HEALTH RISKS FROM CONTAMINATION OF THE RUTHERFORD BUILDINGS, UNIVERSITY OF MANCHESTER

PROVISIONAL REPORT

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September 2009

FOREWORD

The risk assessment that is described in this report would not have been possible without the assistance of a large number of people. I am grateful to all who contributed in any way, including current and former staff and students of the University of Manchester, their families, and the University's management team. Special thanks are due to John Churcher, Neil Todd and Rachel Calam, all of whom devoted substantial time and effort to ensure that important information was not overlooked. Dr David Barker, Head of Compliance and Risk, served as my main point of liaison with the University, and at all times was helpful and constructive. I was also greatly assisted by expert input from teams at the Heath Protection Agency's Radiological Protection Division (in particular Jane Simmonds and Kelly Jones) and the Health and Safety Laboratory (in particular John Cocker and Andrew Easterbrook), with whom it was a pleasure to collaborate.

At the same time, I must make clear that the conclusions and recommendations presented in this report are mine, and do not necessarily reflect the views of these contributors.

David Coggon
September 2009

SUMMARY

This report summarises the findings from an independent inquiry that examined possible health risks from contamination of the Rutherford Buildings* at the University of Manchester. The inquiry was initiated at the request of Professor Alan Gilbert, President of the University, in response to concerns that cases of cancer among former occupants of the Buildings might be related to contamination by radioactive chemicals or mercury that had been used by Sir Ernest Rutherford and his colleagues in research carried out at the beginning of the 20th century. The investigation was conducted in collaboration with teams from the Health Protection Agency's Radiological Protection Division and the Health and Safety Laboratory, and was greatly assisted by input from current and former members of University staff.

Over the course of the investigation it became apparent that at least three cases of pancreatic cancer and two cases of brain cancer had occurred among people who had worked in the Rutherford Buildings. Also causing concern was a case of motor neuron disease. Review of the published scientific literature on these diseases confirmed that ionising radiation is an established cause of brain cancer in humans. Ionising radiation would also be expected to cause pancreatic cancer, although this has not been clearly demonstrable in epidemiological studies to date. Several studies have investigated a possible link between motor neuron disease and mercury, but findings have been inconsistent. There is no reliable epidemiological evidence that ionising radiation causes motor neuron disease. No indications were found of other known or suspected causes of the diseases that might have been encountered through work in the Rutherford Buildings.

Information about possible contaminants of the Rutherford Buildings was obtained from a history (compiled by Dr Neil Todd) of the research carried out by the Department of Physics during its occupation of the Buildings from 1900 to 1967; from a history (provided by the University) of the occupancy of the Buildings after 1967; from recollections offered by various current and former members of University staff; and by review of a) all retained reports of environmental surveys that had been carried out in the Buildings, b) risk assessments that had been carried out prior to remedial and construction projects, c) assessments that had been made of waste removed from the Buildings during remedial work, and d) archived records of the Health and Safety and Radiological Protection Committees.

This information indicated that between 1903 and 1919, the Department of Physics used radionuclides (radioactive forms of elements) from each of the three natural radioactive decay series (uranium-238, uranium-235 and throrium-232), and also substantial quantities of metallic mercury. Breakages and spillages were well documented. After 1919, when Rutherford left Manchester for Cambridge, taking his radioactive sources with him, the only new radioactive substances introduced into the Buildings appear to have been a small quantity of uranium used for research during World War 2, and possibly small quantities of tritium used by one or more medical departments during 1967-73. Apart from radionuclides and mercury, the only other hazardous contaminant identified was asbestos (mainly chrysotile, but also crocidolite and amosite), the presence of which had been documented in several environmental surveys.

* Taken to encompass all parts of what is now known as the Rutherford Building and adjoining buildings that were at one time occupied by the Department of Physics, and also their drains and an underground subway linking the Rutherford Building to the John Owens Building.

To gauge potential health risks from the identified contaminants, exposures were estimated for various reasonable worst-case scenarios, both historically and in the future. The main starting points for the exposure estimates were concentrations of the contaminants measured since 1999, both before and after remedial work to the Buildings. In addition, account was taken of the documented quantities of radionuclides that had been handled by the Department of Physics. Exposure estimates were collated with published data on the relation of relevant health outcomes to levels of exposure, to provide an assessment of risk.

The highest potential risk from contaminant radionuclides was that of lung cancer in long-term past occupants of the most polluted rooms of the Buildings. However, even after allowance for uncertainties in the assessment of historical exposures, the risk of lung cancer from past occupancy of the Buildings is expected to have been small (on average less than 60 excess deaths per 10,000 people, lower than this in non-smokers, and of similar order to the risk from passive smoking).

The excess risks of pancreatic and brain cancer from ionising radiation will have been substantially less than those for lung cancer (less than 1 excess death per 10,000 people).

Maximum potential risks of cancer from ionising radiation in future occupants of the Buildings, and in maintenance workers carrying out intrusive work on the fabric of the Buildings, were calculated to be lower than those in long-term past occupants.

It is unlikely that any harm to human health has occurred in the past 20 years, or will occur in the future, from mercury contamination of the Buildings. In the unlikely event that adverse effects did occur, (perhaps in an individual with relatively high exposures who was unusually susceptible) the impact would probably be minor (subtle cognitive changes and biochemical abnormalities in urine), and potentially reversible following cessation of exposure.

There is more uncertainty about risks from mercury contamination in earlier periods. However, any toxic effects from possibly higher exposures to mercury more than 20 years ago would have been present at the time, and would have tended if anything to resolve as exposures reduced.

Available measurements indicate that any exposure to asbestos through past occupancy of the Rutherford Buildings will have been extremely low, and the consequent lifetime excess risk of cancer in a long-term past occupant of the Buildings is estimated to be less than, and probably well below, 10 per 10,000.

Maintenance workers carrying out intrusive work have potential for higher exposures to asbestos. The main hazards that they face as a consequence of their exposure are lung cancer and pleural mesothelioma, and their risks of developing these diseases will be determined by their cumulative exposure to different forms of asbestos, including exposures from work at other locations as well as the Rutherford Buildings.

In general, any health risks from articles of furniture or furnishings that have been removed from the Rutherford Buildings will be trivial. Possible exceptions to this would be articles that were present in the Buildings before 1919, and articles that are visibly contaminated by mercury.

Theoretical considerations and the limited empirical data that are available suggest that there is no toxic interaction between the identified contaminants in the

Rutherford Buildings that could lead to elevations of risk importantly higher than those estimated for the contaminants individually.

On current evidence, none of the identified contaminants in the Rutherford Buildings could plausibly account for the cases of pancreatic cancer, brain cancer and motor neuron disease that have occurred among past occupants of the Buildings. In particular, the apparent cluster of pancreatic cancer cannot be explained by exposures to radionuclides, mercury or asbestos, either alone or in combination. By far the most likely explanation for the cluster is that it has occurred by chance coincidence.

Epidemiological research to clarify risks further is not a scientific priority. However, recommendations are made to explore further the chemical nature and origins of the mercury contamination in under-floor waste removed from the Rutherford Buildings during 2004-06, and also for limited additional monitoring of mercury levels in air. In addition, 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 control measures are needed to protect those involved in the work.

Given the low potential for risk, no form of health screening or other health intervention is recommended for people who may have been exposed to hazardous contaminants in the Rutherford Buildings.

1 BACKGROUND

This report summarises the findings of an investigation into possible health risks from historical contamination of buildings at the University of Manchester, which had been used by Sir Ernest Rutherford and his colleagues at the beginning of the 20th century in their pioneering research on the structure of the atom and the nature of radioactivity.

In 1999, a survey conducted by the University's Radiological Protection Service (RPS) revealed radioactive contamination in several rooms of what is now known as the Rutherford Building. At that time the Building was occupied in part by the Department of Psychology and in part by the Manchester Museum. Although the level of radiation was considered acceptable for occupancy of 40 hours per week, the RPS advised that it would be prudent to reduce staff and student occupancy of the two most contaminated rooms, and that in any future refurbishment of the floor areas of these two rooms, it would be necessary to ensure that airborne dust was not created.

Over the next four years, further localised surveys, some by RPS and some by external contractors, revealed foci of radioactive contamination in many rooms of the Building, and also in the Basement Room of what was then known as Coupland 2. These surveys were followed by remedial work to remove the contamination identified, although in at least one case, follow-up monitoring indicated that remediation had not been entirely effective, and further decontamination had to be undertaken. In the course of the remedial work, significant quantities of mercury were found on the plasterboard and lagging underneath the floorboards in several rooms.

In 2004-05, the contamination was addressed more systematically and comprehensively, with the aim of removing radiological restrictions on future development and occupation of the Building. This exercise entailed first taking out furniture, shelving and floor coverings ("soft stripping") so that an extensive radiological survey could be undertaken. Then, following initial decontamination of areas that were readily accessible (e.g. the upper surfaces of contaminated floorboards), floorboards and underlying insulation material were systematically removed ("hard stripping"), and the exposed joists were vacuum cleaned. In the course of the work, it was noted that the under-floor contamination by mercury was widespread.

Not surprisingly, these activities caused some consternation among the occupants of rooms that had to be vacated for remedial work. Questions were raised about the exact nature and level of the contamination, and about possible risks to the health of people working in the rooms that were affected. The concerns were set out formally in a report by three members of staff (two recent and one current) from the Department of Psychology (the Churcher report) [Churcher et al, 2008]. As well as highlighting the widespread contamination that had been found in the building, the authors drew attention to the occurrence of cancer in two former colleagues, who had occupied contaminated rooms, and asked whether there might be a causal link. They concluded by calling for an independent review of the risks to past and future occupants of the Building, and of arrangements for protecting their health.

In response, the President of the University, Professor Alan Gilbert, invited me to lead the investigation that is described in this report.

2 TERMS OF REFERENCE

After consultation with various interested parties, it was agreed that the inquiry would address four questions:

i) Is there a material risk to the health of current or future occupants of the Rutherford Buildings from contamination by radioactive materials, and if so, what action is required to address this risk?

ii) Could there be a material risk to health from future intrusive work undertaken on the fabric of the Rutherford Buildings, and if so, what action is required to address this possibility?

iii) Is there any material risk to health from contamination of furniture, furnishings and other articles that have been moved from the Rutherford Buildings to other places?

iv) Could contamination of the Rutherford Buildings by radioactive or chemically hazardous materials contribute (or have contributed) materially to disease incidence or mortality in people who have worked in the Buildings in the past, and if so, what advice should be given to people who have worked in the Buildings in the past?

In this context, the term "Rutherford Buildings" was used to encompass all parts of what is now known as the Rutherford Building and adjoining buildings that were at one time occupied by the Department of Physics, and also their drains and an underground subway linking the Rutherford Building to the John Owens Building.

3 METHOD OF INVESTIGATION

3.1 Scientific strategy

To address the terms of reference, two complementary lines of investigation were pursued.

3.1,1 Diseases of special concern

First, a review was carried out of diseases that had occurred among occupants of the Rutherford Buildings, which stakeholders were concerned might be linked to hazardous contaminants. Two of the diseases (pancreatic and brain cancer) had been highlighted in the Churcher report. In addition, concerns about motor neuron disease emerged from correspondence and discussion with a former member of staff who had worked in the Manchester Museum. The review focused on the epidemiological features of these disorders, their pathogenesis (the pathological processes by which they are thought to arise), and their known and suspected causes. Special attention was paid to causes that might have been encountered through work in the Rutherford Buildings.

3.1.2 Identification of contaminants and assessment of associated risks

In parallel with the review of diseases of special concern, an attempt was made to identify all potentially relevant contaminants that had been present in the Rutherford Buildings, and to assess the risks to health that they posed.

The Churcher report had referred to several radioactive pollutants that had been detected in the Buildings, and to the presence of mercury under floorboards. However, there was a possibility of further unrecognised hazards, and a systematic search was therefore made for evidence of other contaminants.

Once a list of all potentially relevant contaminants had been compiled, their toxicity was reviewed. The aim was to identify and characterise the adverse health effects that they might produce, and the ways in which the risk (probability) of these adverse effects relates to levels and routes* of exposure.

Next, an assessment was made of the levels of exposure to the pollutants that could have occurred during the past 60 years, and of the levels of exposure that might be expected in the future. Various exposure scenarios were considered, including normal occupancy of the Buildings, the performance of maintenance work including some intrusive tasks on the fabric of the Buildings, and living in a house to which articles of furnishing or furniture had been transferred from the Buildings. The cut-point of 60 years was chosen because it would cover the large majority of people who had worked in the Buildings and who were still alive. Moreover, estimates of earlier exposures would be less reliable.

Finally, the estimates of potential exposure were collated with the evidence on risk by levels of exposure to produce assessments of possible risks to health. These assessments did not address the specific circumstances of any named individual. Rather, as a first tier exercise, they considered what would be a reasonable worst-case estimate of risk for each exposure scenario. The option was retained to derive more realistic person-specific assessments where the first tier analysis did not rule out the possibility of material risks to health.

3.1.3 The place of epidemiological investigation

While note was taken of cases of disease that were known to have occurred among current and former occupants of the Rutherford Buildings, no attempt was made to ascertain the morbidity and mortality of occupants systematically, or to evaluate their disease experience epidemiologically. A number of current and former members of staff advocated such investigation, but it would have added substantially to the time needed for the inquiry, and I concluded that it should only be pursued if the initial lines of investigation described above indicated that it would be useful.

It was possible that systematic ascertainment would identify further cases of the diseases that were of particular a priori concern (e.g. pancreatic cancer). However, given what was already known, and the work that was already being undertaken, the discovery of additional cases of disease would not have impacted materially on the initial conduct or interpretation of the investigation. This was because a detailed search was already being carried out for possible hazardous exposures in the Buildings. If, after thorough investigation, no hazardous exposure could be found that could plausibly explain a cluster of, say, pancreatic cancer, then the observation of additional cases would not alter the conclusion that the cluster was unlikely to have been caused by a feature the Buildings or of the work that was carried out in them.

If, on the other hand, a known or suspected cause of pancreatic cancer were identified in the Buildings, then assessment of the level of risk to occupants would be

* Possible routes of exposure to chemicals include inhalation, ingestion, and absorption through in tact skin. Exposure to ionising radiation may be from external sources or through uptake of a radioactive chemical into the body.

determined by collating estimated levels of exposure with what was known from elsewhere about the relation of risk to levels of exposure. For established health hazards, an epidemiological study of staff who had worked in the Buildings was unlikely to add importantly to the evidence from elsewhere on risk by levels of exposure, since the number of people studied would be relatively small (hundreds rather than thousands), and the findings would therefore be subject to substantial statistical uncertainty.

This is illustrated in Table 1, which shows expected numbers of deaths from various causes in a "cohort" of 2,876 workers that was followed for mortality over a period of up to 45 years [Coggon et al, 2003]. The numbers were calculated with the assumption that the cohort had the same sex- and age- and calendar period-specific death rates as the general population of England and Wales.

Table 1 Expected numbers of deaths in a population of 2,876 men and women followed over up to 45 years

Cause of death	Expected number of deaths
All causes	607.6
All cancers	184.2
Lung cancer	51.8
Pancreatic cancer	7.8
Cancer of brain and nervous system	5.3

The exact numbers of deaths that would be expected among past occupants of the Rutherford Buildings would depend not only on the number of men and women who had worked in the Buildings, but also on when they had started such work, and how old they were at the time. Nevertheless, given the much smaller size of the Rutherford Buildings population, we can be confident that the expected numbers of deaths by cause would be substantially less than in Table 1.

The main value of an epidemiological study of past occupants would be to refine risk estimates if the initial assessment for a pollutant indicated that there could be elevations of risk that would be detectable epidemiologically. The design of such a study would then need to be tailored to the health effect of interest. The case for further research of this sort is discussed further in Section 7.1.

3.2 Implementation

Implementing the scientific strategy that has been described depended on input and assistance from various parties.

3.2.1 Input from stakeholders

From the outset, I tried to ensure that the inquiry was a collaborative effort to which all stakeholders could contribute. This was important because the investigation needed to identify and address everyone's concerns, and to take on board all relevant information that stakeholders could provide. To this end, I first consulted on the terms of reference for the inquiry with a broad constituency of individuals, including John Churcher and his co-authors, other current and former members of staff, trades union representatives, University management, the Health and Safety Executive, and the Coroner for Manchester (City) District, who was investigating the death of a former member of staff.

When they had been finalised, the terms of reference were posted on an openly accessible section of the University website that was dedicated to the inquiry [http://www.manchester.ac.uk/rutherfordreview/], together with other relevant material that accrued over the course of the investigation. In addition, John Churcher placed the terms of reference on a website that he had established to provide information about the Rutherford Buildings and their investigation.

To disseminate knowledge of the inquiry more widely, I briefed the media, which generated publicity on television, in national and local newspapers, and in the scientific press. In addition, John Churcher and a current member of the Psychology Department, Rachel Calam, kindly undertook to pass on news of the investigation to those colleagues who had left the Department to work elsewhere, for whom they could find contact details.

As a result of this activity, I received inquiries and information from former students, members of staff and their families in many parts of the UK, and also overseas.

Over the course of the inquiry, I and the colleagues who were assisting me in the investigation visited the University to discuss our plans and interim findings with current and former members of staff and their families. Four plenary presentations were made (in October and December 2008, and March and September 2009), in addition to which, there were opportunities for private meetings with individual stakeholders who wanted one.

Several individuals were especially generous in the time and effort that they gave to assist with the collection and sifting of relevant information. In particular, John Churcher was assiduous in checking archived records held by the University, and Neil Todd produced an invaluable history of Rutherford's work and of activities in the Department of Physics in Manchester after Rutherford's departure in 1919 [Todd, 2008].

3.2.2 External consultants

Early in the course of the inquiry, it became clear that the investigation would be greatly assisted by additional input from experts in ionising radiation and in occupational exposure to mercury. On my advice, the University therefore engaged the Health Protection Agency's Radiological Protection Division (HPA RPD) to help with the assessment of exposures to, and risks from, ionising radiation, and the Health and Safety Laboratory (HSL) to undertake a similar role in relation to mercury.

The HPA RPD and HSL teams both worked closely with me. With the support of the University, and after opportunity for input from other stakeholders, we agreed their terms of reference which were then published on the University website. They have produced their own reports [Oatway et al, 2009; Jones et al, 2009; Rowbotham et al, 2009], which complement the assessment presented in this report, and provide more detail of the underpinning scientific evidence.

4 DISEASES OF SPECIAL INTEREST

Three diseases were identified as being of special interest. The Churcher report had noted the death from pancreatic cancer of a former member of staff in the Department of Psychology who had worked in the Rutherford Building. By the time the inquiry began, a second case was known to have occurred, and subsequently, I was contacted by a third former staff member (now deceased), who had developed

the disease. The Churcher report also drew attention to the occurrence of brain cancer in an ex-member of staff, and before the inquiry began, a second case had been identified. In addition, one former member of staff contacted me early in the course of the inquiry because he had motor neuron disease, and was concerned that it might have been caused by contaminants in the Rutherford Buildings. While no other cases of motor neuron disease were identified, it is a rare disease, and was therefore felt to merit special consideration.

4.1 Pancreatic cancer

Pancreatic cancers are malignant tumours that arise from glandular cells in the pancreas. In England and Wales, there are approximately 13 new cases per 100,000 population per year. The disease is slightly more common in men than in women, and its annual incidence rises steeply with age in both sexes from about 14 per 100,000 at age 55-59 years to 60-70 per 100,000 at 75-79 years. When it occurs, the disease is almost always fatal, and overall, it is responsible for about 1.4% of all deaths nationally.

Like all malignant tumours, pancreatic cancer is thought to result from a series of critical mutations in the DNA of a cell line, which eventually leads the cells to replicate in an uncontrolled manner, invading surrounding tissues and spreading (metastasising) to other parts of the body. Thus, in theory, causal agents might act by causing damage to DNA and inducing mutations (genotoxicity), increasing rates of cell turnover in the pancreas (so that over a given time there is more opportunity for spontaneous mutations to occur during cell division), or inhibiting the body's defence mechanisms that detect and repair damaged DNA or remove abnormal cells,

The best established causes of pancreatic cancer are genetic predisposition, smoking, chronic pancreatitis and diabetes [Ghadirian et al]. Several specific forms of inherited susceptibility to cancer carry an increased risk of tumours in the pancreas, although these are uncommon, and familial occurrence of the disease is rare. Smoking is responsible for a larger number of cases, causing a doubling of risk on average, with higher risks in heavier smokers. Chronic pancreatitis (long-term inflammation of the pancreas) also leads to an approximate doubling of risk, and it appears that risk is particularly high when the pancreatitis results from an inherited genetic susceptibility. Among people with this genetically determined form of pancreatitis, some 40% may eventually develop malignant pancreatic tumours. Clinically diagnosed diabetes has been shown consistently to predispose to pancreatic cancer, risk again being approximately doubled.

In addition to these established causes, various suspected dietary determinants of pancreatic cancer have been investigated. Increased risks have been reported with higher consumption of meat, dairy products and fat, and with lower consumption of fresh fruit and vegetables [Ghadirian et al]. However, these epidemiological findings are not yet sufficiently strong and consistent for firm conclusions. Obesity is another suspected cause [Michaud, 2004].

Epidemiological studies of people exposed to ionising radiation have rarely indicated associations with pancreatic cancer [Ron, 1998], although there are exceptions. For example Smith and Doll [1981] found increased mortality from pancreatic cancer in a follow-up study of British radiologists; Sont and colleagues [2001] reported a significant increase in the incidence of pancreatic cancer with increasing exposure to ionising radiation among a cohort of male radiation workers in Canada (although the incidence of the disease in the cohort as a whole was less than would have been expected from rates in the general population); and in a large population of

underground miners, there was a statistically significant increase in risk of the disease with higher cumulative exposure to radon [Darby et al, 1995]. In addition, analysis of mortality among employees at the Oak Ridge nuclear plant in Tennessee indicated an excess mortality from cancer of the pancreas (34 deaths were observed where 25 would have been expected from death rates in the general population), although this was not statistically significant [Loomis and Wolf, 1996]. As well as low dose internal alpha radiation, some workers in this study had been exposed to other agents, including mercury.

Against these positive findings, other studies have failed to demonstrate a relationship between pancreatic cancer and ionising radiation, and when the totality of evidence was reviewed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) in 2006, their conclusion was that "there is little, if any, evidence for associations between pancreatic cancer and radiation dose" [UNSCEAR, 2008]. Nor was any statistically significant relation apparent in the most important study that has been reported since the UNSCEAR review – a dose-response analysis based on approximately 175,000 radiation workers in the UK [Muirhead et al, 2009].

Thus, while ionising radiation would be expected to cause pancreatic cancer through its well established capacity to damage DNA, the pancreas appears to be less susceptible than other organs such as the lung and bone marrow, making any elevation of risk difficult to detect.

No excess of pancreatic cancer was reported in two studies of workers with relatively high exposures to mercury – one in the Norwegian chloralkali industry [Ellingsen et al, 1993], and the other in mercury miners from four European countries [Boffetta et al, 1998].

4.2 Brain cancer

Brain cancers mostly arise from glial cells that support the nerve cells in the brain. Statistics on their incidence and associated mortality are liable to error because tumours that develop in other organs and spread secondarily to the brain via the blood stream may sometimes be misclassified as primary brain tumours. However, because clinical investigation has become more intensive, this is probably less of a problem now than in the past.

In 2006, the overall incidence of brain cancer in England and Wales was 8.4 per 100,000 per year in men and 5.8 per 100,000 per year in women. Incidence increases with age, although not as steeply as for pancreatic cancer. Most malignant brain tumours are eventually fatal, and in total, they account for about 0.7% of all deaths nationally.

In broad terms, the pathological mechanisms that underlie the development of brain cancer are thought to be similar to those for other cancers, involving the accumulation of critical mutations in the DNA of a cell line.

There is evidence that brain cancer occasionally results from inherited mutations of DNA, but otherwise the only well established cause of the disease is high dose ionising radiation [Schwartzbaum et al, 2006; Davis, 2007]. In particular, there is strong and consistent evidence of elevated risk following childhood exposure of the head to radiation for the treatment of other diseases [Kleinerman, 2006]. However,

as reported by HPA RPD in their Task 1 report, currently available data on risks from lower levels of exposure are limited and equivocal [Oatway et al, 2009].

Many other environmental factors have been investigated as possible causes of brain cancer. They include various infectious agents, head trauma, dietary factors, radiofrequency and power frequency magnetic fields, and a number of chemicals [Wrensch et al, 2002]. Intriguingly, there is a growing body of evidence that brain tumours occur less frequently than normal in people who suffer from "atopic" allergic diseases such as asthma, eczema and hay fever [Schwartzbaum et al, 2006].

Among the possible chemical causes of brain cancer that have been studied is mercury. As already described, the population of workers employed at the nuclear plant in Oak Ridge, Tennessee included some who were exposed to mercury. An analysis of mortality among 2,133 white males who were exposed to mercury vapour at the plant found no significant elevation of mortality from cancers of the brain and central nervous system, although there was an excess in a comparison group of workers who were not exposed to mercury [Cragle et al, 1984]. A study of workers exposed to mercury at eight Swedish chloralkali plants showed a small excess of brain cancers (3 observed as compared with 1.1 expected) that was not statistically significant [Barregård et al, 1990], but there was no excess in a study of Norwegian chloralkali workers [Ellingsen et al, 1993], and a study of some 7000 mercury miners and millers in Spain, Italy, Slovenia and Ukraine found no increase in brain tumours [Boffetta et al, 1998]. As described by HSL in their report, authoritative bodies such as the International Agency for Research on Cancer (IARC) and the Agency for Toxic Substances and Disease Registry (ASTDR) have concluded that there is no sound evidence that mercury causes any form of cancer in humans [Rowbotham et al, 2009].

4.3 Motor neuron disease

Motor neuron disease (also known as amyotrophic lateral sclerosis) is a progressive neurological disorder characterised by degeneration of neurons (nerve cells) that are involved in the control of movement. It affects neurons in the brain and spinal cord, and in the peripheral nerves that connect the brain and spinal cord to muscles. In the UK, there are approximately two new cases per 100,000 population per year, rates in men being approximately 60% higher than in women. The disease is extremely rare before late middle age, incidence peaking at age 75-79 years. Overall, it is responsible for about 0.3% of all deaths nationally.

The pathological mechanisms underlying motor neuron disease are uncertain. Possibilities include toxicity from unusually high concentrations of amino acid neuromodulators that are normally present in the nervous system, and oxidant stress by free radicals [Mitchell and Borasio, 2007]. However, neither of these has been firmly established.

Genetic predisposition is thought to underlie familial clustering that is observed in 5-10% of cases, but no environmental causes of motor neuron disease have been clearly identified [Mitchell and Borasio, 2007]. Among the possible environmental causes that have come under suspicion are heavy metals, including mercury [Roos et al, 2006]. Two case reports have been published describing patients who developed motor neuron disease following unusually high exposure to mercury [Praline et al, 2007] and accidental injection of metallic mercury [Schwarz et al, 1996]. Moreover, experimental exposure of mice to mercury at a concentration of 500 $\mu g/m^3$ for four hours caused damage to nerve cells of the type that can be affected in motor neuron disease [Stankovic, 2006]. However, studies comparing

motor neuron disease patients with unaffected controls have not found significantly higher levels of mercury in the blood [Moriwaka et al, 1993; Pamphlett et al, 2001] or spinal cord [Pamphlett and Waley, 1998]. In summary, therefore, while a link between mercury and motor neuron disease cannot be ruled out, current evidence for a causal role is unconvincing. If there were a causal relationship, the risk would be expected to be small at low levels of exposure.

5 HAZARD IDENTIFICATION AND RISK ASSESSMENT

5.1 Methods

5.1.1 Identification of contaminants

Various sources of information were explored in an effort to ensure that all potentially relevant hazardous contaminants of the Rutherford Buildings were identified.

- Neil Todd, one of the authors of the Churcher report, produced a carefully researched history of experimental work that had been carried out in the Department of Physics, which included details of the radioactive materials used during different time periods. From this it was possible to deduce various contaminants that might have been present as a consequence of spills or breakage of apparatus.
- University management provided a summary history of the occupancy of the Rutherford Buildings after the Department of Physics moved to new premises in 1967. This gave a picture of the type of work that was likely to have been carried out in the Buildings since that time, and the potential for further contamination during this more recent period.
- A number of current and former members of staff offered recollections of work that had been carried out in the Rutherford Buildings at different times.
- University management were asked to provide copies of all retained reports of environmental surveys that had been carried out in the Buildings, of risk assessments that had been carried out prior to remedial and construction projects, of assessments that had been made of waste removed from the Buildings during remedial work, and of the archived records of the Health and Safety and Radiological Protection Committees. These were searched for mention of potentially hazardous contaminants.

5.1.2 Exposure assessment

The assessment of exposures to identified contaminants was based principally on data from past environmental surveys, on new measurements that were made specifically for the purpose of the current inquiry, and on the history of reconstruction and remediation that had been carried out in the Buildings at different times in the past. In addition, in the case of ionising radiation, account was taken of the known rates at which radioactive forms of elements (radionuclides) decay spontaneously to form new elements, and a check was made that exposure estimates were consistent with the level of contamination that might realistically be expected to have occurred, given the nature of the work carried out historically with radioactive chemicals, and the quantities of these materials that were handled. For some radionuclides, no satisfactory environmental measurements were available, but it was possible to make reasonable upper estimates for exposures using information on the quantities of source materials that had been handled.

5.1.3 Identification of health hazards and assessment of risks

The adverse health effects that might result from exposure to identified contaminants, and the ways in which risks of these outcomes relate to levels of exposure, were established from the published scientific literature. Where possible, information was sought from recent, authoritative reviews, but account was taken also of papers describing primary research that post-dated the latest reviews, and of studies that were of special relevance to the inquiry (e.g. on combined exposure to ionising radiation and mercury). The findings were then collated with those on levels of exposure to produce reasonable "upper bound" assessments of risk for relevant exposure scenarios.

5.2 History and potential contamination of the Rutherford Buildings

Originally known as the New Physical Laboratories, the Rutherford Building was opened in 1900, and extended by construction of an Annex in 1912. At that time, it was occupied by the Department of Physics, which began its research on radioactivity in 1903. Various radionuclides were used during the first two decades of the twentieth century, as well as large quantities of elemental mercury.

The Department of Physics, which from 1951 included a sub-department of Astronomy, continued to occupy the premises (renamed in 1945 the Schuster Laboratory and Extension) through to 1967. However, after 1919, the focus of research changed, and with one minor exception during the war years (see below), there appears to have been no further use of radioisotopes during this tenure. Nor has evidence been found of other activities during this later era that could have given rise to persistent hazardous contamination.

During the period from 1967 to 1973, several Departments were located in the Building and its extension at one time or another, including Physiology, Pharmacology, Medical Biochemistry, Medical Computing and Zoology. It is possible that some of the work in the Building at this time entailed the use of radioactive tritium. In 1968, the main part of the Rutherford Building together with the 1912 extension was renamed Coupland 1.

From 1973, a part of the Buildings was occupied by the Department of Psychology, and the remainder by the Manchester Museum. Neither of these departments is thought to have used materials that would cause significant long-term contamination.

The name of the main Building was changed to "Rutherford" in 2006, since when, parts of it have been used to house Student Recruitment, Admissions and the International Development Division. No contamination would be expected from the activities of these groups.

Past environmental surveys indicate that asbestos has been present in the fabric of the Rutherford Buildings. However, records of environmental monitoring do not suggest any persistent contamination from activities carried out in the Building other than that which has already been described.

In summary, therefore, the identified contaminants of concern are radionuclides, mercury and asbestos. The risks to health from these substances are examined in the sections that follow.

5.3 Ionising radiation

5.3.1 Sources of exposure

Historical research by Todd [2008] indicates that during the period from 1903-19, the Department of Physics used radionuclides from each of the three natural radioactive decay chains – uranium-238, uranium-235 and thorium-232. As well as work with higher members of the decay chains such as radium-226 and radon-222, there is evidence that preparations of radon decay products, including lead-210, were handled [Todd 2008].

After 1919, when Rutherford left Manchester for a new position at the University of Cambridge, the research using radioisotopes was discontinued, and Rutherford took his radioactive materials with him to Cambridge. During World War 2, a small quantity of uranium (probably no more than a few grams) was used by the Department of Physics. In addition, it is possible that small quantities of tritium (a radioactive isotope of hydrogen) were used in the Rutherford Buildings between 1967 and 1973. Otherwise, however, there is no historical evidence of other radioactive materials having been used in the Buildings after 1919.

The findings from monitoring for radioactive contamination are consistent with this historical information. The contaminants identified have been radium-226, radon-222, lead-214, bismuth-214, lead-210, actinium-228, lead-212 and bismuth-212. In one room (then known as C1.10), the activity of lead-210 was substantially higher than that of its parent radionuclides such as radium-226, a finding indicative of primary contamination by lead-210 that accords with reports that experiments were carried out with lead-210 preparations. No evidence of tritium has been found in monitoring, but because of its loss by evaporation (most tritium is chemically combined with oxygen in water) and radioactive decay, detectable levels would not be expected after an interval of several decades.

The methods and scope of the monitoring that has been conducted are such that we can be reasonably confident that no other sources of contamination have been present during the last six decades.

5.3.2 Health hazards

HPA RPD have reviewed the health hazards posed by radioactive contamination in the Rutherford Buildings [Oatway et al, 2009]. At the relatively low levels of exposure that could have occurred in the last 60 years, the main adverse effect of concern is an increased risk of various cancers. This occurs through damage to DNA with the induction of mutations.

Other disorders, such as cardiac, respiratory and digestive disease have been reported to occur at increased rates in Japanese atomic bomb survivors, and higher rates of cardiac disease have been described following medical exposures to radiation. However, it is unclear what pathological mechanisms underlie these observations, or whether there is any risk at lower levels of exposure.

In theory, ionising radiation would also be expected to cause congenital abnormalities through its ability to induce mutations of DNA, and there is evidence that this does occur in experimental animals. However, epidemiological research to date has failed to provide direct evidence of such effects in exposed humans, and it appears that if there is a hazard of congenital abnormalities in humans then the risk must be small.

As described in the HPA RPD Task 1 report, there is no reliable epidemiological evidence to support the hypothesis that ionising radiation causes motor neuron disease [Oatway et al, 2009].

5.3.3 Risk in relation to exposure

The excess risk of cancer from ionising radiation depends upon the type of radiation, the dose received by each organ, and the sensitivity of the exposed tissue (tissues with higher turnover of cells, such as the bone marrow, tend to be more vulnerable).

With regard to the contamination in the Rutherford Buildings, the exposures that might impact materially on risk are to gamma radiation from radioactive sources outside the body, and to radiation, especially by alpha particles, from sources that have been taken into the body. Uptake of sources into the body could occur through inhalation of radon gas or of fine particles of contaminated dust, or through ingestion of contaminated dust on food or by hand-to-mouth transfer (e.g. when smoking).

The cumulative dose of external gamma radiation to a specified organ will depend on the level of radioactivity in external sources, their distance from the organ, and the time period over which exposure occurs. Dose falls off rapidly as distance from the source extends.

The dose from internal sources of radiation will depend on the amount and chemical form of each radionuclide that is taken into the body, and its biokinetics (how efficiently it is absorbed, where it goes once it is inside the body, and the extent to which it is metabolised and excreted). Once they are in the body, radionuclides will continue to irradiate tissues until they are excreted or have decayed. Thus, dose calculations must take into account the exposure to tissue that will accumulate over a lifetime (the committed dose). The International Commission on Radiological Protection (ICRP) has developed standard models by which to estimate the doses to specified organs following intake of many radionuclides, and HPA RPD has used similar models for radon.

Any dose of ionising radiation from contamination of the Rutherford Buildings would be superimposed on background exposures to natural sources of radiation (UK annual average 2.2 mSv) and medical exposures for diagnosis (e.g. radiological examinations) and treatment (e.g. radiotherapy).

Evidence on the risk of cancer from specified doses of radiation to different organs comes principally from studies of atomic bomb survivors in Japan and of people exposed through medical uses of radiation. In addition, some information is available from studies of occupational exposure and of residential exposure to radon in buildings.

Some evidence relates directly to radionuclides that were present in the Rutherford Buildings. For example, exposure to high doses of radium-226 has been shown to cause bone cancer in workers applying luminous paint to dials, and there are indications that lower exposures used in the treatment of ankylosing spondylitis have caused leukaemia. Radon (principally radon-222) has been shown to cause lung cancer, both in underground miners and in people with higher levels of the gas in their homes. And thorium (principally thorium-232) has been found to cause liver cancer and leukaemia in patients who were exposed through its use as a radiological contrast medium. Other evidence concerns exposure to ionising radiation from other sources, but can nevertheless be extrapolated to exposures of the type that could have occurred from contamination of the Rutherford Buildings.

Using such data, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has developed specific risk models for selected organs, including the lung, bone marrow and brain. For example, a single acute dose of 100 mSv is estimated to increase the risk of brain cancer by 0.035% over and above that expected in the absence of radiation exposure. For other organs, including the pancreas, a generic risk model has been produced.

These models provide the best available basis on which to estimate the risks of cancer that could have occurred from radioactive contamination of the Rutherford Buildings.

5.3.4 Assessment of exposures

Occupants

To assess potential health risks to past occupants of the Rutherford Buildings, it was assumed as a "reasonable worst-case" scenario (Scenario 1) that:

- An office-based member of staff had occupied a room in the Buildings for 2000 hours per year over the 40-year period from 1950-1989.
- That in the room, a patch of the floorboards with a total area equivalent to a circle 1 metre in diameter was contaminated by radionuclides.
- That this patch of contamination was immediately under the staff member's chair.
- That when working in the office, the staff member spent all of his/her time in this chair.
- That the office also contained a small hotspot of contamination by lead-210 at high activity concentration, of the type that had been identified in two places in Room C1.10. This hotspot was assumed to be a circular area of wall with diameter 10 cm, located at a distance of 30 cm from the body of the staff member when he/she was sitting in his/her chair.
- That 5 cm beneath the patch of contaminated floorboards, there was a circular layer of dust, 1 metre in diameter and 0.5 cm thick, presumed to have originated in part from abrasion of radioactively contaminated surfaces, including the hotspot of activity.
- That the staff member was exposed to ionising radiation through each of:
    - External irradiation by gamma rays arising from radionuclides in the contaminated patch on the floor, in the hotspot on the wall, and in the radioactively contaminated dust under the floor.
    - Inhalation of radon gas and of radionuclides in dust. The inhalation rate was taken as 1.2 m^3 per hour, and the total concentration of dust in air as 10^{-5} g per m^3. The former is a standard rate used by HPA RPD for moderate activity, and the latter, again a standard value used by HPA RPD, is based on a review of monitoring data carried out over recent decades [Jones et al, 2009].
    - Ingestion of radionuclides in dust formed by abrasion of contaminated surfaces, including the hotspot on the wall. The rate of dust ingestion was taken as 1 mg per hour (a standard value used by HPA, as described in a paper by Smith and Jones [2003]).

To derive an upper bound for possible exposure in this scenario, the following assumptions were made (as per the "whole chain" source term in the HPA RPD report [Jones et al, 2009]):

- In radioactively contaminated floorboards, and in dust formed from them, all radionuclides from a given natural decay chain (uranium-235, uranium-238 or thorium-232) were assumed to be present in 2000 with the same level of activity

(except where branching in the relevant decay chain led to a deviation). In the case of the uranium-238 and thorium-232 decay chains, the level of activity was taken as the maximum that had been recorded for any radionuclide in the decay chain in any measured dust sample. For radium-226, this gave the value that would have been expected if approximately 1% of the radium that was known to have been used by Rutherford and his colleagues [Todd, 2008] had been spilt across a total floor area equivalent to a 1 metre diameter circle in each of 20 rooms. As no activity levels had been measured for the uranium-235 decay chain, the level of activity for radionuclides in this series was derived from the quantity of actinium-227 that was thought to have been used by Rutherford. This quantity was taken from data reported by Neil Todd [2008], and the calculation assumed that the proportion of actinium-227 that was lost through spillage was the same as was estimated for radium-226 (i.e. about 1%). The activity level for a radionuclide contaminating a surface (in Bq cm^{-2}) was taken to have the same numerical value as its activity in the surface material, and in dust derived from it, in Bq g^{-1}. The justification for this assumption lay in the ratio of surface activity to activity in dust for radium-226 in two sets of monitoring data from 2000 [Jones et al, 2009].

With rounding up, this method gave values for surface and dust contamination as set out below.

Decay chain	**Surface contamination (Bq cm^{-2})**	**Dust contamination (Bq g^{-1})**
Uranium-235	10	10
Uranium-238	200	200
Thorium-232	1	1

- In the hot spot of contamination on the wall, it was assumed that in 2000, lead-210 and its progeny all had an activity 5000 Bq g^{-1}, and an activity by area of 5000 Bq cm^{-2}. This was based on monitoring data for the two patches of contamination on the wall of Room C 1.10 (rounded up to the next order of magnitude). It was further assumed that there was no contamination by radionuclides higher up the decay chain. This approach was adopted because the activity of lead-210 that was recorded in the hotspots in Room C1.10 was around 100-fold higher than that of predecessor decay chain radionuclides such as radium-226 and bismuth-214, and it therefore seemed clear that the lead-210 in the hotspots arose from primary contamination rather than through radioactive decay of precursor radionuclides.

- In years before 2000, the activities of radionuclides with long half-lives (> 100 years) were assumed to be the same as in 2000, since any decay over 40 years would have been relatively minor. Radionuclides with short half-lives (< 10 years) could only plausibly have arisen through primary contamination by a longer lived parent radionuclide (given what was known about the sources used by Rutherford), and were assumed in each year to have the same level of activity as their most immediate longer lived parent radionuclide. It was possible, however, that some of the radionuclides with intermediate half-lives (e.g. lead-210) that were present in 2000, had resulted in part from primary contamination by the radionuclide in question, rather than from decay of precursors higher in the relevant decay chain. Therefore, the assumed historical activities of these radionuclides were adjusted upwards to account for possible loss by decay over

time. To be sure of obtaining an upper bound estimate of activity, the adjustment was made for all of the radionuclide assumed to be present in 2000. Further details of the calculation are given in the HPA RPD report [Jones et al, 2009].

- The fraction of total inhaled/ingested dust that originated from the patch of floor contamination was assumed to be10%, and the fraction arising from the hotspot on the wall 0.1%.

- The activity concentration of radon-222 in the air of the room throughout 1950-2000 was taken as 180 Bq m^{-3}, which is the level that would be predicted from the highest measured activity concentration of radium-226, and slightly higher than any measured concentration of radon. In the absence of major changes to the Buildings, no important decline in radon-222 concentrations in room air would have been expected over the period 1950-2000.

- The activity concentration of radon-220 (thoron) in the air of the room throughout 1950-2000 was taken as that which would be expected from the highest measured activity concentration of actinium-228. This gave an activity concentration in air at head height of 50 Bq m^{-3}.

- The exposure to external gamma radiation in a given year was taken as that which would occur at a point 30 cm horizontally from the hotspot on the wall and 1 metre vertically above the patch of contamination on the floor, given the levels of radionuclides that were assumed to be present in these areas of contamination, and under the floor, in the year concerned.

To derive an upper bound estimate for future occupants of the Buildings, a similar scenario was considered to that for past occupants, except that the individual was assumed to be first exposed in 2000, and then to be exposed at the same level for the next 40 years (i.e. with no reductions from radioactive decay or removal of radioactive contaminants).

For external irradiation, a dose rate of 2 x 10^{-4} mSv per hour was assumed, this being at the upper end of external dose rates measured in 2001.

For exposures by inhalation and ingestion, the activity by surface area in the floor contamination was taken as 1 Bq cm^{-2} (higher than any level that had been measured since 2005), with a corresponding activity by mass of 1 Bq g^{-1}. These values were assigned to all members of the three natural radioactive decay chains. Any hotspots of contamination on walls were assumed to have been removed by the remediation in 2004-06.

For radon-222 (radon) and radon-220 (thoron) the same activity in air concentration was assumed as prior to remediation – i.e. 180 Bq m^{-3} for radon and 50 Bq m^{-3} for thoron.

This scenario for a future occupant is termed Scenario 2.

Intrusive work

The scenario that was considered in the assessment of risks from intrusive work on the fabric of the Buildings related to a hypothetical maintenance worker who carried out various tasks within the Buildings, most of which would create higher levels of dust than during normal occupancy, and some of which would generate substantial dust levels.

Thus, in estimating possible past exposures (Scenario 3), it was assumed that an employee had worked in the Buildings for 220 hours per year during each of the 40 years from 1950 to 1989. Over the time that he was in the Buildings, the worker was assumed to have exposures to external radiation identical to those in the office worker scenario (i.e. from nearby patches of radioactive contamination on the floor and wall). For most of the time, the ingestion rate of dust was taken to be the same as was assumed for an office worker, and the airborne concentration of inhalable dust tenfold higher (i.e. 10^{-4} g per m^3). However, it was assumed that for 20 hours per year, the worker undertook tasks such as drilling or sanding that produced even higher levels of dust, leading to inhalation of an airborne concentration of 10^{-3} g per m^3, and ingestion of an additional 10 mg per hour. The breathing rate assumed was the same as for the office worker, as were the concentrations of radon-222 and radon-220 in air, the levels of radioactivity in surface patches and dust, and the fractions of dust originating from the contaminated patches on the floor and wall.

To assess risks from intrusive work carried out in the future, the same scenario was assumed, but with the individual first exposed in 2000, and then continuing to be exposed at the same level for the next 40 years (i.e. with no reductions from radioactive decay or removal of radioactive contaminants). Assumed levels of external irradiation, surface and dust contamination, and radon-222 and radon-220 were the same as in the risk assessment for future exposure of an office worker. Again, any hotspots on walls were taken to have been eliminated by remediation. This is denoted Scenario 4.

Articles removed from Building
From the ways in which radioactive contamination of the Rutherford Buildings is thought to have occurred (principally breakage of equipment and spillage prior to 1920), no important contamination would be expected of furniture or furnishings that were first placed in the Buildings after 1920. This is supported by the absence of detectable radioactivity in articles removed from the Buildings that have been monitored by HPA RPD. It thus seems reasonably certain that any exposure to ionising radiation from furniture or furnishings taken from the Buildings would be less than that which could occur from long-term occupancy of the Buildings as an office worker.

5.3.5 Assessment of risk

The risks of cancer from the exposure scenarios set out in Section 5.3.4 were estimated using the relevant UNSCEAR risk models (see Section 5.3.3). The main index of risk that was derived was the “Risk of Exposure-Induced Death” (REID), which is the lifetime risk that an individual will die from a cancer of the type under consideration as a consequence of his/her radiation exposure. Also calculated were “baseline risk” – i.e. the lifetime risk that an individual will die from the cancer in question in the absence of any radiation exposure, and the “loss of life expectancy if death occurs”. The latter measures the average years of life that would be lost by a person who died from a radiation-induced cancer.

For each exposure scenario, the employee was assumed to be a male exposed over a period of 40 years, starting at age 20 years. Lifetime risk for a woman exposed in the same way would be slightly higher for some cancers and slightly lower for others, but overall would be very similar. Lifetime risks for people first exposed at older ages would be lower since on average they would have a shorter future lifetime in which risks could be expressed.

It was further assumed that all of the committed dose of radiation from inhalation or ingestion of radionuclides was incurred in the year in which intake occurred. This will have tended to overestimate risks since in reality, some of the radiation to tissues would not occur until later.

Occupants
For an office-based member staff exposed between 1950 and 1989 in the circumstances specified in Scenario 1, the total committed effective dose of radiation over the period of exposure would be 74.6 mSv. This corresponds to an average of 1.9 mSv per year. To put the figure in context, the average annual dose to a member of the UK population from all sources of radiation, both naturally occurring and artificially generated is 2.7 mSv. Further details of the derivation of this total dose, and of the doses to specific organs, are given in the HPA RPD report [Jones et al, 2009]. The main source of the exposure in 2000 would be inhalation of radon-222 and radon-220 (about 50% of the dose), with exposure from external radiation contributing about 42%.

Table 2 summarises the risks of cancer in various organs that would result from this exposure.

Table 2. Risks of cancer associated with exposures to ionising radiation according to Scenario 1 (an office-based worker employed from 1950 to 1989)

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.94	0.004	13.1
Brain	0.51	0.003	16.5
Liver	0.15	0.005	13.0
Lung	7.05	0.596	12.3
Leukaemia	0.61	0.021	18.2

As indicated in Table 2, the largest 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, on average, approximately 60 in every 10,000 people so exposed would die from a radiation-induced lung cancer (in non-smokers the risk would be lower). This compares with a baseline risk of dying from lung cancer in the general population of about 7% (700 per 10,000). The average years of life lost in an individual who died of lung cancer as a consequence of his exposure would be approximately 12 years.

Next highest is the risk of leukaemia, but with a much lower REID of only 0.021% (less than 3 in every 10,000 deaths).

For pancreatic and brain cancer, the risks are even lower. The REID for pancreatic cancer is 0.004% (less than 1 in every 10,000 deaths), corresponding to slightly less than 0.5% of the baseline risk, while that for brain cancer is 0.003% - approximately 0.6% of the baseline risk.

With regard to exposures from occupation of the Building in the future, the committed effective dose of radiation during 2000-2039 in Scenario 3 (including background radiation) would be 47.7 mSv, and the doses to all organs would be less than in

Scenario 1. It follows that the risks of cancer mortality would also all be less than for Scenario 1.

Intrusive work
For a maintenance worker carrying out intrusive work (Scenarios 2 and 4), ingestion and inhalation of dust would be more significant pathways of exposure than for an office-based worker, but cumulative exposure to external irradiation would be lower (because the duration of exposure was less). Thus, while the total committed effective dose during 1950-89 in Scenario 2 (37.4 mSv) is less than for Scenario 1, some of the doses to specific organs are higher.

Table 3 summarises the risks of cancer mortality in Scenario 2. Risks of liver cancer and leukaemia are slightly higher than for scenario 1, but those for lung, brain and pancreatic cancer are all lower. Risks for Scenario 4 would all be lower than for Scenario 2.

Table 3. Risks of cancer associated with exposures to ionising radiation according to Scenario 2 (a maintenance worker employed from 1950 to 1989)

Cancer	Baseline risk (%)	Risk of Exposure-Induced Death (%) (REID)	Loss of life expectancy if death occurs (years)
Pancreas	0.94	0.001	13.3
Brain	0.51	0.0008	16.8
Liver	0.15	0.006	13.1
Lung	7.05	0.174	12.4
Leukaemia	0.61	0.022	19.2

Articles removed from Building
Given the small potential risks to long-term occupants of the Rutherford Buildings, no significant risk would be expected from radioactive contamination of articles that had been removed from the Buildings and taken to someone's home, provided that the articles were not present in the Buildings before 1920. This is because, as described in Section 5.3.4, exposures from such articles will be relatively trivial.

5.4 Mercury

5.4.1 Sources of exposure

Mercury was an important component of the experimental apparatus used by Rutherford and his colleagues, and was handled in substantial quantities [Todd, 2008]. As would be expected, spills and breakages occurred from time to time, and this caused deposits of mercury to accumulate, especially under floorboards. During renovation work in 2000 and subsequently, quantities of metallic mercury were found beneath the floorboards in many rooms of the Rutherford Buildings. In addition, mercury vapour was detected in the drains from the Buildings. Attempts were made to remove the under-floor mercury during the hard stripping exercise in 2005. However, it is clear that this was not entirely successful, and environmental measurements early in 2009 showed continuing presence of mercury vapour in two rooms (2.62 and 2.63).

Analysis of the waste removed from under the floors in 2005 indicates that a major part of the mercury content was in the form of one or more compounds, which unlike

metallic mercury, only dissolved slowly in 20% nitric acid [communication from Andrew Frith at IRAS Ltd]. It is unclear whether these compounds formed by chemical reaction of spilt metallic mercury, or whether they were a contaminant in manufacture of the cotton insulation material that was used under the floors of the Buildings.

5.4.2 Health hazards

The toxicity of metallic mercury has been investigated quite extensively through epidemiological studies in humans as well as by animal experiments. As described in the HSL report [Rowbotham et al, 2009], the scientific evidence generated has been reviewed at intervals by various reputable national and international bodies, without any major controversies in its interpretation.

Acute toxicity can occur from extremely high exposures of the sort that may arise, for example, when mercury is heated, but there is no reason to expect that such high levels of exposure would at any stage have resulted from contamination of the type that occurred in the Rutherford Buildings. Nor have any reports of acute mercury poisoning been found in the minutes of the University's Health and Safety Committee, or in any other of the University records that were reviewed.

The main concern, therefore, is the possibility of chronic toxicity from long-term lower level exposures, which will have occurred principally through inhalation of mercury vapour (uptake into the body of any highly insoluble mercury compounds that were present in inhaled or ingested dust would be expected to be low). As HSL have described, the organs most susceptible to toxicity from chronic inhalation of mercury vapour are the nervous system (including the brain) and the kidney [Rowbotham et al, 2009].

Neurotoxicity

Neurotoxic effects that have been attributed to mercury include:

- Tremor, initially affecting the hands but sometimes spreading to other parts of the body
- Emotional lability
- Insomnia
- Headaches
- Memory loss and cognitive defects
- Muscle twitching, weakness and wasting
- Numbness and tingling from damage to peripheral nerves

Some of these effects are quite challenging to investigate. Complaints such as headache, insomnia, difficulty with memory and tingling sensations are subjective and occur quite commonly in the general population. Thus, biases may arise from differences in people's awareness of and propensity to report symptoms. Other abnormalities, such as poor performance on tests of cognitive function, may be influenced by the effort that subjects make when being tested, and by other variables that could confound differences between people with and without exposure to mercury. Nevertheless, it is clear that mercury is an important neurotoxicant.

The exact mechanisms by which mercury causes neurotoxicity are as yet unclear, although it is well established that in its elemental form, the metal is able to pass from the blood stream into the brain and central nervous system. Moreover, once it has reached the brain, it can remain there for many years after last exposure.

There is no evidence that neurotoxic effects of mercury can emerge for the first time, or continue to deteriorate, after exposure has ceased. On the contrary, they tend if anything to resolve once exposure has stopped. However, there can still be residual impairment of function many years after a person was last exposed.

The effects of mercury on the kidney are manifest first by biochemical changes such as the presence of proteins in the urine. Symptomatic renal failure occurs only with higher exposures. The toxicity is thought to arise through interaction of mercury ions with thiol groups in proteins, peptides and amino acids – in particular, albumin, metallothionein, glutathione and cysteine [Rowbotham et al, 2009]. Reversal of the urinary excretion of proteins has been observed following cessation of exposure.

In addition to the toxic effects of mercury on the nervous system and kidney, there is some evidence that higher long-term exposures can adversely affect other bodily systems. Effects suggested by individual studies include elevation of blood pressure, increased mortality from heart disease and stroke, biochemical abnormalities of blood cells, and disorders of the menstrual cycle. However, these findings have not been consistently replicated, and the links to mercury can only be regarded as speculative. Overall, most epidemiological studies of reproductive toxicity, have not indicated adverse effects, either in men or in women.

The potential of mercury to cause cancer was reviewed by the International Agency for Research on Cancer (IARC) in 1993, with a conclusion that there was "inadequate evidence" of a cancer hazard in humans or experimental animals [IARC, 1993]. New primary research published since 1993 has not materially altered the balance of evidence on carcinogenicity. The most informative studies are those of occupational populations with relatively high exposures to mercury – chloralkali workers and mercury miners, and these have not indicated clear and consistent elevations of risk for cancer overall or for any specific type of cancer. Moderate excesses of lung cancer in Swedish [Barregård et al,1990] and Norwegian [Ellingsen et al, 1993] cohorts of chloralkali workers may have been caused by concomitant exposure to asbestos, while increased mortality from lung cancer in a cohort of American mercury miners seems to have been driven largely, if not completely, by confounding exposures to silica [Amandus and Costello, 1991]. In a European study of mercury miners, mortality from lung cancer was 19% higher than expected, but the increase in risk was limited to two of the four countries in which the miners worked [Boffetta et al, 1998]. As described in Section 4.2, studies of workers with relatively high exposures to mercury in the chloralkali and mercury-mining industries have not indicated any hazard of pancreatic or brain cancer.

The evidence linking mercury with motor neuron disease has been described in Section 4.3. A link between mercury and motor neuron disease cannot be ruled out, but current evidence for a causal relationship is unconvincing.

5.4.3 Risk in relation to exposure

In the extensive evidence base that is now available, toxic effects of mercury on the nervous system and kidney have not been demonstrable from long-term exposures giving rise to urinary levels of mercury less than 20 µmol/mol creatinine (approximately equivalent to 40 µg/L). On this basis, HSE has set a biological monitoring health guidance value of 20 µmol mercury/mol creatinine (35 µg mercury/g creatinine) in urine [Health and Safety Executive, 1995], while more recently, the equivalent German regulatory authority, DFG, adopted a biological tolerance value of 25 µg mercury/g creatinine [DFG, 2005]. The possibility cannot be

excluded that a small minority of individuals who are unusually vulnerable to the toxic effects of mercury, e.g. because of genetically determined differences in metabolism, might develop adverse effects at lower exposures than the values specified by HSE and DFG. However, if such effects do occur, they are most likely to be minor – subtle abnormalities on neuropsychological testing and increased urinary excretion of various proteins.

Other possible manifestations of toxicity, such as cardiovascular and haematological effects, would not be expected from exposures insufficient to cause toxicity to the nervous system or kidneys.

The HSE health guidance value of 20 µmol mercury/mol creatinine in urine is roughly what would be expected from long-term exposure during working hours to an airborne concentration of 25 µg/m^3 mercury.

5.4.4 Assessment of exposures

Occupants and intrusive work

As described in the HSL report [Rowbotham et al, 2009], the best starting point for assessment of historical exposures to mercury is the set of measurements in the Coupland 1 Building that was made by Casella Winton in 2004, supplemented by under-floor measurements in the Manchester Museum that were made by Diamond Environmental in 2000. These measurements are unlikely to underestimate importantly environmental concentrations over the past ten to twenty years since:

a) The conditions under which the measurements were made were fairly typical in terms of ambient temperature and wind speed.
b) There is no evidence that significant quantities of mercury were removed from the Rutherford Buildings before 2004.
c) Deposits of mercury would not have diminished importantly through evaporation or chemical reaction during the 10-15 years before the measurements were made.
d) There are no reports or records of refurbishment or structural rearrangements between 1990 and 2004 that could have altered ventilation and airflow in a way that significantly reduced airborne concentrations of mercury in rooms.

In the Casella Winton 2004 survey, one room (2.52) had recorded mercury concentrations of 10.7 µg/m^3 and 4.9 µg/m^3. Three other rooms (2.62, 2.63 and 2.53) had measured concentrations between 2 and 6 µg/m^3. All other measurements were less than 2 µg/m^3. Under-floor concentrations of mercury in the Manchester Museum survey by Diamond Environmental were roughly an order of magnitude lower than those subsequently recorded in Coupland 1, making it unlikely that levels in room air would have been higher than the highest value recorded in Coupland 1.

Although a maintenance worker who lifted floor boards or worked close to crevices in the floor might temporarily be exposed to relatively high concentrations of mercury, the highest cumulative exposures would be expected in a member of staff who spent prolonged periods (the equivalent, say, of 8 hours per day, five days per week, 48 weeks per year over 40 years) in a room with the highest concentration of mercury. From the reasoning set out above, the highest average concentrations of mercury in any room since 1980 are likely to have been less than 15 µg/m^3.

There is, however, some uncertainty about possible levels of mercury in earlier times. If the mercury compound(s) in the waste that was removed from under floorboards was formed by chemical reaction of spilt metallic mercury, then it is possible that

larger quantities of metallic mercury, and therefore higher levels of mercury vapour, were present in the past.

During the refurbishment carried out in 2004-06, substantial quantities of mercury were removed from under-floor spaces. This, and any changes to airflow because of the building work, mean that the most reliable guide to potential exposure of future occupants of the Buildings is a set of measurements made by HSL in 2009. This survey covered a total of 114 rooms in the Rutherford Building, Psychology Annex and non-public areas of the Museum, and the highest concentration recorded was 12 $\mu g/m^3$ in room B58 of the Museum. Ten other rooms had concentrations between 2 and 10 $\mu g/m^3$. Again, therefore, no worker in the Rutherford Buildings would be expected to incur a long-term average exposure higher than 15 $\mu g/m^3$, and for most workers, exposures would be well below this figure.

Articles removed from Building
In the absence of visible deposits of mercury, no significant exposure would be expected from contaminated articles that had been removed from the Rutherford Buildings.

5.4.5 Assessment of risk

Occupants and intrusive work
Toxicological evaluation indicates that adverse health effects would not be expected from long-term exposures to mercury during a standard working week at airborne concentrations less than 20 $\mu g/m^3$. The highest long-term exposures among people working in the Rutherford Buildings, either in the past 20 years, or in the future, are very unlikely to exceed 15 $\mu g/m^3$ and in most cases will be well below this level. Thus, it is unlikely that any harm to human health has occurred recently, or will occur in the future, from mercury contamination of the Buildings. In the unlikely event that adverse effects did occur, (perhaps in an individual with relatively high exposures who was unusually susceptible) the impact would probably be minor (subtle cognitive changes and biochemical abnormalities in urine), and potentially reversible following cessation of exposure. Any toxic effects from possibly higher exposures to mercury more than 20 years ago would have been present at the time, and would have tended if anything to resolve as exposures reduced.

Articles removed from Building
In the absence of visible deposits of the metal, no mercury toxicity would be expected from contaminated articles that had been removed from the Rutherford Buildings.

5.5 Asbestos

5.5.1 Sources of exposure

Archived reports held by the University (Appendix 1) clearly document the presence of asbestos – principally chrysotile, but also amosite and crocidolite – in the Rutherford Buildings. For example, in surveys carried out in 1990-91, asbestos was detected in pipe lagging on several floors; in 1998, it was found in the outer lining of a furnace in the Conservation Laboratory of the Museum; and in 2005, asbestos board was noted to be present on the rear of doors in the Cohen Room. At various stages, work was carried out to remove asbestos that had been identified.

5.5.2 Health hazards

The adverse health effects of asbestos arise when small fibres of the mineral are inhaled. Several diseases can result from such exposure, the most important and firmly established hazards being asbestosis (in which the lungs become scarred and stiff, leading to breathlessness), pleural thickening (thickening of the membrane that lines the outer surface of the lung, which again can produce breathlessness), lung cancer, and pleural and peritoneal mesothelioma (malignant tumours of the membranes that line the lungs and the abdominal cavity). As with all toxins, the risk of adverse outcomes depends on the level of exposure. At relatively low exposures, the main risk is from lung cancer and pleural mesothelioma (which often does not develop until thirty or more years after first exposure). Asbestosis and peritoneal mesothelioma are normally associated only with rather higher exposures. There are also differences in the toxicity of different forms of asbestos, crocidolite and amosite tending to be more damaging than chrysotile.

5.5.3 Risk in relation to exposure

The index of exposure to asbestos that is thought to be most relevant to disease risk is cumulative exposure, which is measured in units of (fibre/ml).years. A cumulative exposure of 1 (fibre/ml).year is equivalent to that received by a worker who breathes air containing an average concentration of 1 asbestos fibre per ml over the course of a year (assuming a standard working week and annual holidays). Extensive epidemiological research has been carried out on exposure-response relationships for asbestos-related disease, and in 2000, Hodgson and Darnton from the Health and Safety Executive reviewed the available evidence to derive summary estimates of risk [Hodgson and Darnton, 2000]. For a cumulative exposure of 0.1 (fibre/ml).years, they estimated the excess risks of mesothelioma as 10 deaths per 10,000 exposed for crocidolite, 1.5 deaths per 10,000 exposed for amosite and insignificant for chrysotile. For lung cancer, the corresponding estimates were 0.4 per 10,000 exposed for crocidolite and amosite, and insignificant for chrysotile. Allowing for uncertainties in the data and methods, the highest plausible excess risks for chrysotile were 0.4 deaths per 10,000 exposed for mesothelioma and 1 per 10,000 exposed for lung cancer.

5.5.4 Assessment of exposures

Results are available from a series of surveys between 1987 and 1996, in which airborne concentrations of asbestos were measured at various locations in the Rutherford Buildings (Appendix 1). In all but four cases, levels were below the limit of detection of 0.01 fibres/ml. The exceptions were two samples from the attic above the postgraduate room on 11 November 1990, which had concentrations of 0.01 and 0.02 fibres/ml, and two samples from the tunnel between the Plant Room and the Coupland Building on 25 February 1996, which had concentrations of 0.03 and 0.18 fibres/ml. In the case of the attic above the postgraduate room, repeat samples on the next day had concentrations < 0.01 fibres/ml.

5.5.5 Assessment of risk

Occupants

The available measurements indicate that any exposure to asbestos through past occupancy of the Rutherford Buildings will have been extremely low. Even for a member of staff who was based in the Buildings for 40 years, cumulative exposure will have been < 0.4 (fibre/ml).years. Given that much, if not most, of the asbestos that was present in the Buildings was chrysotile, this implies a lifetime excess risk of cancer of less than, and probably well below, 10 per 10,000. To put this figure in context, environmental tobacco smoke (passive smoking) has been estimated to cause some 3000 deaths per year among non-smokers in the United States [EPA,

1993], which, given that there are some 2.4 million deaths annually in the USA (including deaths in smokers), corresponds to an average lifetime excess risk of more than 12.5 per 10,000 in all non-smokers.

Over the last three decades, some if not most of the asbestos that was originally present in the Buildings has been removed. Thus, provided any remaining asbestos is managed appropriately, with care to ensure that materials containing the mineral are in good condition and not generating dust, and that there are effective controls on exposure if the asbestos is disturbed during building work, the exposures and risks from future occupancy should be even lower than those that could have occurred in past occupants.

Intrusive work
Potentially more important than the risks to occupants, are those to construction and maintenance workers who carry out intrusive work on asbestos materials (e.g. drilling, sawing or grinding) that has the potential to generate much higher fibre concentrations. Although the exposure during any single task may be relatively short in duration, construction workers can be exposed to asbestos repeatedly over the course of their careers. This is evident in elevated death rates from mesothelioma nationally among several construction trades including carpenters, electricians and plumbers. Thus it is important that the University continue to manage carefully any maintenance or construction tasks that are carried out in situations where asbestos might be present, whether in the Rutherford Buildings or elsewhere.

Articles removed from Building
Articles removed from the Rutherford Buildings could give rise to asbestos exposure elsewhere, either if they themselves were made from asbestos, or if they were contaminated by asbestos at the time they were taken from the Buildings. The former seems an unlikely scenario, since the asbestos-containing materials that have been identified in the Buildings have been structural rather than in furniture or equipment. Any contamination of removed objects will have been limited by the relatively low levels of asbestos in the Buildings and would not be expected to cause significant long-term pollution of new places to which they are taken.

5.6 Combined exposures

In the course of the inquiry, several current and former members of staff asked whether there might be toxic interactions between the contaminants that were present in the Rutherford Buildings, and in particular, whether combined or sequential exposure to ionising radiation and mercury might carry a higher risk to health than the sum of any effects that would be expected if the two exposures occurred in isolation.

In recent years, there has been much interest in the possibility of enhanced toxicity from combined exposures to environmental agents (so-called “cocktail effects”). Examples of toxic interactions between chemicals, especially medicines, have been well documented. They can occur because one substance modifies the absorption, distribution (within the body), metabolism or excretion of another, because two substances act on the same biochemical or disease process, or because one substance impairs the body’s mechanisms for protecting against or repairing the damage caused by another. Interactions are also known to occur between ionising radiation and chemical exposures. For example, antioxidants such as melatonin protect against damage to tissues caused by ionising radiation [Vijayalaxmi, 2004]. However, to date, synergistic effects from combined exposures to chemical and/or physical agents have not been clearly demonstrated where the level of exposure to

each agent individually was well below that which would cause toxicity were it encountered in isolation.

The enormous number of possible combinations that can be constructed from even just a few hazardous agents means that it is impossible to generate direct empirical data on combined toxicity for other than a small minority of combined exposures that are of special interest. In the absence of direct data, assessment of combined toxicity is based on deductions from knowledge of the toxicity of agents in isolation, and their modes of action.

One series of epidemiological studies has examined patterns of mortality among employees at the Oak Ridge nuclear plant in Tennessee, some of whom were exposed to both ionising radiation and mercury. In an analysis that focused particularly on a cohort of 2131 men who were exposed to mercury, total mortality from cancer was less than would have been expected from rates in the general population, and lower than in a comparison group of workers from the same plant who were unexposed to mercury [Cragle et al, 1984]. Mortality from lung cancer in the mercury-exposed group was 34% higher than in the general population, but similar to that in the unexposed comparison group. There were only a small number of deaths from cancers of the brain and nervous system, but rates in the workers exposed to mercury were lower than in the comparison group.

Otherwise, no toxicological or epidemiological studies have been found that explored the combined effects of ionising radiation and mercury specifically.

The main target organs and toxic modes of action of ionising radiation and mercury have been summarised in Sections 5.3.2 and 5.4.2, and in the parallel reports by HPA RPD [Oatway et al, 2009] and HSL [Rowbotham et al, 2009]. At low levels of exposure, such as were encountered in the Rutherford Buildings, ionising radiation causes harm by disrupting chemical bonds in DNA, producing genetic mutations, which can in some cases lead eventually to the development of cancer. The organs most at risk of cancer from the exposures in the Rutherford Buildings are the lung, bone marrow and liver. Mercury is most toxic to the nervous system and kidney. Its exact mode of action in the brain is uncertain, but in the kidney, it acts by binding to thiol groups in proteins, peptides and amino acids.

From this understanding of the modes of action, there is no reason to expect unusual synergistic effects from the combination of exposures to ionising radiation and mercury that occurred in the Rutherford Buildings. Nor do the limited empirical data that are available from the Oak Ridge studies suggest any important toxic interaction.

Apart from ionising radiation and mercury, the only other potentially important environmental pollutant that has been identified in the Rutherford Buildings is asbestos. In theory, asbestos and ionising radiation might interact in the production of mesothelioma or lung cancer. However, even if relative risks* were as much as multiplicative, at the low levels of exposure that occurred in the Buildings, the absolute increase in the risk of these diseases would be trivial.

* Relative risk is the ratio of disease risk in people with a given exposure to that in people who do not have the exposure (or in people who are exposed at some other specified level).

6 CONCLUSIONS

6.1 Sources of uncertainty

Inevitably, the assessments of risk that have been made are subject to various uncertainties. The uncertainties stem in part from the limitations of current scientific knowledge, but also from a dearth of historical data on levels of contaminants in the Rutherford Buildings and the absence of firm information on building and maintenance work that was carried out in the Buildings before 1999.

Of the uncertainties that could have caused risks from ionising radiation to be underestimated, perhaps most important is the possibility of maintenance and construction work between 1950 and 1999 that reduced levels of radioactive contamination. It appears that there was no major re-building or alteration during this period, but if, for example, floorboards were sanded before varnishing or fitting of new floor coverings, this might have removed some surface contamination from the boards. Even in the most extreme case, however, it seems unlikely that such activities could have led potential historical exposures to ionising radiation to be underestimated by as much as a factor of five.

Other uncertainties that might have caused risks to be underestimated include the possibility of still unidentified sources of radioactive contamination in the Buildings, and higher rates of spillage of actinium-227 than were assumed in the exposure calculations.

Against this, the risk assessment for ionising radiation incorporated multiple assumptions that would have tended to inflate risk estimates. These include, for example, the assumptions: that a worker occupied a room in the building for 40 consecutive years (in practice the longest occupancy is likely to have been less than this); that his chair was situated immediately above an unusually large patch of floor contamination, and immediately next to a hotspot of contamination on the wall; and that all radionuclides in each decay chain were present in these patches of contamination at the highest levels that had been measured anywhere for any individual radionuclide from the chain. Conservative assumptions of this type will have tended to counter any underestimation of risk because of failure to allow for undocumented and unrecalled maintenance work.

Most important with regard to the risk assessment for mercury is the uncertainty about the exact chemical form of much of the mercury that was present in the waste removed from under the floorboards when the Rutherford Buildings were refurbished during 2004-06. Depending on the origin of this non-metallic mercury, exposures more than 20 years ago may have been higher than those estimated for more recent periods.

The conclusions and recommendations that are presented in this section take account of these various sources of uncertainty.

6.2 Risks from identified pollutants

The inquiry has identified three hazardous contaminants in the Rutherford Buildings – radionuclides, mercury and asbestos.

The highest potential risk from contaminant radionuclides is that of lung cancer in long-term past occupants of the most polluted rooms of the Buildings. However, even after allowance for uncertainties in the assessment of historical exposures, the risk of lung cancer from past occupancy of the Buildings is expected to have been

small (less than 60 excess deaths per 10,000 people, lower than this in non-smokers, and of similar order to the risk from passive smoking).

The excess risks of pancreatic and brain cancer from ionising radiation will have been substantially less than those for lung cancer (each less than 1 excess death per 10,000 people).

Maximum potential risks of cancer from ionising radiation in future occupants of the Buildings, and in maintenance workers carrying out intrusive work on the fabric of the Buildings, are calculated to be lower than those in long-term past occupants.

It is unlikely that any harm to human health has occurred in the past 20 years, or will occur in the future, from mercury contamination of the Buildings. In the unlikely event that adverse effects did occur, (perhaps in an individual with relatively high exposures who was unusually susceptible) the impact would probably be minor (subtle cognitive changes and biochemical abnormalities in urine), and potentially reversible following cessation of exposure.

There is more uncertainty about risks from mercury contamination in earlier periods. However, any toxic effects from possibly higher exposures to mercury more than 20 years ago would have been present at the time, and would have tended if anything to resolve as exposures reduced.

Available measurements indicate that any exposure to asbestos through past occupancy of the Rutherford Buildings will have been extremely low, and the consequent lifetime excess risk of cancer in a long-term past occupant of the Buildings is estimated to be less than, and probably well below, 10 per 10,000.

Maintenance workers carrying out intrusive work have potential for higher exposures to asbestos. The main hazards that they face as a consequence of their exposure are lung cancer and pleural mesothelioma, and their risks of developing these diseases will be determined by their cumulative exposure to different forms of asbestos, including exposures from work at other locations as well as the Rutherford Buildings.

In general, any health risks from articles of furniture or furnishings that have been removed from the Rutherford Buildings will be trivial. Possible exceptions to this would be articles that were present in the Buildings before 1919, and articles that are visibly contaminated by mercury.

Theoretical considerations and the limited empirical data that are available suggest that there is no toxic interaction between the identified contaminants in the Rutherford Buildings that could lead to elevations of risk importantly higher than those estimated for the contaminants individually.

6.3 Explanations for observed patterns of disease

The risk assessment that has been carried out indicates that none of the identified contaminants in the Rutherford Buildings could plausibly account for the cases of pancreatic cancer, brain cancer and motor neuron disease that have occurred among past occupants of the Buildings.

Ionising radiation is an established cause of brain cancer, and is likely also to cause pancreatic cancer (although a hazard of pancreatic cancer has not as yet been demonstrable epidemiologically). However, the relative risks associated with the

highest exposures that could plausibly have resulted from work in the Buildings are less than 1.03 (i.e. less than a 3% increase in risk). Furthermore, if there were an unrecognised hazard of cancer from mercury, the expected relative risk from exposures such as those incurred in the Rutherford Buildings would be small (since no risk has been detectable in epidemiological studies of chloralkali workers and mercury miner with higher exposures). Similarly, the relative risk for any unrecognised hazard of motor neuron disease from ionising radiation or mercury would be expected to be small, since no excess of the disease has been clearly apparent in epidemiological studies of workers with higher exposures to these agents.

If an exposure increases the risk of disease by a relative risk, R, then for every 100 x R exposed cases that develop the disease, 100 would have been expected to do so even in the absence of the exposure, and 100 (R-1) as a consequence of their exposure. Thus, the probability that a case of the disease in an exposed person is attributable to the exposure is 100 (R-1)/(100 x R) = (R-1)/R. It follows that even to attribute disease to exposure on the balance of probabilities (more likely than not), (R-1)/R must be greater than 0.5, and therefore R must be greater than 2. The maximum relative risks from hazardous exposures in the Rutherford Building are calculated to be well below this threshold.

The question then remains as to why some diseases, in particular pancreatic cancer, have occurred more frequently than would be expected among past occupants of the Rutherford Buildings, and in particular of certain more contaminated rooms. Given what is known, we can be reasonably confident that the apparent cluster of pancreatic cancer is not explained by exposure to radionuclides, mercury or asbestos, either alone or in combination. No other significant hazardous contaminants of the Buildings have been identified. Nor are there any known or suspected causes of pancreatic cancer that could explain the cluster. In these circumstances, by far the most likely explanation for the cluster is that it has occurred by chance coincidence.

7 RECOMMENDATIONS

7.1 Further investigation of risk

Given the conclusions that it has been possible to reach in this risk assessment, epidemiological research to clarify risks further is not a scientific priority. Even after allowance for uncertainties, we can be reasonably confident that any health risks from contamination of the Rutherford Buildings have been small, and that risks in the future will be even smaller.

Assuming that adequate records of employment were available, it would be possible to conduct a retrospective epidemiological study of mortality and cancer incidence among current and former members of staff who have worked in the Buildings. An investigation of this sort could provide fuller information on patterns of disease that have occurred in members of staff, but because the population studied would be small, it would not be expected to shed much light on why the observed patterns of disease had occurred. Nor would it help much in refining estimates of future risk. For these reasons, such a study is not recommended.

It would, however, be prudent to explore further the chemical form and origin of the mercury contamination in the waste removed from the Rutherford Buildings during the refurbishment carried out in 2004-06. In particular, it would help to establish

whether the non-metallic mercury that is present in the waste is likely to have resulted from chemical reaction of spilt mercury that collected under the floorboards, or to have been a contaminant of cotton insulation material when it was originally installed. Possible lines of investigation include further chemical analysis of waste samples, and checks to see whether any other buildings in the University that were constructed near the same time as the Rutherford Building contain similar under-floor insulation, and if so, whether it too contains mercury.

7.2 Further monitoring and decontamination

Recent monitoring indicates that the remedial work carried out between 1999 and 2006 did not remove all of the contamination by mercury from the Rutherford Buildings, and may have left some low level contamination by radionuclides.

Further work has since been initiated to address the residual contamination by mercury. After completion of this work, repeat environmental monitoring for mercury should be carried out in the rooms concerned and in those adjacent to them (since changes in under-floor airflow associated with the remedial work might alter levels of mercury in adjacent rooms). In addition, it would be prudent to carry out further monitoring of mercury levels in air in those rooms, which in the most recent HSL survey, had airborne concentrations of mercury in excess of 4 $\mu g/m^3$. The purpose would be to check that the measured values were not unrepresentatively low, with higher levels at other times of year. Thus, this additional monitoring (both post-remediation and in those rooms with measured concentrations above 4 $\mu g/m^3$) should be carried out on four occasions at three-monthly intervals over the course of a year. If concentrations at some times of the year are found to be close to 20 $\mu g/m^3$, options for mitigation should be considered.

Currently, there is no reason to recommend biomonitoring for mercury in any members of staff, although a clinical decision to do so might be made if a staff member were particularly anxious about his or her recent exposure and wanted further reassurance (urinary excretion reflects only exposure in recent weeks). In addition, if future environmental monitoring indicated levels in a room over 10 $\mu g/m^3$, then biomonitoring should be offered to any occupants as a check on their personal levels of exposure.

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 Rutherford 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 control measures are needed to protect those involved in the work. This will require additional radiological measurements.

No routine monitoring of asbestos levels is indicated, but the safety of any future maintenance or construction work that might disturb asbestos-containing materials should be managed according to standard recommended practices.

7.3 Advice for those exposed

Given the low potential for risk, no form of health screening or other health intervention is recommended for people who may have been exposed to hazardous contaminants in the Rutherford Buildings.

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APPENDIX 1

Environmental monitoring of asbestos in Rutherford (previously Coupland 1) Building

Date	Location	Recorded levels (fibres per ml)
19.06.87	Basement store room	< 0.01
03.07.87	Basement plant room	< 0.01
04.05.89	Basement passage	< 0.01
11.11.90	Attic above postgraduate room	0.01, 0.02
12.11.90	Attic above postgraduate room	< 0.01
27.02.91	Basement store room	< 0.01
27.02.91	Basement flint store	< 0.01
17.05.91	Flint store	< 0.01
15.03.95	Basement corridor	< 0.01
09.12.95	Fume cupboard	< 0.01
06.01.96	Museum first floor geology	< 0.01
22.02.96	Tunnel from Coupland plant room to Coupland 1	0.18, 2 x < 0.01
25.02.96	Tunnel from Coupland plant room to Coupland 1	0.03, 3 x < 0.01