potassium-40, which contribute significantly to the dose from the ingestion of food, and that from cosmic radiation. Cosmic radiation originates from outside the atmosphere of the Earth and is composed of energetic particles. These particles pass through the atmosphere and irradiate the entire surface of the planet.

Table 20 Summary of doses to the UK adult population from natural sources (from Watson et al, 2005)

	Average annual UK dose (μSv)	Range (μSv)
Cosmic radiation	330[a]	200 – 400[b]
Terrestrial gamma radiation	350	100 – 1000
Internal radionuclides	250	100 – 1000
Radon[c] (radon-222)	1,200	300 – 100,000
Thoron[c] (radon-220)	100	50 – 500
Total	2,230	1,000 – 100,000

(a) Including an additional 30 μSv from air travel. It should be noted that not all of the population is exposed to this additional source.

(b) Range does not include air travel.

(c) Including decay products.

9 RELEVANCE OF THIS STUDY TO FUTURE REMEDIATION WORK

The doses and risks from exposure to current and future contamination levels were assessed cautiously by including some contribution from natural background. These doses were found to be relatively low and most of the dose is attributable to natural background rather than any residual contamination. However, it is recommended that a risk assessment is carried out before any future work which involves invasive disturbance of the floor or wall materials, such as removing floorboards or walls. Such a risk assessment would require additional radiological measurements which would need to include measurements on the floorboards and inside the floor structure if floorboards are to be removed or ceilings to be taken down. The purpose of such a risk assessment should be to identify what control measures are appropriate to protect workers and others involved in the work, including whether the work should be subject to the requirements of the Ionising Radiations Regulations 1999 (TSO, 2000). The above requirement for a risk assessment should be applied to work affecting the structure of the building, although HPA cannot provide detailed advice at this stage as the risk assessment required will depend on the specifics of the situation.

10 ACKNOWLEDGMENTS

The authors would like to acknowledge valuable contributions from a number of individuals. This work would not have been possible in the available timescale without the large amount of research carried out by Neil Todd and John Churcher. Advice and information from HPA staff is also gratefully acknowledged, in particular from Daryl Dixon, Tracy Gooding, Jan McClure, Jon Miles and Colin Muirhead. Also, discussions have taken place throughout with Professor Coggon and this has also been extremely beneficial. An important aspect in this study was that the concerned staff from the University of Manchester and their families were kept informed and consulted about the work and were given the opportunity to question what was being done. In addition comments received on the provisional report (Jones et al, 2009) were used to refine this report. All of these inputs have improved the study.

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APPENDIX A Basic radiation protection concepts

The first part of the project involved HPA producing a report that described background material on the nature of radioactive contamination at the University of Manchester and the possible health risks (Oatway et al, 2009). Although reference to that report should be made for detailed information in these areas, some material from Oatway et al has been repeated in the following sections to help the reader of this report.

A1 CHARACTERISTICS OF IONISING RADIATION

The energy associated with ionising radiation is released from a radionuclide in one of three main forms: as an alpha particle, as a beta particle or as gamma rays.

Alpha particles are heavy and charged and lose energy rapidly within matter. This means that alpha particles are not particularly penetrating and are stopped by a sheet of paper or the outer layer of skin on the body. If the alpha particle was emitted by a radionuclide outside of the body, then the radiation is unable to irradiate internal organs. Alpha particle radiation is therefore only significant if emitted by radionuclides that are located within the body. As a consequence of depositing its energy within a relatively short distance, travelling only a few cell widths, an alpha particle will deposit most of its energy within a single organ or tissue when compared to other forms of radiation, causing more localised damage.

Beta particles are stopped by a thin sheet of metal and, except for the most energetic particles, are also stopped by the skin. This means that a beta particle emitted outside the body will generally not deposit energy in internal organs but may cause damage to skin. A beta particle, because it is less charged than an alpha particle, may pass through tissues within an organ before it loses all of its energy. A beta particle emitted within the body may therefore cause damage to cells throughout an organ or even in adjacent organs. However, the amount of damage caused per unit distance travelled by a beta particle would be less when compared to that caused by the alpha particle.

Gamma rays are effectively stopped by a thick layer of high density material such as lead. However, this attenuation by matter is strongly dependent on their energy so higher energy gamma rays have a significant probability of passing right through the body. Consequently, they can potentially irradiate any organ regardless of whether they were emitted internally or externally to the body. In general, they may be regarded as being more penetrating than alpha or beta particles, although this is again dependent on the energies of the different types of radiation. Energy is, in general, deposited by secondary electrons, and the dose deposition is equivalent to that of a beta particle, but less intense than that of an alpha particle. Figure A.1 shows the differences in penetrating power.

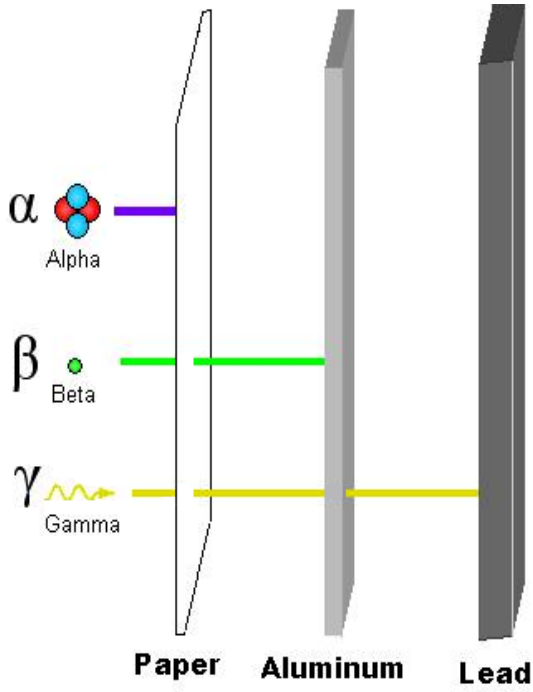

Figure A.1 The penetrating power of ionising radiation

A2 UNITS OF RADIOACTIVITY

The rate at which spontaneous transformations occur in a given amount of a radioactive material (radioactive decay) is known as its activity. Activity is expressed in a unit called **Becquerel** (symbol **Bq**), where 1 Bq is equal to one transformation per second. Multiples of the Bq are often used to describe the number of transformations that a radioactive material will undergo. For example, a mega-Becquerel (**MBq**) is the term used to describe 1 million transformations per second.

The time taken for the activity of a radioactive material to fall to half of its original value is termed half-life. Each radionuclide has its own specific half-life, ranging from fractions of a second to many millions of years. An illustration of radioactive decay is given in Figure A.2 for a radioactive material that has a half-life of 1 hour. From this figure it is evident that the activity decreases by half every hour.

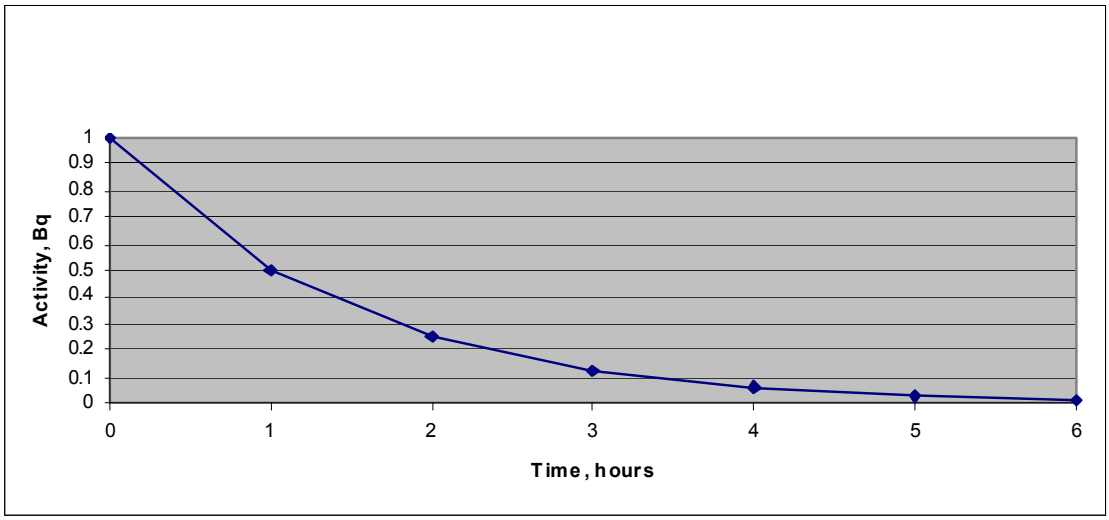

Figure A.2 Radioactive decay curve for a radioactive material with a half-life of 1 hour

A3 RADIOACTIVE DECAY CHAINS

For some radionuclides, radioactive decay does not form a stable atom. The new atom formed may also be radioactive and will undergo radioactive decay. This sequence of radionuclides forms what is called a radioactive decay series or chain; the first radionuclide is called the chain header or 'parent' radionuclide and is followed by a series of 'progeny' radionuclides. Table A.1 to Table A.3 and Figure A.3 to Figure A.5 show the naturally occurring decay series headed by uranium-238, thorium-232 and uranium-235. Within these decay chains are those radionuclides used in experimental work at the University of Manchester.

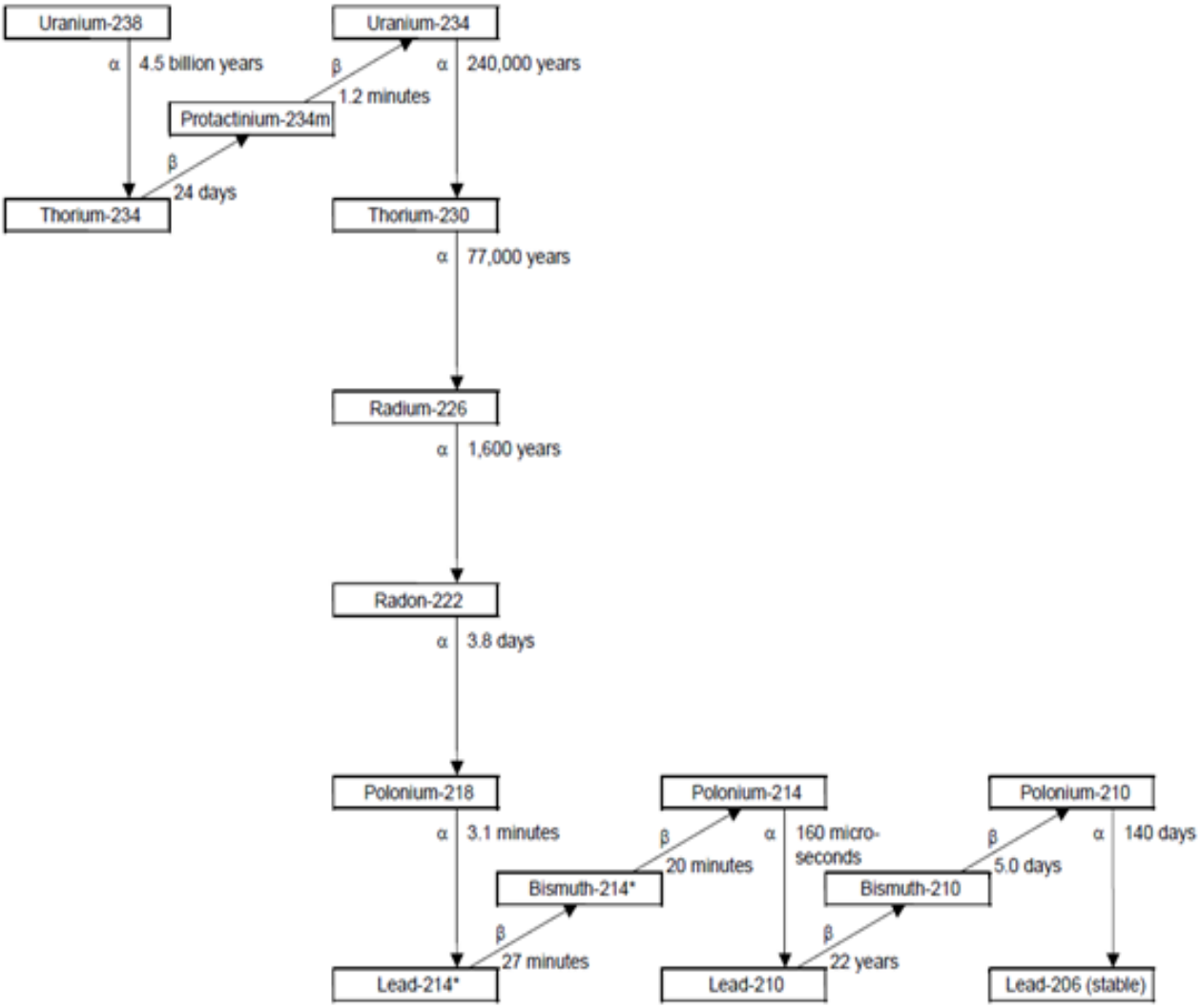

Figure A.3 The uranium-238 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown.

Table A.1 Radiological information about members of the uranium-238 decay chain

Radionuclide	Historical name	Half-life	Decay mode (MeV)[(a)] and intensity (%)[(b)]	Gamma-ray (keV) intensity (%)[(b)]	Product of decay
^{238}U	Uranium	4.5 10^{9} y	α 4.198 (79%) 4.151 (21%)		^{234}Th
^{234}Th	Uranium X1	24.1 d	β 0.199 (70%) 0.107 (19%) 0.106 (8%)	63.3 (5%) 92.4 (3%) 92.8 (3%)	^{234}Pa (0.2%) ^{234m}Pa (99.8%)
^{234}Pa	Uranium Z	6.7 h	β 0.472 (45%) 0.642 (19%) 0.413 (8%)	131.3 (18%) 946.0 (13%) 883.2 (10%)	^{234}U
^{234m}Pa	-	1.2 min	β 2.269 (98%)	1001.0 (1%)	^{234}U (99.87%) ^{234}Pa (0.13%)
^{234}U	Uranium two	2.5 10^{5} y	α 4.775 (71%) 4.722 (28%)		^{230}Th
^{230}Th	Ionium	7.5 10^{4} y	α 4.687 (76%) 4.621 (23%)		^{226}Ra
^{226}Ra	Radium	1. 6 10^{3} y	α 4.784 (94%) 4.601 (5%)	186.2 (4%)	^{222}Rn
^{222}Rn	Radon	3.8 d	α 5.490 (100%)		^{218}Po
^{218}Po	Radium A	3.1 min	α 6.002 (100%)		^{214}Pb (99.98%) ^{218}At (0.02%)
^{218}At	-	1.5 s	α 6.693 (90%) 6.653 (6%) 6.756 (4%)		^{214}Bi
^{214}Pb	Radium B	26.8 min	β 1.024 (6%) 0.729 (42%) 0.672 (49%)	351.9 (38%) 295.2 (19%) 242.0 (7%)	^{214}Bi
^{214}Bi	Radium C	19.9 min	α 5.516 (39%) 5.452 (54%) 5.273 (6%) β 3.272 (18%) 1.542 (18%) 1.507 (17%)	609.3 (46%) 1764.5 (15%) 1120.3 (15%)	^{214}Po
^{214}Po	Radium C'	1.6 10^{-4} s	α 7.687 (100%)		^{210}Pb
^{210}Pb	Radium D	22.3 y	α 3.720 (100%) β 0.017 (84%) 0.063 (16%)	46.5 (4%)	^{210}Bi
^{210}Bi	Radium E	5.0 d	α 4.656 (60%) 4.694 (40%) β 1.162 (100%)		^{210}Po
^{210}Po	Radium F	138.4 d	α 5.304 (100%)		^{206}Pb

(a) For beta particles, maximum energy of the particle is given

(b) Only the 3 highest intensity decays or emissions are shown. The intensity is rounded to nearest significant figure and is only shown if 1% or greater.

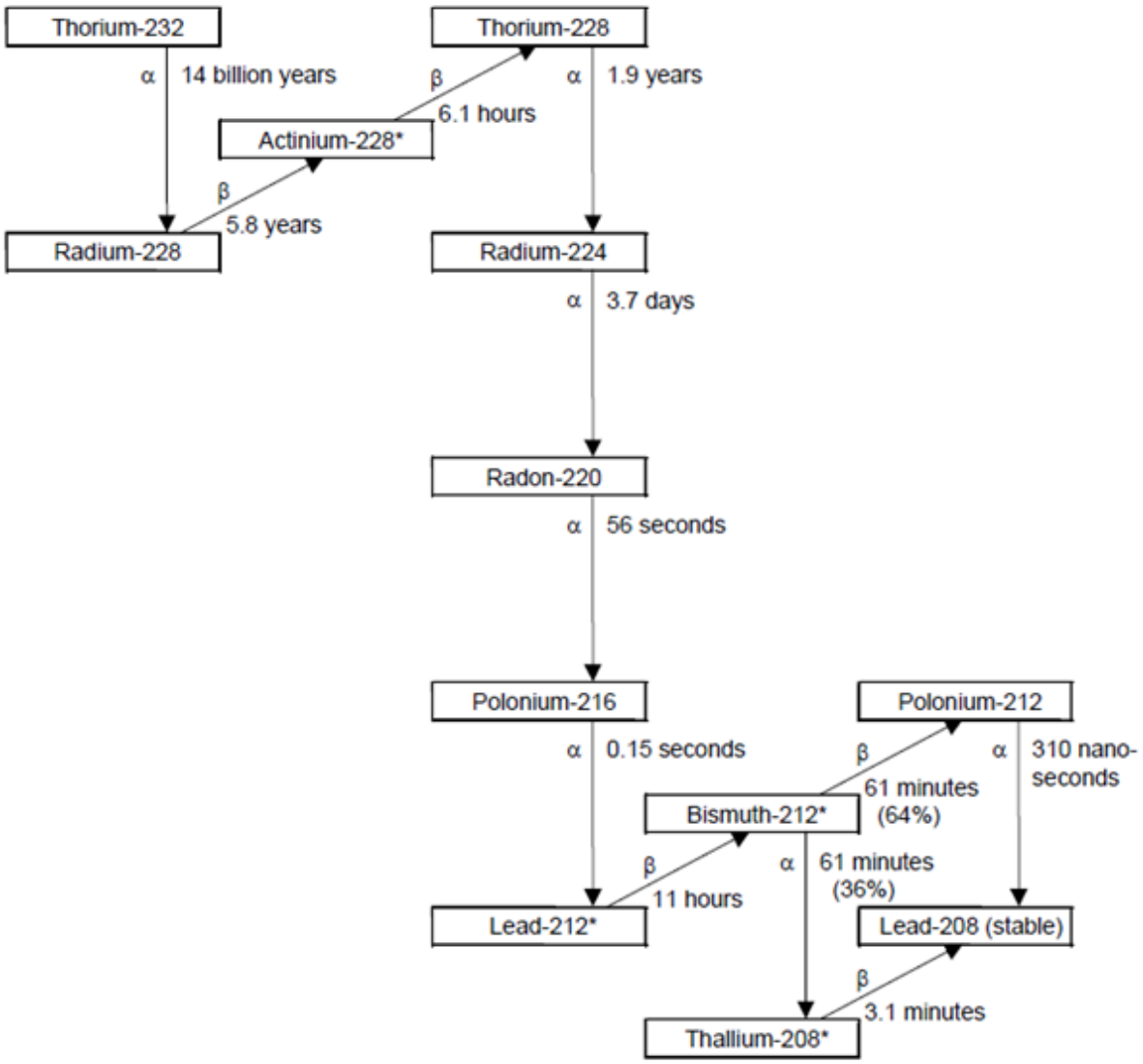

Figure A.4 The thorium-232 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown.

Table A.2 Radiological information about members of the thorium-232 decay chain

Radionuclide	Historical name	Half-life	Decay mode (MeV)[(a)] and intensity (%)[(b)]	Gamma-ray (keV) intensity (%)[(b)]	Product of decay
^{232}Th	Thorium	1.4 10^{10} y	α 4.012 (78%) 3.947 (22%)	63.8 (26%) 140.9 (2%)	^{228}Ra
^{228}Ra	Mesothorium 1	5.8 y	β 0.039 (40%) 0.013 (30%) 0.026 (20%)	13.5 (2%)	^{228}Ac
^{228}Ac	Mesothorium 2	6.2 h	β 1.158 (30%) 1.731 (12%) 2.069 (8%)	911.2 (26%) 969.0 (16%) 338.3 (11%)	^{228}Th
^{228}Th	Radiothorium	1.9 y	α 5.423 (72%) 5.340 (27%)	84.4 (1%)	^{224}Ra
^{224}Ra	Thorium X	3.7 d	α 5.685 (95%) 5.449 (5%)	241.0 (4%)	^{220}Rn
^{220}Rn	Thoron	55.6 s	α 6.288 (100%)		^{216}Po
^{216}Po	Thorium A	0.1 s	α 6.778 (100%)		^{212}Pb
^{212}Pb	Thorium B	10.6 h	β 0.335 (83%) 0.574 (12%) 0.159 (5%)	238.6 (43%) 300.1 (3%)	^{212}Bi
^{212}Bi	Thorium C	60.6 min	α 6.051 (70%) 6.090 (27%) 5.768 (2%) β 2.254 (55%) 1.527 (4%) 0.633 (2%)		^{212}Po (64.06%) ^{208}Tl (35.94%)
^{212}Po	Thorium C'	3 10^{-7} s	α 8.784 (100%)		^{208}Pb
^{208}Tl	Thorium C"	3.1 min	β 1.803 (49%) 1.293 (25%) 1.526 (22%)	2,614.5 (99%) 583.2 (85%) 510.8 (23%)	^{208}Pb

(a) For beta particles, maximum energy of the particle is given

(b) Only the 3 highest intensity decays or emissions are shown. The intensity is rounded to nearest significant figure and is only shown if 1% or greater.

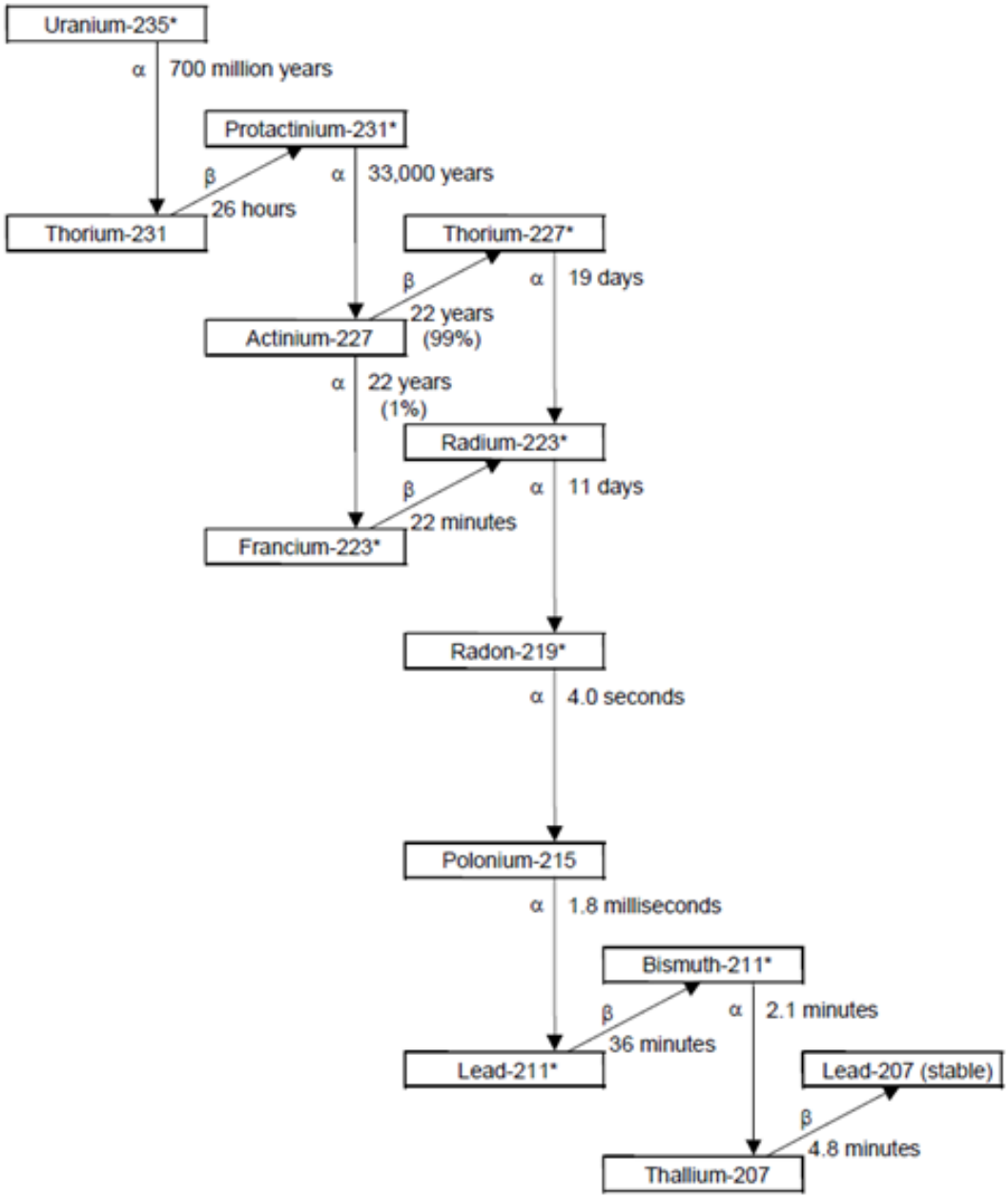

Figure A.5 The uranium-235 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown.

Table A.3 Radiological information about members of the uranium-235 decay chain

Radionuclide	Historical name	Half-life	Decay mode (MeV)[(a)] and intensity (%)[(b)]	Gamma-ray (keV) intensity (%)[(b)]	Product of decay
^{235}U	Actin Uranium	7.0 10^{8} y	α 4.398 (55%) 4.366 (17%) 4.215 (6%)	185.7 (57%) 143.8 (11%) 163.4 (5%)	^{231}Th
^{231}Th	Uranium Y	25.5 h	β 0.288 (37%) 0.305 (35%) 0.206 (13%)	25.6 (15%) 84.2 (7%)	^{231}Pa
^{231}Pa		3.3 10^{4} y	α 5.014 (25%) 4.951 (23%) 5.028 (20%)	27.4 (10%) 300.1 (2%) 302.7 (2%)	^{227}Ac
^{227}Ac	Actinium	21.8 y	α 4.953 (48%) 4.941 (40%) 4.873 (6%) β 0.020 (10%) 0.036 (35%) 0.045 (54%)		^{227}Th (98.62%) ^{223}Fr (1.38%)
^{227}Th	Radioactinium	18.7 d	α 6.038 (24%) 5.978 (24%) 5.757 (20%)	236.0 (12%) 50.1 (8%) 256.3 (7%)	^{223}Ra
^{223}Fr	Actinium K	21.8 min	α 5.340 (100%) β 1.099 (67%) 1.069 (16%) 0.914 (10%)	50.1 (36%) 79.7 (9%) 234.8 (3%)	^{223}Ra
^{223}Ra	Actinium X	11.4 d	α 5.716 (53%) 5.607 (26%) 5.747 (9%)	269.5 (14%) 154.2 (6%) 323.9 (4%)	^{219}Rn
^{219}Rn	Actinon	4.0 s	α 6.819 (79%) 6.553 (13%) 6.425 (8%)	271.2 (11%) 401.8 (6%)	^{215}Po
^{215}Po	Actinium A	1.8 10^{-3}s	α 7.386 (100%)		^{211}Pb
^{215}At		1 10^{-4} s	α 8.026 (100%)		^{211}Bi
^{211}Pb	Actinium B	36.1 min	β 1.372 (91%) 0.540 (6%) 967.2 (2%)	404.9 (4%) 832.0 (4%) 427.1 (2%)	^{211}Bi
^{211}Bi	Actinium C	2.1 min	α 6.623 (84%) 6.278 (16%)	351.1 (13%)	^{207}Tl (99.72%) ^{211}Po (0.28%)
^{211}Po	Actinium C'	0.5 s	α 7.450 (99%)		^{207}Pb
^{207}Tl	Actinium C"	4.8 min	β 1.423 (100%		^{207}Pb

(a) For beta particles, maximum energy of the particle is given

(b) Only the 3 highest intensity decays or emissions are shown. The intensity is rounded to nearest significant figure and is only shown if 1% or greater.

A3.1 Secular equilibrium

Secular equilibrium within a radionuclide decay chain is the situation in which the activities of progeny are the same as that of the parent radionuclide. This state is most rapidly reached when the progeny has a short half-life when compared with the half-life of the parent radionuclide. In the thorium and uranium decay chains, escape of radon gas can result in lower activities of radionuclides at lower positions in the chain; that is, equilibrium with the parent radionuclide cannot be reached.

A3.2 Branching ratios

When a radionuclide decays, it may decay to more than one radionuclide. The fraction of the number of atoms decaying to one of its progenies is known as its branching ratio. This concept is shown schematically in Figure A.6. Branching ratios were taken into account in this study.

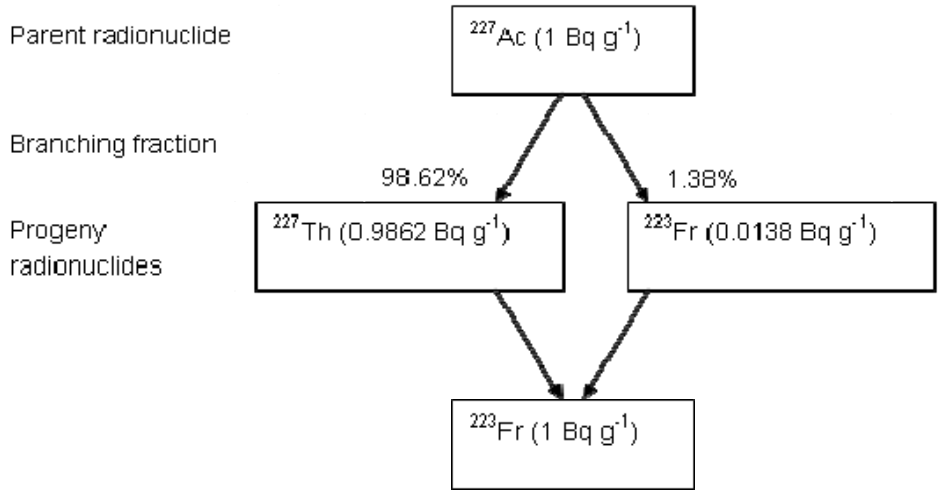

Figure A.6 Representation of decay chain branching and activity of radioactive progeny

A3.3 Dosimetric quantities

The fundamental dosimetric quantity in radiological protection is the **absorbed dose** which is the energy absorbed per unit mass. This quantity has the unit joules per kilogram (J kg^{-1}) and is given the name: gray (Gy). For radiation protection purposes, ICRP (1991, 2007) has devised two additional protection quantities, equivalent and effective dose, given the name, sievert (Sv).

Ionising radiations differ in the way in which they interact with biological materials such that equal absorbed doses (meaning equal amounts of energy deposited per unit mass) from different radiations do not necessarily have equal biological effects. For example, a dose of 1 Gy for alpha particles may be more harmful than 1 Gy from beta particles as the alpha particles, being slower and more heavily charged, lose energy much more

densely along their path resulting in more localised damage. In order to put all ionising radiations on an equal basis with regard to their potential to cause so-called "stochastic effects" (cancer and hereditary effects), the absorbed dose is multiplied by a factor, termed the radiation weighting factor, which accounts for the way a particular type of radiation deposits energy in a tissue. The absorbed dose multiplied by the radiation weighting factor results in a quantity termed the **equivalent dose**, which has the unit of sievert (Sv). For gamma rays and beta particles the radiation weighting factor used is 1, while for alpha particles the factor is set as 20 (ICRP, 1991)

To provide a single quantity for the control of exposures, equivalent doses to organs and tissues are summed after multiplying them by tissue weighting factors which take account of contributions to total risks of cancer and hereditary effects. This doubly weighted quantity is referred to as **effective dose**. This is summarised in Figure A.7.

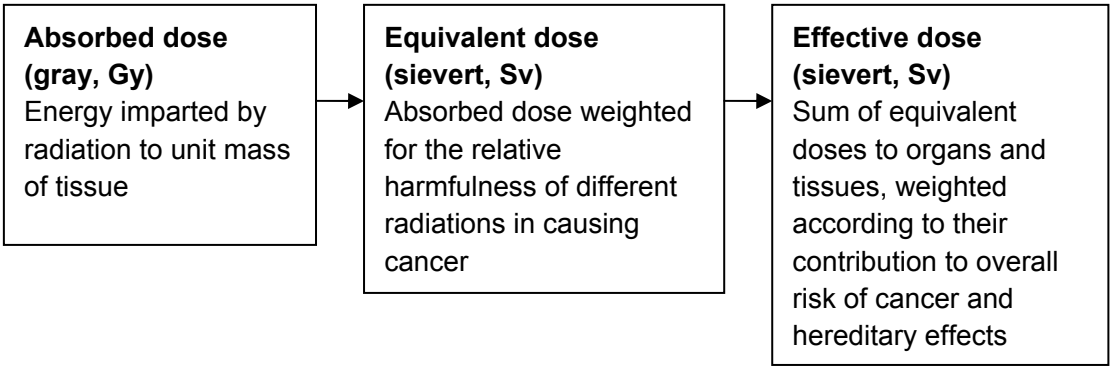

Figure A.7 Summary of dose quantities

APPENDIX B Locations of interest at the University of Manchester

The names of the buildings and the room numbering for the locations of interest have changed a number of times since Professor Rutherford and his colleagues' worked at Manchester University in the early 20th century. The floor plans for the Schuster Building (now Rutherford Building and Manchester Museum) and the Schuster Annex (now Coupland 1 Psychology Annex) shown in Figures B.1 to B.6 have been reproduced from Dr Neil Todd's report (Todd, 2008) which states that the plans are based on Schuster and Hutton plans of 1906. In addition, a floor plan of the basement of Coupland 1 Psychology Annex, provided by the University of Manchester, is given in Figure B.7. The plan for the ground floor of the Coupland 1 Psychology Annex (Figure B.8) has also been included so that the location of the basement below can be understood as the Annex is now part of a larger building.

In the monitoring reports, detailed in APPENDIX C, room numbers have been given. Table B.1 summarises the room names or numbering for the rooms of interest from these monitoring reports.

Table B.1 Different names of rooms over time identified in the monitoring reports as being the most heavily contamination

	Name of the building		
	Physical Laboratory	Coupland 1 Building	Rutherford Building and Manchester Museum
	1900	1968	Present day
Basement	Liquid air and research	CB04, CB05 and CB07	B55, B58 and B57 (Manchester Museum)
	Research (29.2 x 23.5)	CB09	B62 (Manchester Museum)
	Research (27.10 x 19.3)	CB10	B63 (Manchester Museum)
Ground floor	Private laboratory (28.0 x 19.7)	G54 and G55	G.055 (Rutherford Building)
First floor	Balance room (20.10 x 16.6)	C1.10	1.51 & 1.52 (Manchester Museum)
Second floor	Preparation room (20.10 x 16.6)	2.52 2.53	2.052a (Rutherford Building)
	Transit (19.7 x 13.10)	2.62	2.62 (Rutherford Building)
	Research (19.7 x 13.10)	2.63	2.63 (Rutherford Building)

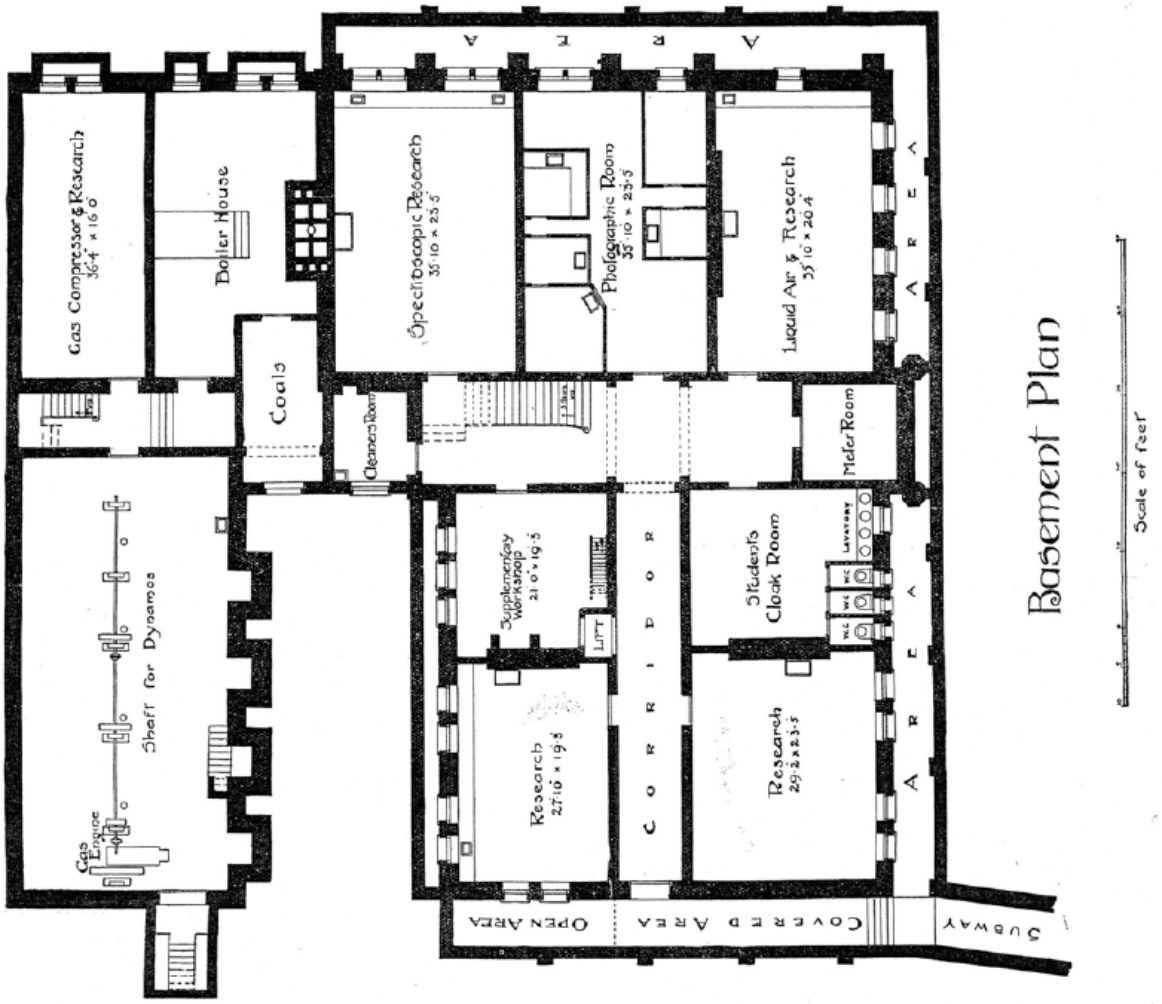

Figure B.1 Basement plan of the Schuster Building (now Manchester Museum)

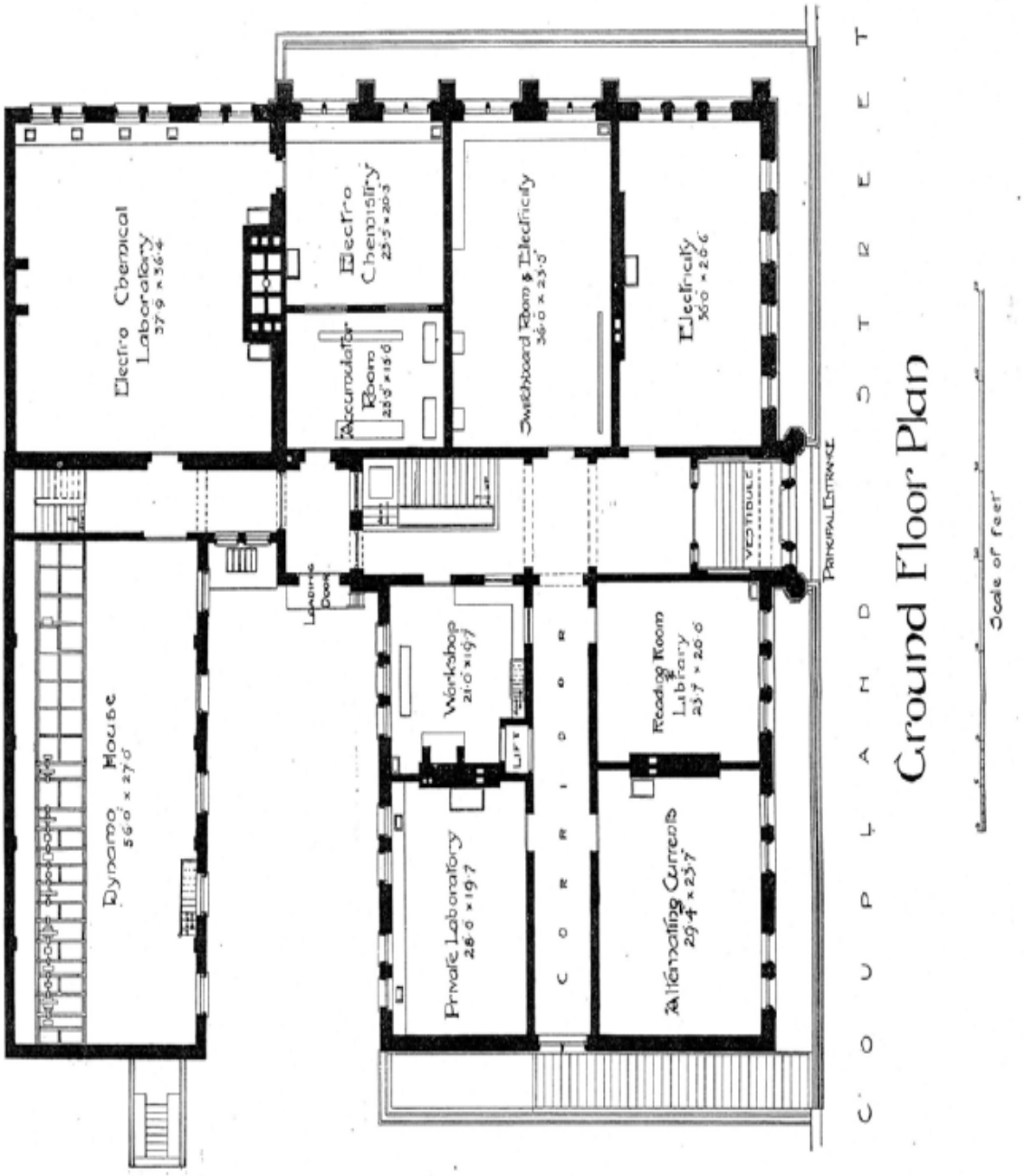

Figure B.2 Ground floor plan of the Schuster Building (now Rutherford Building and Manchester Museum)

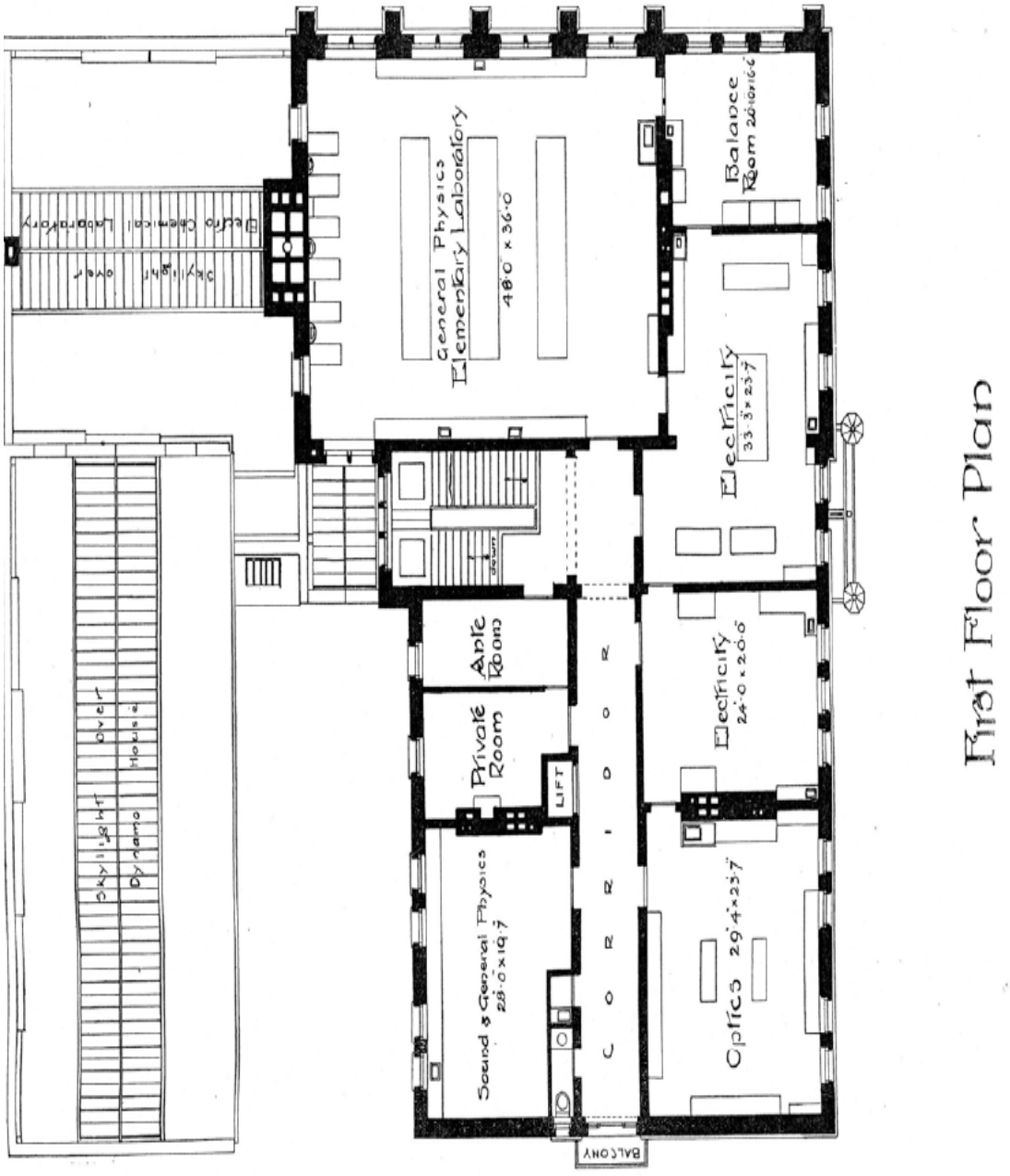

Figure B.3 First floor plan of the Schuster Building (now Rutherford Building and Manchester Museum)

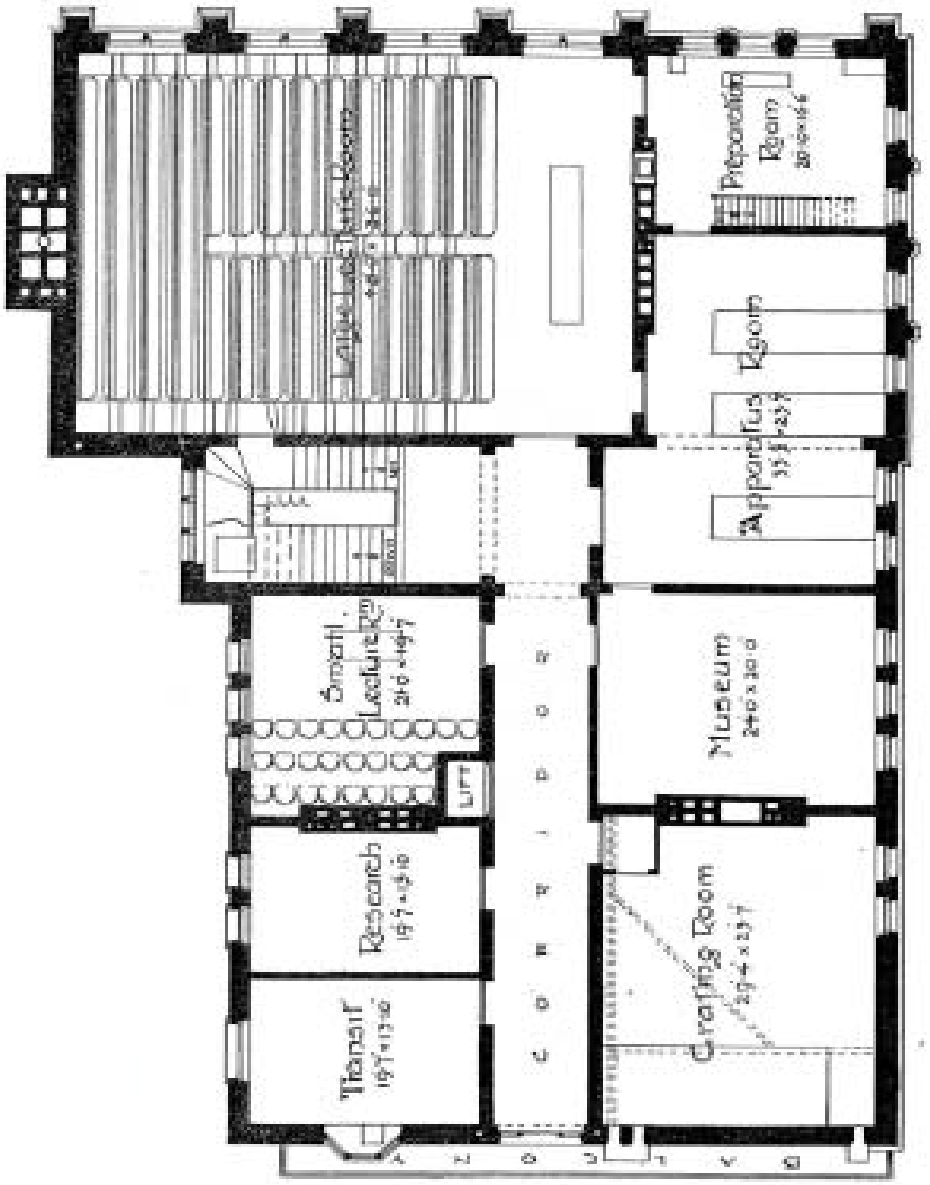

Figure B.4 Second floor plan of the Schuster Building (now Rutherford Building)

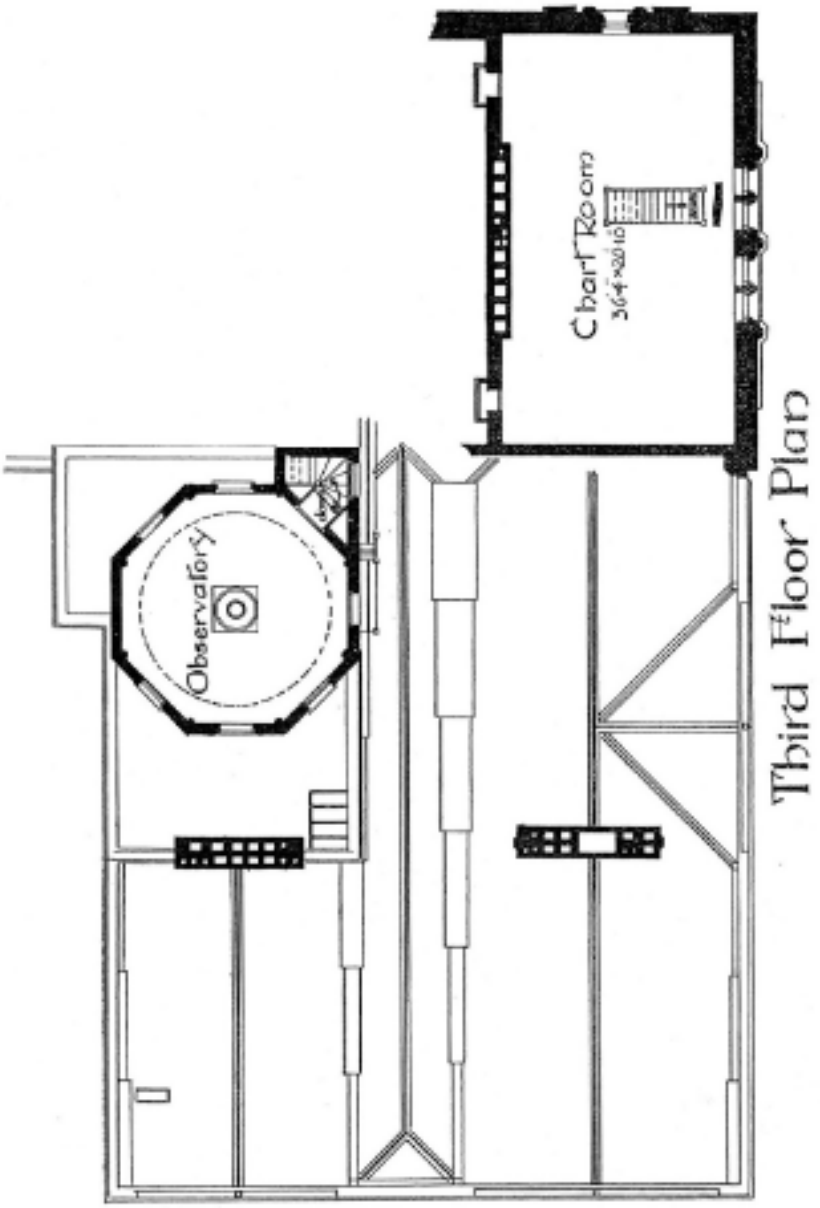

Figure B.5 Third floor plan of the Schuster Building (now Rutherford Building)

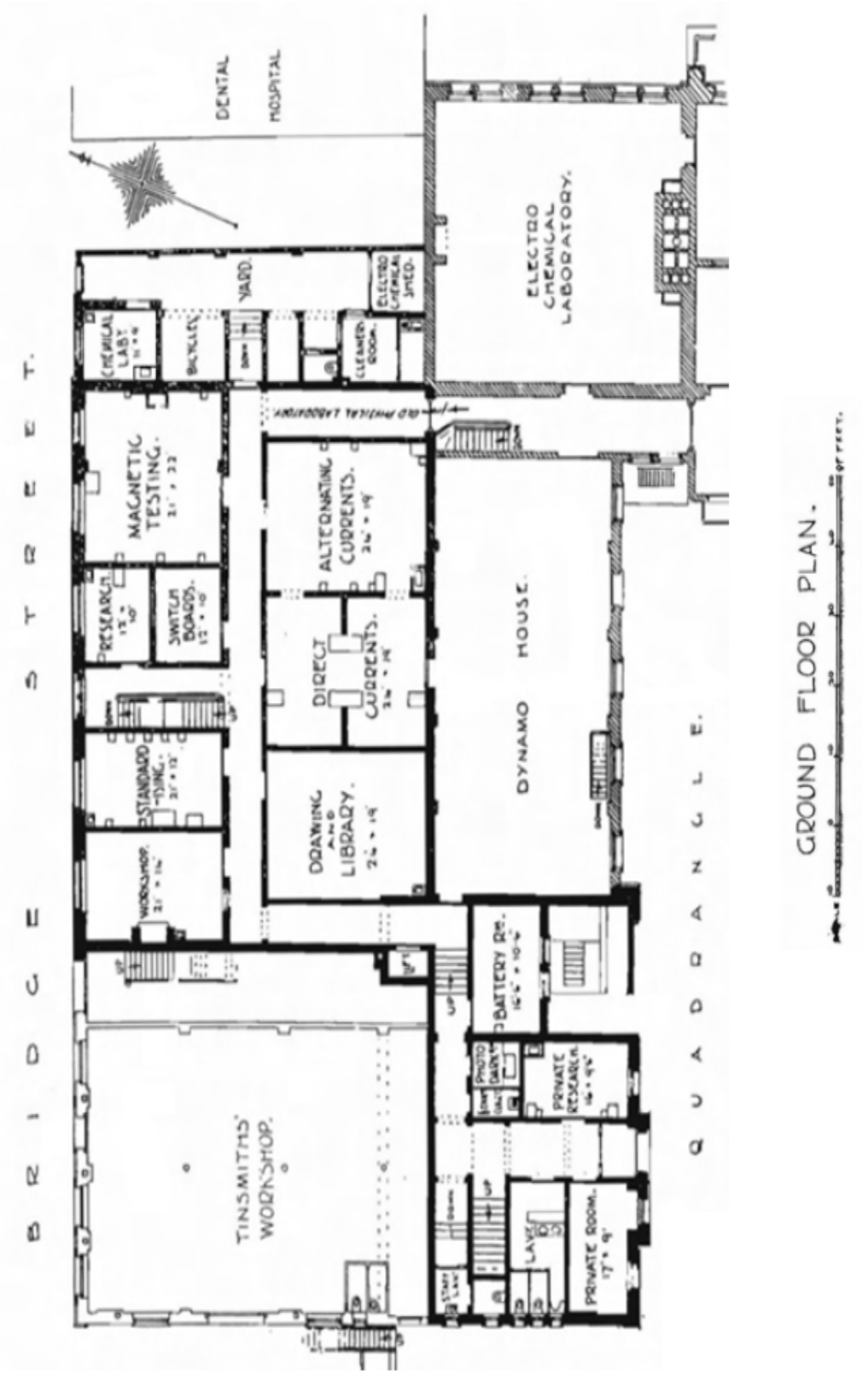

Figure B.6 Ground floor plan of the Schuster Annex Building (now Coupland 1 Psychology Annex)

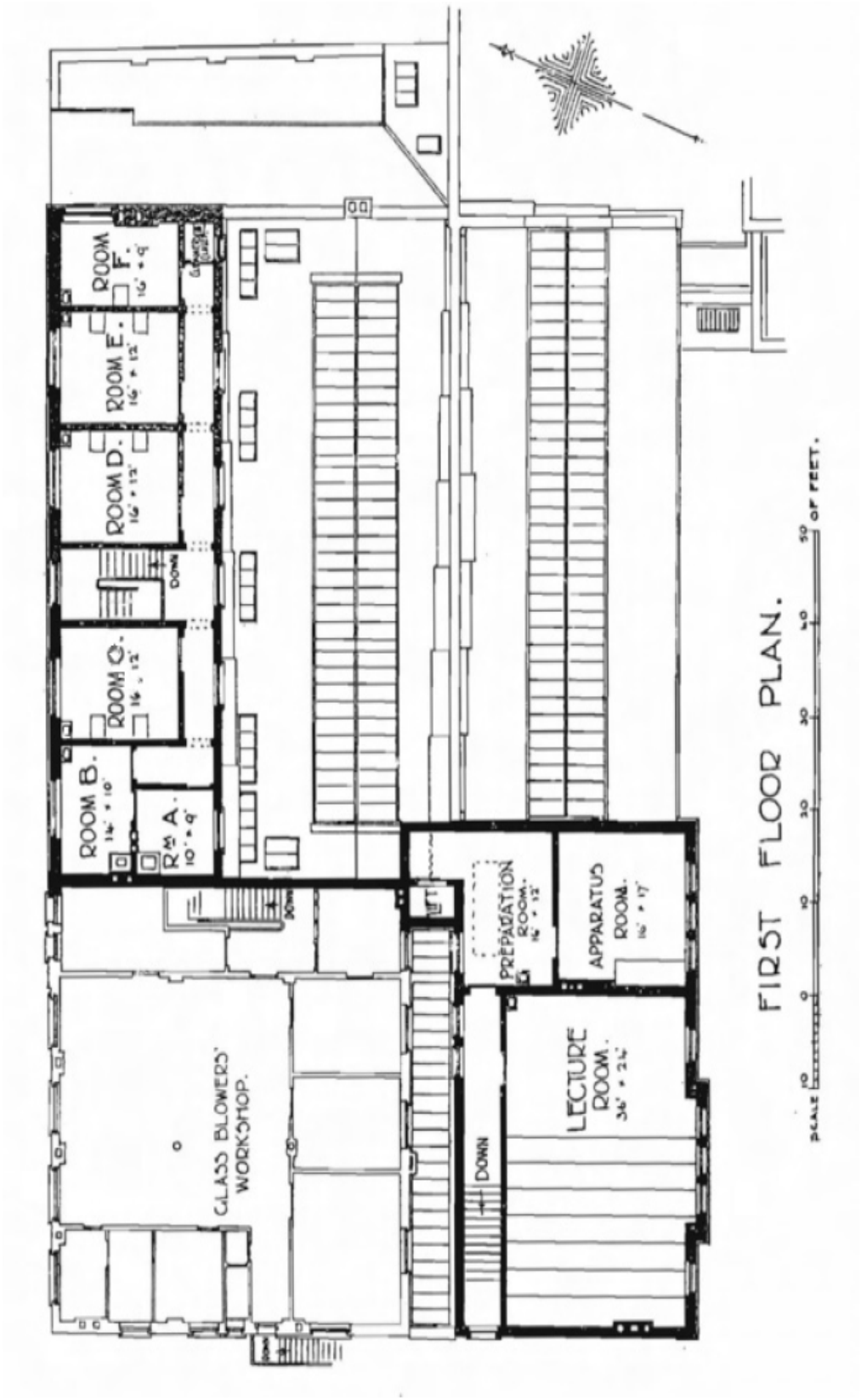

Figure B.7 First floor plan of the Schuster Annex Building (now Coupland 1 Psychology Annex)

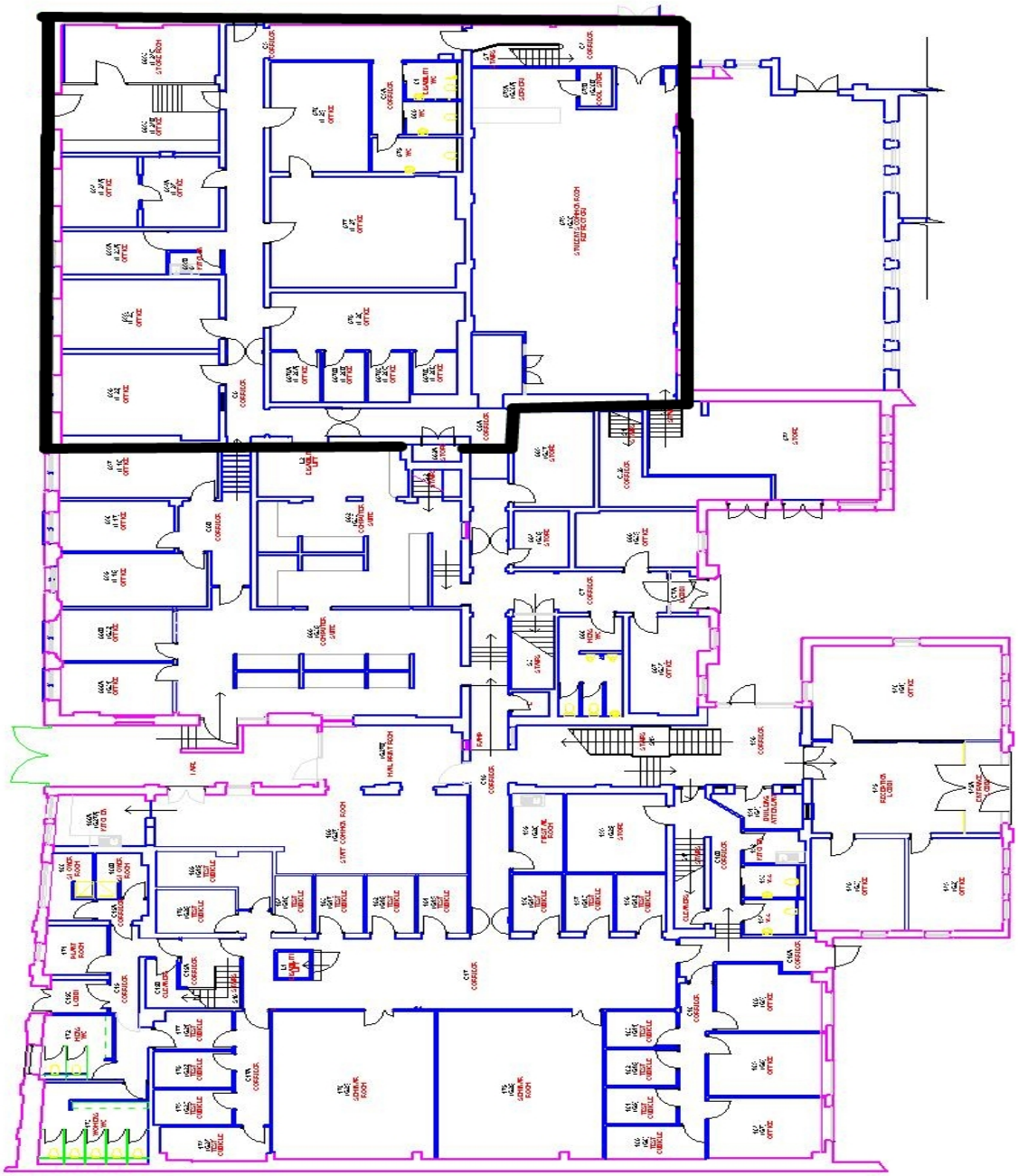

Figure B.8 Ground floor plan of the Coupland 1 Psychology Annex showing location of basement indicated by thick black line

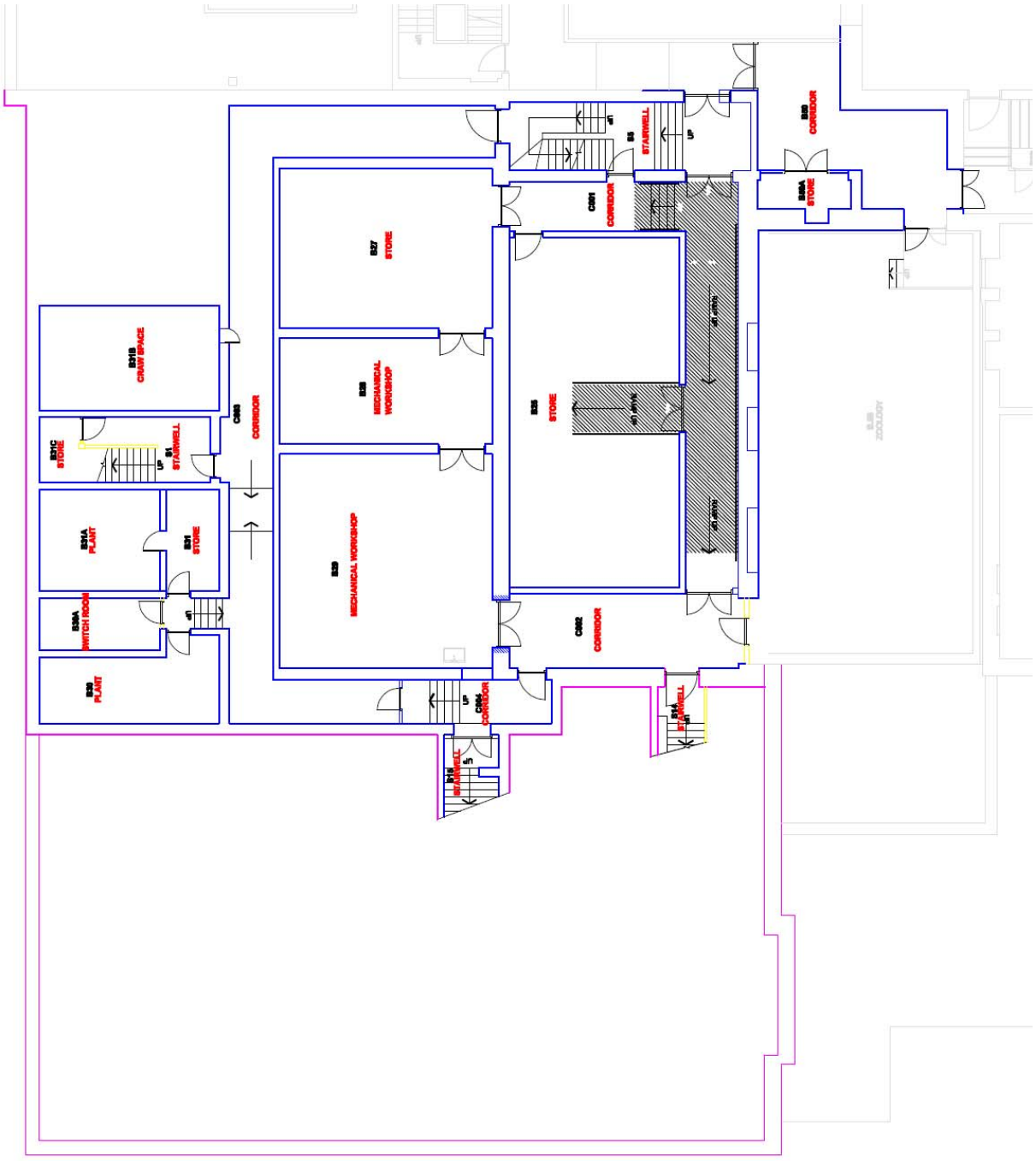

Figure B.9 Basement floor plan of the Coupland 1 Psychology Annex

APPENDIX C Derivation of contamination levels

This appendix details the derivation of contamination levels used in the assessment. Given the lack of monitoring data prior to 1999, most of the contamination levels used in the assessment were based on information from 1999 and later but some were calculated from estimates of the historical inventory.

C1 HISTORICAL INVENTORY

Table C.1 contains a summary of the estimated activities of three radionuclides that were used at the University of Manchester by Professor Rutherford and his colleagues. The following sections describe the estimation of these activities.

Table C.1 Historical inventories based on reports describing the original work at Manchester University[(a)]

Radium-226	$1.2\ 10^{10}$ Bq
Thorium-230	$2\ 10^{8}$ Bq
Actinium-227	$1\ 10^{9}$ Bq

(a) The activities are rounded up from those given in the text

C1.1 Estimation of radium-226 inventory

Dr Todd's report (Todd, 2008) describes Professor Rutherford's and his colleagues' work with radium-226. Professor Rutherford was known to have obtained 500 mg of radium bromide from Vienna, but the exact composition and corresponding activity was unknown. Dr Prise estimated that it comprised 250 mg radium, equivalent to an activity of approximately 10 GBq (Todd, 2008). In addition Professor Schuster had 60-70 mg of radium bromide, which was assumed to have approximately the same composition, which is equivalent to an activity of 1.5 GBq. Therefore the total radium-226 activity was estimated to be 11.5 GBq.

C1.2 Estimation of thorium-230 and actinium-227 inventory

By contrast the thorium-230 and actinium-227 activities given in Table C.1 were recorded in historical reports (Todd, 2008). The activities of thorium-230 and actinium-227 were roughly estimated by Professor Boltwood (Boltwood, 1911) in 1911 by comparison with the activity of an equal weight of uranium oxide, which was at that time expressed in terms of mg of radium. The activities of thorium-230 and actinium-227 were noted as being respectively, three thousand and twenty thousand times the activity of an equal weight of uranium oxide. Professor Boltwood estimated the activity of the 10 mg of precipitate containing actinium-227 as equivalent to 30 mg of radium. In addition, he estimated that the activity of the thorium-230 contained in 1.8 mg of processed thorium oxide to be equal to the activity of 5.3 mg of radium. From this it can

be calculated that Professor Rutherford's inventory included 1.1 10^9 Bq of actinium-227 and 1.9 10^8 Bq of thorium-230.

C1.3 Estimate of surface contamination levels based on historical inventory

Although monitoring data were available for radium-226 ((Robinson, 2000), (Adams, 2000), (Turner, 2000a and b)), this was not the case for thorium-230 and actinium-227. Therefore, contamination levels for thorium-230 and actinium-227 were derived from the historical inventory. A relationship between the historical inventory and measured contamination levels was first developed for radium-226 by making assumptions about the fraction of activity that was likely to have spilt on the floor and the area of floor that the contamination was spread over. This relationship was also assumed to apply to thorium-230 and actinium-227. It is recognised that there are considerable uncertainties associated with this approach. For example, the rate of spillage or area over which spills occurred may depend on the type of experiment being performed, the equipment being used, how often the radionuclide was used, the experimenter, etc and these may vary for each of the different radionuclides.

The potential area of contamination within the Rutherford Building was estimated by considering floor plans cross-referenced with those areas that monitoring indicated were the most heavily affected by contamination. This review indicated that the potential area of concern included offices on 4 floors. The maximum area within these offices was estimated to be approximately 200 m^2. However, the monitoring reports indicated that most of the floors within these offices were not contaminated. It was, therefore, additionally assumed that only 10% of the area within each of the offices was contaminated, ie, 20 m^2.

It is not possible to verify the amount of radioactive material that was spilt. Dr Neil Todd's report (Todd, 2008) describes the work being done with radioactive materials at the University of Manchester. Professor Rutherford noted that only 30 mg of Professor Schuster's radium remained out of the original 60 to 70 mg. However Todd, 2008 states that the difference between these amounts was unlikely to be due to loss through spills, but rather that it was unavailable for use by Professor Rutherford as it was being used in other experiments. It is known that accidents occurred but information is not available on the amount of material lost. There was difficultly in obtaining radium at this time and it is therefore likely that radium samples would have been carefully conserved.

The effect of varying the assumed floor areas and the amounts of activity that ended up as floor contamination was investigated for comparison with the measured levels of radium-226. Table C.2 summarises the results of this process.

Table C.2 Comparison of measured and estimated surface contamination levels

	Estimated inventory in 1903 (Bq)	Surface contamination in 1903 assuming 0.1% of activity lost (Bq/cm^2)[(a)]	Surface contamination in 1903 assuming 1% of activity lost (Bq/cm^2)[(a)]	Estimated contamination levels in the year 2000, used in this assessment (Bq cm^{-2} and Bq g^{-1})	Measured surface activity concentration in the year 2000
Radium-226	1.2 10^{10}	6 (60)	60 (600)	**200**	20 - 87 Bq cm^{-2} (inferred from (Robinson, 2000)) <1 – 200 Bq g^{-1} (Turner, 2000b)
Thorium-230	2 10^8	0.1 (1)	1 (10)	**10**	N/A[(b)]
Actinium-227	1 10^9	0.5 (5)	5 (50)	**10**[(c)]	N/A[(b)]

(a) Values are those based on a floor area of 200 m^2, with the values in brackets being based on a reduced floor area of 20 m^2

(b) No monitoring results were available for these radionuclides and their activity in the year 2000 was based solely on the estimated inventory in 1903.

(c) The activity of actinium-227 present in the year 2000 is approximately a factor of 17 less than that present in the year 1903 due to radioactive decay, this was rounded up to 10 Bq cm^{-2} for use in the assessment.

From Table C.2, it is seen that the measured levels of radium-226 indicate that the proportion of activity lost and the floor area affected was likely to be within the ranges assumed. To estimate the contamination levels for thorium-230 and actinium-227, it was cautiously assumed that 1% of the total activity was distributed over a floor area of 20 m^2, with the values rounded up to the next order of magnitude.

C2 USE OF MONITORING DATA

This section summarises the monitoring data used for the assessment. The data were obtained from monitoring reports made available to the HPA.

To assess past exposures, the results of monitoring undertaken before any remediation work, were used to derive the contamination levels. Table C.3 summarises the dates of the major remediation work known to have been carried out. Information provided by the University (Peters, 2008) indicated that, although the usage of rooms changed over time and there may have been some associated renovation of the rooms, there was no evidence of major building work or remediation prior to 2000.

Table C.3 Summary of dates of major building work

Building	Date
Coupland 1 (CB05, CB09, CB10, G54/G55, C1.10 and 2.52/2.53)	2000
Basement of Coupland 2	2001
Coupland 1 – Cohen Lecture Theatre	2002
Coupland 1 (excluding Cohen Lecture Theatre, main access corridor and lift shaft on each floor)	2004

Table C.4 summarises the types of measurements that were made in the areas of interest. It should be noted that not all of these measurements were used directly in the assessments.

Table C.4 Measurements made prior or during remediation work

Types of measurements	Equipment used	Comments
Surface contamination measurements	LB1210B or LB1210D	In most of the rooms – Coupland 1, Annex and Museum, Coupland 2
Surface gamma dose rate measurement	Mini Instruments 900D	Coupland 1 - G54, G55
Alpha counting of air samples	Equipment not known	Coupland 1 - CB05, CB09, CB10, G54/G55, C1.09, C1.10 and 2.52/2.53, 2.62 and 2.63 Additional measurements made of ground floor/1st floor and 2nd floor
Beta counting of air samples	Equipment not known	Coupland 2 – B6, B9, B10 and corridors
Beta counting of Pb-210/Po-210 contamination	Assessed by direct beta monitoring using a comparison with a 'reference' contamination spot prepared from a known activity Pb/Po-210 solution	31 drums of waste but only 7 of drums identified to have Po/Pb-210 contamination
Gamma counting (following identification of Ra-226 by gamma spectrometry)	Equipment not known	31 drums of waste with 26 drums identified to have Ra-226 contamination
Gamma spectrometry	High purity germanium detector – calibrated for 60 keV to 1836 keV	3 samples (underfloor dust from G55, wall sample from C1.10 and brick dust from room CB05)
Gamma spectrometry	Low resolution gamma spectrometry	G57 (B7), G53, 1.51, 1.52, 1.53, 1.54/1.55, 1.56, 1.57, 2.52, 2.53, 2.54, 2.55, 2.56, Mezzanine Area, 2.60, 2.61, 2.62, 2.63, the Tank Room and the Beekeepers – **These measurements were done in 2004**, ie, post 2000 remediation work for CB05, CB09, CB10, G54/G55, C1.09, C1.10 and 2.52/2.53, 2.62 and 2.63
Liquid scintillation	Equipment not known – sample quenching not accounted for	Coupland 1 – Cohen Lecture Theatre
Radon-222 measurements	Lucas cell measurement	CB05, CB09, CB10, G54/G55, C1.09, C1.10 and 2.52/2.53, 2.62 and 2.63
	HPA passive detectors	Coupland 1 – 2.54, 2.62 and 2.63

The assessment was based on a generic location with exposures and risks estimated for hypothetical groups of people. Therefore, from the many monitoring reports provided by the University the highest reported measured values were used to provide cautious estimates of contamination levels. This section summarises the monitoring results used to obtain the highest measured values.

C2.1 Gamma spectrometry results prior to remediation

Analysis of Manchester University Museum sample, 5 April 2000 (NIRAS, L2000047, (Turner, 2000b)

This measurement report does not state from which room the samples were taken but since the measurement report forms part of the post remediation report for the decontamination and clearance survey of room C1.10 (Collins, 2000) it was assumed that the samples were from room C1.10.

Two samples of wood and one sample of dust were taken and analysed by gamma spectrometry and for gross alpha/beta. The results are reproduced in Table C.5.

Table C.5 Gamma spectrometry measurements (Turner, 2000b)

	Activity concentration (Bq g^{-1})		
	Wood (1)[(a)]	Wood (2)[(b)]	Dust (3)[(c)]
U-238 decay chain			
Ra-226	200.3 ± 8.5	34.5 ± 2.6	< 0.68
Pb-214	89.6 ± 1.2	15.3 ± 0.38	0.0872 ± 0.0046
Bi-214	76.6 ± 1.2	12.99 ± 0.36	0.043 ± 0.042
Pb-210	112.3 ± 9.5	3971± 320	90.0 ± 7.2
Th-232 decay chain			
Ac-228	0.69 ± 0.52	0.41 ± 0.39	< 0.18
Pb-212	0.28 ± 0.12	0.55 ± 0.45	0.05 ± 0.025

(a) NIRAS reference L2000047-1

(b) NIRAS reference L2000047-2

(c) NIRAS reference L2000047-3

The report commented that lead-210 gamma-rays were outside the spectrometry energy calibration range. The additional uncertainty arising from this was estimated at less than 10%. The report commented that it appeared that both wood (2) and dust (3) had lead-210 concentrations in excess of that supported by radium-226.

Analysis of samples from Museum 18 August 2000 (J Turner, 18/7/01, L2000103), (Turner, 2000a)

Three samples were analysed using a high purity germanium detector. The results are shown in Table C.6.

Table C.6 High resolution gamma spectrometry measurements (Turner, 2000a)

	Activity concentration (Bq g^{-1})		
	Under floor dust from room G55 between joists 2-3[(a)]	Wall sample from room C.1.10, Local contamination[(b)]	Brick/mortar dust from brick 5/6, Under window, Room CB 05[(c)]
U-238 decay chain			
Ra-226	70.1 ± 7.7	< 5.5	53.7 ± 8.3
Pb-214	0.44 ± 0.31	< 0.39	12.20 ± 0.73
Bi-214	0.9 ± 0.32	1.00 ± 0.49	14.41 ± 0.65
Pb-210	-	4103 ± 677	-
Th-232 decay chain			
Ac-228	0.571 ± 0.094	0.243 ± 0.094	0.81 ± 0.24
Pb-212	< 0.43	< 0.38	< 0.40
Bi-212	< 3.7	< 2.6	< 2.4

(a) NIRAS reference L2000103-70

(b) NIRAS reference L2000103-85

(c) NIRAS reference L2000103-92

C2.2 Surface contamination measurements prior to remediation

Extensive surface contamination measurements were made by the University's Radiation Protection Service and its contractor, NIRAS. A summary of the highest results are presented in Table C.7 and Table C.8. It should be noted that most results given were in the form of "counts per second". These units are not useful to the assessment and hence a conversion from counts per second to Bq cm^{-2} was made using appropriate conversion factors as noted.

University of Manchester Museum Building Coupland 1. University of Manchester, (Robinson, 2000)

The radium-226 surface contamination given in Table C.7 was estimated from the Berthold LB 1210B measurement (cps). The typical response of a Berthold LB 1210B to a radium-226 alpha particle is 15 cps/Bq/cm^2 (McClure, 2009). Hence the surface contamination in Bq cm^{-2} can be inferred as the instrument reading (cps) divided by the response factor of 15 cps/Bq/cm^2.

Table C.7 Surface contamination and dose rate measurements (Robinson, 2000) and inferred radium-226 surface contamination

Room	Instruments used	Area	Results	Inferred radium-226 surface contamination (Bq cm^{-2})[(a)]
G54[(b)]	Berthold LB 1210B	Floor against partition wall	Approx 1200 cps	80
	900 mini Type D	Floor against partition wall	50 µSv/hr at 1 cm	
G55[(b)]	Berthold LB 1210B	Floor against partition wall	Approx 1300 cps	87
	900 mini Type D	Floor against partition wall	50 µSv/hr at 1 cm	87
	Berthold LB 1210B	Under carpet on lino	300 cps	20
	900 mini Type D	Under carpet on lino	13 µSv/hr at 1 cm	20

(a) It was assumed that contamination was present as a thin layer on the surface and therefore all of the radon-222 has escaped from the floor. Therefore all counts measured by the Berthold detector are due to radium-226 only. This is a cautious assumption

(b) G54 and G55 are located on the ground floor of Rutherford Building

Residual contamination survey of Coupland 1 Building, the Annexe and the Old Dental Hospital. NIRAS MTC/2000/051, Issue 02 (Adams, 2000)

The report is the most detailed one available giving surface contamination and dose rate measurements prior to remediation. Table C.8 gives the highest surface contamination and dose rates measured and the inferred radium-226 surface contamination and activity concentrations.

Table C.8 Highest surface contamination and dose rate measurements given in (Adams, 2000) and inferred radium-226 surface contamination and activity concentration

Room number[(a)]	Berthold LB122 (cps)	Bicron (1 min count) (waist height)	Bicron reading Floor (cpm)	Inferred radium-226 surface contamination (Bq cm^{-2})[(b)]	Inferred radium-226 activity concentration (Bq g^{-1})[(b)]
G54A	No result given	33567	>500000	70	90
G55	Floor 1321	33441	>500000 in corner of room	Floor 70	Floor 90
	Wall 253			Wall 13	
	Other walls 44			Other walls 2	
C1.10	1333			70	

(a) C1.10 is located in the basement of the Manchester Museum and G54 and G55 are located on the ground floor of Rutherford Building

(b) It was assumed that contamination was present as a thin layer on the surface and therefore all of the radon-222 has escaped. Therefore all counts measured by the Berthold are due to radium-226 only. This is a cautious assumption.

The radium-226 surface contamination in Table C.8 was estimated from the Berthold LB122 measurement (cps). The typical response of a Berthold LB122 to a radium-226 alpha particle is 19 cps/Bq/cm^2. Hence the surface contamination can be inferred to be the instrument reading (cps) divided by the response factor of 19 cps/Bq/cm^2 (McClure, 2009).

The inferred activity concentration was then estimated using a density for the wooden floor of 800 kg m^{-3} and a penetration depth of 1 cm for the radium-226.

C2.3 Relationship between surface contamination measurements and activity concentrations in dust

The highest radium-226 surface contamination measurements were 90 Bq cm^{-2} (Table C.7). From Table C.8 an activity concentration of 90 Bq g^{-1} was inferred. These values correspond well to the activity concentration of radium-226 of 70.1 Bq g^{-1} measured in under floor dust from Room G55 as given in the NIRAS report, L2000103 (Turner, 2000a). Therefore, it was assumed that the surface contamination was equivalent to the under floor dust activity concentration, that is 1 Bq g^{-1} is approximately equivalent to 1 Bq cm^{-2}.

C2.4 Radon-222 measurements made prior to remediation

The earliest known radon-222 measurements were reported in the Determination of Rn-222 in air Re: Museum hazard assessments given in Appendix C6 of report by Churcher et al, 2008. However, the measurements were made using a Pylon model AB-5 Portable and calibrated LUCAS LCA-2 scintillation cells. This technique for measuring radon-222 is not thought to be reliable, as it is only based on short time periods.

Table C.9 shows the results of measurements made using HPA passive radon detectors in 2002.

Table C.9 Radon-222 measurements made in Rutherford Building prior to remediation

Room	Measured radon-222 concentration Bq m^{-3}
Room 2-54 2m	23
Room 2-54 Centre of Room 1m	27

The highest measured radon-222 concentration was approximately 30 Bq m^{-3}.

C2.5 Estimation of radium-226 contamination levels for past exposures

The highest measured radium-226 activity concentration, which is 200 Bq g^{-1} is given in the NIRAS report, L2000047 (Turner, 2000b) (see Table C.5). As explained in section C2.3 it was assumed that the surface contamination levels (Bq cm^{-2}) were equivalent to the activity concentrations in dust (Bq g^{-1}) and therefore a surface contamination level of 200 Bq cm^{-2} is implied This is higher than the highest measured surface contamination level of 90 Bq cm^{-2} for G55 (Robinson, 2000). However, it was decided to use this higher value of 200 Bq cm^{-2} in the assessment to avoid any possible underestimation of doses.

C2.6 Estimation of actinium-228 contamination levels for past exposures

The highest measured actinium-228 activity concentration of 0.81 Bq g^{-1} is given in the NIRAS report, L2000103 (Turner, 2000a) (see Table C.6). This value was rounded up to 1 Bq g^{-1} for use in the assessment. Information on actinium-228 was not available for the surface contamination measurements and therefore it was assumed that the surface contamination levels were equivalent to the activity concentration, as had been done for radium-226.

C2.7 Estimation of lead-210 contamination levels for past exposures

The two highest measured activity concentrations for lead-210 were 3971 Bq g^{-1} in wood and 4103 Bq g^{-1} for the brick dust measured in C1.10 (see Table C.5 and Table C.6). The lead-210 gamma peak (46 keV) is generally difficult to detect as it is at the lower end of the detection range for the type of detector used. In addition the lead-210 gamma-rays were outside the energy calibration range of the spectrometer. NIRAS estimated that the additional uncertainty arising from the lead-210 peak being outside the calibration range was less than 10%. Therefore, in the assessment it was cautiously assumed that there was a hotspot of 5000 Bq g^{-1} of lead-210. It was not possible to distinguish lead-210 in the surface contamination measurements and therefore, it was assumed that the surface contaminations levels were equivalent to the activity concentration, as had been done for radium-226.

C2.8 Estimation of radon-222 and radon-220 levels for past exposures

Radon-222 monitoring data prior to remediation were only available from the Rutherford Building (Room 2.54) (see section C2.4). The highest measured concentration in air was approximately 30 Bq m^{-3}. No measurements were available for radon-220 (thoron), but its contribution to dose could not be ignored (Gooding, 2009).

In order to supplement the monitoring data, the radon-222 and radon-220 concentrations in air were also calculated based on the activity concentration of the parent radionuclides radium-226 and radium-224, respectively. The activity concentration for radium-226 was based on measurements as discussed above. However, as radium-224 was not measured directly, its activity was derived from the measured activity concentration of actinium-228.

In order to estimate the influx of radon-222 or radon-220 atoms into a room the number of radon-222 or radon-220 atoms produced per unit time, N_{Rn}, is required. This was estimated as follows:

$$N_{Rn} = (AC_{Ra} * M * L_{Ra}) / L$$

Where

N_{Rn} = Number of radon-222 or radon-220 atoms produced per second from the contaminated layer within the floorboards, number of atoms s^{-1}

AC_{Ra} = Activity concentration of radium-226 (200 Bq g^{-1}) or actinium-228 (1 Bq g^{-1}) in the floorboards (see Table 2 and Table 3)

M = Mass of contaminated floorboards removed during remediation work from G54 and G55 ($7.12\ 10^5$ g) (Adams, 2001b). This was taken to be representative of mass of floorboards from a contaminated room.

L_{Ra} = Thickness of contaminated layer of floorboards, assumed to be 0.1 cm

L = Thickness of floorboards, assumed to be 1 cm

Therefore N_{Rn} = $200\ Bq\ g^{-1} * 7.12\ 10^5\ g * 0.1\ cm/1\ cm$

= $14.24\ 10^6\ s^{-1}$ (radon-222)

= $1\ Bq\ g^{-1} * 7.12\ 10^5\ g * 0.1\ cm/1\ cm$

= $7.12\ 10^4\ s^{-1}$ (radon-220)

The average activity concentration of radon in air, AC_{Rn}, within the representative room was then estimated using the following equation.

$AC_{Rn} = N_{Rn} * \lambda_{Rn} * K_{Rn} * 3{,}600 / (ach * V)$

Where:

AC_{Rn} = Activity concentration of radon-222 or radon-220 in air within the representative room, Bq m^{-3}

N_{Rn} = Number of radon-222 or radon-220 atoms produced per second the contaminated layer within the floorboards, number of atoms s^{-1}

λ_{Rn} = Decay constant radon-222, $2.1\ 10^{-6}\ s^{-1}$ or radon-220, $1.2\ 10^{-2}\ s^{-1}$ (ICRP, 1983)

K_{Rn} = Emanation fraction for the material, assumed to be 0.5 (dimensionless)

ach = Number of air changes per hour in the representative room, assumed to be 1

V = Volume of the representative room, assumed to be 300 m^3

Therefore, AC_{Rn} = $(14.24\ 10^6\ s^{-1} * 2.1\ 10^{-6}\ s^{-1} * 0.5 * 3.6\ 10^3) /1.0 * 300\ m^3$

= 180 Bq m^{-3} (for radon-222)

or = $(7.12\ 10^4\ s^{-1} * 1.2\ 10^{-2}\ s^{-1} * 0.5 * 3.6\ 10^3) /1.0 * 300\ m^3$

= 5100 Bq m^{-3} (for radon-220)

The ability of radon-222 or radon-220 gas to leave a material is related to a parameter termed the emanation fraction. The value of this parameter will depend on the material with which radium has become associated with. Recoil effects, combined with the time it takes free radon atoms to diffuse through the medium in relation to its short half-life, act to reduce the proportion of radon that can escape from the material. In this case it was assumed that the contamination was due to radium in solution soaking into the top layer of the wooden floor. Although the emanation of radon from various media, including materials used in the construction of buildings, has been studied extensively, no data exists for absorbed radium liquid. HPA (Dixon, 2009) gave a conservative estimate of the emanation fraction of 0.3 - 0.5 for radium solution absorbed by wood. In this

assessment the upper value of this range was used, noting that this would result in a radon concentration in air at the higher end of the potential range.

The air concentration of radon within a room will also depend on the room's ventilation rate. The ventilation rate for a typical office is generally between 0.8 and 1.3 air changes per hour, although this varies throughout the day as windows are opened or people move around within the room creating airflows. A reference value of one air change per hour was used in the assessment (Wrixon et al, 1988).

Given the short half life of radon-220 (56 s) two additional factors were taken into account when estimating the air concentration. Tschiersch et al (Tschiersch et al, 2007) reported a ten fold drop in concentration at 1 m above the floor compared to the concentration at the floor surface. Any floor treatment or covering will act to reduce the amount of radon-220 escaping from the surface and an additional ten fold drop in concentration was included to account for this (Miles, 2009).

Revised radon-220 air concentration (1 metre above floor)

= Estimated air concentration

x drop off in concentration with height (1 metre above floor) = 0.1 (Tschiersch et al, 2007)

x reduction due to wax on flooring = 0.1 (Miles, 2009)

This gives a revised radon-220 air concentration of 50 Bq m^{-3}. This value was used in the assessments.

C2.9 External dose rates made following remediation of the buildings

External dose rates made following remediation of the buildings are recorded in two reports (Frith, 2001); (Adams, 2001a)) and are given in Table C.10. The highest dose rate measured was 0.173 μSv h^{-1} (which includes the contribution from natural background).

Table C.10 External dose rates at 1 m above the floor made following remediation

Measurement report	Rooms	Gamma dose rate at 1 m using Mini Instruments 6-80 (μGy hr^{-1})
NIRAS MTC/01/005 (Frith, 2001)	CB.05	0.16
	CB.09	0.15
	CB.10	0.15
	G54/G55	0.14
	C1.10	0.14
	2.52/2.53	0.15
NIRAS MTC/01/024 (Adams, 2001a)	2.62	0.166
	2.63	0.173

The dose rate reported in Table C.10 was rounded up to 0.2 μSv h^{-1} and this value was used in the assessment of current and future exposures.

C2.10 Surface contamination measurements following remediation of the buildings

There are ten monitoring reports which gave surface contamination measurements at different locations in the Rutherford Building and the Coupland 1 Psychology Annex. However, only three monitoring reports found any contamination following remediation and these measurements were only slightly above background as detailed below.

Surface contamination measurements made in Rutherford Annex,, published on Manchester University website (University of Manchester, 2009)

The only measurement reported above background is for room I.38 Child Study Centre of 40 cps using a Berthold LB 122. This relates to a surface contamination level of less than 1 Bq cm^{-2}.

Manchester Museum Survey of Room B-64 Fabric Store Coupland One Building 12/01/05 (Robinson, 2005)

This survey reported 30 cps using a Berthold LB1210 B, which is equivalent to about 1 Bq cm^{-2}.

Radiation monitoring of rooms of Manchester Museum, room B58, B62 and B63 - 5th May 2009 (Robinson, 2009)

This source reported 48 cps in room B-58 using a Berthold LB-122 which relates to about 1 Bq cm^{-2}.

Based on these limited measurements it was decided to cautiously assume a surface contamination level of 1 Bq cm^{-2} after remediation. As detailed in section C2.3 it was assumed that the surface contamination levels were equivalent to the activity concentration. As no information existed that allowed a breakdown of the radionuclides

present it was cautiously assumed that all members of the uranium and thorium decay chains were present at this activity concentration.

C2.11 Radon-222 measurements made after remediation

Following remediation of the buildings further radon-222 measurements were made in the Rutherford Building, the Manchester Museum and the Coupland 1 Psychology Annex (University of Manchester, 2009a-c). The measurement results varied from less than 10 to 410 Bq m^{-3}. The highest radon-222 concentrations in air were measured in a storeroom (G37) in the Coupland 1 Psychology Annex. Since this room is not occupied it was decided that it would not be appropriate to use these measurements for assessing doses. The remaining radon-222 concentrations were below 180 Bq m^{-3}, the value which had been used for the assessment of past exposures. It was decided to cautiously also use this value for the assessment for current and future exposures.

C2.12 Distribution patterns of contamination

The following reports discuss the distribution of the contamination over the floor and walls.

Decommissioning report for decontamination and clearance survey of room C1.10, 21 June 2000 (S Collins, NNC, project ref CB6155) (Collins, 2000)

This states that two contaminated wall/floor joints 1 m and 2 m in length were found in one corner of the room. There was another area of contamination of 100 cm^2.

Proposal for Stage 2 Hard Stripping. A J Frith. 14/12/04. IRAS (Frith, 2004b)

This states that significantly higher levels of contamination are present beneath the floorboards than on the surface, and that to date this has been in the form of dust. The survey identified more than 60 areas of contamination, the majority being low level and discrete contamination. These areas are widely distributed within the building

Radiation survey of Coupland 1 Building upon completion of soft stripping (AJ Frith, December 2004, IRAS Limited, Technical Report E04003/TR/002 Issue 01) (Frith, 2004a)

Photographs taken as part of this survey indicate small discrete patches of contamination.

C2.13 Reference to sanding of the floors

The following document discusses whether sanding of the floor boards had occurred.

Developments in monitoring in Coupland 1 Building, NIRAS (Frith, 2000)

The following text was taken from the reference.

‘Work in room G54/G55 yielded a surprise. When radium contaminated floor boards were removed high level of local lead-210 contamination were found which had not been

detected through the timber of the floor boards. Since we been lead to believe that the floor boards are original, and indeed appeared to be so, we could not explain how lead-210 could be present below the floor boards and not at the surface. We were forced to conclude that the floor boards had in fact been replaced.

We have now part completed decontamination in room 2.52/2.53. We have discovered that here again the lifting of floor boards has exposed hitherto undetected lead-210 (or some other pure beta emitter) that is not readily detected from above the floor boards.

The only conclusion I can come to is that the original floor boards, heavily contaminated with spilled lead-210 (some of which leaked through the gaps) were sanded during the laying of lino or other floor covering.'

APPENDIX D Methodology for assessing doses

This appendix details the methodology used for assessing the doses from past exposures and from current and future exposures. The methodologies for the two assessments are the same except for the calculation of dose from external irradiation and also that different activity levels were used in the two cases. For the assessment of doses from past exposures the contamination levels used are given in Table 2 to Table 5 of the main text, with the hotspot contamination level being given in Table 6. For the assessment of doses from current and future exposures the contamination levels used are given in Table 8. It was assumed that all of the lead-210 contamination hotspots were removed during remediation of the buildings and therefore exposure from a hotspot was not considered in the assessment of doses from current and future pathways.

D1 DOSES FROM EXTERNAL IRRADIATION

D1.1 Assessment of doses from past exposures

In assessing the exposure of a person to external irradiation from contamination, radionuclides were assumed to be located both on the floor boards, represented by a surface layer of activity, and as a layer of dust under the floor boards. For material under the floor boards, account was taken of the effect of any shielding provided by the overlying wood of the floor. The dose from external irradiation from radionuclides located on or under the floor boards and from the hotspot, shown schematically in Figure 1 of the main text, D_{ext} (Sv y^{-1}), was estimated using the following equation.

$$D_{ext} = T_{floor} * (DR_{surface} * AC_{surface} + DR_{dust} * \rho * AC_{dust}) + T_{HS} * DR_{HS} * AC_{HS}$$

Where

T_{floor} = Length of time exposed to radionuclides on or under the floor. For office-based staff this equals 2000 h y^{-1} whilst for the maintenance worker this equals 220 h y^{-1}

T_{HS} = Length of time exposed to radionuclides located within a hotspot. For office-based staff this equals 2000 h y^{-1} whilst for the maintenance worker this equals 220 h y^{-1}

$DR_{surface}$ = External irradiation dose rate derived using the model Microshield version 7.02 (Negin, 1986) for exposure to a 1 m diameter circular patch of contamination on the surface of the floor, Sv h^{-1} per Bq cm^{-2}. Rotational geometry was used.

$AC_{surface}$ = Activity concentration within contamination on the surface of the floor, Bq cm^{-2} (see Table 2 to Table 5)

DR_{dust} = External irradiation dose rate derived using the model Microshield version 7.02 (Negin, 1986) for exposure to a 1 m diameter circular patch of

contaminated dust under the floor, Sv h^{-1} per Bq cm^{-3}. Rotational geometry was used.

ρ = Assumed density of dust under the floor, 0.5 g cm^{-3}

$AC_{surface}$ = Activity concentration of contamination in dust under the floor, Bq g^{-1} (see Table 2 to Table 5)

DR_{HS} = External irradiation dose rate derived using the model Microshield version 7.02 (Negin, 1986) for exposure to contamination within a hotspot, Sv h^{-1} per Bq cm^{-2}. Anterior/posterior geometry used.

AC_{HS} = Activity concentration of contamination within a hotspot, see Table 6. Bq cm^{-1}

The above equation was summed over all radionuclides present.

For contamination on the floor, both on the floor boards and under the floor within the dust layer, the floor area covered by the contamination was considered to be represented by a circular patch of diameter 1 m. The external dose rate from radionuclides within dust located under the floor boards was modelled assuming that the dust layer was 0.5 cm thick, with an air space above of 5 cm and above that floor boards made of wood of thickness 1 cm. The receptor point, representing the location where the dose rate was estimated, was at a distance 1 m above the floor board and was located above the centre of the contaminated area; see Figure 1 of the main text for a schematic of the model. For exposure from contamination on the floor, the irradiation field was described by a rotational geometry, representing an exposure along the body length. Wood was assumed to be composed of CH_2O with a density of 0.75 g cm^{-3}. Dust was assumed to be composed of carbon with a density of 0.5 g cm^{-3}.

It was assumed that the exposed individual would spend their entire time at work above the contamination, that is, no time was spent in areas outside of the patch of contamination. This was considered cautious but may occur if, for example, a chair was located at this location and an individual spent most of their time sitting.

In addition to exposure to contamination on or under the floor all exposed groups were considered to be exposed to a small patch of contamination on the wall. This is the ‘hotspot’ contamination profile given in Table 6.

D1.2 Assessment of doses from current and future exposures

For assessing the dose from external irradiation for current and future exposures, D_{ext} (Sv y^{-1}), the equation below was used.

D_{ext} = DR * T

Where

DR = Dose rate within an office, 2 10^{-4} mSv per hour, taken from the monitoring reports (see section C2.9)

T = Exposure time, hours per year. For office-based staff this equals 2000 h y^{-1} whilst for the maintenance worker this equals 220 h y^{-1}

It should be noted that external irradiation from the contamination was considered to irradiate all organs within the body equally. Therefore, no equivalent dose to individual organs was estimated for this pathway with the dose to each organ being assumed to be equal to the effective dose experienced by the whole body.

D2 DOSES FROM INHALATION

D2.1 Doses from inhalation of radon gas

The effective dose or the equivalent dose to the lungs from the inhalation of radon-222 or radon-220 gas, D_{Rn} (mSv y^{-1}), was estimated using the following equation.

$$D_{Rn} = DC_{Rn} * T * AC_{Rn}$$

Where

DC_{Rn} = Effective dose coefficient or the lung equivalent dose coefficient, mSv per h per Bq m^{-3}. See text below.

T = Length of time in the office inhaling radon gas. For the office-based staff worker this equals 2000 h y^{-1} whilst for the maintenance worker this equals 220 h y^{-1}

AC_{Rn} = Average activity concentration of radon-222 or radon-220 in air within the representative room, 180 Bq m^{-3} for radon-222 and 50 Bq m^{-3} for radon-220. See section C2.8 for details.

For radon-222, the effective dose coefficient was derived from European Commission Basic Safety Standards (European Commission, 1996) and ICRP Publication 65 (ICRP, 1993) whilst for radon-220, the effective dose coefficient was obtained from (UNSCEAR, 2000). To obtain the lung equivalent dose coefficient, the effective dose coefficients were divided by the tissue weighting factor as defined by ICRP (ICRP, 1991), which for the lung is equal to 0.12.

An equilibrium factor is used as a measure of the degree of disequilibrium between radon gas and its short-lived progeny. The equilibrium factor is one when the activities of the radon progeny and radon gas are equal, meaning that the decay products have stayed close to the radon parent long enough for equilibrium to be reached (a couple of hours). Progeny of radon adhere to objects or dust particles because of their electrostatic charge, whereas gaseous radon does not, so the equilibrium factor in the atmosphere is usually less than one. The equilibrium factor is lowered by air circulation or air filtration devices and it is increased by airborne dust particles (such as cigarette smoke). The recommended equilibrium factor for radon-222 for indoor air is 0.4 (ICRP, 1993) and for radon-220 is 0.1 (UNSCEAR, 2000).

Table D.1 Summary of the dose coefficients used in this assessment for the inhalation of radon-222 and radon-220

	Radon-222 Sv per h per Bq m^{-3}	Radon-220 Sv per h per Bq m^{-3}
Effective dose coefficient	$1.5\ 10^{-9}$	$2.4\ 10^{-8}$
Equivalent dose coefficient for the lung	$1.3\ 10^{-8}$	$2.0\ 10^{-7}$

D2.2 Inhalation of contaminated dust

Dust containing radioactive material could be resuspended from the floor and subsequently inhaled. The activity concentration of the dust, expressed as Bq g^{-1}, was assumed to be the same regardless of whether it was in the air or on surfaces. The amount of dust in air was discussed in section 5.3. The dose from inhaling dust, D_{dust} (Sv y^{-1}), to the two exposed groups was assessed using the following equations.

For the office-based staff member

$$D_{dust} = DC_{inh} * INH * F_{ix} * T * DL * (F_{floor} * AC_{floor} + F_{HS} * AC_{HS})$$

Where

DC_{inh} = Effective and organ specific equivalent dose coefficients for the inhalation of radionuclides of interest, Sv Bq^{-1}, from (ICRP, 1996) and (ICRP, 2001) respectively

INH = Inhalation rate for a working adult, 1.2 m^3 h^{-1} (Smith KR and Jones AL, 2003)

F_{ix} = Fraction of the contamination on surfaces that was not fixed, assumed to be 1. See section 5.5.3 for more details.

T = Time spent in the office, 2000 h y^{-1}

DL = Normal working levels of dust loading, see Table 10

AC_{floor} = Activity concentration of dust originating from areas of widespread contamination, Bq g^{-1}

AC_{HS} = Activity concentration of dust originating from the hotspot where applicable, Bq g^{-1}

F_{floor} = Fraction of inhaled dust that was contaminated with radionuclides from large areas of contamination, 0.1. See section 5.5.2 for more details.

F_{HS} = Fraction of inhaled dust that was contaminated with radionuclides from a hotspot, 0.001. See section 5.5.2 for more details.

For the maintenance worker

$$D_{dust} = DC_{inh} * INH * F_{ix} * (T_{nw} * DL_{nw} * [F_{floor} * AC_{floor} + F_{HS} * AC_{HS}] + DL_{wr} * T_{wr} * F_{wr} * AC_{HS})$$

Where

DC_{inh} = Effective and organ specific equivalent dose coefficients for the inhalation of radionuclides of interest, Sv Bq^{-1}, from (ICRP, 1996) and (ICRP, 2001) respectively

INH = Inhalation rate for a working adult, 1.2 m^3 h^{-1} (Smith KR and Jones AL, 2003)

F_{ix} = Fraction of the contamination on surfaces that was not fixed, assumed to be 1. See section 5.5.3 for more details.

T_{nw} = Time spent with normal working levels of dust loading, 220 h y^{-1}, see Table 10

DL_{nw} = Normal working levels of dust loading, see Table 10, 1 10^{-4} g m^{-3}

F_{floor} = Fraction of inhaled dust that was contaminated with radionuclides from large areas of contamination, 0.1. See section 5.5.2 for more details.

AC_{floor} = Activity concentration of dust originating from areas of widespread contamination, Bq g^{-1}

F_{HS} = Fraction of inhaled dust that was contaminated with radionuclides from a hotspot, 0.001. See section 5.5.2 for more details.

AC_{HS} = Activity concentration of dust originating from the hotspot, where applicable, Bq g^{-1}

DL_{wr} = Work raised levels of dust loading, see Table 10, 1 10^{-3} g m^{-3}

T_{wr} = Time spent with a work raised level of dust loading, 20 h y^{-1}, see Table 10

F_{wr} = Fraction of inhaled dust that was contaminated with radionuclides from a hotspot generated when working with a drill, 0.1. See section 5.5.2 for more details.

AC_{HS} = Activity concentration of dust originating from the hotspot, where applicable, Bq g^{-1}

The above equations were summed over all radionuclides present.

D3 DOSES FROM INADVERTENT INGESTION OF CONTAMINATED DUST

Inadvertent ingestion of dust contaminated with radioactive materials was considered to occur when an occupant of a room placed food or other objects in the mouth that had been picked up off surfaces on which contaminated dust was present. The inadvertent ingestion rate of dust was based on a representative ingestion rate for an adult at home of 10 mg d^{-1} (Smith and Jones, 2003). A summary of the ingestion rate of dust is given in section 5.4. The dose, D_{ing} (Sv y^{-1}), from the inadvertent ingestion of radionuclides associated with dust was calculated for each of the exposed groups using the following equations.

For the office-based staff member

$D_{dust} = DC_{ing} * ING * F_{ix} * T * (F_{floor} * AC_{floor} + F_{HS} * AC_{HS})$

Where

DC_{ing} = Effective and organ specific equivalent dose coefficients for the inhalation of radionuclides of interest, Sv Bq^{-1}, from (ICRP, 1996) and (ICRP, 2001) respectively

ING = Inadvertent ingestion rate of dust. See Table 10

F_{ix} = Fraction of the contamination on surfaces that was not fixed, assumed to be 1. See section 5.5.3 for more details.

T = Time spent in the office, 2000 h y^{-1}, See Table 10

AC_{floor} = Activity concentration of dust originating from areas of widespread contamination, Bq g^{-1}

AC_{HS} = Activity concentration of dust originating from the hotspot, where applicable, Bq g^{-1}

F_{floor} = Fraction of ingested dust that was contaminated with radionuclides from large areas of contamination, 0.1. See section 5.5.2 for more details.

F_{HS} = Fraction of ingested dust that was contaminated with radionuclides from a hotspot, 0.001. See section 5.5.2 for more details.

For the maintenance worker

$D_{dust} = DC_{ing} * F_{ix} * (T_{nw} * ING_{nw} * [F_{floor} * AC_{floor} + F_{HS} * AC_{HS}] + ING_{wr} * T_{wr} * F_{wr} * AC_{HS})$

Where

DC_{ing} = Effective and organ specific equivalent dose coefficients for the inhalation of radionuclides of interest, Sv Bq^{-1}, from (ICRP, 1996) and (ICRP, 2001) respectively

F_{ix} = Fraction of the contamination on surfaces that was not fixed, assumed to be 1. See section 5.5.3 for more details.

T_{nw} = Time spent at normal work, 220 h y^{-1}, See Table 10

ING_{nw} = Inadvertent ingestion rate of dust during normal work, 1 mg h^{-1}

T_{wr} = Time spent actively working, 20 h y^{-1}, See Table 10

AC_{HS} = Activity concentration of dust originating from the hotspot, where applicable, Bq g^{-1}

ING_{wr} = Inadvertent ingestion rate of dust during periods of active working, 10 mg h^{-1}

AC_{floor} = Activity concentration of dust originating from areas of widespread contamination, Bq g^{-1}

AC_{HS} = Activity concentration of dust originating from the hotspot, where applicable, Bq g^{-1}

F_{floor} = Fraction of ingested dust that was contaminated with radionuclides from large areas of contamination, 0.1. See section 5.5.2 for more details.

F_{HS} = Fraction of ingested dust that was contaminated with radionuclides from a hotspot, 0.001. See section 5.5.2 for more details.

F_{wr} = Fraction of ingested dust that was contaminated with radionuclides from a hotspot generated when working with a drill, 0.1. See section 5.5.2 for more details.

The above equations were summed over all radionuclides present.

APPENDIX E Details of calculations of risk of exposure induced death and other risk measures

The measure of lifetime detriment used in this risk assessment was the 'Risk of Exposure Induced Death' (REID). This measure is widely used in the radiation epidemiology community for measuring the lifetime risk of radiation induced cancer. It is appropriate for this risk assessment as it takes account of both radiation risks over the whole of a person's life and risks from competing causes of death. There are a number of similar measures of lifetime detriment that could have been used, for example, the 'Excess lifetime risk' (ELR). The ELR differs from the REID measure in that, for a population calculation, it excludes that proportion of people that it predicts would die from a radiation induced cancer who would have died of that cancer anyway in the absence of a radiation exposure. In view of the size of radiation doses being considered in this assessment, the differences in the values of these measures would be expected to be very small. A good review of lifetime detriment measures has been published by Thomas et al (1992).

E1 DEFINITION OF THE RISK MEASURES

Comprehensive definitions of these measures are defined in Thomas et al (1992) and also in Appendix B of Annex A of the 2006 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 2008). The definitions given below are simplifications that are appropriate when the quantities used in the calculations (such as the underlying death rates) are available on an annual basis and not as continuous mathematical functions.

Risk of Exposure Induced Death

For a particular cancer type, *C*, the REID for an individual who receives a single acute organ specific dose, D, at age e is defined as:

$$REID_c(e,D) = \sum_{a=e}^{99} \left[\mu_c(a \mid e,D) - \mu_c(a)\right] S(a \mid e,D)$$

Where

$\mu_c(a)$ is the annual death rate from cancer type *C* at age *a*

$\mu_c(a \mid e,D)$ is the annual death rate from cancer type *C* at age *a* conditional on exposure *D* received at age *e*.

$S(a \mid e,D)$ is the probability of the individual surviving to age *a* conditional on exposure *D* received at age *e.*

Expanding these functions further gives

$$\mu_c(a|\, e,D) = \mu_c(a).\ (1 + \mathrm{ERR}(a)_{C,e,D})$$

where $\mathrm{ERR}(a)_{C,e,D}$ is the appropriate excess relative risk value for cancer type *C* for a subject who is age *a* and who received dose *D* at age *e.* For these calculations the ERR functions are taken from the most recent set of models proposed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 2008).

For cancers of the brain, liver, lung and leukaemia, models specific to these cancers were used. However, for pancreatic cancer where no type-specific model was available, a general 'all solid cancer' model was used.

$$S(a \mid e,D) = \prod_{age=e}^{a} S(a-1 \mid e,D) \times \left[1 - \text{Probability(death in year } a \mid e,D)\right]$$

Where

Probability (death in year $a \mid e,D$) = $\mu(a) - \mu_c(a) + \mu_c(a|e,D)$ and

$\mu(a)$ is the annual all cause death rate at age *a*.

Note: For a subject who receives *n* annual doses $d_e, \ldots d_{e+n}$ starting at age *e* then *D* can be considered as representing the set of doses $d_e, \ldots d_{e+n}$ and the excess relative risk function becomes

$$ERR(a)_{C,e,D} = \sum_{d=d_e}^{d_t} ERR(a)_{C,e,d}$$

Where t = e+n if a ≥ e+n

t = e+a if a < e+n.

Loss of life expectancy (LOLE)

This is calculated as the sum, from age at first exposure to end of life (99 years), of the difference in annual survival probabilities between the exposed individual and a similar but unexposed individual.

$$LOLE_c(e,D) = \sum_{a=e}^{99} S(a \mid e) - S(a \mid e,D)$$

Loss of life expectancy per radiation induced death (LOLE/REID)
For an individual this measures the predicted loss of life expectancy if they succumb to an exposure induced cancer. It is simply calculated as the LOLE divided by the REID.

The second of these loss of life expectancy measures is the more useful as the first does not take account of the fact that an individual will either succumb to a radiation-induced cancer – in which case they do lose some of their life expectancy – or the individual does not die of a radiation-induced cancer in which case they do not lose any life expectancy.

Baseline risk
This is the lifetime risk that the individual will die of the specified cancer in the absence of any excess radiation exposure. It is derived from the age and sex specific demographic data about the population from which the individual is drawn.

Life expectancy (unexposed)
This is the individual's predicted life expectancy in the absence of any excess radiation exposure. It is derived from the age and sex specific demographic data about the population from which the individual is drawn.

Life expectancy (exposed)
This is the individual's predicted life expectancy following the specified radiation exposure. It is derived from the age and sex specific demographic data about the population from which the individual is drawn and the radiation risk model for the cancer in question.

It should also be noted that none of these measures corresponds to the measure of attributable risk.

E2 DETAILS OF THE TWO EXPOSURE SCENARIOS CONSIDERED

Two scenarios have been considered: one retrospective and one prospective.

In each case a hypothetical male subject was assumed to receive annual doses in each of forty years following first exposure at age twenty years. In the retrospective scenario the exposure was considered to start in 1950 while in the prospective scenario the first exposure was in the year 2000. In each case the subject was assumed to have the potential to live to 100 years. The risk values calculated for this subject represent an upper limit for the male population as anyone who started work at a more advanced age would have less time for any risk to be expressed.

England and Wales underlying specific cancer death rates and the overall age specific mortality rate, obtained from the Office for National Statistics (ONS), were used for the years 1950 to 1999. For all subsequent years the 1999 rates were used (ONS, 2007).

The annual radiation induced cancer risk was derived from the set of relative risk models published by UNSCEAR (2008). For most cancer types the appropriate cancer site specific risk model was used. However, for pancreatic cancer, no model specific to this cancer is available so the ‘all solid cancer’ model was used.

These models calculate risk based on annual doses. For the purposes of these calculations, the committed effective dose to 50 years, from each annual intake was assumed to be expressed all in the year of the intake.

While this assumption is not strictly accurate, it does represent the ‘worst case scenario’ for the actual dose distribution and so provides an upper bound on the variation in the radiation risk related to this factor.

APPENDIX F Assessed dose and risks

This Appendix gives tables containing the assessed dose and risks for past exposures and current and future ones.

Table F.1 Annual effective and organ equivalent doses for 1950 to 2000 (Radon in this context means radon-222 and radon-220)

year		Integrated dose (1950 to 1989)	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1974
Office worker																											
Base case	**mSv/y**																										
Bone surface	Equivalent	236.3	3.4	3.1	8.9	8.6	8.4	8.2	8.0	7.7	7.5	7.3	7.2	7.0	6.8	6.6	6.4	6.3	6.1	6.0	5.8	5.7	5.5	5.4	5.3	5.1	5.0
Brain	Equivalent	29.7	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Effective (inc radon)	Effective	72.2	2.1	2.1	2.0	2.0	2.0	2.0	2.0	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.8	1.8	1.8	1.8	1.8	1.8	1.8	1.8	1.8	1.7	1.7
Liver	Equivalent	86.6	3.3	3.2	3.1	3.0	3.0	2.9	2.8	2.8	2.7	2.6	2.6	2.5	2.4	2.4	2.3	2.3	2.2	2.2	2.1	2.1	2.0	2.0	2.0	1.9	1.9
Pancreas	Equivalent	29.7	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Red marrow	Equivalent	61.4	2.2	2.1	2.1	2.0	2.0	1.9	1.9	1.9	1.8	1.8	1.8	1.7	1.7	1.7	1.6	1.6	1.6	1.5	1.5	1.5	1.5	1.4	1.4	1.4	1.4
Lungs (inc radon)	Equivalent	295.6	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4
Whole chain	**mSv/y**																										
Bone surface	Equivalent	273.0	10.3	10.0	9.8	9.5	9.3	9.1	8.9	8.7	8.5	8.3	8.1	7.9	7.7	7.5	7.4	7.2	7.0	6.9	6.7	6.6	6.5	6.3	6.2	6.1	5.9
Brain	Equivalent	31.2	0.9	0.9	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Effective (inc radon)	Effective	74.6	2.1	2.1	2.1	2.1	2.1	2.0	2.0	2.0	2.0	2.0	2.0	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.8	1.8	1.8	1.8	1.8	1.8
Liver	Equivalent	88.7	3.3	3.2	3.2	3.1	3.0	2.9	2.9	2.8	2.7	2.7	2.6	2.6	2.5	2.4	2.4	2.3	2.3	2.2	2.2	2.1	2.1	2.1	2.0	2.0	1.9
Pancreas	Equivalent	31.2	0.9	0.9	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Red marrow	Equivalent	64.7	2.2	2.2	2.2	2.1	2.1	2.0	2.0	2.0	1.9	1.9	1.8	1.8	1.8	1.7	1.7	1.7	1.7	1.6	1.6	1.6	1.6	1.5	1.5	1.5	1.5
Lungs (inc radon)	Equivalent	299.5	7.6	7.6	7.6	7.6	7.6	7.6	7.6	7.6	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5
Maintenance worker																											
Base case	**mSv/y**																										
Bone surface	Equivalent	358.3	15.3	14.9	14.4	14.0	13.6	13.1	12.7	12.4	12.0	11.6	11.3	10.9	10.6	10.3	10.0	9.7	9.4	9.1	8.8	8.5	8.3	8.0	7.8	7.6	7.3
Brain	Equivalent	7.5	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Effective (inc radon)	Effective	36.6	1.5	1.4	1.4	1.3	1.3	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.1	1.0	1.0	1.0	0.9	0.9	0.9	0.9	0.9	0.8	0.8	0.8	0.8
Liver	Equivalent	116.5	5.0	4.8	4.7	4.5	4.4	4.3	4.1	4.0	3.9	3.8	3.7	3.5	3.4	3.3	3.2	3.1	3.0	3.0	2.9	2.8	2.7	2.6	2.5	2.5	2.4
Pancreas	Equivalent	7.5	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Red marrow	Equivalent	69.2	2.9	2.8	2.8	2.7	2.6	2.5	2.4	2.4	2.3	2.2	2.2	2.1	2.0	2.0	1.9	1.9	1.8	1.8	1.7	1.7	1.6	1.6	1.5	1.5	1.4
Lungs (inc radon)	Equivalent	82.3	3.0	2.9	2.8	2.8	2.7	2.6	2.6	2.5	2.5	2.4	2.4	2.3	2.3	2.2	2.2	2.2	2.1	2.1	2.0	2.0	2.0	1.9	1.9	1.9	1.8
Whole chain	**mSv/y**																										
Bone surface	Equivalent	374.0	15.7	15.3	14.8	14.4	13.9	13.5	13.1	12.7	12.4	12.0	11.7	11.3	11.0	10.7	10.4	10.0	9.8	9.5	9.2	8.9	8.7	8.4	8.2	7.9	7.7
Brain	Equivalent	7.6	0.3	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Effective (inc radon)	Effective	37.4	1.5	1.4	1.4	1.4	1.3	1.3	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.0	1.0	1.0	1.0	0.9	0.9	0.9	0.9	0.9	0.8	0.8	0.8
Liver	Equivalent	116.9	5.0	4.8	4.7	4.5	4.4	4.3	4.1	4.0	3.9	3.8	3.7	3.6	3.4	3.3	3.2	3.1	3.1	3.0	2.9	2.8	2.7	2.6	2.5	2.5	2.4
Pancreas	Equivalent	7.6	0.3	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Red marrow	Equivalent	70.3	3.0	2.9	2.8	2.7	2.6	2.5	2.5	2.4	2.3	2.3	2.2	2.1	2.1	2.0	1.9	1.9	1.8	1.8	1.7	1.7	1.6	1.6	1.5	1.5	1.5
Lungs (inc radon)	Equivalent	85.1	3.0	3.0	2.9	2.8	2.8	2.7	2.7	2.6	2.6	2.5	2.5	2.4	2.4	2.3	2.3	2.2	2.2	2.1	2.1	2.1	2.0	2.0	2.0	1.9	1.9

Table F.1 continued

year		1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Office worker																											
Base case	**mSv/y**																										
Bone surface	Equivalent	4.9	4.8	4.7	4.6	4.5	4.4	4.3	4.2	4.1	4.0	3.9	3.8	3.7	3.7	3.6	3.5	3.4	3.4	3.3	3.2	3.2	3.1	3.0	3.0	2.9	2.9
Brain	Equivalent	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Effective (inc radon)	Effective	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
Liver	Equivalent	1.8	1.8	1.8	1.7	1.7	1.7	1.6	1.6	1.6	1.5	1.5	1.5	1.5	1.4	1.4	1.4	1.4	1.3	1.3	1.3	1.3	1.3	1.2	1.2	1.2	1.2
Pancreas	Equivalent	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Red marrow	Equivalent	1.4	1.3	1.3	1.3	1.3	1.3	1.2	1.2	1.2	1.2	1.2	1.2	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Lungs (inc radon)	Equivalent	7.4	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3	7.3
Whole chain	**mSv/y**																										
Bone surface	Equivalent	5.8	5.7	5.6	5.5	5.4	5.3	5.2	5.1	5.0	4.9	4.8	4.7	4.7	4.6	4.5	4.4	4.3	4.3	4.2	4.1	4.1	4.0	4.0	3.9	3.8	3.8
Brain	Equivalent	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Effective (inc radon)	Effective	1.8	1.8	1.8	1.8	1.8	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.6	1.6	1.6	1.6	1.6
Liver	Equivalent	1.9	1.9	1.8	1.8	1.8	1.7	1.7	1.7	1.6	1.6	1.6	1.5	1.5	1.5	1.5	1.4	1.4	1.4	1.4	1.4	1.3	1.3	1.3	1.3	1.3	1.2
Pancreas	Equivalent	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Red marrow	Equivalent	1.4	1.4	1.4	1.4	1.4	1.3	1.3	1.3	1.3	1.3	1.3	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.1
Lungs (inc radon)	Equivalent	7.5	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4
Maintenance worker																											
Base case	**mSv/y**																										
Bone surface	Equivalent	7.1	6.9	6.7	6.5	6.3	6.1	5.9	5.7	5.6	5.4	5.2	5.1	4.9	4.8	4.6	4.5	4.4	4.2	4.1	4.0	3.9	3.8	3.6	3.5	3.4	3.3
Brain	Equivalent	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Effective (inc radon)	Effective	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.4	0.4
Liver	Equivalent	2.3	2.2	2.2	2.1	2.1	2.0	1.9	1.9	1.8	1.8	1.7	1.7	1.6	1.6	1.5	1.5	1.4	1.4	1.4	1.3	1.3	1.2	1.2	1.2	1.1	1.1
Pancreas	Equivalent	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Red marrow	Equivalent	1.4	1.3	1.3	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.0	1.0	1.0	0.9	0.9	0.9	0.9	0.8	0.8	0.8	0.8	0.8	0.7	0.7	0.7	0.7
Lungs (inc radon)	Equivalent	1.8	1.8	1.7	1.7	1.7	1.7	1.6	1.6	1.6	1.6	1.5	1.5	1.5	1.5	1.4	1.4	1.4	1.4	1.4	1.4	1.3	1.3	1.3	1.3	1.3	1.3
Whole chain	**mSv/y**																										
Bone surface	Equivalent	7.5	7.3	7.1	6.9	6.7	6.5	6.3	6.1	6.0	5.8	5.6	5.5	5.3	5.2	5.0	4.9	4.8	4.6	4.5	4.4	4.3	4.2	4.0	3.9	3.8	3.7
Brain	Equivalent	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Effective (inc radon)	Effective	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Liver	Equivalent	2.3	2.3	2.2	2.1	2.1	2.0	1.9	1.9	1.8	1.8	1.7	1.7	1.6	1.6	1.5	1.5	1.4	1.4	1.4	1.3	1.3	1.2	1.2	1.2	1.1	1.1
Pancreas	Equivalent	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Red marrow	Equivalent	1.4	1.4	1.3	1.3	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.0	1.0	1.0	0.9	0.9	0.9	0.9	0.8	0.8	0.8	0.8	0.8	0.7	0.7	0.7
Lungs (inc radon)	Equivalent	1.9	1.8	1.8	1.8	1.8	1.7	1.7	1.7	1.7	1.6	1.6	1.6	1.6	1.5	1.5	1.5	1.5	1.5	1.4	1.4	1.4	1.4	1.4	1.4	1.3	1.3

Table F.2 Effective and organ equivalent doses from current and future exposures (summed over 40 years)

Office worker	Dose, mSv
Bone surface	20
Brain	16
Effective (inc radon[a])	48
Liver	17
Pancreas	16
Red marrow	16
Lungs (inc radon)	279
Maintenance worker	
Bone surface	49
Brain	2
Effective (inc radon[a])	7
Liver	9
Pancreas	2
Red marrow	6
Lungs (inc radon)	33

(a) Radon in this context means radon-222 and radon-220

Table F.3 Office based staff member for *base* contamination scenario from exposure from 1950 over 40 years (assuming male and that exposure starts at age of 20 years)

					Rate fraction = (RR-1/RR)*100					
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0027	0.001	16.5	0.438	0.556	0.644	0.513	73.501	73.5
Pancreas	UNSCEAR 2006 ERR Solid	0.0043	0.001	13.14	0.429	0.476	0.49	0.937	73.501	73.5
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0046	0.001	13	2.108	2.796	3.301	0.148	73.501	73.5
Lung	UNSCEAR 2006 ERR INC Lung	0.5886	0.072	12.28	4.735	6.812	8.593	7.075	73.501	73.429
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.0203	0.004	18.31	6.543	5.962	3.284	0.607	73.501	73.497

Table F.4 Office based staff member for *whole chain* contamination scenario from exposure from 1950 over 40 years (assuming male and that exposure starts at age of 20 years)

					Rate fraction = (RR-1/RR)*100					
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0028	0.001	16.49	0.455	0.585	0.673	0.513	73.501	73.5
Pancreas	UNSCEAR 2006 ERR Solid	0.0045	0.001	13.13	0.444	0.5	0.513	0.937	73.501	73.5
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0047	0.001	12.99	2.146	2.856	3.382	0.148	73.501	73.5
Lung	UNSCEAR 2006 ERR INC Lung	0.5965	0.073	12.28	4.796	6.897	8.699	7.075	73.501	73.428
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.0215	0.004	18.24	6.862	6.272	3.476	0.607	73.501	73.497

Table F.5 Maintenance worker for the *base case* contamination scenario from exposure from 1950 over 40 years (assuming male and that exposure starts at age of 20 years)

					Rate fraction = (RR-1/RR)*100					
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0007	0.001	16.81	0.127	0.159	0.172	4.159	73.501	73.5
Pancreas	UNSCEAR 2006 ERR Solid	0.0012	0	13.37	0.124	0.136	0.132	0.937	73.501	73.501
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0063	0.001	13.1	2.993	3.837	4.4	0.148	73.501	73.5
Lung	UNSCEAR 2006 ERR INC Lung	0.1686	0.021	12.5	1.585	2.133	2.552	7.075	73.501	73.48
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.0223	0.004	19.25	7.681	6.668	3.424	0.607	73.501	73.497

Table F.6 Maintenance worker for the *whole chain* contamination scenario from exposure from 1950 over 40 years (assuming male and that exposure starts at age of 20 years)

					Rate fraction = (RR-1/RR)*100					
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0008	0.001	16.79	0.13	0.164	0.178	0.513	73.501	73.5
Pancreas	UNSCEAR 2006 ERR Solid	0.0012	0	13.35	0.128	0.14	0.137	0.937	73.501	73.501
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0063	0.001	13.1	3.001	3.848	4.411	0.148	73.501	73.5
Lung	UNSCEAR 2006 ERR INC Lung	0.1746	0.022	12.49	1.634	2.203	2.643	7.075	73.501	73.479
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.0226	0.004	19.23	7.767	6.77	3.478	0.607	73.501	73.497

Table F.7 Office based staff member for exposure from 2000 over 40 years (assuming male and that exposure starts at age of 20 years)

				Rate fraction = (RR-1/RR)*100						
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0014	0.001	16.18	0.224	0.292	0.343	0.513	75.239	75.238
Pancreas	UNSCEAR 2006 ERR Solid	0.0024	0	12.62	0.219	0.249	0.26	0.967	75.239	75.239
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0009	0	12.89	0.339	0.5	0.644	0.164	75.239	75.239
Lung	UNSCEAR 2006 ERR INC Lung	0.5346	0.06	11.27	4.451	6.434	8.149	6.491	75.239	75.179
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.0059	0.001	16.67	1.703	1.668	0.995	0.64	75.239	75.239

Table F.8 Maintenance staff for exposure from 2000 over 40 years (assuming male and that exposure starts at age of 20 years)

				Rate fraction = (RR-1/RR)*100						
Cancer	Model	REID %	LOLE (Y)	LOL if death occurs (y)	Age 50	Age 60	Age 70	Base risk %	Life exp (unexposed) (y)	Life exp (exposed) (y)
Brain	UNSCEAR 2006 ERR INC Brain	0.0001	0.001	16.18	0.022	0.029	0.034	0.513	75.239	75.239
Pancreas	UNSCEAR 2006 ERR Solid	0.0002	0	12.62	0.022	0.025	0.026	0.967	75.239	75.239
Liver mort	UNSCEAR 2006 ERR INC Liver	0.0005	0	12.89	0.19	0.281	0.362	0.164	75.239	75.239
Lung	UNSCEAR 2006 ERR INC Lung	0.0624	0.007	11.25	0.538	0.793	1.021	6.491	75.239	75.232
Leukaemia	UNSCEAR 2006 ERR Leukaemia	0.002	0	16.67	0.588	0.576	0.342	0.64	75.239	75.239