

The Dangers of Ionising Radiation

*A Scientific Guide to Radiation Risks for
Government Agencies, Legal
Professionals and Medical Clinicians*

by

Ian Fairlie

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and Medical Clinicians**

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In Memoriam

This book is dedicated to former scientists whose critical insights on radiation risks guided the writing of this book: Professor Tom Wheldon (2000†), Professor Alice Stewart (2002†), Dr Philip Day (2009†), and Professor Steve Wing (2016†).

Tom Wheldon was head of the CRC Beatson Laboratories in Glasgow, Scotland and co-author of a previous book on radiation risks - 'Radiation Risks: An Examination' (1994). He was my external examiner during my MSc studies at the Medical College of St Bartholomew's Hospital, London UK in 1992.

Alice Stewart was head of the Department of Social Medicine at the University of Birmingham UK where she carried out the famous Oxford Survey of Childhood Cancer from the 1950s to the 1990s.

Phil Day was Reader at the Department of Chemistry at the University of Manchester UK and a member of the UK government's CERRIE Committee on internal radiation risks (2000-2005).

Steve Wing was Professor of Epidemiology at the University of North Carolina, US (1985-2016).

"Science sans conscience n'est que ruine de l'âme."

(Science without conscience is but the ruin of the soul.)

François Rabelais (circa 1483–1553)

"Radiation, in its simplest terms - figuratively, literally and chemically - is poison.... there is no amount of radiation so small that it has no ill effects at all on anybody. There is actually no such thing as a minimum permissible dose.

Perhaps we are talking about only a very small number of individual tragedies - the number of atomic age children with cancer, the new victims of leukemia, the damage to skin tissues here and reproductive systems there - perhaps these are too small to measure with statistics. But they nevertheless loom very large indeed in human and moral terms."

Former US President John F. Kennedy (1917-1963)

Speech given in Milwaukee, WI, April 1960

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I particularly acknowledge the out-of-print book 'Radiation Risks : An Examination' (1991) Tarragon Press, Scotland by David Sumner, Tom Wheldont† and Walter Watson.

Any errors or oversights are the sole responsibility of the author.

London, United Kingdom

January 2026

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Executive Summary

This book provides an independent scientific examination of the health effects of ionising radiation, in response to reports suggesting that it may be less dangerous than currently thought. It draws attention to the fact that radiation causes not just cancer but other ill-effects such as cardiovascular disease and dementia not acknowledged in existing radiation protections. The book shows that radiation's cancer risks are not fully recognised quantitatively-speaking, and that existing cancer risk coefficients need to be increased. It highlights the continued increase in UK cancer incidence among children in recent years.

Recent authoritative epidemiological studies and corroborative reports provide strong evidence that current risk factors for radiation do not overestimate its risks. Indeed the opposite is the case: the new evidence indicates we need to increase our perceptions of radiation risks and tighten current radiation limits.

Official reports state that radiation risks in children are approximately ten times greater than in adults, but these are poorly addressed in UK or US radiation protection systems: no risks or limits are published for radiation exposures to children. A similar position exists for women who are 50% more sensitive to radiation than men, but few protections exist for them.

Low levels of radiation are serious matters. For example this book estimates that about 10% of all cancer deaths in the UK are from natural background + medical exposures. In addition, about 20% of all childhood leukemias are estimated by experts to be caused by

background radiation. Childhood cancer is a worrisome issue: the annual death toll of children in the UK from cancer is about six times greater than from road accidents. Just as worrying is the fact that childhood cancer incidence has been rising in the past two decades and we are little closer to determining its cause(s) than we were 20 years ago. Radiation is the only external environmental factor for which incontrovertible evidence exists of its potential to cause cancers, especially leukemias.

Although many improvements have been made in treatments to reduce death tolls from cancer, the same is not true for reducing the number of new cancer cases each year. Our society appears to be better at treating cancer than preventing it from arising in the first place, despite the truism that an ounce of prevention is worth a pound of cure.

From cellular studies and epidemiological evidence, this book suggests that over half of infant and childhood leukemias arise from radiation exposures *in utero*. This means that radiation, like thalidomide, is a teratogen. The main difference is that with thalidomide the effects – limb malformations – are visibly apparent at birth whereas with radiation the effects – mutations in blood-forming tissues - only become apparent months and years after birth.

It is recommended that the Precautionary Principle be used when assessing radiation risks - especially in children and women. Consideration should be given to establishing a national cancer preventative programme to reduce childhood cancer incidence levels.

Chapter 1

Introduction

This book is about ionising radiation¹ and its close relative radioactivity, neither of which we can register with our senses but which are ever present in our lives. For example, we are continually exposed to small amounts of background radiation, and radiation. And we shall show that radioactive substances are increasingly used in medicine, industry and academia.

The main types of radiation are shown in Appendix A; major sources of radiation are listed in Appendix B; radioactivity is explained in Appendix C; internal and external radiation is discussed in Appendix D, and radiation effects are explained in Appendix E.

Conventionally (and in the UK, legislatively) radiation is divided into two categories - ionising radiation (IR) and non-ionising radiation (NIR). This book concerns the former, including gamma rays and X-rays. Less energetic non-ionising radiation, that is electromagnetic radiation and electromagnetic fields, eg from mobile phones, is discussed briefly in Appendix F. However recent research indicates a blurring between the two types: this is discussed in Appendix G. For those in a hurry, facts about radiation in a nutshell are listed in Appendix H.

¹ radiation types are described in Appendix A

Why this book is relevant

Epidemiological studies (discussed for example by Kitahari et al, 2015) using computerised record linkage and pooled cohorts have allowed larger, more powerful, assessments of cancer and other radiation-related effects at low radiation doses even below ~ 50 mGy². The resulting risk estimates for radiation demonstrate beyond reasonable doubt that radiation's health risks are greater than presently perceived. As the new findings are statistically significant, we can be confident they are reliable for use in revising radiation protection standards and in setting safer limits.

The new studies also indicate that radiation causes not just cancers but also cardiovascular diseases, strokes, dementia and other illnesses³. Current risk factors recommended by the International Commission on Radiological Protection (ICRP) only address fatal cancer. Few people and, it would appear, few politicians appear to be aware of the new dangers from radiation exposures. A main conclusion of this book is that existing risk factors and exposure limits for radiation need to be substantially revised and brought up-to-date.

A second reason for this book is that recent research indicates new radiation-related effects in tissue. These are termed non-targetted effects and they include genomic instability, bystander effects, minisatellite mutations (now called tandemly-repeated DNA), apoptosis⁴, and hormetic effects. These observations, together with

² dose units are set out in Annex 2

³ they also question past perceptions of possible thresholds for cardiovascular disease and eye cataracts.

⁴ apoptosis is a controlled process whereby a cell self-destructs to remove itself without causing inflammation

new insights about radiation-related epigenetic effects, profoundly deepen our understanding of radiation's effects but they have not been taken into account by official agencies in radiation protection, as we shall see below.

A third reason is that many new studies indicate raised incidences of cancer near nuclear power facilities: these are discussed in Chapter 16. They pose inconvenient questions about recent government proposals to expand our reliance on nuclear power in the future.

Radiation and radioactivity: Some history

The discoveries of radiation by Röntgen and radioactivity by Becquerel were made at the end of the 19th century. Quite soon afterwards, adverse health effects from exposures to them began to be observed. In addition, an epidemic of leukemia occurred among UK army radiation teams after the first World War, mainly due to the use of rudimentary X-ray equipment by surgeons to find shrapnel in wounded infantry (March, 1944). Sadly this epidemic existed for decades until the 1940s and 1950s, and according to March resulted in the deaths of an estimated 500 army surgeons. (References at end of each chapter).

To deal with this alarming finding from WW1, the then International Society of Radiology established an International Committee on Radiation Units (ICRU) to formulate ideas on radiation doses. Despite its title, this was an unofficial body unconnected to any governments. Later in 1928, the International Congress of Radiology (a grouping of academics whose purpose was to facilitate best international practices in radiology)

established the International X-ray and Radium Protection Committee. In 1950, this was renamed the International Commission on Radiological Protection (ICRP) which still publishes recommendations on radiological protection. Again the ICRP, like the ICRU, is an unofficial body without formal connections to governments.

The ICRU and ICRP are voluntary bodies although their recommendations are followed by most governments. One of the main findings of this book is that the ICRP's recommendations are often slow in being formulated – long after new evidence of new effects from radiation exposures has appeared. The ICRP's record is discussed further in Appendix U.

Radiation risk - a controversial issue

The history of radiation risks – to be precise the perceptions of these risks in Europe and North America - reveals a long-running battle of scientific interpretations, values and politics. This was commented upon by US epidemiologists Wing and Richardson who stated

“Although controversies over scientific findings are common, the topic of health effects of ionizing radiation has generated an exceptional amount of heat. Despite a century of research since Roentgen's discovery of X rays, fundamental disagreements exist over biophysical mechanisms, dose-response assumptions, analytical strategies, interspecies extrapolations, and the representativeness of studies of select human populations.” (Wing et al, 1997)

Broadly speaking, over the past seven decades a gulf has existed - and often still does - between official radiation risks used by

government and industry and the risks observed by scores of independent scientists. The latter found that radiation risks, including cancer, were greater than official estimates, but they and their scientific reports were often harshly treated by officialdom. A recent book (Fairlie I and Folkers C, 2024) contains succinct biographies of 40 of these independent scientists and whistle-blowers.

The present book examines recent evidence on radiation's dangers and risks in more detail. It is aimed at professionals, academics and clinicians who are well-informed in their own disciplines but not necessarily in radiation risks. This is because radiation science tends to be quite separate from other areas of science, largely for historical reasons.

Critical viewpoint

With radiation, much depends on whether its effects are considered harmful or not, which means that viewpoints, ethical principles and legal provisions are involved. For that reason and for the sake of transparency, this book is written from a public health point of view. As stated by the Roman consul Cicero over two thousand years ago:

“Salus populi suprema lex esto”

Translated as: the health and safety of the people is the supreme law. We discuss public health in Appendix N. Adopting its viewpoint necessarily requires a critical stance.

Dozens of books have been written on radiation and its effects, almost⁵ all from an institutional point of view. That is, they tend to reflect the interests of the institutions at which authors were employed or under contract in many instances. These authors are entitled to their views of course, but what may be missing are critical views and insights, which would give a more comprehensive picture. Seeing both sides of contentious matters leads to better judgments and policy decisions. This book presents these critical views and insights.

Structure and Content

Since radiation is a complex subject we start with simpler matters, then progress to more complicated ones. To free up the narrative flow, subject areas which are lengthy to explain are moved to the Appendices. Scientific references are presented at the end of each chapter and also collected in Annex 4 at the end of the book.

Many accounts of radiation risks use jargon words, units and terms little used outside the radiation world. More often than not they are untransparent and are sometimes difficult to grasp at first sight. This book avoids jargon and explains all terms, in order to present a readily accessible account of radiation's dangers and risks. The field of radiation biology is also awash with abbreviations and acronyms little used outside it. The technical Annexes spell these out and explain them. A glossary contains commonly-used terms in radiation and radioactivity studies.

⁵ see Bibliography at the end for recommended literature

Epidemiology

Epidemiological studies often were a poor tool for discovering the adverse effects of radiation exposures in the past (Smith, 2001). Some suffered from poor case identification, non-uniform registration, variable or uncertain diagnostic criteria and uncertainties in the uniformity of data collation. Predicted excess deaths were often uncertain due to confounding factors, competing causes of death and the use of different risk projection models. In almost all epidemiology studies which assessed risks, difficulties existed with estimating radiation doses accurately.

An important aspect is the type of epidemiology study, as some types are more reliable than others. This is outlined in Appendix P on the hierarchy of types of epidemiological evidence. Another aspect is size as the larger the number of cases in a study the more reliable its findings. Meta-studies which group together such studies in order to strengthen their statistical strength are a solution, but relatively few have been carried out. In addition, various factors can produce bias and/or confounding: for example, smoking and alcohol cause major increases in overall mortality⁶, morbidity⁷, and cancer and other diseases.

In recent years, the quality of epidemiology studies has been improving, due to their larger sizes, rigorous attention to controls and to possible confounding factors, use of statistical tests (see Appendix S), treatments of uncertainty and their analyses of previous studies, and the various factors involved in possible

⁶ numbers of deaths

⁷ numbers of cases of the illness

causalities. For example, the INWORKS studies (considered in chapter 10) are marked improvements over epidemiology studies in the previous century. The recent epidemiology study by Wakeford and Bithell (2021) on childhood leukemia increases following *in utero* exposures sets a high bar for future studies to match.

Older Studies

It will be noted that many of the studies referenced in this book are relatively old. This is mostly due to the lack of modern studies in the relevant areas. However just because a study is from the past does not mean that its content is unreliable or should be discounted. It just means that up-to-date studies do not exist or at least have not been discovered from the literature searches carried out. It is quite noticeable for example that after about 2010 many UK databases on radiation exposures were discontinued (for unclear reasons). Indeed one of the reasons for writing this book has been to resurrect older studies (and preserve their memories) in order to make them available to younger generations of scientists.

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Chapter 2

Cancer

Cancer is a major stochastic¹ effect of low exposures to ionising radiation. The WHO's International Agency on Research on Cancer has classified ionising radiation as a Group 1 carcinogen which means that sufficient evidence exists of carcinogenicity in humans (IARC, 2011). IARC has also published reports indicating that the following are all also Group 1 carcinogens:

- Ionizing radiation (all types)
- Neutron radiation
- Phosphorus-32, as phosphate
- Plutonium
- Radioiodines, including iodine-131
- Nuclear fission products, including strontium-90
- Radionuclides, α -particle-emitting, internally deposited
- Radionuclides, β -particle-emitting, internally deposited
- Radium-224 and its decay products
- Radium-226 and its decay products
- Radium-228 and its decay products
- Radon-222 and its decay products
- Solar radiation
- Thorium-232 and its decay products
- Ultraviolet radiation (wavelengths 100-400 nm, encompassing UVA, UVB, and UVC)

¹ stochastic means having a random probability distribution

- X-ray and gamma radiation

Aetiology of Cancer

Four decades ago, Upton and Albert (1986) were among the first scientists to state that extremely small radiation exposures to a few molecules or to a single molecule could lead to cancer. They explained this was because

- most cancers could be traced back to a single cell of origin
- most carcinogens were also mutagens
- a clear association existed between cancers and specific mutations/chromosome aberrations, and
- clear experimental evidence indicated a no-threshold dose-response relationship

Since then, their conclusions have continued to be supported by the available evidence, as cancer is now known to arise from radiation damage to a single stem cell.

Stem cells

Stem cells create new differentiated² cells in an organ while staying undifferentiated themselves. In adults, they act as a repair system for the body, i.e. replenishing cells when they die. Most stem cells are found in the gut lining, bone marrow and skin where they regenerate intestinal cells, blood and skin. In adults, the production of new cells is closely controlled and just keeps pace with cell loss.

² differentiated cells have specialised functions, eg skin cells or nerve cells.

Undifferentiated cells in adults on the other hand are multipotent, that is they have the ability to generate more cells in that organ while staying unchanged themselves

This means that stem cells divide only when new cells are needed; if new cells are not needed, the division of stem cells is prevented by signals received from nearby cells.

However when a stem cell is damaged by radiation it may fail to respond to these signals telling it to stop. As a result, the cell and its descendants continue to divide producing a mass of cells, i.e. a tumour, which if untreated can be fatal. The type of cancer depends on which organ the stem cell is found. Stem cell damage can occur at low radiation doses - with correspondingly low probability.

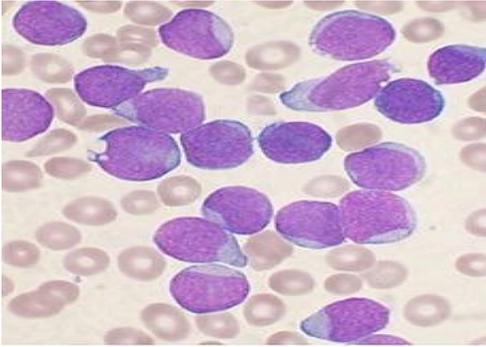
For some adult cancers, as many as five or six separate DNA mutations may be needed for a cancer to result, though with childhood tumours, only one may be needed. The need for several mutations may explain why cancers often appear many years after the original exposure. Possibly an initial exposure to radiation or other agent causes the first of several required mutations, with the other mutations being caused in later years - perhaps by our continual exposure to background radiation.

Cancer is a very common disease - causing more than a third of all UK deaths: cancer incidence is increasing throughout the Western world.

Blood cancers

Blood cancers including leukemias are characterized by abnormal increases in the numbers and sizes of white blood cells. They do not result in tumours but in deformed blood cells which are unable to carry out their normal functions, i.e. protection from bacterial and viral infections. See BOX and figure 2.1

Figure 2.1 Bone marrow cells (stained mauve) exhibiting acute lymphocytic leukemia



Source <http://en.wikipedia.org/wiki/Leukaemia>

Leukemia types

Leukemias are first divided into acute and chronic forms. Acute leukaemia means the rapid increase of immature (malignant) blood cells which results in the bone marrow being unable to produce healthy blood cells. Due to the rapid accumulation of cells many enter the bloodstream and spread to other organs. Immediate treatment is required. Acute leukemia is the most common form of leukemia in children.

Chronic leukaemia means the increase of mature, abnormal, white blood cells over months or years. These cells are produced more quickly than normal cells, resulting in many abnormal white blood cells in blood. Whereas acute leukemia must be treated immediately, chronic forms are monitored for some time before treatment. Chronic leukemia mostly occurs in older people.

Secondly, leukaemias are divided according to which kind of blood cell is affected, either lymphocytic (lymphoblastic) leukaemias or myeloid (myelogenous) leukaemias. In lymphocytic leukemias, the cancer occurs in marrow cells that form lymphocytes, i.e. infection-fighting cells. In myeloid leukemias, the cancer change takes place in cells that form red blood cells, some other types of white cells, and platelets.

Combining these two classifications provides a total of four main categories as follows:

	Acute	Chronic
Lymphocytic leukemia	Acute lymphoblastic leukemia (ALL)	Chronic lymphocytic leukemia (CLL)
Myeloid leukemia	Acute myelogenous leukemia (AML)	Chronic myelogenous leukemia (CML)

The cause(s) of cancer?

Although the cause or causes of most cancers are unknown at present, some childhood leukemias are now thought to be due to background radiation. British scientists Mobbs et al (2009) and Wakeford et al (2009) have estimated that 15% - 20% of spontaneous childhood leukemias are due to naturally-occurring background radiation. In the past, Stewart (1958) had suggested that the figure may be higher – and may even be 100% (Baverstock, 2003)

Whatever the percentage figure, it is widely accepted that leukemia is particularly associated with exposures to radiation, as it is the only external environmental factor for which incontrovertible

evidence exists of its potential to cause cancers, especially leukemias.

The rationale for this statement is as follows. Dividing cells are particularly sensitive to radiation damage, and the greatest mass of reproducing cells (apart from embryos and fetuses) is found in bone marrow stem cells where they create billions of blood cells every day³. These vital tissues are located in the middle tissues of our bones especially the femur. These tissues are not thought to be affected by chemicals or pathogens, but are vulnerable to external radiation and to permeable (i.e. highly soluble) molecules which may be radioactively-labelled, eg water molecules labelled with radioactive hydrogen-3 (i.e. tritiated water) or carbon molecules labelled with radioactive carbon-14. These types of radiation may damage marrow stem cells so that they produce deformed white blood cells, i.e. leukaemia.

Childhood leukemia is currently thought to result from a two-stage process as shown in Figure 4 reproduced below from Rössig and Jürgens (2008). The first genetic defect occurs in a cell of the embryo/foetus. This may be due to background radiation or to exposures to (man-made) radiation or to chemicals in the womb. A second defect in the same cell happens after birth, also perhaps due to background radiation or exposure to bacteria or viruses.

³ these are called haematopoietic tissues, and the whole process the haematopoietic system.

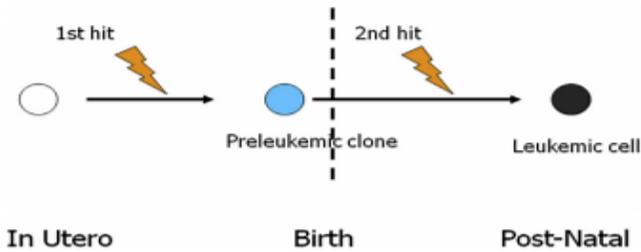


Fig. 4. Scheme for leukemogenesis after Rössig and Jürgens (2008).

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Chapter 3

Cardiovascular Disease including Stroke

Cancer is the most studied effect of radiation, but increased risks of coronary heart disease, arteriosclerosis¹, atherosclerosis², and strokes³ have now been observed following radiation exposures. These are collectively termed cardiovascular disease (CVD). Strokes occur as a result of blood supplies to the brain being cut off or restricted and are usually included in studies and data on CVD, as they are often fatal.

Until the mid-1960s, heart tissue had been thought to be radio-resistant. Even as late as the 1990s, the issue of whether radiation exposures led to CVD was controversial and the relationship was not really examined in depth until the turn of the 21st century. Little et al (2008) were among the first to report that cardiovascular risks were raised after moderate exposures to radiation. Increased radiation-associated mortality from heart disease and stroke was observed among the Japanese atomic bomb survivors (Shimizu et al, 2010, Takahashi et al, 2017) and observed for stroke among those exposed in early childhood (Tatsukawa et al, 2008). Bruno et al (2013) found an early peculiar pattern of pre-clinical vascular

¹ arteriosclerosis is the thickening, hardening and loss of elasticity of artery walls. This restricts the blood flow to organs and tissues and leads to severe health risks

² atherosclerosis is a specific form of arteriosclerosis in which an artery wall thickens as a result of invasion of white blood cells and proliferation of intimal smooth muscle cells creating a fibro-fatty plaque

³ stroke occurs when low blood flow (often from atherosclerosis) to the brain results in damage and death to brain cells. Strokes are often fatal: essentially from brain malfunction.

involvement after radiation exposures which supported the view that low-level radiation contributed to cardiovascular disease.

In fact, for many years, US physicians had considered that it was cardiovascular disease, not cancer, that limited the survival times of their cancer patients after they had received radiation therapy treatment (Heidenreich and Kapoor, 2009).

In 2015, Kreuzer et al (2015) stated that low radiation doses could increase the long-term risk of cardiovascular disease. They stated

“This would have major implications for radiation protection with respect to medical use of radiation for diagnostic purposes and occupational or environmental radiation exposure. Therefore, it is of great importance to gain information about the presence and possible magnitude of radiation-related cardiovascular disease risk at doses of less than 0.5 Gy”.

The INWORKS studies (see Chapter 10) also observed increased radiogenic risks of cardiovascular disease and strokes (Gillies et al, 2017).

Perhaps the most important study here is the recent large meta-analysis⁴ of 93 health studies on cardiovascular diseases published in the influential British Medical Journal (Little et al, 2023). The authors concluded

⁴ a meta-analysis combines data from several studies on the same issue to produce a single estimate of effect. This usually increases its statistical power compared with a single study.

“Our findings suggest that radiation detriment might have been significantly underestimated, implying that radiation protection and optimisation at low doses should be rethought.”

“This finding has considerable implications for the system of radiological protection, assuming that the extrapolation is permissible, even, for example, over the restricted dose range 0-0.5 Gy. This added risk would nearly double the low dose detriment.”

Their conclusions were supported in an accompanying BMJ editorial (BMJ, 2023).

This new study confirms and strengthens previous studies because it is large and powerful, statistically speaking. The BMJ article discusses risks to medically-exposed patients as medical exposures are larger than environmental and worker ones. Nevertheless in the list of studies (set out in Supplement S3, Tables S3.2-S3.5 in the study’s web appendix 1), about half of the studies examined were of nuclear workers and populations near nuclear facilities. Therefore the report’s conclusions extend to occupational and environmental exposures as well as medical ones.

No widespread consensus exists at present to explain how radiation causes CVD, but these diseases have longish latency periods, do not appear to have a threshold, and are progressive. In other words, they have similar characteristics to radiogenic cancer effects.

Little et al (2009) have stated that multiple small exposures to radiation cause monocyte chemoattractant protein-1⁵ (MCP-1) concentrations to increase linearly with dose. MCP-1 is important in the pathogenesis of many diseases, allergic conditions, atherosclerosis, nephropathies and tumours. The main driver for the increase in MCP-1 is monocyte death and consequent reduction in MCP-1 degradation. The radiation-induced risks predicted by this model are consistent with those observed in a number of occupationally-exposed groups. Lowered MCP-1 concentrations with low density lipoprotein cholesterol concentrations are also consistent with experimental and epidemiologic data.

Hildebrandt (2010) has stated

“In recent years, there is growing epidemiological evidence of excess risk of late occurring cardiovascular disease at much lower radiation doses and occurring over much longer intervals after radiation exposure without a clear-cut threshold. ... The mechanisms of radiation-induced vascular disease induction are far from being understood. However, it seems to be very likely that inflammatory responses are involved. If ... inflammatory response is ... the most likely cause of radiation-induced cardiovascular disease after low exposures, this ... implies a role for non-targetted radiation effects.”

If the latter point about non-targetted⁶ effects is correct, this could be significant for low-dose CVD risks, as they could be greater than we currently think. See discussion in Kadhim et al (2013). Further discussion on radiogenic CVD risks can be found here

⁵ monocyte chemoattractant protein-1 is a cytokine—a signaling protein—that attracts immune cells to sites of inflammation, infection, or injury. They play a major role in initiating immune responses and contributing to various diseases.

⁶ meaning that radiation’s effects are not on the DNA molecule. See chapter 6