Committee on Medical Aspects of Radiation in the Environment (COMARE)

THIRTEENTH REPORT

The health effects and risks arising from exposure to ultraviolet radiation from artificial tanning devices.

Chairman: Professor A Elliott

Produced by the Health Protection Agency for the
Committee on Medical Aspects of Radiation in the Environment

ISBN 978-0-85951-645-7

CONTENTS

FOREWORD

i The Committee on Medical Aspects of Radiation in the Environment (COMARE) was established in November 1985 in response to the final recommendation of the report of the Independent Advisory Group chaired by Sir Douglas Black (Black, 1984). The terms of reference for COMARE are:

> 'to assess and advise Government and the devolved authorities on the health effects of natural and man-made radiation and to assess the adequacy of the available data and the need for further research'

In over 20 years of providing advice to Government and the devolved authorities COMARE has produced to date 12 major reports and many other statements and documents mainly related to exposure to naturally occurring radionuclides, such as radon and its progeny, or to man-made radiation, usually emitted by major nuclear installations. The most recent COMARE report focused on the impact of personally initiated X-ray computed tomography scanning in the health assessment of asymptomatic individuals. The current report addresses the health effects and risks associated with the use of artificial tanning devices.

ii The incidence of skin cancer in the UK has continued to rise. In 2006, more than 10,400 new cases of malignant melanoma and over 81,500 cases of non-melanoma skin cancer were registered in the UK (Cancer Research UK, 2008a). An association has been established between increased melanoma incidence and the use of artificial ultraviolet (UV) radiation devices (IARC Working Group, 2005, 2007). However, regulation of the artificial UV radiation tanning industry in the UK is currently inconsistent and largely unmonitored, due to the widespread availability of such equipment for domestic use and unsupervised coin-operated outlets.

iii In 1992, COMARE produced a statement on the adverse health effects of UV radiation within the context of existing public health advice on minimising UV radiation exposure and the need for further research, in response to a request from the UK health departments (UK Parliament, 1992). COMARE noted with concern the significant rise in malignant melanoma incidence (over 2,500 cases in England and Wales in 1986) and considered this to be a significant health issue in the UK. It supported the improvement of skin cancer registration procedures, the work of the (then) National Radiological Protection Board (NRPB) in solar UV radiation monitoring and the Department of Health's proposal to reinforce existing public health advice on UV radiation.

iv In 2007 the Welsh Assembly Government (WAG), supported by the Scottish Government (SG), the Department of Health, Social Services and Public Safety for Northern Ireland (DHSSPS NI) and the Department of Health (DH), requested that COMARE provide advice regarding the safety of UV sunbeds in the UK. To achieve an appropriate review of this topic, COMARE established its UV Sunbed Subcommittee (USS). This subcommittee

incorporates members from COMARE and scientific experts, and a patient representative as well as representatives from government departments.

v This subcommittee's terms of reference are:

> 'to assess and advise COMARE on the health effects and risks arising from the exposure to UV radiation from artificial tanning devices and to advise on the adequacy of the appropriate controls and the need for further research'

When the subcommittee finished its deliberations, the review was passed to COMARE for consideration by the full committee with the aim that in due course COMARE would present its advice to the UK health departments. That advice is contained in this, our Thirteenth Report.

vi A number of initiatives involving UV sunbeds have been running concurrently with the work of the subcommittee. In 2007 the Department of Health launched its Cancer Reform Strategy to set a clear direction for cancer services for the next five years. One area of focus was skin cancer, with an expansion of the SunSmart Campaign and the review of options for regulation of the sunbed industry (Department of Health, 2007). Part of the 2008 SunSmart Campaign run by Cancer Research UK aimed to raise the awareness of the dangers of sunbeds (Cancer Research UK, 2008b). The Health and Safety Executive (HSE) performed a consultation in 2008 on its revised guidelines on controlling the health risks associated with working with UV tanning equipment. The latest guidelines were published in May 2009 (HSE, 2009). The work of the subcommittee will enable the formulation of clear strategies by Government and will take into account the reports from the other initiatives.

vii The aim of this COMARE report has been to provide advice for the UK health departments on the health effects and the risks from exposure to UV radiation from artificial tanning devices, such as sunbeds or sunlamps that are used on commercial premises or are available for purchase or hire to be used in the home. However, the interest in this issue extends beyond the remit of the health departments and the recommendations made in this report will be pertinent to other government departments.

viii In this report, the term 'sunbed' is used to represent all types of artificial UV tanning devices utilised for cosmetic purposes.

CHAPTER 1

INTRODUCTION

1.1 Non-ionising ultraviolet (UV) radiation originates primarily from the sun, but also from artificial sources. The UV radiation region covers the wavelength range 100–400 nm and is divided into three bands: UVA (315–400 nm), UVB (280–315 nm) and UVC (100–280 nm), which are listed as anticipated to be human carcinogens (National Toxicology Program, 2005).

1.2 There is scientific evidence relating to cumulative UV radiation exposure and the potential it has to cause damage to the skin, such as sunburn, skin cancer and photokeratitis of the eyes. There is an increased risk of skin cancer and cataracts, and ageing of the skin occurs at a greater rate. The immune system can also be suppressed. Therefore, the UV radiation from the sun and artificial sources is of considerable public health concern. The risk is also greater in younger people.

1.3 Skin cancers are extremely common, with two main categories: malignant melanomas and non-melanoma skin cancers (NMSCs). Over 81,500 cases of NMSCs were registered in the UK in 2006, but registration is known to be incomplete. The majority of NMSCs are either basal cell carcinomas (BCCs) or squamous cell carcinomas (SCCs), both of which are highly treatable and survival rates are very high. Both tend to be slow growing, appearing commonly on the face or sun-exposed areas. The main treatment is through surgery, which can result in disfigurement. Although uncommon, it is possible for NMSCs to be fatal and in 2007 there were over 500 deaths attributed to NMSCs (Cancer Research UK, 2008a). Treatment of NMSCs places a significant burden on the NHS. Recent data from the South West Public Health Observatory show that in 2001–2006 the number of admissions to English hospitals as an in-patient or as a day-case was approximately 6,000 per year for melanoma but 49,000 per year for NMSCs (SWPHO, personal communication, 2009). These admissions annually required 13,000 bed-days for melanoma and 31,000 bed-days for NMSCs over the period 2001–2006.

1.4 Malignant melanomas are less common (with over 10,400 new cases in the UK in 2006) but are more frequently fatal. In 2007 over 2,000 deaths were attributed to melanoma (Cancer Research UK, 2008c). Episodes of burning UV radiation exposure, even brief ones, at an early age are implicated as a major risk factor for melanoma (Mackie, 2006).

1.5 Patients with certain skin diseases can benefit from therapeutic UV radiation exposure, given under carefully controlled conditions and often combined with photoactivated drugs. Therapeutic UV radiation exposure is used in the treatment of psoriasis, and with photoactivated drugs for the treatment of certain lymphoid malignancies of the skin; however, these procedures should only be carried out in a clinical setting under medical supervision. The use of artificial UV radiation in the treatment of acne should also only be performed under these conditions.

1.6 The scientific evidence for the potentially harmful effects of UV radiation has been reviewed by a number of scientific expert groups, which include the International Agency for Research on Cancer (IARC, 1992), the World Health Organization (WHO, 1994, 2003), the International Commission on Non-Ionizing Radiation Protection (ICNIRP, 2003) and the European Society for Skin Cancer Prevention (EUROSKIN, 2005). In the UK, the independent Advisory Group on Non-ionising Radiation (AGNIR, 1995, 2002) has produced reports outlining the scientific evidence and potential guidelines.

1.7 According to IARC (IARC Working Group, 2005), there is evidence to suggest an increase in melanoma risk in later life associated with use of sunbeds by young people in their teens and twenties. The data show a prominent and consistent increase in risk for melanoma in people who initially used sunbeds in their first three decades: a 75% increase in risk of melanoma was calculated for such users of artificial tanning appliances. In addition, there is an increase in risk of squamous cell carcinoma of the skin associated with use of sunbeds by young people in their teens. There is also information suggesting detrimental effects from use of sunbeds on immune response, which may have repercussions on the aggressiveness of squamous cell carcinoma (AGNIR, 2002).

1.8 For the general public using commercial outlets, there are perceived beneficial health effects from exposure to UV radiation, which are largely psychological and cosmetic. There is little value in the use of sunbeds in terms of protection from sunburn. Vitamin D synthesis is promoted by some outlets as justification for the use of sunbeds, yet vitamin D can be nutritionally supplied without the risks associated with exposure to artificial UV radiation. The usefulness of sunbeds in the induction of vitamin D synthesis is dependent on the level of UVB emissions; however, UVA is usually the predominant emission from sunbeds. There is evidence that although use of sunbeds can increase vitamin D levels, this reaches a plateau after a few sessions (Thieden et al, 2008). Given that there are wholly safe alternatives, the benefit of sunbed use as a source of vitamin D is outweighed by the risks.

1.9 EUROSKIN, ICNIRP and the WHO recommend that UV radiation appliances are not used for tanning or other non-medical purposes; however, the level of control employed varies between countries. There are particularly strict controls for sunbed use in France, which were introduced in 1998. In all, 15 member states of the European Union have some measures in place with either guidance or legislation. Germany, like the majority of the UK, uses a voluntary self-regulation control system. Initial take-up of the scheme was extremely low, which has led to a revamping of the system (Böttger, 2007). The only UK authority to currently regulate sunbed use with legislation is Scotland, through the Public Health etc (Scotland) Act 2008 (Scottish Parliament, 2008). The Act prohibits use of sunbeds by people under 18 years of age and also the sale or hire of sunbeds to people under 18. There are currently no regulations in place for England, Wales or Northern Ireland.

1.10 The Public Health etc (Scotland) Act 2008 also prohibits the unsupervised use of sunbeds. This is the result of the major public health concern in the increase in the number of unsupervised commercial outlets and the increasing trend by many outlets (both supervised and unsupervised) to operate with promotional pricing strategies, such as unlimited sessions within a specific timeframe and free starter sessions. In addition, the Act requires operators to provide users with information on the health effects of sunbed use. Health information on the use of sunbeds is also provided in the guidance issued by the Health and Safety Executive (HSE, 2009).

1.11 The Sunbed Association (TSA) was founded in 1995 as the trade association for the UK sunbed industry. Membership is voluntary and currently TSA represents approximately 20% of the UK operators. It is a requirement for members of TSA to work to its code of practice, which incorporates aspects of the British and European Standard (BS EN 60335-2-27: 2003, British Standards Institution, 2003) and the HSE guidance note on UV tanning equipment (The Sunbed Association, 2008).

1.12 Another area for concern is the use of sunbeds in the home, either through hire or purchase. The duration of UV radiation exposure under these circumstances is therefore up to the discretion of the individual and may exceed the recommendation of the British Photodermatology Group (BPG) of a maximum of 20 sessions per year (Diffey et al, 1990).

1.13 Sunbeds emit predominately UVA radiation. There is wide variety in the style (lie-down units and stand-up units) and in the UVA and UVB output of sunbeds. The British Standard (BS EN 60335-2-27: 2003) classifies sunbeds according to their UVA and UVB effective irradiances. More recently, due to the demand to have a tan for fashion or cosmetic reasons, manufacturers have developed devices that produce higher levels of UVB, at all the various spectral distributions and power levels. Many sunbeds now exceed the effective irradiance of midday southern European sun, giving a greater carcinogenic potential (Oliver et al, 2007).

1.14 The purpose of this report is to provide COMARE's advice to Government on the health effects and risks from UV sunbeds, recommending actions that could be taken forward by all four UK health departments and other government departments with an interest in this issue.

CHAPTER 2

ULTRAVIOLET RADIATION INDUCED DNA DAMAGE: EFFECTS IN CELLS AND ORGANISMS

Sunlight and light from sunbeds causes DNA and tissue damage.

DNA damage causes mutation.

Mutation causes cancer and ageing.

The incidence of all skin cancers is increasing.

10,400 new cases of melanoma in 2006 (and rising) in the UK.

2.1 Since life first began on Earth, organisms have been subjected to DNA damage. The causes of DNA damage include exposure to radiations and chemicals (Friedberg et al, 2006; Goodhead, 1989; Hutchinson, 1985; Lawley, 1966; Mitchell et al, 1991; Setlow, 1974). For organisms exposed to sunlight, solar UV radiation has been a source of such damage (Friedberg et al, 2006; Mitchell et al, 1991; Peak et al, 1987; Setlow, 1974). Components in DNA, especially the pyrimidine bases, absorb UV energy and the spectrum of DNA damage induced depends on the UV radiation wavelength. The absorption peak for DNA lies at 260 nm; this is in the UVC portion of the UV radiation spectrum and solar UVC never reaches the Earth's surface. UVC predominantly induces two adjacent pyrimidines in the same strand of the double helix to become covalent linked to distort the DNA structure (Friedberg et al, 2006; Mitchell et al, 1991; Setlow, 1974).

2.2 Solar UV radiation also induces oxidative damage to DNA and cell components. These events have been linked to the process of cancer induction either via the direct creation of DNA damage such as 8-oxoguanine, which is highly mutagenic, or via the instigation of cell signalling events that play roles in cancer induction (reviewed in Friedberg et al, 2006).

2.3 Many of the lesions induced by UVC are also observed following exposure to solar UV radiation (UVA and UVB), but they are induced with a lower efficiency (Mitchell et al, 1991; Setlow, 1974).

2.4 The DNA double helix consists of two DNA strands and each strand contains four different DNA bases – two types of pyrimidines (thymine and cytosine) and two types of purines (guanine and adenine). The two strands of the double helix are held together by adenine from one strand pairing with thymine in the other strand and vice versa, plus guanine from one strand pairing with cytosine in the other strand and vice versa (Watson and Crick, 1953). The genetic code that determines how an organism functions is held in one strand as a triplet code via the specific order of the four different DNA bases in that strand. The other strand of the double helix holds complementary information via the precise base pairing properties of the double helix (Watson

and Crick, 1953). The DNA sequence of an organism is arranged into functional units termed genes. Each gene holds the information for a specific protein or gene product which in turn has a precise role in that organism.

2.5 This complementarity is essential when the DNA in a cell is replicated for cell division; the two strands of the double helix separate and each has a new strand made using the specific base pairing properties. This ensures the genetic information in cells is maintained during cell division and remains relatively constant. When two adjacent pyrimidines in the same strand absorb UV radiation to become covalently linked the precise pairing properties between these bases and those in the opposite strand of the helix become disrupted and can result in mutation (Friedberg et al, 2006; Leroi, 2003; Maki, 2002; Taggart and Starr, 2006). Mutations are permanent changes in the genetic code that change the functionality of a gene's product.

2.6 Mutations occur when such damage is misreplicated by the DNA replication machinery which is responsible for duplicating the genome when cells divide. As mentioned above, it is essential to replicate the DNA accurately when cells divide so as to maintain the integrity of the genome. If the damaged bases are not recognised during DNA replication, then misreplication will occur to change the genetic code permanently. In other words, damage that may have been repairable is now converted into a permanent change – a mutation (Leroi, 2003; Maki, 2002; Taggart and Starr, 2006).

2.7 Irrespective of the UV radiation wavelength, the DNA damage induced causes mutation and skin cancer incidence is related to exposure (see paragraph 2.10).

2.8 Cell division is highly regulated in complex organisms. For example, in adult humans some of the organs have little cell division (eg the brain), whereas in organs such as the skin and gut, cells are constantly replicating to replace those cells that are lost by wear and tear. If mutations arise in genes that control cell division, then diseases resulting from uncontrolled cell division, including cancer, can be the outcome. These cancers can affect the functioning of the organ in which they arise, and if the cancer cells spread to other tissues then secondary tumours may arise with dramatic consequences for the individual (Weinberg, 2006).

2.9 Because of the deleterious effects of DNA damage, living organisms developed DNA repair mechanisms at an early stage of evolution; such mechanisms operate in simple bacteria through to humans (Friedberg et al, 2006; Reed and Waters, 2003). However, these mechanisms are not perfect and damage is not always removed efficiently from the genome. Hence, because un-repaired DNA damage can cause cancer and because DNA damage also has a role in ageing, the avoidance of exposure to sources of DNA damage is recommended so as to reduce the risks of cancer and premature ageing.

2.10 A classic illustration of the importance of repairing UV-radiation-induced DNA damage is seen in the genetic disease xeroderma pigmentosum. Children born with this rare condition cannot repair solar UV-radiation-induced DNA damage because they have mutations in genes coding for components of DNA repair. As a result they have a 2,000-fold elevated risk of skin cancer from exposure to sunlight and they frequently die from these cancers at an early age unless sunlight exposure is severely restricted (Friedberg et al, 2006; Reed and Waters, 2003).

2.11 We have known for some time that solar UV radiation induces DNA damage and in 1992 the International Agency for Research on Cancer classified solar UV radiation as a human carcinogen (IARC, 1992). Broad-spectrum UV radiation was also listed as known to be a human carcinogen in the 11th Report on Carcinogens of the US National Toxicology Program (2005). This classification was based on a great deal of scientific data that showed solar UV radiation causes increases in mutations in numerous organisms (Friedberg et al, 2006). Some of these mutations occur in genes that regulate cell division and as a result they can cause cancer. Epidemiological studies link sun exposure to increases in skin cancer (IARC, 1992).

2.12 Today there is extensive information on the induction of skin cancer by solar UV radiation (see Chapter 3), be it from the sun or from sunlamps that emit either different or the same spectrum of light as that from the sun. These events have been studied in animal models (Rass and Reichrath, 2008) and in humans (Cleaver and Crowley, 2002; de Gruijl et al, 2001), and a substantial effort has gone into identifying the contributions of the various components of the solar UV radiation spectrum to the different types of skin cancer (Cleaver and Crowley, 2002; de Gruijl et al, 2001; Rass and Reichrath, 2008).

2.13 There are three common forms of skin cancer – basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and malignant melanoma. BCC and SCC are also classified as non-melanoma skin cancers (NMSCs). BCC is a slow growing, locally invasive skin cancer and is the most common skin cancer as well as being the most common cancer of fair-skinned populations. Most BCCs are relatively benign and metastasis is rare. A major risk factor for BCC development is a patient's cumulative exposure to UV radiation. Significant local tissue destruction and disfigurement can occur and surgical excision is often employed in the treatment of BCCs. As the majority occur on the face, facial surgery with subsequent scarring can be a serious undertaking (Ceilley and Del Rosso, 2006). SCC is the second most common skin cancer and is more frequently found in older people. The most likely sites of cancer are the areas of skin often exposed to the sun. SCCs are also capable of metastatic spread, and when this occurs it complicates treatment and can pose a threat to life. For SCC, chronic UV-radiation-induced skin damage such as photoageing is an accepted predisposing factor. SCCs can be treated with surgery or radiotherapy (Mackie, 2006). Malignant melanoma is one of the most dangerous types of cancer as it is fast growing and spreads quickly, and can be lethal. UV radiation is the principal environmental cause of malignant melanoma. A recent study by Quintana et al showed that around 25% of melanoma cells had the potential to proliferate extensively and produce new tumours, even from single cells. However, it is not known if this level of disease progression would occur within patients (Quintana et al, 2008). Approximately 95–98% of melanomas are sporadic; however, 2–5% develop in melanoma families. Superficial spreading melanoma is the most common form of melanoma, with evidence implicating short, sharp episodes of burning exposure at an early age as a major risk factor (Mackie, 2006).

2.14 The incidence of skin cancer in humans has increased in recent times in a number of countries. A classic example, applicable to the sunny climate and personal behaviour patterns, is the incidence of skin cancer in Australians, which has steadily increased due to sun exposure. In the UK we have seen rises also, with at least 81,500 cases of basal and squamous cell carcinomas and 10,400 cases of malignant melanoma in 2006. Over the last 20 years incidence rates have more than doubled. This makes skin cancer the most frequently occurring form of cancer in the UK, particularly as the actual number of people with non-melanoma skin cancer is estimated as at least 100,000 per year as this

form of cancer tends to be under-reported (Cancer Research UK, 2008a). The incidence of basal and squamous cell carcinoma results in a substantial burden on the NHS for diagnosis and treatment, estimated at almost £58 million in 2002, compared with £13 million for melanoma (Morris et al, 2005). The costs of eye disease, to which solar UV radiation and sunbeds can contribute, are also substantial.

2.15 Melanoma often occurs at a younger age than many other cancers, resulting in a higher average number of years of life lost for deaths from this aggressive and often fatal disease. The average years of life lost in the UK for melanoma has been calculated as over 15 years per death, indicating that the impact is high (Burnet et al, 2005). If the indirect costs (patient costs, morbidity and mortality costs) are included with the costs to the NHS, the total estimated cost of skin cancer in the UK for 2002 was in excess of £190 million, with 63% of this value due to malignant melanoma (Morris et al, 2005). Over a period of six years, six cases of deaths attributed to NMSC among people below the age of 24 years were reported by the Office for National Statistics in England and Wales, against 72 cases of deaths due to melanoma (SWPHO, personal communication, 2009). The increase in all skin cancers is mainly attributable to increases in sunbathing, although there is undoubtedly a component of increased risk due to sunbed usage – the topic of this report and on which further information is contained in the subsequent chapters.

2.16 Solar UV radiation additionally induces cell or tissue damage and, in contrast to cancer induction, this affects almost all individuals who have excessive exposure, resulting in signs of premature skin ageing. In the dermis, UV radiation causes collagen to break down at a higher rate than with chronological ageing. Sunlight damages collagen fibres and results in the accumulation of abnormal elastin. When sun-induced elastin accumulates, metalloproteinase enzymes are produced in large quantities. Normally, metallo-proteinases remodel sun-injured skin by manufacturing and reforming collagen. This process does not always work effectively and some of metalloproteinases can actually break down collagen. The result of this is the formation of disorganised collagen fibres, namely solar scars. When this imperfect rebuilding process is repeated many times it results in the development of wrinkles (Fisher et al, 2002). Although mainly thought of as a cosmetic effect, skin damaged by UV radiation from natural or artificial sources can heal less well and it has an increased frequency of issues related to the ageing of this tissue (Fisher et al, 2002).

2.17 In addition to effects on the skin, exposure to solar UV radiation is related to the induction of other diseases, notably cataracts, pterygia and cold sores (WHO, 2009). Cataracts are an eye disease where the lens becomes increasingly opaque, resulting in impaired vision and eventual blindness. Long-term sun exposure to the eye increases the risk of developing a specific cataract type called cortical cataract, and 5% of the cataract-related disease burden is directly attributable to UV radiation exposure (Neale et al, 2003). UV radiation exposure is also related to pterygia (McCarty et al, 2000). This is a growth progressively encroaching across the surface of the eye and UV radiation exposure is accountable for 40–70% of this disease. Lastly, reactivation of herpes of the lip is also attributable to UV radiation exposure, which causes immunosuppression and reactivation of the herpes simplex virus, resulting in ‘cold sores’ (Ichihashi et al, 2004). Up to 50% of the manifestation of this disease is attributable to UV radiation exposure.

CHAPTER 3

DETRIMENTAL HEALTH IMPLICATIONS OF ULTRAVIOLET RADIATION EXPOSURE

The severity and consequences of UV radiation damage vary considerably between individuals.

Effects of UV radiation exposure may take years to develop and persist permanently.

High dose rate UV radiation exposure is one of most important risk factors for melanoma.

Skin type, increased numbers of moles and family history are also melanoma risk factors.

UV radiation exposure exacerbates the normal ageing processes of skin resulting in photoageing.

UV radiation exposure can also result in immunosuppression and eye damage, including cataract formation.

Biological basis of health effects

3.1 The effect of UV radiation on biological systems and melanoma induction is mediated by an incompletely understood array of mechanisms of great complexity. Some of these are discussed, albeit in simplified form, in order to outline the processes which lead to tumour formation. Attention will also be drawn to the other effects of UV radiation exposure which have implications for health beyond the induction of neoplasia, and which are less well known to the general public.

3.2 A particular difficulty of assessing melanoma risk in relation to external factors is that there are a large number of variables, some known, and many unknown, which contribute to risk, both to the population and to the individual. Some of these are understood, and can be used to guide advice. Many are suspected, but not proven, and although evidence may sometimes be of poor quality, adoption of a precautionary approach (the precautionary principle) requires all evidence to be critically assessed and taken into account.

3.3 The biological effects of UV radiation depend on such factors as skin type; degree of natural pigmentation; genetic factors; age; dose, frequency and duration of exposure; and severity of acute damage. Cellular repair mechanisms, which may be subject to individual variation, are known to be of considerable importance. Individuals with defective DNA repair, such as sufferers from the disease xeroderma pigmentosum (XP), are highly susceptible to skin cancers, with a 1,000-fold increase in risk of developing basal and squamous cell carcinomas and malignant melanoma before the age of 20 years (Kraemer et al, 1994). Such patients have extreme sensitivity to UV radiation, and develop many skin tumours at a young age unless rigorously protected. There is likely to be variation between individuals which moderates risk of skin cancer. Individuals not suffering from XP repair most of the DNA

damage from UV radiation, with the repair generally being efficient. Thus, the findings with XP patients demonstrate that UV radiation is able to cause tumours in man.

3.4 Animal models, including the platy fish *Xiphophorus*, show that UV radiation exposure is capable of inducing melanomas. The mechanisms may differ from human melanoma, however, but the model is a proof of principle. There are additional data from transgenic mice models which have allowed researchers to investigate the relative roles of UVA and UVB in carcinogenesis, albeit with as yet inconclusive results (Besaratinia et al, 2008; Li et al, 2009; Wang et al, 2008). However, XP mouse models have clear increases in UV-radiation-induced skin cancer and reflect the human situation (Marchetto et al, 2004; van Steeg et al, 2001).

3.5 UV radiation effects on DNA in man include strand breaks, damage from reactive free radicals, and alterations to the DNA bases themselves. Damage to genes occurs, and although some degree of repair takes place, damage of this type is well established as a precursor to malignant transformation. UV radiation damage effectively leaves a damage signature on a number of key genes in keratinocytes, including *p53*, which are known to play fundamental roles in human cancer. Genomic studies in melanoma have identified a number of mutations important in carcinogenesis, such as V-raf murine sarcoma viral oncogene homologue B1 (BRAF), and some evidence indicates that these genetic events are related to UV radiation exposure (Dhomen and Marais, 2007). Between 50% and 70% of human melanomas may be triggered by the acquisition of a mutation in BRAF, as a founder event in melanomagenesis (Dhomen et al, 2009).

3.6 Other biological effects of UV radiation exposure include immunosuppression. Experiments in mice show that cutaneous hypersensitivity reactions to chemical sensitisers are reduced as a result of UV radiation exposure, and grafted tumours from syngeneic mice are not rejected. The key work in the field was first reported by Fisher and Kripke who described work in animals which suggested that exposure to UV radiation may result in local and systemic immunosuppression (Fisher and Kripke, 1977, 1982) and that the nature of the immunosuppression is particularly towards tumour antigens induced during photocarcinogenesis (Fisher and Kripke, 2002). This tumour graft model is especially interesting. It was found that in certain mouse strains, tumours could be induced in the skin by UV radiation exposure. These should in theory be transplantable to other mice with the same genetic makeup (syngeneic) but in fact the majority were rejected, showing that the tumours were sufficiently immunogenic to elicit a rejection response from the recipient. If recipients were exposed to UV radiation prior to the transplant, the establishment rate of transplanted tumours was much higher. More recently, the mechanisms of UV-radiation-induced immunosuppression have been explored at a cellular level in man and it has been established that UV-radiation-induced T regulatory cells play a role, reviewed by Norval et al (2007).

3.7 Skin tumours in transplant patients who are immunosuppressed can sometimes grow unusually rapidly, and behave aggressively. The animal models discussed above suggest a mechanism by which UV irradiation might also suppress the immune control of tumours, or of cells in transition to an immunogenic malignant phenotype. The skin of the mice used in these experiments is close to human skin in terms of minimum erythemal dose, but because the experiments cannot be done in man, this work must remain somewhat conjectural in terms of possible significance to human malignancies.

3.8 Therefore, it is established that UV radiation can and does damage DNA, can induce melanomas in animal models and humans, and produces characteristic damage in key genes known to be implicated in human cancer. It can also produce immunosuppression which has implications for tumorigenesis and the aggressiveness of the malignant phenotype. The data available until 2002 on the immunosuppressive effects of sunlight were reviewed by the Advisory Group on Non-ionising Radiation (AGNIR, 2002).

Clinical aspects of UV radiation injury

3.9 Skin pigment is produced in melanocytes, a cell type which is distributed in the basal layer of the epidermis. The pigments are termed melanins, and several subtypes have been characterised such as eumelanin and phaeomelanin. Melanosomes are intracellular structures that contain the melanins, and the melanocytes themselves have dendritic processes along which the melanosomes travel to keratinocytes as skin pigmentation increases. Dark-skinned people do not necessarily have more melanocytes, but they do have more pigment distributed within the melanocytes than fair-skinned people, and the pigment is differently distributed in the cell. The melanin is thought to act as an optical filter, and provides protection at the cellular level from some of the damaging effects of UV radiation.

3.10 The amount and distribution of melanin in tanned fair skin differs markedly from the melanin in a natural dark skin. Tans do not provide the same degree of protection as a naturally pigmented skin (Emmett et al, 2008). Dark-skinned individuals are not by any means immune to sunburn, but tend to suffer less damage and are at a much lower risk of skin cancer than their fair-skinned counterparts. *De novo* production of melanin is a response to UV-radiation-induced skin injury. Other types of response to skin injury occur, including skin thickening and loss of elasticity, and the induction of types of skin cancer such as basal and squamous cell carcinomas.

3.11 Variation in the effectiveness of UV radiation repair mechanisms between individuals occurs to a lesser degree in the normal population; however, some normal individuals may still be at greater risk than others, especially those from fair-skinned populations. Such variation in the capacity for DNA repair is seen in susceptibility to sunburn, and occasionally in unexpectedly severe skin responses to radiotherapy.

3.12 War veterans, who served in the Far East and were prisoners of war (PoW) during World War II are often recognisable by the skin damage resulting from solar UV radiation exposure. Such skin damage exacerbates the normal changes of thinning and atrophy, which take place as skin ages and is termed photoageing. PoW skins often have pre-malignant and frankly malignant changes in many sites owing to the field changes in exposed areas of skin (Page et al, 2000). Cutaneous effects of large cumulative sun exposure in these individuals took many years to manifest as skin damage and there are therefore concerns that short-term perceived ‘benefits’ of tanning may carry a significant price later in life in susceptible people.

3.13 It is not only the skin that sustains damage from UV radiation exposure. Long-term eye damage can also occur. Eye damage, including cataract formation, may occur where eye protection is not worn, and has been reported in populations at special risk, such as Aboriginal communities in Australia (Taylor, 1980). Ocular damage from radiation is reflected by pathologies known to be related to UV radiation exposure, which include pterygia (a soft tissue overgrowth on the surface of the eye), cataracts and melanomas of both anterior and posterior ocular segments. Eye irritation, photokeratitis and conjunctivitis may occur in intense sunshine and in sunbed

users who reject eye protection. Long-term eye damage has been demonstrated in Aboriginal communities in Australia (Taylor, 1980, 1981), and in those occupationally exposed to UV radiation, such as welders (Guenel et al, 2001; Tenkate and Collins, 1997; Zlateva et al, 1996). There are data to suggest that sun exposure increases the risk of some eye diseases such as cataracts, and conjunctival neoplasms, reviewed by Gallagher and Lee (2006) and by the AGNIR (2002).

Epidemiological data on melanoma risk in relation to UV radiation exposure

3.14 The relationship between sun exposure and melanoma risk has been covered in detail elsewhere (AGNIR, 2002), but we will summarise the epidemiological observations made and the more recent progress in the field. This report is intended to review the current state of knowledge about the effects of artificial UV radiation exposure, with special reference to human health risks. This is therefore an overview with conclusions which embody the precautionary principle, where a degree of irreducible uncertainty in the data remains.

3.15 Melanoma is largely a cancer of fair-skinned people so that geographical variation in incidence is marked and the areas of highest incidence are those in which fair-skinned people live in close proximity to the equator (IARC, 1997). In England there is a significant relationship between decreasing latitude and the incidence of melanoma once ethnicity is taken into account (Brown et al, 2004). Furthermore, the more common types of melanoma (superficial spreading, nodular and lentigo maligna melanomas) are all seen almost exclusively in the pale skinned. Melanoma which occurs in darker-skinned people is usually of the acral lentiginous type: occurring on the sole of the foot or arising from the nail plate. The aetiology of acral lentiginous melanoma is poorly understood, not least because its rarity precludes epidemiological studies of sufficient size.

Incidence and mortality

3.16 There has been a steady rise in the incidence of melanoma of the skin in many areas of Australia, New Zealand, North America and Europe since the 1950s. The most recent incidence figures are available online from the Globocan 2002 project at the Cancer Mondial Statistical Information website (http://www-dep.iarc.fr/) through the International Agency for Research on Cancer (IARC). The highest incidence is seen in Australia and New Zealand. There has been a levelling off in incidence in many areas of Western Europe in recent years, but in Eastern and Southern Europe levels are still increasing (de Vries et al, 2003). In the UK, there is some evidence for a levelling off in some areas but not in all (Downing et al, 2006). Despite the increase in incidence in the north of the UK recently, mortality seems to be levelling off (Downing et al, 2006). Within England, there are notable regional differences in incidence. In 2004, the South East and South West showed the highest incidence levels for melanoma, although the North East had the largest increase over the ten-year period to 2004 (SWPHO, 2008).

3.17 It is widely reported that melanoma is relatively common among younger adults and tends to stabilise after about the mid-50s. This is in contrast to many other cancers where there is a steady increase in incidence throughout most of life. However, the incidence of melanoma, unlike that of many other cancers, is confounded by birth-cohort effects and, if adjustment is made for this effect (Dennis, 1999), melanoma rates do increase throughout life due to cumulative effects on the immune system and damage due to UV radiation exposure.

3.18 The crude incidence in the USA increases from around 1 in 100,000 at age 20 years to 5 in 100,000 at age 50 (Lachiewicz et al, 2008). Data from

Australia show an incidence of 3.4 in 100,000 in children, with the highest incidence ever reported in Queensland of 9.2 in 100,000. Most of the cases were in 13 and 14 year olds. Cases occurring before puberty, however, are exceedingly rare. With other types of injurious radiation, the evidence indicates that damage may have an inverse relationship to age at exposure. The effects of UV irradiation may take years to develop, and persist for years and remain irreversible for the rest of life. This lag time effect would be expected to produce very few tumours close to the time of exposure. Data show that melanoma accounts for 1.3% of all cases of cancer in patients under the age of 20 years. However, in 15–19 year olds, melanoma accounts for up to 7% of all cancers. Data from the South West Public Health Observatory cancer registry show that the percentage of all cancers diagnosed as melanoma in 15–19 year olds has risen over the last five years from 4.6% in 2002 to 13.33% in 2006 with an average over the five years of 9.6% (SWPHO, personal communication, 2009). The risk of sun exposure may in fact be greatest in the youngest age groups, but the effects may not be evident until later in life. These factors should also be considered to commend application of the precautionary principle (McWhirter and Dobson, 1995).

At-risk phenotypes

3.19 Within the fairer-skinned populations, those individuals with skin which is more susceptible to sunburn, are at greater risk of melanoma. The Fitzpatrick skin type scale is usually used to categorise skin so that type I (skin which always burns in the sun) is particularly associated with skin cancer risk (see Table 3.1). Phenotypes which correlate with sunburn risk have been established as risk factors for melanoma in many case–control studies, and 60 of these studies have recently been subject to a meta-analysis by Gandini et al (2005c). Gandini et al reported that Fitzpatrick skin type was associated with increased risk. For Fitzpatrick skin type I, the increased relative risk (RR) was 2.09 (95% confidence interval, CI, 1.67–2.58) compared with type IV. Freckling is an acquired phenotype in people with very fair skin who have been exposed to the sun and a high density of freckles was also associated with risk (RR = 2.10 for the highest freckling score compared with the lowest, 95% CI 1.80–2.45). Eye colour was less strongly associated with risk (blue vs dark: RR = 1.47, 95% CI 1.28–1.69) but hair colour was a good predictor of risk (red vs dark: RR = 3.64, 95% CI 2.56–5.37). These analyses were univariable ones and these risk factors are correlated and are putatively controlled by the same genes.

Table 3.1: Fitzpatrick skin type scale

Skin type	Skin colour	Characteristics
I	White; very fair; red or blond hair; blue or hazel eyes; freckles	Always burns, never tans
II	White; fair; red or blond hair; blue, hazel, or green eyes	Usually burns, tans with difficulty
III	Cream white; fair with any eye or hair colour; very common	Sometimes mild burn, gradually tans
IV	Brown; typical Mediterranean skin	Rarely burns, tans with ease
V	Dark brown; Middle Eastern skin types	Very rarely burns, tans very easily
VI	Black	Never burns, tans very easily

3.20 The Fitzpatrick scale is a useful tool for standardisation of skin types and has a correlation with risk from UV radiation. However, a recent study showed that white fair-skinned children were seven times as likely to sunburn as a group, matched for Fitzpatrick type, with children whose parents described their ethnicity as Hispanic. This suggests that even a small amount of skin pigmentation due to ethnic origin may have a significant protective effect against the acute effects of UV radiation exposure (Emmett et al, 2008).

3.21 The best understood of the genes which control the expression of phenotypes associated with melanoma risk, is that which codes for the melanocortin receptor, MC1R. The melanotropic hormones ACTH and MSH bind to this receptor and stimulate pigmentation (Suzuki et al, 1996) and melanocyte proliferation. The agouti protein (ASIP) also binds to the receptor (Abdel-Malek et al, 1999) where it acts as an antagonist (Jackson et al, 2006). There are numerous publications linking inheritance of variants at the MC1R locus and some of the melanoma phenotypic risk factors, namely red hair and freckles. However, there are many genes known to modulate pigmentation in mammalian species and the complexity of the genetic determination of pigmentation remains to be elucidated fully (Rouzaud and Hearing, 2005). A recent genome-wide association study (Sulem et al, 2007), for example, performed in samples from Northern Europeans, provided further supportive evidence for a role for MC1R in controlling human pigmentation and for variants in the oculocutaneous gene previously identified as a gene having a role in determining eye colour (Duffy et al, 2004), but also identified the tyrosinase gene and other loci as those controlling pigmentation.

3.22 The relationship between inheritance of MC1R variants and red hair was subject to a meta-analysis recently published (Raimondi et al, 2008). Nine studies on MC1R and phenotype were included in the analysis. The MC1R variants p.R160W and p.D294H were associated with both red hair and fair skin, while p.D84E, p.R142H, and p.R151C were strongly associated with red hair only – odds ratios (ORs) ranged from 2.99 (95% CI 1.51–5.91) for p.D84E to 8.10 (95% CI 5.82–11.28) for p.R151C. Inheritance of variants at the MC1R locus have also been shown to correlate with the presence of freckling (Bastiaens et al, 2001). MC1R variation is notably absent in African populations, which has been attributed to strong functional constraint near the equator where red hair and sun-susceptible skin types would be very deleterious (Harding et al, 2000). There are therefore good data to support the view that variants at the MC1R locus play a significant role in determining the presence of red hair and freckles, which are two correlated phenotypic risk factors for melanoma; however, there are other genes whose contributions to risk are yet to be fully understood.

3.23 The inheritance of MC1R variants also appears to be associated with melanoma risk as would be expected, and newer studies have attempted to investigate the biological basis of this relationship. In a recent meta-analysis, eleven studies were included in a study of the relationship between MC1R variants and melanoma risk (Raimondi et al, 2008). The seven variants, p.D84E, p.R142H, p.R151C, p.I155T, p.R160W, p. R163Q and p.D294H, were significantly associated with melanoma development, with ORs ranging from 1.42 (95% CI 1.09–1.85) for p.R163Q to 2.45 (95% CI 1.32–4.55) for p.I155T. No association with melanoma or phenotype was found for p.V60L and p.V92M variants. In conclusion this meta-analysis provides evidence that some MC1R variants are associated with both melanoma and phenotype, while others are only associated with melanoma development. These results suggest that MC1R variants could play a role in melanoma development via both pigmentary and non-pigmentary pathways.

3.24 The single most potent phenotypic risk factor for melanoma is, however, the presence of increased numbers of melanocytic naevi (moles). An abnormal naevus phenotype has been recognised in families with melanoma and in apparently sporadic melanoma. In families, this was first recognised in the 19th century (Norris, 1820), but was described in detail by Wallace Clark and by Lynch in the 1970s (Lynch et al, 1975; Wallace et al, 1973). Within families with mutations in high penetrance melanoma susceptibility genes such as CDKN2A, gene carriers are more likely to have increased numbers of naevi than non-mutation carriers (Newton Bishop et al, 1994), but the correlation between the presence of the abnormal naevus phenotype and melanoma risk/ mutation carrier status is poor (Bergman et al, 1986; Newton Bishop et al, 1994). It seems likely that CDKN2A controls the naevus phenotype only in part and that there are likely to be other naevus genes co-segregating in these families.

3.25 In population terms, the presence of increased numbers of naevi and clinically atypical naevi (those which are larger in diameter than 5 mm with an irregular or diffuse edge and variable pigmentation) are associated with increased melanoma risk. Many studies have investigated the relationship in different countries. Again, a meta-analysis has been published recently of data extracted from 46 studies published before September 2002. The number of common naevi was confirmed to be an important risk factor with a substantially increased risk associated with the presence of 101–120 naevi compared with less than 15 (pooled relative risk (RR) = 6.89, 95% CI 4.63–10.25) as was the number of atypical naevi (RR = 6.36, 95% CI 3.80–10.33; for 5 versus 0) (Gandini et al, 2005a). Twin studies have provided good evidence that the naevus phenotype is largely genetically determined (Bataille et al, 2000; Easton et al, 1991; Wachsmuth et al, 2001; Zhu et al, 1999), although with some additional effect of recreational sun exposure (Wachsmuth et al, 2005). Naevus genes are therefore hypothesised to be low penetrance melanoma susceptibility genes.

3.26 Family history of melanoma is a risk factor for the disease: in a meta-analysis the presence of any family history was associated with a relative risk of 1.74 (95% CI 1.41–2.14) (Gandini et al, 2005c). In a population study, based upon data from Swedish cancer registries, the standardised incidence ratios for offspring of a melanoma case were 2.40 (95% CI 2.10–2.72) when only the parent had melanoma and 2.98 (95% CI 2.54–3.47) when only a sibling was affected; when both a parent and a sibling were affected the standardised incidence ratio was 8.92 (95% CI 4.25–15.31) (Hemminki et al, 2003).

3.27 A list of at-risk groups advised not to use sunbeds is given in Table 3.2.

Table 3.2: At-risk groups for melanomas and NMSCs

Individuals with Fitzpatrick skin type I
Individuals with Fitzpatrick skin type II and freckles
The presence of increased numbers of melanocytic naevi (moles)
Individuals with a family history of skin cancer
Individuals with a history of sunburn, particularly in childhood
Individuals under the age of 18 years
Individuals taking medicines or using creams that sensitise the skin to sunlight
Individuals with a medical or genetic condition that predisposes them to skin cancer
Individuals with extensive skin damage due to sunlight

Melanoma risk associated with sun exposure

3.28 The geographical variation in melanoma incidence is indicative of a relationship between sun exposure and risk, the incidence being greatest where fair-skinned populations live in greatest proximity to the equator. UV flux (and personal dose) increases with proximity to the equator, in high altitude terrain and in circumpolar regions. There are large variations in daily personal erythemal exposure, more so for indoor workers living in northern Europe than for those resident in locations closer to the equator, which are due not only to seasonal changes in ambient conditions, but just as importantly to seasonal variation in behaviour. Not surprisingly, holiday and summer weekend exposures account for the largest daily UV radiation doses. For indoor workers living in northern Europe, a typical annual exposure is estimated to be about 150 standard erythemal doses (SED), with a corresponding estimate of 400 SED for indoor workers in Florida (Diffey, 2008). A relationship between increased sun exposure and melanoma risk within populations has been reported in a number of case–control studies. Very importantly, most studies have shown that high dose rate UV radiation exposure, such as is experienced on holiday, is the most important risk factor for melanoma, and that occupational sun exposure is either neutral or protective at least in temperate climes (Gandini et al, 2005b). The exception may be in areas of extreme sun exposure such as Queensland, Australia, where there are some data suggestive of total (cumulative) sun exposure as a risk factor for melanoma (Green et al, 1985). The lack of a dose–response curve for melanoma and sun exposure has proved controversial and confusing, but the data are consistent across studies. Furthermore, in the case–control studies the relationship between vacational or recreational studies and risk has been consistently accompanied by a clear relationship between reported sunburn and melanoma risk (Gandini et al, 2005b; Green et al, 1985), which in many studies has been stronger for reported sunburn in childhood. Thus, the epidemiological studies have provided evidence that the pattern of sun exposure most strongly associated with melanoma risk is intermittent and that those with fair skin who have burnt in the sun are at risk. Moreover, sunburn in childhood is a particular risk.

3.29 It should be stated that a view is emerging that there are, however, two routes to melanoma, the more common being associated with high dose rate UV radiation exposure, sunburn and for which increased numbers of naevi are a risk factor, and a less common type associated with chronic sun exposure in continuously exposed body sites such as the head and neck (Bataille et al, 1998; Whiteman et al, 2003).

Risk of squamous cell carcinoma (SCC)

3.30 Squamous cell carcinoma of the skin has a much more simple relationship with sun exposure than has melanoma, such that there is a dose–response relationship: SCC is associated with increased cumulative exposures (Armstrong and Kricker, 1993; Leiter and Garbe, 2008). Other aetiological factors of importance include chronic immunosuppression as is seen increasingly frequently in organ transplant recipients. SCC occurring in such patients may exhibit unusually rapid growth, presumably because of a lack of normal immunological control mechanisms (Kuijken and Bouwes Bavinck, 2000).

Health implications of UV radiation exposure

3.31 In summary, the severity and consequences of UV radiation damage vary considerably between individuals. UV radiation exposure in man leads to skin cancer of at least three types, two of which can be life-threatening, and also to photoageing of skin. Significant immunosuppression occurs in mouse models, and if a similar effect exists in man, it has important implications. Several types of serious eye pathology are also related to UV radiation exposure, including malignant melanomas of the eye itself. There is good evidence that the dominant pattern of sun exposure associated with risk of melanoma internationally, is intermittent sun exposure such as occurs in indoor

workers who spend vacational time or leisure time in the sun. The evidence suggests that the risks are greater in those with skin which is susceptible to burning (and for which the phenotypic markers include red hair and freckles), and in those with larger numbers of melanocytic naevi and naevi which are clinically atypical. Family history of melanoma increases the risk. Reported sunburn is furthermore associated with risk and the biological data suggest that severe responses to sun exposure, such as occurs with sunburn, lead to increased risk and it is hypothesised that immunological changes following sunburn may be important in melanoma pathogenesis. There are data to suggest that chronic or cumulative sun exposure is important for cancer risk, particularly for melanoma of the head and neck in the elderly and, importantly, squamous cell carcinoma. In addition, this type of exposure is directly related to other important health effects such as photoageing, pterygium, cataracts and other eye pathologies.

CHAPTER 4

HEALTH RISKS AND PERCEIVED BENEFITS RESULTING FROM SUNBED USE

Modern sunbeds are capable of producing irradiation equivalent to Mediterranean sunlight.

First use of sunbeds before the age of 35 years increases the risk of melanoma.

Extensive sunbed use is also associated with photoageing of the skin.

UVB exposure can synthesise vitamin D in the skin but this is dependent on a number of factors including age. Dietary supplements are the preferred alternative source.

Modern sunbeds can emit as low as 0.5% UVB and are not recommended for increasing vitamin D levels. Synthesis of vitamin D can reach a plateau after a few sunbed sessions.

Using a model, it is estimated that sunbed use accounts for approximately 370 new cases of melanoma and 100 deaths each year in the UK.

Sunbeds

4.1 Sunbeds are the most common type of artificial UV tanning device, consisting of a collection of UV radiation emitting lamps arranged horizontally so that the user can lie under (and/or on) the lamps. Most modern sunbeds expose the user from above and below simultaneously, to reduce overall session time. There are other types of tanning device, such as stand-up booths and tabletop facial solaria. These differ from sunbeds only in the body area exposed and in the physical orientation of the user.

4.2 The user is exposed to a quantity of UV radiation which is determined by factors such as the number of lamps, the power of each lamp, their position relative to the body, and the duration of the exposure session. These factors are all quantitative. The choice of lamps with differing output spectra may also result in quantitative effects on sunbed performance, as lamps with spectra richer in the shorter wavelengths can produce a greater erythemal effect for the same power consumption.

4.3 Most of the tanning lamps incorporated into sunbeds are of the low pressure mercury discharge type. These are outwardly similar to ordinary fluorescent lamps – the only difference being that the sunbed lamps have a fluorescent phosphor coating and outer envelope material chosen to maximise production and emission of long wavelength UV radiation. Many tanning units (sunbeds and walk-in types) consist of nothing more than large banks of long (up to 2 metres) low pressure lamps, usually mounted in a curved housing to try and ensure reasonably even exposure of the whole skin area.

4.4 Some sunbeds make use of high pressure lamps to irradiate the face. These lamps are more compact, and run at a higher temperature. They produce a spectrum that is richer in the more energetic shorter wavelengths of UV

radiation, and so it is essential that adequate filtration is provided. This is usually achieved by a combination of filters built into the device and provision of protective eyewear.

4.5 As products which may be used in a domestic setting, sunbeds fall within the scope of the current European Directive on Low Voltage (European Commission, 2006). This Directive stipulates, in an annex, that devices placed on the market (in Europe) must not produce radiation which could cause a danger. The definition of a dangerous level of radiation has been left to standardisation bodies. The current British and European Standard (BS EN 60335-2-27: 2003, British Standards Institution, 2003) contains a scheme for classification of sunbeds by the manufacturer: sunbeds which fit into this scheme are considered to be fit for the market. At the time of writing, the British Standard specifies that the spectral irradiance of a sunbed must be measured, from 250–400 nm, and then erythemally weighted. Sunbeds are then assigned to a type depending on their effective irradiance, as described in Table 4.1.

Table 4.1: UV appliance types

	Effective irradiance (W m^{-2})	
UV appliance type	**250–320 nm**	**320–400 nm**
1	<0.0005	≥0.15
2	0.0005–0.15	≥0.15
3	<0.15	<0.15
4	≥0.15	<0.15

4.6 The UV radiation types can be described as:

Type 1: biological effect is caused mainly by wavelengths above 320 nm, and irradiance is relatively high in the range 320–400 nm.

Type 2: biological effect is caused by all wavelengths, and irradiance is relatively high in the range 320–400 nm.

Type 3: biological effect is caused by all wavelengths, and irradiance is relatively low.

Type 4: biological effect is caused mainly by wavelengths below 320 nm.

4.7 The British Standard is intended for use by manufacturers of sunbeds, and does not make stipulations about the operation of sunbeds once they have been purchased. However, it does advise that type 1 and 2 sunbeds are considered appropriate for use in solaria, type 3 sunbeds are considered safe for home use, whilst type 4 sunbeds are intended for use only following medical advice.

4.8 Most of the sunbeds currently available produce irradiances which place them in the less powerful reaches of type 2 or the more powerful reaches of type 3. In the last few years, sunbeds which are too powerful to fit into type 3 have been developed, as a result of which the process of modifying the Standard is currently under way. In Europe (and hence in the UK), this work is

informed by the conclusion of the Scientific Committee on Consumer Products (SCCP, 2006) that the upper limit for sunbed irradiance should be 0.3 W m^{-2} (or 11 standard erythemal doses (SED) per hour, erythemally effective), the equivalent of 'tropical sun'. Regulatory bodies responsible for Marketing Authorisations in the various EU member states have already adopted this value as a performance limit for devices allowed on to the market, and the sunbed industry seems to be moving to comply with this.

4.9 A Swiss study surveyed the emission spectra of sunbeds and found that while the spectrum was similar to natural sunlight, the emission of UVA was increased by 1,015-fold, with an effective UV index* of 13, compared with the UV index of 8.5 for sunlight at noon at intermediate latitudes (Gerber et al, 2002). A survey of sunbed output showed variation in both UVA and UVB between beds, and also variation in output along the length of the tube in individual beds. The UVB output varied by a factor of 60 (Wright et al, 1996).

4.10 Modern sunbeds are capable of producing irradiation of at least the equivalent of Mediterranean sunlight. Furthermore, there is evidence of widespread breach of the British Standard, BS EN 60335-2-27: 2003, with a significant percentage of sunbeds exceeding the limits for a type 3 device (Oliver et al, 2007).

Sunbeds and melanoma risk

4.11 The well-established link between solar UV radiation exposure and melanoma incidence has raised concerns of similar risks to users of sunbeds. Since there is no satisfactory treatment for advanced melanoma, it is prudent to reduce risk as much as can reasonably be achieved or is reasonably practicable.

4.12 A recent systematic review emphasised the increased risk to sunbed users (IARC Working Group, 2007). First use of sunbeds before the age of 35 years increased the risk of malignant melanoma by 75% and an increased risk of squamous cell carcinoma was also determined. Higher risk behaviour has been documented in teenagers, with 60% experiencing a burn within the last year (De Vries et al, 2006) with repeated sunbed use in 26%. A survey of 4,000 members of the public by Cancer Research UK found that 82% of sunbed users first used a sunbed before the age of 35 years (Cancer Research UK, 2008d).

4.13 There are data from many studies, such as migrant studies, which suggest that early (childhood) sun exposure is particularly important in melanoma aetiology (Whiteman et al, 2001). If risk of melanoma is related to age at exposure to UV radiation, teenagers and younger children may be at significantly increased long-term risk. This is a cause for concern as it is not known whether the risk relates to the age at exposure or the age when sunburnt; however, use of sunbeds at a young age will increase the risk and the cumulative exposure. It is therefore of special concern that sunbed use occurs in very young primary school children (Hamlet and Kennedy, 2004), in whom the risks are unknown, but are likely to be as high or higher than in other groups.

4.14 A case–control study demonstrated significant risk for people aged under 45 years with fair skin, and highlighted a median time of seven years from first use of a sunbed to melanoma development (Bataille et al, 2004). The

* The UV index is a measure of the level of UV radiation, used to alert people about the need to use sun protection. The values of the index range from zero upward – the higher the value, the greater the potential for damage to the skin and eye, and the less time it takes for harm to occur (WHO, 2009).

interpretation of clinical studies would require a significant lag time from exposure. It will be years before the effects of current UV radiation exposures are seen, which emphasises the need for adequate and sustained follow-up of study populations.

4.15 A variety of commonly used medical drugs can sensitise the skin to UV radiation in susceptible patients (see Appendix B), as can the saps and extracts of various plants, some of which are common in the environment.

4.16 In addition, a number of products are advertised as tanning accelerators for use with sunbeds. These are normally topical creams designed to keep skin moisturised during the tanning process. Some contain collagen and claim to prevent premature ageing, while others produce a 'tingle', which apparently increases circulation to the skin surface, enhancing the tanning process. Some tanning accelerators contain the amino acid tyrosine, in the belief that it stimulates and increases melanin formation, thereby accelerating the natural tanning process.

4.17 A recent development for the tanning industry is the use of Melanotan and Melanotan II, analogues of the α-melanocyte stimulating hormone, as tanning accelerators. The products are designed to be injected and are promoted as increasing skin pigmentation without exposure to UV radiation; however, the process of tanning using these products is greatly expedited by UV radiation exposure. Both products are undergoing clinical trials but are not currently licensed in Europe or the USA. The US Food and Drug Administration issued a warning against the use of Melanotan II as an unapproved product in September 2007 (US FDA, 2007a). In August 2008 the Danish Medicines Agency published a warning over the use of Melanotan, as the effects and side-effects had not been fully investigated (Danish Medicines Agency, 2008). In the UK, the Medicines and Healthcare products Regulatory Agency has warned people not to use these unlicensed medicines (MHRA, 2008). There has been significant media coverage on the use of these products and the associated side-effects. A letter in the *British Medical Journal* drew attention to a complicating factor in presenting patients, with rapid changes in the appearance of pre-existing melanocytic naevi after injection of Melanotan and Melanotan II (Langan et al, 2009).

4.18 The epidemiological data on sunbed use and risk are much more limited than for sun exposure and risk. The number of studies which have addressed this potential risk factor is much smaller, and on the whole the detail collected about the duration and type of use is poorer. Furthermore, the UV radiation emissions of sunbeds are very variable and therefore it has been very difficult to account for this in the studies reported. A confounding factor in the studies is sun exposure, as it is difficult to disaggregate UV radiation exposure from sunbathing and from use of sunbeds.

4.19 Individual studies have produced variable estimates of risk associated with sunbed use. In a study of all comers to skin clinics, sunbed use was associated with an increased risk of melanoma, especially for women under the age of 45 years and where tanning sessions were longer than 20 minutes (Ting et al, 2007), although recruitment via skin clinics may reflect findings applicable to a somewhat selected group. Case–control studies were summarised in a review of 19 studies identified by literature search by the IARC Working Group (2007). Overall in this analysis, there was a positive association with ever having used a sunbed and melanoma (summary relative risk (RR) = 1.15, 95% CI 1.00–1.31), although evidence of a dose–response relationship was scant. First use of sunbeds before the age of 35 years significantly increased the

risk of melanoma, based on seven informative studies (summary RR = 1.75, 95% CI 1.35–2.26) (IARC Working Group, 2007). These data are persuasive of an effect of UV radiation exposure from sunbeds, but there were concerns that users of sunbeds were also likely to be sunbathers. In a recent study of sun-seeking behaviours in 7,200 French adults, for example, sunbed users also reported having had more sunburns after sun exposure (Ezzedine et al, 2008). Moreover, the sunbed users were more likely to have other risk factors for melanoma such as fair skin and indoor working. The IARC review of the 19 studies found eight studies in which the risk was adjusted for confounders related to sun exposure and sun sensitivity. In these studies the summary relative risk was similar to that from the overall analysis, giving a positive association with melanoma.

4.20 The data therefore on the risk of melanoma resulting from sunbed use are limited. Sunbeds, however, have been in widespread use for a relatively short period of time and there are concerns that as melanoma has a long latency period that the full measure of risk is not yet established (Autier and Boyle, 2008). Furthermore, use of a sunbed over long periods of time may result in huge doses of UVA and UVB exposure, equivalent to very large cumulative doses which could be greater than that achievable through holiday sun exposure.

4.21 The IARC data suggested that there was increased risk associated with sunbed use for individuals under the age of 35 years compared with older individuals (IARC Working Group, 2007) and there are data from elsewhere supportive of a special effect of sun exposure early in life. Melanoctytic naevi are a risk factor for melanoma as discussed above and the emergence and proliferation of these naevi is a feature of early life so that Autier and Boyle recently concluded that access to sunbeds should be prohibited for those under the age of 18 years (Autier and Boyle, 2008). A prohibitive approach may prove to be most effective as adherence to a voluntary industry code designed to limit access to sunbeds of populations at special risk in Australia was found to be widely ignored (Dobbinson et al, 2006; Paul et al, 2005).

Estimation of annual melanoma deaths attributable to sunbed use

4.22 Whilst definitive data on mortality due to sunbed use will remain unknown due to the confounding effect of sun exposure, it is possible by mathematical modelling to make a crude estimate of the number of melanoma deaths per year attributable to the cosmetic use of sunbeds. Using such a model it has been estimated (Diffey, 2003) that sunbed use might account for around 370 new cases of melanoma and 100 deaths (range 50–200) each year in the UK.

4.23 Despite some important caveats, the model attempts to estimate the health burden from cosmetic tanning and has the advantage of being applicable to other settings if the parameters are adjusted accordingly. As such, Gordon et al (2008) have replicated the model to derive an estimate of the annual incidence and mortality of melanoma as a consequence of sunbed use in Australia. They estimated 281 new melanoma cases and 43 melanoma-related deaths in Australia each year attributable to sunbed use. From the models, the estimated rates for melanoma-related deaths for the population as a consequence of sunbed use are consistent between the two countries.

4.24 A subsequent analysis assuming enforcement of solarium regulations, thereby effectively restricting use by young people under 18 years and prohibiting people with fair skin that burns easily, estimated between 18 and 31 melanomas would be avoided each year per 100,000 people (Hirst et al, 2008).

Other risks associated with sunbed use

Photoageing to skin

4.25 The association of extensive sunbed use with photoageing of the skin is seen regularly in clinical practice but is poorly documented in the literature. Pigmentary changes similar to sun-induced lentigos are, however, well described in patients treated with therapeutic UV radiation for skin diseases, such as psoralen and UVA therapy (PUVA) (Holzle, 1992).

4.26 Exposure to large amounts of artificial UV radiation in animals is associated with photoageing in the laboratory (Pfau et al, 1986). In humans there is evidence that long-term damage to mitochondrial DNA in elderly skin is related more to photodamage than chronological age (Berneburg and Krutmann, 1998) and more recently sunbed usage by volunteers who had previously not used these machines was shown to induce the same changes (Reimann et al, 2008). These biological data therefore provide measurable evidence of changes in the skin which are usually associated with ageing.

Risk of squamous cell carcinoma (SCC)

4.27 As noted previously SCC is associated with increased cumulative sun exposures (Armstrong and Kricker, 1993; Leiter and Garbe, 2008). Cohort studies of patients treated with PUVA have established a clearly increased dose determined risk of SCC (Lindelof et al, 1991; Stern, 1992).

4.28 The data on sunbed usage and SCC risk are compromised by a marked lack of large studies. Although the IARC Working Group pooled data on three studies and produced a summary relative risk of 2.25 (95% CI 1.08–4.70) for any use of a sunbed (IARC Working Group, 2007), the data overall are few.

Risk of ocular damage

4.29 The principal result of UV radiation exposure on the eye is corneal damage. The damage is generally limited to the epithelial cells of the cornea and the condition tends to be short lived.

4.30 Although the primary emission from sunbeds is UVA, which has a greater transmission than UVB, it is exposure to radiation in the 295–325 nm wavelength range of UVB that has been shown to induce cataract formation. Sunbeds produce limited 295–325 nm radiation, but the health risks should still be taken into account. It also should not be inferred that UVA is safe with respect to lens exposure. UVA can play a part in crystalline lens ageing with the loss of elasticity and browning.

4.31 The crystalline lens blocks UV radiation wavelengths below 400 nm and the cornea blocks wavelengths below 300 nm, protecting the retina from most of the UV radiation emitted from sunbeds. However, trace amounts of UVB radiation between 300 and 315 nm may still reach the retina. The use of protective goggles will prevent exposure of the eyes to harmful levels of UV radiation and the risk of ocular damage (ICNIRP, 2003).

Short-term adverse effects

4.32 UVA radiation emitted from sunbeds is quite capable of producing burns, as well as tanning, particularly in skin that normally burns in sunlight. One hour or more of UVA sunbed exposure can produce erythema, and sometimes blistering, in susceptible individuals, occasionally requiring admission to a hospital burns unit (B L Diffey, personal communication, 2008). In 2009 a 14 year old suffered first degree burns to 70% of her body after spending 19 minutes on a coin-operated sunbed in an unstaffed outlet (*BBC News,* 2009). Sunbed burns can result if the integral optical filters associated with arc lamps slip out of place, as was the reason for superficial to medium partial thickness burns reported in three women successively using such a faulty appliance (Eltigani and Matthews, 1994). Furthermore, the use of certain medications (Hawk, 1984) or lotions applied to the skin (Larsen, 1985) may increase the chance of an erythematous and/or eczematous reaction. Minor

side-effects such as itching, skin rashes or nausea have also been reported after using UVA tanning appliances (Devgun et al, 1982; Diffey, 1986; Rivers et al, 1989). Sunbeds can also cause the common photodermatosis polymorphic light eruption (Devgun et al, 1982; O'Toole and Barnes, 1995; Rivers et al, 1989) – a transient, irritating, papular reaction – and they exacerbate light aggravated dermatoses, such as systemic lupus erythematosus (Stern and Docken, 1986).

4.33 A more serious side-effect is the onset of skin frailty and blistering occurring in people who tan poorly in sunlight and who have used UVA sunbeds twice a week or more for one or more years (Farr et al, 1988; Murphy et al, 1989).

Sunbeds and sunburn

4.34 A history of sunburn has been reported as a risk factor in melanoma (Dennis et al, 2008). Since burning is not a common feature when tanning using UVA sunbeds, proponents of cosmetic tanning have taken this to imply that tanning with sunbeds is safer than in sunlight. Marked reddening of the skin ('sunburn') from sunlight occurs when the skin has received an unweighted UV radiation (290–400 nm) dose of 15 J cm^{-2} (equivalent to about three times the dose required to produce a just-perceptible reddening in unacclimatised white skin) or more. A similar exposure is delivered during each UVA sunbed session (McGinley et al, 1998), but the reason the skin does not generally burn is because of the low emission [around 0.5–1.5% of total UV radiation emission (Diffey, 1997)] at wavelengths less than 320 nm (the UVB component) from most UVA sunbeds. In comparison, the emission in this spectral region (below 320 nm) in summer sunlight from temperate to tropical latitudes is 4–6% of the total UV radiation energy.

4.35 However, lack of burning with sunbeds should not be taken as evidence that tanning with sunbeds is safer than in sunlight since burning *per se* is not necessarily associated with increased risk of melanoma but is merely a marker of a high dose of solar radiation exposure (principally UVA exposure). It is of note that patients undergoing courses of UVB phototherapy for psoriasis and other skin diseases, in which marked erythema ('burning') is a common feature of the treatment, do not appear to be at increased risk of skin cancer (Lee et al, 2005).

Perceived benefits of sunbed use

4.36 There seem to be psychological benefits of sunbed use and this probably drives their usage. The most popular reason for using sunbeds appears to be improving appearance. One study from Bradford, UK, suggested that usage was associated with perceptions of 'looking healthy' or 'looking better' (Amir et al, 2000). Similarly, a study from Sweden suggested that usage was greater in young people who were least satisfied with their body image (Brandberg et al, 1998). It might be concluded therefore that use of sunbeds to acquire a tan has psychological benefits associated with a perception that the person's appearance is improved, although it is clear that the psychological factors that influence the use of sunbeds are complex.

Psychological

Protection from sunburn

4.37 Many people use sunbeds before holidays in sunny countries in the belief that the sunbed-acquired tan will afford them protection from the sun, as well as 'improving' their appearance. The level of protection afforded by a sunbed tan is, however, small. In one study, the effects of exposure to a UVA sunbed three times a week for four weeks was compared in 31 normal subjects with those seen in nine control subjects exposed to sunbeds emitting visible light (Rivers et al, 1989). The mean protection factor against later UVB-induced erythema was 3.2 ± 0.3 after the UVA sunbed course and 1.6 ± 0.2 among the controls. During the course of the study significantly more adverse effects, such as pruritus, erythema, freckling, burning sensation, dryness and

polymorphic light eruption, were seen in the individuals exposed to a UVA sunbed. The changes found in both groups were attributed to small amounts of UVB emission from both active and control lamps. The level of protection was therefore limited and moreover was associated with morbidity.

Vitamin D synthesis

Background: vitamin D

4.38 Vitamin D is a prohormone which is essential to human calcium absorption and physiology and hence important for good health. It is available to humans in two forms: vitamin D_3 (cholecalciferol), which is formed very efficiently in the skin when exposed to UV radiation or is ingested in the form of fatty fish, and vitamin D_2, which is present in plants and is much less potent. The vitamin D precursor in the skin, 7-dehydrocholesterol, is activated by exposure to light to produce vitamin D_3. Vitamin D_3 produced in the skin or absorbed from the gut (supplied in fatty fish or dietary supplements) is then further hydroxylated in the liver to 25(OH)D and then in the kidney to produce the active hormonal form, 1.25-dihydroxyvitamin D_3 (Webb, 2006).

4.39 The relative contributions of dietary sources of vitamin D_3 or vitamin D_3 derived from sun exposure vary between populations and by season (at least at higher latitudes). In some areas of the world, such as North America therefore, many foods are fortified with vitamin D_3 and in others a high intake of fatty fish, such as herring, accounts for a significant proportion of the total contribution. However, despite the high levels of fortification in North America intakes are considered to be insufficient (Whiting et al, 2007). Within Europe, where the fortification is much lower, the reported levels have been lowest in the Republic of Ireland and the Netherlands, with the highest levels in Nordic countries where fish is an important part of the diet (Ovesen et al, 2003). Even in Australia, season was found to be a strong determinant of vitamin D status and vitamin D insufficiency was reported to occur over wide latitudes as dietary intake is so low (van der Mei et al, 2007). Insufficiency was reported to occur in some months when sun exposure protection would be advised.

4.40 Given the low dietary intake of vitamin D in most populations, humans are dependent on exposure to UV radiation for their serum levels of vitamin D_3. Thus, rickets resultant from vitamin D_3 deficiency was recognised as an English disease when pollution in the industrial north reduced exposure to the sun. Indeed Jablonski and Chaplin have argued persuasively that the need to synthesise sufficient vitamin D_3 in the skin was so great that it has driven the evolution of skin colour (Jablonski and Chaplin, 2000). The use of supplements varies widely between countries and data suggest that use of supplements in Europe is greater in Nordic countries than elsewhere (Ovesen et al, 2003). Bates et al suggested that in the UK only 16% of the elderly living at home and 3% living in care homes took supplements (Bates et al, 2003). A cross-sectional measure of the 1958 birth cohort measured at the age of 45 years, reported a high prevalence of vitamin D insufficiency during the winter and spring, when 25(OH)D concentrations of below 25, 40 and 75 nmol L^{-1} were found in 15.5%, 46.6% and 87.1% of participants, respectively; the proportions were 3.2%, 15.4% and 60.9%, respectively, during the summer and autumn (Hypponen and Power, 2007). The ability of the skin to activate 7-dehydrocholesterol (and therefore determine vitamin D_3 levels resultant from sun exposure) varies according to latitude, time of day, season, time outdoors, age (being reduced with increased age) and clothing worn. Serum vitamin D_3 levels in individuals are therefore determined by a complex interaction between diet, supplementation and sun exposure, but individual variation is as yet unexplored.

4.41 The optimal levels of vitamin D in the blood remain a little controversial: certainly the views as to what is ideal have changed over time.

Severe deficiency sufficient to produce rickets is associated with levels below 10 nmol L^{-1} but over time opinion has changed, so that the significance of higher levels in which rickets does not occur but at which measurable harmful effects on health can be demonstrated, has been recognised, and this has led to a re-classification. However, there is still variation and controversy with this, and this is discussed by Ovesen et al (2003).

4.42 Suboptimal vitamin D levels have been associated in recent years with muscle weakness (Gerdhem et al, 2005), an increased risk of some cancers (Giovannucci et al, 2006; John et al, 2007; Schwartz and Skinner, 2007) and an increased risk of autoimmune disease (Lips, 2006). A potentially important link to cardiovascular disease has also been identified (Dobnig et al, 2008; Giovannucci et al, 2008; Pilz et al, 2008). Furthermore, a recent meta-analysis of 18 randomised controlled trials suggested that vitamin D supplementation may reduce overall mortality (Autier and Gandini, 2007). A recent four-year randomised trial of supplementation with 1,100 international units of vitamin D (equivalent to two servings of fatty fish) and calcium in post-menopausal women in North America appeared to reduce the risk of cancer (Lappe et al, 2007). The supplementation was sufficient to raise serum vitamin D levels to more than 80 nmol L^{-1}.

4.43 Whilst the issues around level of risk remain highly controversial, there does seem to be sufficient data to suggest that low vitamin D levels should be avoided.

4.44 The data, however, suggest that suboptimal levels of vitamin D are common (Hypponen and Power, 2007; Reginster, 2005). In a recent Scottish study of older patients attending outpatient clinics for example, 72.6% had insufficient vitamin D levels (25(OH)D < 50 nmol L^{-1}) and 27.5% had levels which were frankly deficient (25(OH)D < 25 nmol L^{-1}) (Burleigh and Potter, 2006). Many health agencies have recognised this as a potentially very important health issue and there are discussions currently in Europe on dealing with this by dietary fortification.

Sunbeds and vitamin D levels

4.45 It has been suggested that use of sunbeds might be a useful means of correcting low levels of vitamin D in the population as it is proposed that a ten-minute session yields 2,000 to 4,000 international units (equivalent to four to eight servings of fatty fish or two to four pills of supplement) (Grant and Holick, 2005). However, the use of sunbeds to induce vitamin D synthesis is dependent on the level of UVB emissions, which can be variable as the primary emission from sunbeds is UVA radiation (Gerber et al, 2002; Oliver et al, 2007). A recent study showed that sunbeds emitting 0.5% and 1.4% UVB increased vitamin D levels, but this increase reached a plateau after a few sessions with later sessions hardly contributing to the effect (Thieden et al, 2008). In the trial up to 64% of the sunbed sessions resulted in additional side-effects, including erythema and polymorphic light eruption increasing with UVB dose and exposure time. It should be noted that six of the 33 subjects in the trial showed no increase in vitamin D levels during the sunbed treatments, possibly due to genetic factors reducing their ability to synthesise vitamin D. Although sunbeds could be used to augment vitamin D synthesis in some people, the study could not recommend this practice due to potential carcinogenicity and the high frequency of the acute side-effects.

4.46 The importance of having optimal levels of vitamin D in the serum for many aspects of health, and the demonstration that population levels of vitamin D in the serum are low in the UK as in much of Europe, means that supporting measures to raise levels should be addressed. Use of fatty fish and

supplements are a preferable means of increasing vitamin D levels in multi-ethnic populations around the world (Van Der Meer et al, 2008). Use of sunbeds is associated with vitamin D synthesis in the skin; however, ageing can decrease the ability of skin to synthesise vitamin D from UV radiation exposure, reducing the effectiveness of the sunbed session (MacLaughlin and Holick, 1985). Compared with suberythemal sun exposure and dietary intake, the use of sunbeds to augment vitamin D synthesis is, in relative terms, associated with too much risk in terms of skin damage, to be recommended.

4.47 Holick et al have suggested that artificial light sources may be useful for the induction of vitamin D synthesis in the skin for patients with fat malabsorption syndromes who are unable to absorb oral vitamin D (Holick et al, 2007); however, this uses high UVB lamps in a clinical setting and gives different UV radiation exposures to those received from a sunbed.

4.48 In 2005 the Advertising Standards Agency received a complaint against a leaflet from The Sunbed Association (TSA) on 'Vitamin D essential for good health – Sunbeds sessions ARE good for you'. The complaint was upheld, as it was understood that the claims for the beneficial effects of sunbed use were not generally agreed and it was not considered appropriate for TSA to advocate the use of sunbeds to prevent the development of serious medical conditions. TSA was asked to remove all claims relating to the medical efficacy of sunbed use (Advertising Standards Agency, 2005).

Balance of health risks and perceived benefits from the use of sunbeds

4.49 The perceived benefits of sunbed use are largely psychological and cosmetic. In terms of other health benefits, there is little value in terms of protection from sunburn. The practice of using sunbeds to increase vitamin D synthesis has not been recommended and indeed members of society who might benefit from additional vitamin D synthesis (the elderly housebound, long-stay hospital patients, ethnic groups with an indoor lifestyle, and those wearing sun-excluding clothing), are groups extremely unlikely to visit commercial sunbed outlets.

4.50 The epidemiological data described above have identified risk factors for melanoma. These include skin which tends to burn in the sun, red hair and freckles, the presence of large numbers of melanocytic naevi, and a family history of melanoma. It is likely that these risk factors would also apply to users of sunbeds and therefore sunbed usage should be denied to individuals with these phenotypes. The association between a history of sunburn and melanoma risk is especially strong for sunburn in youth. Use should also be discouraged by young people below the age of majority, since data associating sunbed use with melanoma risk are strongest for individuals under the age of 35 years. The known lead time needs to be taken into account in setting age limits, with a precautionary margin.

CHAPTER 5

SUNBED USE BY CHILDREN AND YOUNG PEOPLE

Reports are accumulating on the use of sunbeds by children and young people in both Europe and the USA.

Reported sunbed use is rare in the first decade but rises rapidly in the second decade.

Girls are more frequent users than boys.

Childhood sunbed use is more common in relatively deprived areas.

Childhood sunbed use is more common in households where adults also use sunbeds.

5.1 It is well established for solar UV radiation exposure that excessive exposure in the first two decades of life increases the risk of melanoma developing later in life. It is therefore possible that exposure to UV radiation emissions from sunbeds in childhood and adolescence could be even more damaging to the skin in the long term, than use after the age of 20 years. Moreover there are significant concerns about the ability to accumulate very large doses over time (see paragraph 4.20) and use of sunbeds earlier in life may result in greater lifetime accumulation.

Studies from the UK

5.2 There is longstanding advice that young people should not use sunbeds, from professionals and organisations within the health community (Cancer Research UK, 2004; Diffey et al, 1990; HSE, 1998, 2009) and the responsible sector of the sunbed industry (The Sunbed Association, 2008). There remains, however, abundant evidence that this advice is ignored by many children and young people, especially girls. To this effect, part of the 2008 SunSmart campaign was directed at raising awareness at the dangers of using sunbeds, especially by the under 35s (Cancer Research UK, 2008d).

5.3 The first UK-wide survey of sunbed users carried out in the mid-1980s found that 19% of users of commercial sunbed premises were young people aged under 20 years, with a small number (0.6%) aged 15 years or less (Diffey, 1986).

5.4 A survey of 1,405 primary school children in Scotland (Hamlet and Kennedy, 2004) found that that almost 7% of children aged 8–11 years have used a sunbed in the previous five or six months and 1.3% may be using one as regularly as every fortnight.

5.5 A questionnaire survey to assess the awareness of tanning guidelines, the use of sunbeds and the attitude towards tanning in 499 adolescents aged between 14 and 16 years was carried out in two schools in a mixed urban part of Merseyside (Mackay et al, 2007). The investigators found that sunbeds had been used by 43% of respondents; girls had used them much more than boys,

with use increasing by age for both sexes. Overall, 65% reported that they were aware of guidelines and about one-half thought that guidelines advising people how often to use a sunbed were a good thing. One in ten of those using a sunbed also said they had experienced problems with their eyes or skin. Most users thought using sunbeds made them look healthy, made them more attractive and confident, that they created a base tan before a holiday, and that they were a good treatment for acne.

5.6 A recent study from the West Midlands on 872 responses from 900 children aged 11–16 years reported that 49 (6%) had used sunbeds and that 70% of these were female. Of the users, 30% had freckles, and 35% had had at least one sunburn (Suchak et al, 2008). Of the devices used, 45% were coin-operated, and 92 % of the child users also had an older family member using sunbeds, emphasising the need for family education.

Studies from other countries

5.7 A review of indoor tanning by adolescents (Lazovich and Forster, 2005) found that prevalence is consistently higher among girls than boys and increases with age in both Europe and the USA. What actually constitutes prevalence in this context has been defined variously as any use, use in the past 6 or 12 months, or frequent use in the past 12 months and this varying definition explains why, in their review of 12 studies, Lazovich and Forster (2005) found that prevalence use by young female adolescents varied from 14% to 75%, with a mean value weighted by sample size of 43%. A comparative weighted mean prevalence for young males was 18%.

5.8 According to the most recent studies, based on nationally representative samples from Europe (Boldeman et al, 2001) and the USA (Demko et al, 2003), 30% of Swedish and 24% of American adolescents aged 13–19 years, reported any use of indoor tanning; frequent use of indoor tanning (ten or more times) was reported by 7.5% and 11.7% of adolescents in Sweden and the USA, respectively. Geller et al (2002) studied 10,079 males and females aged between 12 and 18 years from 50 US states and found that overall 10% had used a sunbed in the previous year, with a significant difference in use between girls (14.4%) and boys (2.4%). Among the girls, sunbed use was much more common in those aged 15–18 years, at 24.6%, compared with those aged 12–14 years, at 4.7%. A further study by O'Riordan et al (2006) of 6,373 females aged 12–18 years in the USA reported that 14% had used a sunbed in the previous year, and that frequent use of sunbeds was associated with additional health risk behaviours including bulimia, smoking or use of recreational drugs. A third US study reported on 5,274 adolescent parent pairs drawn from the 100 largest cities in the USA (Hoerster et al, 2007). Eleven per cent of the adolescents had used a sunbed in the previous year, and significant determinants of sunbed use were use of sunbeds by parents, and parental enthusiasm for tanned skin.

Psychological factors

5.9 The observation that the prevalence of sunbed use by girls is much greater than that by boys is evident from many of the above studies. Quite what makes young women such ardent users of sunbeds was examined in a psychological study which concluded that a tanned skin from sunbed use in this cohort, despite being aware of the hazards, helps them to achieve their ideal of beauty (Fiala et al, 1997).

5.10 That children and adolescents should not use sunbeds has received wide media coverage – see, for example, the BBC News website (*BBC News,* 2008). As one example, following a report that young people under 18 years should be banned from using sunbeds, the BBC TV youth programme *Newsround* featured an item on this topic on 20 January 2004 and followed this

up with an online question: 'Do you agree that kids should be banned from using them?' (*BBC Newsround,* 2004). Whilst many respondents did agree that youngsters should not use sunbeds, there were an appreciable number who disagreed, as illustrated by the following quote from a 13 year old girl: 'No way – I use one and I won't live without it. I know the risks but I still choose to go on one. As long as the kids know the risks I think it's ok'.

Prohibition

5.11 The 'Regulation of Provision of Sunbeds' forms Part 8 of Public Health etc (Scotland) Act 2008, which successfully completed its final Stage 3 consideration on 12 June 2008 and received Royal Assent on 16 July 2008 (Scottish Parliament, 2008). The implementation plans have yet to be finalised, but the provisions of the Act are expected to come into effect in the autumn of 2009. Part 8 of the Act includes provisions for operators of sunbed premises that will directly affect children and young people:

(i) Prohibition on allowing use of sunbeds by persons under 18.

(ii) Prohibition on sale or hire of sunbeds to persons under 18.

5.12 The prohibition on the use of sunbeds by the under 18s is under consideration by the Department of Health and Children in the Republic of Ireland, and is also being called for by the British Association of Dermatologists (personal communication, 2009).

CHAPTER 6

SUNBED USE IN THE UK

The number of commercial sunbed outlets is increasing. Of particular concern is the growth of unstaffed outlets, unsupervised sunbeds, non-traditional outlets and coin-operated sunbeds.

The UV irradiance of commercial sunbeds is increasing.

The distribution of sunbed outlets varies geographically, with particularly high concentrations in some local authorities in the north of England.

The concentration is higher in deprived urban areas, even after taking into account their more densely concentrated populations. This finding is consistent across all four countries in the UK.

The lack of registration of commercial outlets hampers the monitoring of trends in their numbers, type, power and distribution. This means that the risk they are potentially posing to the population cannot be monitored.

Population risks need to be quantified

6.1 The incidence of all skin cancers is rising. There is clear evidence that a major risk factor is UV radiation exposure. While traditionally this UV radiation exposure has taken place out of doors, over the past decades increased numbers of commercial outlets have installed sunbeds (Oliver et al, 2007) and an increasing proportion of the population, especially young people, are using them (Cancer Research UK, 2008e).

6.2 Around a quarter of adults in the UK have used a sunbed, as have around 6% of young people aged 11–17 years. The proportion of young people who had used a sunbed was higher in 15–17 year olds and in 11–17 year olds resident in cities (11%). Eighteen per cent of young people who have not used a sunbed, would consider doing so in future (Cancer Research UK, 2008e). A Mintel report in 2007 on the ownership and use of beauty aids showed that 4% of a sample of people aged 16 years or over own a sunbed/solarium or sunlamp and 7% use one (Mintel, 2007).

6.3 From a public health perspective, the key concern is that artificial sources of UV radiation not only have the same acute and long-term effects on skin of structural damage and risk of skin cancer, but more importantly it is possible to easily expose the skin to levels of UV radiation equivalent to Mediterranean sun (Oliver et al, 2007), in intense doses and over very prolonged periods.

6.4 Not only is the possibility of acute burning eminently feasible with new powerful devices etc, but extreme levels of chronic exposure can occur that are much greater than the average UK population could achieve by holidaying abroad or living in the South West.

6.5 There is good evidence on the effect of chronic exposure of fair skin to tropical sun over a period of three to four years in World War II prisoners of

war in the Far East (Page et al, 2000). Regular sunbed use could mimic or surpass this.

6.6 Given this evidence of the very significant potential for harm, it is important from a public health perspective to be able to quantify the risk to the population. Ideally there would be a clear method of identifying how many sunbeds and lamps are in use, where these are located, in commercial outlets or in home use, and who is using them and for how long. From this, a more precise assessment of population risk based on epidemiological evidence could be made.

6.7 The number of commercial outlets is increasing (Oliver et al, 2007; Redcar & Cleveland Borough Council, 2002). Commercial sunbeds are increasingly being placed in unusual locations, eg video rental shops and small local supermarkets.

6.8 Of particular concern is the growth of unstaffed commercial outlets with coin-operated sunbeds that have been described as 'the high street equivalent of the launderette' (Scott, 2003). There is concern that these outlets are particularly popular in low-income areas (*Environmental Health Journal*, 2005).

6.9 At present, only some incomplete fragments of this population risk assessment jigsaw can be put together and, given the clear risks, this argues strongly for registration so that use can be quantified, trends monitored and regulations implemented.

What is the current status of commercial sunbed availability in the UK?

6.10 Sunbeds are available to the public through commercial outlets and, more unusually, vertical sunbeds (which can fit into small spaces) have been located in nail bars and even cheque exchanges.

6.11 Sunbeds in commercial outlets may be supervised, or unstaffed (coin-operated). If well run some advice is being given to clients; however, in unstaffed commercial outlets no face to face advice is given and there are no controls on use, providing unlimited access for clients.

6.12 As discussed in Chapter 7, in the UK very few local authorities have licensing in place. Where licensing does occur, local authorities can create registers of the numbers and locations of commercial outlets in their area (CIEH, 2005; Oliver et al, 2007). However, even in these areas there is no legal requirement to keep records of clients, doses, skin type, etc.

6.13 In the absence of a national registration scheme no comprehensive information is routinely collected. The total number of commercial outlets providing sunbed usage throughout the UK is not known.

6.14 The Sunbed Association (TSA), representing operators, manufacturers and hirers, which has an operating code based on many of the Health and Safety Executive recommendations, estimates there are around 8,000 tanning facilities nationally, only a fifth of whom are members of TSA (K Banks, Chief Executive, The Sunbed Association, personal communication, 2006). However, TSA had 1,171 UK registered members in 2007.

6.15 A study conducted by the South West Public Health Observatory sought to identify outlets providing sunbed facilities by a desk-top UK-focused search utilising internet directories. This identified a total of 5,350 sunbed outlets across England, Scotland, Wales and Northern Ireland (SWPHO, 2009), and corresponds to approximately two-thirds of the 8,000 sunbed outlets estimated by TSA to be operating across the UK. Of the 5,350 sunbed outlets

located, 4,492 were in England, 171 were in Northern Ireland, 484 were in Scotland and 203 were in Wales.

6.16 A comparison between 1,149 TSA members for whom postcode data were available and the outlets identified using the internet directory search revealed matches for only 496 of the 5,350 (9%). Membership varied by country. The lowest percentage membership was found in Northern Ireland (4%) followed by Scotland (7%) and England (9%). Wales had the highest percentage membership with 17% of outlets registered with TSA. Conversely, from the membership list for TSA, 496/1,149 outlets (43%) were identified by the internet search strategy.

6.17 A register compiled by Bradford Local Authority identified 94 outlets, compared with 45 identified by the SWPHO internet search. Of these 45, 24 were not on the Local Authority's list – therefore, the two studies combined found 118 outlets. Thus, the internet search study identified 38% of the total number of outlets identified by both studies combined (SWPHO, 2009).

6.18 It is not possible to derive a definitive estimate of the degree of sunbed outlet coverage across the whole UK from these matching studies but on the basis of the above discussion it would appear that the likely approximate completeness of ascertainment by the internet search is at best around 50%.

6.19 The current study primarily identified commercial sunbed outlets through internet searches, and so was likely to capture a higher proportion of relatively new, multi-unit, efficiently operated establishments. It is less likely to identify older, more marginal establishments, often providing sunbed facilities as an additional feature to their main business activities (eg hairdressers). These businesses are more likely to be operating in economically marginal areas, with relatively high levels of local area deprivation. Thus, it might be anticipated that had a greater degree of coverage been obtained by employing additional survey methods, the proportion of outlets found in more deprived areas may have been even higher.

6.20 Finally, the unit of analysis was based upon commercial sunbed outlets, and did not take into account variation in numbers of tanning units within outlets. Again, it is more likely that the survey search methods would have identified a larger proportion of large, multi-unit outlets.

6.21 Using local authority registration data or a database, such as that constructed from internet directories by the South West Public Health Observatory (SWPHO, 2009), it is possible to study where these outlets are distributed by area characteristics, eg urban or rural, concentration of population, affluence or deprivation of area. This can be used to surmise the likely client group using the outlets. However, as no records are kept it is not possible to give precise information on gender, age group or skin type or most importantly on acute and cumulative doses. In contrast, in some European countries, such as France and Sweden, commercial outlets need to be registered and/or hold a licence and there is a similar situation in Victoria in Australia. These comprehensive data could be used for monitoring trends and looking at distributions.

6.22 The South West Public Health Observatory undertook an analysis of the distribution of the commercial sunbed outlets by geography, deprivation and high risk group defined as an estimated number of white persons aged 15 to 34 years in local areas. Sunbed outlet data were mapped for each local authority area to illustrate variations across the UK. The concentration of commercial sunbed outlets varies significantly by geography, see Figure 6.1.

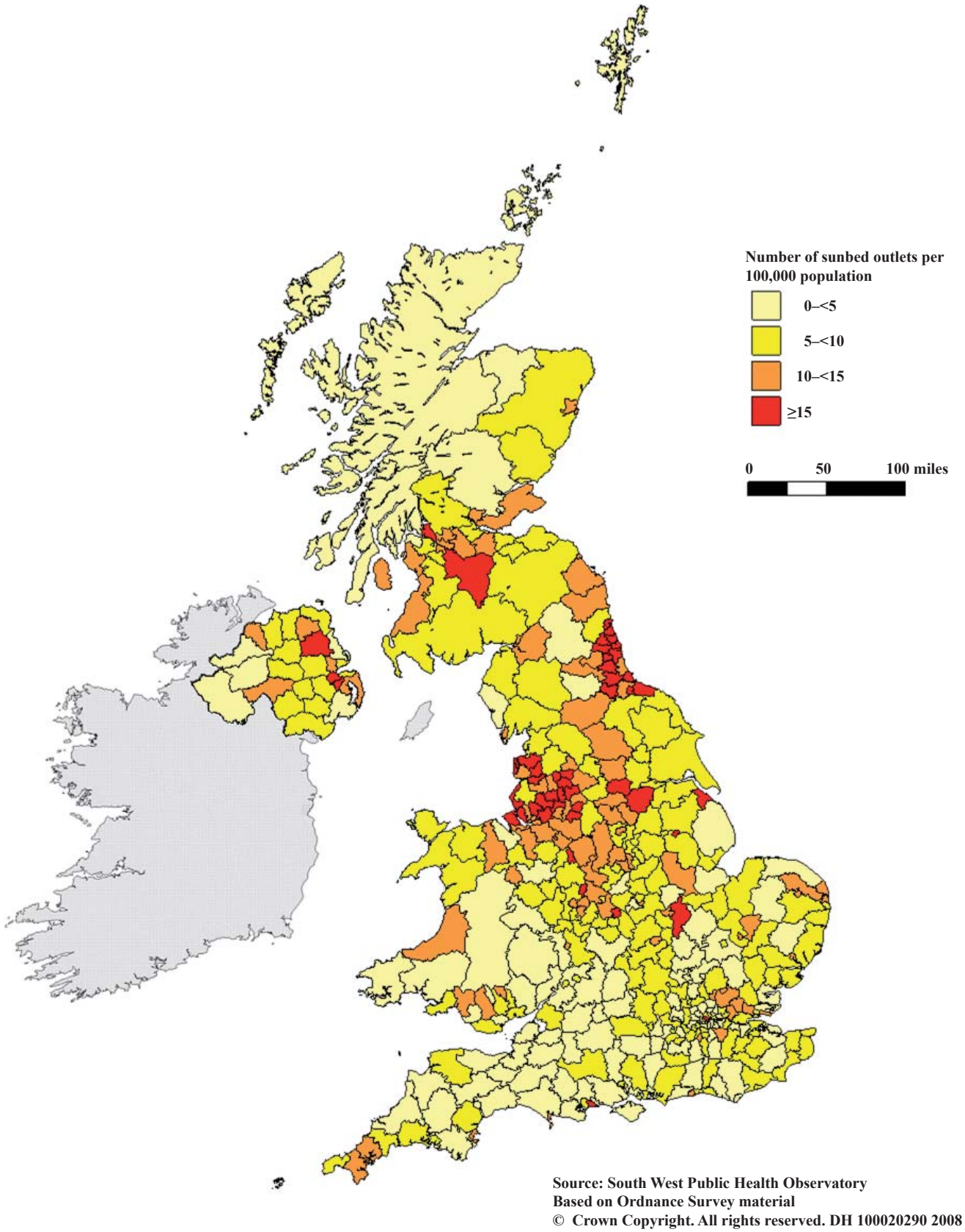

Figure 6.1: Sunbed outlets per 100,000 total population – UK local authority areas, 2006

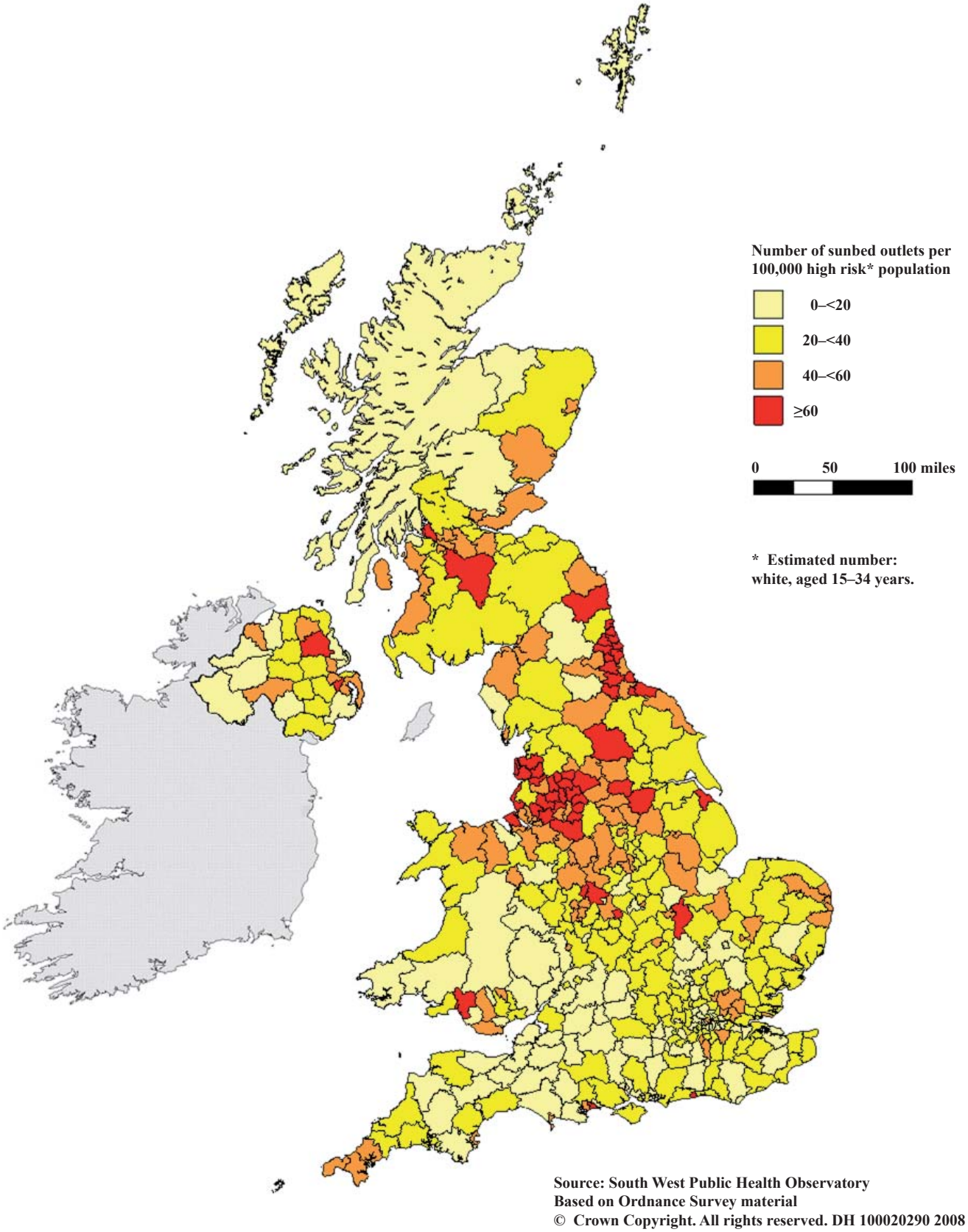

Figure 6.2: Sunbed outlets per 100,000 high risk population – UK local authority areas, 2006

6.23 Concentrations of local authorities with high sunbed outlet rates per 100,000 population are found in the urban areas of North West and North East England. Rates in southern England were relatively low except in Cornwall.

6.24 The City of London Local Authority is an exception. It had by far the highest outlet rate per 100,000 total (the outlet rate for the City of London was 156 per 100,000 total population compared with an average of 8.9 for England, and was almost six times the rate of the next highest local authority). The City of London Local Authority has a very small resident population (under 8,000 in 2005), but is the place of work for approximately 340,000 people.

6.25 Rates in Scotland were highest in West Dunbartonshire and South Lanarkshire Local Authorities, and lowest in the North and West regions. No overall pattern was found in Wales or in Northern Ireland.

6.26 The distribution in the variations of density of sunbed outlets across the UK, when analysed in relation to the high risk group, generally resembled that observed for the total population, although levels in individual local authorities differed, see Figure 6.2.

6.27 In each country, sunbed outlets are predominantly located in urban areas, with relatively few outlets sited in rural locations. Commercial sunbed outlets are most commonly found in 'secondary retail areas', where the combination of rental affordability and potential customer access is most advantageous for this type of enterprise.

6.28 Within each country a strong trend can be observed between the number of sunbed outlets and level of area deprivation, with an approximate doubling of number of sunbed outlets in the most deprived quintiles compared with the most affluent ones.

6.29 The following observations are consistent across all countries within the UK.

(i) Urban areas have much higher total outlet rates than rural areas.

(ii) Total outlet rates generally increase with growing levels of area deprivation.

(iii) The largest increase in total outlet rates occurs between deprivation quintile 3 (average) and deprivation quintile 4 (second most deprived).

6.30 Country-specific observations are as follows.

England

(i) Strong, consistent trends of increasing outlet rates with increasing levels of area deprivation for both urban and town and fringe areas.

(ii) No discernable trend for rural areas.

Wales

(i) Generally increasing outlet rates with increasing deprivation for urban areas, the highest outlet rate was observed in the second most deprived quintile (DQ4).

(ii) No discernable trend for town and fringe areas.

(iii) Increasing rates with increasing levels of affluence for rural areas, though the total number of outlets for this area type was small (n = 12).

Scotland

(i) Generally increasing outlet rates with increasing deprivation for urban areas, the highest outlet rate was observed in the second most deprived quintile (DQ4).

(ii) Increasing rates with increasing levels of affluence for rural areas, though the total number of outlets for this area type was small (n = 15).

Northern Ireland

(i) Generally increasing outlet rates with increasing deprivation for urban areas, the highest outlet rate was observed in the second most deprived quintile (DQ4).

(ii) Generally increasing rates with increasing levels of affluence for rural areas; however, the most deprived quintile (DQ5) has the second highest rate. The total number of outlets for this area type was again small (n = 22).

6.31 Although the sunbed outlet rates per 100,000 high risk population (white people aged 15 to 34 years) are approximately four times those for the total population, the relative distributions of rates by area type and deprivation quintile for each country are almost identical to those observed for the whole population.

6.32 The most consistent gradient was observed in England (which had the largest number of sunbed outlets), with steadily increasing rates of sunbed outlets with increasing national quintile of deprivation.

6.33 In Wales, Scotland and Northern Ireland, the highest outlet rates were observed within the second most deprived quintile (after adjusting for urban/rural mix). In each country, however, the two most deprived quintiles had higher rates than the two most affluent quintiles.

Implications for public health policies

6.34 There have been calls for the mandatory licensing of sunbed outlets (Mackintosh, 2006). This would enable the collection of more complete and accurate data on which to base future research in this area. It is crucial that, if introduced nationally, sunbed registers are properly maintained to ensure completeness, accuracy, reliability and timeliness.

6.35 Public health policies need to be based on robust empirical evidence. This study suggests that the rate of sunbed locations per 100,000 population varies by area deprivation in the UK, with those living in poor areas more likely to encounter sunbed outlets in their locality. However, the risk from sunbed use is not simply determined by age, ethnicity and proximity to sunbed outlets. Further research is needed to accurately determine the 'at-risk population'. Additionally, there needs to be a more robust data source on the location of UK sunbed outlets. The findings merit further investigation because they suggest a possible source of health inequalities that could be addressed by effective public health policy.

6.36 The contribution of increased access to sunbeds in more deprived areas to the changing socioeconomic profile of female melanoma cases, in particular, needs to be investigated.

6.37 Similarly, the relationship between the rapid increase in incidence of melanoma in young people in the North West and East of England and the high density of commercial sunbed outlets and high teenage use rates should be researched.

6.38 Very few of the sunbed outlets identified were registered members of The Sunbed Association, which operates a voluntary code of practice. Membership levels were especially poor in Northern Ireland. Uptake of voluntary self-regulation schemes has been low in other countries (Böttger, 2007) and compliance with voluntary codes of practice has been shown to be poor (Dobbinson et al, 2006; *Which?,* 2008).

6.39 The British Association of Dermatologists firmly believes that a voluntary code of practice is largely ineffective (British Association of Dermatologists, personal communication, 2009) and therefore urges that the formal regulation is introduced, including:

(i) A ban on sunbed use for the under 18s.

(ii) A ban on coin-operated, unstaffed sunbeds. Currently, at unstaffed facilities anyone, including children, may use the tanning devices. There is no limit imposed on the dose per session or the number of sessions allowed.

(iii) A requirement on operators to provide information to clients on the health risks of sunbed use, to allow people to make a more informed decision. Many salons do not provide adequate information on the health risks, but instead advertise somewhat spurious health 'benefits'.

(iv) The removal of sunbeds from all local authority health facilities such as gyms and sports centres, as providing sunbeds at such venues sends conflicting messages and can lead to the perception that tanning facilities are 'healthy'.

(v) Inspection of premises operating sunbeds commercially, and the power of those inspecting to enforce regulations through fines/licence revocation.

CHAPTER 7

APPROACHES TO THE CONTROL OF SUNBEDS IN THE UK AND OTHER COUNTRIES AND ASSOCIATED LEVELS OF BUSINESS COMPLIANCE

Specific legislation on sunbed use exists in Belgium, Finland, France, Norway, Portugal, Spain, Sweden, USA, Australia and New Zealand.

Legislation was passed in Scotland in 2008; however, the UK does not have national legislation.

Control can be through legislation, voluntary code, licensing, registration or guidance.

Poor compliance is found against a variety of control measures where strict legislative controls do not exist.

Approaches to control

7.1 The mechanisms employed to control the use of sunbeds vary from country to country. For some, the risks posed by such devices, when used for cosmetic purposes, have justified strict regulatory control, whilst others have relied upon voluntary codes of practice in attempts to achieve specific standards.

7.2 The UK does not have specific national legislation aimed at controlling the cosmetic use of sunbeds. It does, however, have certain more general legislative provisions that may be applied to the use of sunbeds.

7.3 The Health and Safety at Work etc Act 1974 and the Management of Health and Safety at Work Regulations 1999 are generic health and safety legislation that require businesses and individuals to:

(i) Assess the health and safety risks created by their work activities, including the risks to employees and members of the public.

(ii) Take measures to control those risks as far as is reasonably practicable.

7.4 To provide more specific requirements, the Health and Safety Executive (HSE) issued (voluntary) guidance in the 1990s (HSE, 1998). Since the guidance was published there has been considerable technological change in the sunbed industry as well as expansion in the use of tanning devices. In 2008 the HSE conducted a consultation on a draft revision of the guidance. Subsequently, updated guidance was published in May 2009 (HSE, 2009).

7.5 A number of local authorities have 'adopted' specific legislation that permits the introduction of licensing regimes for certain cosmetic treatments that can include the use of sunbeds. The legislation that allows the licensing of premises using sunbeds is restricted in its geographical application. The London Local Authorities Act 1991 allows the licensing of premises offering artificial UV radiation tanning (London Local Authorities, 1991) (see Appendix C). Similar Acts have allowed licensing of such premises in

Nottinghamshire (Nottinghamshire County Council, 1985), Birmingham (Birmingham City Council, 1990), Dorset (Dorset for You, 2008) and in Essex (Essex County Council, 1987). Liverpool City Council is currently looking at introducing a voluntary code raising the restriction on sunbed use to the under 18s (Liverpool City Council, personal communication, 2008).

7.6 The Sunbed Association (TSA) has voluntary membership but currently only represents approximately 20% of the operator market. All members commit to complying with TSA code of practice, which advises that children under 16 years, people with unsuitable skin types, people with excessive moles or freckles, and people with a history of skin cancer should not use sunbeds. The code requires that all sunbeds must be used under supervision of appropriately trained staff and protective goggles must be provided and worn. TSA provide training courses and the programme includes UV radiation, sunbed lamps and their service life, sunbeds – features, maintenance and cleaning, the skin and how it tans, sunbed sessions and skin types, health and safety guidelines, and the provision of information for customers. Members must demonstrate compliance with the code of practice during inspections of their premises (The Sunbed Association, 2008).

7.7 In other areas, controls are reliant upon the generic health and safety legislation and the voluntary guidance. However, in light of the growing incidence of skin cancer and concerns about the contribution of sunbeds to this health burden, Mr Kenneth Mackintosh (MSP) launched a consultation on the regulation of sunbed parlours in Scotland in 2006 (Mackintosh, 2006). The proposals were subsequently integrated into the Public Health etc (Scotland) Act 2008 for Scotland (Scottish Parliament, 2008), which is expected to be implemented in the autumn of 2009. This Act regulates the provision of sunbeds through:

(i) Prohibition on allowing use of sunbeds by persons under 18.

(ii) Prohibition on the sale or hire of sunbeds to persons under 18.

(iii) Prohibition on allowing unsupervised use of sunbeds.

(iv) A duty on operators to provide information to sunbed users.

(v) A duty on operators to display an information notice.

The Scottish Ministers may prescribe the information which operators have to provide to users, as well as the form and manner in which it is to be provided. It is hoped that this information will clearly state the necessity for the use of protective eyewear.

7.8 The Department of Health and Children in the Republic of Ireland launched a public consultation in 2008 on proposed legislation to regulate sunbeds. This included proposals to prohibit the use of sunbeds by the under 18s, controls on the sale and/or rental of sunbeds, inspections to ensure compliance, registration with the competent authority, and exemptions for medical use. The responses to the consultation are being considered in how the proposals will be taken forward (Department of Health and Children, 2008).

7.9 In addition to controls directed at the use of sunbeds, there are also other generic standards that seek to ensure harmonisation across the European Union for electrical equipment designed for use within certain voltage limits (50 and 1,000 V AC and 75 and 1,500 V DC). The current European Directive on Low Voltage (European Commission, 2006) sets health and safety standards for construction, installation, maintenance and use that would apply to artificial

tanning devices and replaced the original directive from 1973 (European Commission, 1973).

7.10 In light of the safeguard procedures in the original low voltage directive, the Spanish Authorities notified the European Commission that the European Standard EN 60335-2-27: 1997 (European Standards, 1997) did not entirely cover the health and safety aspects which needed to be considered during the design phase of the electrical appliance, ie it did not provide limit values on the maximum effective irradiance of UV radiation for the types of tanning devices covered by the scope of the standard.

7.11 As a consequence, the European Commission's Scientific Committee on Consumer Products (SCCP) was asked to provide an opinion on the 'biological effects of ultraviolet radiation relevant to health with particular reference to sunbeds for cosmetic purposes' (SCCP, 2006)*, which would form a basis for any revisions to the European Standard. One specific recommendation arising from the opinion was that the maximum erythemally weighted irradiance should not exceed 11 standard erythemal doses per hour (or 0.3 W m^{-2}), the equivalent of 'tropical sun'.

7.12 The SCCP made a number of other recommendations in relation to restricting the usage of sunbeds – in particular that:

(i) People with known risk factors for skin cancer should be advised not to use UV tanning devices – this would include those with Fitzpatrick skin types I and II and the presence of freckles, atypical and/or multiple moles and a family history of melanoma.

(ii) UV tanning devices should not be used by the under 18s.

(iii) Eye protection should be worn if sunbeds are used.

7.13 In July 2006 the European Commission called upon member states and the sunbed industry to ensure that appropriate warnings and instructions were provided to prevent misuse of sunbeds. The Commission also requested the introduction of UV irradiance limits within product standards and associated provision of guidance for both industry and consumers. Alongside this the Commission asked all member states to ensure that solaria applied any such guidance. Member state consensus supported the adoption of the SCCP recommendations (and in particular the irradiance limits) to be applied from 22nd July 2007. Subsequently the Commission has requested that EN 60335-2-27: 1997 (European Standards, 1997) be amended accordingly (Straszburger, 2007).

7.14 Various pieces of work have been carried out to establish the position in the member states regarding controls applied to sunbeds. From a Commission questionnaire (Straszburger, 2007) it has been established that 15 member states have some measures in place (legislation or guidance) to control the use of UV irradiation for cosmetic purposes. Nine countries (Austria, Belgium, Cyprus, Finland, France, Germany, Spain, Sweden and the UK) have applied the SCCP recommendation to limit the maximum irradiance to 0.3 W m^{-2}.

7.15 Specific legislation controlling the use of sunbeds exists in the following European countries: Belgium (Service Public Federal Economie,

* In the SCCP opinion the term 'sunbed' refers to all types of UV tanning devices for cosmetic purposes.

2007), Finland (Ministry of Health and Social Affairs, 2002), France (Légifrance, 1997), Norway (NRPA, 2003), Portugal (Piazena, 2007), Spain (Ministry of the Presidency – Spain, 2002) and Sweden (Swedish Radiation Protection Institute, 1998). Outside Europe, legislative controls exist in a number of US states (National Tanning Training Institute, 2008) and in Australia (Government of South Australia, 2008a,b; State Government of Victoria, 2007).

7.16 The content of legislation in Europe varies from country to country (Piazena, 2007) but the core elements addressed include:

(i) Technical requirements for appliances.

(ii) Limits on spectral distribution and irradiance.

(iii) Limits on dose and frequency of exposure.

(iv) Operational requirements.

(v) Information and advice for consumers.

(vi) Staff training.

(vii) Equipment maintenance.

(viii) Supervision, inspections and sanctions.

Some of these elements are covered by the HSE guidance on the use of UV tanning equipment (HSE, 2009).

7.17 Amongst the European states, France has a particularly robust system of legislative controls that were introduced in 1998. Equipment requirements are based around European Standard EN 60335-2-27: 1997 but differ primarily in limiting the usage of appliance by UV radiation type, eg UVB should be less than 1.5% of total UV radiation. In addition, type 1 appliances should only be used by professionals, type 3 alone should be available for home use, and types 2 and 4 should only be used by medical practitioners (see Table 4.1 for UV appliance types).

7.18 There is a mandatory requirement for operators to declare appliances to the regional health authorities, which triggers an initial inspection. Initial inspections and follow ups every second year are carried out by certified organisations, with annual reports submitted to the health authorities. There are specific requirements for approved training of operators, including refresher courses every five years. There are requirements for the display of consumer information. Automated equipment is prohibited, as is use of sunbeds by the under 18s.

7.19 France intends to update and review its legislation in 2008, in part as a result of the European Commission mandate. Particular consideration is likely to be given to restricting the use of UV radiation type 3 equipment, increasing operator training courses from 8 to 20 hours' duration, and the introduction of 'informed consent' forms for customers (Césarini, 2007). In the current legislation, minors under the age of 18 years are banned from using UV radiation emitting appliances, all appliances must be declared to the health authority, and any claim of health benefit is prohibited (Légifrance, 1997).

7.20 Germany, like the majority of the UK, relies upon 'voluntary' controls to maintain consumer protection in the use of sunbeds. The requirements cover the core elements included in the legislation in other member states but these are not mandatory. The requirements were initially identified in 2003 and

tanning outlet operators were invited to sign up to the standards. Once such a commitment had been made the business would be supplied with a certificate that they could then display (which might be used for marketing advantage). Take up was extremely poor (less than 100 of some 6,000 known premises) and this together with the European Commission mandate led to the revamping of the scheme (Böttger, 2007).

7.21 Regulations on the UV radiation emissions of sunbeds were introduced in Sweden in 1991 (and updated in 1999) requiring licensing of certain sunbed appliances and registration of premises with the local environmental and health authorities. The UVB component of the output is also limited to 1.5% of total UV radiation, eye protectors must be provided, and advice and information available.

7.22 In the USA, the Food and Drug Administration (FDA) provides extensive regulation of the indoor tanning industry via various aspects of sunlamp products, which is aimed mainly at equipment manufacturers. The US FDA regulations require each sunbed to carry a label detailing consumer information about avoiding overexposure and warning that certain medications or cosmetics may increase sensitivity to UV radiation. A condition is also made for the provision of suitable eye protection. There should also be recommended exposure schedules for skin types II–V attached to each device (US FDA, 2007b).

7.23 By April 2008, 28 US states regulated tanning facilities for minors through state laws and a further five states were introducing legislation (National Conference of State Legislatures, 2008). Age restrictions varied between states from under the age of 13 years to under the age of 18 years. A preliminary study of tanning operators in Kansas, which does not impose any age restrictions, found that the majority of operators believed that age restrictions should be required (Apollo and Muma, 2007).

7.24 Australia and New Zealand have a voluntary code of practice in the joint standard, AS/NZS 2635: 2002, approved on behalf of the Council of Standards Australia on 1st March 2002 and on behalf of the Council of Standards New Zealand on 21st March 2002 (Australia/New Zealand Standard, 2002). This sets out requirements for installing and maintaining and operating sunbeds and for the content of warning notices and client consent forms. Although the Standard is primarily directed towards commercial establishments, some operational requirements are equally applicable as a guide for domestic use.

7.25 In 2001, the Australian Competition and Consumer Commission (ACCC) ruled that under the Trade Practices Act 1974 solaria operators could not advertise their services as being safe or healthy and that they had a duty of responsibility to ensure that consumers were adequately informed of the risk from using solaria (ACCC, 2001).

7.26 The State Government of Victoria in Australia has introduced regulations on the operation of solaria. The Radiation (Tanning Units Amendment) Interim Regulations 2007 (State Government of Victoria, 2007) came into effect in February 2008 via the Radiation Act 2005 and require any person possessing, selling or maintaining sunbeds to hold a management licence. Conditions included in the licence cover displays of health warnings, mandatory skin type assessments and an age limit of 16 years, with 16–17 year olds requiring parental consent. Similarly, South Australia has issued two regulations under the Radiation Protection and Control Act 1982. The

Cosmetic Tanning Units Regulations 2008 (Government of South Australia, 2008a) came into operation in March 2008 and require sunbed owners and operators to abide by the Australian Standard AS/NZS 2635: 2002. The Non-ionising Radiation Regulations 2008 (Government of South Australia, 2008b) came into effect in March 2009 and require anyone operating a sunbed to be licensed.

Business compliance

7.27 Evidence of levels of business compliance is somewhat patchy. The exception is in France, which through its mandatory requirement for registration and regular inspection can provide a clear indication of business compliance. Since the implementation of the legislation in 1998, the compliance level at initial inspection has risen from around 50% to 70%. Subsequent controls show compliance levels running at around 80%. A compliance survey carried out in 2004 showed that the failures were mainly due to absence of registration and use of uncertified operators (Césarini, 2007).

7.28 By contrast, the initial German voluntary approach (2003) has proven less successful. Some 30 months after the introduction of voluntary certification less than 100 of more than 6,000 commercial tanning outlets had signed up to the scheme. Investigation into the lack of take up revealed a number of problems:

(i) Operators did not understand the need for certification.

(ii) Operators did not believe that increased risk of cancer was an issue that would concern customers.

(iii) The costs of certification were seen to be too high.

(iv) The public was not aware of the scheme, thus membership would give no particular business advantage.

A revamped scheme, again voluntary, was launched in September 2007 and will complement moves to legislate on sunbed irradiance and prohibition of use by the under 18s (Böttger, 2007; Levine et al, 2005).

7.29 A number of studies carried out in the USA, Canada and Poland have found poor levels of compliance with control requirements, including the use of sunbeds by the under 18s (Lazovich and Forster, 2005). A study in Australia showed poor compliance with specific aspects of the voluntary code introduced in 2002 (Dobbinson et al, 2006).

7.30 In the UK surveys have been carried out to investigate levels of compliance. Some have focused specifically on usage of sunbeds by children (*BBC News,* 2005; Hamlet and Kennedy, 2004; *The Guardian,* 2005; *Which?,* 2008), while others have been more generalised. A general survey was carried out by the Royal Environmental Health Institute of Scotland (REHIS) in 2006 (REHIS, 2006). This found that not only were there limited controls on the age of users but there were also problems in the provision of advice about skin type and suitability for tanning as well as failures to offer or ensure use of eye protection.

7.31 The Chartered Institute of Environmental Health (CIEH) recorded the findings of a number of English surveys in its 'Saving our skins toolkit' (CIEH, 2005) which was aimed at supporting local authorities to take action to reduce the incidence of skin cancer. The surveys identified similar shortcomings to those found in the REHIS survey, with one local authority (Chichester District Council) finding that over 60% of the premises inspected failed to follow the 1998 HSE guidance on sunbeds.

7.32 More recently, a survey commissioned by the CIEH (Wales) involved visits to 69 different premises in South East Wales (CIEH (Wales), 2008). The research was carried out on the basis of 'mystery shopper' visits, with operators being asked a standardised range of questions. Five unstaffed studios were visited amongst the larger sample. Of those interviewed, 30% (21) were members of The Sunbed Association.

7.33 As part of the interview businesses were asked about age restrictions for use, frequency of use and time between sessions, use of eye protection, and advice given regarding illness and use of specific medication as well as skin type. An assessment of any warning information or notices was also carried out.

7.34 The survey found that the majority of those questioned would allow a person under 16 years of age to use a sunbed, although in some cases this was conditional upon the consent of an adult. In addition, in observational studies unstaffed commercial outlets appeared to be used by the under 16s.

7.35 There was considerable variation in views about appropriate intervals between sessions, with some operators allowing consecutive treatments with less than a 24-hour gap. In addition, a large proportion of premises used coin-operated equipment (apart from unstaffed facilities), potentially allowing disregard of any advice about session length. There was little investigation into the health of prospective customers and little information on medication that would preclude sunbed usage. Fewer than 25% of operators carried out a formal skin type assessment.

7.36 Recommendations arising from the survey were that:

(i) Operation of sun tanning equipment should be controlled by statute.

(ii) Commercial sunbed outlets should be licensed.

(iii) Unstaffed premises should be prohibited.

(iv) Eye protection should be compulsory.

(v) Use by the under 18s should be prohibited.

7.37 A similar exercise has also recently been carried out in Northern Ireland by the Sunbed Working Group of the Northern Ireland Melanoma Strategy Implementation Group. The survey involved inspector visits to all known premises (400) and examination of consumer safety and health and safety standards (Devereux et al, 2008). The results are currently being analysed but some of the key findings are that:

(i) Only 16% were members of The Sunbed Association.

(ii) Some 86% sold tanning accelerators.

(iii) Staff generally received training, although content and delivery was variable, eg 14% did not cover skin type assessment.

(iv) Where skin type assessment was carried out, 47% would not advise a skin type I customer to avoid using sunbeds.

(v) Some 10% did not provide any safety information for customers.

(vi) There were various mechanisms for controlling levels, duration and frequency of exposure; however, a small proportion of businesses did not set limits on or regulate exposure (2% and 5%, respectively).

7.38 A small survey undertaken by the All Party Parliamentary Group on Skin in 2008 found that commercial outlets are often ignoring the 1998 HSE

guidance (All Party Parliamentary Group on Skin, 2008). The group made a number of recommendations based on the findings, including bans on the use of sunbeds by the under 18s and on unstaffed, coin-operated sunbeds.

Enforcement

7.39 Surveys of officials charged with controlling the use of sunbeds (primarily environmental health practitioners) have revealed the difficulties created by the lack of specific legislation. Whilst the Health and Safety at Work etc Act 1974, the Management of Health and Safety at Work Regulations and equivalent legislation in the devolved administrations create general provisions, these are primarily focused on the health, safety and welfare of employees rather than customers who choose to use sunbeds. It has been argued that given the main focus of the generic health and safety legislation, there could be considerable difficulties in taking formal action where private individuals have voluntarily chosen treatments. To date, formal action does not appear to have been taken, despite considerable concerns and specific cases of harm being caused (primarily burns due to inappropriate exposure). It has been suggested that failure to observe voluntary standards for use of sunbeds is a matter of public health, rather than health and safety. If so, there would appear to be no obvious route for redress or prevention of harm. The exception to this is in areas where licensing exists, although action is limited by the variable licensing conditions.

Use of licensing conditions as a control mechanism

7.40 The London Local Authorities Act allows UV tanning facilities to be licensed as 'special treatments'. There are general provisions for a range of 'special treatments' that cover matters such as cleanliness, facilities, operator personal hygiene and record keeping, as well as specific requirements for the different types of treatment. An example of the specific conditions for sunbeds that are currently applied by Islington Council is shown in Appendix C.

7.41 Through the use of such licensing conditions consistent standards can be established, monitored and enforced. Annual inspections ensure that compliance is monitored and where non-compliances are identified proportionate sanctioning may be applied. Consistent failures may result in suspension or withdrawal of the licence.

7.42 The licensing regime would normally involve a fee from the operator wishing to offer a licensable activity. This would generally be structured to cover the administration and enforcement costs accrued in ensuring standards are maintained to protect public health.

7.43 The 'special treatments' regime applied in London also requires the registration of operators which allows standards to be set for training and competence, areas identified as often deficient in surveys and investigations.

7.44 The approach applied in Scotland, through the amendment of the Public Health etc (Scotland) Act 2008, imposes specific prohibitions and introduces a limited number of requirements. The use of a licensing regime would appear to provide a wider range of controls, although a cost–benefit analysis of the two approaches does not appear to exist.

7.45 The opportunities gained through use of a 'special treatments' approach, which includes existing and emerging cosmetic treatments where evidence exists of harm occurring if inadequately controlled, could be considered in a cost–benefit analysis of a licensing approach. This might be a mechanism to address the need for controls on the cosmetic use of lasers if proposals for de-regulation are taken forward. It could also include other emerging issues such as thread vein treatments and semi-permanent make up application, amongst others.

CHAPTER 8

CONCLUSIONS

8.1 In this, our Thirteenth Report, we have considered and reviewed evidence from a wide range of sources to provide advice on the health effects and the risks associated with exposure to UV radiation from artificial tanning devices, such as sunbeds, whether in commercial outlets or for home use.

8.2 Exposure to UV radiation is capable of inducing DNA damage; accelerated ageing (photoageing); all types of skin cancer including melanomas; other diseases such as cataracts, pterygia and cold sores; and immunosuppression.

8.3 The incidence of skin cancer is continuing to rise. Skin cancers are now the most common form of cancer in the UK, with 10,400 malignant melanoma cases and at least 81,500 non-melanoma skin cancers (NMSCs) recorded in 2006. The cost to the NHS is considerable with an estimated spend in 2002 of almost £58 million on diagnosis and treatment of NMSCs and £13 million for malignant melanoma.

8.4 Intermittent high dose rate UV radiation exposure is associated with increased risk of melanoma at all ages of life. Current sunbed technology allows exposure of skin to doses of UV radiation, equivalent to or exceeding the effective irradiance of midday Mediterranean sun, in intense doses and over prolonged periods of time.

8.5 High dose UV radiation exposure is not always associated with visible burns. Lack of burning with sunbeds should not be taken as evidence that the use of sunbeds for tanning is safer than exposure to sunlight.

8.6 Increased melanoma risk is also associated with skin phenotype (classified by the Fitzpatrick scale), melanocytic naevi (moles) and with family history of melanoma and other genetic factors.

8.7 According to the International Agency for Research on Cancer first use of sunbeds before the age of 35 years increases the risk of malignant melanoma by 75%.

8.8 A mathematical model estimates that sunbed use could account for approximately 370 new cases of melanoma and 100 deaths each year in the UK, resulting in a melanoma-related death rate for the population as a consequence of sunbed use consistent with that estimated for Australia.

8.9 Cumulative UV radiation exposure from sunbeds is associated with photoageing.

8.10 There is evidence of sunbed use by children and young people in the UK. Girls are more frequent users than boys. Childhood and adolescent usage has been recorded in both supervised and unstaffed (coin-operated) commercial outlets.

8.11 The irradiance from sunbeds can vary greatly. Most fall into type 2 or 3 based on the classification in the British and European Standard, BS EN 60335-2-27: 2003. Type 3 is the only class intended for unskilled use and therefore suitable for general use in commercial sunbed outlets. In recent years, sunbeds have been produced that are too powerful for the type 3 classification. A significant percentage of sunbeds exceed the recommended maximum erythemally weighted irradiance of 0.3 $W\,m^{-2}$ (equivalent to 11 standard erythemal doses, SED, per hour). COMARE cannot recommend any cosmetic use of sunbeds; however, the British Photodermatology Group in 1990 recommended a limit of 20 sessions per year for practical reasons.

8.12 The health risks associated with sunbed use outweigh the perceived benefits. The majority of perceived benefits from sunbed use are psychological and cosmetic. The use of sunbeds is not associated with added protection for sun exposure.

8.13 Artificial UV radiation sources are used medically to treat a variety of skin conditions, predominantly psoriasis; however, this should only be carried out in a clinical setting under medical supervision.

8.14 Vitamin D may be synthesised in the skin via exposure to UV radiation, although nutritional supplements are the preferred source for increasing vitamin D levels. The value of using sunbeds for vitamin D synthesis is dependent on the level of UVB emissions from the sunbed, which can be as low as 0.5%, and on other factors such as the age of the individual. The vitamin D levels achieved can also reach a plateau after a few sunbed sessions. The practice of using sunbeds to synthesise vitamin D is not recommended due to the potential carcinogenicity and the high frequency of acute side-effects (erythema and polymorphic light eruptions).

8.15 The number of commercial sunbed outlets in the UK is estimated at 8,000 by The Sunbed Association and this number is increasing. Only a fifth of the outlets are believed to be registered with TSA. The distribution of sunbed locations varies geographically, with particularly high concentrations in some local authorities in the north of England. There are concerns that commercial outlets are particularly concentrated in low income areas. Currently there is no national registration scheme for commercial outlets.

8.16 The level of control over the use of sunbeds varies between countries and can exist through legislation, voluntary code, licensing or guidance. In 2008, the Public Health etc (Scotland) Act 2008 was passed, which includes the prohibition of the use of sunbeds by the under 18s, the prohibition of the use of unsupervised sunbed parlours, and also requires operators to provide information to users. The UK, as a whole, does not have national legislation specifically aimed at regulating the cosmetic use of sunbeds. The Health and Safety Executive has revised its guidance on the use of UV tanning equipment, which covers sunbed operators and gives advice for clients. A number of local authorities have introduced licensing regimes for specific cosmetic treatments that can include sunbeds. Similar legislation measures to those passed in Scotland are currently being considered by the Department of Health and Children in the Republic of Ireland.

8.17 The British Association of Dermatologists is recommending a ban on the use of sunbeds by the under 18s and a ban on unstaffed, coin-operated sunbeds, and recommending that sunbeds should be removed from sites such as local authority sports centres. There should be a mandatory requirement for better information at the point of sale on the health risks associated with sunbed

use and sunbed providers should be prevented from undertaking any positive healthcare advertising. These recommendations are endorsed by the All Party Parliamentary Group on Skin and are in line with the recommendations from COMARE.

8.18 The lack of a national legislation scheme in the UK controlling the cosmetic use of sunbeds and the other points highlighted above are addressed in the recommendations of this report.

CHAPTER 9

RECOMMENDATIONS

In this report we have reviewed the literature regarding the health effects and risks arising from exposure to UV radiation from sunbeds* and we have also considered the controls in place in the UK at this time. We wish to make the following recommendations.

Recommendation 1

Regulation is required on the commercial use of sunbeds. Clinically prescribed use of sunbeds should be carried out only under medical supervision. Currently in the UK, legislation is only in place in Scotland. The recommendations presented here may exceed the requirements of this legislation and therefore should be considered by all UK health departments and government departments with an interest in this area. Legislation to regulate the use of sunbeds should focus on the following areas.

(i) We recommend that the commercial use of sunbeds by the under 18s is prohibited. This is in line with both the Public Health etc (Scotland) Act 2008 and the recommendations of the World Health Organization, and also the proposed legislation by the Department of Health and Children in the Republic of Ireland. Introducing an age restriction of 18 years brings the use of sunbeds in line with the sale of a number of other age-restricted goods, eg tobacco and alcohol. We recommend that the sale or hire of sunbeds to the under 18s should also be prohibited.

(ii) In order to support (i) above we recommend the prohibition of unsupervised use and/or self-determined operation of sunbeds in commercial outlets.

(iii) We recommend that all staffed commercial outlets should be licensed and registered, including registration of the types and power of machines on the premises. Licensing will allow control and checks of adherence to standards. Registration will permit monitoring of trends and distribution of commercial outlets and of machine types.

(iv) We recommend that legislation should include a requirement for commercial outlets to ensure that adequate protective eyewear is provided for users. The use of protective eyewear by clients should be compulsory.

(v) We recommend that detailed written information on the health risks associated with the use of sunbeds must be provided to users and should be clearly and easily visible on machines, both in commercial settings and for home use. Informed consent should be obtained from the clients prior to use. The use of sunbeds by persons in at-risk groups should be discouraged.

* In this report, the term ‘sunbed’ is used to represent all types of artificial UV tanning devices utilised for cosmetic purposes.

(vi) We recommend that commercial outlets and sunbed retailers should be prohibited from using information promoting unproven and/or net health benefits of sunbed use.

(vii) All sunbeds should adhere to both the British and European Standard (BS EN 60335-2-27: 2003) and the recommendations from the Scientific Committee on Consumer Products, in particular not exceeding a sunbed irradiance of 0.3 W m^{-2}.

Recommendation 2

We believe that it is important that inspections are carried out of commercial outlets to determine compliance with whatever level of regulation is imposed. We recommend that local authorities have a duty to inspect commercial outlets periodically and are given the appropriate powers of entry to premises and access to relevant information (eg maintenance records, staff on duty and accident reports). If licensing is enforced, the local authorities should be provided with sanctioning powers.

We recommend that the need for appropriate operator training is recognised, covering both the technology and safety of the sunbeds. Commercial outlets should be required to show local authorities that a standard level of competence is being met and that the outlet is staffed at all times with trained, competent personnel.

Recommendation 3

Skin cancer is the most common form of cancer in the UK and its incidence is continuing to rise, placing an increasing economic burden on the NHS. Historically, the budget allocated to raising the awareness of risk factors for skin cancer has been small. We recommend that funding for such campaigns is reviewed, taking into consideration that spent on other national health campaigns.

We recommend that stronger publicity campaigns on the risks from UV radiation exposure, and in particular sunbeds, are directed towards children, as users or potential users of sunbeds. Such campaigns could focus on photoageing effects from sunbeds to enhance the message.

We also recommend that the appropriate authorities strictly review the advertising employed by the sunbed industry.

Recommendation 4

The complete risks associated with the use of sunbeds have not been fully established due to the long latency period of skin cancers and the relatively recent widespread usage of sunbeds. We recommend that further research is required into sunbed usage and the risk and aetiology of malignant melanomas and non-melanoma skin cancers (NMSCs). This research should include detailed investigations into skin damage from melanomas and NMSCs, with particular reference to ageing.

Additional research is also recommended into the potential and reported ocular damage resulting from the use of sunbeds without adequate eye protection.

We recommend that population-based research should be undertaken to correlate skin damage and sunbed use (ie number of sessions, duration and strength of machine) and control for holiday exposure. This should investigate socio-economic factors, access to sunbeds and age of use, where possible.

There is also a requirement for research to establish why some fair-skinned people find tanning desirable and to determine how behaviour may be changed. The recent tanning phenomenon could be correctable with a different approach to body image; however, background knowledge of the psychology for tanning needs to be determined.

REFERENCES

Abdel-Malek Z, Suzuki I, Tada A, Im S, and Akcali C (1999). The melanocortin-1 receptor and human pigmentation. *Ann NY Acad Sci* **885**, 117–133.

ACCC (2001). ACCC crackdown on solarium claims. Release # MR 194/01. Australian Competition and Consumer Commission. http://www.accc.gov.au/content/index.phtml/itemId/87813/fromItemId/378012 (accessed May 2009).

Advertising Standards Association (2005). Non-broadcast Adjudications on an objection to a leaflet from The Sunbed Association. Advertising Standards Association. http://www.asa.org.uk/asa/adjudications/non_broadcast/Adjudication+Details.htm?Adjudication_id=40251 (accessed May 2009).

AGNIR (1995). Health Effects from Ultraviolet Radiation (Report of an Advisory Group on Non-ionising Radiation). *Doc NRPB* **6**, 7–190.

AGNIR (2002). Health Effects from Ultraviolet Radiation (Report of an Advisory Group on Non-ionising Radiation). *Doc NRPB* **13**, 11–268. Available at www.hpa.org.uk (accessed May 2009).

All Party Parliamentary Group on Skin (2008). Skin Cancer – Improving Prevention, Treatment and Care. All Party Parliamentary Group on Skin, London.

Amir Z, Wright A, Kernohan E E, and Hart G (2000). Attitudes, beliefs and behaviour regarding the use of sunbeds amongst healthcare workers in Bradford. *Eur J Cancer Care (Engl)* **9**, 76–79.

Apollo M and Muma R (2007). A study of tanning operators in the state of Kansas: their attitudes and stated practices regarding minors and tanning. In *Proceedings of the 3rd Annual GRASP Symposium. Wichita State University* http://soar.wichita.edu:8080/dspace/bitstream/10057/703/1/grasp+62.pdf (accessed May 2009).

Armstrong B and Kricker A (1993). Sun exposure causes both non-melanocytic skin cancer and malignant melanoma. In *Proceedings on Environmental UV Radiation and Health Effects,* pp 106–113.

Australia/New Zealand Standard (2002). Solaria for cosmetic purposes. AS/NZS 2635:2002. http://www.public.health.wa.gov.au/cproot/1487/2/key_requirements_of_ASNZS2635 2002_solaria_for_cosmetic_purposes.pdf (accessed May 2009).

Autier P and Boyle P (2008). Artificial ultraviolet sources and skin cancers: rationale for restricting access to sunbed use before 18 years of age. *Natl Clin Pract Oncol* **5**, 178–179.

Autier P and Gandini S (2007). Vitamin D supplementation and total mortality: a meta-analysis of randomized controlled trials. *Arch Intern Med* **167**, 1730–1737.

Bastiaens M, ter Huurne J, Gruis N, Bergman W, Westendorp R, Vermeer B J, and Bouwes Bavinck J N (2001). The melanocortin-1-receptor gene is the major freckle gene. *Hum Mol Genet* **10**, 1701–1708.

Bataille V, Grulich A, Sasieni P, Swerdlow A, Newton B J, McCarthy W, Hersey P, and Cuzick J (1998). The association between naevi and melanoma in populations with different levels of sun exposure: a joint case–control study of melanoma in the UK and Australia. *Br J Cancer* **77**, 505–510.

Bataille V, Snieder H, MacGregor A J, Sasieni P, and Spector T D (2000). Genetics of risk factors for melanoma: an adult twin study of nevi and freckles. *J Natl Cancer Inst* **92**, 457–463.

Bataille V, Winnett A, Sasieni P, Newton Bishop J A, and Cuzick J (2004). Exposure to the sun and sunbeds and the risk of cutaneous melanoma in the UK: a case–control study. *Eur J Cancer* **40**, 429–435.

Bates C J, Carter G D, Mishra G D, O'Shea D, Jones J, and Prentice A (2003). In a population study, can parathyroid hormone aid the definition of adequate vitamin D status? A study of people aged 65 years and over from the British National Diet and Nutrition Survey. *Osteoporos Int* **14**, 152–159.

BBC News (2005). Young children 'using sun beds'. BBC News Website. http://news.bbc.co.uk/1/hi/england/merseyside/4527590.stm (accessed May 2009).

BBC News (2008). Sunbed use 'puts lives at risk'. BBC News Website. http://news.bbc.co.uk/1/hi/health/7334385.stm (accessed May 2009).

BBC News (2009). Girl, 14, burnt in tanning salon. BBC News Website. http://news.bbc.co.uk/1/hi/wales/7899199.stm (accessed May 2009).

BBC Newsround (2004). Should kids be banned from using sunbeds? BBC News Website. http://news.bbc.co.uk/cbbcnews/hi/chat/your_comments/newsid_3405000/3405619.stm (accessed May 2009).

Bergman W, Palan A, and Went L N (1986). Clinical and genetic studies in six Dutch kindreds with the dysplastic naevus syndrome. *Ann Hum Genet* **50**, 249–258.

Berneburg M and Krutmann J (1998). Mitochondrial DNA deletions in human skin reflect photo- rather than chronologic aging. *J Invest Dermatol* **111**, 709–710.

Besaratinia A, Kim S I, and Pfeifer G P (2008). Rapid repair of UVA-induced oxidized purines and persistence of UVB-induced dipyrimidine lesions determine the mutagenicity of sunlight in mouse cells. *FASEB J* **22**, 2379–2392.

Birmingham City Council (1990). Licensing: Massage and Special Treatments – Birmingham City Council Act. http://www.birmingham.gov.uk/generatecontent?content_item_id=6636&content_item_type=0&menu_id=0 (accessed May 2009).

Black D (1984). Investigation of the possible increased incidence of cancer in West Cumbria. Report of the Independent Advisory Group. HMSO, London.

Boldeman C, Branstrom R, Dal H, Kristjansson S, Rodvall Y, Jansson B, and Ullen H (2001). Tanning habits and sunburn in a Swedish population age 13–50 years. *Eur J Cancer* **37**, 2441–2448.

Böttger A (2007). Experiences with voluntary regulation – Germany. Presented at EUROSKIN 5th International Conference, Hamburg.

Brandberg Y, Ullen H, Sjoberg L, and Holm L E (1998). Sunbathing and sunbed use related to self-image in a randomized sample of Swedish adolescents. *Eur J Cancer Prev.* **7**, 321–329.

British Standards Institution (2003). Specification for safety of household and similar electrical appliances. Particular requirements for appliances for skin exposure to ultraviolet and infrared radiation. BS EN 60335-2-27: 2003.

Brown P, Sandhu J, and Verne J (2004). Ptolemy's View on Melanoma in England. Presented at Association of Public Health Observatories Annual Conference.

Burleigh E and Potter J (2006). Vitamin D deficiency in outpatients: a Scottish perspective. *Scott Med J* **51**, 27–31.

Burnet N G, Jefferies S J, Benson R J, Hunt D P, and Treasure F P (2005). Years of life lost (YLL) from cancer is an important measure of population burden – and should be considered when allocating research funds. *Br J Cancer* **92**, 241–245.

Cancer Research UK (2004). Sunbed summit seeks to ban tanning for under 16s. Cancer Research UK press statement. http://info.cancerresearchuk.org/news/archive/pressreleases/2004/march/38926 (accessed May 2009).

Cancer Research UK (2008a). CancerStats – UK skin cancer incidence statistics. http://info.cancerresearchuk.org/cancerstats/types/skin/incidence/ (accessed September 2008).

Cancer Research UK (2008b). SunSmart campaign. http://info.cancerresearchuk.org/healthyliving/sunsmart/ (accessed May 2009).

Cancer Research UK (2008c). CancerStats – UK skin cancer mortality statistics. http://info.cancerresearchuk.org/cancerstats/types/skin/mortality/ (accessed September 2008).

Cancer Research UK (2008d). Using sunbeds under 35 could prove fatal. Cancer Research UK press statement. http://info.cancerresearchuk.org/news/archive/pressreleases/2008/april/422513 (accessed May 2009).

Cancer Research UK (2008e). Underage Use of Sunbeds – Scoping Study. Work for the Department of Health, London.

Ceilley R I and Del Rosso J Q (2006). Current modalities and new advances in the treatment of basal cell carcinoma. *Int J Dermatol* **45**, 489–498.

Césarini J-P (2007). Experience with legal regulations: France. Presented at EUROSKIN 5th International Conference, Hamburg.

CIEH (2005). Saving our skins toolkit. Raising Awareness of the Risk of Skin Cancer. Chartered Institute of Environmental Health. http://www.cieh.org/library/knowledge/public_health/skin_cancer/saving%20our%20skins%20toolkit.pdf (accessed May 2009).

CIEH (Wales) (2008). The Sunbed Control Study. Chartered Institute of Environmental Health (Wales). http://www.cieh-cymruwales.org/uploadedfiles/core/policy/research/sunbed_control_study.pdf (accessed May 2009).

Cleaver J E and Crowley E (2002). UV damage, DNA repair and skin carcinogenesis. *Front Biosci* **7**, d1024–d1043.

Danish Medicines Agency (2008). Warning against the product Melanotan. http://www.dkma.dk/1024/visUKLSArtikel.asp?artikelID=13865 (accessed May 2009).

de Gruijl F R, van Kranen H J, and Mullenders L H (2001). UV-induced DNA damage, repair, mutations and oncogenic pathways in skin cancer. *J Photochem Photobiol B* **63**, 19–27.

de Vries E, Bray F I, Coebergh J W, and Parkin D M (2003). Changing epidemiology of malignant cutaneous melanoma in Europe 1953–1997: rising trends in incidence and mortality but recent stabilizations in western Europe and decreases in Scandinavia. *Int J Cancer* **107**, 119–126.

de Vries H, Willems K, Mesters I, and Reubsaet A (2006). Skin cancer prevention behaviours during summer holidays in 14 and 18-year-old Belgian adolescents. *Eur J Cancer Prev* **15**, 431–438.

Demko C A, Borawski E A, Debanne S M, Cooper K D, and Stange K C (2003). Use of indoor tanning facilities by white adolescents in the United States. *Arch Pediatr Adolesc Med* **157**, 854–860.

Dennis L K (1999). Increasing risk of melanoma with increasing age. *JAMA* **282**, 1037–1038.

Dennis L K, Vanbeek M J, Beane Freeman L E, Smith B J, Dawson D V, and Coughlin J A (2008). Sunburns and risk of cutaneous melanoma: does age matter? A comprehensive meta-analysis. *Ann Epidemiol* **18**, 614–627.

Department of Health (2007). Cancer Reform Strategy. http://www.dh.gov.uk/en/publicationsandstatistics/publications/publicationspolicyandguidance/dh_081006 (accessed May 2009).

Department of Health and Children (2008). Public Consultation – Proposed Legislation to Regulate Sunbeds. http://www.dohc.ie/consultations/closed/sunbed_consultation/ (accessed May 2009).

Devereux C, O'Hagan A, Furey B, McElwee G, McEvoy G, Cuaghey J, McPeak L, Fitzsimmons L, Smart L, McMahon N, Loan P, Martin S, Gordon S, Crossin T, and Gavin A (2008). Study of operating practices in tanning bed parlours in Northern Ireland. Presented at *Irish Association of Dermatologists – Spring Meeting* http://www.irishdermatologists.ie/incs/pdf/abstract4_2008.pdf (accessed May 2009).

Devgun M S, Johnson B E, and Paterson C R (1982). Tanning, protection against sunburn and vitamin D formation with a UV-A 'sun-bed'. *Br J Dermatol* **107**, 275–284.

Dhomen N and Marais R (2007). New insight into BRAF mutations in cancer. *Curr Opin Genet Dev* **17**, 31–39.

Dhomen N, Reis-Filho J S, da Rocha D S, Hayward R, Savage K, Delmas V, Larue L, Pritchard C, and Marais R (2009). Oncogenic Braf induces melanocyte senescence and melanoma in mice. *Cancer Cell* **15**, 294–303.

Diffey B L (1986). Use of UV-A sunbeds for cosmetic tanning. *Br J Dermatol* **115**, 67–76.

Diffey B L (2003). A quantitative estimate of melanoma mortality from ultraviolet A sunbed use in the U.K. *Br J Dermatol* **149**, 578–581.

Diffey B (1997). Sunbeds: what are they, who uses them and what are the health effects? Health Education Authority, London.

Diffey B (2008). A behavioral model for estimating population exposure to solar ultraviolet radiation. *Photochem Photobiol* **84**, 371–375.

Diffey B L, Farr P M, Ferguson J, Gibbs N K, deGruijl F R, Hawk J L, Johnson B E, Lowe G, Mackie R M, and McKinlay A F (1990). Tanning with ultraviolet A sunbeds. *BMJ* **301**, 773–774.

Dobbinson S, Wakefield M, and Sambell N (2006). Access to commercial indoor tanning facilities by adults with highly sensitive skin and by under-age youth: compliance tests at solarium centres in Melbourne, Australia. *Eur J Cancer Prev* **15**, 424–430.

Dobnig H, Pilz S, Scharnagl H, Renner W, Seelhorst U, Wellnitz B, Kinkeldei J, Boehm B O, Weihrauch G, and Maerz W (2008). Independent association of low serum 25-hydroxyvitamin d and 1,25-dihydroxyvitamin d levels with all-cause and cardiovascular mortality. *Arch Intern Med* **168**, 1340–1349.

Dorset for You (2008). Massage and special treatment licence (MST). http://www.dorsetforyou.com/index.jsp?articleid=387212 (accessed May 2009).

Downing A, Newton-Bishop J A, and Forman D (2006). Recent trends in cutaneous malignant melanoma in the Yorkshire region of England; incidence, mortality and survival in relation to stage of disease, 1993–2003. *Br J Cancer* **95**, 91–95.

Duffy D L, Box N F, Chen W, Palmer J S, Montgomery G W, James M R, Hayward N K, Martin N G, and Sturm R A (2004). Interactive effects of MC1R and OCA2 on melanoma risk phenotypes. *Hum Mol Genet* **13**, 447–461.

Easton D F, Cox G M, Macdonald A M, and Ponder B A (1991). Genetic susceptibility to naevi – a twin study. *Br J Cancer* **64**, 1164–1167.

Eltigani E and Matthews R N (1994). An unusual cause of sunbed burns. *Burns* **20**, 87–88.

Emmett A, Uchida T, and Wagner R F, Jr (2008). Sunburn risk factors for beachgoing children. *Dermatol Online J* **14**, 28.

Environmental Health Journal (2005). Skin cancer – the next burning issue. *EHJ* 8–10.

Essex County Council (1987). Essex Act – Part VI – Establishments for Massage or Special Treatment. http://www.opsi.gov.uk/acts/localact1987/pdf/ukla_19870020_en.pdf (accessed May 2009).

European Commission (1973). Low Voltage Directive 73/23/EEC (1973). L77. Luxembourg, European Commission.

European Commission (2006). Low Voltage Directive 2006/95/EC. L374. http://ec.europa.eu/enterprise/electr_equipment/lv/index.htm (accessed May 2009).

European Standards (1997). Specification for safety of household and similar electrical appliances. Particular requirements. Skin exposure to ultraviolet and infrared radiation. EN 60335-2-27.

EUROSKIN (2005). Proposed code of practice for artificial tanning – Euroskin-DD-01. European Society of Skin Cancer Prevention. http://www.euroskin.eu/downloads/proposedcodeof.pdf (accessed May 2009).

Ezzedine K, Malvy D, Mauger E, Nageotte O, Galan P, Hercberg S, and Guinot C (2008). Artificial and natural ultraviolet radiation exposure: beliefs and behaviour of 7200 French adults. *J Eur Acad Dermatol Venereol* **22**, 186–194.

Farr P M, Marks J M, Diffey B L, and Ince P (1988). Skin fragility and blistering due to use of sunbeds. *BMJ (Clin Res Ed)* **296**, 1708–1709.

Fiala B, Kopp M, and Gunther V (1997). Why do young women use sunbeds? A comparative psychological study. *Br J Dermatol* **137**, 950–954.

Fisher G J, Kang S, Varani J, Bata-Csorgo Z, Wan Y, Datta S, and Voorhees J J (2002). Mechanisms of photoaging and chronological skin aging. *Arch Dermatol* **138**, 1462–1470.

Fisher M S and Kripke M L (1977). Systemic alteration induced in mice by ultraviolet light irradiation and its relationship to ultraviolet carcinogenesis. *Proc Natl Acad Sci USA* **74**, 1688–1692.

Fisher M S and Kripke M L (1982). Suppressor T lymphocytes control the development of primary skin cancers in ultraviolet-irradiated mice. *Science* **216**, 1133–1134.

Fisher M S and Kripke M L (2002). Systemic alteration induced in mice by ultraviolet light irradiation and its relationship to ultraviolet carcinogenesis. 1977. *Bull World Health Organ* **80**, 908–912.

Friedberg E C, Walker G C, Siede W, Wood R D, Schultz R A, and Ellenberger T (2006). *DNA Repair and Mutagenesis.* 2nd Edition. ASM Press.

Gallagher R P and Lee T K (2006). Adverse effects of ultraviolet radiation: a brief review. *Prog Biophys Mol Biol* **92**, 119–131.

Gandini S, Sera F, Cattaruzza M S, Pasquini P, Abeni D, Boyle P, and Melchi C F (2005a). Meta-analysis of risk factors for cutaneous melanoma: I. Common and atypical naevi. *Eur J Cancer* **41**, 28–44.

Gandini S, Sera F, Cattaruzza M S, Pasquini P, Picconi O, Boyle P, and Melchi C F (2005b). Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *Eur J Cancer* **41**, 45–60.

Gandini S, Sera F, Cattaruzza M S, Pasquini P, Zanetti R, Masini C, Boyle P, and Melchi C F (2005c). Meta-analysis of risk factors for cutaneous melanoma: III. Family history, actinic damage and phenotypic factors. *Eur J Cancer* **41**, 2040–2059.

Geller A C, Colditz G, Oliveria S, Emmons K, Jorgensen C, Aweh G N, and Frazier A L (2002). Use of sunscreen, sunburning rates, and tanning bed use among more than 10 000 US children and adolescents. *Pediatrics* **109**, 1009–1014.

Gerber B, Mathys P, Moser M, Bressoud D, and Braun-Fahrlander C (2002). Ultraviolet emission spectra of sunbeds. *Photochem Photobiol* **76**, 664–668.

Gerdhem P, Ringsberg K A, Obrant K J, and Akesson K (2005). Association between 25-hydroxy vitamin D levels, physical activity, muscle strength and fractures in the prospective population-based OPRA Study of Elderly Women. *Osteoporos Int* **16**, 1425–1431.

Giovannucci E, Liu Y, Hollis B W, and Rimm E B (2008). 25-hydroxyvitamin D and risk of myocardial infarction in men: a prospective study. *Arch Intern Med* **168**, 1174–1180.

Giovannucci E, Liu Y, Rimm E B, Hollis B W, Fuchs C S, Stampfer M J, and Willett W C (2006). Prospective study of predictors of vitamin D status and cancer incidence and mortality in men. *J Natl Cancer Inst* **98**, 451–459.

Goodhead D T (1989). The initial physical damage produced by ionizing radiations. *Int J Radiat Biol* **56**, 623–634.

Gordon L G, Hirst N G, Gies P H, and Green A C (2008). What impact would effective solarium regulation have in Australia? *Med J Aust* **189**, 375–378.

Government of South Australia (2008a). Radiation Protection and Control (Cosmetic Tanning Units) Regulations under the Radiation Protection and Control Act 1982. http://www.legislation.sa.gov.au/lz/c/r/radiation%20protection%20and%20control%20(cosmetic%20tanning%20units)%20regulations%202008/current/2008.23.un.pdf (accessed May 2009).

Government of South Australia (2008b). Radiation Protection and Control (Non-ionising Radiation) Regulations under the Radiation Protection and Control Act 1982. http://www.legislation.sa.gov.au/lz/c/r/radiation%20protection%20and%20control%20(non-ionising%20radiation)%20regulations%202008/current/2008.24.un.pdf (accessed May 2009).

Grant W B and Holick M F (2005). Benefits and requirements of vitamin D for optimal health: a review. *Altern Med Rev* **10**, 94–111.

Green A, Siskind V, Bain C, and Alexander J (1985). Sunburn and malignant melanoma. *Br J Cancer* **51**, 393–397.

Guenel P, Laforest L, Cyr D, Fevotte J, Sabroe S, Dufour C, Lutz J M, and Lynge E (2001). Occupational risk factors, ultraviolet radiation, and ocular melanoma: a case–control study in France. *Cancer Causes Control* **12**, 451–459.

Hamlet N and Kennedy K (2004). Reconnaissance study of sunbed use by primary school children in Lanarkshire. *J Public Health (Oxf)* **26**, 31–33.

Harding R M, Healy E, Ray A J, Ellis N S, Flanagan N, Todd C, Dixon C, Sajantila A, Jackson I J, Birch-Machin M A, and Rees J L (2000). Evidence for variable selective pressures at MC1R. *Am J Hum Genet* **66**, 1351–1361.

Hawk J L (1984). Photosensitizing agents used in the United Kingdom. *Clin Exp Dermatol* **9**, 300–302.

Hemminki K, Zhang H, and Czene K (2003). Familial and attributable risks in cutaneous melanoma: effects of proband and age. *J Invest Dermatol* **120**, 217–223.

Hirst N, Gordon L, Gies P, and Green A C (2008). Estimation of avoidable skin cancers and cost-savings to government associated with regulation of the solarium industry in Australia. *Health Policy* **89**(3), 303–311.

Hoerster K D, Mayer J A, Woodruff S I, Malcarne V, Roesch S C, and Clapp E (2007). The influence of parents and peers on adolescent indoor tanning behavior: findings from a multi-city sample. *J Am Acad Dermatol* **57**, 990–997.

Holick M F, Chen T C, Lu Z, and Sauter E (2007). Vitamin D and skin physiology: A D-lightful story. *J Bone Mineral Res* **22**, V28–V33.

Holzle E (1992). Pigmented lesions as a sign of photodamage. *Br J Dermatol* **127**(Suppl 41), 48–50.

HSE (1998). Controlling health risks from the use of UV tanning equipment. Sudbury, Health and Safety Executive.

HSE (2009). Reducing health risks from the use of ultraviolet (UV) tanning equipment (INDG209). Sudbury, Health and Safety Executive. http://www.hse.gov.uk/pubns/indg209.pdf (accessed May 2009).

Hutchinson F (1985). Chemical changes induced in DNA by ionizing radiation. *Prog Nucleic Acid Res Mol Biol* **32**, 115–154.

Hypponen E and Power C (2007). Hypovitaminosis D in British adults at age 45 y: nationwide cohort study of dietary and lifestyle predictors. *Am J Clin Nutr* **85**, 860–868.

IARC (1992). *Solar and Ultraviolet Radiation. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* **55**, pp 73–138. http://monographs.iarc.fr/eng/monographs/vol55/volume55.pdf (accessed May 2009).

IARC (1997). *Cancer Incidence in Five Continents.* Volume II. D M Parkin et al, Eds. Lyon, International Agency for Research on Cancer.

IARC Working Group (2005). Exposure to artificial UV radiation and skin cancer/views and expert opinions of an IARC Working Group. *International Agency for Research on Cancer* **1**, http://www.iarc.fr/en/publications/pdfs-online/wrk/wrk1/ArtificialUVRad&SkinCancer.pdf (accessed May 2009).

IARC Working Group (2007). The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers: a systematic review. *Int J Cancer* **120**, 1116–1122.

Ichihashi M, Nagai H, and Matsunaga K (2004). Sunlight is an important causative factor of recurrent herpes simplex. *Cutis* **74**, 14–18.

ICNIRP (2003). Health issues of ultraviolet tanning appliances used for cosmetic purposes. *Health Phys* **84**, 119–127.

Jablonski N G and Chaplin G (2000). The evolution of human skin coloration. *J Hum Evol* **39**, 57–106.

Jackson P J, Douglas N R, Chai B, Binkley J, Sidow A, Barsh G S, and Millhauser G L (2006). Structural and molecular evolutionary analysis of Agouti and Agouti-related proteins. *Chem Biol* **13**, 1297–1305.

John E M, Koo J, and Schwartz G G (2007). Sun exposure and prostate cancer risk: evidence for a protective effect of early-life exposure. *Cancer Epidemiol Biomarkers Prev* **16**, 1283–1286.

Kraemer K H, Lee M M, Andrews A D, and Lambert W C (1994). The role of sunlight and DNA repair in melanoma and nonmelanoma skin cancer. The xeroderma pigmentosum paradigm. *Arch Dermatol* **130**, 1018–1021.

Kuijken I and Bouwes Bavinck J N (2000). Skin cancer risk associated with immunosuppressive therapy in organ transplant recipients: epidemiology and proposed mechanisms. *BioDrugs* **14**, 319–329.

Lachiewicz A M, Berwick M, Wiggins C L, and Thomas N E (2008). Epidemiologic support for melanoma heterogeneity using the surveillance, epidemiology, and end results program. *J Invest Dermatol* **128**, 1340–1342.

Langan E A, Ramlogan D, Jamieson L A, and Rhodes L E (2009). Change in moles linked to use of unlicensed 'sun tan jab'. *BMJ* **338**, b277.

Lappe J M, Travers-Gustafson D, Davies K M, Recker R R, and Heaney R P (2007). Vitamin D and calcium supplementation reduces cancer risk: results of a randomized trial. *Am J Clin Nutr* **85**, 1586–1591.

Larsen W G (1985). Perfume dermatitis. *J Am Acad Dermatol* **12**, 1–9.

Lawley P D (1966). Effects of some chemical mutagens and carcinogens on nucleic acids. *Prog Nucleic Acid Res Mol Biol* **5**, 89–131.

Lazovich D and Forster J (2005). Indoor tanning by adolescents: prevalence, practices and policies. *Eur J Cancer* **41**, 20–27.

Lee E, Koo J, and Berger T (2005). UVB phototherapy and skin cancer risk: a review of the literature. *Int J Dermatol* **44**, 355–360.

Légifrance (1997). Décret no 97-617 du 30 mai 1997 relatif à la vente et à la mise à disposition du public de certains appareils de bronzage utilisant des rayonnements ultraviolets. http://www.legifrance.gouv.fr/affichtexte.do?cidtexte=jorftext000000567033&datetexte=&fastpos=1&fastreqid=27011572&oldaction=rechtexte (accessed May 2009).

Leiter U and Garbe C (2008). Epidemiology of melanoma and nonmelanoma skin cancer – the role of sunlight. *Adv Exp Med Biol* **624**, 89–103.

Leroi A M (2003). *Mutants: On the Form, Varieties and Errors of the Human Body.* Harper Collins.

Levine J A, Sorace M, Spencer J, and Siegel D M (2005). The indoor UV tanning industry: a review of skin cancer risk, health benefit claims, and regulation. *J Am Acad Dermatol* **53**, 1038–1044.

Li Y, Gudjonsson J E, Woods T L, Zhang T, Johnston A, Stoll S W, and Elder J T (2009). Transgenic expression of S100A2 in hairless mouse skin enhances Cxcl13 mRNA in response to solar-simulated radiation. *Arch Dermatol Res* **301**(3), 205–217.

Lindelof B, Sigurgeirsson B, Tegner E, Larko O, Johannesson A, Berne B, Christensen O B, Andersson T, Torngren M, and Molin L (1991). PUVA and cancer: a large-scale epidemiological study. *Lancet* **338**, 91–93.

Lips P (2006). Vitamin D physiology. *Prog Biophys Mol Biol* **92**, 4–8.

London Local Authorities (1991). London Local Authorities Act – Chapter xiii. Part ii – Special Treatment Premises. http://www.opsi.gov.uk/acts/localact1991/ukla_19910013_en_1#legislation-preamble (accessed May 2009).

Lynch H T, Frichot B C, Lynch P, Lynch J, and Gurigis H A (1975). Family studies of malignant melanoma and associated cancer. *Surg Gynecol Obstet* **141**, 517–522.

Mackay H, Lowe D, Edwards D, and Rogers S N (2007). A survey of 14 to 16 year olds as to their attitude toward and use of sunbeds. *Health Education Journal,* **66**, 141–152.

Mackie R M (2006). Long-term health risk to the skin of ultraviolet radiation. *Prog Biophys Mol Biol* **92**, 92–96.

Mackintosh K (2006). The Regulation of Sunbed Parlours Bill: A Consultation. http://www.scottish.parliament.uk/business/bills/pdfs/mb-consultations/sunbedparloursbillconsultation.pdf (accessed May 2009).

MacLaughlin J and Holick M F (1985). Aging decreases the capacity of human skin to produce vitamin D3. *J Clin Invest* **76**, 1536–1538.

Maki H (2002). Origins of spontaneous mutations: specificity and directionality of base-substitution, frameshift, and sequence-substitution mutageneses. *Ann Rev Genet* **36**, 279–303.

Marchetto M C, Muotri A R, Burns D K, Friedberg E C, and Menck C F (2004). Gene transduction in skin cells: preventing cancer in xeroderma pigmentosum mice. *Proc Natl Acad Sci USA* **101**, 17759–17764.

McCarty C A, Fu C L, and Taylor H R (2000). Epidemiology of pterygium in Victoria, Australia. *Br J Ophthalmol* **84**, 289–292.

McGinley J, Martin C J, and Mackie R M (1998). Sunbeds in current use in Scotland: a survey of their output and patterns of use. *Br J Dermatol* **139**, 428–438.

McWhirter W R and Dobson C (1995). Childhood melanoma in Australia. *World J Surg* **19**, 334–336.

MHRA (2008). Press Release. 'Tan jab' is an unlicensed medicine and may not be safe – warns medicines regulator. Medicines and Healthcare products Regulatory Agency, London. http://www.mhra.gov.uk/newscentre/pressreleases/con031009 (accessed May 2009).

Ministry of Health and Social Affairs (Finland) (2002). Decree on the Limitation of Public Exposure to Non-ionizing Radiation (294/2002). http://www.stuk.fi/sateilytietoa/sateilevat_laitteet/en_GB/solarium/_files/12222632510024445/default/STMasetus294-2002english.pdf (accessed May 2009).

Ministry of the Presidency – Spain (2002). Crown Decree No. 102/2002 of 27 September 2002 regulating the sale and use of suntan equipment using ultraviolet radiations. http://www.boe.es/boe/dias/2002/10/10/pdfs/A35771-35774.pdf (accessed May 2009).

Mintel (2007). Electronic Beauty Aids. http://oxygen.mintel.com/sinatra/oxygen/search_results/show&/display/id=220131 (accessed May 2009).

Mitchell D L, Jen J, and Cleaver J E (1991). Relative induction of cyclobutane dimers and cytosine photohydrates in DNA irradiated *in vitro* and *in vivo* with ultraviolet-C and ultraviolet-B light. *Photochem Photobiol* **54**, 741–746.

Morris S, Cox B, and Bosanquet N (2005). Cost of Skin Cancer in England. *Tanaka Business School Discussion Papers.* TBS/DP05/39. http://www3.imperial.ac.uk/pls/portallive/docs/1/43013.pdf (accessed May 2009).

Murphy G M, Wright J, Nicholls D S, McKee P H, Messenger A G, Hawk J L, and Levene G M (1989). Sunbed-induced pseudoporphyria. *Br J Dermatol* **120**, 555–562.

National Conference of State Legislatures (2008). Tanning Restrictions for Minors: A State-by-State Comparison. http://www.ncsl.org/programs/health/tanningrestrictions.htm#statelws (accessed May 2009).

National Tanning Training Institute (2008). A Guide to State Radiation Control Offices. http://www.tanningtraining.com/reginfo/state.html (accessed May 2009).

National Toxicology Program (2005). Report on Carcinogens. Eleventh Edition. US Department of Health and Human Services, Public Health Service. http://ntp-server.niehs.nih.gov/index.cfm?objectid=32BA9724-F1F6-975E-7FCE50709CB4C932 (accessed May 2009).

Neale R E, Purdie J L, Hirst L W, and Green A C (2003). Sun exposure as a risk factor for nuclear cataract. *Epidemiology* **14**, 707–712.

Newton Bishop J A, Bataille V, Pinney E, and Bishop D T (1994). Family studies in melanoma: identification of the atypical mole syndrome (AMS) phenotype. *Melanoma Res* **4**, 199–206.

Norris W (1820). A case of fungoid disease. *Edin Med Surg J* **16**, 562–565.

Norval M, Cullen A P, de Gruijl F R, Longstreth J, Takizawa Y, Lucas R M, Noonan F P, and van der Leun J C (2007). The effects on human health from stratospheric ozone depletion and its interactions with climate change. *Photochem Photobiol Sci* **6**, 232–251.

Nottinghamshire County Council (1985). Nottinghamshire County Council Act – Part IV – Massage or Special Treatment.

NRPA (2003). Regulations No. 1362. Norwegian Radiation Protection Authority. http://www.nrpa.no/archive/Internett/Publikasjoner/Annet/act_eng.pdf (accessed May 2009).

O'Riordan D L, Field A E, Geller A C, Brooks D R, Aweh G, Colditz G A, and Frazier A L (2006). Frequent tanning bed use, weight concerns, and other health risk behaviors in adolescent females (United States). *Cancer Causes Control* **17**, 679–686.

O'Toole E and Barnes L (1995). Sun-bed-induced vesiculobullous polymorphic light eruption. *Br J Dermatol* **132**, 171.

Oliver H, Ferguson J, and Moseley H (2007). Quantitative risk assessment of sunbeds: impact of new high power lamps. *Br J Dermatol* **157**, 350–356.

Ovesen L, Andersen R, and Jakobsen J (2003). Geographical differences in vitamin D status, with particular reference to European countries. *Proc Nutr Soc* **62**, 813–821.

Page W F, Whiteman D, and Murphy M (2000). A comparison of melanoma mortality among WWII veterans of the Pacific and European theaters. *Ann Epidemiol* **10**, 192–195.

Paul C L, Stacey F, Girgis A, Brozek I, Baird H, and Hughes J (2005). Solaria compliance in an unregulated environment: the Australian experience. *Eur J Cancer* **41**, 1178–1184.

Peak M J, Peak J G, and Carnes B A (1987). Induction of direct and indirect single-strand breaks in human cell DNA by far- and near-ultraviolet radiations: action spectrum and mechanisms. *Photochem Photobiol.* **45**, 381–387.

Pfau R G, Hood A F, and Morison W L (1986). Photoageing: the role of UVB, solar-simulated UVB, visible and psoralen UVA radiation. *Br J Dermatol.* **114**, 319–327.

Piazena H (2007). Sunbed regulations in Europe – a survey. Presented at EUROSKIN 5th International Conference, Hamburg.

Pilz S, Dobnig H, Fischer J E, Wellnitz B, Seelhorst U, Boehm B O, and Marz W (2008). Low vitamin D levels predict stroke in patients referred to coronary angiography. *Stroke* **39**, 2611–2613.

Quintana E, Shackleton M, Sabel M S, Fullen D R, Johnson T M, and Morrison S J (2008). Efficient tumour formation by single human melanoma cells. *Nature* **456**, 593–598.

Raimondi S, Sera F, Gandini S, Iodice S, Caini S, Maisonneuve P, and Fargnoli M C (2008). MC1R variants, melanoma and red hair color phenotype: a meta-analysis. *Int J Cancer* **122**, 2753–2760.

Rass K and Reichrath J (2008). UV damage and DNA repair in malignant melanoma and nonmelanoma skin cancer. *Adv Exp Med Biol* **624**, 162–178.

Redcar & Cleveland Borough Council (2002). Redcar and Cleveland Borough Council Sun Tanning Survey.

Reed S H and Waters R (2003). DNA repair. In *The Encyclopedia of the Human Genome.* D Cooper, ed. Wiley Publishing Group.

Reginster J Y (2005). The high prevalence of inadequate serum vitamin D levels and implications for bone health. *Curr Med Res Opin* **21**, 579–586.

REHIS (2006). Survey of Sunbed Salons in Scotland. The Royal Environmental Health Institute of Scotland. http://www.rehis.org/media/news299.html (accessed May 2009).

Reimann V, Kramer U, Sugiri D, Schroeder P, Hoffmann B, Medve-Koenigs K, Jockel K H, Ranft U, and Krutmann J (2008). Sunbed use induces the photoaging-associated mitochondrial common deletion. *J Invest Dermatol* **128**, 1294–1297.

Rivers J K, Norris P G, Murphy G M, Chu A C, Midgley G, Morris J, Morris R W, Young A R, and Hawk J L (1989). UVA sunbeds: tanning, photoprotection, acute adverse effects and immunological changes. *Br J Dermatol* **120**, 767–777.

Rouzaud F and Hearing V J (2005). Regulatory elements of the melanocortin 1 receptor. *Peptides* **26**, 1858–1870.

SCCP (2006). Opinion on biological effects of ultraviolet radiation relevant to health with particular reference to sunbeds for cosmetic purposes. Scientific Committee on Consumer Products of the European Commission. SCCP/0949/05. http://ec.europa.eu/health/ph_risk/committees/04_sccp/docs/sccp_o_031b.pdf (accessed May 2009).

Schwartz G G and Skinner H G (2007). Vitamin D status and cancer: new insights. *Curr Opin Clin Nutr Metab Care* **10**, 6–11.

Scott P (2003). Coin-operated tanning salons – what's the true cost? *Unpublished work.* South West Public Health Observatory.

Scottish Parliament (2008). Public Health etc (Scotland) Act. http://www.opsi.gov.uk/legislation/scotland/acts2008/pdf/asp_20080005_en.pdf (accessed May 2009).

Service Public Federal Economie (2007). Royal Decree. Arrêté royal modifiant l'arrêté royal du 20 juin 2002 relatif aux conditions d'exploitation des centres de bronzage. http://www.staatsbladclip.be/lois/2007/12/28/loi-2007011544.html (accessed May 2009).

Setlow R B (1974). The wavelengths in sunlight effective in producing skin cancer: a theoretical analysis. *Proc Natl Acad Sci USA* **71**, 3363–3366.

State Government of Victoria (2007). Radiation (Tanning Units Amendment) Interim Regulations 2007. S.R. No. 148/2007. Australia. http://www.health.vic.gov.au/environment/downloads/solarium_regs.pdf (accessed May 2009).

Stern R S (1992). Risks of cancer associated with long-term exposure to PUVA in humans: current status – 1991. *Blood Cells* **18**, 91–97.

Stern R S and Docken W (1986). An exacerbation of SLE after visiting a tanning salon. *JAMA* **255**, 3120.

Straszburger G (2007). Sunbeds and solarium services: state of play. Presented at EUROSKIN 5th International Conference, Hamburg.

Suchak R, Pinto A, Devlin J, Limb P, and Cerio R (2008). Exposure to artificial tanning devices by young teenagers at a science college in Dudley, West Midlands. *Br J Dermatol* **159**(s1), 129.

Sulem P, Gudbjartsson D F, Stacey S N, Helgason A, Rafnar T, Magnusson K P, Manolescu A, Karason A, Palsson A, Thorleifsson G, Jakobsdottir M, Steinberg S, Palsson S, Jonasson F, Sigurgeirsson B, Thorisdottir K, Ragnarsson R, Benediktsdottir K R, Aben K K, Kiemeney L A, Olafsson J H, Gulcher J, Kong A, Thorsteinsdottir U, and Stefansson K (2007). Genetic determinants of hair, eye and skin pigmentation in Europeans. *Nat Genet* **39**, 1443–1452.

Suzuki I, Cone R D, Im S, Nordlund J, and Abdel-Malek Z A (1996). Binding of melanotropic hormones to the melanocortin receptor MC1R on human melanocytes stimulates proliferation and melanogenesis. *Endocrinology* **137**, 1627–1633.

Swedish Radiation Protection Institute (1998). The Swedish Radiation Protection Institute's Regulation on Sun Beds. http://www.stralsakerhetsmyndigheten.se/Global/Publikationer/Forfattning/Stralskydd/1998/ssifs-1998-2e.pdf (accessed May 2009).

SWPHO (2009). Sunbed outlets and area deprivation in the UK: A geographical investigation. *Unpublished work.* South West Public Health Observatory.

Taggart R and Starr C (2006). Mutated genes and their protein products. In *Biology: The Unity and Diversity of Life.* 11th Edition. Thompson Brookes Cole.

Taylor H R (1980). The prevalence of corneal disease and cataracts in Australian aborigines in Northwestern Australia. *Aust J Ophthalmol* **8**, 289–301.

Taylor H R (1981). Climatic droplet keratopathy and pterygium. *Aust J Ophthalmol* **9**, 199–206.

Tenkate T D and Collins M J (1997). Personal ultraviolet radiation exposure of workers in a welding environment. *Am Ind Hyg Assoc J* **58**, 33–38.

The Guardian (2005). Children as young as 11 use sunbed salons. http://www.guardian.co.uk/uk/2005/dec/14/health.topstories3 (accessed May 2009).

The Sunbed Association (2008). Written submission to Health and Sport Committee on Public Health etc. (Scotland) Bill. http://www.scottish.parliament.uk/s3/committees/hs/PublicHealthBill/PHB08SunbedAssociation.pdf (accessed May 2009).

Thieden E, Jorgensen H L, Jorgensen N R, Philipsen P A, and Wulf H C (2008). Sunbed radiation provokes cutaneous vitamin D synthesis in humans – a randomized controlled trial. *Photochem Photobiol* **84**, 1487–1492.

Ting W, Schultz K, Cac N N, Peterson M, and Walling H W (2007). Tanning bed exposure increases the risk of malignant melanoma. *Int J Dermatol* **46**, 1253–1257.

UK Parliament (1992). Statement of advice from COMARE to Health Departments. Health effects of ultra violet radiation. Written Answers to Questions. Hansard, cols 633–635, 1 July 1992. http://www.publications.parliament.uk/pa/cm199293/cmhansrd/1992-07-01/Writtens-6.html#Writtens-6_spnew3 (accessed May 2009)

US FDA (2007a). FDA issues warning letter to Melanocorp Inc for illegal sale of Melanotan II. US Food and Drug Administration. http://www.fda.gov/ForConsumers/default.htm (accessed May 2009).

US FDA (2007b). Performance Standards for Light-emitting Devices. 21CFR1040.20. 21CFR1040.20. US Food and Drug Administration. http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfCFR/CFRSearch.cfm?fr=1040.20 (accessed May 2009).

Van Der Meer I M, Boeke A J P, Lips P, Grootjans-Geerts I, Wuister J D, Deville W L, Wielders J P M, Bouter L M, and Middelkoop B J C (2008). Fatty fish and supplements are the greatest modifiable contributors to the serum 25-hydroxyvitamin D concentration in a multiethnic population. *Clin Endocrinol* **68**, 466–472.

van der Mei I A, Ponsonby A L, Engelsen O, Pasco J A, McGrath J J, Eyles D W, Blizzard L, Dwyer T, Lucas R, and Jones G (2007). The high prevalence of vitamin D insufficiency across Australian populations is only partly explained by season and latitude. *Environ Health Perspect* **115**, 1132–1139.

van Steeg H, de Vries A, van Oostrom C T, van Benthem J, Beems R B, and van Kreijl C F (2001). DNA repair-deficient Xpa and Xpa/p53+/- knock-out mice: nature of the models. *Toxicol Pathol* **29**(Suppl), 109–116.

Wachsmuth R C, Gaut R M, Barrett J H, Saunders C L, Randerson-Moor J A, Eldridge A, Martin N G, Bishop T D, and Newton Bishop J A (2001). Heritability and gene-environment interactions for melanocytic nevus density examined in a UK adolescent twin study. *J Invest Dermatol* **117**, 348–352.

Wachsmuth R C, Turner F, Barrett J H, Gaut R, Randerson-Moor J A, Bishop D T, and Bishop J A (2005). The effect of sun exposure in determining nevus density in UK adolescent twins. *J Invest Dermatol* **124**, 56–62.

Wallace D C, Beardmore G L, and Exton L A (1973). Familial malignant melanoma. *Ann Surg* **177**, 15–20.

Wang Y, Zhou X, Weinstein E, Maryles B, Zhang Y, Moore J, Gao D, Atencio D P, Rosenstein B S, Lebwohl M, Chen H D, Xiao T, and Wei H (2008). *p53* gene mutations in SKH-1 mouse tumors differentially induced by UVB and combined subcarcinogenic benzo[a]pyrene and UVA. *Photochem Photobiol* **84**, 444–449.

Watson J and Crick F (1953). Molecular structure of nucleic acids; a structure for deoxyribose nucleic acid. *Nature* **171**, 737–738.

Webb A R (2006). Who, what, where and when – influences on cutaneous vitamin D synthesis. *Prog Biophys Mol Biol* **92**, 17–25.

Weinberg R A (2006). *The Biology of Cancer.* Garland Science.

Which? (2008). The heat is on. *Which?* Magazine May 2008, 38–39.

Whiteman D C, Watt P, Purdie D M, Hughes M C, Hayward N K, and Green A C (2003). Melanocytic nevi, solar keratoses, and divergent pathways to cutaneous melanoma. *J Natl Cancer Inst* **95**, 806–812.

Whiteman D C, Whiteman C A, and Green A C (2001). Childhood sun exposure as a risk factor for melanoma: a systematic review of epidemiologic studies. *Cancer Causes Control* **12**, 69–82.

Whiting S J, Green T J, and Calvo M S (2007). Vitamin D intakes in North America and Asia–Pacific countries are not sufficient to prevent vitamin D insufficiency. *J Steroid Biochem Mol Biol* **103**, 626–630.

WHO (1994). *Ultraviolet Radiation* (2nd Edition). *Environmental Health Criteria* **160**. Geneva, World Health Organization. http://www.inchem.org/documents/ehc/ehc/ehc160.htm (accessed May 2009).

WHO (2003). Artificial Tanning Sunbeds – Risks and Guidance. Geneva, World Health Organization. http://www.who.int/uv/publications/en/sunbeds.pdf (accessed May 2009).

WHO (2009). Ultraviolet Radiation and the INTERSUN Programme – Health Effects of UV Radiation. Geneva, World Health Organization. http://www.who.int/uv/health/en/ (accessed May 2009).

Wright A L, Hart G C, Kernohan E, and Twentyman G (1996). Survey of the variation in ultraviolet outputs from ultraviolet A sunbeds in Bradford. *Photodermatol Photoimmunol Photomed* **12**, 12–16.

Zhu G, Duffy D L, Eldridge A, Grace M, Mayne C, O'Gorman L, Aitken J F, Neale M C, Hayward N K, Green A C, and Martin N G (1999). A major quantitative-trait locus for mole density is linked to the familial melanoma gene CDKN2A: a maximum-likelihood combined linkage and association analysis in twins and their sibs. *Am J Hum Genet* **65**, 483–492.

Zlateva V, Toncheva R, and Andreev A (1996). Epidemiological studies on occupational eye pathology. *Eur J Ophthalmol* **6**, 440–445.

ACKNOWLEDGEMENTS

Ms Kim Willis, Research Project Officer at the Chartered Institute of Environmental Health, for researching the controls in place for sunbeds outside the UK.

THE APPENDICES

APPENDIX A

GLOSSARY

Aetiology	The cause(s) of a disease.
Agouti protein (ASIP)	The agouti signalling peptide (also referred to as Agouti) acts as an antagonist at melanocortin receptors, specifically MC1.
Antagonist	A chemical substance that interferes with the physiological action of another through a blocking mechanism.
Artificial UV radiation source	A UV radiation source other than the sun.
Basal cell carcinoma (BCC)	The most common non-melanoma skin cancer, originating from basal cells, usually occurs as a pearly nodule or plaqued with central depression. It begins in the lowest layer of the epidermis, called the basal cell layer. It usually develops on sun-exposed areas, especially the head and neck, but may also be common on the trunk. Basal cell carcinomas are slow growing and rarely spread to other parts of the body. They are almost always curable.
Benign	Not malignant. Incapable of invading tissue boundaries or to metastasise. Unlikely *per se* to lead to death.
Bulimia	An eating disorder characterised by episodic binge eating and often followed by compensatory behaviours, such as purging, and associated with feelings of guilt and depression.
Carcinogen	An agent that causes cancer.
Carcinogenesis	Production and development of cancer.
Cataract	An opacity, partial or complete, on the lens of the eye which may impair vision and, if dense enough, can cause blindness.
Collagen	The insoluble structural protein making up the bulk of connective tissue. It is composed of a triple helix of strong fibres.
Conjunctivitis	Inflammation of the conjunctiva of the eye characterised by redness and often accompanied by a discharge.
Cutaneous	Of the skin.
Dermis	A layer of skin below the epidermis that consists of connective tissue and contains nerve endings, sweat and sebaceous glands and blood and lymph vessels.
DNA	A chemical made up of a linear sequence of different molecules called bases (adenine, thymine, cytosine and guanine) constituting the genetic material of organisms. There are four bases and the permuted sequence of these is read as a code which determines the composition and properties of the organism. The simplest organisms such as bacteria have nearly five million bases in their genetic material; humans have more than three-hundred million bases.

Elastin	A protein similar to collagen that is the principal structural component of elastic fibres.
Emission spectrum	The spectrum of bright lines, bands or continuous radiation characteristic of, and determined by, a specific emitting substance subjected to a specific kind of excitation.
Epidemiology	The study of factors affecting health and illness of populations, regarding the causes, distribution and control.
Erythema	A redness of the skin.
Fitzpatrick skin type	A numerical classification scheme for the colour of skin, developed in 1975 by T B Fitzpatrick, as a way to classify the response of different types of skin to UV radiation. It measures several components: genetic disposition, reaction to sun exposure and tanning habits.
Fluorescence	The emission of electromagnetic radiation, particularly visible light, which is stimulated when a substance absorbs incident radiation.
Heterogeneous	Consisting of elements that are not of the same kind or nature.
Hydroxylation	The chemical process that introduces one or more hydroxyl groups (-OH) into a compound, thereby oxidising it.
Immune system	Complex physiological mechanisms consisting of specialised cells and organs that have evolved to defend the body against attacks by foreign invaders.
Immunomodulator	An agent that alters the immune system response either by suppression by or enhancement.
Immunosuppression	Suppression of the immune response.
Intentional exposure	Trying to achieve a tan by lying in the sun, or in a sunbed/booth with minimal clothing, to maximise skin exposure.
Irradiation	The process by which an item is exposed to radiation, either intentionally or accidentally.
Isomerisation	The process by which a molecule is transformed into different form or configuration (which may have different properties) by a rearrangement of its atoms.
Keratinocyte	The major cell type of the epidermis that constitutes approximately 90% of epidermal cells.
Lentigo	A flat brownish pigmented spot on the skin, due to increased deposition of melanin. When malignant, it is known as ‘Lentigo maligna’. Acral lentigos are more common in dark-skinned individuals, but may be present in light-skinned individuals.
Malignant	Synonymous with cancerous. Malignant neoplasms or tumours can invade and destroy other tissues and spread to other parts of the body via the bloodstream or lymphatics (metastasis).
Median	The middle value in a distribution.
Melanin	Group of black, dark-brown, or reddish pigments present in the skin. Produced in melanocytes and stored in melanosomes.
Melanocortins	A group of pituitary peptide hormones that act through a multitude of specific receptors.

Melanocyte	Dendritic clear cell of the epidermis that synthesises the pigment melanin.
Melanocytic naevi	A small dark spot on the skin formed mainly from melanocytes, also known as a mole. It can be either subdermal (composed of melanin) or pigmented growth on the skin.
Melanoma	A tumour that arises in the melanocyte system of the skin and other organs (the cells that produce pigment) and that may spread rapidly to other parts of the body if not diagnosed and treated early. A melanoma may begin as a mole. Melanomas are the most dangerous type of skin cancer and the main cause of death from skin cancer.
Meta-analysis	A statistical method that combines the results of several studies addressing a set of related research hypotheses, to achieve a more accurate data analysis. It is widely used in epidemiological studies.
Metalloproteinase	An enzyme that conducts proteolysis and contains a metal in its active site.
Metastasis	The spread of a disease from one organ or part to a non-adjacent organ or part. Only malignant tumour cells and infections have the capacity to metastasise.
Minimum erythemal dose (MED)	The UV radiation dose that produces a just noticeable erythema on previously unexposed skin.
Morbidity	The incidence or prevalence of a disease.
Mutation	A permanent transmissible change in the genetic material, which may alter a characteristic of an individual or manifest as disease.
Naevi	A sharply circumscribed and chronic lesion of the skin, commonly named birthmarks and moles. Naevi are by definition benign. They differ from lentigos by the presence of nests of melanocytes, which are absent in lentigos.
Non-melanoma skin cancer (NMSC)	A malignant growth of the external surface or epithelial layer of the skin that most often originates from the external skin surface as a squamous cell carcinoma or a basal cell carcinoma. It is the most common form of cancer in the UK.
Phenotype	The entire physical, biochemical and physiological characteristics of an organism, as determined by both genetic make-up and environmental influences.
Photoageing	Premature and accelerated ageing of the skin as a result of excessive exposure to UV radiation. Effects of photoageing on the skin include dryness, loss of elasticity, wrinkles, discolouration and changes in texture.
Photokeratitis	A burn of the cornea by UVB rays, which may also be known as snow blindness. The condition can also be caused by artificial UVB sources, such as sunbeds or a welder's arc.
Pigmentation	The deposition of colouring matter or the colouration or discolouration of a part by a pigment.
Pruritus	Severe itching, often of undamaged skin.
Psoriasis	A chronic, non-contagious disease characterised by red inflamed lesions covered with silvery-white scabs of dead skin.
Pterygium	An abnormal wing-like mass of tissue arising from the conjunctiva of the inner corner of the eye that obstructs vision by growing over the cornea.

Purine	An organic compound that is the fundamental form of the purine bases in DNA and RNA including adenine and guanine.
PUVA	A type of phototherapy that combines the oral or topical photosensitising chemical psoralen with long-wave UVA.
Pyrimidine	An organic compound that is the fundamental form of the pyrimidine bases in DNA and RNA, including uracil, cytosine and thymine.
Rickets	A disease predominantly caused by a vitamin D deficiency. It most commonly occurs in children suffering from severe malnutrition, although it can occur in adults. It results in the softening of bones, which in children potentially leads to fractures and deformities.
Solaria	Commercial establishments that contain one or more sunbed units.
Squamous cell carcinoma (SCC)	Scaly or ulcerative malignant tumour derived from squamous cells, which are constituents of the skin and line the upper aerodigestive tract. Other organ cavities can develop squamous epithelium as a precursor to malignant change. Skin squamous carcinomas can closely resemble basal cell carcinomas, but are much more likely to spread and metastasise. The majority can be cured, but where spread has taken place this is much more difficult and the tumours can on occasion prove ultimately to be lethal.
Standard erythemal dose (SED)	A measure of erythemal UV radiation equivalent to an erythemal effective radiant exposure of 100 joules per metre squared ($J\ m^{-2}$).
Sunbed	An electrically powered appliance or installation intended to produce tanning of the human skin by utilising UV radiation.
Sunbed operator	A person or corporation having ultimate control and management of one or more sunbeds in a commercial establishment.
Sun protection factor	The ratio of the least amount of UV radiation required to produce a minimal erythema on sunscreen protected skin to the amount of energy required to produce the same erythema on unprotected skin.
Sunscreen	A topical preparation, commonly a lotion or cream, used to protect the skin from UV radiation.
Syngeneic	Genetically identical or related closely enough to allow tissue transplantation.
Tumorigenesis	The process of initiation and the progression of a tumour.
Tyrosinase	A copper-containing enzyme that catalyses the oxidation of phenols. It catalyses the production of melanin from tyrosine by oxidation.
Ultraviolet (UV) radiation	Electromagnetic radiation in the wavelength range 100–400 nm.
UVA radiation	UV radiation in the long wavelength range 315–400 nm. Not significantly filtered by the atmosphere. Approximately 97% of UV radiation that reaches the Earth's surface.
UVB radiation	UV radiation in the medium wavelength range 280–315 nm. Approximately 3% of UV radiation that reaches the Earth's surface.
UVC radiation	UV radiation in the short wavelength range 100–280 nm. All solar UVC radiation is absorbed by the ozone layer.

UV radiation dose	The amount of UV radiation to which a person is exposed. The UV radiation dose depends on the intensity of UV radiation and exposure time. It is expressed in joules per metre squared (J m^{-2}). In general, the greater the dose, the greater the likelihood of an effect.
Vitamin D	Helps to form and maintain strong bones. It is found in food – in particular in fish, milk, and dairy products – and can also be made by the body after exposure to UV radiation. A deficiency of vitamin D leads to decalcified bones and the development of rickets. Suboptimal vitamin D levels can also result in bone frailty in the elderly and have also been associated with increased cardiovascular risk, muscle weakness and an increased risk of some cancers and autoimmune disease.
Wavelength	The distance between two similar and successive and successive points on an alternating wave. The unit for optical radiation is the nanometre (nm) or 10^{-9} m.
Xeroderma pigmentosum	A rare hereditary skin disorder caused by a defect in the enzymes that repair DNA damaged by UV radiation. It results in hypersensitivity to the carcinogenic effect of UV radiation.

APPENDIX B

PHOTOSENSITISING MEDICINES AND AGENTS

Photosensitising agents* can result in two types of reaction – phototoxic and/or photoallergic.

Phototoxic reactions result from direct damage to tissue from activation of the agent by exposure to UV radiation. The process is usually acute and the skin's appearance can resemble sunburn. UVA radiation is most commonly associated with phototoxicity; however, UVB and visible light can also contribute to this reaction. The reactions usually improve once the medicines have been discontinued and cleared from the body.

Photoallergic reactions are a cell-mediated immune response in which the antigen is the light-activated photosensitising agent. The reactions usually resemble eczema and are generally chronic. They tend to be less common than phototoxic reactions and are commonly caused by topical agents.

Phototoxic drugs	*Common examples include:*
Antibiotics	the quinolones – eg ciprofloxacin, levofloxacin
	tetracyclines – eg tetracycline, doxycycline
	sulphonamides – eg sulphamethoxazole and trimethoprim; cotrimoxazole, sulphamethoxazole
	metronidazole
Antihistamines	diphenhydramine
Malaria medications	quinine
	chloroquine
	hydroxychloroquine
Cancer chemotherapy drugs	5-fluorouracil
	vinblastine
	dacarbazine
Cardiac drugs	amiodarone
	nifedipine
	quinidine
	diltiazem
Diuretics	furosemide
	thiazides – eg hydrochlorothiazide
Diabetic drugs	sulphonylureas – eg chlorpropamide, glyburide

* http://www.medicinenet.com/sun-sensitive_drugs_photosensitivity_to_drugs/article.htm

Painkillers	non-steroidal anti-inflammatory drugs – eg naproxen, piroxicam
Skin medications	photodynamic therapy for skin cancer – eg ALA or 5-aminolevulinic acid, methyl-5-aminolevulinic acid
Acne medications	isotretinoin
	acitretin
Psychiatric drugs	phenothiazines – eg chlorpromazine
	tricyclic antidepressants – eg desipramine, imipramine

Photoallergic drugs	*Common topical photoallergic agents include:*
Sunscreens	para-aminobenzoic acid (PABA)
	oxybenzone
	cyclohexanol
	benzophenones
	salicylates
	cinnamate
Anti-microbials	chlorhexidine
	hexachlorophene
	dapsone
Painkillers	celecoxib
Cancer chemotherapy drugs	5-fluorouracil
Fragrances	musk
	6-methylcoumarine

APPENDIX C

SECTIONS FROM THE STANDARD CONDITIONS FOR SPECIAL TREATMENT PREMISES ISSUED BY ISLINGTON COUNCIL

PUBLIC PROTECTION DIVISION

222 UPPER STREET LONDON N1 1XR

STANDARD CONDITIONS FOR SPECIAL TREATMENT PREMISES

London Local Authorities Act 1991 - 2000

GENERAL

These conditions apply to all premises for which a special treatment licence has been granted.

LICENCE

1. The licence is personal to its holder. The licence cannot be transferred by the licence holder to any other person unless the licence holder has followed the procedures for transfer prescribed in Part II of the Act and the Council's Rules Governing Applications.
2. The licence is only valid in respect of the premises named on the licence. The licence cannot be transferred by the licence holder to any other premises unless the licence holder has followed the procedures for transfer prescribed in Part II of the Act and the Council's Rules Governing Applications.
3. Licences are normally granted for a maximum period of twelve months. This period is either from 1 April to 31 March or 1 October to 30 September.
4. The establishment specified in the licence may only carry out treatments which are specified on the licence. If any alteration is required an application for the variation of the licence must be made to the Council in the manner specified in the Council's Rules Governing Applications.
5. The licensee, if a sole proprietor or a partnership, shall at once notify the Council in writing of any change in the name or private address of the licensee(s) or if a company within the meaning of the Companies Act 1985, or any Act amending the same shall forthwith notify the Council in writing of any change in the registered office address or in the constitution of the directorate of such company during the currency of this licence.
6. Proposed changes in the name, title or style of the premises licensed as a special treatment establishment shall be notified to the Council and shall not be put into effect until an amended licence is issued. This can only be carried out where there is no change in any other circumstances other than for example the name or title of the premises. If any other circumstances change for example the type of treatment offered, or the address of the premises, an application for a variation or transfer of licence is required.
7. The licence shall be displayed in a prominent position within the licensed premises at all times.

ULTRA VIOLET TANNING EQUIPMENT: SUNBEDS AND TANNING BOOTHS

57. The licensee shall draw up a schedule of maximum exposure times based on information supplied by the manufacturer and the operator shall advise clients of suitable exposure levels to avoid over-exposure particularly during initial sessions.

58. Warning notices and guidance notes approved by the Council shall be clearly displayed near the machine informing users of the equipment of the dangers of over-exposure.

59. Suitable goggles for the protection of the eyes of users of the equipment must be provided and each user must be advised of the possible dangers of failing to properly protect the eyes from ultra violet light. No user of the equipment should be allowed to undertake treatment without such protection.

60. Records must be kept of the hours of use of each machine and these records shall show when the tubes are replaced. Tubes must be replaced at intervals recommended by the manufacturer, together with the ultra violet transmitting plastic sheet if fitted.

61. Ultra violet lamps should be effectively protected from persons coming into contact with the lamps. Suitable means of achieving this protection would be the completion of the lamp enclosure with ultra violet radiation transmitting material, embedding the lamps within reflectors, or by covering with a grille or mesh. The protection should be of adequate mechanical strength which should not be impaired through repeated exposure to ultra violet radiation. In the case of lamps that might explode, the protection should be capable of containing fragments.

62. Only replacement tubes completely compatible with those supplied by the manufacturer of the appliance and of the same spectral output and energy emission as the original equipment fitted shall be used.

63. A suitable readily identified emergency device shall be fitted within easy reach of a person using the equipment. The device, when operated, should switch off ultra violet lamps, summon assistance, and where an upper canopy or door is electrically operated raise or open the canopy or door. Canopies/doors not electrically operated must rise/open freely.

64. The surface of the bed/booth must be cleansed after each use with a suitable cleanser as recommended by the manufacturer of the appliance, or covered with a disposable impervious film which is changed between each client.

65. Prospective users of the equipment shall be asked to complete a confidential questionnaire before using the appliance to establish whether any conditions exist which would indicate that use of the appliance could have an adverse effect on the health or safety of the user.

66. An automatic timer shall be fitted to the equipment and shall be of good quality with an accuracy of + - 10% and shall be such that the user is unable to increase the duration of treatment.

67. Equipment must be situated in a suitable room or cubicle and so positioned that adequate ventilation and cooling is provided, so that the temperature rise in the enclosure due to the operation of the equipment does not exceed 5°C.

68. These conditions, in so far as they relate to matters of health, hygiene and safety, are subject to amendment in accordance with any change in the requirements of the relevant statutory provisions or on the recommendation of the Health and Safety Executive.

APPENDIX D

COMMITTEE ON MEDICAL ASPECTS OF RADIATION IN THE ENVIRONMENT

CHAIRMAN

Professor A Elliott BA PhD DSc CPhys FInstP FIPEM
Western Infirmary, Glasgow

DEPUTY CHAIRMAN

Professor R Waters BSc PhD DSc
Pathology Department
University of Wales College of Medicine, Cardiff

PRESENT MEMBERS

Professor T C Atkinson BSc PhD
Department of Earth Sciences
University College London

Dr H R Baillie-Johnson MB BS FRCR FRCP
Department of Oncology
Norfolk and Norwich University Hospital

Professor R Dale MSc PhD FInstP FIPEM FRCR(Hon)
Radiation Physics & Radiobiology
Imperial College, Charing Cross Hospital, London

Dr C J Gibson BA MSc PhD FIPEM
Churchill Hospital
Oxford

Professor S V Hodgson BM BCh DM FRCP
Department of Clinical Development Sciences
St George's University of London

Professor P Jeggo BSc PhD
Genome Damage and Stability Centre
University of Brighton

Dr G Maskell MA FRCP FRCR
Department of Radiology
Royal Cornwall Hospital, Truro

Professor M D Mason MD FRCP FRCR
Oncology and Palliative Medicine
University of Wales College of Medicine

Dr C D Mitchell PhD FRCP
Paediatric Haematology/Oncology Unit
John Radcliffe Hospital, Oxford

Dr M Murphy BA MB BChir MSc FFPH
Childhood Cancer Research Group
Oxford

Dr R A Shields MA MSc PhD FIPEM
Medical Physics Department
Manchester Royal Infirmary

Professor I Stratford BSc PhD
School of Pharmacy & Pharmaceutical Sciences
University of Manchester

Dr J Verne BSc MSc MB BS PhD FFPH
Regional Public Health Group
Government Office for the South West (Bristol)

Professor R Wakeford BSc PhD CSci CPhys FInstP CStat CEng MINucE CRadP FSRP
Dalton Nuclear Institute
University of Manchester

FORMER MEMBERS WHO SERVED DURING THE PREPARATION OF THIS REPORT

Professor G McKenna BSc MD PhD FRCR FMedSci
Department of Radiation Oncology and Biology
Churchill Hospital, Oxford (Until February 2009)

Professor P McKinney BSc PhD MFPHM(Hon)
Paediatric Epidemiology Group
University of Leeds (Until March 2009)

Professor E Wright HNC BSc PhD CBiol MIBiol MRCPath FRCPath
Department of Molecular and Cellular Pathology
University of Dundee (Until March 2007)

SECRETARIAT

Dr R Hamlet BSc PhD CBiol MIBiol (Scientific – *until February 2009*)

Mr S Ebdon-Jackson BSc MSc HonFRCP (Scientific)

Dr E Petty BSc PhD (Scientific and Minutes)

Dr K Broom BSc DPhil CBiol MIBiol (Minutes)

Miss K Stonell (Minutes)

Miss J Kedward (Administrative)

ASSESSORS IN ATTENDANCE REPRESENTING THE FOLLOWING ORGANISATIONS

Department for Children, Schools and Families

Department for Communities and Local Government

Department of Energy and Climate Change

Department of the Environment, Food and Rural Affairs

Department of Health

Department of Health, Social Services and Public Safety (Northern Ireland)

Department for Innovation, Universities and Skills

Environment Agency

Food Standards Agency

Health Protection Agency – Radiation Protection Division

Health and Safety Executive

Information Services Division, NHS Scotland

Medical Research Council

Ministry of Defence

Nuclear Decommissioning Authority

Office for National Statistics

Scottish Environment Protection Agency

Scottish Government

Welsh Assembly Government

UV SUNBEDS SUBCOMMITTEE

CHAIRMAN

Professor R Waters BSc PhD DSc
Pathology Department
University of Wales College of Medicine, Cardiff

MEMBERS

Dr H R Baillie-Johnson MB BS FRCR FRCP
Department of Oncology
Norfolk and Norwich University Hospital

Dr J Verne BSc MSc MB BS PhD FFPH
Regional Public Health Group
Government Office for the South West (Bristol)

Professor R Mackie CBE FRSE MD DSc
Department of Public Health and Health Policy
University of Glasgow

Professor B Diffey BSc AKC PhD DSc
Dermatological Sciences, Institute of Cellular Medicine
Newcastle University

Professor J Newton-Bishop MD FRCP
Section of Epidemiology and Biostatistics
Leeds Institute of Molecular Medicine
University of Leeds

Ms J Lockhart
Patient Representative

Ms S Woolnough
Cancer Research UK

Dr S Gupta
Department of Health

ASSESSORS

Mr G Hooker
Department of Health

Mr C Brereton
Welsh Assembly Government

Dr A Johnston
Scottish Government

Mr N McMahon
Department of Health, Social Services and Public Safety (Northern Ireland)

Ms J Morris
Chartered Institute of Environmental Health)

SECRETARIAT

Dr E Petty

Dr K Broom

Mr A Pearson

APPENDIX E

DECLARATION OF MEMBERS' INTERESTS CODE OF PRACTICE

1 Introduction

This code of practice guides members of COMARE as to the circumstances in which they should declare an interest in the course of the Committee's work.

To avoid any public concern that commercial interests of members might affect their advice to Government, Ministers have decided that information on significant and relevant interests of members of its advisory committees should be on the public record. The advice of the Committee frequently relates to matters which are connected with the radiation industry generally and, less frequently, to commercial interests involving radioactivity. It is therefore essential that members should comply with the code of practice which is set out below.

2 Scope and definitions

This code applies to members of COMARE and its subcommittees, subgroups, working groups and working parties which may be formed.

For the purposes of this code of practice, the 'radiation industry' means:

(a) companies, partnerships or individuals who are involved with the manufacture, sale or supply of products processes or services which are the subject of the Committee's business. This will include nuclear power generation, the nuclear fuel reprocessing industry and associated isotope producing industries, both military and civil and also medical service industries;

(b) trade associations representing companies involved with such products;

(c) companies, partnerships or individuals who are directly concerned with research or development in related areas;

(d) interest groups or environmental organisations with a known interest in radiation matters.

This excludes government departments, professional bodies, international organisations and agencies.

It is recognised that an interest in a particular company or group may, because of the course of the Committee's work, become relevant when the member had no prior expectation this would be the case. In such cases, the member should declare that interest to the Chairman of the meeting and thereafter to the Secretariat.

In this code, 'the Department' means the Department of Health, and 'the Secretariat' means the secretariat of COMARE.

3 Different types of interest – definitions

The following is intended as a guide to the kinds of interests which should be declared. Where a member is uncertain as to whether an interest should be declared they should seek guidance from the Secretariat or, where it may concern a particular subject which is to be considered at a meeting, from the Chairman at that meeting. Members of the Committee and the Secretariat are under no obligation to search out links between one company and another, for example where a company with which a member is connected has a relevant interest of which the member is not aware and could not reasonably be expected to be aware.

If members have interests not specified in these notes but which they believe could be regarded as influencing their advice they should declare them to the Secretariat in writing and to the Chairman at the time the issue arises at a meeting.

3.1 *Personal interests*

A personal interest involves current payment to the member personally. The main examples are:

(a) *Consultancies and/or direct employment:* any consultancy, directorship, position in or work for the radiation industries which attracts regular or occasional payments in cash or kind.

(b) *Fee-paid work:* any work commissioned by those industries for which the member is paid in cash or kind.

(c) *Shareholdings:* any shareholding in or other beneficial interest in shares of those industries. This does not include shareholdings through unit trusts or similar arrangements where the member has no influence on financial management.

(d) *Membership or affiliation:* any membership role or affiliation that the member or close family member has to clubs or organisations with an interest or involvement in the work of the Department. This will not include professional bodies, organisations and societies.

3.2 *Non-personal interests*

A non-personal interest involves current payment which benefits a department to which a member is responsible, but is not received by the member personally. The main examples are:

(a) *Fellowships:* the holding of a fellowship endowed by the radiation industry.

(b) *Support by industry:* any payment, other support or sponsorship by the radiation industry which does not convey any pecuniary or material benefit to a member personally but which does benefit their position or department, eg:

(i) a grant from a company for the running of a unit or department for which a member is responsible;

(ii) a grant or fellowship or other payment to sponsor a post or a member of staff in a unit or department for which a member is responsible. This does not include financial assistance for students, but does include work carried out by postgraduate students and non-scientific staff, including administrative and general support staff.

(iii) the commissioning of research or work by, or advice from, staff who work in a unit or department for which a member is responsible.

(c) *Support by charities and charitable consortia:* any payment, other support or sponsorship from these sources towards which the radiation industry has made a specific and readily identifiable contribution. This does not include unqualified support from the radiation industry towards the generality of the charitable resource.

(d) *Trusteeships:* where a member is trustee of a fund with investments in the radiation industry, the member may wish to consult the Secretariat about the form of declaration which would be appropriate.

3.3 *Specific interests*

A specific interest relates explicitly to the material, product, substance or application under consideration by the Committee.

A member must declare a personal, specific interest if they currently receive a payment, in any form, for any significant fundamental development work undertaken previously or at this time, on a material, product or substance or its application under consideration. This will include the production of radioactive substances and devices designed to use ionising or non-ionising radiation for diagnostic, treatment or other purposes.

A member must declare a non-personal, specific interest if they are aware that the department to which they are responsible currently receives payment for significant fundamental development work undertaken previously or at this time, on a material, product or substance or its application under consideration but they have not personally received payment for that work in any form. This will include the production of radioactive substances and devices designed to use ionising or non-ionising radiation for diagnostic, treatment or other purposes.

3.4 *Non-specific interests*

A non-specific interest relates to a company or associated material, product, substance or application, but not to the specific material, product, substance or application under consideration by the Committee.

A member must declare a personal, non-specific interest if they have a current personal interest with a material, product, substance or application from a particular company, which does not relate specifically to the material, product, substance or application from that company under consideration.

A member must declare a non-personal, non-specific interest if they are aware that the department to which they are responsible is currently receiving payment from the company concerned which does not relate specifically to a material, product, substance or application under discussion.

If a member is aware that a material, product, substance or their application under consideration is or may become a competitor of a material, product or substance manufactured, sold or supplied by a company in which the member has a current personal interest, they should declare their interest in the company marketing the rival material, product or substance.

Members are under no obligation to seek out knowledge of such work done for or on behalf of the radiation industry within departments to which they are responsible if they would not reasonably expect to be informed. This applies to all non-personal, specific and non-specific interests.

4 Declaration of interests

4.1 *Declaration of interests to the Secretariat*

Members should inform the Secretariat in writing when they are appointed of their current personal and non-personal interests and annually in response to a Secretariat request. Only the name of the company (or other body) and the nature of the interest is required; the amount of any salary, fees, shareholding, grant, etc, need not be disclosed. An interest is *current* if the member has a continuing financial involvement with the industry, eg if they hold shares in a radiation company, have a consultancy contract, or if the member or the department to which they are responsible is in the process of carrying out work for the radiation industry. Members are asked to inform the Secretariat at the time of any change in their personal interests, and may be invited to complete a form of declaration when required. It would be sufficient if changes in non-personal interests are reported at the next annual declaration following the change. (Non-personal interests involving less than £5000 from a particular company in the previous year need not be declared.)

The register of interests should be kept up-to-date and be open to the public.

4.2 *Declaration of interests at meetings and participation by members*

Members are required to declare relevant interests at Committee meetings and to state whether they are personal or non-personal interests. The declaration should include an indication of the nature of the interest.

(a) If a member has a current (personal or non-personal) interest in the business under discussion, they will not automatically be debarred from contributing to the discussion subject to the Chairman's discretion. The Chairman will consider the nature of the business under discussion and of the interest declared (including whether it is personal or non-personal) in deciding whether it would be appropriate for the relevant member to participate in the item.

(b) If a member has an interest which is not current in the business under discussion, this need not be declared unless not to do so might be seen as concealing a relevant interest. The intention should always be that the Chairman and other members of the Committee are fully aware of relevant circumstances.

A member, who is in any doubt as to whether they have an interest which should be declared, or whether to take part in the proceedings, should ask the Chairman for guidance. The Chairman has the power to determine whether or not a member with an interest shall take part in the proceedings.

If a member is aware that a matter under consideration is or may become a competitor of a product, process or service in which the member has a current personal interest, they should declare the interest in the company marketing the rival product. The member should seek the Chairman's guidance on whether to take part in the proceedings.

If the Chairman should declare a current interest of any kind, they should stand down from the chair for that item and the meeting should be conducted by the Deputy Chairman or other nominee if the Deputy Chairman is not there.

4.3 *Members' declarations of interests – 2009*

Member	Company	Personal interest	Company	Non-personal interest
Prof T C Atkinson		None	UKAEA	Consultancy
Dr H R Baillie-Johnson		None		None
Prof R Dale				
Prof A Elliott		None		None
Dr C J Gibson		None		None
Prof S V Hodgson		None	CR-UK	Support for research
Prof P Jeggo				
Dr G Maskell		None		None
Prof M D Mason		None		None
Dr C D Mitchell		None		None
Dr M Murphy		None		None
Dr R A Shields		None		None
Prof I Stratford	1 Oxford Biomedica	Shares		None
	2 Astrazeneca	Grants and consultancy		
	3 UCB/Celltech	Grants		
Dr J Verne		None		None
Prof R Wakeford	1 Sellafield Ltd	Consultancy		
	2 Compensation Scheme for Radiation-linked Diseases	Consultancy		
	3 Canadian Nuclear Safety Commission	Contract		
Prof R Waters		None		None